Article

Marine heatwaves impact organism developmental time

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Understanding the effect of marine heatwaves on organisms is central for improving climate change predictions. Even moderate heatwave events are likely to drive performance of organisms especially if they are long relative to the life cycle duration. In ectotherms, such events will affect biological time on a stage-dependent basis; they could alter the timing of life cycle events (e.g. spawning, reproduction) and cause reproductive failure. We use a mathematical framework to explore three different scenarios for the causal relationship between temperature and developmental time and help future experimental research. Here, we highlight the need to experimentally test for (1) stage-dependent responses to temperature and (2) plastic responses to the thermal history. (3) Consider traits linked to developmental time (e.g. body size) and (4) integrate across levels of organization to develop stronger explanatory models. Experiments need to manipulate the timing, duration, and magnitude of warm events.

Understanding the causal relationships between temperature and development in marine animals is central for predictions of climate change effects on populations and ecosystems. This is true, for instance, in the case of effects of heatwaves and other warm events on critical life history traits such as biological time. Heatwaves are extreme events characterised by a period where temperature exceeds a predetermined threshold; marine heatwaves have become more frequent over the past decades and further increases are predicted in response to climate change[1–4]. While long periods and severe heatwaves have been associated with mass mortalities[5], moderate events are likely to affect ecosystems through changes in organismal traits, for example, by driving body size or the capacity to tolerate food limitation[6]. Biological time is defined as the time occurring between developmental stages of the life cycle, or between birth and death, i.e. as generation time[7]. Biological time has a central role in biology[8–12]: generation time sets a fundamental constraint for cell division, differentiation, growth, and reproduction if organisms are to produce offspring before they die ('pace of death hypothesis'[11]). Because most organisms are ectotherms (i.e. they cannot control their body temperature except through behaviour[13]), generation time (or any other biological time) is usually shortened by increased body temperature[14].

The effect of temperature on populations and ecosystems, as mediated by biological time are pervasive, as shown by shifts in the timing of blooming, reproduction, or metamorphosis experienced after long-term warming in many organisms across the world[9]. Temperature effects on generation time must be accounted for, to better understand drivers of performance and population dynamics under heatwaves or seasonal variations in temperature[15,16]. One of the current concerns in climate change

biology is the potential effect of warming on aging through increases in developmental and growth rates[17].

A deeper understanding of causal relationships requires explanatory models, i.e. formal systems where the hypothesised relationship between variables in the model (the inferential structure) reflects a causal relationship in a natural system[18–20]. Such a model would encode the interaction between temperature and some element of the organismal genome or phenotype, driving developmental time. Explanatory models differ from phenomenological models (or simulations) in that the latter only simulate the data. Phenomenological models usually make biased predictions when applied outside the original environmental range (e.g. the temperature range used to fit the model). However, the development of explanatory models of effects of heatwaves on biological time is challenging[21–23]. In principle, one could model the effects of heatwaves on developmental time, through the concept of degree days[24–26] but it would be desirable to have models that more closely reflect the biological processes driving developmental rates. Importantly, explanatory models are usually derived from data obtained in static experiments, consisting of keeping organisms at constant temperatures. However, organismal performance at constant temperatures do not always match observed responses after exposure to thermal fluctuations[15,27,28]. Static experiments do not provide information on historical effects, for example, how plasticity or carry-over effects drive biological time. Instead, explanatory models considering, for instance, the role of heatwave duration, timing, and intensity would be desirable.

Here, we explore three scenarios differing in how temperature drives developmental time across stages of the life cycle. The scenarios are

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represented by different types of mathematical models; our analysis follows the idea that mathematically oriented thinking can be combined with quantitative experimentation to increase understanding of how embryonic development proceeds[29] and is driven by temporal changes in temperature. Our aims are: (1) the formulation of alternative models to guide experimental design or the reanalysis of existing data; (2) to establish relationships between model components and biological processes occurring at different levels of organization, as the organism develop. (3) To better identify models with different perspectives on how we view development. A better understanding of the effects of body temperature on biological time requires that we deal with two main perspectives. On one side, there is a perspective where temperature operates on some characteristic of the organism that does not change in association with the phenotypic change observed at the organismal level (including organs and systems). For example, at the molecular level, aerobic organisms show a common structural basis of metabolism, underpinned by enzymes and associated reactions which drive metabolic rates[30–32] according to laws of thermodynamics[33]. Hence, in that perspective, interactions between temperature and such molecules would drive biological rates irrespective of the stage of development (Fig. 1a) and development is viewed as a revelation of enzyme reactions at the macroscopic level[26] Alternatively, such characteristic varies in response to the important amount of phenotypic change, associated to the formation of tissues and organs during development (e.g. crustaceans[34], Fig. 1b). Perhaps, regulatory processes, controlling the timing of key life cycle events can also modify effects of temperature, known to occur at the molecular level. In the 'Results' we introduce the concept of symmetry, which provides us a way to consider the above-mentioned perspective. We then introduce three hypothetical scenarios of the effects of temperature on developmental time and consider each scenario in a separate section, followed by the discussion.

Results
Symmetries
We use the concept of symmetry[35–39] as a guide to account for the above-mentioned perspectives. The most common usage of the term symmetry refers to a change in shape of a geometrical object (Fig. 2a, b), after we perform a change in its orientation: for example, a jellyfish (seen from above) possesses radial symmetry because its shape does not change (= it is
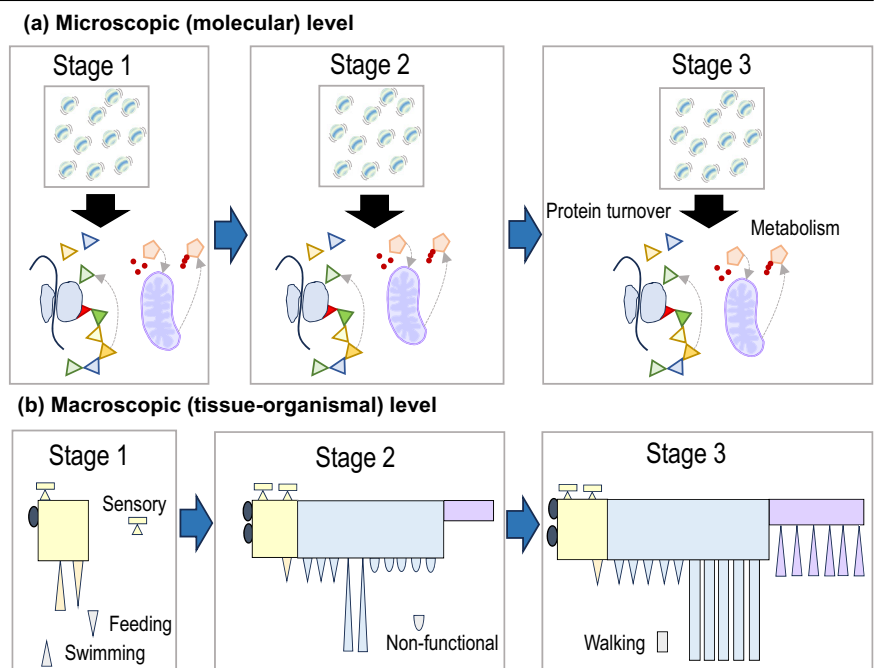
said to be 'invariant') after a rotation of its body. There is an analogous usage in developmental biology (Fig. 2c) where we observe symmetry breaking[37,40–43]: early-stage embryos consist of a homogeneous group of cells, but they develop into a spatially complex structure[44] through asymmetric cell division. Symmetries occur also with respect to time; they are called 'time-translation symmetry': as we observe the change of a system through time, some quantities (e.g. energy in a conservative physical system[45]) or properties remain unchanged (Fig. 2d, e). Predictability of outcomes of experiments also implies a time-translation symmetry where the same scientific law (governing the experimental outcome) applies at different points of time[36]. In all those cases, symmetries involve some 'immunity' to a change[36] occurring in a system (e.g. rotation in space, translation in time). We apply this concept to our different perspectives: for instance, the first perspective (Fig. 1a) provides an example of time-translation symmetry, where some property of an organism remains unchanged (it is preserved) despite the phenotypic changes experienced during development.

Organisms and modelling scenarios
We partition the life cycle into a set of stages, i.e. discrete units (different phenotypes), of variable duration. During development, temperature is allowed to change between stages, but the temperature within a given stage is considered constant. In realistic scenarios, temperature would change continuously over time; in our approximation, we consider that the life cycle can be divided into a sufficiently large number of stages of short duration such that the most important changes in temperature occur between stages and an average temperature approximates well the conditions experienced within a stage.

We consider organisms as systems made of (biological) components, defined here as the collection of traits and processes, which interact with temperature and determine developmental time. Components may be defined at the different levels of organization[46,47], i.e. from molecular to organ and the systems they compose. We follow the so-called relational approach[18–20,48,49] (Supplementary Note 1) in that components are defined by their outcomes (=developmental time in most of the components considered here) rather than by their structure. Hence, we do not try to associate components to parts of organisms; instead, we try to infer from the equations, whether components should vary among developmental stages.

Fig. 1 | Two different perspectives about the effects of temperature on biological time. Perspectives are for an ectothermic organism, with a crustacean developing through three stages as an example. **a** At all stages, and irrespective of morphological change, organisms are viewed as a collection of molecules (abstracted as spheres in a box). Higher temperatures (increased kinetic energy) result in increased rates of molecular contact, protein turnover (protein synthesis in ribosomes) and metabolism (ATP synthesis in mitochondria), which instead drive developmental time. Developmental time responds mainly to laws of thermodynamics irrespective of phenotypic change; note that the same processes are represented at all stages (panel based on Dorrity et al.[31] and Dias-Cuadros[32]). **b** Development exemplified as the formation of new body parts and remodelling appendages in decapod crustaceans. From this perspective, the effect of temperature on developmental time should be stage-specific. Main morphological changes are abstracted as the appearance of new geometric shapes representing new body parts or changes in appendage morphology and function (detailed morphology in ref. [34]). Nauplius (stage 1): single eye; zoea (stage 2): thorax and abdomen with new appendages; decapodid (stage-3): new abdominal appendages.

(a) Microscopic (molecular) level

Stage 1 | Stage 2 | Stage 3

Protein turnover | Metabolism

(b) Macroscopic (tissue-organismal) level

Stage 1 | Stage 2 | Stage 3

Sensory | Feeding | Swimming | Non-functional | Walking

Fig. 2 | Simple cases of symmetry transformations and symmetry breaking. a Some triangle rotations (e.g. with angle θ = 120°) are symmetric because the triangle orientation does not change; the rotation is identified by looking at the vertex labels. **b** In an object with parallel stripes (e.g. an idealised tiger skin) some stripe permutations (see labels) constitute symmetric transformations because they leave the pattern unchanged. **c** Embryonic development (sea urchin as example) can be viewed as a series of symmetry breaking transformations: here, individuals develop from an embryo of four equally sized cells, towards a form with increasing spatial structure: a 60-cell embryo, where cells located at different positions, give rise to different cell layers and tissues in the larval stage (simplified from Fig. 17.5 in ref. [43]). **d** Loop in music: the same pattern of four chords (Am, Em, G, D, made of half-tone notes) is repeated over time. **e** Emmy Noether's theorem shows that energy conservation through time (where forces are conservative) implies a time-translation symmetry[45]. For example, in an idealised oscillating pendulum (no dissipative forces), the total energy is constant through time, although the kinetic and potential energy will vary.
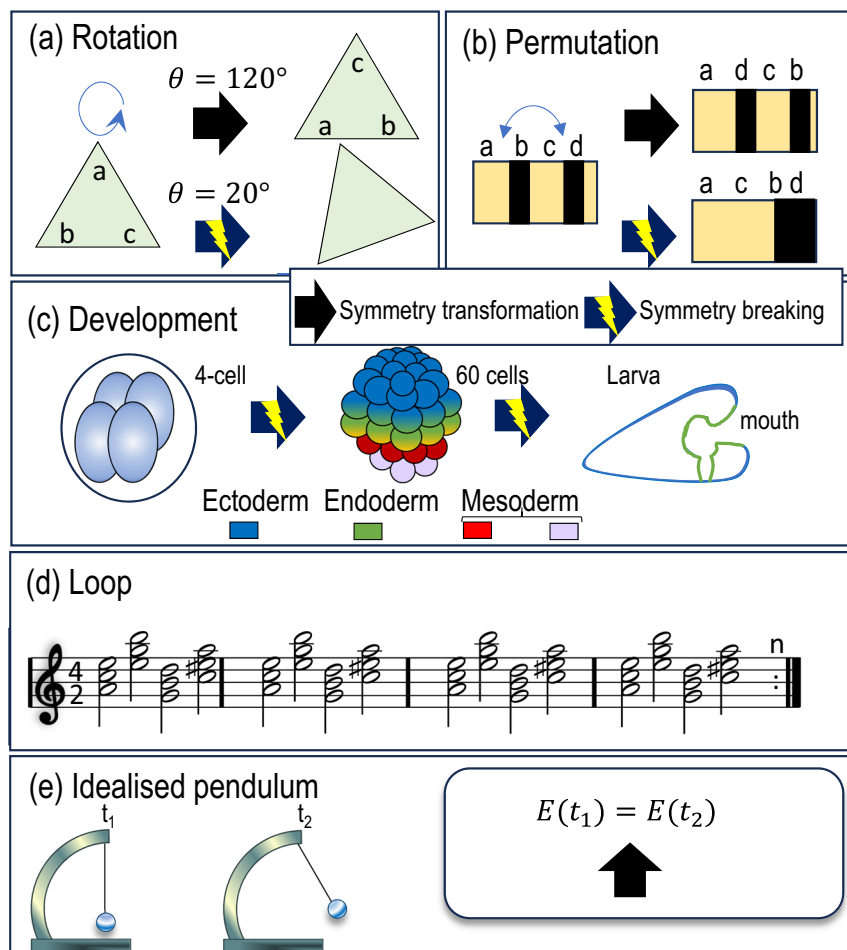

Importantly, because temperature has pervasive effects on all organism features, components do not have a specific location in the body. Components may not only correspond to specific phenotypic traits but also to the genome. This is because, temperature does not only affect the stability (or activity) of proteins and lipids (comprising the phenotype) but the properties of DNA[50], including a type of damage called oxidative stress[51] and transposon activity (via heat shock), which is thought to contribute to gene mutations and result in the generation of phenotypic variants[52,53].

We explore three hypothetical scenarios of increasing complexity (Fig. 3). Scenario 1 (Fig. 3a) has the simplest model where there is a pre-existing 'functional component' of the organism, so named because it is represented by a mathematical function, $f(T)$, of temperature, T. This component remains unchanged along development, i.e. it drives development time, irrespective of phenotypic change. Hence, this scenario accounts for the first perspective (Fig. 1a), where at all stages, temperature drives kinetic energy and contact rates among molecules $f(T)$. The component would represent the collection of macromolecules that respond to increased temperature by increasing reaction rates and shortening developmental time. There will be structural differences among molecules (and macroscopic differences) at different developmental stages; however, in this scenario, they are not required to explain how temperature drives developmental time. Any effect of those differences will be introduced as a stage-dependent constant (see below).

Scenarios 2 and 3 account for the second perspective, where the functional component changes along development, i.e. phenotypic changes along development cannot be simply accounted for by a multiplying constant. In scenario 2 (Fig. 3b), there is a pre-existing set of stage-specific functional components and associated mathematical functions $f_s(T)$. Here, the component would represent various features, such as signalling

networks or other factors driving the timing of specific developmental events. For example, for embryonic development (some vertebrates and invertebrates), include signalling networks based on molecular clocks[18,54,55] and mechanical interactions among groups of cells[56]. Moulting through different larval stages is regulated by the action of moulting hormones in e.g. crustaceans[57]. In addition, for some arthropods, availability of oxygen to cells appears to mediate the effect of temperature on stage duration in cases where stages are completed once individuals reach a critical mass[58–60]. Irrespective of the details, under scenario 2, the timing of events, driven by those processes, will have to be modified by temperature in a stage-dependent manner.

In the first two scenarios, the components are fixed ('pre-existing'). In the third scenario (Fig. 3c), temperature operates in two steps, affecting the characteristics of the functional components (i.e. they are considered as 'plastic'), leading to historical effects. First, temperature interacts at a given stage (S₁ in Fig. 3c) with a so-called "operator component" responsible for the formation of the functional component. Subsequently, temperature interacts with the functional component (at stages S₂ or S₃ in Fig. 3c) to drive developmental time. An example would be a plastic response where temperature triggers gene expression programs (from off to on), subsequently driving the formation of physiological phenotypes. Here, the operator component would represent the transformation of a cue to a signal leading to the formation of a new phenotype (the functional component), driving the effect of temperature on developmental time. Such a form of plasticity implies that the causal relationship between temperature and developmental time cannot be solely encoded as a set of stage-specific functions $f_s(T)$. Instead, the encoding should also include an expression of the action of the 'operator component', given by a mathematical mapping (details in section 'Scenario 3: Plasticity').
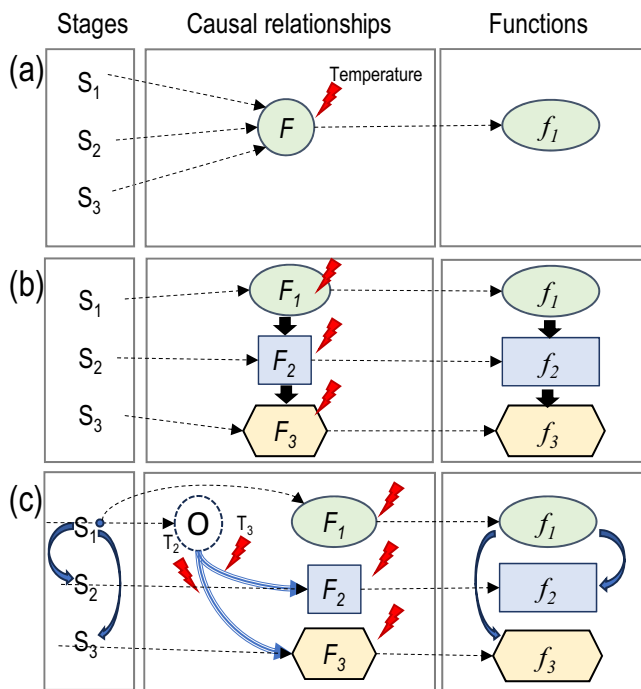
Fig. 3 | Scenarios of causal relationships between temperature and developmental time. Dashed arrows show the correspondence between stages, components and mathematical functions. F: functional components of the organism interacting with temperature at a given stage and leading to the developmental time at the same stage. The example is based on three stages, S_1–S_3 that occur either sequentially (**a**, **b**: black arrows) or defining alternative pathways (**c**: blue arrows). **a** Component conserved along development: All stages share the same functional component, leading to a single function. **b** Stage-specific components: functional components (and functions) are stage-specific. Organisms develop through a fixed pathway. **c** Plasticity: Stages do not necessarily coincide with morphological stages. The manifestation of a given functional component (F_2 or F_3) depends on the temperature (indicated as T_2, T_3) experienced in the previous stage (S_1) and interacting with an 'operator component' (O). Hence, organisms develop through alternative pathways (blue arrows), also reflected in a sequence of functions driving the effect of temperature on developmental time.

Equations

The effect of temperature on stage-specific developmental time (D_s) is expressed as a product of two terms; i.e. the mathematical function $f_s(T)$, multiplied by a constant ($=a_s$), such that $D_s = a_s \times f_s(T)$, where s indexes stage number ($s = 1,...,n$). The constants a_s provide the units of time while $f_s(T)$ may be a unitless quantity. In all scenarios, the multiplying constants, a_s are allowed to differ among stages. For example, a_s may respond to body mass, but the central point is that each a_s does not depend (either implicitly or explicitly) on the temperature. Stage-specific functional components differ in $f_s(T)$ only in scenarios 2 and 3. The $f_s(T)$ functions may vary in the functional forms or by different parameter values within a given functional form.

Several mathematical functions are used to model developmental time[26,61–69] (examples in Table 1). For instance, in the model developed in the context of the metabolic theory of ecology (UTD)[61,69], developmental time is driven by biochemical kinetics underpinning metabolic rates and can accommodate the effect of body mass[70]. The UTD model was fitted to larval developmental time[14]. Subsequently, an extension of the previous model was proposed that was able to capture non-monotonic responses to temperature[64]. This latter model fits different types of biological rates and is a candidate for scenario 1, i.e. applying to all stages of development (as in Fig. 3a). There are three conditions that validate the model at the organismal level of organization[63]. For example, within an organism, there will be different cell types, and the formation of tissues depends on timely cell division

and differentiation[71]. Suppose that the time for division and differentiation of a given subgroup of cells, associated with a given structure, defines the time to a given stage. If cells only differ in the multiplying constant (k_1), but not in the function $f(T)$, the expected duration of development is given by $E(D) = K_1 f(T)$, where K_1 is the average of the cell-specific constants. Hence, the functional relationship between temperature and developmental time appears at the level of a group of cells. Under small variations in the parameters of the function (e.g. k_2 in Table 1) one can approximate the average value of $f(T)$ as a function of the average of such parameter[64].

Because the focus of the current approach is not on the specific within-stage process, but on whether it remains preserved along development, the specific functional form (or parameter value) is not important here. Instead, the focus is on the different forms taken by the expression $D_s = a_s \times f_s(T_s)$, where the temperature and the functions can be either constant or vary among stages. Temperature could also change within a given stage, but we focus on the cases where changes in temperature occur in association with changes in the developmental stage, where the function $f(\cdot)$ is allowed to be stage-specific. Whenever the effect of temperature is driven by a single function within stages (both $f(\cdot)$ and a_s are constant), but temperature varies through time ($=t$), the stage-specific developmental time is given by $D_s = a_s E[f_s(T(t))]$, where $E[]$ is the expectation operator giving the mean value.

The process of development will be represented as a matrix and a vector in a configuration space. In the matrix (Table 2) rows are developmental sequences and columns are stages. Through this approach, we will be able to visualise the consequences of the different scenarios and relate biological processes to permutations of either temperatures or whole terms, across columns or rows. The state of the organism is described as a vector in the configuration space (Fig. 4) defined by the identity and number of stages ($s = 1,...,n$). The coordinates in each dimension of such space are functions of Ds. For an organism passing through 2 stages, where $D = D_1 + D_2$, there is a 2D space (Fig. 4a). Biologists usually represent stage-dependent development as plots of pairs of stage-specific developmental times (e.g. plot D_2 vs. D_1 in the above example, Fig. 4b) to explore different developmental processes[72,73]. However, because the emphasis is on the relationships between the stages, the absolute value of the duration of development (giving the vector length) does not matter for our current analysis. Instead, we will consider the 'normalised' vector (of length $\Phi = 1$), such that the stage-dependent developmental times are proportions of the total duration of development. Thus, we have $1 = \Phi = \Phi_1 + \Phi_2 + ... + \Phi_n$, where each proportional time is $\Phi_i = D_i/D$; proportional time is the square of the i-coordinate (φ_i) of the vector in the i-axis ($\Phi_i = \varphi_i^2$). For an organism developing through only two stages ($n = 2$), we would get $1 = \Phi = \Phi_1 + \Phi_2 = \varphi_1^2 + \varphi_2^2$. A rotation of the vector in the configuration space (Fig. 4a) corresponds to a permutation of temperatures across stages in the matrix; a rotation of the axes in the configuration stage, corresponds to a permutation of columns in the matrix and to the so-called passive symmetry transformation[33].

Scenario 1: component preserved along development. In the first scenario, we consider the simplest model (Fig. 3a), where the functional component, represented by the function $f(T)$, persists across stages:

$$D_{Ts} = \sum_{s=1}^{s=n} a_s f(T_s) \tag{1}$$

Recall that, in this model, any effect of the phenotype on developmental time is captured in the multiplying constant, which is allowed to be stage-specific. For example, such a constant may scale with body mass[69]. In such a case, mathematically, we have a separation of variables; for Eq. (1), we re-write $a_s = g(B_s)$, i.e. as a function of stage-specific body mass, B_s, and obtain stage-specific terms $g(B_s)f(T_s)$.

When temperatures are constant, Equation-1 predicts that we can ignore stage-dependent development to understand the effect of temperature on the total developmental time, because we can replace the multiplying constant by a single number: this is because $D_{Ts} = f(T) \times nE(a_s)$ where such

Table 1 | Example of mathematical functions used to model the effect of temperature on developmental time

Name	Function	Reference number
Linear degree day	$k_1/(T-k_2)$	Ref. 26
Arrhenius	$k_1 \exp(k_2/T)$	Ref. 61 Equation 5
Power	$k_1 T^{-k_2}$	Ref. 62
Belehrádek	$k_1(T-k_2)^{-k_3}$	Ref. 63
Eyring	$k_1 T^{-k_2} \exp(k_3/T)$	Ref. 64

In all cases, T is temperature and k_1, k_2, k_3 are equation-specific constants. All constants labelled as k_1 are either multiplying constants as defined above in the main text, or they are rate constants (when the associated function has units). This difference is not relevant as long as they do not depend on temperature (hence, becoming a variable).

number is given by the product the number of stages (n) and the average of the stage-specific multiplying constants $E(a_s)$. The model also implies that stage-specific proportional time Φ_s does not depend on temperature, if all stages experience the same temperature over development, we obtain:

$$\Phi_s = \frac{a_s f(T)}{D_{Ts}} = \frac{a_s}{\sum_{s=1}^{s=n} a_s} = b_s \quad (2)$$

The change in temperature is represented in the matrix (Table 2, rows 1 and 2) by a permutation of all temperatures T_1 of row 1 for all T_2 of row 2, which does not change any of the Φ_s in Eq. 2. In terms of the configuration space (Fig. 3), the replacement of temperatures leaves the vector unchanged (it does not rotate). Furthermore, in this model, we can permute terms across columns without any change in the outcome. The first permutation reflects a scaling symmetry (ratio of stage-specific to total development does not change), implying that the same causal relationship is sustained through time, despite the phenotypic change occurring through development. The second permutation shows that the temporal order of the stages does not matter for how temperature drives developmental time.

When an organism develops under varying temperatures, the symmetry in proportional time is not manifested because:

$$\Phi_s = \frac{a_s f(T_s)}{D_{Ts}} \quad (3)$$

In the matrix (Table 2), this case is represented by rows 3 and 4, and any change in temperature is expressed as permutations of temperatures across rows: those permutations are equivalent to the creation of a thermal fluctuation; permutations of temperatures across rows change the timing of the fluctuation. In the vector representation, there is now a rotation by an angle. However, in the matrix (Table 2), we can still permute the entire columns without changing the value of the total duration of development; in the configuration space, a rotation in the axes still does not change the total duration of development; hence, the passive symmetry transformation is still valid. Those transformations are therefore symmetric and highlight that (for this case) the temporal order of the stages does not matter for the calculation of developmental time.

The full stage-specific effect of temperature can be still represented by an average because we can re-write Eq. 1 as if the organisms were passing through stages characterised by equal values of the multiplying constants. By doing such a modification, the total duration of development is:

$$D_{Tj} = \sum_{j=1}^{j=M} c f(T_j) = cM E[f(T_j)] \quad (4)$$

In Eq. 4, there are M substages all with the same multiplying constant ($=c$). Hence, there is no need to know the order in which stages occur, nor the order in which temperature changes.

This model is the most symmetric, and developmental stages can be thought of as interchangeable modules. Evidence of scaling symmetry has

been found in studies monitoring developmental time at constant temperatures (and given different names), in some copepods[74], zebrafish[75], fruit fly embryos[76], crab *Hemigrapsus sanguineus*[22] and in nematode *C. elegans*[72] (Fig. 3b and Supplementary Note 2). However, there is also evidence against proportional symmetry in many arthropods[77] (90% of 78 species) and the nematode *C. elegans*[73]. For *C. elegans*, different temperatures resulted in the permutation in the order of developmental events (division in target cells and moulting)[72] and lack of proportional symmetry was found in the intermoult periods[73]. More generally, some scaling laws associated to the effect of body mass on biological rates appear to vary[78–80]. The Arrhenius model does not fully match effects of temperature on invertebrate larval development[14] nor on a stage-specific basis[81], but perhaps more recent models[63] will produce better fits. Recent studies[72,73] highlight the need for experiments of highly controlled temperature where organisms are monitored individually, where developmental time is quantified with high precision.

An important contribution comes from studies of molecular and organismal timekeepers within cells[12,32,71,80,82] and at the organ level[83,84]. Within cells, molecular motion drives cycles of synthesis and degradation of proteins and production and degradation of ATP, but cells also possess signalling mechanisms, controlling endogenous timekeepers. In multicellular organisms, hormones and other developmental regulators synchronise processes among tissues[57,84]. The latter mechanism defines checkpoints at different stages of development; their existence does not necessarily point towards stage-specific functions (e.g. nematodes[83]), but they raise the possibility of stage-dependent responses to temperature if stages are separated by checkpoints. The need to consider different types of clocks and the deviations from scaling symmetry[73,77] point towards a possible stage-specific regulation of the effect of temperature on post-embryonic development (scenario 2).

Scenario 2: stage-specific hardwired components. We now consider a fixed pathway of development, as defined by the set of functions (f_1,… f_n), (Fig. 2a). Here, the pathway of development is 'hardwired' in the sense that the functional components are pre-existing and not affected by temperature. Stage-specific biological time, D_{fs}, is given by functions, $f_s(T)$, such that:

$$D_{fs} = \sum_{s=1}^{s=n} a_s f_s(T_s) \quad (5)$$

This new model leads to the breaking of the scaling symmetry: for a given stage, Eq. 5 becomes:

$$\Phi_s = \frac{a_s f_s(T)}{D_{fs}} \quad (6)$$

The stage-specific function, $f_s(T)$ cannot be factored out and eliminated from the equation. Besides, we cannot replace the stage-specific effects of temperature by a single average value, as in Eq. 4. Instead, the best we can do is to express developmental time as:

$$E[D_{fs}] = \sum_{s=1}^{s=n} E[a_s f_s(T_s)] \quad (7)$$

In Eq. 7, the average function is applied on a stage-by-stage basis; it cannot be taken out of the summation even if the multiplying constants were all the same across all stages. Averaging as in Eq. 4 would be possible (as an approximation) only if the stage-dependent functions have the same form (e.g. all following the Arrhenius equation) and if the variation in the parameter characterising the stage-dependent functions are small[63]. Otherwise, we require information on the stage-specific functions, because changes in the phenotype modifies the effect of temperature on developmental time beyond those considered in the constants 'a_s'.

Table 2 | Matrix representing development sequences (rows) progressing through different stages (columns: 1,2,..s, N)

	1	2	s	N	Φ
(a) Scenario 1: Component conserved along development					
T_1	$b_1 \times f(T_1)$	$b_2 \times f(T_1)$	$b_s \times f(T_1)$	$b_n \times f(T_1)$	1
T_2	$b_1 \times f(\boldsymbol{T_2})$	$b_2 \times f(\boldsymbol{T_2})$	$b_s \times f(\boldsymbol{T_2})$	$b_n \times f(\boldsymbol{T_2})$	1
T_1 and **T_2**	$b_1 \times f(T_1)$	$b_2 \times f(\boldsymbol{T_2})$	$b_s \times f(T_1)$	$b_n \times f(T_1)$	1
T_1 and **T_2**	$b_1 \times f(\boldsymbol{T_2})$	$b_2 \times f(T_1)$	$b_s \times f(\boldsymbol{T_2})$	$b_n \times f(\boldsymbol{T_2})$	1
T_1 and **T_2**	$b_1 \times f(\boldsymbol{T_2})$	$b_2 \times f(\boldsymbol{T_2})$	$b_s \times f(T_1)$	$b_n \times f(\boldsymbol{T_2})$	1
(b) Scenario 2: Stage-specific - hardwired components					
T_1	$b_1 \times f_1(T_1)$	$b_2 \times f_2(T_1)$	$b_s \times f_j(T_1)$	$b_n \times f_n(T_1)$	1
T_2	$b_1 \times f_1(\boldsymbol{T_2})$	$b_2 \times f_2(\boldsymbol{T_2})$	$b_s \times f_j(\boldsymbol{T_2})$	$b_n \times f_n(\boldsymbol{T_2})$	1
T_1 and **T_2**	$b_1 \times f_1(T_1)$	$b_2 \times f_2(\boldsymbol{T_2})$	$b_s \times f_j(T_1)$	$b_n \times f_n(T_1)$	1
T_1 and **T_2**	$b_1 \times f_1(\boldsymbol{T_2})$	$b_2 \times f_2(T_1)$	$b_s \times f_j(\boldsymbol{T_2})$	$b_n \times f_n(\boldsymbol{T_2})$	1
T_1 and **T_2**	$b_1 \times f_1(\boldsymbol{T_2})$	$b_2 \times f_2(\boldsymbol{T_2})$	$b_s \times f_j(T_1)$	$b_n \times f_n(\boldsymbol{T_2})$	1
(c) Scenario 3: Stage-specific - plastic components					
Identity\Sequence	$j=1$	2	3	4	5
$i=1,2$	$b_1 \times f_1(T_1)$	$b_2 \times f_2(T_1)$			
$i=1,3$	$b_1 \times f_1(\boldsymbol{T_2})$		$b_3 \times f_3(T_1)$		
$i=1,4,5$	$b_1 \times f_1(T_3)$			$b_4 \times f_4(T_1)$	$b_5 \times f_5(T_1)$

Rows also represent different scenarios of temperature (T_1, T_2, T_3). The last column in (a) and (b) highlight that the vector associated to each case is normalised. (a) Scenario 1, conservation along development; for constant (rows 1,2) and variable temperature (rows 3-5). Arrows show permutation of temperatures across rows (creation of a thermal fluctuation) and columns (change in the timing of a fluctuation). (b) Scenario 2: stage specific components. (c) Scenario 3: plasticity; the temperature experienced by the first stage determines the stage identity (=i) and the subsequent path of development. Temperature drives the number of stages, with a switch between pathways of two vs. three stages. In (c) there is no notion of (the normalised) vector Φ.

In the model of Eq. 5, stages still act as independent modules. We can still obtain permutation and rotation symmetries, by normalising the stage-specific developmental time but recognising that the duration of development, D_{fs}, reflects a collection of stage-specific functions. A permutation of temperatures across columns (Table 2b) leads to vector rotation but conserves the vector length; rotation occurs now despite the temperatures being constant across stages. However, the permutation of full column terms and rotation of axes in the configuration space (Fig. 4) can still be carried out without changing the duration of development, showing that the order in the developmental sequence does not matter for the calculations.

Scenario 3: plastic components. In nature, the hardwiring of the internal systems driving developmental time may be counterproductive under situations of thermal stress. Instead, some compensatory-repair system may be necessary to deal with stress at the expense of a delay in development. When extreme temperatures perturb gene expression programs, cells do not progress in the cell cycle until damage is repaired[31,85]. Evidence of effects of a compensatory process on developmental time is provided by experiments showing that transfer from a low to a high temperature resulted in shorter developmental time than individuals exposed to that high temperature for the full larval development[86]. Hence, in response to heatwaves experienced at a given stage, developmental time may have to be adjusted through compensatory responses, perhaps operating at a subsequent stage. Compensatory responses may be activated by additional factors: for instance, in response to food limitation, many organisms attempt to maintain size at metamorphosis at the expense of extending developmental time, presumably because fitness costs of reduced body size are larger than that of delaying metamorphosis[87]. In addition, some organisms acclimate to temperature as they develop (developmental acclimation[88]), and temperatures experienced at a given stage could hypothetically drive the developmental responses in subsequent stages. Another important response is developmental plasticity, where organisms develop through alternative pathways, characterised by different numbers and types of stages. Developmental plasticity is a well-known phenomenon occurring, for instance, in nematodes, crustaceans, insects, and amphibians[89]. For example, shrimps develop through alternative developmental pathways characterised by different number of instars depending on e.g. temperature conditions experienced in early stages[90]. In those cases, the identity of the stage in a developmental sequence depends on the temperature experienced at the previous stage.
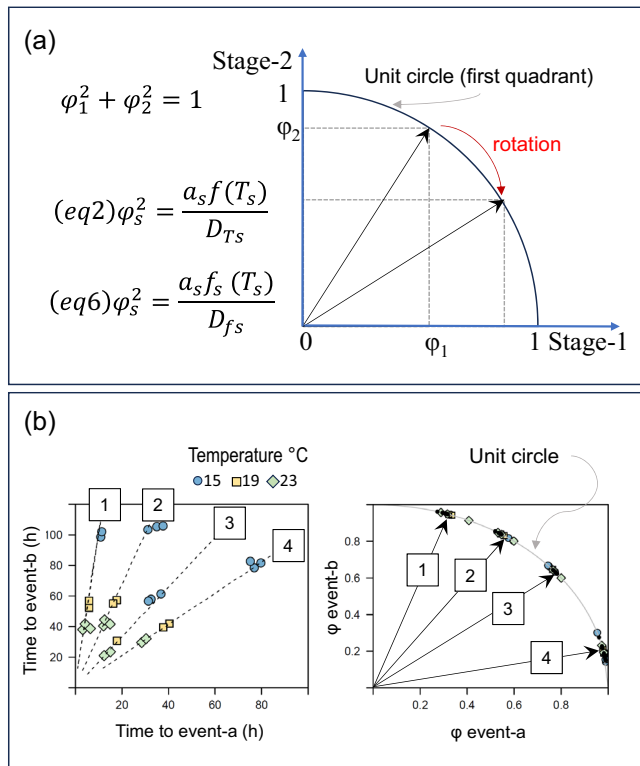
Fig. 4 | The configuration space representing the effect of temperature on development. **a** A case of two stages is given as an example for simplicity. Each axis of the configuration space is represented by a stage. The 'state' is given as the position of a normalised vector (length = 1). The vector coordinates ($\varphi_s > 0$) reflect the proportional time of each stage, which results in the vector being normalised (absolute time of development is not considered). This configuration applies to models of Eqs. 1 and 5. Changes in temperature leads to changes in the proportional time of each stage, which makes the vector rotate. The rotation occurs because temperature changes the proportional time. **b** Example based on an experiment on the effect of temperature (constant conditions, three levels) on the development of nematode *C. elegans* to different stages (=events); source: Fig. 2c of ref. 72. A rotation of the vector would correspond to a change in slope in Fig. 6C of ref. 72. Right panel: reconstruction of a subset of data; Left panel: normalised vector for each pair of events and for the dashed line (black dots). Details of methods are given in the Supplementary Note 2; data points are given in Supplementary Data 1.

Accounting for different forms of phenotypic plasticity motivates our third step (Fig. 3c), where temperature conditions at some stage shape the functional components in subsequent stages. In this case, there may be 'acclimation stages', not necessarily correlated with macroscopic morphological changes in the organism. In scenarios 1 and 2, the pathway of development, as defined by the set of functions ($f_1,...f_n$), are maintained irrespective of temperature (Fig. 5a), i.e. temperature affects the developmental time but not the functional components. By contrast, the forms of phenotypic plasticity described here, constitute a branching of pathways along development (Fig. 5b) where individuals will take one of the possible alternative routes.

In scenario 3, one must account for the fact that multiplying constants at a given stage may be driven by temperatures experienced at previous stages. In such case, development at a given stage D_s can be written as $D_s = h_{s,T(S-1)} f_{s,T(S-1)}$ implying that the 'constant' now becomes a function $h_{s,T(s-1)}$ of the temperature conditions experienced in a previous stage. However, because we are not focusing on details of the form of functions, we simplify the model in that any historical effect of temperature is accounted for a re-defined function $h_{T(s-1)} f_{s,T(S-1)} = f_{s,T(S-1)}$ such that $D_s = a_s f_{s,T(S-1)}$. Hence, any historical effect of temperature is accounted for by f_s. This approach is in line with the fact that components (as defined previously) can

be considered as composites of sub-components. Experiments will be needed to determine which sub-component and which parameter or term within $f_{s,T(S-1)}$ is driven by historical effects.

In a scenario of plasticity, permuting temperatures (Table 2c) can result in individuals going through a different pathway (= changing rows); hence, a single row no longer describes the correct developmental sequence. In addition, permuting full columns would mean retro causality because the branching of pathways cannot occur after organisms are set into a particular pathway. There is, therefore, time asymmetry highlighted by the preferred order of stages (i.e. we need to consider the order in which the stages occur as organisms develop). Because of such a preferred order, there should also be a preferred direction of orientation of axes in the configuration space, which leads to the breaking of passive rotation symmetry. The direction of how development proceeds is given by the stage where branching occurs (Fig. 5b).

Plasticity results in that stage identity and the number of stages become variables, reflecting the fact that the functional components are shaped by temperature (Fig. 3c). Therefore, the configuration space is not fixed for all temperatures experienced during development and the vector representation of Fig. 4 does not fully capture the effect of temperature on developmental time. All symmetries remaining under scenario 2 are broken in a scenario of plasticity.

The development of a specific mathematical theory for this mechanism is beyond the scope of this paper; here, we present a potential way to achieve more plasticity. This is a generic model for adaptive plasticity (Supplementary Notes 3), meaning that it captures the three main steps leading to the formation of the (adaptive) phenotype[91,92]. The model is based on the so-called relational approach to system theory, used to model anticipatory systems[18–20]. The causal structure driving the effect of temperature on developmental time is now expressed in the diagram.

$$T \xrightarrow{\eta} H(T, D)$$
$$\downarrow$$
$$T \xrightarrow{f_i} D$$

where η is a mathematical transformation (represented as an arrow) mapping temperatures, T, to the set of functions $H(T,D)$. Given a value of temperature, T_i, we get $\eta(T_i) = f_i$ a function. In the lower part of the diagram, the function, maps temperature to developmental time (D) as in scenarios 1 and 2. We must find a representation of the set of possible but not yet realised functional components as well as a mathematical representation of the 'operator component'; here we use an approach based on vector spaces and operators[93]. The set of potential responses to temperature (=n) is represented as a vector ($\hat{\Psi}$) in a vector space, based on an orthonormal basis, $\hat{e}_1, ..\hat{e}_i, .., \hat{e}_n$.

$$\hat{\Psi} = \sum_i f_i \hat{e}_i$$

The time course of a plastic response may be summarised in three general steps[91,92]: (1) an environmental cue is converted into a signal; (2) the signal triggers the formation of a new phenotype and (3) the new phenotype drives organismal performance. The operator must encode steps 1 and 2; it may represent two 'sub-components' acting in sequence. The first sub-component is represented as a 'step function' mapping temperature values into n-categories corresponding to the dimension of the vector space mentioned above. For example, with $n = 3$, the organism would classify temperatures in one of three categories (e.g. 'high', 'normal' and 'low') and respond to temperature through three different phenotypes. Each category corresponds to a signal represented by a vector, $\hat{e}_i^*$ such that $\hat{e}_i^* \hat{e}_j = 1$ if and only if $i = j$ (otherwise =0). The second sub-component is represented as the inner product of the signal vector, $\hat{e}_i^*$ with $\hat{\Psi}$ such that:

$$\hat{e}_i^* \cdot \hat{\Psi} = f_i$$
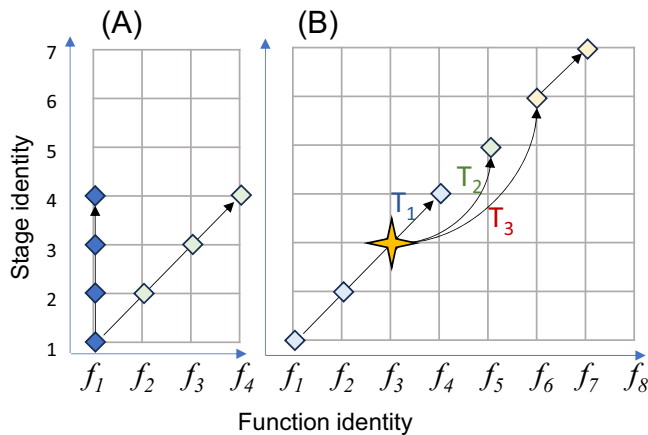
Fig. 5 | Developmental trajectories for the different scenarios. a Scenarios 1 (blue symbols) and 2 (green symbols) of fixed trajectories and configuration space. **b** Scenario 3: Trajectories are not fixed and instead depend on the temperature (T_1, T_2, T_3) experienced in stage-3 (star); when stage-3 is exposed to T_3, development occurs through an extra stage, which results in an increase in the dimension of the configuration space.

For example, the inner product would represent the action of an intracellular signal in triggering transcription of a specific region of the genome, initiating the formation of the new phenotype. Step-3 (from phenotype to performance) is represented by the function f_i, where performance is given as developmental time.

Discussion

Over many decades, scientists have discussed and attempted to derive explanatory models of the effects of temperature on biological time. Those models are now central for correct predictions of effects of climate change events (e.g. heatwaves) as organismal development affects population phenology, leading to potential mismatches with the timing of prey abundance or with periods of optimal growth or reproduction[9]. Developmental time can also have important indirect effects on fitness through the influence on, e.g. body size at metamorphosis or maturation[87,94]. This is particularly important in the context of climate change because, for example, most animals develop through complex life cycles with some form of metamorphosis[95]. Most tests of theories explaining timing and size at metamorphosis in marine organisms have been developed by manipulating food levels[94]. A major outcome of these experiments is the stage-dependent plasticity in developmental time, which would point towards scenario 3 in our framework (albeit, with regard to food). Longer developmental time seems to be part of a compensatory response to sustain body size at metamorphosis. However, increased temperatures can result in a failure of the compensatory effect[6]. Overall, those studies highlight the need for explanatory models linking temperature and developmental time. For example, the above-mentioned theory of metamorphosis still needs to incorporate results of experiments manipulating temperature on a stage-dependent basis to predict responses to heatwaves. Given the evidence presented here, a wide perspective considering potential stage-dependent effects and phenotypic plasticity will be appropriate.

It would be ideal to combine experiments with mathematical models representing the action of biological clocks on development[29], but such a level of detail is known for only a few species[54,55]. However, given that many marine organisms develop through distinct morphological stages, it is straightforward to experimentally test scenarios of different complexity. Studies based on static experiments, i.e. exposing organisms to constant temperatures, can be helpful to differentiate between scenarios 1 vs. either 2–3, but an appropriate range of temperatures should be tested. If data are

consistent with scenario 1 one could try to fit specific models. Recent models[64] are a starting point to develop tests on a stage-by-stage basis. This point can be seen from the following example: Suppose three species develop through two stages according to the three different scenarios; consider a static experiment (temperatures are kept constant over time) where different individuals are kept under different temperatures. Now assume that the response at each temperature depends on the following constants (a_i) and functions, $f_i(T)$, which are not known to the experimenter. For each species, we have:

$$\text{Species}_1 D_{Sp1} = a_1 f_1 \quad a_2 f_1$$
$$\text{Species}_2 D_{Sp2} = a_1 f_1 \quad a_2 f_2$$
$$\text{Species}_3 D_{Sp3, T < Th} = a_1 f_1 \quad a_2 f_2$$
$$\text{Species}_3 D_{Sp3, T > Th} = a_1 f_1 \quad a_2 f_3$$

where T_h is a thermal threshold (for species 3 only). In this case, it is easy to separate scenario 1 from 2 and 3. For example, for stage 2, in case of species 1, the stage-specific proportional developmental time is given by $\Phi_2 = a_2/(a_1 + a_2)$ irrespective of temperature, while in the remaining cases proportional times will depend on temperature: In species 2, we have $\Phi_2 = a_2 f_2/(f_1 a_1 + f_2 a_2)$, while in species 3 we have, for the range of temperatures above the thermal threshold T_h: $\Phi_2 = a_2 f_3/(f_1 a_1 + f_3 a_2)$. Preliminary tests of stage-dependent causal relationships may be carried out after systematic literature reviews, covering studies where the effects of temperature on developmental time were quantified on a stage-by-stage basis[76]. There are two important points to consider. First, whether studies have been carried out through group or individual rearing. Experiments using groups of individuals as replicate units will provide only average values of developmental time; in replicates, where mortality rates are high, the estimations of developmental times for early stages will be based on individuals that are not available for estimation at advanced stages. Some mitigation may be made by choosing replicate units (and thermal treatments) where survival rates are high. By contrast, experiments using individual rearing and having a tight control of temperature[72,73] will provide the strongest tests. The second point considers that, in testing the above-mentioned scenarios, it is assumed that each stage experiences a constant temperature. Most static experiments will start with all individuals at the same temperature, some of which will be distributed over the different temperature treatments. In such a case, it is important to identify the developmental stage (stage 1) where the change in temperature is experienced, and then remove such stage from the calculations.

While static experiments will help to test for stage-specific causal relationships, it will be very difficult to determine whether such a relationship is fixed or plastic (scenario 2 vs. 3) without knowledge of the functions before the experiment is performed. There might be indirect evidence pointing to scenario 3: for example, through graphical analysis, it may be possible to observe breaks in the overall functional response to temperature because the slopes of the functions will vary across the thermal threshold. In the above-mentioned example, in species 3, for stage 2, the slope of the relationship between temperature and developmental time will shift from $a_2 \partial f_2 / \partial T$ to $a_2 \partial f_3 / \partial T$ at the thermal threshold T_h. However, the detection of such a break will demand experiments using many temperature treatments on each side of the thermal threshold.

A full evaluation of scenario 3, which involves historical effects such as hardening, acclimation and carry-over effects[90,96,97] requires pulse experiments manipulating the duration, magnitude, and timing of a warm event, in relation to the stages under study[28,98–100]. The development of such experiments should also motivate studies quantifying how heatwaves drive biological time and phenology through causal links operating at different levels of organisation or on different traits (e.g. body size). Species characterised by developmental plasticity are crucial; among those, shrimps stand out because they sustain fisheries worldwide. Overall, the increasing frequency of marine heatwaves, demand a flexible approach towards understanding the causal links between temperature and biological time.

Data availability

Code availability

R code is provided as in the Supplementary Notes and can be accessed without restrictions.

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Acknowledgements

Author contributions

L.G. and G.T. conceptualised the study, created the figures, edited, reviewed and agreed on the final manuscript. LG wrote the graphical abstract, wrote the first draft and created the background and conducted data analysis. We thank Dr. Claudia Piccini and Falco Nozar for help with the musical score in Fig. 2d. We also thank the anonymous referees for their comments, which helped us to clarify our arguments and strengthen our article.

Funding

Competing interests

The authors declare no competing interests.

Additional information

Supplementary information The online version contains supplementary material available at https://doi.org/10.1038/s43247-025-02469-w.

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Peer review information *Communications Earth & Environment* thanks the anonymous reviewers for their contribution to the peer review of this work. Primary Handling Editors: Christopher Cornwall and Alice Drinkwater. A peer review file is available.

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