A framework for modelling thermal load sensitivity across life

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Abstract

Forecasts of vulnerability to climate warming require an integrative understanding of how species are exposed to, are damaged by, and recover from thermal stress in natural environments. The sensitivity of species to temperature depends on the frequency, duration, and magnitude of thermal stress. Thus, there is a generally recognised need to move beyond physiological metrics based solely on critical thermal limits and integrate them with natural heat exposure regimes. Here we propose the Thermal Load Sensitivity (TLS) framework, which integrates biophysical principles for quantifying exposure with physiological principles of the dynamics of damage and repair processes in driving sublethal impacts on organisms. Building upon the established Thermal Death Time (TDT) model, which integrates both the magnitude and duration of stress, the TLS framework attempts to disentangle accumulation of damage and subsequent repair processes that alter responses to thermal stress. With the aid of case studies and reproducible simulation examples, we discuss how the TLS framework can be applied to enhance our understanding of the ecology and evolution of heat stress responses. These include assessing thermal sensitivity across diverse taxonomic groups, throughout ontogeny, and for modular organisms, as well as integrating additional stressors in combination with temperature. We identify critical research opportunities, knowledge gaps, and new ways of integrating physiological measures of thermal sensitivity to improve forecasts of thermal vulnerability.

I. Introduction

Climate change is exposing species not just to gradual warming but also increases to the frequency and severity of extreme heat events that impose physiological stress on organisms. Thermal vulnerability to heat stress depends on two key processes – exposure and sensitivity (Huey et al., 2012; Williams et al., 2008). Exposure reflects the extent to which organisms experience a potentially stressful environmental change. It is the outcome of the interaction between environmental factors and characteristics of the organism that determine body temperature. Exposure also incorporates the organism's ability to select microenvironments. New developments in the field of biophysical ecology have largely resolved the conceptual and technical barriers to predicting exposure to heat stress, though uptake of these methods has been gradual (Briscoe et al., 2023; Buckley & Kingsolver, 2021). Sensitivity describes the thermal responsiveness of an organism to temperature stress that are leads to physiological damage or death (Clusella-Trullas et

al., 2021; Jørgensen et al., 2022); it depends on life history and physiology (Buckley & Kingsolver, 2021). These factors are not independent: sensitivity can be moderated by the dynamics of exposure (intensity and duration) and by the capacity of species' thermal physiology to recover from thermal stress. We therefore need a general, quantitative framework to capture the physiological mechanisms of both damage and recovery if we are to effectively predict how increasingly erratic thermal regimes will impact function, survival, and reproduction of organisms.

Approaches to assessing thermal sensitivity vary across taxonomic groups and research fields (Bennett et al., 2018; Geange et al., 2021). Assessments of static endpoints, such as critical thermal limits and the quantification of the cumulative impact of prolonged exposure to different (potentially stressful) temperature regimes, are common procedures (Klockmann et al., 2017). The large variation in body size and lifespan among organisms affect the feasibility of measuring thermal tolerance consistently; it is necessarily assessed on vastly different life stages (e.g., fruits, seeds, eggs, larvae, adults) and on different scales, from components of an individual (e.g., leaves, flowers), to whole individuals, and populations (e.g., bacterial colonies, *Drosophila* populations, soil seed banks) (Klockmann et al., 2017; Wahid et al., 2007).

Effective assessment of the thermal vulnerability of populations thus requires an integrated knowledge of the mechanisms by which temperature-induced damage leads to functional incapacitation, reproductive failure, or death in individuals. In many cases, assessing lethal limits is not possible for logistical or ethical reasons (e.g., in vertebrates, or rare and long-lived species) and may not even be desired, given that we should be interested in detecting vulnerability at ecologically relevant thresholds prior to thermal death. To overcome this, researchers apply a range of proxies, such as thermal limits of biological processes, changes to activity budgets, and assessment of damage and mortality during extreme climatic events in nature (Marchin et al., 2022b; Sinervo et al., 2010; Welbergen et al., 2008). There is need for developing integrative probabilistic and mechanistic models to characterise physiological responses to temperature with predictions that can be empirically tested and validated.

Here we demonstrate the potential to combine physiological models of thermal sensitivity with general models of exposure dynamics to enhance our ability to understand and predict the effects of temperature on organisms. We use example cases to illustrate why considering repair together with damage is essential, and to highlight potential uses for the framework across disparate taxonomic groups and life stages to generate useful and testable predictions in the face of rapid global change. We identify key targets for focussed research, whereby taking a unified

approach with standardised terminology should improve predictive capacity.

II. Thermal Death Time (TDT) models explicitly incorporate duration of heat exposure

Critical thermal limits (e.g., CT_{max}) have been used widely as static point thresholds or endpoints to represent the temperature at which physiological processes cease to function (Bennett et al., 2018). In some cases, critical temperatures are explicitly lethal (Lutterschmidt & Hutchison, 1997), but they can also range from the temperature at which an insect can no longer right itself or is knocked down (van Heerwaarden et al., 2016), to onsets of spasms in lizards (Taylor et al., 2021), loss of equilibrium in fish (Ern et al., 2023), or dysfunction of photosynthetic machinery in plants (Arnold et al., 2021). For some comparative research questions, there are benefits to using point estimates as they are relatively easy to obtain, which permits large comparisons of thermal tolerances among taxa (Bennett et al., 2021; Camacho et al., 2024; Sunday et al., 2011) or sites (Dewenter et al., 2024; Sunday et al., 2019). The use of different indices and limitations of point estimates and endpoints, like CT_{max}, have been comprehensively reviewed and critiqued since at least the 1990s (Clusella-Trullas et al., 2021; Jørgensen et al., 2021; Jørgensen et al., 2019; Lutterschmidt & Hutchison, 1997; Ørsted et al., 2022; Rezende et al., 2020; Rezende et al., 2014; Santos et al., 2011; Terblanche et al., 2011). The consistent opinion from these works is that derived point estimates – which are often collapsed into a mean lethal temperature for a population – can be dependent on methodological differences (e.g. in heating rate; Arnold et al., 2021; Payne et al., 2025). Consequently, variance from non-biological sources can be high, and calls into question the validity of broad comparative studies that use vastly different methods without adjusting for these (discussed in Perez et al., 2021).

Finding a singular temperature threshold to define thermal limits inherently overlooks the interplay between the intensity and duration of temperature exposure that leads to compounding physiological dysfunction (Hochachka & Somero, 2002; Jørgensen et al., 2021; Michaelsen et al., 2021; Rezende et al., 2020; Rezende et al., 2014). The need to explicitly capture the intensity and duration of exposure (also referred to as thermal dosage, cumulative heat sum, heat load, or heat dose), along with integrating such information with dynamic, realistic thermal environments, have all led to the rise of the Thermal Death Time (TDT) model in ecology.

The TDT is not a new concept – it was first explicitly introduced in the 1920s to ensure

147 that bacteria were killed during the canning process of food (Ball, 1923). Subsequently, it has been
148 applied to ectothermic animals to estimate survival times under various acclimation and exposure
149 temperatures (Maynard Smith, 1957; Mellanby, 1954). Relating thermal tolerance with exposure
150 time re-emerged as a contemporary tool in thermal ecology in the past two decades (Armstrong et
151 al., 2009; Rezende et al., 2014; Santos et al., 2011). Essentially, the TDT became an extension of
152 the typical thermal performance curve – which provides insight into the optimal temperature,
153 upper and lower limits, and temperature breadth for performance (Angilletta, 2006; 2009) – with
154 the added dimension of exposure time to thermal stress (Rezende et al., 2014). The TDT model
155 has since been applied to several insects to understand thermal impacts on fertility and survival
156 (e.g., Ørsted et al., 2024; Youngblood et al., 2025). It has also been applied to plants to optimise
157 weed management in agriculture where thermal treatments were applied to soil to eradicate weed
158 seeds (Dahlquist et al., 2007) and to determine the effects of thermal load on the function of
159 photosystems (Cook et al., 2024).
160 The TDT explicitly models how both exposure time and exposure temperature affect lethal
161 limits (e.g., LT_{50} – the lethal temperature limit when 50% mortality occurs), which captures such
162 relationships as:
163
164 $$T = CT_{max1h} - z \cdot \log_{10}(t) \quad \text{(Equation 1)}$$
165
166 where, T = temperature for, say, 50% mortality (LT_{50}), CT_{max1h} is the critical thermal maximum
167 (°C), z = thermal sensitivity and t = time (in hours) before reaching the 50% damage threshold.
168 Note that because $\log_{10}(1) = 0$, the intercept of Equation 1, CT_{max1h}, corresponds to the lethal
169 temperature for 1 h of exposure. While we standardise CT_{max} to 1 h, time can be scaled to other
170 units (e.g., minutes) depending on what is biologically relevant to the organism's ecology. Given
171 that survival follows a typical dose-response curve, logarithmic transformation makes the
172 relationship between lethal temperature and time approximately linear (Rezende et al., 2014).
173 Alternatively, we can flip the axes to account for the fact that temperature is the main
174 factor manipulated in experiments allowing one to re-parametrise the TDT curve as follows:
175
176 $$\log_{10}(t) = \alpha + \beta \cdot T \quad \text{(Equation 2)}$$
177
178 In the above equation, time to reach 50% mortality, t, is on the y-axis and temperature, T, on the x-

axis. We can recover CT_{max1h} and z by back-transformation using the new slope (β) and intercept (α) from this relationship as follows: $CT_{max1h} = -\frac{\alpha}{\beta}$ and $z = -\frac{1}{\beta}$. The parameterisation of the TDT curve as in Equation 2 is useful because it allows one to capture how damage accumulates over time as follows (see Jørgensen et al., 2021; Ørsted et al., 2024):

$$Accumulated\ damage = \sum_{i=1}^{T_e>T_c} \frac{100 \cdot (t_{i+1} - t_i)}{10^{(\beta \cdot \max(T_i; T_{i+1}) + \alpha)}} \quad \text{(Equation 3)}$$

where the equation calculates the accumulated damage (as a %) from time, t_i to time t_{i+1}, using the parameters from the TDT curve (Equation 2). The accumulated damage function assumes overheating risk and injury occurrence when T_e (the exposure temperature) exceeds T_c (the assumed critical temperature above which heat injury accumulates) (Ørsted et al., 2024). When the accumulated damage reaches 100%, the lethal limit (that is, the defined threshold; LT_{50} in this example) has been reached.

Potential for extending the TDT model to explore sublethal effects

Generally, TDT models are sensitive to the chosen endpoint, are phenomenological in nature, are usually quantified at the whole-organism level, and they assume that survival declines exponentially with exposure duration. However, mortality may not occur immediately under moderately stressful temperatures and there can be both direct and immediate effects on other fitness components (Buckley & Huey, 2016). It is also possible that organisms can cope with moderately stressful temperatures for a relatively long time, where survival remains at 100%, before they suddenly succumb to the stress (e.g., Gómez-Gras et al., 2022). The thermal conditions that organisms are exposed to during their development and at crucial life stages prior to – or in conjunction with – heat stress can substantially alter fitness outcomes beyond simple mortality. Generating predictions from dose-response curves could allow for a range of different limit thresholds to be used. For example, sublethal measurements (e.g., critical fertility limits and functional inhibition thresholds) can be used in conjunction with and can extend the value of TDT models (Cook et al., 2024; Faber et al., 2024; Ørsted et al., 2024).

While predictions for mortality thresholds align well with empirical data in ramping assays they may not predict the survival probability curve if temperatures fluctuate (Rezende et al., 2020). This is partly due to the unknown capacity for repair processes to offset damage or injury

accumulation during reprieves from damaging temperatures (Huey & Kearney, 2020; Jørgensen et al., 2021; Ørsted et al., 2022). Dynamic, probabilistic modelling approaches attempt to circumvent this problem, and they seem to predict mortality under fluctuating conditions quite well when the empirical survival curves obtained at constant temperatures are adequately described (Rezende et al., 2020). These approaches offer exciting potential and will require additional empirical study to validate the net effect of damage-repair processes on physiological function that determine survival probability, and what other impacts these – and other natural, interacting processes – have on the fitness of individuals and populations. TDT does not provide much insight into the amelioration of thermal stress (although recent studies are exploring acclimation, e.g., Baeza Icaza et al., 2025; Wehrli et al., 2024; Youngblood et al., 2025), which is a function of damage, repair, and acclimation. For this reason and for linguistic accuracy as the framework is used for broader applications, we propose a conceptual renaming of Thermal *Death* Time to Thermal Load Sensitivity (TLS) when used as a general framework that is inclusive of non-lethal measures and applied to different organisms.

III. Damage and repair: The physiological cost of extreme temperatures

The TLS framework allows for modelling approaches to be integrated with, or used to predict, both lethal and sublethal limits (i.e., not necessitating *death* as in Thermal Death Time). It places specific emphasis on *disentangling* the processes of damage and repair through time in dynamic environmental conditions. Specifically, we make the distinction that damage accumulates *during* stress, and may be increasingly apparent *following* stress, while repair occurs *during* as well as *between* stresses, and the relative magnitude of these processes determines the extent to which the organism recovers at a given time point (Buckley et al., 2025; Williams et al., 2016). The shift to a TLS perspective is important as we progress our understanding of the effects of thermal stress accumulation, variability, and extremes on vital physiological processes that in turn affect demographic and ecological processes. There is growing empirical evidence of the important role of recovery from physiological damage following thermal stress (Bai et al., 2019; Curtis et al., 2014; Malmendal et al., 2006).

Ørsted et al. (2022) reviewed the nature of damage processes in ectotherms that occur beyond the 'permissive' temperature range in which normal function is possible (i.e., the 'stressful' range). As homeostasis is disrupted under thermal stress, there is a balance of two

antagonistic processes: damage (injury accumulation) and repair. It is assumed that these processes may occur simultaneously; they both depend on the severity and duration of the thermal stress and legacy or carryover effects of environmental conditions prior to and following thermal stress (Buckley et al., 2025; Ørsted et al., 2022). If damage accumulates from a given heat load, it will need to be partially or completely repaired to re-establish homeostasis, both during and after cessation of stressful conditions. The buildup of heat load over longer time periods will not only result in damage accumulation but will also limit the extent of repair (Ørsted et al., 2022). Life processes are governed by complex chemical transformations within and between cells mediated by protein and membrane integrity. Mechanisms of heat damage generally involve increasingly misfolded or unfolded proteins (Feder & Hofmann, 1999; Wahid et al., 2007) and oxidative damage to DNA, lipids, and proteins that ultimately compromises cellular function (Georgieva & Vassileva, 2023; Hasanuzzaman et al., 2013; Ritchie & Friesen, 2022; Tuteja et al., 2001). All these phenomena are influenced by temperature through the laws of thermodynamics (Michaletz & Garen, 2024).

The TDT model captures repair and damage occurring simultaneously in the stressful range (where damage outweighs repair) through z, but repair will be far more important and impactful outside of the stressful range. Repair mechanisms can be diverse, but many are thought to be conserved between plants and animals (Tuteja et al., 2001). They include the regulation of shock proteins and other chaperone proteins to refold or to degrade misfolded proteins (Liu & Howell, 2016; Wahid et al., 2007). Repair pathways are known for excising damaged DNA resulting from bursts of oxidative stress (e.g., base excision repair; Tuteja et al., 2001) and replacement of oxidised fatty acids (e.g., Wagner & Chitnis, 2023), but the details of repair are less well understood compared to factors contributing to damage. Repair rates are known to be temperature-dependent in flies (Bowler & Kashmeery, 1979; Dingley & Maynard Smith, 1968; Ørsted et al., 2022), bacteria (Iandolo & Ordal, 1966; McKellar et al., 1997), and plants (Curtis et al., 2014).

Theoretical advances allow for simulations of the dynamics of physiological damage and repair depending on temperature (Michaletz & Garen, 2024), which are needed to predict sensitivity and vulnerability to stress in nature (Ørsted et al., 2024). For example, Klanjscek et al. (2016) developed a damage and repair model for oxidative stress that could potentially be applied to heat stress. Jørgensen et al. (2021) developed a mathematical model for estimating accumulated injury from thermal stress using static and dynamic knockdown data in TDT models. Rezende et al. (2020) also showed that dynamic TDT models, which assume that individuals that survived the

thermal stress can repair damage between bouts of heat stress (e.g., overnight), could estimate survival probability of drosophilids in the laboratory and field. Such studies are foundational to test and validate that there is a dynamic interplay between damage and repair processes through exposure to thermal stress that varies in frequency, duration, and intensity.

Modelling the dynamics of damage and repair using the Thermal Load Sensitivity (TLS) framework

Modelling damage and repair in natural ecosystems requires us to connect physiological sensitivity with realistic thermal exposure at sufficiently fine resolution. Biophysical models can now approximate microclimates at hourly resolution globally, which can be coupled with models of thermoregulatory behaviours to predict operational temperatures of organisms (Kearney et al., 2020; Kearney & Leigh, 2024; Klinges et al., 2022; Meyer et al., 2023). This relatively new capacity to predict the temperatures to which organisms are exposed can be combined in the TLS framework to make more nuanced predictions of risk to thermal stress at fine scales or under scenarios with dynamic and extreme environmental conditions. The inclusion of damage and repair enables the cumulative impacts of thermal stress to be modelled under natural, fluctuating conditions including stress and reprieve. The rate of repair and the decay in the rate of repair, resulting from temperature stress or reduced physiological condition, can both be explicitly incorporated into simulations using the TLS framework. Such feedback processes are expected to alter organism function and homeostasis during exposure to heat stress and benign temperatures that facilitate repair (e.g., overnight or during periods of reprieve from heat).

To illustrate how the feedback processes of damage and repair could play out theoretically, we simulated the effects of temperature on physiological function while altering repair rates and their dependence on physiological function (additional details in Supporting Information). We estimated the thermal sensitivity of a hypothetical ectotherm (Fig. 1a), then simulated damage rate increasing rapidly with temperature (Fig. 1b). We applied a Sharpe-Schoolfield Arrhenius model to simulate repair rates based on a repair rate coefficient ($\dot{k}$) to set the rate of repair at 20°C (Fig. 1c).

It is essential to recognise that damage and repair have non-linear relationships with temperature and that both processes will occur simultaneously. Outside the stressful range of temperatures, repair outstrips damage, whereas inside the stressful range, damage outstrips repair. TDT focuses mainly on the balance within the stressful zone but ignores repair outside the

303 stressful range, within the permissive range. Although damage may be the net result of exposure to
304 high temperature, repair processes such as protein synthesis and chaperoning to limit protein
305 misfolding, are occurring whenever temperatures permit (Santra et al., 2019). Therefore, we
306 calculated the damage/repair ratio (Fig. 1d), and the net damage rate (Fig. 1e), based on the
307 balance between damage and repair at different temperatures, to predict the range of temperatures
308 across which damage outweighs repair and *vice versa*. The processes that facilitate repair are
309 likely also dependent on physiological condition, such that the repair rate itself declines when an
310 organism is in poor physiological condition from accumulating thermal damage (Fig. 1f).
311 We applied this model to gridded hourly estimates of air temperature from the
312 *microclimOZ* dataset (Kearney, 2019) to predict body temperatures of our hypothetical ectotherm
313 for four weeks, including three days that reach damaging extreme temperatures (Fig. 1g).
314 Predicted body temperatures were assumed to equal shaded air temperature, as in a small insect
315 (note that heat budgets can be computed with the ectotherm model of *NicheMapR* (Kearney &
316 Porter, 2020) for more complex scenarios where this simplifying assumption would not hold).
317 Next, we integrated repair rate into probabilistic dynamic thermal 'tolerance landscape' models
318 (Rezende et al., 2020). Note that the actual magnitude of the thermal stress is contingent on the
319 temperature trajectories throughout the day. Thus, we simulate how the cumulative dosage of
320 sublethal heat stress compromises physiological function, which is altered by (and further alters)
321 the balance between damage and repair during the thermal regime (Fig. 1h). Finally, we visualised
322 the assumed dependence of repair rate on physiological condition as a feedback process that
323 reduces the repair rate coefficient ($\dot{k}$) when damage accumulates from exposure to heat (Fig. 1i;
324 details in Supporting Information). Box 1 provides an example application of the TLS framework
325 incorporating damage and repair feedback for *Drosophila suzukii*, and an additional example for
326 weed seeds is provided in the Supporting Information.
327

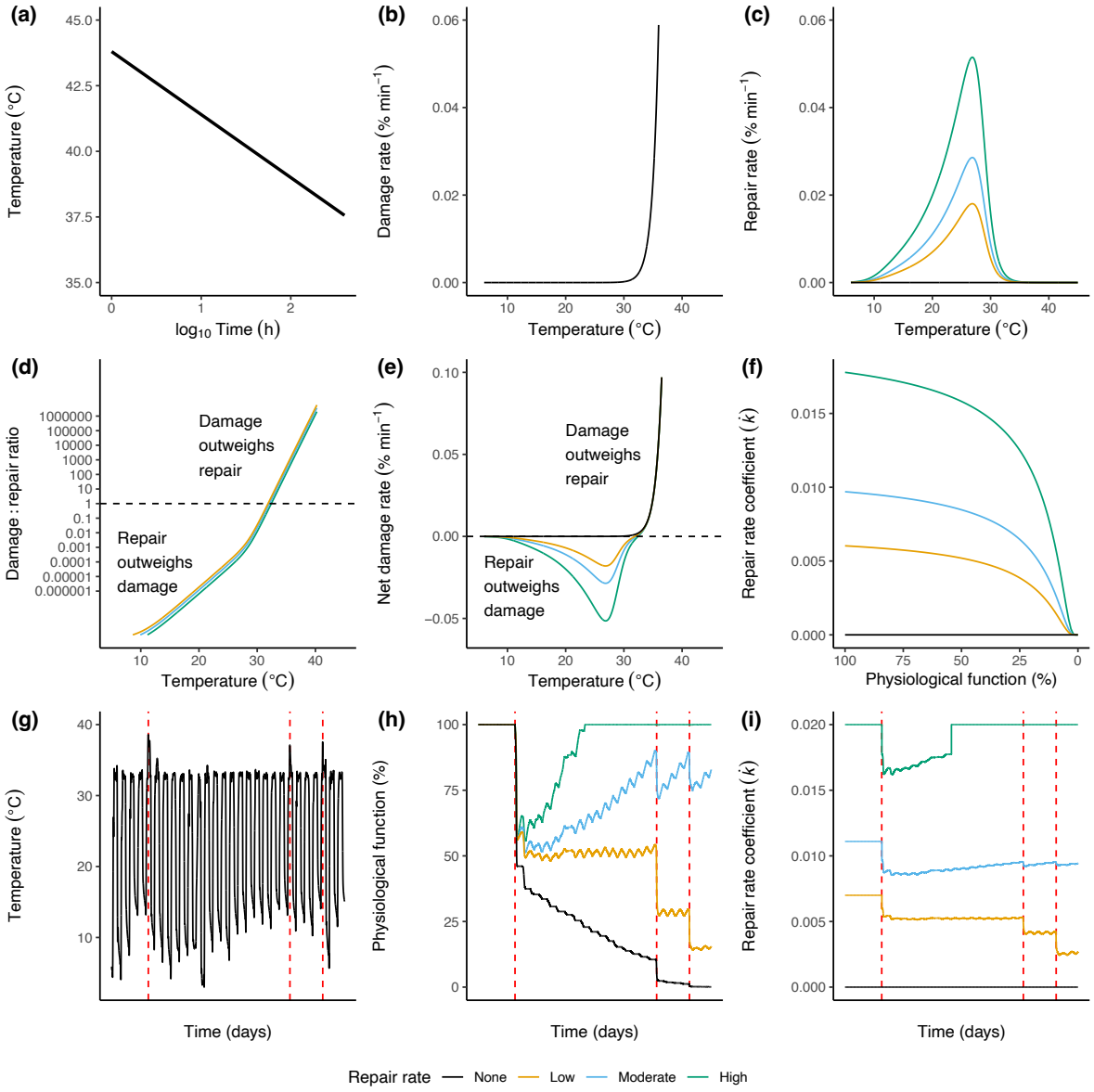


Figure 1. Simulations of the counteracting processes of damage and repair during heat exposure of a hypothetical ectotherm. (**a**) The underlying thermal sensitivity curve for the ectotherm with intercept CT_{max1h} (critical thermal maximum of 1 h of exposure) and slope z (thermal sensitivity) parameters. (**b**) Damage rate as an approximately exponential function of temperature. (**c**) Repair rates as a function of temperature, simulated for the hypothetical ectotherm with no (black), low (orange), moderate (blue), and high (green) repair capacity using Arrhenius functions. (**d**) The damage/repair ratio as a function of temperature, where the dashed black line represents a 1:1 damage/repair ratio. (**e**) The net damage rate as a function of temperature (the balance between damage and repair processes), where the dashed black line represents equal damage and ratio. (**f**) The repair rate coefficient ($\dot{k}$), which is the rate of repair at 20°C, as a function of the organism's physiological function. (**g**) The modelled body temperature during summer over a four-week time course. Dashed red lines in panels **g–i** represent extreme heat days during the time course. (**h**) Physiological function (%), the proportion of full performance possible following exposure to physiological stress that accumulates over the time course, simulated with different repair rates using the TLS framework, illustrating how this response may substantially impact the outcome of thermal stress events over time. (**i**) The dependence of repair rate coefficient ($\dot{k}$) on physiological function over the four-week time course.

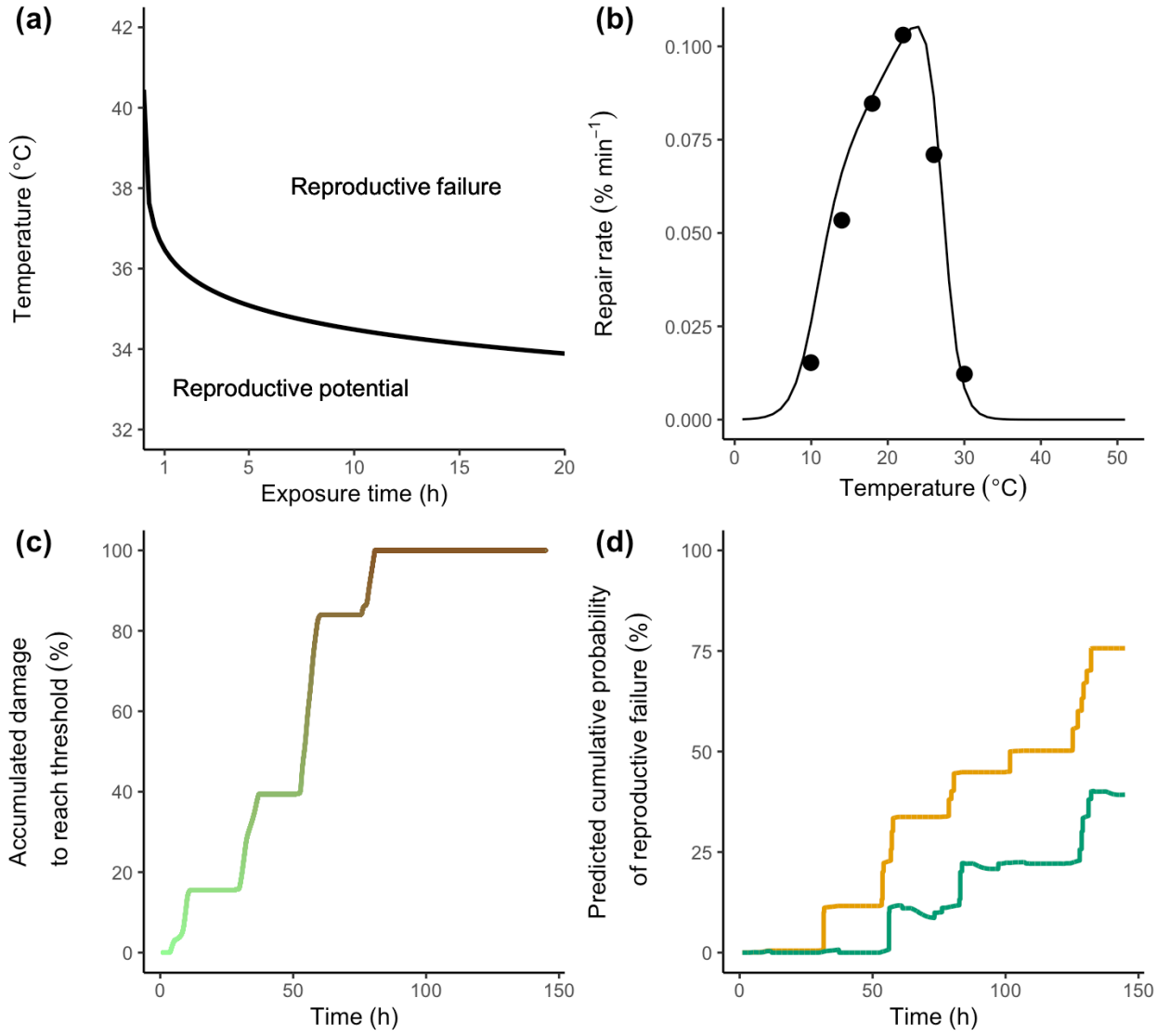
Box 1. Application of the TLS framework to *Drosophila suzukii*

Drosophila suzukii is a globally invasive pest that is a prime candidate species for studies of thermal load sensitivity. We used raw data for productivity of female flies from Ørsted et al. (2024) to explore damage accumulation and repair under combinations of temperature and exposure duration. Productivity of females is a crucial (sublethal) contributor to population viability that is more sensitive to temperature than thermal coma or death.

Using these data, we show how the relationship between temperature and exposure duration determines the conditions under which reproduction can potentially occur or fail (Box 1 Figure a). To illustrate the potential for repair to alter heat failure rates and outcomes, we used *metaDigitise* (Pick et al., 2019) in the R Environment for Statistical Computing v4.3.1 (R Core Team, 2023) to digitise Fig. 5c from Ørsted et al. (2022), extract preliminary repair values (%) at six 'repair temperatures' for *D. suzukii*, and convert them to repair rate per minute (% min^{-1}). These repair values correspond to the improvement of knockdown time relative to a first heat exposure after 6 h of recovery at different temperatures to allow for repair before another knockdown assay. We recognise that these data are preliminary and correspond to knockdown rather than reproductive viability (Ørsted et al., 2022), but there is little empirical data on temperature-dependent repair rates available. We developed a simple model to simulate repair rates, where repair is modelled using the Sharpe-Schoolfield Arrhenius model (Schoolfield et al., 1981) that uses a repair rate coefficient ($\dot{k}$) to set the rate of repair at 20°C (*de facto* optimum), such that instantaneous repair rates are high at optimal temperatures but drop rapidly at thermal extremes (equation and fitted parameters in Supporting Information). The six reported repair rate data points derived from Ørsted et al. (2022) correspond closely with the Arrhenius model for repair rate (Box 1 Figure b).

Using a six-day simulation of realistic body temperatures (that ranged 6-34°C; Fig. S1) derived from *NicheMapR* (Kearney & Porter, 2020), we applied the damage accumulation function (Equation 3) to demonstrate the accumulation of damage up to the T_{50} threshold (50% reproductive viability), which is reached after around 81 h (Box 1 Figure c). With no repair, a dynamic 'tolerance landscape' function (Rezende et al., 2014) shows that 50% probability of reproductive failure is reached around 100 h. Accounting for repair reduces the probability of reproductive

failure to below 50% for the entire simulation (Box 1 Figure d). Thus, these models using data for heat failure with and without repair provide markedly different fitness outcomes.



Conceptual and practical application of the Thermal Load Sensitivity (TLS) framework to female *Drosophila suzukii* reproduction. (**a**) Regression between temperature (*y*-axis) and time (h) to event (in this case T_{50}, *x*-axis) data is then used to estimate the CT_{max1h} (intercept of curve) and thermal sensitivity *z* (slope of the $\log_{10}$-linear relationship). (**b**) Repair rates as a function of temperature. Points are estimates for *D. suzukii* repair rate from Ørsted et al. (2022) and the curve is modelled repair rates using an Arrhenius function. (**c**) Simulating temperature exposure across six days with cool nights and applying the accumulated damage model (Equation 3) to illustrate how damage accumulates up to reach the threshold T_{50}. (**d**) Predicted cumulative probability of reproductive failure as using dynamic tolerance landscape models without repair (orange) and with repair (green) that is occurring both during stress and also outside of the stressful range of temperatures.

While these process-based simulations of TLS are useful for generating plausible predictions about the balance between damage and repair on physiological function for a broad range of organisms, heat exposure scenarios, and different scales, they need further empirical characterisation and validation. Both the general shape of the recovery curve as a function of temperature and the dependence of recovery on physiological function or temperature are, to our knowledge, still largely unknown (although the Arrhenius function in Box 1 appears to capture this well for *D. suzukii*). Various mathematical functions could be used to model the assumed temperature-dependence of damage and repair, much like the suite of plausible functions that can be fit to thermal performance curves (Padfield et al., 2021); the most appropriate function will likely differ among life forms (Ørsted et al., 2022). It will therefore be necessary to design experiments to quantify damage and repair rates to parameterise and to validate these models, which remains challenging for real organisms (Bai et al., 2019; Huey & Kearney, 2020; Kingsolver & Woods, 2016; Klanjscek et al., 2016). Broad taxonomic groups might have similar sensitivity responses due to evolutionary conserved mechanisms of cellular damage and repair, but this is yet to be tested. We recognise that varying these damage and repair assumptions could significantly alter model outcomes (e.g., Youngblood et al., 2025), and this is an exciting area for investigation for which we advocate targeted investigations into damage-repair processes across diverse taxa.

IV. TLS could help address key outstanding questions in global change biology and thermal ecology

Global change biology and thermal ecology inherently need to consider multiple stressors in combination, and the impacts of the timing and magnitude of these stressors in an organisms' life. Below we provide an exploratory, conceptual overview of some of the emerging areas of research for which the TLS framework could be used for both theoretical and empirical insight.

A. Sublethal measures of thermal sensitivity and impacts on modular systems of an organism

The role of heat exposure in causing sublethal detrimental effects on organism fertility has come

into sharp focus as an important climate change impact on population growth, extinction risk, and species distributions (Bretman et al., 2024; van Heerwaarden & Sgrò, 2021; Walsh et al., 2019). The TLS framework allows investigations into potential spatial distributions based on thermal effects on sublethal traits (Box 2). Thermal sensitivity to heat stress in animals usually focuses on whole-organism physiology and ignores more vulnerable modular organs and life stages (Bennett et al., 2018). The thermal sensitivity of essential organs and primary biological functions like reproduction are arguably more ecologically valuable to understanding the potential vulnerability of organisms to global change stressors than are their lethal endpoints (van Heerwaarden & Sgrò, 2021).

In plants, most of the thermal vulnerability indices are calculated for leaves or cut leaf sections and thus describe thermal limits at the functional level at a very fine scale (e.g., photosynthetic machinery). The temperature ranges realised in most plant species' geographic range are far narrower than measured thermal limits (Lancaster & Humphreys, 2020) and there is little evidence that extreme temperatures alone kill adult plants, especially trees (Marchin et al., 2022a). Both the onset of functional impairment of photosystems and the damage to leaf tissue are clearly dependent on thermal exposure time (Cook et al., 2024; Faber et al., 2024; Neuner & Buchner, 2023). However, we know little about how accumulated thermal damage to modular organs like leaves then affects the state of larger components such as a tree crown or the entire tree, and what the resource or energy costs are for repair or discarding dead tissue and regenerating. To illustrate these concepts, we used data from a heatwave during the dry summer of 2020 in Sydney, Australia. Daily maximum air temperature exceeded 45°C on multiple occasions during a period of no rainfall, within which it is too dry to repair the damage from heat stress (orange area of Fig. 2a), resulting in crown dieback (Fig. 2b). Although there were then small rainfall events, extreme temperatures were still occurring and these conditions remain unfavourable for substantial repair (blue area of Fig. 2a), but crown cover loss was less dramatic (Fig. 2b). Larger rainfall events coupled with a reduction in maximum air temperature then provided conditions that allow repair of damage (green area of Fig. 2a) and then at least two species of urban trees had capacity to regenerate their crown, while others were too damaged (Fig. 2b).

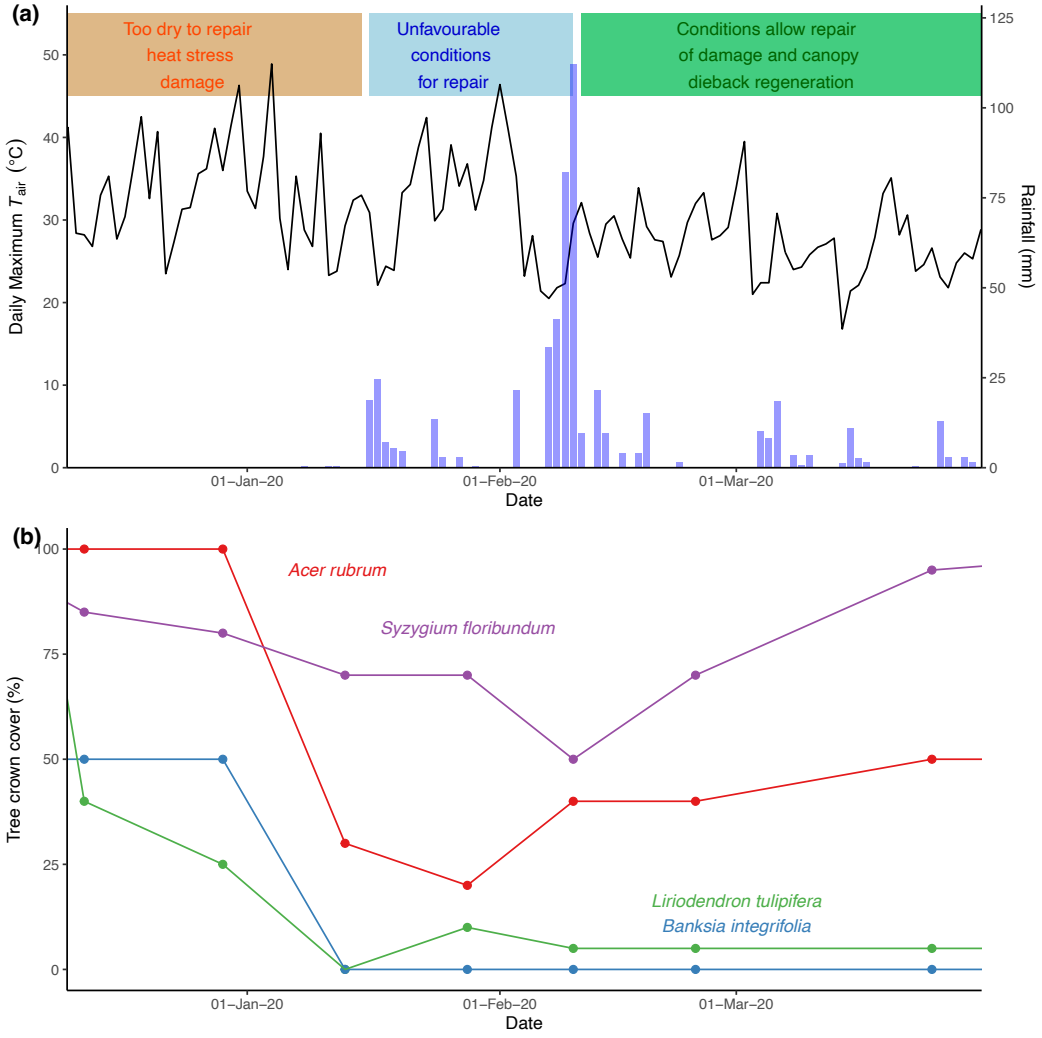
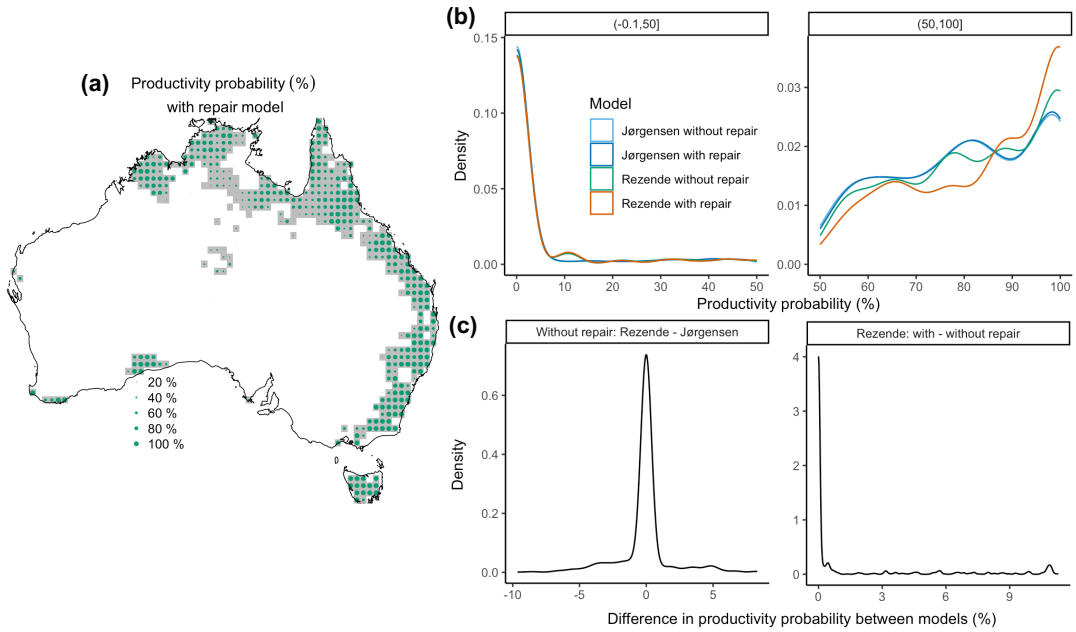


Figure 2. Example of (mostly) sublethal effects of heat on modular components of organisms (e.g., leaves on trees). (**a**) Extreme heat during dry conditions in Sydney, Australia during the 2019-2020 austral summer. Black line is the daily maximum air temperature and blue bars are rainfall events. (**b**) Recovery of tree crown foliage from heat stress in urban tree species during this time was conditional on heat tolerance and water availability. Responses were species-specific: some trees died when maximum air temperature surpassed physiological thresholds (*Banksia integrifolia*, blue), while surviving trees began recovering by resprouting new leaves in the weeks after rainfall (*Acer rubrum*, red; *Syzygium floribundum*, purple). The young leaves of some species were vulnerable to further heat damage (*Liriodendron tulipifera*, green), and full recovery of lost foliage of trees that accumulated substantial heat damage took multiple years for many individuals (data adapted from Marchin et al. (2022b)).

Box 2: Estimating the potential spatial distribution of the invasive pest *Drosophila suzukii* as a function of damage accumulation and repair capacity

Drosophila suzukii is a globally invasive pest that would have devasting consequences to agricultural industries if it were to establish in Australia. Current pest risk analysis reports indicate it would have major impacts on berry, stone fruit, and viticulture; collectively worth at least $5.4 billion AUD (DAFF, 2013). To identify regions where *D. suzukii* could maintain productivity (positive population growth), we extend the example from Box 1 to estimate the spatial extent in which female *D. suzukii* could remain productive for seven days in January in Australia (summer) using gridded microclimate data from *microclimOZ* (Kearney, 2019). First, we fitted a traditional static 50% threshold (CT_{max1h} = 36.3°C) model to determine the spatial extent within which *D. suzukii* could remain productive (grey background area in Box 2 Figure a; Fig. S2). Then, we fitted dynamic thermal landscape models from Rezende et al. (2020) and dynamic CT_{max} models from Jørgensen et al. (2021), each with and without implementing the damage-repair feedback (details in Supplementary Information), applied to each grid cell. The size of the green circles in Box 2 Figure a indicate the probability of females producing offspring based on the dynamic tolerance landscapes model with repair (for maps of each model see Fig. S3). Box 2 Figure b shows the density (proportion of grid cells) of producing offspring according to the four models. This shows that the different models generally behave similarly, while including repair increases the proportion of locations with productivity above 85%. Box 2 Figure c left panel shows that there is relatively little difference between the Rezende and Jørgensen modelling approaches (also shown by: Youngblood et al., 2025), while the right panel shows that there is up to 12% difference in productivity probability when repair is included. The damage accumulated over the seven-day simulation was reduced when we included damage-repair dynamics. Thus, applying the TLS damage-repair model provides a more detailed perspective on the intensity of sublethal heat stress, highlighting geographic areas where persistence of *D. suzukii* may depend on repair processes. Such insights could be used to more effectively identify growing regions that might be susceptible to incursion and population establishment. Our model examples suggest that even during a hot week in summer, female *D. suzukii* could still reproduce in large portions of Australia's most productive agricultural regions. For example, the predicted distribution of the area where the fly could reach high productivity includes significant areas for growing strawberry in southeast

Queensland, and grape and stone fruit growing regions in eastern New South Wales, eastern Victoria, and much of Tasmania.



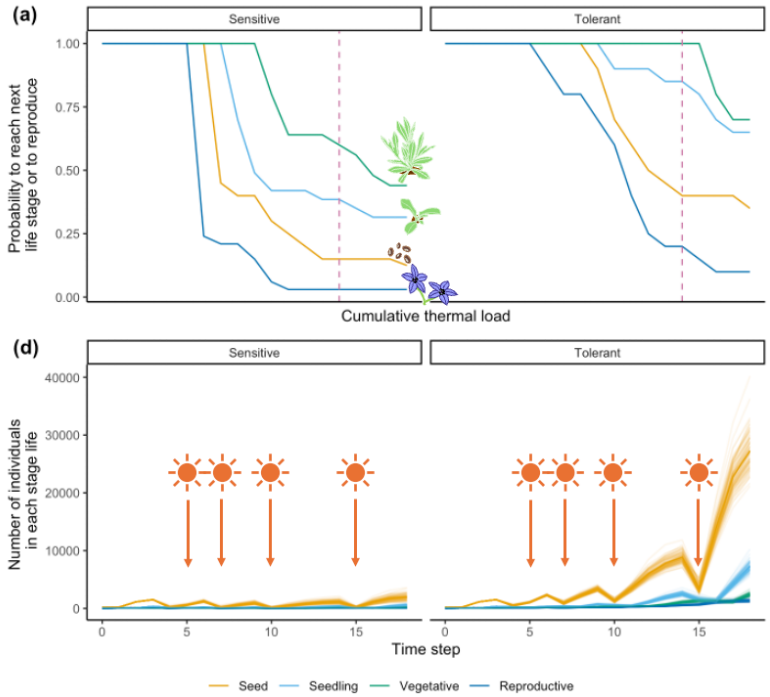
(a) Spatial map for potential extent for *Drosophila suzukii* to remain productive during a hot week in summer in Australia. **(b)** Density plots of productivity probability across the grid cells and **(c)** of pairwise comparisons between different models with and without repair.

B. Demographic scaling across life stages

The need for more ecologically relevant measures of temperature stress has given rise to the adoption of other less extreme (sublethal) indices of thermal vulnerability, like thermal fertility limits (Walsh et al., 2019). Different life stages clearly have different temperature stress thresholds, typically with pollen development and seedling stages being the most thermally sensitive in plants (Ladinig et al., 2015; Rosbakh et al., 2018) and sperm the most thermally sensitive in animals (Dahlke et al., 2020; van Heerwaarden & Sgrò, 2021). Early life stages that are sessile can be more vulnerable to overheating and may have lower heat tolerance (e.g., butterfly eggs (Klockmann et al., 2017), tadpoles (Ruthsatz et al., 2022), and intertidal gastropods (Truebano et al., 2018)). However, in other cases, less mobile instars and pupal stages of insects can be more tolerant than eggs or adults due to their reliance on inherent heat resistance rather than

behavioural heat avoidance (Bowler & Terblanche, 2008; Kingsolver et al., 2011). Small and large organisms (including the same species at different stages of growth) can have size-dependent body temperature and thermal resistance due to thermal inertia and changes to boundary layer properties (Kearney et al., 2021). Organisms around the millimetre scale, including larval stages of invertebrates, may have very subtle and fine-scale microclimates available to them to avoid overheating (Pincebourde & Woods, 2020). In plants, the life stage at which the plant is exposed to thermal stress is crucial in determining the impact of that stress on individual plant responses, their reproductive success, and subsequent population dynamics (Everingham et al., 2021; Notarnicola et al., 2021; 2023; Satyanti et al., 2021). However, most available thermal tolerance data are measured on adults, largely ignoring earlier life stages or actively reproducing individuals, both of which are crucial for assessing the true vulnerability of a population to environmental stress (Bennett et al., 2018).

Climate warming will expose different life stages to different intensity of heat events due to variation in microclimates, sessility, and thermoregulatory behaviour (Levy et al., 2015). In reptiles with temperature-dependent sex determination, nesting habitats that are exposed to consistently warmer temperatures or fluctuating extreme heat events may no longer support balanced sex ratios necessary for population stability (Valenzuela et al., 2019). Shifts in developmental rates and timing of reproduction could also dissociate species' trophic interactions or interspecific dependencies that make environments viable (Kronfeld-Schor et al., 2017). Ecologically relevant evaluations of thermal sensitivity and vulnerability across life stages are needed to effectively model impacts on population demographics. As an illustrative example, we simulated life-stage specific sensitivity to thermal load in a hypothetical plant (Fig. 3a,b) and applied a simple matrix population model (Fig. 3c) to simulate demographic projections (Fig. 3d,e). This approach (see also Salguero-Gómez et al., 2015) is a basis for allowing TLS to alter probabilities for transition within matrices (Fig. 3b,c) if thermal stress occurs during a given life stage (see also Wiman et al., 2014). Further integrations of sublethal thermal effects on growth and reproduction informed by TLS could be built into trait-based demographic models (e.g., Falster et al., 2016).



(b) Probability to reach next life stage at indicated thermal load

	Sensitive	Tolerant
Seed	0.15	0.4
Seedling	0.385	0.85
Vegetative	0.6	1
Reproductive	0.03	0.2

(c) Life stage transition matrix

	Seed	Seedling	Vege.	Repro.
Seed	0.1	0	0	20
Seedling	0.3	0.1	0	0
Vege.	0	0.4	0.3	0.3
Repro.	0	0	0.5	0.6

(e) Predicted population outcomes

	Initial	Sensitive	Tolerant
Seed	200	1,972	27,290
Seedling	100	551	7,265
Vege.	100	187	2,430
Repro.	10	92	1,331

Figure 3. Simulation of how thermal load sensitivity can differ across life stages in sensitive and tolerant populations of a hypothetical plant species with four distinct life stages: seed, seedling, vegetative (non-reproductive adult), and reproductive (actively flowering adult). **(a)** As cumulative thermal load increases toward prolonged high temperature, the probability of progression to later life stages and reproducing is reduced. Left panel shows probability declining with cumulative thermal load in a sensitive population and the right panel shows the same for a tolerant population. **(b)** Vectors of probabilities for transition to next life stage in the populations at the thermal load indicated by the dashed line. **(c)** Life stage transition matrix showing the proportion of each life stage transitioning to the next life stage or reproducing at each time step (e.g., that 10% of seeds remain seeds, 30% become seedlings, which implies 60% fail to establish as seedlings, while 60% of reproductive plants remain in reproductive stage, 30% stop flowering and return to vegetative stage, 10% die, and each reproductive plant in the reproductive stage at the time step produces 20 viable seeds that return to the seedbank). **(d)** Predicted population dynamics through time as the number of individuals in each life stage from 100 simulations under a scenario where a heat event equivalent to the thermal load indicated in **(a)** occurs at four of the time steps (indicated by sun symbol with arrows). **(e)** Initial population size at time step 0 and the final population at time step 20, showing the persistent effects of different sensitivity of life stage to cumulative thermal load that could have persistent or lag effects on population dynamics.

C. Phenotypic plasticity and thermal legacies

Prior exposure to stressors can result in plastic changes that make organisms (intragenerational) or their offspring (intergenerational) less sensitive to future stress events through acclimation, or developmental or transgenerational plasticity. For example, acclimation through heat hardening is expected to mitigate damage through ‘resistance’ mechanisms that protect cells, such as upregulation of heat shock proteins (Moseley, 1997). Early growth environments alter development of offspring (Monaghan, 2007) through developmental plasticity – the ability for an organism to alter its phenotype in response to its environment during development (West-

Eberhard, 2003). Thus, exposure to heat stress early in life could lead to altered sensitivity to heat stress (i.e., thermal load) later in life via stress priming (e.g., Hoffman et al., 2018; Hossain et al., 2018). A comprehensive meta-analysis of ectotherms found that developmental temperatures often slightly increased heat tolerance but did not consistently result in persistent effects on later life stages (Pottier et al., 2022a). It is not always the case that developmental environments shift responses to temperature, and it is not yet clear if and how thermal sensitivity is altered by marginally stressful thermal histories. Thermal tolerance and plasticity can have complex patterns throughout ontogeny, further altered by the history of exposure to chronic or acute thermal stress. These 'thermal legacy' effects can alter threshold-based thermal tolerance and physiological plasticity (Geange et al., 2021; Lancaster & Humphreys, 2020; Marasco et al., 2023; Payne et al., 2025), and will therefore likely also modify the rates and sensitivity of both damage and repair processes (Burton et al., 2022; Einum & Burton, 2023).

D. Multi-stressor integration

The TLS framework can be extended to understand the combined effects of multiple stressors, whether biotic (e.g., competition, disease) or abiotic (e.g., salinity, nutrient, water). Such an approach is feasible given that exposure to additional stressors may affect similar underlying physiological processes of damage and repair through cross-tolerance (Bryant et al., 2024; Hossain et al., 2018; Katam et al., 2020). In natural environments, a range of potential abiotic and biotic stressors frequently co-occur and interact with thermal stress, increasing the challenge of predicting cumulative effects of thermal stress. Thermoregulation in plants is complex and highly dynamic, with significant differences in realised temperatures and leaf-to-air offsets that depend on canopy structure and scale (Arnold et al., 2025; Dong et al., 2017; Guo et al., 2023), however it is clear that water availability will moderate responses to high temperatures (Ruehr et al., 2016). For example, heatwaves often occur during droughts. Experiments have found that at moderate levels of water stress, plants may exhibit a priming response that increases heat tolerance but, at extreme levels, water stress greatly decreases the ability of plants to cool their leaves and so may exacerbate heat stress (Cook et al., 2021; Marchin et al., 2022a). Other biotic interactions such as pathogen infection that occurs simultaneously with heat stress can not only suppress resilience to the pathogen but also reduce the heat tolerance of the host in invertebrates (Hector et al., 2021) and plants (Desaint et al., 2021).

Ultimately, when organisms are exposed to two or more stressors the cumulative effect of all stressors can be additive, synergistic, or antagonistic (Orr et al., 2020) (Fig. 4a,b), and disentangling multi-stressor effects on heat tolerance should be a major focus of future work. Exposure to additional stressors will alter the TLS parameters, along with damage and repair rates, and thresholds for enzyme inactivity, potentially in complex or non-linear ways. As a simple (linear) example, a change in intercept (CT_{max1h}) with no change in slope (z) with the addition of non-thermal stressors implies an additive effect of the stressors (Fig. 4b). Changes in slope, with or without changes in intercept, imply an interactive effect, either synergistic or antagonistic (Fig. 4b), as the extra effect of the non-thermal stress can also be temperature-dependent (Duncan & Kefford, 2021). For these simplified examples, multi-stressor effects on CT_{max1h} and z can be evaluated by including interaction terms in statistical models. The limited empirical data available with multiple abiotic stressors (e.g., Enriquez & Colinet, 2017; Maynard Smith, 1957; Verberk et al., 2023; Youngblood et al., 2025) suggest the slope can change, implying an interactive effect.

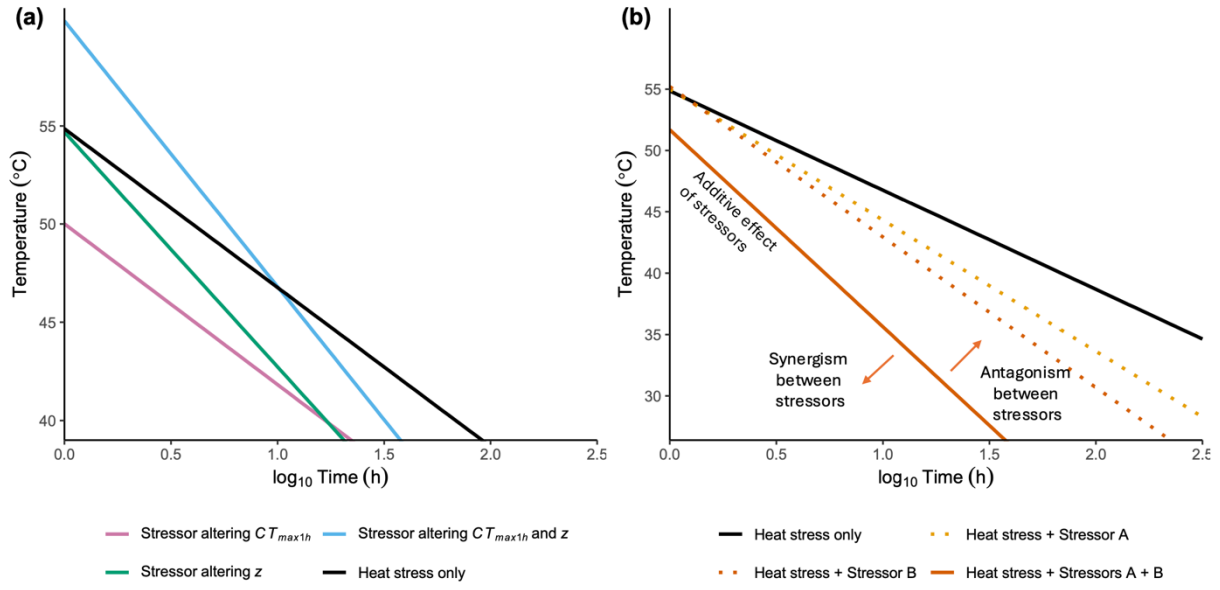


Figure 4. Conceptual depiction of the effects of heat stress in combination with additional stressors within the TLS framework. (**a**) Different coloured lines represented potential changes in the CT_{max1h} and/or z parameters of the TLS curves when subject to additional stressors. (**b**) The difference between the TLS curves with heat stress alone (black solid line) and the TLS curves of heat stress with other stressors individually (A, yellow dotted line and B, orange dotted line). From these lines we would predict that the effect of all three stressors (heat, A, and B) is additive by summing the difference between heat stress only and heat stress with one stressor (orange solid line). If the net effect of the three stressors is more extreme than the additive effect, then the stressors accumulate synergistically, but if the effect of all three is less than the additive effect, then the stressors are antagonistic, and the net effect is less than the sum of their individual effects.

V. Conclusions and agenda

The Thermal Load Sensitivity framework provides a step towards reconciling the ways in which organisms deal with natural dynamics of heat stress, whether that be temperature alone or in combination with other stressors. The TLS framework achieves this through the integration of the dynamics of thermal stress exposure with the dynamics of physiological damage and repair. The following five key areas, as discussed above, stand out as being particularly important foci for investigation, extension, and application of the TLS framework to better understand and predict thermally mediated impacts on plants and animals:

1. ***Adopt TLS terminology because it is inclusive of sublethal effects and applies across developmental states and the tree of life.*** As discussed above, we advocate a shift in language and inherent focus on lethal effects to sublethal effects that are more ecologically relevant, which includes taxonomic groups for which it is difficult or undesirable to estimate whole organism death. Large datasets for diverse thermal tolerance limits are emerging, mostly for ectothermic animals (e.g., Bennett et al., 2018; Lancaster & Humphreys, 2020; Pottier et al., 2022b). While these provide a foundation, there is a need to expand them to cover a more representative sample of life. Improving understanding of the biological processes that underpin a given sublethal effect and testing assumptions to better parameterise models will improve the efficacy of thermal vulnerability predictions for a given species.
2. ***Apply emerging tools to identify universal damage and repair mechanisms that impact recovery from thermal stress.*** Disentangling damage and repair mechanisms is crucial (Ørsted et al., 2022). Integrative computational models for genome-scale protein folding and stress responses are emerging for microbes (Chen et al., 2017; Zhao et al., 2024), however empirical data and understanding of these dynamic biological process remains very limited for complex life forms. Developing effective methods for measuring rates of damage and repair in plants and animals could be tackled with multifaceted flow cytometry approaches using consensus panel markers of stress, damage, and repair (Buerger et al., 2023). By determining the conditions under which proteins unfold and inactivate, oxidative stress responses are expressed, and by mapping programmed cell death pathways during and after thermal stress (Chen et al., 2020; Roychowdhury et al., 2023), we can begin to

understand mechanisms of damage and repair reciprocity. Repair will be particularly important when damage is low, and we therefore need to better understand the trade-offs between repairing or replacing damaged cells and tissues, and how these depend on metabolic repair costs (Rennolds & Bely, 2023). Linking bioenergetics at the cellular level to physiological and ecological functions and fitness is a crucial research frontier (Sokolova, 2021). We need, however, empirical data to build a deeper understanding of the complex cellular processes underlying damage and repair to construct and evaluate mechanistic models.

3. ***Ascertain principles determining how multiple stressors, both abiotic and biotic, affect thermal load sensitivity*.** Different stressors and biological interactions are expected to impact the damage and repair processes by acting through common mechanisms across plants and animals (Wek et al., 2023). However, combinations of stressors and/or biotic interactions and their timing may have complex effects on damage accumulation that must be factored into assessments of *vulnerability* (Georgieva & Vassileva, 2023; Prasch & Sonnewald, 2015; Taborsky et al., 2022). Few studies have evaluated how additional stressors modify thermal load sensitivity and given that stresses co-occur in nature, this is an essential avenue for future investigations.
4. ***Integrate plasticity in response to past stress to determine mechanisms and scale of stress priming.*** A clearer understanding is required of the biological mechanisms and environmental cues that contribute to priming and the plasticity of responses to stress. Plasticity in damage and repair processes, and the time course or rates of these plastic responses can alter sensitivity and lead to differences in vulnerability of populations (Burton et al., 2022; Dupont et al., 2024; Einum & Burton, 2023). Thus, exploring timing of stresses and rates of plastic responses will be pivotal to being able to model and predict how environmental exposure affects individuals throughout ontogeny and then scales up to affect the vulnerability of populations.
5. ***Improving understanding of the plastic and evolutionary potential of thermal tolerance will inform conservation and management decision making, and breeding for food security.*** Finally, we need a better understanding of genetic variation in stress tolerance across diverse taxa. The genetic variation underlying thermal sensitivity likely depends on multiple complex mechanisms acting over different time scales (González-Tokman et al., 2020; Logan & Cox, 2020). Quantitative genetics can reveal evolutionary constraints,

selection, and heritability of thermal load sensitivity parameters (Leiva et al., 2024). Understanding phenotypic and genetic variation in thermal sensitivity among populations is essential for predicting how they could adapt to future environmental conditions, which facilitates strategic conservation planning and adaptive management (Bennett et al., 2019; Rilov et al., 2019). Breeding crops that are resilient to thermal extremes from climate change while maintaining yield to meet food security demands will rely on building a deep understanding of the adaptive signatures and genetic mechanisms underlying thermal sensitivity before making use of synthetic biology tools and quantitative genomics (Lohani et al., 2020; Razzaq et al., 2021).

Researchers need to recognise the cumulative effects of thermal load on damage and repair processes and how they will interact to affect biological responses to global change. The TLS framework builds on the established principles of the TDT model used in ecophysiology (Ørsted et al., 2022; Rezende et al., 2014), forming a strong basis for further research into additional dimensions (e.g., sublethal effects, tissue types, life stages, spatial models, multiple stressors), that impact sensitivity and the underlying molecular and genetic architecture. We hope that a broader focus through the TLS framework will provide opportunities to better predict organism vulnerability in a time of profound global change. Death is just one, albeit severe, consequence of thermal stress; predicting loss of individual reproduction and ecological function while realistically incorporating dynamic environmental and biological processes is much more challenging but arguably more important. Integrating these essential components into our theoretical and modelling frameworks is a step towards better understanding organism vulnerability to significant environmental stressors.

Competing interests

The authors declare no competing interests.

Author contributions

P.A.A. and J.M.B. led the development of the workshop and co-led writing of manuscript drafts with substantial input from D.W.A.N., A.B.N., M.R.K., and E.L.R. Funding for the workshop was acquired by V.F.B., P.A.A., J.M.B., and O.J.R. The simulations, functions, and R scripts were developed by M.R.K., E.L.R., D.W.A.N. and P.A.A. All authors contributed substantially to the workshop and ideas presented in the perspective and critically revised the draft manuscript.

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Supplementary Information

A framework for modelling thermal load sensitivity across life

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Note that a digital tutorial-style version of this supplementary information, which includes all R code and modified functions to recreate the developed TLS models, as well as figures in the main text and supporting information is available here:
https://pieterarnold.github.io/thermalloadsensitivity/.
The data and other files that support the analyses and figures are available here:
https://github.com/pieterarnold/thermalloadsensitivity.

Notes on repair rate model

Potential repair rate (Fig. 2c of main text) was modelled using a typical four-parameter Sharpe-Schoolfield Arrhenius model (Schoolfield et al., 1981) with an additional repair rate parameter, in the form of:

$$y(T) = \dot{k}_{ref} \cdot \exp\left(\frac{T_A}{T_{ref}} - \frac{T_A}{T}\right) \cdot \frac{\left(1 + \exp\left(\frac{T_{AL}}{T_{ref}} - \frac{T_{AL}}{T_L}\right) + \exp\left(\frac{T_{AH}}{T_H} - \frac{T_{AH}}{T_{ref}}\right)\right)}{\left(1 + \exp\left(\frac{T_{AL}}{T} - \frac{T_{AL}}{T_L}\right) + \exp\left(\frac{T_{AH}}{T_H} - \frac{T_{AH}}{T}\right)\right)}$$

(Equation S1)

To parameterise our models, we fitted a Thermal Death Time (TDT) function based on parameters for female *Drosophila suzukii* productivity reported in Ørsted et al. (2024). We fitted a '*thermal landscape*' function to estimate survival probability over time of exposure to heat stress to derive the intercept (α) and slope (β) parameters. We use α and β terminology here for clarity because different applications of the TDT model refer to intercept, slope, CT_{max} and z differently due to fitting either temperature or time as the response variable, which substantially alters how one would interpret the parameters.

Fitted model parameters for simulation in Figure 1.

Parameter	Description, unit	Value
T_A	Arrhenius temperature, K	14065
T_L	Arrhenius temperature lower threshold, K	283.65
T_H	Arrhenius temperature upper threshold, K	301.65
T_{AL}	Arrhenius temperature lower, K	50000
T_{AH}	Arrhenius temperature upper, K	100000
T_{ref}	reference temperature, K	293.15
T	sequence of temperatures over which to model, K	273.15 - 323.15
$\dot{k}_{ref}$	repair rate at T_{ref}, % min^{-1}	0.007; 0.0111; 0.02
$\dot{k}_{dec}$	repair rate decline, dimensionless	3

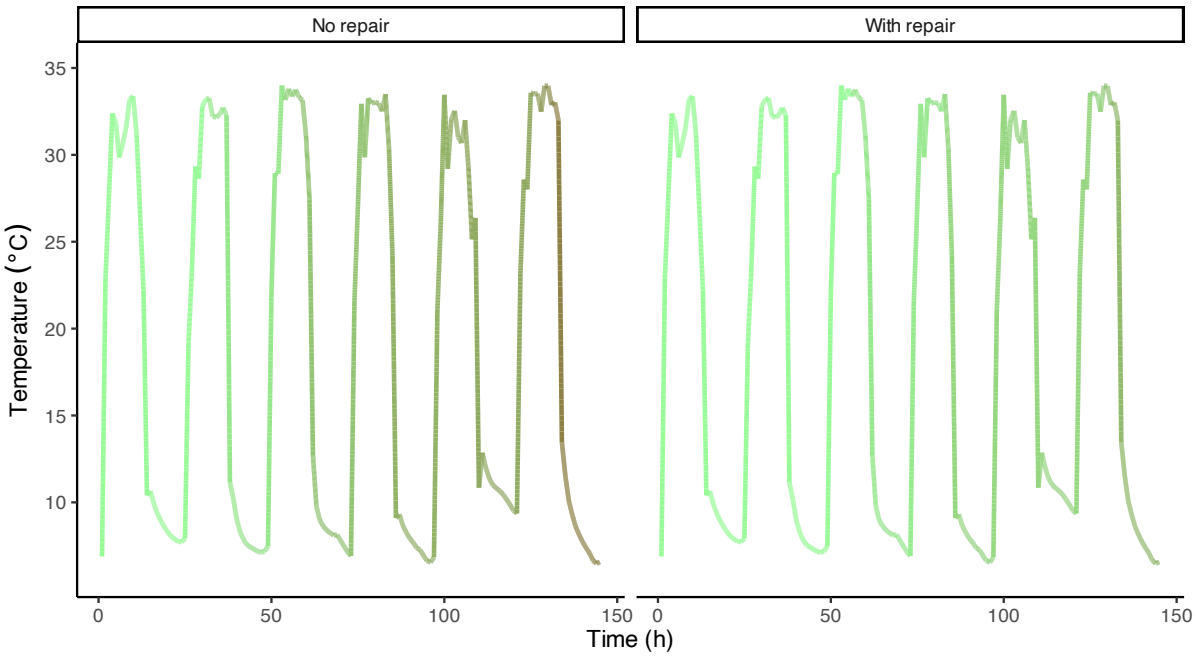

Fig. S1. Simulation of body temperatures over six days for the *D. suzukii* example in Box 1. Left panel is damage without allowing repair and the right panel includes repair as in Box 1 figure b. Colours correspond to accumulated damage as in Box 1 figure c. The increase in 'brown' colouration shows how damage is higher at the end of the six-day period without considering repair compared with the repair scenario.

Extended Box 2: Estimating the potential spatial distribution of the invasive pest *Drosophila suzukii* as a function of damage accumulation and repair capacity

To identify regions where *D. suzukii* could maintain productivity (positive population growth), we extend the example for Box 1 to estimate the spatial extent in which female *D. suzukii* could remain productive for seven days in January in Australia (summer) using gridded microclimate data from *microclimOZ* (Kearney, 2019). The maximum air temperatures at 120 cm during this seven-day period are shown in Fig. S2a. We fitted a traditional static threshold (CT_{max1h} = 36.3°C) model to determine the spatial extent within which *D. suzukii* could remain productive at first (Fig S2b).

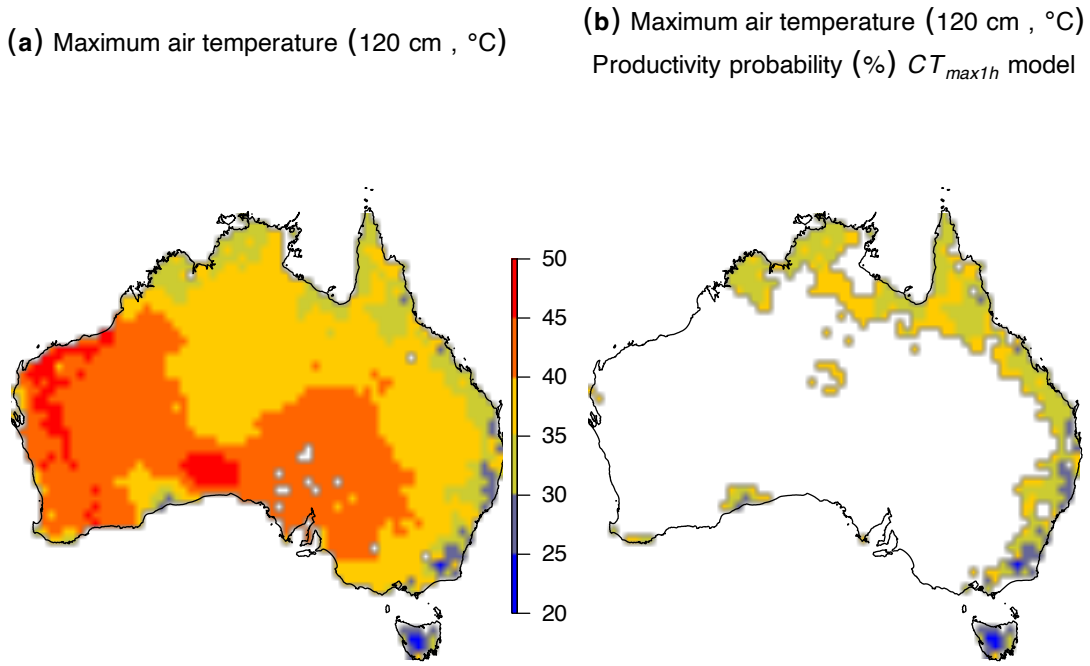

Fig. S2. (a) Map of maximum air temperatures at 120 cm (°C) across Australia over the seven-day period used for the simulation for *Drosophila suzukii*. **(b)** Subset map of Australia showing the areas that did not exceed CT_{max1h} for productivity during the simulation.

Application of Jørgensen et al. and Rezende et al. models

For applying Jørgensen et al. (2021) models for Box 2, we integrated equation 2 from Jørgensen et al. (2021) by fixed time steps. The lethal dose (d_L) function integrates the parameters for the critical temperature causing 50% mortality in 24 hours (CT_{max24h}) and the temperature causing 50% mortality in 1 hour (CT_{max1h}) to determine the lethal dose of heat, such that:

$$d_L = \exp^{k \cdot \left(\frac{CT_{max1h}}{CT_{max24h}} - 1\right)}$$

(Equation S2)

$$k = \frac{\log(10)}{-\beta^{-1}}$$

(Equation S3)

$$CT_{max24h} = \frac{\log_{10}(24) - \alpha}{\beta}$$

(Equation S4)

$$CT_{max1h} = \frac{\log_{10}(1) - \alpha}{\beta}$$

(Equation S5)

Specifically, the parameters for the *D. suzukii* model used in Box 2 were:
Intercept (α) = 11.902, slope (β) = –0.3058, z = 3.27.

The Rezende et al. (2020) models apply the *ad hoc dynamic.landscape* function (details in Supplementary Information, p. 12 of Rezende et al. (2020). We then modified this function (*dynamic.landscape2*) to add the Sharpe-Schoolfield Arrhenius model for repair to the 'alive' term (range: 0-100) to indicate the status of the organism or sublethal component thereof. As the value of 'alive' reduced below 0.99, the function implements a decay in repair rate ($\dot{k}_{ref_i}$) to simulate the decline in repair capacity due to accumulation of injury or physiological damage.

$$\dot{k}_{ref_i} = \dot{k}_{ref} \cdot \left(\dot{k}_{ref}^{\frac{\dot{k}_{dec}}{alive}}\right)$$

(Equation S6)

Where $\dot{k}_{dec}$ is an arbitrary parameter for defining the steepness of the decay in repair rate. Each iteration of model fitting then uses the $\dot{k}_{ref_i}$ term as the repair parameter for each time step.

Fitted model parameters

Fitted model parameters for simulation in Box 1 Figure.

Parameter	Description, unit	Value
T_A	Arrhenius temperature, K	3516.25
T_L	Arrhenius temperature lower threshold, K	283.65
T_H	Arrhenius temperature upper threshold, K	300.15
T_{AL}	Arrhenius temperature lower, K	50000
T_{AH}	Arrhenius temperature upper, K	83333
T_{ref}	reference temperature, K	293.15
T	sequence of temperatures over which to model, K	273.15 - 323.15
$\dot{k}_{ref}$	repair rate at T_{ref}, % min^{-1}	0.095
$\dot{k}_{dec}$	repair rate decline, dimensionless	3

In the Box 2 models, we applied the repair rate Arrhenius model (shown in Box 1 Figure, which includes the decay in repair rate based on damage accumulation impacting physiological function) to the dynamic tolerance landscape model (modified R function). Allowing the probability of successful productivity to increase when temperature conditions facilitated partial repair at a rate that is dependent on temperature and accumulated damage (see Box 1 Figure) reduced the damage accumulated over the seven-day simulation. Maps for the distribution of productivity are based on the dynamic tolerance landscape model without repair (Rezende et al., 2020) (Fig. S3a), the dynamic CT_{max} model without repair (Jørgensen et al., 2021) (Fig. S3b), the dynamic tolerance landscape model with repair rate (Fig. S3c), and productivity probability difference between the tolerance landscape models with and without repair (Fig. S3d).

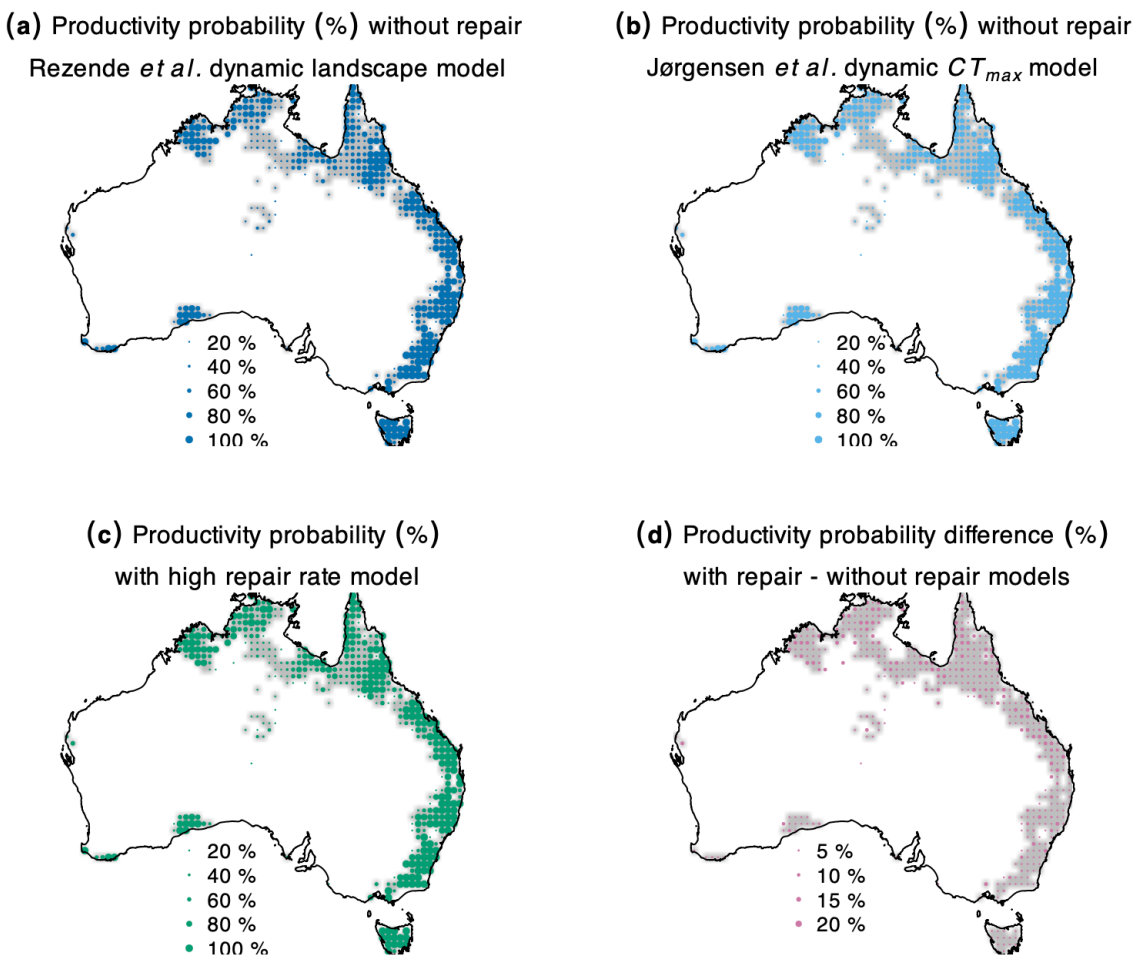

Fig. S3. Maps of Australia showing areas of viable productivity of *Drosophila suzukii* based on different models over the seven-day period used for the simulation. In all maps, the grey background area is based on CT_{max1h} shown in Fig S1b. Circles of different sizes show productivity probability (%), where larger symbols indicate higher probability of producing offspring. Maps are based on **(a)** the dynamic tolerance landscape model without repair (Rezende et al., 2020), in dark blue and **(b)** the dynamic CT_{max} model without repair (Jørgensen et al., 2021), in light blue; **(c)** the dynamic tolerance landscape model with repair rate (estimated in Box 1 and modelled with damage-repair feedback from Fig. 1), in green. **(d)** Productivity probability difference between models shown in **(a)** and **(c)**, in pink.

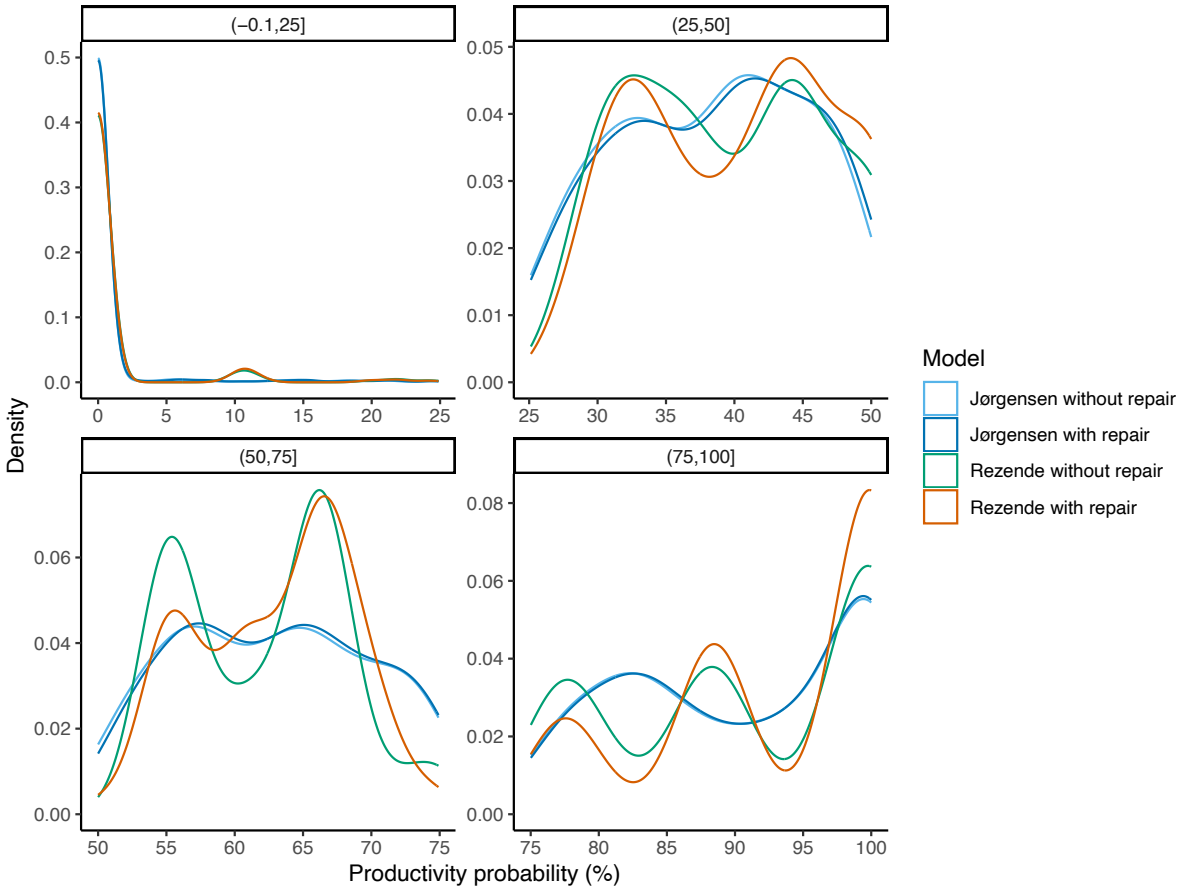

Fig. S4. Density plots of productivity probability across the grid cells, faceted into quartiles.

Additional detailed example of the TDT model and TLS framework: Weed management by solarisation

The TDT model can already be applied broadly. For example, it can be used to optimise weed management in agriculture where thermal treatments are applied to soil to eradicate weed seeds. Developing strategies to deplete weed seed banks and their germination potential is a grand challenge in agronomic management (Chauhan, 2020). Soil solarisation is a non-chemical approach to biocide that uses passive solar heating under plastic to disinfect soil of crop pests and weeds (Stapleton, 2000).

As an example of applying the TDT model, we illustrate the relationship between heat exposure over time and seed mortality or germination failure. We extracted LT_{80} (temperature at which 80% of seeds are killed and fail to germinate) data from Dahlquist et al. (2007) and used *metaDigitise* (Pick et al., 2019) in the R Environment for Statistical Computing v4.3.1 (R Core Team, 2023) to estimate TDT curves to evaluate thermal sensitivity for three weed species. The proportion of seed mortality in *Sisymbrium irio* (London rocket) is strongly dependent on both temperature and time (Fig. S5a). To achieve 80% seed mortality at 42°C requires about 85 h of cumulative heat treatment, whereas, at 50°C, this time required reduces drastically to 4 h. Applying the TDT model to three weed species predicts that, on average, treatment temperatures need to reach 57°C for at least 1 h for seed mortality to reach LT_{80} (Fig. S5b). Seed mortality has the same qualitative response to thermal dosage but there are interspecific differences in critical thermal maximum at 1 h (CT_{max1h}) and thermal sensitivity (z) resulting from differences among species (Fig. S5b). If we simulate a heat treatment ramping to approximately 50°C, then it would need to be maintained for 20 h on average to eradicate 80% of all seeds, but each species requires very different thermal dosages to accumulate the target ('100% damage'), which here actually refers to reaching LT_{80} (Fig. S5c). Damage accumulates at different rates based on both CT_{max1h} and z: *S. irio* seeds reach LT_{80} by the treatment after 10 h of treatment, while *Solanum nigrum* (black nightshade) seeds reach LT_{80} after 27 h, and *Amaranthus albus* (tumble pigweed) do not reach LT_{80} even after 30 h of heat treatment (Fig. S5d). While *A. albus* has higher CT_{max1h} than *S. nigrum*, the greater sensitivity z of *S. nigrum* (Fig. S5b) results in slower damage accumulation under the treatment regime (Fig. S5c,d). The effectiveness of solarisation techniques therefore relies heavily on the thermal dosage being applied at the necessary intensity and duration.

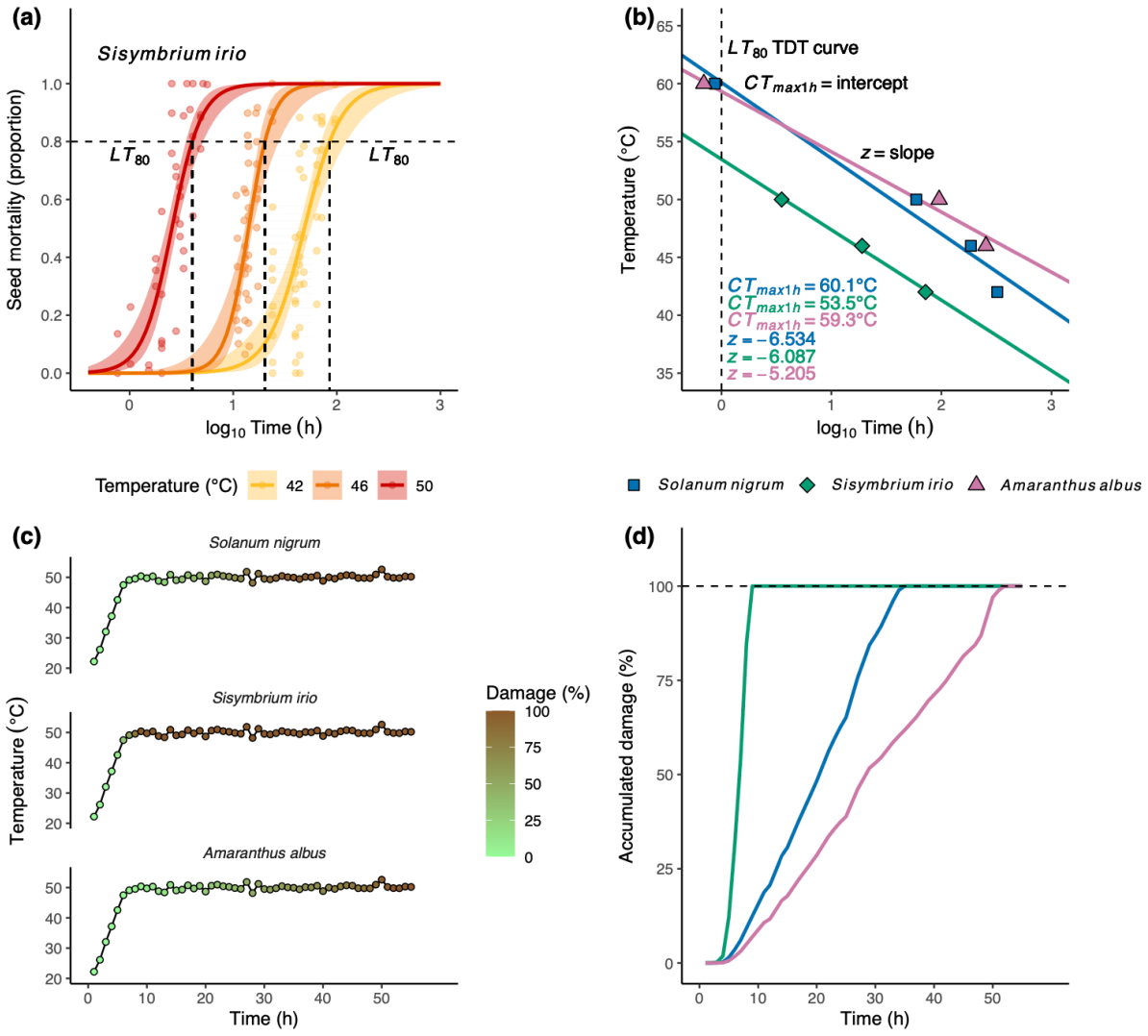

Figure S5. Conceptual and practical application of the Thermal Death Time (TDT) model to seed mortality. (**a**) Experiments measure seed mortality (as % not germinating after heat stress application) for varying amounts of time and across a range of temperature treatments. A dose-response curve can be estimated for each temperature via logistic regression. Fitted response curves to estimate LT_{80} (time for 80% mortality) of an example weed species taken from Dahlquist et al. (2007). (**b**) Biologically relevant thresholds, such as LT_{80}, that are derived from (**a**) are then used to estimate a TDT curve for three weed species that differ in their thermal ecology. A linear regression between temperature (y-axis) and $\log_{10}$ Time (h) to event (in this case LT_{80}, x-axis) data is then used to estimate the CT_{max1h} (intercept of TDT curve) and thermal sensitivity z (slope of the TDT curve). (**c**) Simulating a ramping temperature profile for an approximate 50°C heat solarisation treatment for 30 h and applying the damage model (Equation 3) predicts how accumulating damage could differ by species. (**d**) As temperature slowly increases to approximately 50°C, damage accumulates towards LT_{80} at different rates depending on species thermal sensitivity.

Different threshold values can be used for estimating the effects of thermal damage. As an example, 10%, 50% and 90% mortality could be useful to understand the efficacy of a treatment. We applied the TDT models for two of the weed species (Fig. S6a,b) across a natural cyclic temperature regime that reaches high temperatures during the day but cools overnight, allowing for damage to stop accumulating (Fig. S6c,d). Finally, we can add in a simple version of the Arrhenius repair function to allow the accumulated damage to be reduced when temperatures permit repair to occur (Fig. S6e,f). In reality, it may be unlikely for a seed to repair damage inflicted by high temperatures, however in this example, we use a relatively high value of $\dot{k}$ purely for illustrative purposes. Repair can be seen to ameliorate the damage accumulation, and it delays the time that it takes for *S. irio* to reach any *LT* threshold by 24 h, but the end result of the 120 h temperature regime is the same due to the extreme temperatures reached.

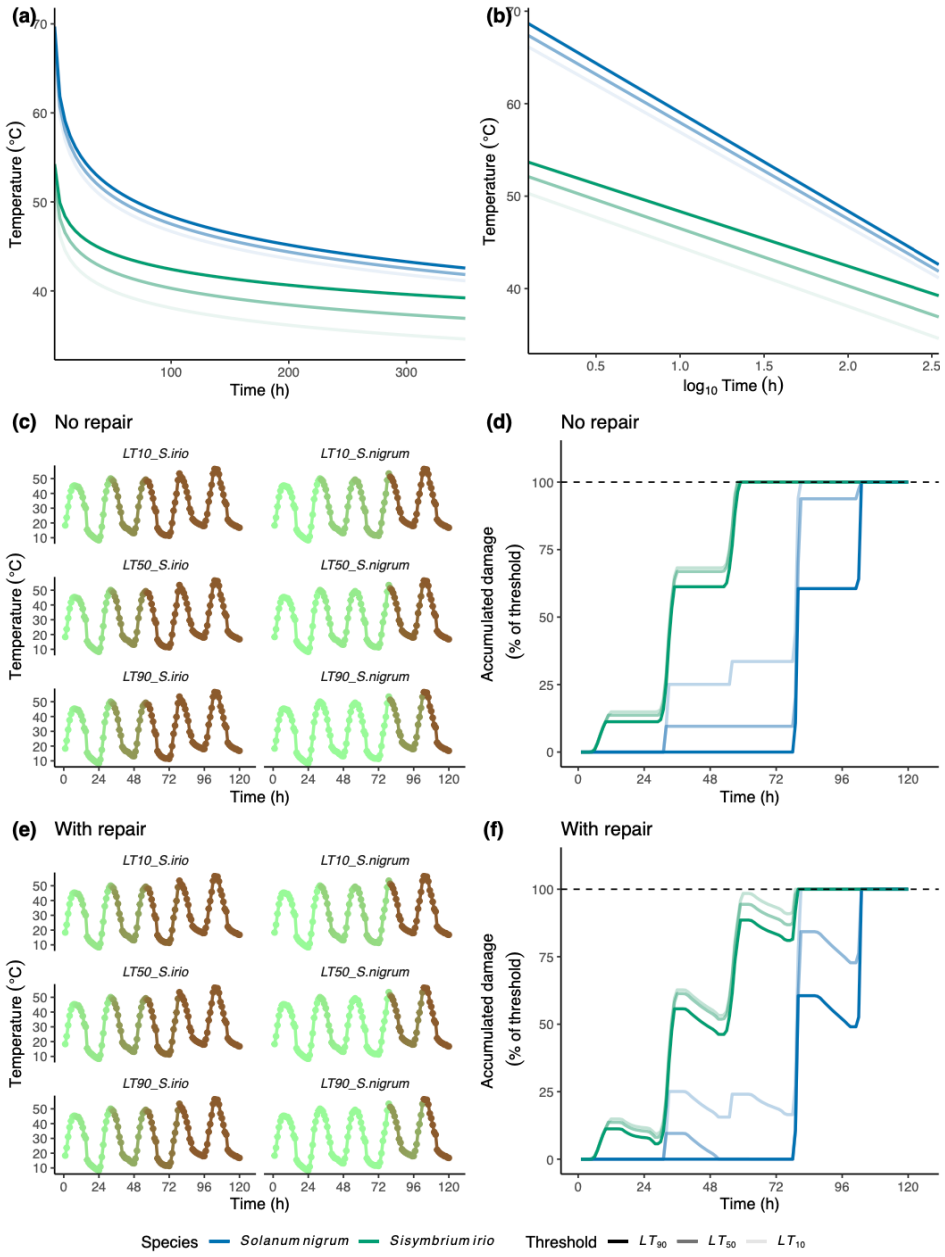

Figure S6. Conceptual and practical application of the Thermal Load Sensitivity (TLS) framework to seed mortality, including a basic repair function, using TDT estimates derived from dose-response curves in Fig. S5,, this time using fitted response curves to estimate three thresholds: LT_{10}, LT_{50}, and LT_{90} (time for 10%, 50%, and 90% mortality, respectively) of two weed species (*Solanum nigrum* and *Sisymbrium irio*) from Dahlquist et al. (2007). **(a)** Relationship between temperature (*y*-axis) and time (h, *x*-axis) to reach the three thresholds. **(b)** Log-linear relationship between temperature (*y*-axis) and $\log_{10}$ Time (h, *x*-axis) to reach the three thresholds. **(c)** Simulating a dynamic, cyclic temperature profile for 120 h and applying the damage model predicts how accumulating damage could differ by species. **(d)** Accumulated damage rate over time differs between species depending on thermal sensitivity, and depending on the threshold used. **(e)** Simulating the same temperature profile for 120 h and applying the damage model predicts how accumulating damage could differ by species and threshold, while repair is allowed to reduce damage that has accumulated. **(f)** Accumulated damage over time differs between species depending on thermal sensitivity, the threshold used, and damage is ameliorated by repair occurring during temperatures that are not stressful.

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