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Snook, R.R., Bretman, A., Dougherty, L.R. et al. (2026) The consequences of rising temperatures for animal fertility. Nature Reviews Biodiversity. ISSN: 3005-0677

https://doi.org/10.1038/s44358-026-00142-4

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 sub-lethal 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 shape thermal sensitivity. Fertility-based thermal limits can predict species distributions and extinction risk better than survival-based measures, although tested across a limited taxonomic range. Evolutionary responses to fertility loss under warming appear constrained but increased mutational variation, local adaptation and hybridization may increase fertility resilience. Key research priorities include broader taxonomic evaluation of evolutionary potential and ecological outcomes, including population effects under multiple stressors and impacts on community and ecosystem dynamics as fertility-sensitive taxa shift range or go extinct. Recognising and addressing fertility-based vulnerability is essential for anticipating biodiversity change and designing more effective responses to climate impacts.

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 **heatwaves**[1]. While much attention has focused on lethal impacts of warming[2] reproductive processes are well-known to be negatively impacted at sub-lethal thermal stress in both sexes[3]. The thermal range over which **fertility** and **fecundity** (hereafter also referred to holistically as **reproductive output**) is possible is typically narrower than the survival range[4] and can fail at temperatures that do not affect survival[5,6]. While fertility and fecundity are major drivers of population persistence, these non-lethal reproductive effects remain underrepresented in predictions of species vulnerabilities and responses to climate change[3]. Recent measures of upper **Thermal Fertility Limits** (TFLs)[7] (**Box 1)** better match current species ranges and predict temperature-related extinction[5,6], and so are likely of critical importance to biodiversity. Moreover, societal impacts are potentially far-reaching as heat-induced sterility occurs in livestock[8], pollinators[9], and even humans[10] (**Fig. 1**). Thus, the impact of temperature-mediated effects on fertility and fecundity merit closer examination given the 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, fertilisation 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, fertilisation and fecundity. We highlight factors influencing variation in thermal impacts on reproduction, such as sex, fertilisation mode, and exposure duration and how **phenotypic plasticity** may mediate responses. To gauge potential impacts on biodiversity, we assess the ecological and evolutionary consequences of thermal limits to fertility, including on predictions of species re-distribution, extinction risk, and sexual selection and the potential for adaptive evolutionary responses to increased warming. Finally, we outline research priorities for better predicting the extent to which thermally-induced reproductive decline will impact biodiversity under ongoing climate change. Although temperature also alters traits that influence reproductive success - such as mating behaviour[11], reproductive timing/ phenology[12] or parental care[13] – our emphasis is on thermal effects on parental fertility and fecundity. Temperature can also influence post-fertilisation mortality of embryos or offspring with experiments frequently not disentangling effects of heat stress on parental fertility and fecundity from offspring viability[14]. To minimize this problem, we highlight studies where gametes or parents, rather than embryos or offspring, experienced heat stress.

Patterns of heat-induced sterility

Research on thermal impacts on fertility is dominated by terrestrial invertebrates (particularly insects) and laboratory animal models[14] (**Supplementary Table1**). Nevertheless, there are examples from amphibians[15] (**Fig. 1A**), reptiles[16](**Fig.** 1B), birds[17] (**Fig. 1C**) and wild mammals[18,19] (**Fig.** 1**D**). There has also been focus on economically important livestock[8] (**Fig. 1E**; **Supplementary Table1**). Even human sperm quality is declining, some of which is epidemiologically linked to temperature variability[10] (**Fig. 1F**). Conservation and captive breeding programs may also be at risk when animals are kept outside at ambient temperatures due to the negative thermal effects on fertility and fecundity[20]. Warming can affect pollinators by reducing sperm viability[21], which is compounded by mated queens storing sperm for months or years[9]. Conversely, heat suppression of fertility and fecundity in pests or disease vectors may offer economic benefits[22].

However, responses are heterogenous (**Fig.1**) – temperature-induced changes in fecundity and fertility may be positive, negative or absent depending on a variety of factors such as species, sex, and life stage and type of thermal exposure(e.g. constant, fluctuating, extreme). For example, in the mussel, *Mytilus galloprovincialis*, warmer temperature increases fertilization rate[23] whereas the opposite occurs in the clam, *Tridacna maxima*[24]. Both sexes may be negatively impacted (**Fig. 1C**), or one sex may be more susceptible. Effects on fecundity may result from complex interactions with species-specific ecological traits (**Fig. 1D**) or life history traits, such as body size[16] but see[25,26]. Higher winter temperatures can also impact spring fecundity, such as in the wood frog, *Rana sylvatica*[15] (**Fig. 1A**). Predicting whether fertility and fecundity *per se* is negatively impacted, and for which species under what thermal conditions, remains challenging, especially given different approaches to measure the impacts of heat stress (**Box 1)**[7,27,28].

Mechanisms of heat-induced fertility loss

High temperature stress damages fertility and fecundity through diverse physiological and molecular pathways, including impacts on: gamete quantity and quality; reproductive fluids; sperm storage; rate of fertilisation; 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 is vitally important for predicting fertility resilience, conservation, human health, and protection of agriculture, along with the potential for fertility recovery, 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 slower[38-40], have reduced viability[29,40-42], and sperm and eggs that exhibit more DNA damage[34,37,43]. In some species, brief heat shock causes immediate and long-lasting male sterility, consistent with irreparable damage to spermatogenesis (**Supplementary Table 1**). In other species, males remain fertile initially but become sterile days later[5]. 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 with by sperm that were developing during the heat stress, leading to a delayed onset of sterility[5,44,45](**Supplementary Table 1**).

Heat stress can induce oxidative stress through disruption of mitochondrial function, generating Reactive Oxygen Species (ROS). Increase in ROS 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 *D. melanogaster* laboratory lines, fertility sensitivity to heat stress was correlated with oxidative stress effects on lifespan[47], and these patterns were mirrored at the genetic level, where susceptibility to heat-induced male sterility was associated with genes involved in DNA damage repair and autophagy[48]. Similarly, DNA damage was linked to heat stress effects on spermatogenesis in *Caenorhabditis elegans*, but notably not oogenesis[43]. ROS and heat stress can also impact transposable element activity[43,49], which affects sex-specific gene regulation, and can generate novel genetic and phenotypic variation[50]. In *D. subobscura*, heat stress resulted in different transposable element expression patterns between the testes and ovaries[51] although whether this difference is associated with reduced fertility and DNA damage is unknown. Links between ROS 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.

Reproductive fluid effects

As well as gametes, both sexes produce reproductive fluid that impact fertilisation 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 increasing egg-laying[52]. 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 proteins[41]. In the lizard, *Tropidurus spinulosus*, female ovarian fluid mitigated negative temperature effects on sperm velocity, a trait mediating male fertilisation success[53]. In the fish, *Symphodus ocellatus,* warmer water decreases the extent to which ovarian fluid biases fertilisation towards high-performing sperm[54]. Thus, the potentially buffering roles of sex-specific reproductive fluid to high temperature may vary among taxa.

Sperm storage and fertilisation failure

In species that store sperm, heat stress may 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 *D. virilis*, female fertility is recovered following remating after heat stress, suggesting damage to previously stored sperm rather than to the female reproductive tract[55].

Even if sperm and eggs meet, heat stress can decrease fertilisation success[23,24] (**Fig. 2**; **Supplementary Table 1**). In *D. melanogaster*, developmentally heat-stressed males fertilised fewer eggs and, when fertilisation occurred, embryos were at an earlier stage of development compared to eggs fertilised by non-heat stressed males[56]. In mammals, *in vitro* heat stress increases sperm-egg fusion failure[57] and increases lethal **polyspermy**[58]. Increases in polyspermy were associated with decreased transcript abundance of a gene involved in preventing polyspermy[58].

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 pathways[59]. Hsps are essential for gametogenesis[60]. Transcriptome analysis of testes of pigs[61], mice[31], rats[36], rabbits[62], chickens[63] and octopus[64] and ovaries in rabbits[65] 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 there is sex-specific variation in the strength of transcriptional responses that underlies sex differences in heat-induced declines of reproductive output[40,48] is unknown. Male *D. subobscura* exhibited greater Hsp gene expression changes after acute heat exposure than females, which were associated with increased transposable element expression patterns between the testes and ovaries although this was not directly linked to effects on fertility[51]. Mechanistically, Hsp70 drives transposable element activation in gonads under heat stress in *D. melanogaster*[49]. Hsps in females from monogamous species or those that store sperm for long periods, such as in honeybees, may protect stored sperm. For example, honeybee females increase expression of Hsps in their spermathecae after heat shock[9]. Likewise, anti-oxidative responses in females could be expected to protect stored sperm from heat stress. However, no increase in enzymes functioning in anti-oxidative stress or in GAPDH, which helps maintain stored sperm, was found in heat-shocked honeybee females[9]. Whether regulation of Hsps relates to species differences in thermal responses for fertility and fecundity is unknown, though peak Hsp induction has been linked to differences in CT_{max} between populations of killifish[66] and *Drosophila* species[67].

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 expression changes even after six weeks in benign conditions[68]. In contrast, acute exposure elicited transient Hsp responses that returned to baseline within hours with reduced ovarian damage[68]. However, the link between the gene expression kinetics and reproductive damage is untested. Generally, upregulation of heat shock responses for an extended period is likely to be costly[69], and potentially suggestive of poor adaptation to the thermal environment[70]. Exposure regime may also alter Hsp expression. In killifish (*Austrofundulus limnaeus*) liver tissue, small Hsps altered gene expression more strongly to fluctuating temperatures, while larger chaperones like Hsp70 and Hsp90 were more active under chronic heat[71]. 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 may be caused by heat stress causing various types of epigenetic modifications (e.g., DNA or chromatin methylation or acetylation). Epigenetic 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. In both sexes, genes with methylation shifts were associated with sex-specific gametogenesis, although heat-stress effects on fertility and fecundity were not examined[72]. 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*[73]. In cows, heat stress in the F_0 generation leads to a reduced conception rate in F_1 daughters and increased stillbirths in F_3 great-granddaughters[74]. What epigenetic mechanisms caused 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 and population dynamics, such as sexual selection (**Box 2**). Partial or complete recovery following heat stress can occur in some species under some conditions[41,75-77] (**Supplementary Table 1**). In the insect pest, *Monochamus alternatus*, 15d heat stressed individuals showed full recovery of fecundity, sperm number and sperm viability within four weeks, although recovery time varied across traits[78]. Recovery was mediated in part by Hsp20, which appeared to help refold reproductive proteins during the recovery phase, suggesting that Hsps may also influence longer term recovery after heat stress (see subsection "*Conserved heat shock response")*.

Recovery potential may depend on the severity and duration of heat stress, the mechanism behind sterility, and the exposed life stage. For instance, adult exposure duration influenced recovery rates in female *D. melanogaster*, although fertility was never restored to control levels[32]. In this same species, the extent of recovery was reduced as temperatures increased, but only when heat stress was applied to the adult, not pupal, stage[29]. 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 for better predicting how thermal variability impacts 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 predict which organisms may be more at risk. Here we examine four factors that mediate thermal stress impacts on fertility and fecundity – sex-specific effects and its downstream consequences on sexual selection (**Box 2**), fertilisation mode, life history stage exposed, and exposure regime. We also consider how different types of phenotypic plasticity influence variation in reproductive output responses across these factors.

Sex-specific effects

A key outstanding question is whether fertility and fecundity is more sensitive to warming in males or females[79] 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 males[80]. However, most research has exposed either only one sex or both sexes simultaneously to heat stress, with the latter typically not delineating sex-specific effects as heat-stressed individuals are not mated to control mates[14]. 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 due to sperm being particularly heat sensitive[5,6,29,47,81] (see section "Mechanisms of heat-induced fertility loss"). However, a meta-analysis spanning 241 aquatic invertebrates and ectothermic vertebrates found that the sexes are responding similarly for temperature effects on fecundity and gamete traits[82]. In terrestrial organisms, sex differences for the impact of thermal stress on fecundity appear variable[83], being more negative for males[29,40,47,84] or females[17,85-87], or show no sex difference[22,88]. Determining the extent of sex-specific variation and what factors contribute to this variation is crucial given the impact on **operational sex ratio** (OSR), population growth and subsequent ecological and evolutionary consequences (e.g., **Box 2**; see section “Ecological and Evolutionary Impacts”).

Fertilisation mode

The mode of fertilisation alters the environment experienced by gametes, which could influence the impact of heat stress on fertility and fecundity. External fertilisers release gametes into the environment, directly exposing them to the abiotic environment, whereas this is avoided in internal fertilisers. Additionally, the sperm of internal fertilizers may be stored inside the female, raising the possibility that the female reproductive tract could protect sperm from heat stress, though this does not appear to be the case in some insects[9,55]. A recent meta-analysis examining effects of temperature (but not necessarily heat stress) on fertility and fecundity in aquatic external and internal fertilising invertebrates and ectothermic vertebrates found weak support for fertilisation mode moderating thermal fertility effects[82]. Whether stronger effects would be found when only using studies that applied thermal stress, or comparing fertilisation mode effects for terrestrial ectotherms that may experience greater thermal variation compared to aquatic ectotherms, is untested.

Life stage

Thermal performance curves (**Box 1**) vary across life cycle stages[89]. This ontogenetic variation can generate demographic bottlenecks, in which a vulnerable stage may constrain population growth, leading to overestimates of population resilience when only more tolerant life stages are tested[90]. In two insect species, heat stress during pupal and adult stages reduced subsequent offspring production more than exposure during earlier developmental stages[29,45,75,91]. In externally fertilizing fish, spawners are more heat sensitive, with lower **Thermal Safety Margins** (TSMs) than non-reproductives and juveniles[90]. The meta-analysis across aquatic invertebrates and ectothermic vertebrates found that the gamete stage was more thermally susceptible than when developing or adult individuals were exposed[82]. In contrast, the gamete stage in other taxa is more robust to thermal stress than later stages[23,92,93]. Determining the relative impact of thermal stress across the life cycle and why this varies across taxa will improve predictability of where and when demographic bottlenecks due to heat induced loss of fertility and fecundity can occur and effects on other evolutionary and ecological processes (e.g. **Box 2**)– key information relevant for conservation.

Exposure – duration, frequency and type

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 constant mean high temperature throughout development but fluctuating temperatures with the same mean reduced fertility in all lines[94]. A meta-analysis across 241 species of aquatic invertebrates and ectothermic vertebrates found that experimental increases in temperature had more negative effects on reproductive traits than natural thermal exposure, but exposure duration, magnitude of temperature increase, and whether temperature treatment was constant or fluctuating had no effect[82]. Ørsted and colleagues[85] used the Thermal Death Time approach (**Box 1**) to examine effects on both survival and reproductive output in *D. suzukii*. 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, generating more days in which high temperature would impair fecundity relative to survival.

Multiple stressors

While studies show thermal effects on reproductive output loss (**Supplementary Table 1**), climate change also modifies other abiotic factors like UV radiation (which may 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[95,96], humidity[97], pollutants[98] and salinity[23] have shown stressor interactions influence fertility and fecundity. For example, studies examining the effects of climate change on marine organisms often test the impact of both rising temperature and ocean acidification caused by higher dissolved CO_2 levels[99]. The combined effect of these two stressors can be complex, having negative synergistic effects on fertilisation success in oysters[100] and sperm swimming speed in mussels[23] whereas in the subarctic copepod, increasing acidification partly ameliorates the harmful effects of warming on fecundity[101]. Expanding studies that combine heat stress with other stressors is important for better increased 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 improves tolerance to subsequent extreme temperatures[102]. This can mitigate fertility loss in some species (e.g. *Drosophila*[69] but see[77]; sea urchins[103]). Second, heat exposure during development often impairs adult reproduction even when adults experience benign conditions[48,75,88,104] though such exposure sometimes enhances adult thermal tolerance[105,106]. Overall, however, most evidence indicates lasting negative consequences, with a meta-analysis finding no sex difference in acclimation capacity[107]. Third, parents exposed to heat can alter offspring phenotype via non-genetic mechanisms, a form of transgenerational plasticity that may be adaptive when environmental conditions persist across generations[108] (see section, "Mechanisms of heat-induced fertility loss"). Such plasticity improves offspring thermal resilience in some species[109,110] but reduces offspring reproductive output even when offspring develop under benign temperatures in other species[30,40]. Understanding the evolutionary impact of phenotypic plasticity on heat-induced loss of reproductive output requires additional research.

Ecological and evolutionary impacts

While thermal effects on fertility and fecundity are increasingly well-documented, the broader ecological and evolutionary impacts remain underappreciated. Here we explore how heat-induced reductions in fertility and fecundity can shape species distributions, extinction risk, sexual selection (**Box 2**) and how range shifts due to suppressed fertility and fecundity from heat stress may impact evolutionary and ecological responses.

Species distributions

Understanding how climate change will affect where species can live is a crucially important research question. Species distribution models (SDMs) predict where species currently live and incorporate thermal tolerance data in different ways: mechanistic SDMs use physiological limits directly , statistical or envelope SDMs infer limits from matching species' occurrence data with climate variables that are assumed to correlate with physiological limits, and hybrid models that combine the two[111]. This information can then be used to map areas of predicted suitable habitat for future climate change scenarios.

Whether fertility and fecundity limits (TFLs; **Box 1, Fig. 3**) are better predictors of current species distributions than survival limits (either lethal temperatures or CTmax; **Box 1, Fig. 3**), thereby providing better resolved data for forecasting climate change responses, has been tested in two separate studies, both using multiple *Drosophila* species[5,6]. Parratt and colleagues[5] compared TFL_{80} (the temperature rendering 80% of the population sterile after a 4-hour static heat shock) with LT_{80}(static 4-hour heat shock) and CT_{max} (ramping temperature). Across 43 species, over 25% had TFLs lower than survival limits the day after heat shock, rising to 44% seven days later (**Fig. 3A**). The temperature causing 80% of males to be sterile vs 80% to die was species-specific, spanning from 0ºC to 4.3ºC, changing the species rank of thermal tolerance and producing narrower estimates of each species' TSM (**Fig. 3B**). Despite being measured under laboratory conditions, TFL_{80} values improved predictions of current species distributions by over 35% compared to using CT_{max}, indicating the ecological relevance of this trait. This improvement occurred even when using TFL_{50} (**Box 1**), a result confirmed in study by van Heerwaarden and Sgrò[6], which used chronic and lower temperature conditions. These findings indicate many species live closer to a reproductive threshold than previously appreciated, making some species even more vulnerable to climate change than when considering survival data alone. While TFL_{80}, CT_{max} and LT_{80} were positively correlated across species, there was a weaker association between fertility and survival limits, suggesting distinct underlying processes. However, a TDT study on *D. suzukii* found that fertility and survival limits were strongly correlated, even though fertility failed at lower temperatures[85]. Together, both TFL and TDT approaches show 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 suggests negative consequences for population persistence, assuming evolutionary potential is limited (see section "Evolutionary Potential and Constraint"). Whether TFLs predict population extinction risk has only been directly tested in Drosophila[6]. Experimental evolution of six

Drosophila species under gradual warming (0.2ºC every two weeks) found that all species went extinct within 46 weeks, with tropical species going extinct sooner and thus at cooler temperatures than temperate species. Male TFLs best explained extinction probability compared to female TFLs, lethal temperatures, 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 29.2ºC[6] consistent with independent evidence that constant or fluctuating regimes at this developmental exposure severely reduces both male fertility and fecundity with low heritability for resistance[94]. Yet other *D. melanogaster* populations persist in regions that regularly exceed 29ºC[112] suggesting **local adaptation** via mechanisms that mitigate heat damage to reproductive output[76] (see section "Evolutionary Potential and Constraint").

Further insight comes from the sterile insect technique (SIT), in which mass release of sterile males, either through ionizing radiation or, more recently, through genetic manipulation, is used to suppress pest populations[113]. Population crashes can be rapid, with guidelines suggesting release of between 90 and 99% sterile males[113]. 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 – this could reduce the demographic cost of male sterility[114]. Moreover, as SIT eliminates reproduction for most males, this will produce extreme variance in male reproductive success, which could select for increased female discrimination against sterile males[115] (**Box 2**). Whether this evolutionary response could emerge under natural heat-induced male sterility is unexplored. The second factor is fertility recovery after heat stress[114] (see the Mechanisms subsection, "Fertility recovery after heat stress"). Demography will be negatively impacted when sterility is long-lasting relative to the organism's life span or irreversible.

Sympatric species and adaptive introgression

Species are shifting polewards in response to rising temperature[116] potentially bringing closely related species into secondary contact. If reproductive isolation barriers are incomplete, then such shifts into sympatry may result in increased gene flow and recombination arising from hybridization between taxa[117]. Hybridisation could have either negative, positive or a mix of fitness consequences.

Negative hybridization consequences can occur when increased sympatry generates **reproductive interference**[118], 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 more sensitive species, who may be sterile, this could negatively impact female reproductive success. On the island of São Tomé, two sister species, *D. santomea* and *D. yakuba*, exhibit different thermal fertility sensitivities and meet in a contact zone, generating some hybrids[119]. Because matings between *D. yakuba* females and the more thermally sensitive *D. santomea* males are more likely than the reciprocal[120], high temperatures could exacerbate reproductive interference by generating more (costly) hybrids. Increased hybridization could either result in reduced species barriers or select for **reinforcement** of stronger prezygotic barriers[121]. Experimental evolution with these two species showed reinforcement of prezygotic gamete barriers that reduced hybridization cost[122], but temperature was not used to change hybridization costs, leaving the role of differential thermal fertility between sympatric species an open question. In the marine tubeworm, *Galeolaria*, reproductive isolation is strong at cooler temperatures between two sympatric species but higher temperatures increase cross-fertilisation success[123]. Whether this hybridization is costly or beneficial is unknown.

Hybridization may be beneficial by allowing acquisition of thermal fertility resilience alleles from a more warm-adapted species. Such **adaptive introgression** may provide a route for rapid evolutionary change, although this could also blur species boundaries[124]. For example, in the copepod *Tigriopus californicus*, experimental hybridization of two populations with divergent heat tolerance followed by resequencing found increased survival heat tolerance in hybrid lines associated with introgression from the more heat-tolerant southern population[125]. Fertility effects were not tested.

Invasive species

Invasive species have multiple ecological and evolutionary consequences on communities and ecosystems[126]. They harm native species through ecological competition and can exhibit superior thermal performance and phenotypic plasticity[127]. Despite this, 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 assessed. However, in the round goby, an invasive species that has adapted to local salinity by improving sperm velocity has enabled population spread[128]. This 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 thermal responses of factors influencing reproductive output.

Evolutionary potential and constraint

Thermal fertility limits place an upper bound on the ability of populations to reproduce under climate warming. Niche tracking to higher altitudes or latitudes may 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

Evolutionary potential to increase thermal fertility tolerance (e.g. shifting TFLs to the right; **Box 1, Fig. C**) requires heritable genetic variation in relevant physiological processes (see section “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 (i.e., methodological, life stage, population) that need to be considered when predicting potential adaptive responses[129]. A study in mice (*Mus musculus*) found genetic variation in fertility of heat-stressed males, with resistant males being superior in several traits related to offspring production, with heritability per trait ranging from 0.09-0.13 after heat stress[130]. In *D. melanogaster,* developmental exposure to chronically high temperatures indicated low heritability for fertility tolerance, but broad-sense heritability rose as stress increased, revealing **cryptic genetic variation (CGV)**[94]. The release of CGV is a common response under genetic or environmental stress, resulting in increased trait variance due to DNA repair mechanisms losing fidelity, subsequently generating phenotypic plasticity and extreme phenotypes that could facilitate adaptation to novel environments[131].

Despite there being evidence for CGV in the heat tolerance of reproductive traits, multi-generational selection experiments in several *Drosophila* species failed to elicit evolutionary shifts in male fertility limits[6] or female fecundity[132] under steadily increasing temperatures. The lack of heat tolerance response echoes patterns for non-fertility traits[133], in which upper thermal tolerances are typically more constrained than lower tolerances[134]. However, in *T. castaneum* males undergoing experimental evolution under constant high temperature were found to have improved fertility under subsequent heatwave conditions at even higher temperatures[135], suggesting that sustained selection can sometimes yield increased fertility tolerance.

Genetic architecture

The genetic architecture of adaptive traits can also impact 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 pleiotropy**[136]. For example, a negative genetic correlation between heat and cold reproductive tolerance in ostriches (*Struthio camelus*) may constrain adaptation to increasing fluctuating temperatures[17]. In *C. maculatus*, experimental lines evolving under climate warming had increased longevity but reduced reproduction, indicating a trade-off in life history investment[137]. However, in nearly-isogenic *D. melanogaster* lines, genetic variation associated with thermal fertility effects was uncorrelated with many other stress responses, including CTLs, suggesting their independent evolution[47,94]. The typically positive relationship between body size and fecundity[138] but the negative relationship between high developmental temperature and body size in ectotherms[102] may impact the ability for 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 size[139]. Moreover, fecundity under high thermal stress can be reduced independently of body size[26] and experimental warming studies show conflicting results on evolution of body size, including its relationship to reproductive output[140,141]. So whether changes in body size, which has been suggested to represent a third universal response to climate warming[142], genetically influences the evolution of reproductive output as temperatures continue to rise requires substantial experimental dissection.

Sex-specific genetic architecture may also impact 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 for that perform well at multiple temperatures[17].

In *C. maculatus*, selection for male sexual competitiveness resulted in increased **thermal sensitivity of fertility (TSF; Box 1)** in both sexes, suggesting that sexually selected traits carry hidden costs for fertility under heat stress and that heat sensitivity of female reproductive output is genetically correlated with sexually selected reproductive traits in males[44] (**Box 2**). Conversely, *D. melanogaster* lines revealed a weak correlation between thermal sensitivity of male and female fertility[47], suggesting potential for sex-specific evolution in some systems.

Thermal induction of mutations

While 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 and/or decreased DNA repair - processes related to compromised fertility under heat stress (see section "Mechanisms of heat-induced fertility loss"). While most mutations are, on average, deleterious, higher temperatures can increase mutational variance, enhancing selection efficiency and accelerating adaptation[143]. Heat stress can trigger increased transposable element activity that can generate mutations[50]. Additionally, heat stress may interact with other mutation inducing stressors. For example, in *C. maculatus*, lines evolving under warming had enhanced germline repair after exposure to mutation-inducing radiation[137], hinting that selection to warming can affect mutational robustness. However, whether elevated mutation rates in response to increasing temperature can provide **evolutionary rescue** to fertility of animal populations is untested, although there is evidence that increased mutation rate can result in thermal adaptation in *Escherichia coli*[144].

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 thermal limits (i.e. have a narrower TSM), such that both increases in the mean temperature and its variability are predicted to make tropical species vulnerable to even modest warming[2]. While limited in scope, across six *Drosophila* species, TFL_{50} occurs at lower temperatures in tropical species, indicating lower fertility resilience and faster extinction compared to temperate species[6]. Generally, tropical species also exhibit narrower thermal fertility safety margins[6] while having higher warming tolerance and CT_{max}-based thermal safety margins[145]. However, whether tropical species are more vulnerable to temperature increases induced by climate change is debated[146]. Analysis of 38 ectotherm species suggested higher extinction risk in temperate species owing to greater thermal variance and longer extreme events (i.e., longer periods of heat stress in the summer) compared to tropical species[147]. Note though that this study could not disentangle temperature effects on fertility from survival.

Local adaptation

Thermal tolerance studies often use one population to represent a species, which may 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*[87,148] and *D. melanogaster*[76], in which low latitude populations exhibit higher fertility resilience than higher latitude populations. In the ostrich, females with higher egg laying rates under hotter temperatures regulated their head temperature better[149]. This response was both heritable and exhibited a signature of local adaptation since 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 since 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 can 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[3,81]. Reproductive failure can occur at temperatures well below lethal temperatures, shown across diverse animal taxa. Research has focused on model organisms or livestock, but heat stress consistently reduces gamete quality, reproductive fluids, and fertilization success, often via DNA damage and oxidative stress. Hsps and other stress-related genes seem to play central roles. Male fertility appears more sensitive to heat stress than female fertility, but sex-specific effects on fecundity are more variable. External fertilizers may be more vulnerable than internal fertilizers although data are sparse. Life history stages vary in both heat sensitivity and potential for recovery, with important implications for population dynamics. Plastic responses may be limited for buffering fertility.

Ecological and evolutionary impacts of heat-induced sterility are under-studied and taxonomically limited. *Drosophila* studies show thermal fertility limits generate more narrow thermal safety margins, increase extinction risk, and better predict current species distributions compared to death temperatures. However, similar studies in other taxa are lacking, the link between fertility loss and range limits remains correlative, and the impact of other factors such as behavior, acclimation, and the use of microclimates[111] in buffering fertility impacts is unknown. While there is low heritability of fertility resilience, little is known about the role of genetic architecture, mutations, or hybridization in shaping its adaptive potential. This potential is also contingent on demographic and ecological context with small populations, species with narrow ranges, and geography (i.e., tropical versus temperate habitats) impacting vulnerability. These taxa also face other anthropogenic stressors, reinforcing the need for multi-stressor experiments. Invasive species may prove more resilient but this requires study. Impacts of heat-induced sterility on sexual selection remains strikingly under-studied.

Thus, as a burgeoning field, important knowledge gaps remain. Below we highlight six research priorities to facilitate knowledge that will provide better predictive abilities of future fertility and fecundity costs and responses to climate change.

1. **Expand taxonomic breadth**. Much of the mechanistic and ecological effects of 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 factors causing some taxa to be more vulnerable. Surveying multiple populations is critical to quantify genetic variation that could respond to thermal selection for fertility and fecundity resilience.

2. **Integrate functional and ecological endpoints**. Understanding what magnitude and duration of thermal exposure is most relevant for each taxon, and integrating these data into models of population persistence (e.g. species ranges, extinction risk) is needed. Modelling efforts are essential to simulate diverse scenarios predicting male and female responses across life history stages, identifying conditions that promote population persistence vs those leading to extinction. Experimental studies could validate these models, deepening understanding of species-specific vulnerabilities and informing conservation strategies.

3. **Incorporate multi-stressor scenarios**. Interactions between heat and other stressors could mean that lower temperatures with milder fertility and fecundity effects (e.g., TFL_{30}) generates a larger effective reproductive loss (e.g. TFL_{50}) under the same temperature. Multi-stressor experiments are necessary.

4. **Evaluate broader community and ecosystem consequences**. Range shifts are a common climate change response[116], and male TFLs may partly underlie observed patterns. Whether variability in thermal fertility tolerance, including invasive species and hybridization potential, has community effects is unknown. Studies assessing species-specific CTmax effects on community interactions have found range shifts and variability in critical thermal tolerance across trophic levels alters species composition and community interactions, changes food webs[150], and modifies density-dependent competition processes[151]. Similar studies could illuminate whether species-specific TFLs also have broad community and ecosystem consequences.

5. **Determine interactions with other evolutionary processes and the impact of genetic architecture**. Both natural selection (e.g. differences across life history stages in how heat stress impacts trait expression and fitness consequences) and sexual selection, along with genetic correlations between traits within and between the sexes, impact evolutionary response. More sophisticated experiments across taxa quantifying how these factors impact the evolution of reproductive output tolerance under a warming world are necessary.

6. **Translate mechanistic insights into applied solutions**. Shared pathways of thermal reproductive damage across species offer targets for conservation, agriculture and aquaculture interventions. Agricultural practices have incorporated cooling and selective breeding to prevent fertility loss[152], but this has not translated to considerations for wild organisms. However, despite shared pathways, related taxa clearly vary in thermal fertility resilience, that may be locally adaptive. Genomic screening for such resilience along with selective breeding or adaptive introgression could provide management solutions for vulnerable or economically important taxa.

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

Figure legends

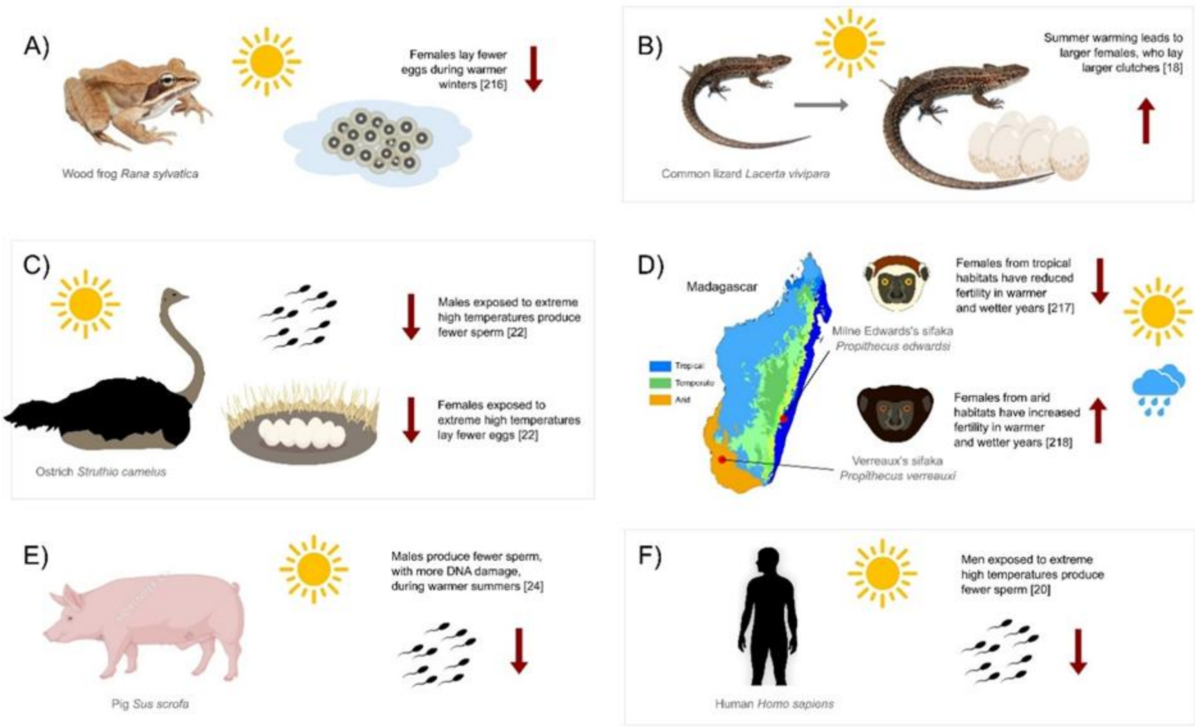


Fig. 1. Temperature effects on reproductive output are variable and taxonomically widespread. **A**) Warmer winters in the midwestern United States reduce female fecundity in the wood frog, *Rana sylvatica*[15]. **B**) In contrast, female common lizards (*Lacerta vivipara*) from southern France born in warmer years mature at a larger size and so produce larger clutches[16]. **C**) Ostriches (*Struthio camelus*) breeding in South Africa are regularly exposed to large temperature extremes, and extreme high temperatures cause females to lay fewer eggs, and males to transfer fewer sperm during mating[17]. **D**) In Madagascar, the effect of climate on breeding in sifakas differs across species. Female Verreaux's sifakas (*Propithecus verreauxi*) living in the arid south of the island have higher fertility in warmer and wetter years[18]. In contrast, female Milne Edward's sifakas (*Propithecus edwardsi*) living in the tropical, mountainous east of the island have reduced fertility in warmer and wetter years[19]. **E**) High temperatures can also impact 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 summers[8]. **F**) High temperatures also impair the fertility of human men. For example, Chinese men exposed to high temperatures 0-90 days before they visited a sperm bank had reduced sperm quality[10].

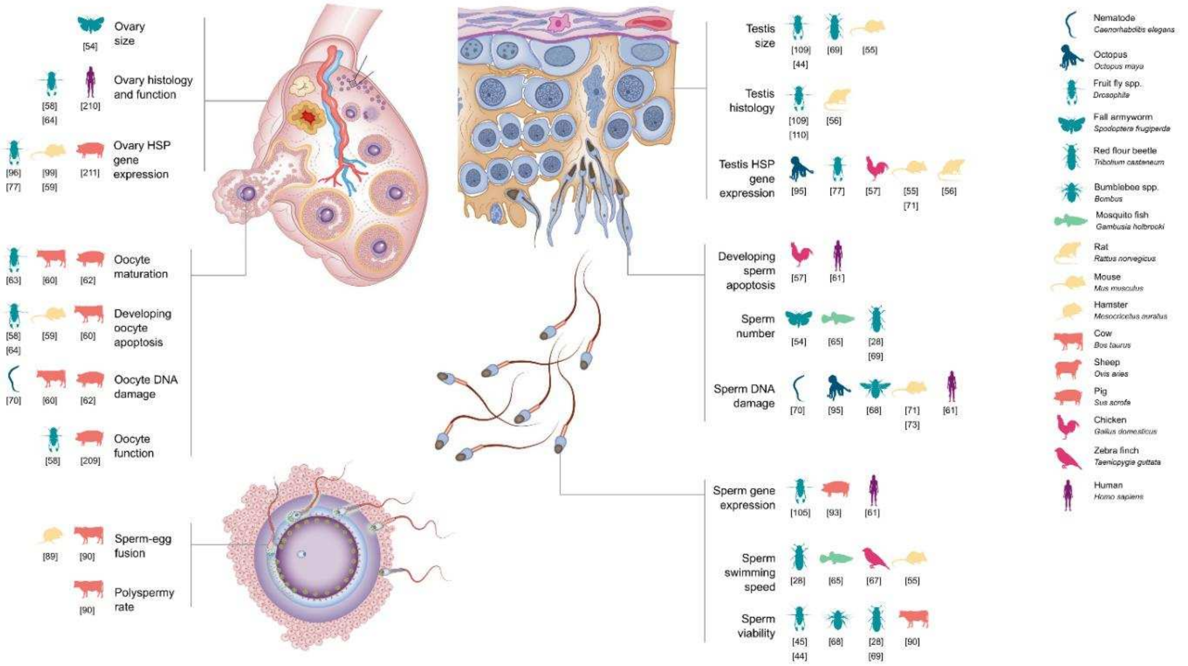


Fig. 2. Generalised schematic of animal reproduction, identifying the timing and mechanism of heat stress impacts. We focus on processes from gamete formation to fertilisation, including gonad function. Annotations are where evidence exists from studies in **Supplementary Table 1** that processes are abnormal under heat stress compared to benign conditions.

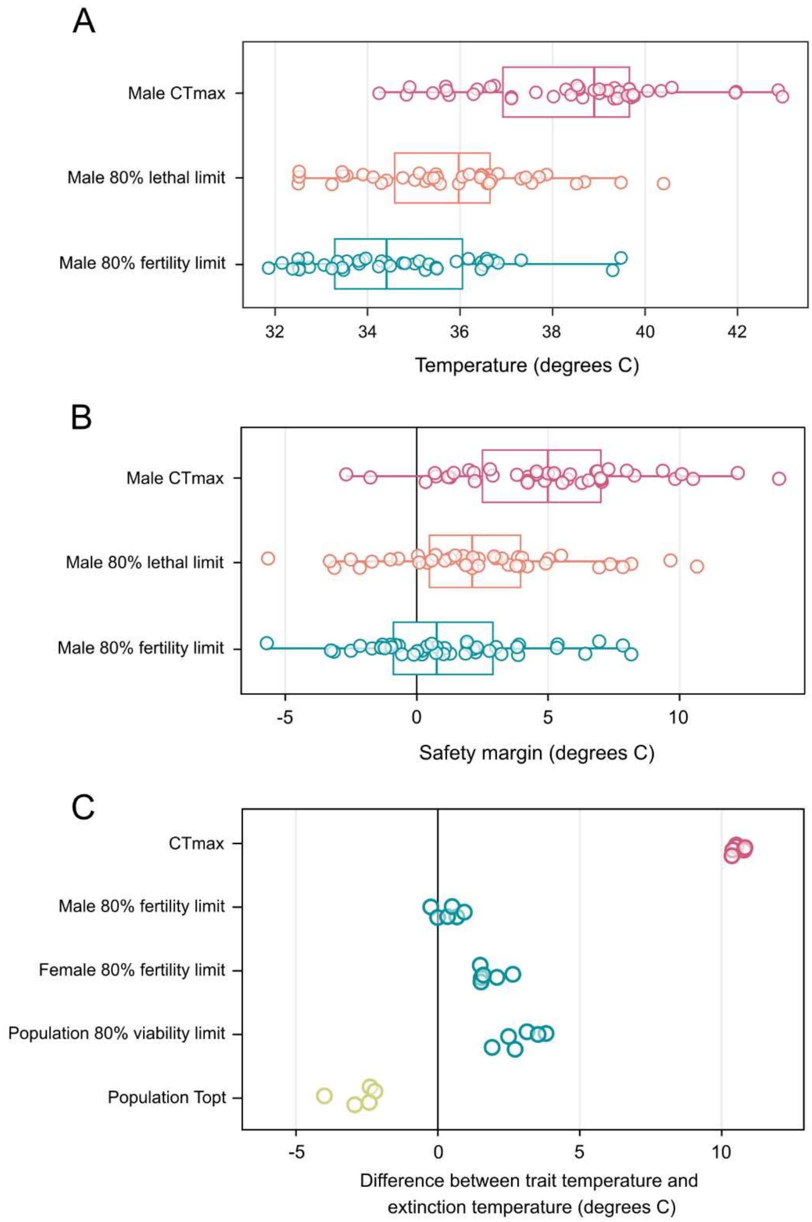
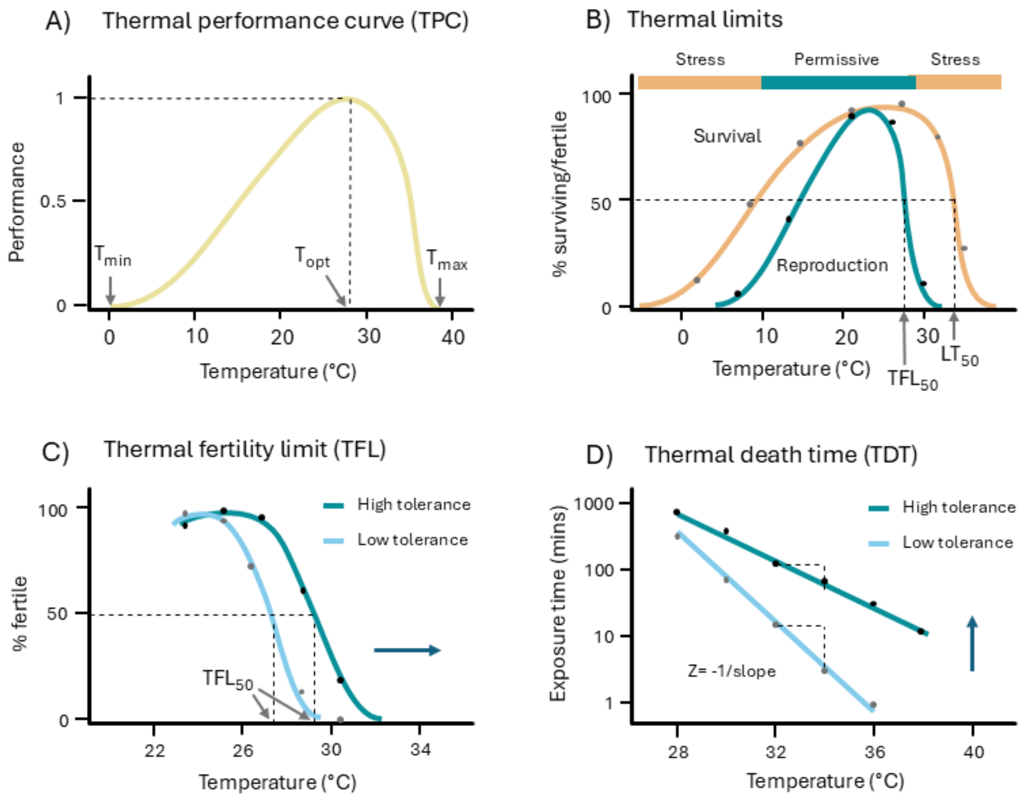


Fig. 3. The ecological relevance of thermal fertility limits. A) Across 43 species of Drosophila fruit fly, the 80% male Thermal Fertility Limit (TFL; green points) tends to occur at a lower temperature than either the 80% Lethal Limit (LT; orange points) or the Critical Thermal Maximum (CT_{max}; red points). **B)** Across 43 species of *Drosophila* fruit fly, the 80% male Thermal Fertility Limit (TFL; green points) has a narrower Thermal Safety Margin than either the 80% Lethal Limit (LT; orange points) or the Critical Thermal Maximum (CT_{max}; red points). Data in A) and B) adapted from [5]. **C)** Across six species of *Drosophila* fruit fly, the temperature at which laboratory populations go extinct is almost identical to the 80% male Thermal Fertility Limit (TFL). The 80% female TFL, or any other metrics of population viability, are less strongly related to extinction temperature. Data adapted from[6].

Box 1. Measures of thermal tolerance



Thermal tolerance can be summarized by a **Thermal Performance Curve**(TPC), which plots trait performance across temperatures[153](**Fig. a**; note, curves in all panels are hypothetical). TPCs are typically unimodal and asymmetric, with performance declining steeply at higher temperatures[154,155]; **Fig. a**). TPCs describe thermal tolerance by determining the temperature that maximizes performance (T_{opt}; dashed line **Fig. a**), and the lower (T_{min}) and upper (T_{max}) thermal limits at which performance fails (**Fig. a**). Intervening temperatures describe the 'permissive' region which allows life-cycle completion, including reproduction and anything outside of this region is stressful[4,156](**Fig. b**). TPCs have been used to predict how organisms will respond to climate change[134,145].

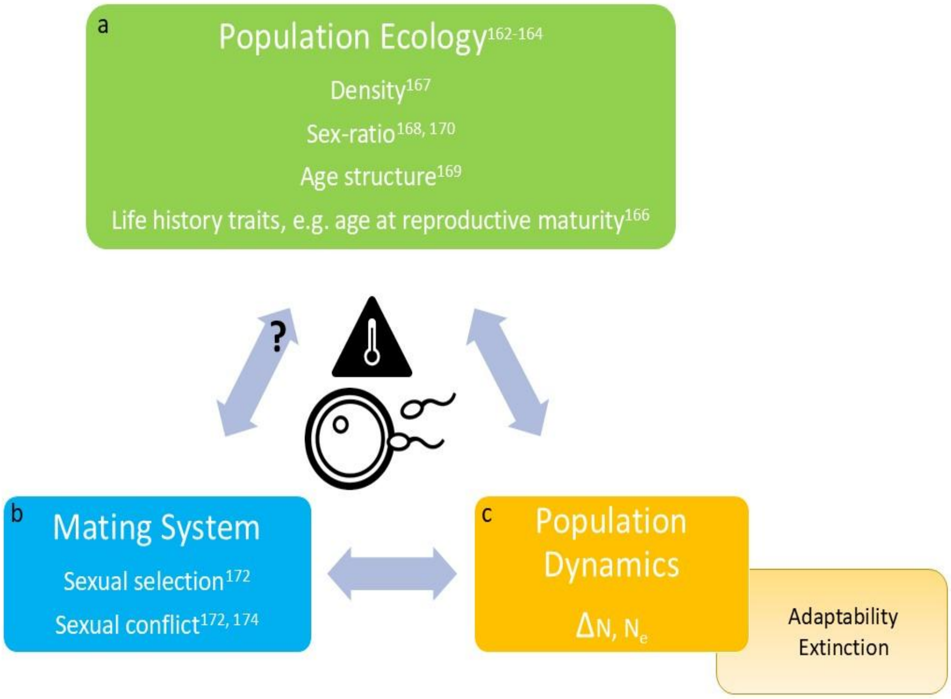
Survival (binary trait) can be measured using **Critical Thermal Limits**, the temperature at which individuals lose coordination or motor function(**Critical thermal maximum** [**CT_{max}**] and Critical thermal minimum [CT_{min}]), or via **Lethal Temperatures**(LTs), the temperature at which individuals die[2]. For example, LT_{50} indicates the temperature at which 50% of a cohort dies (**Fig. b**; dashed LT_{50} line, orange curve). These assays usually apply heat acutely, either statically for a set time period at a particular temperature or through ramping the temperature at some rate until physiological failure occurs.

Similarly, fertility endpoints can be assessed using **Thermal Fertility Limits**[7] (TFLs; **Fig. b**), which measures the proportion of individuals that are **sterile/infertile** (e.g. 50% for TFL_{50}; **Fig. b**, dashed TFL_{50} line, green curve) after heat exposure. A higher TFL indicates greater heat tolerance (**Fig. c**, arrow showing shift to right, dark blue line). 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 exposures[7].

Heat stress effects on reproductive output can also be assessed using **Thermal Sensitivity of Fertility** (**TSF**) in which fecundity (e.g. egg or offspring number) at a small number of test temperatures is determined[7,83]. 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 climate[7,14]).

This variation links to the issue that thermal injury may accrue over time in a dose-dependent way[27], which these methods do not test. The **Thermal Death Time** approach (TDT) explicitly addresses this by integrating exposure time and temperature with log-transformed exposure duration resulting in some performance outcome(e.g. 50% loss of fertility) plotted against temperature[4] (**Fig. d**). This captures how milder stress over longer periods can cause similar damage to acute extremes. Higher thermal tolerance (**Fig. d**, arrow) is indicated by a shallower negative slope[4] (**Fig. d**, dark blue line) with sensitivity quantified by z (**Fig. d**, dashed lines). TDT studies improve predictions of organismal response to high temperatures[157,158]. Only one TDT study has assessed fertility, finding that heat-induced sterility in *D. suzukii* followed a dose-dependent relationship, similar to survival traits, but occurring at lower temperatures[85]. However, debate remains on the most ecologically relevant way to measure thermal tolerance, including other approaches that integrate exposure duration and thermal intensity[28,159].

Box 2. Sexual selection and heat-induced sterility



Rising temperatures can influence sexual selection via heat-induced reductions in fertility and fecundity. This is important because sexual selection may promote adaptation by purging deleterious alleles or hinder it by escalating sexual conflict and reducing female fitness[160,161]. Ecological factors are key influencers of mating systems[162,163], shaping both the strength and sex-specific direction of sexual selection and sexual conflict and sexually antagonistic coevolution[163,164]. Previous reviews have highlighted that the interaction between thermal ecology and sexual selection is of major interest[165], but here we focus on how heat-induced sterility sexual selection, an area largely overlooked.

Heat stress reductions in fertility and fecundity (**Fig.**, central image) directly changing population size (ΔN) and effective population size (N_e), impacting genetic variation and evolutionary consequences of these changes (**Fig. c**), while altering ecological parameters (**Fig. a**). These population-level parameters influence mating systems[166-170], and both sexual selection and sexual conflict (**Fig. b**). All interact to determine adaptability and extinction probability, although whether there is a reciprocal effect of sexual selection on ecological parameters is uncertain[171] (**Fig. b**, ? going toward **Fig. a**). Some research has begun to ask how heat-induced sterility impacts these ecological components. In *D. virilis*, pupal heat stress delays reproductive maturity, leaving many adults temporarily infertile whereas adult stress causes permanent sterility[77,166], which can age-restructure populations. Moreover, if the sexes differ in thermal sensitivity, the OSR shifts[166], modifying sex-specific variance in reproductive success and the strength and direction of sexual selection.

Heat-induced sterility may also select for different mating behaviours, impacting sexual selection. Mating with sterile males often triggers facultative polyandry as a fertility assurance strategy[172], intensifying sexual conflict and changing sex-specific variance in reproductive success. Condition-dependent male signals[173] may be reduced by heat stress, favouring female choice for fertile males[174]. Conversely, reduced female fecundity may drive male mate choice toward more fecund partners[174]. These behavioural and OSR-mediated effects alter the strength and sex-bias of sexual selection. A meta-analysis suggests warming generally reduces variance in male fitness while increasing variance in females[165], though data specific to heat-induced sterility were lacking.

Sexual selection may change how the sexes invest in reproductive success and heat stress may reveal trade-offs in this investment. In two insect species, males evolving under regimes promoting greater sexual selection showed reduced progeny production [44] or sperm competitiveness[175] after heat stress. This may reflect a trade-off between investment in intensified male-male competition and germline maintenance[176]. In contrast, multigenerational experimental sexual selection in other taxa indicates polyandry can benefit populations: when both sexes experience warming, populations with a history of strong sexual selection have higher fecundity[177-179].

Overall, heat-induced fertility and fecundity declines has 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.

Glossary

Acclimation - Physiological adjustment to an increase in temperature, particularly where longer-term exposure to a mildly stressful temperature can increase the 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.

Cryptic Genetic Variation (CGV) – 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.

Critical Thermal Maximum (CT_{max}) - the highest temperature at which a physiological function is lost (often measured as motor control or coordinated movement), or death occurs.

Evolutionary rescue – selection for evolutionary change enabling individuals to survive under stressful or novel conditions and is sufficiently rapid to save a population from extinction

Fecundity - Number of eggs or viable offspring produced by an individual. 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 effects of heat treatment on offspring survival is not conflated. However, in many taxa, only offspring number is reported. In cases where 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 parental effect.

Fertility - Ability to produce viable offspring. Note that in 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.

Heatwave (or heat wave) – A meteorological term for a sustained period of abnormally warm weather that last for multiple days, measured relative to the normal climate for a given location and time of year. There is no universally agreed definition for the duration and severity of a heatwave; 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,

Heat Shock Proteins (Hsps) – 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 Hsps function as chaperone proteins, stabilising the structure of other proteins which are sensitive to heat stress.

Infertility/sterility - Inability to produce viable offspring but can specifically mean inability to produce gametes.

Lethal Temperature (LT) - the temperature at which there is, for example, 80% (LT_{80}) or 50% (LT_{50}) mortality in a population/ 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 which stay cool.

Operational Sex Ratio (OSR) – the ratio of males to females within the mating pool (fertile and ready to mate).

Polyspermy – entrance of more than one sperm into the egg; typically lethal.

Phenotypic plasticity – the ability of a genome to produce multiple phenotypes depending on the environment.

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.

Thermal Fertility Limit (TFL) - The temperature at which individuals become (at least temporarily) sterile (scored as a binary 0/1 outcome). As for LT, measured as TFL_{80}/TFL_{50}, the temperature at which either 80% or 50% of individuals in a population/ set of experimental organisms are infertile.

Thermal Death Time (TDT) - The integration of stress intensity (i.e. temperature) and exposure time. Through cumulative effects organisms for example may suffer more from a mildly stressful high temperature experienced for a long time versus an extreme temperature for a short time. As the relationship is exponential, a small increase in temperature may lead to a large decrease in tolerance time.

Thermal Performance Curve (TPC) - 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 Sensitivity of Fertility (TSF) - The relationship between temperature and reproductive output. The number of offspring produced or proxies thereof (e.g. sperm velocity, follicle number) are a measure of fertility rather than the binary 0/1 score of fertility.

Thermal Safety Margin (TSM) - the difference between the thermal limit and the maximum ambient temperature experienced in the environment

Acknowledgments

We would like to thank the European Society for Evolutionary Biology for funding the Special Topics Network (STN) on The Evolutionary Ecology of Thermal Fertility Limits and the members of the STN whose work and collaboration on this topic we value immensely. We would like to thank our funders for support (Vetenskapsrådet 2022-03116 and 2018-04598 to RRS; Natural Environment Research Council NE/X011550/1 to LD; the Biotechnology and Biological Research Council (BB/W016753/1 to AB and RRS; UKRI1927 to AB, RRS and CF) and a Heisenberg fellowship from the Deutsche Forschungsgemeinschaft (FR 2973/11-1 to CF).

Competing interests

The authors declare no competing interests.

Author contributions

All authors contributed equally to all aspects of the writing.

References

1 IPCC. Climate Change 2023: Synthesis Report. Contribution of Working Groups I, II and III to the Sixth Assessment Report of the Intergovernmental Panel on Climate Change. 35-115 (Geneva, Switzerland, 2023).

2 Bennett, J. M. *et al.* GlobTherm, a global database on thermal tolerances for aquatic and terrestrial organisms. *Scientific Data* **5**, 180022, doi:10.1038/sdata.2018.22 (2018).

3 Walsh, B. S. *et al.* The Impact of Climate Change on Fertility. *Trends In Ecology & Evolution* **34**, 249-259 (2019).

4 Ørsted, M., Jørgensen, L. B. & Overgaard, J. Finding the right thermal limit: a framework to reconcile ecological, physiological and methodological aspects of CTmax in ectotherms. *Journal of Experimental Biology* **225**, jeb244514, doi:10.1242/jeb.244514 (2022).

5 Parratt, S. R. *et al.* Temperatures that sterilize males better match global species distributions than lethal temperatures. *Nature Climate Change* **11**, 481-484, doi:10.1038/s41558-021-01047-0 (2021).

6 van Heerwaarden, B. & Sgrò, C. M. Male fertility thermal limits predict vulnerability to climate warming. *Nature Communications* **12**, 2214, doi:10.1038/s41467-021-22546-w (2021).

7 Bretman, A. *et al.* Systematic approaches to assessing high-temperature limits to fertility in animals. *Journal of Evolutionary Biology* **37**, 471-485, doi:10.1093/jeb/voae021 (2024).

8 Peña, S. T. *et al.* Tropical summer induces DNA fragmentation in boar spermatozoa: implications for evaluating seasonal infertility. *Reproduction, Fertility and Development* **31**, 590-601, doi:10.1071/RD18159 (2019).

9 McAfee, A. *et al.* Vulnerability of honey bee queens to heat-induced loss of fertility. *Nature Sustainability* **3**, 367-376, doi:10.1038/s41893-020-0493-x (2020).

10 Xiao, L. *et al.* Effects of temperature anomaly on sperm quality: A multi-center study of 33,234 men. *Heliyon* **10**, e26765, doi:10.1016/j.heliyon.2024.e26765 (2024).

11 Leith, N. T., Macchiano, A., Moore, M. P. & Fowler-Finn, K. D. Temperature impacts all behavioral interactions during insect and arachnid reproduction. *Current Opinion in Insect Science* **45**, 106-114, doi:10.1016/j.cois.2021.03.005 (2021).

12 Neate-Clegg, M. H. C., Tonelli, B. A. & Tingley, M. W. Advances in breeding phenology outpace latitudinal and elevational shifts for North American birds tracking temperature. *Nature Ecology & Evolution* **8**, 2027-2036, doi:10.1038/s41559-024-02536-z (2024).

13 Wiley, E. M. & Ridley, A. R. The effects of temperature on offspring provisioning in a cooperative breeder. *Animal Behaviour* **117**, 187-195, doi:10.1016/j.anbehav.2016.05.009 (2016).

14 Dougherty, L. R. *et al.* A systematic map of studies testing the relationship between temperature and animal reproduction. *Ecological Solutions and Evidence* **5**, e12303, doi:10.1002/2688-8319.12303 (2024).

15 Benard, M. F. Warmer winters reduce frog fecundity and shift breeding phenology, which consequently alters larval development and metamorphic timing. *Global Change Biology* **21**, 1058-1065, doi:10.1111/gcb.12720 (2015).

16 Chamaillé-Jammes, S., Massot, M., Aragón, P. & Clobert, J. Global warming and positive fitness response in mountain populations of common lizards *Lacerta vivipara*. *Global Change Biology* **12**, 392-402, doi:10.1111/j.1365-2486.2005.01088.x (2006).

17 Schou, M. F. *et al.* Extreme temperatures compromise male and female fertility in a large desert bird. *Nature Communications* **12**, 666, doi:10.1038/s41467-021-20937-7 (2021).

18 Campos, F. A. *et al.* Does climate variability influence the demography of wild primates? Evidence from long-term life-history data in seven species. *Global Change Biology* **23**, 4907-4921, doi:10.1111/gcb.13754 (2017).

19 Dunham, A. E., Erhart, E. M. & Wright, P. C. Global climate cycles and cyclones: consequences for rainfall patterns and lemur reproduction in southeastern Madagascar: Climate effects on lemur demography. *Global Change Biology* **17**, 219-227, doi:10.1111/j.1365-2486.2010.02205.x (2011).

20 Van Heezik, Y., Jalme, M. S., Hémon, S. & Seddon, P. Temperature and egg-laying experience influence breeding performance of captive female houbara bustards. *Journal of Avian Biology* **33**, 63-70, doi:10.1034/j.1600-048X.2002.330110.x (2002).

21 Sturup, M., Baer-Imhoof, B., Nash, D. R., Boomsma, J. J. & Baer, B. When every sperm counts: factors affecting male fertility in the honey bee *Apis mellifera*. *Behavioral Ecology* **24**, 1192-1198, doi:10.1093/beheco/art049 (2013).

22 Weaving, H., Terblanche, J. S. & English, S. Heatwaves are detrimental to fertility in the viviparous tsetse fly. *Proceedings of the Royal Society B: Biological Sciences* **291**, 20232710, doi:10.1098/rspb.2023.2710 (2024).

23 Eads, A. R., Kennington, W. J. & Evans, J. P. Interactive effects of ocean warming and acidification on sperm motility and fertilization in the mussel *Mytilus galloprovincialis*. *Marine Ecology Progress Series* **562**, 101-111, doi:10.3354/meps11944 (2016).

24 Armstrong, E. J., Dubousquet, V., Mills, S. C. & Stillman, J. H. Elevated temperature, but not acidification, reduces fertilization success in the small giant clam, *Tridacna maxima*. *Marine Biology* **167**, 8, doi:10.1007/s00227-019-3615-0 (2020).

25 Romano, A. *et al.* Shrinking body size under climate warming is not associated with selection for smaller individuals in a migratory bird. *Journal of Animal Ecology* **94**, 958-970, doi:10.1111/1365-2656.70027 (2025).

26 Klepsatel, P. *et al.* Variation in thermal performance and reaction norms among populations. *Evolution* **67**, 3573-3587, doi:10.1111/evo.12221 (2013).

27 Rezende, E. L., Castañeda, L. E. & Santos, M. Tolerance landscapes in thermal ecology. *Functional Ecology* **28**, 799-809, doi:10.1111/1365-2435.12268 (2014).

28 Buckley, L. B., Huey, R. B. & Ma, C.-S. How Damage, Recovery, and Repair Alter the Fitness Impacts of Thermal Stress. *Integrative And Comparative Biology*, icaf019, doi:10.1093/icb/icaf019 (2025).

29 Meena, A. *et al.* Life stage-specific effects of heat stress on spermatogenesis and oogenesis in *Drosophila melanogaster*. *Journal of Thermal Biology* **125**, 104001, doi:10.1016/j.jtherbio.2024.104001 (2024).

30 Reshma, R. *et al.* Transgenerational effects of thermal stress on reproductive physiology of fall armyworm, *Spodoptera frugiperda*. *Journal of Pest Science* **96**, 1465-1481, doi:10.1007/s10340-023-01660-2 (2023).

31 Rizzoto, G., Boe-Hansen, G., Klein, C., Thundathil, J. C. & Kastelic, J. P. Acute mild heat stress alters gene expression in testes and reduces sperm quality in mice. *Theriogenology* **158**, 375-381, doi:10.1016/j.theriogenology.2020.10.002 (2020).

32 Gandara, A. C. P. & Drummond-Barbosa, D. Warm and cold temperatures have distinct germline stem cell lineage effects during *Drosophila* oogenesis. *Development* **149**, dev200149, doi:10.1242/dev.200149 (2022).

33 Li, J. *et al.* Effects of chronic heat stress on granulosa cell apoptosis and follicular atresia in mouse ovary. *Journal of Animal Science and Biotechnology* **7**, 57, doi:10.1186/s40104-016-0116-6 (2016).

34 Rao, M. *et al.* Transient scrotal hyperthermia affects human sperm DNA integrity, sperm apoptosis, and sperm protein expression. *Andrology* **4**, 1054-1063, doi:10.1111/andr.12228 (2016).

35 Roth, Z. & Hansen, P. J. Disruption of nuclear maturation and rearrangement of cytoskeletal elements in bovine oocytes exposed to heat shock during maturation. *Reproduction* **129**, 235-244, doi:10.1530/rep.1.00394 (2005).

36 Yadav, S. K. *et al.* The thermo-sensitive gene expression signatures of spermatogenesis. *Reproductive Biology and Endocrinology* **16**, 56, doi:10.1186/s12958-018-0372-8 (2018).

37 Barati, F. *et al.* Meiotic competence and DNA damage of porcine oocytes exposed to an elevated temperature. *Theriogenology* **69**, 767-772, doi:10.1016/j.theriogenology.2007.08.038 (2008).

38 Adriaenssens, B., Damme, R., Seebacher, F. & Wilson, R. S. Sex cells in changing environments: can organisms adjust the physiological function of gametes to different temperatures? *Global Change Biology* **18**, 1797-1803, doi:10.1111/j.1365-2486.2012.02672.x (2012).

39 Boni, R., Gallo, A., Montanino, M., Macina, A. & Tosti, E. Dynamic changes in the sperm quality of *Mytilus galloprovincialis* under continuous thermal stress. *Molecular Reproduction and Development* **83**, 162-173, doi:10.1002/mrd.22604 (2016).

40 Sales, K. *et al.* Experimental heatwaves comprise sperm function and cause transgenerational damage in a model insect. *Nature Communications* **9**, 4771 (2018).

41 Canal Domenech, B. & Fricke, C. Recovery from heat-induced infertility—A study of reproductive tissue responses and fitness consequences in male *Drosophila melanogaster*. *Ecology and Evolution* **12**, doi:10.1002/ece3.9563 (2022).

42 Martinet, B. *et al.* Mating under climate change: Impact of simulated heatwaves on the reproduction of model pollinators. *Functional Ecology* **35**, 739-752, doi:10.1111/1365-2435.13738 (2021).

43 Kurhanewicz, N. A., Dinwiddie, D., Bush, Z. D. & Libuda, D. E. Elevated Temperatures Cause Transposon-Associated DNA Damage in *C. elegans* Spermatocytes. *Current Biology* **30**, 5007-5017.e5004, doi:10.1016/j.cub.2020.09.050 (2020).

44 Baur, J., Zwoinska, M., Koppik, M., Snook, R. R. & Berger, D. Heat stress reveals a fertility debt owing to postcopulatory sexual selection. *Evolution Letters* **8**, 101-113, doi:10.1093/evlett/qrad007 (2024).

45 Meena, A. *et al.* Fertility loss and recovery dynamics after repeated heat stress across life stages in male *Drosophila melanogaster*: patterns and processes. *Royal Society Open Science* **11**, 241082, doi:10.1098/rsos.241082 (2024).

46 Boni, R. Heat stress, a serious threat to reproductive function in animals and humans. *Molecular Reproduction and Development* **86**, 1307-1323, doi:10.1002/mrd.23123 (2019).

47 Zwoinska, M. K., Rodrigues, L. R., Slate, J. & Snook, R. R. Phenotypic Responses to and Genetic Architecture of Sterility Following Exposure to Sub-Lethal Temperature During Development. *Frontiers in Genetics* **11**, 573, doi:10.3389/fgene.2020.00573 (2020).

48 Rodrigues, L. R., Zwoinska, M. K., Wiberg, R. A. W. & Snook, R. R. The genetic basis and adult reproductive consequences of developmental thermal plasticity. *Journal of Animal Ecology* **91**, 1119-1134, doi:10.1111/1365-2656.13664 (2022).

49 Cappucci, U. *et al.* The Hsp70 chaperone is a major player in stress-induced transposable element activation. *Proceedings of the National Academy of Sciences* **116**, 17943-17950, doi:doi:10.1073/pnas.1903936116 (2019).

50 Mojica, E. A. & Kültz, D. Physiological mechanisms of stress-induced evolution. *Journal of Experimental Biology* **225**, jeb243264, doi:10.1242/jeb.243264 (2022).

51 Bodelón, A., Fablet, M., Siqueira De Oliveira, D., Vieira, C. & García Guerreiro, M. P. Impact of Heat Stress on Transposable Element Expression and Derived Small RNAs in *Drosophila subobscura*. *Genome Biology and Evolution* **15**, evad189, doi:10.1093/gbe/evad189 (2023).

52 Poiani, A. Complexity of seminal fluid: a review. *Behavioral Ecology and Sociobiology* **60**, 289-310, doi:10.1007/s00265-006-0178-0 (2006).

53 Rossi, N., Lopez Juri, G., Chiaraviglio, M. & Cardozo, G. Oviductal fluid counterbalances the negative effect of high temperature on sperm in an ectotherm model. *Biology Open* **10**, bio058593, doi:10.1242/bio.058593 (2021).

54 Kustra, M. C. *et al.* Warm waters undermine cryptic female choice. doi:10.1101/2025.04.10.648287 (2025).

55 Walsh, B. S. *et al.* Female fruit flies cannot protect stored sperm from high temperature damage. *Journal of Thermal Biology* **105**, 103209, doi:10.1016/j.jtherbio.2022.103209 (2022).

56 Gandara, A. C. P. & Drummond-Barbosa, D. Chronic exposure to warm temperature causes low sperm abundance and quality in *Drosophila melanogaster*. *Scientific Reports* **13**, 12331, doi:10.1038/s41598-023-39360-7 (2023).

57 Hirao, Y. & Yanagimachi, R. Temperature dependence of sperm-egg fusion and post-fusion events in hamster fertilization. *Journal of Experimental Zoology* **205**, 433-437, doi:10.1002/jez.1402050312 (1978).

58 Sakatani, M., Yamanaka, K., Balboula, A. Z., Takenouchi, N. & Takahashi, M. Heat stress during in vitro fertilization decreases fertilization success by disrupting anti-polyspermy systems of the oocytes. *Molecular Reproduction and Development* **82**, 36-47, doi:10.1002/mrd.22441 (2015).

59 Morimoto, R. I. The Heat Shock Response: Systems Biology of Proteotoxic Stress in Aging and Disease. *Cold Spring Harbor Symposia on Quantitative Biology* **76**, 91-99, doi:10.1101/sqb.2012.76.010637 (2011).

60 Neuer, A. The role of heat shock proteins in reproduction. *Human Reproduction Update* **6**, 149-159, doi:10.1093/humupd/6.2.149 (2000).

61 Gòdia, M. *et al.* A RNA-Seq Analysis to Describe the Boar Sperm Transcriptome and Its Seasonal Changes. *Frontiers in Genetics* **10**, 299, doi:10.3389/fgene.2019.00299 (2019).

62 Pei, Y., Wu, Y. & Qin, Y. Effects of chronic heat stress on the expressions of heat shock proteins 60, 70, 90, A2, and HSC70 in the rabbit testis. *Cell Stress and Chaperones* **17**, 81-87, doi:10.1007/s12192-011-0287-1 (2012).

63 Wang, S.-H. *et al.* Acute Heat Stress Induces Differential Gene Expressions in the Testes of a Broiler-Type Strain of Taiwan Country Chickens. *PLOS ONE* **10**, e0125816, doi:10.1371/journal.pone.0125816 (2015).

64 López-Galindo, L. *et al.* Transcriptomic Analysis Reveals Insights on Male Infertility in *Octopus maya* Under Chronic Thermal Stress. *Frontiers in Physiology* **9**, 1920, doi:10.3389/fphys.2018.01920 (2019).

65 Tang, L. *et al.* Negative effects of heat stress on ovarian tissue in female rabbit. *Frontiers in Veterinary Science* **Volume 9 - 2022**, doi:10.3389/fvets.2022.1009182 (2022).

66 Fangue, N. A., Hofmeister, M. & Schulte, P. M. Intraspecific variation in thermal tolerance and heat shock protein ge ne expression in common killifish, *Fundulus heteroclitus*. *Journal of Experimental Biology* **209**, 2859-2872, doi:10.1242/jeb.02260 (2006).

67 Krebs, R. A. A comparison of Hsp70 expression and thermotolerance in adults and larvae of three *Drosophila* species. *Cell Stress & Chaperones* **4**, 243, doi:10.1379/1466-1268(1999)004<0243:ACOHEA>2.3.CO;2 (1999).

68 Bei, M., Wang, Q., Yu, W., Han, L. & Yu, J. Effects of heat stress on ovarian development and the expression of HSP genes in mice. *Journal of Thermal Biology* **89**, 102532, doi:10.1016/j.jtherbio.2020.102532 (2020).

69 Krebs, R. A. & Loeschcke, V. Costs and Benefits of Activation of the Heat-Shock Response in *Drosophila melanogaster*. *Functional Ecology* **8**, 730, doi:10.2307/2390232 (1994).

70 Ware-Gilmore, F., Novelo, M., Sgrò, C. M., Hall, M. D. & McGraw, E. A. Assessing the role of family level variation and heat shock gene expre ssion in the thermal stress response of the mosquito *Aedes aegypti*. *Philosophical Transactions of the Royal Society B: Biological Sciences* **378**, 20220011, doi:10.1098/rstb.2022.0011 (2023).

71 Podrabsky, J. E. & Somero, G. N. Changes in gene expression associated with acclimation to constant tem peratures and fluctuating daily temperatures in an annual killifish *Austrofundulus limnaeus*. *Journal of Experimental Biology* **207**, 2237-2254, doi:10.1242/jeb.01016 (2004).

72 Velikaneye, B. A. & Kozak, G. M. Epigenomic Changes in *Ostrinia* Moths Under Elevated Pupal and Adult Temperature. *Molecular Ecology* **34**, e17676, doi:10.1111/mec.17676 (2025).

73 Khan, S. & Mishra, R. K. Multigenerational Effect of Heat Stress on the *Drosophila melanogaster* Sperm Proteome. *Journal of Proteome Research* **23**, 2265-2278, doi:10.1021/acs.jproteome.4c00205 (2024).

74 Laporta, J., Khatib, H. & Zachut, M. Review: Phenotypic and molecular evidence of inter- and trans-generati onal effects of heat stress in livestock mammals and humans. *animal* **18**, 101121, doi:10.1016/j.animal.2024.101121 (2024).

75 Canal Domenech, B. & Fricke, C. Developmental heat stress interrupts spermatogenesis inducing early male sterility in *Drosophila melanogaster*. *Journal of Thermal Biology* **114**, 103589, doi:10.1016/j.jtherbio.2023.103589 (2023).

76 Rohmer, C., David, J. R., Moreteau, B. & Joly, D. Heat induced male sterility in *Drosophila melanogaster*: adaptive genetic variations among geographic populations and role of the Y c hromosome. *Journal of Experimental Biology* **207**, 2735-2743, doi:10.1242/jeb.01087 (2004).

77 Walsh, B. S. *et al*. Plastic responses of survival and fertility following heat stress in pupal and adult *Drosophila virilis*. *Ecology and Evolution* **11**, 18238-18247, doi:10.1002/ece3.8418 (2021).

78 Li, H. *et al*. The fertility recovering from heat stress and interactions of heat sho ck protein 20 with reproduction-related proteins in *Monochamus alternatus*. *Insect Science*, 1744-7917.13470, doi:10.1111/1744-7917.13470 (2024).

79 Iossa, G. Sex-Specific Differences in Thermal Fertility Limits. *Trends in Ecology & Evolution* **34**, 490-492, doi:10.1016/j.tree.2019.02.016 (2019).

80 Clutton-Brock, T. H. & Parker, G. A. Potential Reproductive Rates and the Operation of Sexual Selection. *The Quarterly Review of Biology* **67**, 437-456, doi:10.1086/417793 (1992).

81 David, J. R. *et al*. Male sterility at extreme temperatures: a significant but neglected phenomenon for understanding *Drosophila* climatic adaptations. *Journal of Evolutionary Biology* **18**, 838-846, doi:10.1111/j.1420-9101.2005.00914.x (2005).

82 Chatten, A., Grieve, I., Meligoniti, E., Hayward, C. & Pilakouta, N. Predicting the Effects of Climate Change on the Fertility of Aquatic Animals Using a Meta-Analytic Approach. *Ecology Letters* **28**, e70054, doi:10.1111/ele.70054 (2025).

83 Baur, J., Jagusch, D., Michalak, P., Koppik, M. & Berger, D. The mating system affects the temperature sensitivity of male and female fertility. *Functional Ecology* **36**, 92-106, doi:10.1111/1365-2435.13952 (2022).

84 Parrett, J. M., Kulczak, M. & Szudarek-Trepto, N. Fertility loss under thermal stress: males have lower fertility limits but no evidence of sex differences in sensitivity. *Oikos* **2024**, e10329, doi:10.1111/oik.10329 (2024).

85 Ørsted, M., Willot, Q., Olsen, A. K., Kongsgaard, V. & Overgaard, J. Thermal limits of survival and reproduction depend on stress duration: A case study of *Drosophila suzukii*. *Ecology Letters* **27**, e14421, doi:10.1111/ele.14421 (2024).

86 Janowitz, S. A. & Fischer, K. Opposing effects of heat stress on male versus female reproductive success in *Bicyclus anynana* butterflies. *Journal of Thermal Biology* **36**, 283-287, doi:10.1016/j.jtherbio.2011.04.001 (2011).

87 Santos, M. A. *et al*. Sex and population differences underlie variation in reproductive success in a warming environment. *Evolution* **77**, 1842-1851, doi:10.1093/evolut/qpad104 (2023).

88 Piyaphongkul, J., Pritchard, J. & Bale, J. Heat Stress Impedes Development and Lowers Fecundity of the Brown Plan thopper *Nilaparvata lugens* (Stål). *PLoS ONE* **7**, e47413, doi:10.1371/journal.pone.0047413 (2012).

89 Kingsolver, J. G. *et al*. Complex Life Cycles and the Responses of Insects to Climate Change. *Integrative and Comparative Biology* **51**, 719-732, doi:10.1093/icb/icr015 (2011).

90 Dahlke, F. T., Wohlrab, S., Butzin, M. & Pörtner, H.-O. Thermal bottlenecks in the life cycle define climate vulnerability of fish. *Science* **369**, 65-70, doi:10.1126/science.aaz3658 (2020).

91 Zhang, W., Chang, X.-Q., Hoffmann, A., Zhang, S. & Ma, C.-S. Impact of hot events at different developmental stages of a moth: the closer to adult stage, the less reproductive output. *Scientific Reports* **5**, 10436, doi:10.1038/srep10436 (2015).

92 Rebolledo, A. P., Sgrò, C. M. & Monro, K. Thermal performance curves reveal shifts in optima, limits, and breadth in early life. *Journal of Experimental Biology*, jeb.233254, doi:10.1242/jeb.233254 (2020).

93 Collin, R. & Chan, K. Y. K. The sea urchin *Lytechinus variegatus* lives close to the upper thermal limit for early development in a tropical lagoon. *Ecology and Evolution* **6**, 5623-5634, doi:10.1002/ece3.2317 (2016).

94 Rodrigues, L. R. *et al.* Fluctuating heat stress during development exposes reproductive costs and putative benefits. *Journal of Animal Ecology* **91**, 391-403, doi:10.1111/1365-2656.13636 (2022).

95 Betini, G. S., Wang, X., Avgar, T., Guzzo, M. M. & Fryxell, J. M. Food availability modulates temperature-dependent effects on growth, reproduction, and survival in *Daphnia magna*. *Ecology and Evolution* **10**, 756-762, doi:10.1002/ece3.5925 (2020).

96 Donelson, J. M., Munday, P. L., McCormick, M. I., Pankhurst, N. W. & Pankhurst, P. M. Effects of elevated water temperature and food availability on the reproductive performance of a coral reef fish. *Marine Ecology Progress Series* **401**, 233-243 (2010).

97 Costa, E. A. P. D. A., Santos, E. M. D. M., Correia, J. C. & Albuquerque, C. M. R. D. Impact of small variations in temperature and humidity on the reproductive activity and survival of *Aedes aegypti* (Diptera, Culicidae). *Revista Brasileira de Entomologia* **54**, 488-493, doi:10.1590/S0085-56262010000300021 (2010).

98 Ricupero, M. *et al.* Combined thermal and insecticidal stresses on the generalist predator *Macrolophus pygmaeus*. *Science of The Total Environment* **729**, 138922, doi:10.1016/j.scitotenv.2020.138922 (2020).

99 Harvey, B. P., Gwynn-Jones, D. & Moore, P. J. Meta-analysis reveals complex marine biological responses to the inter active effects of ocean acidification and warming. *Ecology and Evolution* **3**, 1016-1030, doi:10.1002/ece3.516 (2013).

100 Parker, L. M., Ross, P. M. & O'Connor, W. A. Comparing the effect of elevated pCO2 and temperature on the fertiliza tion and early development of two species of oysters. *Marine Biology* **157**, 2435-2452, doi:10.1007/s00227-010-1508-3 (2010).

101 Pedersen, S. A. & Hanssen, A. E. Ocean acidification ameliorates harmful effects of warming in primary consumer. *Ecology and Evolution* **8**, 396-404, doi:10.1002/ece3.3526 (2018).

102 Angilletta, M. J. *Thermal Adaptation - A theoretical and empirical synthesis*. (Oxford University Press, 2009).

103 Suckling, C. C. *et al.* Adult acclimation to combined temperature and pH stressors significantly enhances reproductive outcomes compared to short-term exposures. *Journal of Animal Ecology* **84**, 773-784, doi:10.1111/1365-2656.12316 (2015).

104 Porcelli, D. *et al.* Gene expression clines reveal local adaptation and associated trade-offs at a continental scale. *Scientific Reports* **6**, 32975, doi:10.1038/srep32975 (2016).

105 Weaving, H., Terblanche, J. S., Pottier, P. & English, S. Meta-analysis reveals weak but pervasive plasticity in insect thermal limits. *Nature Communications* **13**, 5292, doi:10.1038/s41467-022-32953-2 (2022).

106 Pottier, P. *et al.* Developmental plasticity in thermal tolerance: Ontogenetic variation, persistence, and future directions. *Ecology Letters* **25**, 2245-2268, doi:10.1111/ele.14083 (2022).

107 Pottier, P., Burke, S., Drobniak, S. M., Lagisz, M. & Nakagawa, S. Sexual (in)equality? A meta-analysis of sex differences in thermal acclimation capacity across ectotherms. *Functional Ecology* **35**, 2663-2678, doi:10.1111/1365-2435.13899 (2021).

108 Donelson, J. M., Salinas, S., Munday, P. L. & Shama, L. N. S. Transgenerational plasticity and climate change experiments: Where do we go from here? *Global Change Biology* **24**, 13-34, doi:10.1111/gcb.13903 (2018).

109 Cavieres, G. *et al.* Rapid within- and transgenerational changes in thermal tolerance and fitness in variable thermal landscapes. *Ecology and Evolution* **10**, 8105-8113, doi:10.1002/ece3.6496 (2020).

110 Jarrold, M. D. *et al.* Life-history trade-offs and limitations associated with phenotypic adaptation under future ocean warming and elevated salinity. *Philosophical Transactions of the Royal Society B: Biological Sciences* **374**, 20180428, doi:10.1098/rstb.2018.0428 (2019).

111 Buckley, L. B. *et al.* Characterizing biological responses to climate variability and extremes to improve biodiversity projections. *PLOS Climate* **2**, e0000226, doi:10.1371/journal.pclm.0000226 (2023).

112 Rivera-Rincón, N. *et al.* A comparison of thermal stress response between *Drosophila melanogaster* and *Drosophila pseudoobscura* reveals differences betw een species and sexes. *Journal of Insect Physiology* **153**, 104616, doi:10.1016/j.jinsphys.2024.104616 (2024).

113 Dyck, V. A., Hendrichs, J. & Robinson, A. S. *Sterile insect technique: principles and practice in area-wide integrated pest management*. 2nd edition edn, (CRC Press, 2021).

114 Barclay, H. J. in *Sterile insect technique: principles and practice in area-wide integrated pest management* 201-244 (CRC Press, 2021).

115 Shuster, S. M., Pyzyna, B., Mayer, L. P. & Dyer, C. A. The opportunity for sexual selection and the evolution of non-responsiveness to pesticides, sterility inducers and contraceptives. *Heliyon* **4**, e00943, doi:https://doi.org/10.1016/j.heliyon.2018.e00943 (2018).

116 Chen, I. C., Hill, J. K., Ohlemüller, R., Roy, D. B. & Thomas, C. D. Rapid Range Shifts of Species Associated with High Levels of Climate Warming. *Science* **333**, 1024-1026, doi:10.1126/science.1206432 (2011).

117 Arce-Valdés, L. R. & Sánchez-Guillén, R. A. The evolutionary outcomes of climate-change-induced hybridization in insect populations. *Current Opinion in Insect Science* **54**, 100966, doi:10.1016/j.cois.2022.100966 (2022).

118 Kyogoku, D. When does reproductive interference occur? Predictions and data. *Population Ecology* **62**, 196-206, doi:10.1002/1438-390X.12041 (2020).

119 Matute, D. R., Novak, C. J. & Coyne, J. A. Temperature-based extrinsic reproductive isolation in two species of *Drosophila*. *Evolution* **63**, 595-612, doi:10.1111/j.1558-5646.2008.00588.x (2009).

120 Coyne, J. A., Kim, S. Y., Chang, A. S., Lachaise, D. & Elwyn, S. Sexual isolation between two sibling species with overlapping ranges *Drosophila santomea* and *Drosophila yakuba*. *Evolution* **56**, 2424-2434, doi:10.1111/j.0014-3820.2002.tb00168.x (2002).

121 Servedio, M. R. & Noor, M. A. F. The Role of Reinforcement in Speciation: Theory and Data. *Annual Review of Ecology, Evolution, and Systematics* **34**, 339-364, doi:10.1146/annurev.ecolsys.34.011802.132412 (2003).

122 Matute, D. R. Reinforcement of Gametic Isolation in *Drosophila*. *PLoS Biology* **8**, e1000341, doi:10.1371/journal.pbio.1000341 (2010).

123 Gallegos, C., Hodgins, K. A. & Monro, K. Temperature and sex shape reproductive barriers in a climate change hotspot. *Evolution* **78**, 906-918, doi:10.1093/evolut/qpae012 (2024).

124 Taylor, S. A. & Larson, E. L. Insights from genomes into the evolutionary importance and prevalence of hybridization in nature. *Nature Ecology & Evolution* **3**, 170-177, doi:10.1038/s41559-018-0777-y (2019).

125 Griffiths, J. S., Kawji, Y. & Kelly, M. W. An Experimental Test of Adaptive Introgression in Locally Adapted Populations of Splash Pool Copepods. *Molecular Biology and Evolution* **38**, 1306-1316, doi:10.1093/molbev/msaa289 (2021).

126 Roy, H. E. *et al.* Curbing the major and growing threats from invasive alien species is urgent and achievable. *Nature Ecology & Evolution* **8**, 1216-1223, doi:10.1038/s41559-024-02412-w (2024).

127 Kelley, A. L. The role thermal physiology plays in species invasion. *Conservation Physiology* **2**, cou045-cou045, doi:10.1093/conphys/cou045 (2014).

128 Green, L., Havenhand, J. N. & Kvarnemo, C. Evidence of rapid adaptive trait change to local salinity in the sperm of an invasive fish. *Evolutionary Applications* **13**, 533-544, doi:10.1111/eva.12859 (2020).

129 Hoffmann, A. A., Sgrò, C. M. & Van Heerwaarden, B. Testing evolutionary adaptation potential under climate change in inve rtebrates (mostly *Drosophila*): findings, limitations and directions. *Journal of Experimental Biology* **226**, jeb245749, doi:10.1242/jeb.245749 (2023).

130 Cammack, K. M., Mesa, H. & Lamberson, W. R. Genetic variation in fertility of heat-stressed male mice. *Theriogenology* **66**, 2195-2201, doi:10.1016/j.theriogenology.2006.06.011 (2006).

131 Kovuri, P., Yadav, A. & Sinha, H. Role of genetic architecture in phenotypic plasticity. *Trends in Genetics* **39**, 703-714, doi:10.1016/j.tig.2023.04.002 (2023).

132 Santos, M. A. *et al.* No evidence for short-term evolutionary response to a warming environment in *Drosophila*. *Evolution* **75**, 2816-2829, doi:10.1111/evo.14366 (2021).

133 Kellermann, V. & Van Heerwaarden, B. Terrestrial insects and climate change: adaptive responses in key traits. *Physiological Entomology* **44**, 99-115, doi:10.1111/phen.12282 (2019).

134 Bennett, J. M. *et al.* The evolution of critical thermal limits of life on Earth. *Nature Communications* **12**, 1198, doi:10.1038/s41467-021-21263-8 (2021).

135 Sales, K., Gage, M. J. G. & Vasudeva, R. Experimental evolution reveals that males evolving within warmer thermal regimes improve reproductive performance under heatwave conditions in a model insect. *Journal of Evolutionary Biology* **37**, 1329-1344, doi:10.1093/jeb/voae116 (2024).

136 Walsh, B. & Lynch, M. *Evolution and selection of quantitative traits*. (Oxford university press, 2018).

137 Berger, D., Stångberg, J., Grieshop, K., Martinossi-Allibert, I. & Arnqvist, G. Temperature effects on life-history trade-offs, germline maintenance and mutation rate under simulated climate warming. *Proceedings of the Royal Society B: Biological Sciences* **284**, 20171721, doi:10.1098/rspb.2017.1721 (2017).

138 Blanckenhorn, W. U. The Evolution of Body Size: What Keeps Organisms Small? *The Quarterly Review of Biology* **75**, 385-407, doi:10.1086/393620 (2000).

139 Chakraborty, A. *et al.* Within-population variation in body size plasticity in response to combined nutritional and thermal stress is partially independent from var iation in development time. *Journal of Evolutionary Biology* **36**, 264-279, doi:10.1111/jeb.14099 (2023).

140 Antunes, M. A. *et al.* Body size decline during thermal evolution is only detected at mild temperature. *Proceedings of the Royal Society B: Biological Sciences* **291**, 20241498, doi:10.1098/rspb.2024.1498 (2024).

141 Rogell, B. *et al.* Sex-dependent evolution of life-history traits following adaptation to climate warming. *Functional Ecology* **28**, 469-478, doi:10.1111/1365-2435.12179 (2014).

142 Gardner, J. L., Peters, A., Kearney, M. R., Joseph, L. & Heinsohn, R. Declining body size: a third universal response to warming? *Trends in Ecology & Evolution* **26**, 285-291, doi:10.1016/j.tree.2011.03.005 (2011).

143 Berger, D., Stångberg, J., Baur, J. & Walters, R. J. Elevated temperature increases genome-wide selection on de novo mutations. *Proceedings of the Royal Society B: Biological Sciences* **288**, 20203094, doi:10.1098/rspb.2020.3094 (2021).

144 Swings, T. *et al.* Adaptive tuning of mutation rates allows fast response to lethal stress in *Escherichia coli*. *eLife* **6**, e22939, doi:10.7554/eLife.22939 (2017).

145 Deutsch, C. A. *et al.* Impacts of climate warming on terrestrial ectotherms across latitude. *Proceedings of the National Academy of Sciences* **105**, 6668-6672, doi:10.1073/pnas.0709472105 (2008).

146 Hoffmann, A. A., Chown, S. L. & Clusella-Trullas, S. Upper thermal limits in terrestrial ectotherms: how constrained are they? *Functional Ecology* **27**, 934-949, doi:10.1111/j.1365-2435.2012.02036.x (2013).

147 Duffy, K., Gouhier, T. C. & Ganguly, A. R. Climate-mediated shifts in temperature fluctuations promote extinction risk. *Nature Climate Change* **12**, 1037-1044, doi:10.1038/s41558-022-01490-7 (2022).

148 Porcelli, D., Gaston, K. J., Butlin, R. K. & Snook, R. R. Local adaptation of reproductive performance during thermal stress. *Journal of Evolutionary Biology* **30**, 422-429, doi:10.1111/jeb.13018 (2017).

149 Svensson, E. I. *et al.* Heritable variation in thermal profiles is associated with reproductive success in the world's largest bird. *Evolution Letters* **8**, 200-211, doi:10.1093/evlett/qrad049 (2024).

150 Katzenberger, M., Duarte, H., Relyea, R., Beltrán, J. F. & Tejedo, M. Variation in upper thermal tolerance among 19 species from temperate wetlands. *Journal of Thermal Biology* **96**, 102856, doi:10.1016/j.jtherbio.2021.102856 (2021).

151 Diamond, S. E. *et al.* Heat tolerance predicts the importance of species interaction effects as the climate changes. *Integrative and Comparative Biology* **57**, 112-120, doi:10.1093/icb/icx008 (2017).

152 Toledo, I. M., Dahl, G. E. & De Vries, A. Dairy cattle management and housing for warm environments. *Livestock Science* **255**, 104802, doi:10.1016/j.livsci.2021.104802 (2022).

153 Angilletta, M. J. Estimating and comparing thermal performance curves. *Journal of Thermal Biology* **31**, 541-545, doi:10.1016/j.jtherbio.2006.06.002 (2006).

154 Buckley, L. B., Huey, R. B. & Kingsolver, J. G. Asymmetry of thermal sensitivity and the thermal risk of climate chang e. *Global Ecology and Biogeography* **31**, 2231-2244, doi:10.1111/geb.13570 (2022).

155 Rezende, E. L. & Bozinovic, F. Thermal performance across levels of biological organization. *Philosophical Transactions of the Royal Society B: Biological Sciences* **374**, 20180549, doi:10.1098/rstb.2018.0549 (2019).

156 Jørgensen, L. B., Ørsted, M., Malte, H., Wang, T. & Overgaard, J. Extreme escalation of heat failure rates in ectotherms with global war ming. *Nature* **611**, 93-98, doi:10.1038/s41586-022-05334-4 (2022).

157 Rezende, E. L., Bozinovic, F., Szilágyi, A. & Santos, M. Predicting temperature mortality and selection in natural *Drosophila* populations. *Science* **369**, 1242-1245, doi:doi:10.1126/science.aba9287 (2020).

158 Verberk, W. C. E. P., Hoefnagel, K. N., Peralta-Maraver, I., Floury, M. & Rezende, E. L. Long-term forecast of thermal mortality with climate warming in riverine amphipods. *Global Change Biology* **29**, 5033-5043, doi:https://doi.org/10.1111/gcb.16834 (2023).

159 Arnold, P. A. *et al.* A framework for modelling thermal load sensitivity across life. *Global Change Biology* **31**, e70315, doi:https://doi.org/10.1111/gcb.70315 (2025).

160 Candolin, U. & Heuschele, J. Is sexual selection beneficial during adaptation to environmental change? *Trends Ecol Evol* **23**, 446-452, doi:10.1016/j.tree.2008.04.008 (2008).

161 Parrett, J. M. *et al.* Genomic evidence that a sexually selected trait captures genome-wide variation and facilitates the purging of genetic load. *Nature Ecology & Evolution* **6**, 1330-1342, doi:10.1038/s41559-022-01816-w (2022).

162 Emlen, S. T. & Oring, L. W. Ecology, sexual selection, and the evolution of mating systems. *Science* **197**, 215-223, doi:10.1126/science.327542 (1977).

163 Willink, B., Ho, T. A. T. & Svensson, E. I. Ecology and sexual conflict drive the macroevolutionary dynamics of female-limited color polymorphisms in damselflies. *Proceedings of the National Academy of Sciences* **122**, e2503400122, doi:doi:10.1073/pnas.2503400122 (2025).

164 Perry, J. C. & Rowe, L. Sexual conflict in its ecological setting. *Philosophical Transactions of the Royal Society B: Biological Sciences* **373**, 20170418, doi:doi:10.1098/rstb.2017.0418 (2018).

165 García-Roa, R., Garcia-Gonzalez, F., Noble, D. W. A. & Carazo, P. Temperature as a modulator of sexual selection. *Biological Reviews* **95**, 1607-1629, doi:10.1111/brv.12632 (2020).

166 Walsh, B. S., Mannion, N. L. M., Price, T. A. R. & Parratt, S. R. Sex-specific sterility caused by extreme temperatures is likely to create cryptic changes to the operational sex ratio in *Drosophila virilis*. *Current Zoology* **67**, 341-343, doi:10.1093/cz/zoaa067 (2020).

167 Winkler, L., Eilhardt, R. & Janicke, T. Population density affects sexual selection in an insect model. *Functional Ecology* **37**, 2734-2747, doi:https://doi.org/10.1111/1365-2435.14410 (2023).

168 Costa, S. G., Magalhães, S. & Rodrigues, L. R. Multiple mating rescues offspring sex ratio but not productivity in a haplodiploid exposed to developmental heat stress. *Functional Ecology* **37**, 1291-1303, doi:https://doi.org/10.1111/1365-2435.14303 (2023).

169 Cook, P. A., Costello, R. A., Brodie III, E. D. & Formica, V. Population age structure shapes selection on social behaviour in a long-lived insect. *Philosophical Transactions of the Royal Society B: Biological Sciences* **379**, 20230331, doi:doi:10.1098/rstb.2023.0331 (2024).

170 Schacht, R. *et al.* Adult sex ratios: causes of variation and implications for animal and human societies. *Communications Biology* **5**, 1273, doi:10.1038/s42003-022-04223-w (2022).

171 Svensson, E. I. Eco-evolutionary dynamics of sexual selection and sexual conflict. *Functional Ecology* **33**, 60-72, doi:https://doi.org/10.1111/1365-2435.13245 (2019).

172 Pembury Smith, M. Q. R., Latkova, L. & Snook, R. R. Facultative polyandry under heat stress and the evolutionary potential for climate-driven shifts in mating systems. *Heredity*, doi:10.1038/s41437-025-00795-5 (2025).

173 Kotiaho, J. S., Simmons, L. W. & Tomkins, J. L. Towards a resolution of the lek paradox. *Nature* **410**, 684-686, doi:10.1038/35070557 (2001).

174 Pembury Smith, M. Q. R., Trojmar, D., Gotthard, K., Wiklund, C. & Snook, R. R. Sex-specific variation in thermal sensitivity has multiple negative effects on reproductive trait performance. *Journal of Animal Ecology* **94**, 943-957, doi:https://doi.org/10.1111/1365-2656.70026 (2025).

175 De Nardo, A. N. *et al.* Experimental evolution reveals trade-offs between sexual selection and heat tolerance in *Drosophila prolongata*. *Evolution* **79**, 823-836, doi:10.1093/evolut/qpaf035 (2025).

176 Koppik, M., Baur, J. & Berger, D. Increased male investment in sperm competition results in reduced maintenance of gametes. *PLOS Biology* **21**, e3002049, doi:10.1371/journal.pbio.3002049 (2023).

177 Plesnar-Bielak, A., Skrzynecka, A. M., Prokop, Z. M. & Radwan, J. Mating system affects population performance and extinction risk under environmental challenge. *Proceedings of the Royal Society B: Biological Sciences* **279**, 4661-4667, doi:10.1098/rspb.2012.1867 (2012).

178 Parrett, J. M. & Knell, R. J. The effect of sexual selection on adaptation and extinction under increasing temperatures. *Proceedings of the Royal Society B: Biological Sciences* **285**, 20180303, doi:10.1098/rspb.2018.0303 (2018).

179 Iglesias-Carrasco, M. *et al.* Sexual selection buffers the negative consequences of population fragmentation on adaptive plastic responses to increasing temperatures. *Evolution* **78**, 86-97, doi:10.1093/evolut/qpad193 (2024).