A simple demographic explanation for the evolution of the dietary restriction response and its ecological relevance

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Abstract

Considerable life history plasticity is observed in response to variation in food availability and composition. This is perhaps best known in the context of dietary restriction, which consistently induces lower reproduction and higher survival across taxa, with nutritional geometry studies further demonstrating the importance of food composition as well as amount. Although there is a huge amount of mechanistic work on understanding this response, models for how and why this may have evolved are limited and highly debated. Here, we show that a simple demographic model of evolution predicts that the dietary restriction response should evolve as adaptive life history plasticity in a fluctuating environment. This model involves less restrictive assumptions than previous theories, most notably that individuals do not need to return to good resources within their lifetime to gain a benefit from the dietary restriction response. We then explore how this approach might help us to understand and predict life history shifts in response to rapid environment changes in wild populations, and discuss how this perspective affects quantification of individual fitness in wild populations. By considering how population growth maps on the nutritional landscape, a nutritional geometry approach may provide deeper insight into the evolution of this plasticity.

Keywords: Caloric restriction, selection, generation time, population growth, density regulation, fitness

Introduction

Environmentally induced variation in the timing of important life events, known as life history plasticity, is observed across many contexts. Perhaps the best studied and taxonomically consistent life history plasticity occurs in response to changes in food availability and composition. In the laboratory, where food is typically provided *ad libitum*, this plasticity is demonstrated by dietary restriction (DR) studies, in which total amount of food or nutritional content is reduced. Across taxa, dietary restriction is generally found to increase lifespan (Nakagawa et al., 2012; Ivimey-Cook et al., 2025) and reduce reproduction (Moatt et al., 2016). Nutritional geometry studies further show that this may be driven by changes in availability of protein or micronutrients (Moatt et al., 2020). There is also experimental evidence in the wild that food supplementation increases reproduction (Ruffino et al., 2014), although no synthesis that we are aware of focuses on the impact of food supplementation on adult survival. Notably, this response is remarkably consistent across many animal taxa, with model systems for this response ranging from nematode worms, to flies, to vertebrates (Nakagawa et al., 2012; Ivimey-Cook et al., 2025; Moatt et al., 2016).

The dietary restriction (DR) response is well studied in the field of ageing, although typically from a mechanistic perspective. There are several hypotheses regarding the evolution of the DR response, with the most cited theory arguing that it represents adaptive plasticity (Holliday, 1989; Shanley & Kirkwood, 2000). Under this hypothesis, the reduction in reproduction and increase in longevity in response to DR allows an individual to 'wait' until environmental conditions improve. The DR response is therefore argued to represent evolutionary adaptation to deal with periods of famine, either in terms of overall amount or particular nutrients (Moatt et al., 2020). This model is reasonably complex and rests on the assumption of an individual returning to good resources during its lifetime. This has been criticised, most notably by Adler & Bonduriansky (2014), who argue that in the wild individuals are unlikely to survive to recommence reproduction later, and that the DR response is instead, a laboratory artifact.

Although typically conceptualised as pausing reproduction to wait out a period of famine, in life history terminology the DR response represents a plastic response to food availability that shifts the timing of reproduction, from high output early in life (high reproduction and low survival; i.e., a fast life history) in high food environments to reproduction spread through a longer life in food limited environments (low reproduction and high survival; a slow life history). Whilst we do not typically link food availability and population growth in laboratory studies, resource availability is intrinsically linked to population growth rate in the wild, with populations typically growing when food is plentiful and shrinking when food is limited. Using simple principles from evolutionary demography, we show that there is a selective advantage to reproducing early in a growing population and delaying reproduction in a declining population. This demonstrates how the DR response may represent adaptive plasticity, whereby individuals that show variation in life history strategy in response to fluctuations in food availability will contribute more to future generations, regardless of the speed of those fluctuations. Importantly, the benefit of this plasticity is not dependent on a return to good resources during an individual's lifetime, and holds regardless of whether an individual spends all their life in one environment, or whether the environment fluctuates across their life. Our model is simpler than previous ones (Shanley & Kirkwood, 2000) and addresses criticisms of the DR response representing adaptive plasticity (Adler & Bonduriansky, 2014). Importantly, this model considers the benefits of life history plasticity in terms of adopting the best strategy within a given environment (i.e. relative fitness), rather than considering absolute fitness over a lifetime. As this plasticity is pervasive, the benefits of this plasticity are intrinsically hard to experimentally demonstrate. We therefore consider where we might find support for this model in the wild. Finally, we consider the importance of this plasticity for the assumptions made when using lifetime measures of individual fitness to quantify selection in wild populations.

Dietary Restriction in an Evolutionary Demography Framework

The problem that we are trying to address here is why the timing of reproduction would plastically shift in response to resource availability. Although we do not typically think about populations declining in the benign conditions of the laboratory, resource availability is likely intrinsically linked to population growth rate in the wild. It is well established that the population growth rate impacts how and whether the timing of breeding is under selection (Charlesworth, 1994; de Vries et al., 2023). To understand this, we can consider that it is beneficial for an individual to put its offspring into the population when it is smallest, as its offspring will take up a larger proportion of that future population, and so that individual's descendants will spread through the population faster. This argument focusses on an individual's relative fitness within an environment rather than its absolute fitness across environments. To emphasise this point, we can assume that all individuals have the same number of offspring over their life. When the population is stationary (i.e., not growing or shrinking, with growth rate $= 1$), it does not matter whether an individual breeds early or late, because there is not a time when the population is smaller, and so there is no optimal time to put their offspring into the population (Figure 1a). However, when a population is growing (growth rate > 1), given the same overall offspring production, it is beneficial to breed earlier, because the offspring (and its descendants) will form a larger proportion of the future population than if it breeds later (Figure 1b). There is, therefore, selection for earlier breeding or a fast life history. Conversely, when a population is declining (growth rate < 1), it is beneficial to breed later, as the offspring will be a larger proportion of the population if they are put in later (Figure 1c). There is therefore selection for a slower life history in declining populations. This latter example is hard to conceptualise, because given a constant negative growth rate eventually this population will go extinct. Put another way, given the same total offspring production, a slow life history strategy will go extinct slower than a fast life history strategy; it is therefore *relatively* better to have a slow strategy. The timing of

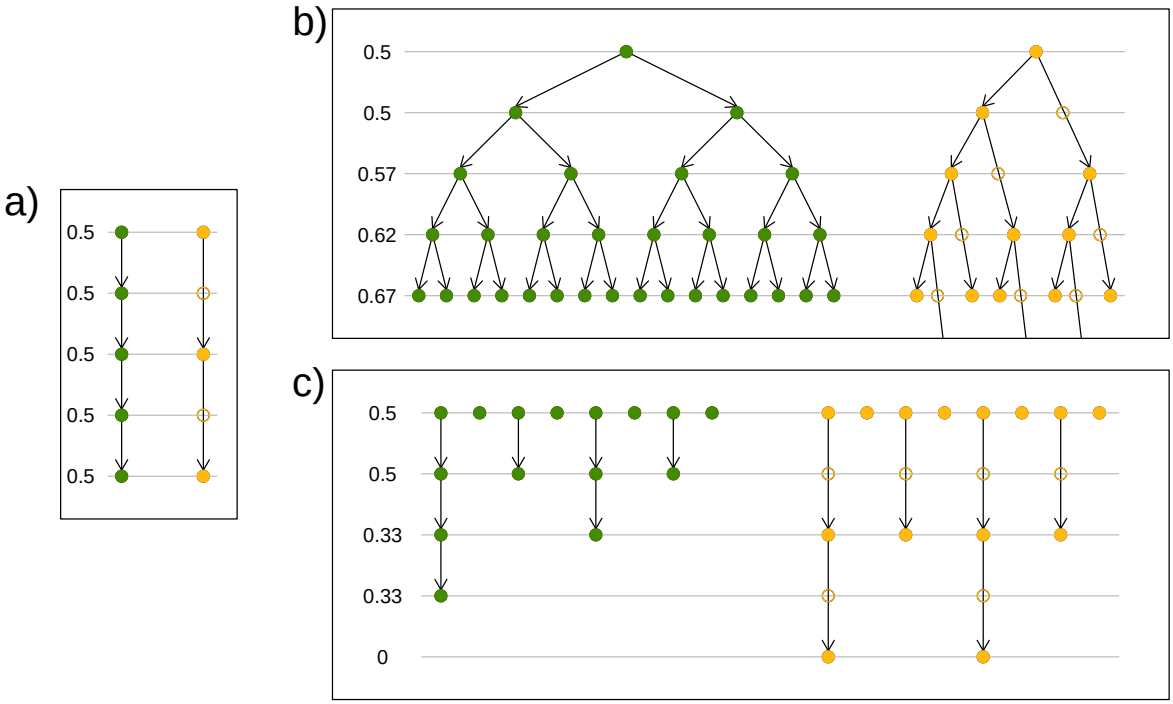

Figure 1: Simple representation of the effect of timing of reproduction on the spread of different strains depending on the growth rate of the population. Here, we consider two strains: 'fast' (green) with individuals (represented by filled circles) that reproduce immediately (represented by arrows to new individuals) and die, and 'slow' (yellow) with individuals that reproduce the same amount, but also survive one time step (represented by empty circles) and spread reproduction out. The numbers on the left hand side of each panel show the proportion of the population that is green. In a), the population is stationary with all individuals having one offspring, the fast strain immediately, and then slow strain after one time point. In b), the population is growing, with all individuals having 2 offspring, the fast strain having both in one time point, and the slow strain spreading them out over two time points. In c), the population is declining, with individuals having 0.5 offspring on average (i.e. half of the individuals reproduce once), with the fast strain immediately and the slow strain after one time point.

breeding therefore determines a 'strains' capacity to invade a population, depending on the population growth rate at that time.

To our knowledge, most previous work on life history strategies has focussed on the evolution of fixed strategies rather than explaining the existence of life history plasticity. Here, we put forward a simple model to demonstrate the benefits of such plasticity. We first use our model to confirm that the timing of reproduction is under selection dependent on the population growth rate in a constant environment, with low reproduction and high survival favoured whenever the population is declining, and *vice versa*. We then consider a fluctuating environment, and the relative benefits of fixed and plastic strategies. We show that as population growth fluctuates, we expect the evolution of plastic strategies, similar to

the DR response, even if fluctuations do not happen within an individual's lifetime. Thus, we can explain the plastic DR response without the need for a return to good conditions (i.e. population growth) within an individual's lifetime. We then validate this model using simulations. Whilst it is unsurprising that we might expect plasticity to be optimal in a fluctuating environment, our purpose here is simply to demonstrate that the plasticity that we commonly observe is consistent with the plasticity that we would expect.

Model

Let us consider a discrete-time model of a population of an asexual clonal species with overlapping generations and multiple strains. This population experiences different environments which affect survival and reproduction (vital rates) and, thus, the population growth rate.

All individuals within a strain have the same expected vital rates. We assume that individuals have constant vital rates within a given environment. In other words, if an individual is within the same environment for its whole life, it will always have the same expected fecundity and survival and so its expected total reproductive output (R_0) is given by $\frac{f}{1-s}$, where f is fecundity rate and s is survival rate across a time-step. We can therefore calculate the expected instantaneous growth rate (C) of a strain across time points within a given environment as $f + s$. We therefore assume there is no age structure (i.e. vital rates do not vary with age) and no senescence.

Here, we specifically want to explore selection on the timing of reproduction (i.e. when individuals put their offspring into the population). We therefore assume that within a given environment, all strains have the same expected total reproduction across their life (R_0), but they vary in their timing of reproduction. By holding R_0 constant, we ensure that there is no global selection across strains driven simply by differences in total offspring production, and so any differences between strains in their respective growth rates is due solely to differences in their timing of reproduction. This model formulation has been used elsewhere for similar

purposes (de Vries et al., 2023). Variation in the timing of reproduction could be conceptualised in several ways, and we choose to define this in terms of generation time. Given a constant rate of fecundity and survival across life, the expected generation time for a strain is $\frac{1}{(2-2s)}$ (or half the expected lifespan). We can therefore alter the timing of breeding by varying the survival rate of a strain. For strains to have the same R_0 in a given environment, a difference in survival among strains requires an opposite difference in fecundity. This generates a strong negative relationship between survival and fecundity across stains; individuals from a short generation time strain produce all their offspring early and then die early, whilst individuals from a long generation time strain live longer and spread out their reproduction across their lives (we assume that there is no post-reproductive lifespan). This is analogous to thinking about fast and slow life histories, meaning that the strains vary across this continuum. We argue that this is not necessarily synonymous with a reproduction-survival trade-off, as higher reproduction does not cause lower survival (i.e. we assume no cost to higher reproduction) and there is no energy allocation between different functions, the variation in timing is simply conceptualised this way. We also note that these results can also be achieved using other, perhaps less biologically realistic ways of varying life-history (for example, all individuals having the same lifespan and varying in their year of reproduction; we present this model in the Supplements). Our model is therefore consistent with, but does not require, a reproduction-survival trade-off.

Although DR is typically conceptualised in terms of two environments: DR (bad environment; food or nutrient restriction) and no DR (good environment; often *ad libitum* food), we would realistically expect an environmental gradient. In our model, we assume that the environment alters the expected total reproductive output over an individual's life (R_0; Moatt et al., 2016). In a 'good' environment, $R_0 < 1$ and if the environment is 'bad' then $R_0 > 1$. Although the overall growth rate of the population will be some combination of the growth rate of individual strains, as our model has no age structure, we know that when $R_0 > 1$ the overall population will be growing, when $R_0 < 1$ the overall population will be declining, and when the expected $R_0 = 1$ the overall population will be stationary.

Although we consider this in the context of food availability or nutritional quality here, we note that this could be driven by any environmental variable that impacts total reproduction and so population growth rate (e.g. temperature).

In Figure 2, we show analytically how selection acting on the timing of reproduction depends on whether the overall population is growing (in a good environment) or declining (in a bad environment). For a given expected total reproduction (R_0; x-axis), we can see that there is variation in the possible growth rates of different strains (yellow area in Figure 2), except when the population is stationary, where total reproduction and growth rate are always equal to one. This area is bound by the 1:1 line, which happens when a strain is semelparous and individuals breed at age one and then die, and the nearly flat line with intercept near zero, representing a strain in which individuals have infinitesimally high survival and reproduce at an infinitesimally small rate (approximating individuals that never reproduce and never die).

The lines in Figure 2 then represent strains with different generation times. Given that

$$R_0 = f/1 - s \tag{1}$$

and

$$C = f + s \tag{2}$$

, we can show that

$$C = 1/(1 - s) * R_0 - s/(1 - s) \tag{3}$$

. As shown above, the generation time is a simple function of s, and holding s constant (giving a fixed generation time) gives a linear relationship between R_0 and C, a strain's instantaneous growth rate. Figure 2 shows that when the population is declining (i.e., a bad environment, R_0¡1) slow strains will spread faster than faster strains, and vice versa when the population is growing (when the environment is good). In other words, there is selection on timing of breeding based on the overall population growth rate. This model also shows that it is best to spread reproduction throughout a longer life when resources are low and

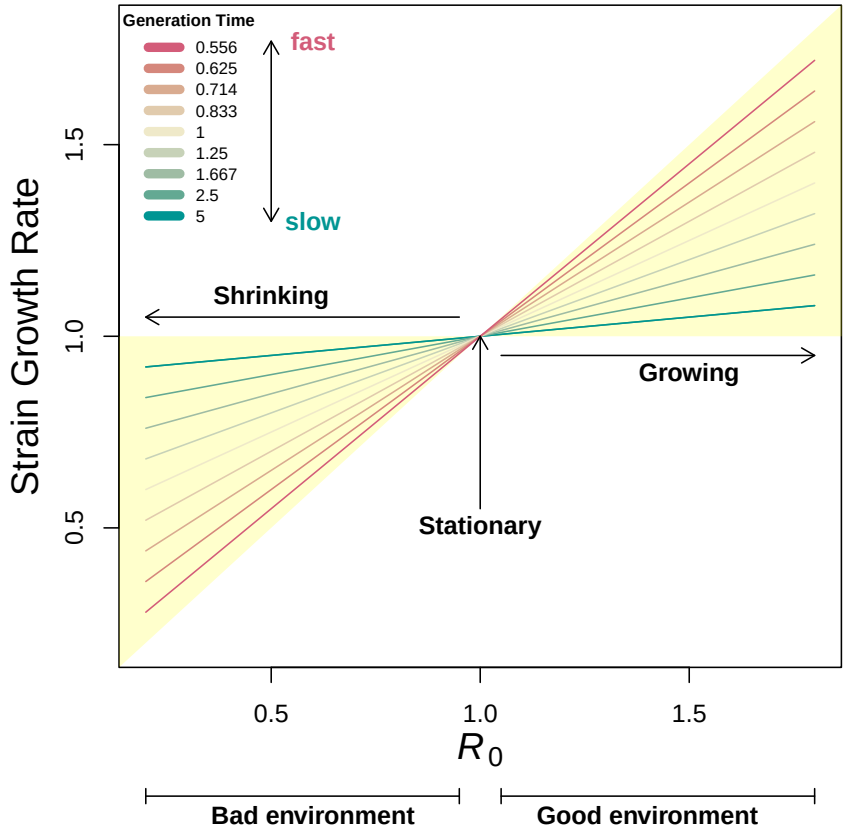

Figure 2: How variation in the timing of reproduction affects strain growth rate. For a given total reproductive output (R_0; x axis), there is variation in the potential growth rate across strains (yellow area). This variation is caused by the effect of the timing of reproduction on growth rate, illustrated by different strains that vary in their generation time (coloured lines). When the overall population is declining, for the same total reproduction, strains with a slower life history have a relatively faster growth rate. When the overall population is growing, for the same total reproduction, strains with a faster life history have a relatively faster growth rate.

the population is declining (as in the DR response), regardless of whether you return to good resources within your lifetime.

Fluctuating environments

In natural systems, environments are not constant. We can imagine a variable environment, where on average the environment gives an expected R_0 of 1, and so the expectation of population growth rate would also be 1 (i.e. a stationary population). Looking at Figure 2, we can see that in a fluctuating environment it would be optimal to be plastic, and switch from a fast strategy when the environment was good and the population is growing, to a

slow strategy when the environment was bad and the population declining. Note that this is exactly the plasticity that we see in the dietary restriction response (low reproduction and high survival in a low food environment, high reproduction and low survival in a high food environment).

To conceptualise this in more detail, we can first consider a scenario where the fluctuations are very slow and the time spent within one environment is very long (i.e., positive temporal autocorrelation). In this scenario, the average individual spends its entire life in one environment. Here, we would expect that a plastic strategy will be optimal, where individuals adopt a fast strategy in good environments and a slow strategy in bad environments, as this would allow them to achieve the highest growth rate given the environment (i.e. the highest *relative* fitness). In the context of very slow fluctuations, a form of developmental plasticity might be selected for, where individuals adopt a particular strategy (fast or slow) at birth depending on the prevailing conditions. Although reversible plasticity would also achieve this optimisation, it would not be strongly selected for, given the infrequent fluctuations, and that most individuals would experience a single environment during their life. As discussed above, established models of DR as adaptive plasticity (Shanley & Kirkwood, 2000) assume that the benefit of the DR response comes when individuals *return* to resources within their lifetime. This model shows that the return to resources is not necessary, as it is still best to adopt a slow life history when the population is declining, regardless of whether you return to good resources within your lifetime, as this strategy maximises an individual's *relative* fitness.

Similarly, plasticity would also be favoured in a rapidly fluctuating environment (negative temporal autocorrelation). Here we might expect more dynamic, reversible plasticity, where individuals are selected to shift between strategies when the environment changes. Although most DR experiments expose individuals to one food environment, some experiments switch between them, and show that organisms rapidly adjust their life history accordingly (Mair et al., 2003; McCracken et al., 2020; Sultanova et al., 2021). Selection on this rapid

plasticity is about maximising relative fitness within each time point, rather than the absolute fitness over an individual's whole life.

Model validation through simulations

Above we show analytically that selection on the timing of reproduction varies with population growth rate and verbally argue that a plastic life history strategy, like that we see in the DR response, is optimal given any level of environmental fluctuation. To validate this argument, we ran some simple individual based simulations, based on the model outlined above, where the environment directly affects the expected R_0.

We simulated three scenarios in which the environment fluctuated with varying levels of temporal autocorrelation: one with positive autocorrelation, so slow changes between environments and long periods in one environment; one stochastic; and one with negative temporal autocorrelation where the environment rapidly fluctuates (Figure S2). Environmental values were drawn from a log normal distribution with mean of 1 and standard deviation of 0.1 on the observed scale, and temporal autocorrelation was simulated with a first-order autoregressive function, with correlation 0.75, 0 and -0.75 on the latent scale for positive temporal autocorrelation, stochastic and negative temporal autocorrelation scenarios. In the Supplements, we also present three scenarios with a fixed environment as a validation of the basic model.

Within each scenario, we simulated four strains, three with a fixed strategy and one with a plastic strategy. As in the model above, the expected R_0 of all strains within an environment was the same. In the fixed strategy strains, we varied the generation time by holding survival between time-steps constant across environments and letting fecundity change. We simulated one 'fast' strain with a survival rate of 0.5, an 'intermediate' strain with a survival of $\frac{2}{3}$ and a 'slow' strain with a survival rate of 0.75, giving expected lifespans of 2, 3, and 4 time-steps and expected generation times of 1, 1.5 and 2 time-steps, respectively. These were chosen to be within a relatively small range that maybe feasible

within a population and are similar to the survival rates of many species studied in the wild (e.g. many small vertebrates). The plastic strain had the fast strategy in environments with expected $R_0 > 1$, intermediate strategy when the population was stationary, and slow strategy in the environments where expected $R_0 < 1$ and the population was declining.

An individual's survival and reproduction at each time point was sampled from Bernoulli and Poisson distributions, respectively, with rates determined by the combination of environment and strain. Within the simulations, individuals did not interact. 200 simulations were run per scenario, and each simulation was started with 1000 individuals from each strain and run for 100 time points. We recorded the number of individuals of each strain at each time point and measured the relative spread of each strain as the proportion of each strain at each time point. To assess how the frequency with which the plastic strain 'won', we also recorded the proportion of simulations in which the plastic strain had the highest population size in the 100th time point.

In support of our verbal argument, the plastic strain spread at the expense of those with a fixed strategy, regardless of the duration of the fluctuations (Figure 3; the plastic strategy was dominant in 94.5% of the simulations with positive autocorrelation, and 100% of simulations in the other scenarios). This further supports our argument that a DR-like plastic response is selected for whether individuals will return to food within their lifetime.

Caveats

For anyone studying plasticity, it will seem unsurprising that plasticity is selected for in a fluctuating environment. Our purpose here is simply to demonstrate that the substantial plasticity commonly observed across many taxa is consistent with the plasticity that you would expect if resource availability acts as a cue for population growth rate. There are important caveats we would like to address in our model of plasticity.

First, we assume there is no cost of plasticity, which may be unrealistic. Whilst there is a considerable amount of research focussing on the costs of plasticity (DeWitt et al., 1998;

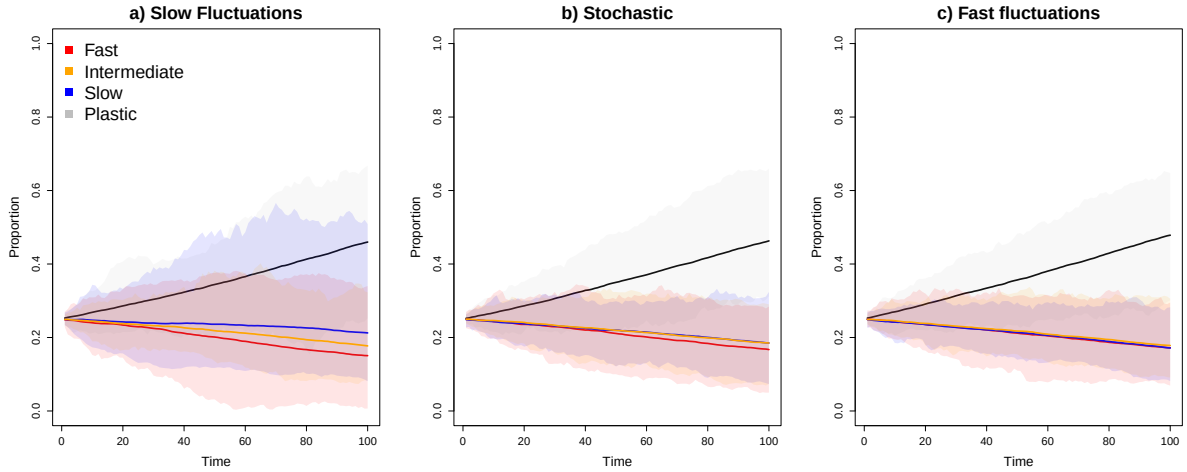

Figure 3: Spread of four different strains, three with fixed life history strategies and one with a plastic strategy, adopting a fast strategy in a good environment and a slow strategy in a bad environment. Panels show different speeds of environmental fluctuations, with a) positive temporal autocorrelation, b) stochastic variation and c) positive temporal autocorrelation.

Auld et al., 2009; Little & Seebacher, 2025), here we simply seek to demonstrate that the plasticity we commonly observe would be expected to be selected for, and so reasonably could represent adaptive plasticity. Given that this strategy appears to be highly conserved (Nakagawa et al., 2012; Ivimey-Cook et al., 2025; Moatt et al., 2016), we can then assume that it is not too costly to have widely evolved. The costs of this plasticity would also be hard to measure empirically, given that this plasticity is so widespread. This would have to be done by looking for variation in this plasticity within populations, and then assessing the costs and benefits of this variation.

Second, we assume that resource availability is a reliable cue for population growth rate and that individuals respond immediately to this cue. Again, there is a substantial amount of literature covering these points (e.g. DeWitt et al., 1998). In the absence of reliable cues, plasticity is unlikely to evolve. Equally if the plastic response is slower than the environmental fluctuations, then the plasticity will provide limited benefits. Again, given that this plasticity has evolved and is conserved across taxa, this suggests that the cues are reliable, and that the plastic response is faster than typical environmental fluctuations. Here, the cue is an environment that the individual directly experiences (i.e. food availability or quality) and that relates to the growth rate of the population at that time, rather than some

proxy (e.g. whether the temperature predicts the food availability in the future). The reliability of this cue at signalling population level changes in growth rate then relates to how much variation in food availability there is between individuals in a population within a time point, versus variation among time points. If the variation within time points is higher than among time points, then how much food an individual has is not a good cue of what the population is doing. In a competitive environment, it is likely that all individuals end up with lots of resources when resources are abundant, whereas in a low resource environment, it would depend on the form of competition; asymmetric competition would result in few individual having lots of resource and many having little, and symmetric competition would result in all individuals have little. Under both scenarios, the average individual would experience less food in a bad environment, suggesting that for many individuals the resource environment that they experience would be broadly representative of the overall environment, and so represents a reliable cue for the population growth rate. In terms of the speed of response, studies that have altered the food environment during an experiment have shown that the individuals can rapidly alter their response (Mair et al., 2003; McCracken et al., 2020; Sultanova et al., 2021).

Relatedly, our model assumes that individuals suddenly switch between strategies at a set point, knowing exactly when the population moves from growing to declining. We make this assumption partly for simplicity, and partly as many DR studies present this response as a dichotomy. A more realistic model would allow a more continuous change in strategy across an environmental gradient. Although the optimal strategy would be a sudden switch, mistiming this switch (i.e. adopting a slow strategy when the population was still growing) would be strongly selected against. Food availability is unlikely to be so good a cue for population growth rate that such a dramatic shift in strategy would be selected for. While DR studies typically dichotomise the environment, studies of nutritional geometry consider continuous changes over a much broader range of diets, providing a much clearer picture of how plasticity in the timing of breeding manifests as diet quality changes (Raubenheimer et al., 2023).

Finally, we assume a very simplistic life history, with no age-structure and no senescence. This is unrealistic. We chose this model deliberately to demonstrate the importance of well-known concepts in demography for the evolution of adaptive plasticity, in as accessible way as possible. One recent study with a more complex model from a different perspective that considers age structure, comes to a complimentary conclusion (Irish et al., 2025). Due to the lack of age structure, our model also does not consider demographic momentum, in which a population can continue along a previous growth trajectory even after a change in vital rates, temporarily decoupling expected R_0 and instantaneous growth rate (Koons et al., 2006). This phenomenon depends heavily on which vital rates are affected and predominantly affects populations with slow-life histories (Koons et al., 2006). For example, a species with late reproduction might be expected to continue to decline after an event that caused increased mortality of mature adults, until the younger individuals mature. However, the plasticity we describe may mitigate this effect, as it would lead to individuals shifting to earlier reproduction.

Adaptive life history plasticity in the wild

We have presented a simple model that explains why there is a selective advantage to the life history plasticity seen in the commonly observed DR response, and so why this response likely represents adaptive plasticity in a fluctuating environment. It has previously been argued that this response may be a laboratory artifact (Adler & Bonduriansky, 2014). We therefore explore whether and where we might see evidence for such a response in wild populations. We first consider a widespread phenomenon, density regulation, where we argue that this plasticity should be common, and so may provide an explanation for its evolution and conservation across taxa. Second, we consider extreme reactions in response to environmental hardship, and how these fit with our model. Finally, we consider human altered environments; anthropogenic changes often impact food availability and quality, providing natural experiments from which would expect predictable plastic responses.

Density Regulation

Density regulation is common in wild populations (Brook & Bradshaw, 2006). It is often assumed to work via resource availability; when populations are above the carrying capacity, resources are limited (whether in terms of absolute amounts, or the availability of more high-quality resources), leading to a declining population growth rate, and vice versa. Density regulated populations therefore present a system in which rapid, short-term fluctuations in resource availability (i.e. negative temporal autocorrelation) predict rapid fluctuations in population growth. Our model suggests that under this regime, there is strong, persistent, directional selection for life history plasticity. In other words, there is density-dependent selection on generation time. Given how widespread density regulation is thought to be, it seems plausible that the ubiquity of this force may have facilitated the widespread evolution and conservation of this life history plasticity.

Theoretical work on density dependent selection on generation time is best exemplified by the literature on R and K selection, where R is a fast life history strategy selected for at low density, and K is a slow strategy selected for at carrying capacity (MacArthur, 1962; MacArthur & Wilson, 1967; Pianka, 1970; Reznick et al., 2002). Assuming fixed strategies and no phenotypic plasticity, this density dependent selection has been suggested as a mechanism that can explain the maintenance of genetic variation in life history within populations (Sæther et al., 2016; Araya-Ajoy et al., 2021; Sæther et al., 2021). As we show here, this density dependent selection would also select for adaptive life history plasticity, which thus far has attracted much less attention.

Although the DR response is rarely considered in terms of density regulation, recent work has shown that the same physiological mechanisms that are associated with the DR response (e.g., IGF-1) also respond to food availability (Dantzer & Swanson, 2012; Lodjak & Verhulst, 2020) and density (Ravindran et al., 2026) in the wild .

Responding to harsh environments

In the extreme case, our model predicts that when the population is declining, organisms should optimally have infinitely high survival and infinitely low reproduction. There are many example in which we see such extreme life history plasticity in response to food availability in the wild, where low food availability causes organisms undergo complete cessation of reproduction and even take measures to increase survival by shutting themselves down.

Perhaps the most obvious example of this is seasonal breeding. In temperate regions, there is a predictable food pulse during spring and summer, in which animals reproduce. In such systems, we tend to discretise time into years, and focus on the breeding season. In the context of our model, we may then consider this plasticity working across breeding seasons with organisms changing their life history on that scale. However, we can also consider this on a finer scale, viewing these populations in continuous time. In this context, we can see considerable life history plasticity across a year, with organisms ceasing reproduction during periods of low food availability and then initiating reproduction during periods of high food availability. At the extreme, some species hibernate, increasing survival probability by downregulating physiological processes and hiding away. Supplementary feeding has also been shown to advance the onset of breeding in these systems (Ruffino et al., 2014), suggesting that food availability acts as a trigger for reproduction.

Outside of seasonal breeding, we see many similar examples on shorter timescales. Opportunistic breeders present a clear example of this. Zebra finches, for example, breed following rainfall, which signals an increase in food availability (Zann et al., 1995). Similarly, small mammals go into torpor during periods of low food availability (Vuarin & Henry, 2014; Vuarin et al., 2015; McAllan & Geiser, 2014), a shorter-term version of hibernation and insects go into diapause when food sources are limited (Hahn & Denlinger, 2011). If exposed to dietary restriction in a juvenile stage, *Caenorhabditis elegans* goes into a developmentally arrested dauer state (Golden & Riddle, 1984). A similar response is even seen in microbes, which appear to enter a dormant state when density is high and resources

are limited (Bergkessel et al., 2016; Jõers & Tenson, 2016; Pelusi et al., 2020). Regan et al. (2020) noted that the mechanisms underlying processes such as torpor and diapause are the same as those underlying the dietary restriction response, suggesting a common physiological underpinning, and so shared evolutionary history.

There may be other factors impacting these responses, such as physiological constraints and selection against putting offspring into bad environmental conditions they are unlikely to survive (although this is also indicative of population decline). From a purely demographic perspective, however, given that food availability likely linked to population growth, these responses can also be seen in the same context of adaptive plasticity.

Human altered environments

Anthropogenic change in the last few decades has been rapid and dramatic, with large impacts upon wild populations (Hendry et al., 2008). Studying changes in these human altered environments provides large scale, often replicated natural experiments. Two clear examples of this are harvesting and urbanisation.

Generally, harvesting results in a massive reduction in population density, moving the population below its carrying capacity, resulting it in a state where it is always growing. Although this is a direct effect on population growth rate, we could well imagine that this is mediated by harvesting generating high resource availability by reducing density. Under our model, we would expect a plastic shift to a fast life history, with high and early reproduction. Interestingly, this is exactly what has been repeatedly observed in fisheries; a life history shift toward earlier maturation and higher fecundity (Darimont et al., 2009; Audzijonyte et al., 2013). Previous work has interpreted this as evolutionary change in response to size (and so age)-specific harvesting, inducing selection on faster life histories (Heino & Godø, 2002; Heino & Dieckmann, 2008; Heino et al., 2015). However, there is very little direct evidence of evolutionary change, as this is extremely difficult to demonstrate in such systems (Heino & Dieckmann, 2008). Indeed, Audzijonyte et al. (2013) showed that the phenotypic

response in fisheries is far larger than might be expected via adaptive evolution alone, suggesting that at least part of this response may be due to plasticity, and notably is *consistent* with adaptive plastic response we describe in our model. Harvesting is a form of extreme predation, and it is worth noting that natural predation is also well known to impact life history in the same way (Reznick et al., 1990, 2001).

Urbanisation represents a systematic shift away from 'natural' habitats. For many species (e.g. non-commensal and feral species), this likely represents a worse environment, with lower availability and quality of food. Under these environments, we might therefore expect a shift towards slower life histories. Interestingly, there is repeated evidence of lower reproduction in urban versus rural areas (Chamberlain et al., 2009; Sepp et al., 2018; Capilla-Lasheras et al., 2022), and some evidence that survival might additionally increase (Sepp et al., 2018).

Measuring fitness in a fluctuating environment

Fitness is a key measure in evolutionary biology that allows us to measure selection and so predict evolutionary change. Although there are many ways of measuring fitness, all represent a combination of survival and reproduction and so plasticity in these components may affect how we should measure fitness. In individual-based studies of wild populations, we typically measure the fitness of an individual as the culmination of survival and reproduction over their lifetime, with metrics such as lifetime reproductive success (LRS; total offspring produced across an individual's life) representing the gold standard. However, we show that strategies can have the same expected LRS (described as R_0 in our model), but differentially spread through the population depending on their life histories. Although this has already been acknowledged (McGraw & Caswell, 1996; Brommer et al., 2002; Moorad, 2014), metrics that aim to deal with this are still predominantly lifetime metrics and so implicitly assume that environment is constant across life. However, resources often fluctuate in natural environments, and so the plasticity of individuals in response to this is

also important.

Measures of fitness relate to spread through a population and so fitness is often considered as relative to the others around you. Lifetime metrics pose this as the relative fitness against all others in the population across time points. Our simplistic model here suggests that, in a fluctuating environment, it is more important to consider your fitness relative to those around you at a particular time point. Relative fitness may vary across environments within a lifetime, meaning that any metric calculated across a lifetime would miss this subtlety. If there is variation in plasticity among individuals, some individuals will match their environment better than others, and therefore spread more through the population. For example, two individuals may have the same LRS (i.e., same absolute fitness across their lives), but if one individual better matched their reproduction to the environmental fluctuations (i.e., high relative fitness within environments), then they will likely contribute more to the future population. Indeed, in our simulations, individuals from all strains had the same expected LRS, but with environmental fluctuations the plastic strains always spread. Whilst the presence of plasticity itself seems conserved, the degree of plasticity may vary within a population, meaning that lifetime metrics may fail to fully capture fitness variation. Indeed, variation in the dietary restriction response has been observed across strains in mice and drosophila (Liao et al., 2010; Rikke et al., 2010; McCracken et al., 2020). In this case, lifetime metrics will not work as intended within a fluctuating environment.

Our model therefore illustrates the problem of using lifetime metrics of fitness in a fluctuating environment. The clearest alternative is to use time-step metrics such as individual contribution to the next time-step, measured as fecundity/2 + survival in a diploid population (Sæther & Engen, 2015), which can be relativised within a time-step.

Discussion

We present a simple demographic model that shows how life history plasticity, such as that seen in the dietary restriction response, is selected for in a fluctuating environment, if the

population growth rate varies between declining and growing. We show that the environmental fluctuations can be much longer than an individual's lifespan, meaning that the assumption of returning to resources within a lifetime as set out in previous models (Holliday, 1989; Shanley & Kirkwood, 2000) is not necessary to explain this response as adaptive plasticity. We further argue that this response may have been selected for by the common occurrence of density regulation, and that there are many similar responses to dietary restriction in natural conditions, addressing previous criticisms of the DR response representing adaptive plasticity (Adler & Bonduriansky, 2014). Finally, the widespread occurrence of this plasticity in varying environments common in natural populations suggests that we should reconsider how to measure fitness in the wild.

Previous studies have suggested that the dietary restriction response may be sex-specific(Nakagawa et al., 2012; Moatt et al., 2016), although this may be driven by invertebrates (Ivimey-Cook et al., 2025). Whilst we consider an asexual population here, a simple two-sex extension of our model may help explain this; if we assume female demographic dominance (in which we assume that all eggs are fertilised by males), there may be no particular advantage to males altering their survival, as males are not directly impacting upon population growth. In cases where females completely cease reproduction, however, there are no longer any reproductive opportunities and so this may favour male plasticity.

It has also been suggested that the survival response to dietary restriction may be weaker in longer lived species (Demetrius, 2004; Deere et al., 2023, although note that this has not been quantitatively assessed). This may link to the ideas of demographic buffering and lability, where some vital rates may vary to track the environment to increase growth rate (lability), whilst selection may act on those with the largest impact upon growth rate (known as sensitivity) to remain constant (buffering) (Koons et al., 2009). The key point of our model is to demonstrate that it is adaptive to relatively shift contribution to reproduction and survival depending on the population growth rate. Although it would be

optimal to shift from semelparous to dormant depending on the environment, there is a defined range for reproduction and survival within a species, and so clear physiological constraints for a given species in how plastic they can be. Together, this may suggest that species may shift their generation time through different changes in reproduction or survival, dependent on their overall life history. As species with faster overall life history generally have higher sensitivity in reproduction, we may expect them to alter survival more, whereas slower species have higher sensitivities in survival, and so may be more likely to alter reproduction, matching this prediction. More empirical and theoretical work would have to be done to explore this further. Importantly, it is not necessary that we explicitly see survival increase and reproduction decrease with an change in the environment, rather that there is a shift in the relative rates, within an impact on generation time.

To date, the focus in life history evolution has been on fixed strategies, and how varying environmental conditions may maintain life history variation among individuals, for example, research on R and K selection. Interestingly, our models suggest an additional reason why slow life histories may be selected for in stationary populations within a fluctuating environment (for example, fluctuating around a carrying capacity). Classic work in demography shows that in the long term, growth rate is sensitive to variation (Tuljapurkar, 1982), with more variation leading to a reduction in the long term, stochastic growth rate. As can be seen in Figure 2, slower life histories have lower variation in growth rate across environments, and so under a fluctuating environment with a stationary population growth rate on average, slower life-histories might be more likely to spread, as their stochastic growth rate is higher. Put another way, fast life histories are much more likely to crash in bad years than slow ones. Although this provides a similar result to the theory on K-selection (slow strategies would be selected for at carrying capacity), as far as we are aware, this argument has not been made previously. As we noted earlier, there has been a general lack of work on how a fluctuating environment may also select for life history plasticity. Interestingly, there is a conspicuous lack of evidence for a negative relationship between survival and reproduction among individuals (Winder et al., 2025), including at the

genetic level (Chang et al., 2024). This suggests that rather than maintaining variation in fixed strategies, this selection may have resulted in plasticity. Empirical evidence for this is currently scarce, and so this would provide a fruitful avenue for further research.

Whilst we focus on food availability, our model would hold for any environment that was closely matched to population growth rate. Indeed, Regan et al. (2020) highlighted that the mechanisms underlying the DR response are often the same as those for many other environmental cues. Obvious other environments include temperature and parasitism, which are both known to affect life history (Nylin & Gotthard, 1998; Snook et al., 2026; Agnew et al., 2000). Temperature is a more complex environment with a strongly non-linear and asymmetric relationship between temperature and fitness, with clear thermal optima. Whilst many studies focus on either survival or reproduction, our model demonstrates the utility of considering the combined plasticity of these two vital rates, and how they vary in response to environmental condition that may predict population growth.

Dietary restriction experiments often compare two environments, *ad libitum* food versus some form of restriction. However, as discussed above, we do not realistically expect the response to food availability to be dichotomous. Indeed, the degree of dietary restriction has been linked to size of the plastic response in reproduction (Moatt et al., 2016). Moreover, food availability in the wild is likely a much more complex picture than simply presence or absence of food, but also about the relative abundance of high-quality food. Nutritional geometry provides a more complex framework that allows much more detailed exploration of this plasticity. Taking a nutritional geometry approach would allow us to map population growth rate onto a nutritional landscape, and compare this with changes in survival and reproduction across this same landscape. By incorporating information about the projected growth rate, this will help us to understand the dynamics of survival and reproduction across this landscape, and how individuals may be optimising their strategy according to the available resources. This would provide much more fine-scale picture about life history plasticity, and so allow further refinement of this model.

Data and Code availability

Code for this study is available at

https://github.com/joelpick/adaptive_plasticity

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Author Contributions

Conceptualization: JLP Writing: Original Draft: JLP Writing: Editing: JLP, YAA, HF, EIC, SR, CAW Formal analysis: JLP Visualisation: JLP Supervision: CAW

Conflicts of Interest

We declare no conflicts of interest.

References

Nakagawa, S., Lagisz, M., Hector, K. L. & Spencer, H. G. 2012 Comparative and meta-analytic insights into life extension via dietary restriction. Aging Cell **11**, 401–409. (doi:10.1111/j.1474-9726.2012.00798.x)

Ivimey-Cook, E. R., Sultanova, Z. & Maklakov, A. A. 2025 Rapamycin, Not Metformin,

Mirrors Dietary Restriction-Driven Lifespan Extension in Vertebrates: A Meta-Analysis. Aging Cell **n/a**, e70131. (doi:10.1111/acel.70131)

Moatt, J. P., Nakagawa, S., Lagisz, M. & Walling, C. A. 2016 The effect of dietary restriction on reproduction: a meta-analytic perspective. BMC Evolutionary Biology **16**, 199. (doi:10.1186/s12862-016-0768-z)

Moatt, J. P., Savola, E., Regan, J. C., Nussey, D. H. & Walling, C. A. 2020 Lifespan Extension Via Dietary Restriction: Time to Reconsider the Evolutionary Mechanisms? BioEssays **42**, 1900241. (doi:10.1002/bies.201900241)

Ruffino, L., Salo, P., Koivisto, E., Banks, P. B. & Korpimäki, E. 2014 Reproductive responses of birds to experimental food supplementation: a meta-analysis. Frontiers in Zoology **11**, 80. (doi:10.1186/s12983-014-0080-y)

Holliday, R. 1989 Food, reproduction and L'ongevity: Is the extended lifespan of calorie-restricted animals an evolutionary adaptation? BioEssays **10**, 125–127. (doi:10.1002/bies.950100408)

Shanley, D. P. & Kirkwood, T. B. L. 2000 CALORIE RESTRICTION AND AGING: A LIFE-HISTORY ANALYSIS. Evolution **54**, 740–750. (doi:10.1111/j.0014-3820.2000.tb00076.x)

Adler, M. I. & Bonduriansky, R. 2014 Why do the well-fed appear to die young? BioEssays **36**, 439–450. (doi:10.1002/bies.201300165)

Charlesworth, B. 1994 *Evolution in age-structured populations*. Cambridge studies in mathematical biology, Cambridge: Cambridge University Press, 2 edn.

de Vries, C., Galipaud, M. & Kokko, H. 2023 Extrinsic mortality and senescence: a guide for the perplexed. Peer Community Journal **3**. (doi:10.24072/pcjournal.253)

614 Mair, W., Goymer, P., Pletcher, S. D. & Partridge, L. 2003 Demography of Dietary
615 Restriction and Death in Drosophila. Science **301**, 1731–1733.
616 (doi:10.1126/science.1086016)

617 McCracken, A. W., Adams, G., Hartshorne, L., Tatar, M. & Simons, M. J. P. 2020 The
618 hidden costs of dietary restriction: Implications for its evolutionary and mechanistic
619 origins. Science Advances **6**, eaay3047. (doi:10.1126/sciadv.aay3047)

620 Sultanova, Z., Ivimey-Cook, E. R., Chapman, T. & Maklakov, A. A. 2021 Fitness benefits of
621 dietary restriction. Proceedings of the Royal Society B: Biological Sciences **288**,
622 20211787. (doi:10.1098/rspb.2021.1787)

623 DeWitt, T. J., Sih, A. & Wilson, D. S. 1998 Costs and limits of phenotypic plasticity.
624 Trends in Ecology & Evolution **13**, 77–81. (doi:10.1016/S0169-5347(97)01274-3)

625 Auld, J. R., Agrawal, A. A. & Relyea, R. A. 2009 Re-evaluating the costs and limits of
626 adaptive phenotypic plasticity. Proceedings of the Royal Society B: Biological Sciences
627 **277**, 503–511. (doi:10.1098/rspb.2009.1355)

628 Little, A. G. & Seebacher, F. 2025 A review of the empirical evidence for costs of plasticity
629 in ectothermic animals. Journal of Experimental Biology **228**, jeb249226.
630 (doi:10.1242/jeb.249226)

631 Raubenheimer, D., Hou, R., Dong, Y., Ren, C. & Cui, Z. 2023 Towards an integrated
632 understanding of dietary phenotypes. Philosophical Transactions of the Royal Society B:
633 Biological Sciences **378**, 20220545. (doi:10.1098/rstb.2022.0545)

634 Irish, S. D., Kimberley, A., Immler, S., Moorad, J. & Maklakov, A. A. 2025 Mutation
635 accumulation underpins evolution of lifespan extension by dietary restriction.
636 (doi:10.1101/2025.06.18.660314)

637 Koons, D. N., Grand, J. B. & Arnold, J. M. 2006 Population momentum across vertebrate
638 life histories. Ecological Modelling **197**, 418–430. (doi:10.1016/j.ecolmodel.2006.03.034)

Brook, B. W. & Bradshaw, C. J. A. 2006 Strength of Evidence for Density Dependence in Abundance Time Series of 1198 Species. Ecology **87**, 1445–1451. (doi:10.1890/0012-9658(2006)87[1445:SOEFDD]2.0.CO;2)

MacArthur, R. H. 1962 Some generalized theorems of natural selection. Proceedings of the National Academy of Sciences **48**, 1893–1897. (doi:10.1073/pnas.48.11.1893)

MacArthur, R. H. & Wilson, E. O. 1967 *The theory of island biogeography*, vol. 1. Princeton university press

Pianka, E. R. 1970 On r- and K-Selection. The American Naturalist **104**, 592–597. (doi:10.1086/282697)

Reznick, D., Bryant, M. J. & Bashey, F. 2002 r- AND K-SELECTION REVISITED: THE ROLE OF POPULATION REGULATION IN LIFE-HISTORY EVOLUTION. Ecology **83**, 1509–1520. (doi:10.1890/0012-9658(2002)083[1509:RAKSRT]2.0.CO;2)

Sæther, B.-E., Visser, M. E., Grøtan, V. & Engen, S. 2016 Evidence for .- and .-selection in a wild bird population: a reciprocal link between ecology and evolution. Proceedings of the Royal Society B: Biological Sciences **283**, 20152411. (doi:10.1098/rspb.2015.2411)

Araya-Ajoy, Y. G., Niskanen, A. K., Froy, H., Ranke, P. S., Kvalnes, T., Rønning, B., Le Pepke, M., Jensen, H., Ringsby, T. H., Sæther, B.-E. et al. 2021 Variation in generation time reveals density regulation as an important driver of pace of life in a bird metapopulation. Ecology Letters **24**, 2077–2087. (doi:10.1111/ele.13835)

Sæther, B.-E., Engen, S., Gustafsson, L., Grøtan, V. & Vriend, S. J. G. 2021 Density-Dependent Adaptive Topography in a Small Passerine Bird, the Collared Flycatcher. The American Naturalist **197**, 93–110. (doi:10.1086/711752)

Dantzer, B. & Swanson, E. M. 2012 Mediation of vertebrate life histories via insulin-like growth factor-1. Biological Reviews **87**, 414–429. (doi:10.1111/j.1469-185X.2011.00204.x)

Lodjak, J. & Verhulst, S. 2020 Insulin-like growth factor 1 of wild vertebrates in a life-history context. Molecular and Cellular Endocrinology **518**, 110978. (doi:10.1016/j.mce.2020.110978)

Ravindran, S., Corripio-Miyar, Y., Pick, J. L., Bal, X., Pilkington, J. G., Pemberton, J. M., Nussey, D. H. & Froy, H. 2026 Plasma insulin-like growth-factor 1 (IGF-1) concentrations predict early life-history traits in a wild mammal. Functional Ecology **n/a**. (doi:10.1111/1365-2435.70339)

Zann, R. A., Morton, S. R., Jones, K. R. & Burley, N. T. 1995 The Timing of Breeding by Zebra Finches in Relation to Rainfall in Central Australia. Emu **95**, 208–222. (doi:10.1071/MU9950208). _eprint: https://doi.org/10.1071/MU9950208

Vuarin, P. & Henry, P.-Y. 2014 Field evidence for a proximate role of food shortage in the regulation of hibernation and daily torpor: a review. Journal of Comparative Physiology B **184**, 683–697. (doi:10.1007/s00360-014-0833-0)

Vuarin, P., Dammhahn, M., Kappeler, P. M. & Henry, P.-Y. 2015 When to initiate torpor use? Food availability times the transition to winter phenotype in a tropical heterotherm. Oecologia **179**, 43–53. (doi:10.1007/s00442-015-3328-0)

McAllan, B. M. & Geiser, F. 2014 Torpor during Reproduction in Mammals and Birds: Dealing with an Energetic Conundrum. Integrative and Comparative Biology **54**, 516–532. (doi:10.1093/icb/icu093)

Hahn, D. A. & Denlinger, D. L. 2011 Energetics of Insect Diapause. Annual Review of Entomology **56**, 103–121. (doi:10.1146/annurev-ento-112408-085436)

Golden, J. W. & Riddle, D. L. 1984 The *Caenorhabditis elegans* dauer larva: Developmental effects of pheromone, food, and temperature. Developmental Biology **102**, 368–378. (doi:10.1016/0012-1606(84)90201-X)

688 Bergkessel, M., Basta, D. W. & Newman, D. K. 2016 The physiology of growth arrest:
689 uniting molecular and environmental microbiology. Nature Reviews Microbiology **14**,
690 549–562. (doi:10.1038/nrmicro.2016.107)

691 Jõers, A. & Tenson, T. 2016 Growth resumption from stationary phase reveals memory in
692 Escherichia coli cultures. Scientific Reports **6**, 24055. (doi:10.1038/srep24055)

693 Pelusi, A., Margiotta, F., Passarelli, A., Ferrante, M. I., Ribera d'Alcalà, M. & Montresor, M.
694 2020 Density-dependent mechanisms regulate spore formation in the diatom Chaetoceros
695 socialis. Limnology and Oceanography Letters **5**, 371–378. (doi:10.1002/lol2.10159)

696 Regan, J. C., Froy, H., Walling, C. A., Moatt, J. P. & Nussey, D. H. 2020 Dietary restriction
697 and insulin-like signalling pathways as adaptive plasticity: A synthesis and re-evaluation.
698 Functional Ecology **34**, 107–128. (doi:10.1111/1365-2435.13418)

699 Hendry, A. P., Farrugia, T. J. & Kinnison, M. T. 2008 Human influences on rates of
700 phenotypic change in wild animal populations. Molecular Ecology **17**, 20–29.
701 (doi:10.1111/j.1365-294X.2007.03428.x)

702 Darimont, C. T., Carlson, S. M., Kinnison, M. T., Paquet, P. C., Reimchen, T. E. &
703 Wilmers, C. C. 2009 Human predators outpace other agents of trait change in the wild.
704 Proceedings of the National Academy of Sciences **106**, 952–954.
705 (doi:10.1073/pnas.0809235106)

706 Audzijonyte, A., Kuparinen, A. & Fulton, E. A. 2013 How fast is fisheries-induced evolution?
707 Quantitative analysis of modelling and empirical studies. Evolutionary Applications **6**,
708 585–595. (doi:10.1111/eva.12044)

709 Heino, M. & Godø, O. R. 2002 Fisheries-Induced Selection Pressures in the Context of
710 Sustainable Fisheries. Bulletin of Marine Science **70**, 639–656

711 Heino, M. & Dieckmann, U. 2008 Detecting Fisheries-Induced Life-History Evolution: An
712 Overview of the Reaction-Norm Approach. Bulletin of Marine Science **83**, 69–93

713 Heino, M., Pauli, B. D. & Dieckmann, U. 2015 Fisheries-Induced Evolution. Annual Review
714 of Ecology, Evolution, and Systematics **46**, 461–480.
715 (doi:10.1146/annurev-ecolsys-112414-054339)

716 Reznick, D. A., Bryga, H. & Endler, J. A. 1990 Experimentally induced life-history evolution
717 in a natural population. Nature **346**, 357–359. (doi:10.1038/346357a0)

718 Reznick, D., Butler, I. M. J., Rodd, H. & null, n. 2001 Life-History Evolution in Guppies.
719 VII. The Comparative Ecology of High- and Low-Predation Environments. The American
720 Naturalist **157**, 126–140. (doi:10.1086/318627)

721 Chamberlain, D. E., Cannon, A. R., Toms, M. P., Leech, D. I., Hatchwell, B. J. & Gaston,
722 K. J. 2009 Avian productivity in urban landscapes: a review and meta-analysis. Ibis **151**,
723 1–18. (doi:10.1111/j.1474-919X.2008.00899.x)

724 Sepp, T., McGraw, K. J., Kaasik, A. & Giraudeau, M. 2018 A review of urban impacts on
725 avian life-history evolution: Does city living lead to slower pace of life? Global Change
726 Biology **24**, 1452–1469. (doi:10.1111/gcb.13969)

727 Capilla-Lasheras, P., Thompson, M. J., Sánchez-Tójar, A., Haddou, Y., Branston, C. J.,
728 Réale, D., Charmantier, A. & Dominoni, D. M. 2022 A global meta-analysis reveals higher
729 variation in breeding phenology in urban birds than in their non-urban neighbours.
730 Ecology Letters **25**, 2552–2570. (doi:10.1111/ele.14099)

731 McGraw, J. B. & Caswell, H. 1996 Estimation of Individual Fitness from Life-History Data.
732 The American Naturalist **147**, 47–64. (doi:10.1086/285839)

733 Brommer, J. E., Merilä, J. & Kokko, H. 2002 Reproductive timing and individual fitness:
734 Reproductive timing and fitness. Ecology Letters **5**, 802–810.
735 (doi:10.1046/j.1461-0248.2002.00369.x)

736 Moorad, J. A. 2014 Individual fitness and phenotypic selection in age-structured populations
737 with constant growth rates. Ecology **95**, 1087–1095. (doi:10.1890/13-0778.1)

738 Liao, C.-Y., Rikke, B. A., Johnson, T. E., Diaz, V. & Nelson, J. F. 2010 Genetic variation in
739 the murine lifespan response to dietary restriction: from life extension to life shortening.
740 Aging Cell **9**, 92–95. (doi:10.1111/j.1474-9726.2009.00533.x)

741 Rikke, B. A., Liao, C.-Y., McQueen, M. B., Nelson, J. F. & Johnson, T. E. 2010 Genetic
742 dissection of dietary restriction in mice supports the metabolic efficiency model of life
743 extension. Experimental Gerontology **45**, 691–701. (doi:10.1016/j.exger.2010.04.008)

744 Sæther, B.-E. & Engen, S. 2015 The concept of fitness in fluctuating environments. Trends
745 in Ecology & Evolution **30**, 273–281. (doi:10.1016/j.tree.2015.03.007)

746 Demetrius, L. 2004 Caloric Restriction, Metabolic Rate, and Entropy. The Journals of
747 Gerontology: Series A **59**, B902–B915. (doi:10.1093/gerona/59.9.B902)

748 Deere, J. A., Xu, C., Adelmant, C., Aboobaker, A. & Salguero-Gómez, R. 2023 The Hunger
749 Games as the Key to Happily Ever After? The Journals of Gerontology: Series A **78**,
750 1116–1124. (doi:10.1093/gerona/glad100)

751 Koons, D. N., Pavard, S., Baudisch, A. & Jessica E. Metcalf, C. 2009 Is life-history buffering
752 or lability adaptive in stochastic environments? Oikos **118**, 972–980.
753 (doi:10.1111/j.1600-0706.2009.16399.x)

754 Tuljapurkar, S. D. 1982 Population dynamics in variable environments. III. Evolutionary
755 dynamics of r-selection. Theoretical Population Biology **21**, 141–165.
756 (doi:10.1016/0040-5809(82)90010-7)

757 Winder, L. A., Simons, M. J. & Burke, T. 2025 No evidence for a trade-off between
758 reproduction and survival in a meta-analysis across birds. eLife **12**, RP87018.
759 (doi:10.7554/eLife.87018)

760 Chang, C.-c., Moiron, M., Sánchez-Tójar, A., Niemelä, P. T. & Laskowski, K. L. 2024 What
761 is the meta-analytic evidence for life-history trade-offs at the genetic level? Ecology
762 Letters **27**, e14354. (doi:10.1111/ele.14354)

763 Nylin, S. & Gotthard, K. 1998 Plasticity in Life-History Traits. Annual Review of
764 Entomology **43**, 63–83. (doi:10.1146/annurev.ento.43.1.63)

765 Snook, R. R., Bretman, A., Dougherty, L. R. & Fricke, C. 2026 The consequences of rising
766 temperatures for animal fertility. Nature Reviews Biodiversity pp. 1–17.
767 (doi:10.1038/s44358-026-00142-4)

768 Agnew, P., C. Koella, J. & Michalakis, Y. 2000 Host life history responses to parasitism.
769 Microbes and Infection **2**, 891–896. (doi:10.1016/S1286-4579(00)00389-0)

Supplementary Material for:

A simple demographic explanation for the evolution of the dietary restriction response and its ecological relevance

Joel L. Pick, Yimen G. Araya-Ajoy, Hannah Froy, Edward R. Ivimey Cook, Sanjana Ravindran & Craig A. Walling

S1 Model with no reproduction-survival trade-off

We can have a model where all individuals have the same lifespan (here 10 time-steps), and only breed in one year. Strains vary systematically in the year in which they breed creating variation in generation time, as the generation time will be equal to age of reproduction of that strain. We don't consider a scenario in which individuals have the same survival rate with varying year of reproduction, as this induces selection on generation time even when the population is stationary, as those with a later generation time are more likely to have died before reaching the point of reproduction. This means that R_0 is not constant across strains.

Similar to the main model, R_0 is dependent on the the environment, and all strains have the same R_0 in a given environment. Here, we calculate the growth rate as λ, the first eigenvalue of a Leslie matrix, with all ones on the off-diagonal, and R_0 in the element of the top row of the matrix for the given year of reproduction.

As shown in Figure S1, this gives a qualitatively similar pattern to our main model, whereby fast strains do better when the population is growing and slow strains when the population is shrinking.

It is worth noting that post-reproductive lifespan does not impact λ, and so in this scenario, λ is the same whether the individuals in a strain live after reproducing, or do not. The later case, with strains varying in their year of reproduction and then dying, would also generate a negative reproduction-survival correlation.

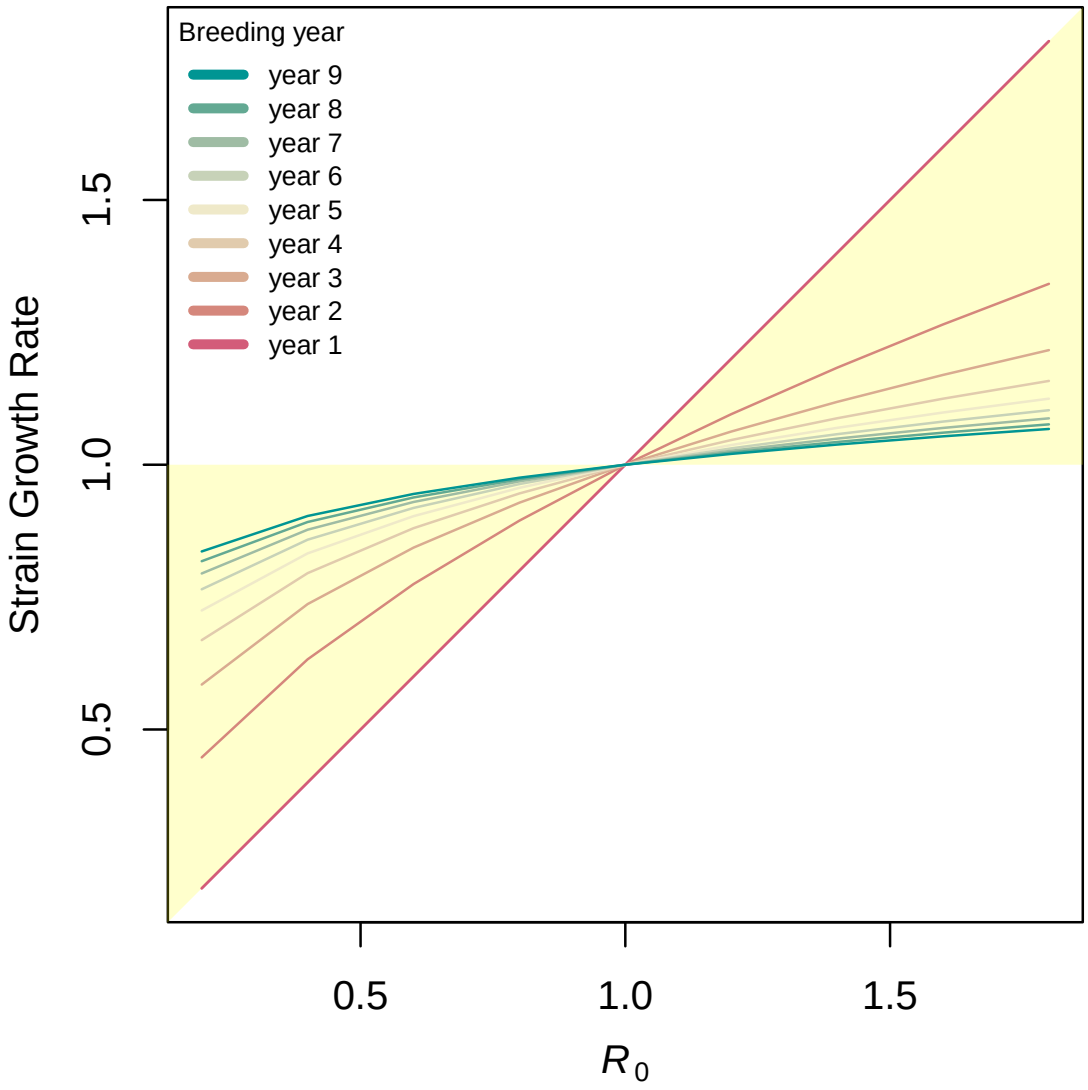

Figure S1: How variation in the timing of reproduction affects strain growth rate, using a different way of varying generation time. For a given total reproductive output (R_0; x axis), there is variation in the potential growth rate across strains (yellow area). This variation is caused by the effect of the timing of reproduction on growth rate, illustrated by different strains that vary in their generation time (coloured lines). Here, variation in generation time is generated by varying timepoint at which different strains reproduce. When the overall population is declining, for the same total reproduction, strains with a slower life history have a relatively faster growth rate. When the overall population is growing, for the same total reproduction, strains with a faster life history have a relatively faster growth rate.

S2 Temporal autocorrelation

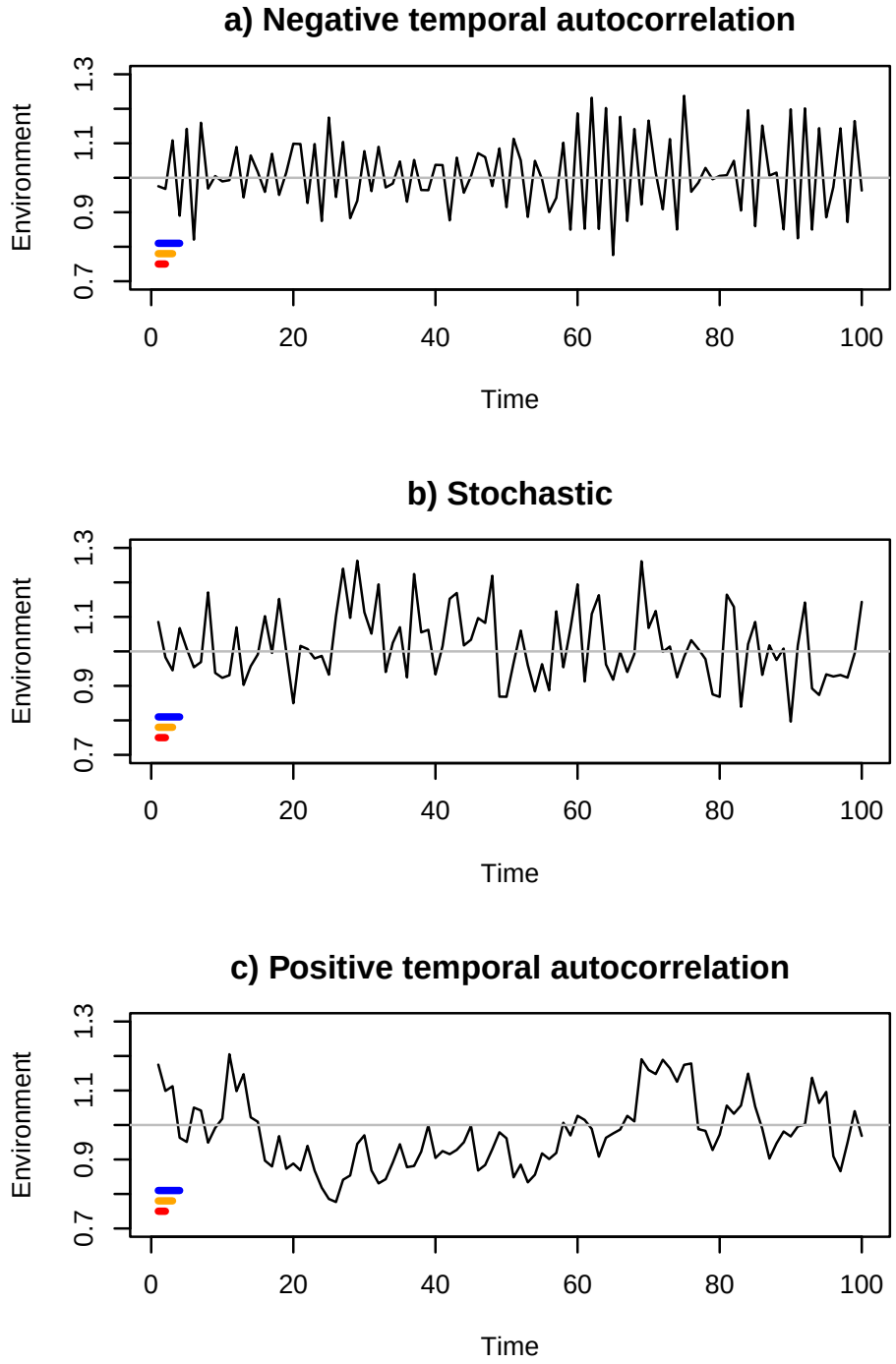

Figure S2: Example of three scenarios with fluctuating environments; a) negative temporal autocorrelation leading to rapid fluctuations, b) stochastic fluctuations and c) negative temporal autocorrelation leading to very rapid fluctuations. Environments were simulated with the same parameters used in the simulations in the main text. The coloured lines show the expected lifespan of the three strains in the simulation; blue - slow, orange - intermediate, red - fast.

S3 Fixed environment simulations

in addition to the simulations in the main text, we simulated three scenarios with a fixed environment: one scenario with a good environment resulting in an expected R_0 of 1.1 and so a growing population, one scenario with an stationary environment resulting in an expected R_0 of one and so a stationary growth rate, and one scenario with a bad environment resulting in an expected R_0 of 0.9, and so a declining population. These fixed environment simulations were run to demonstrate that timing of reproduction is under selection when populations are not stationary.

As expected, both the strain that already possessed the optimal strategy for that specific environmental (e.g the fast strategy in the good environment) and the plastic strain, which adopted an optimal strategy, spread (Figure S3 a-c).

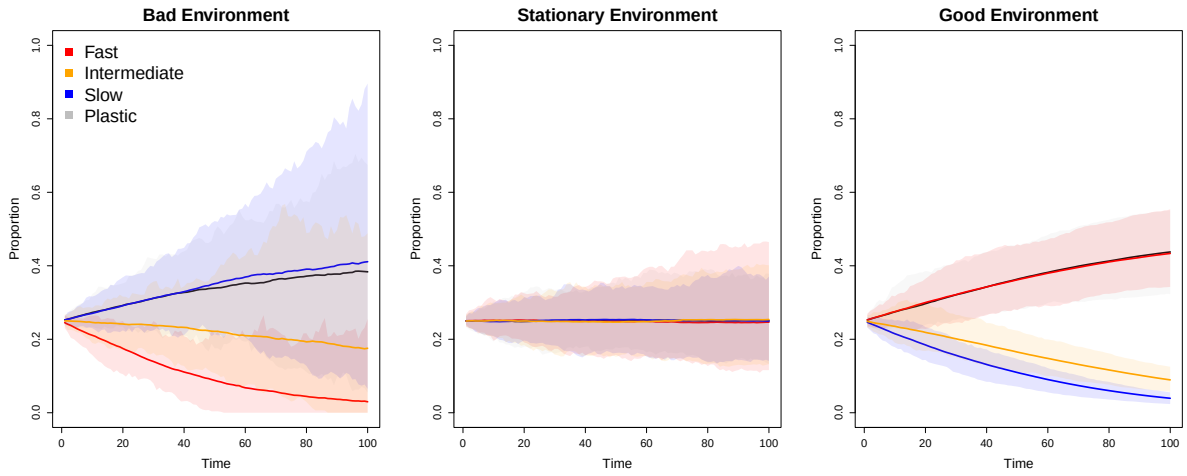

Figure S3: Spread of four different strains, three with fixed life history strategies and one with a plastic strategy, adopting a fast strategy in a good environment and a slow strategy in a bad environment. Panels show different fixed environments resulting in a) a growing population with an R_0 of 1, b) a stationary population with an R_0 of 1 and c) a declining population with an R_0 of 0.9.