

Biases in common methods used to quantify ageing and selective disappearance- a guide

Running title: Diagnosing selective disappearance

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Contributions

Conceptualisation: KS, MVDP, EIC, SB, IS

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Data and code accessibility statement

Entire simulation code and HTML output, along with all associated empirical data, and disappR, are available at Open science framework and DRYAD.

Their associated permanent links (not anonymized)

are: <https://doi.org/10.5061/dryad.dz08kpsbw>; and <https://osf.io/kevnrm/files/osfstorage/10.17605/OSF.IO/KEVNM>).

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Conflict of interest

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Abstract

1. Observed population-level patterns of age-dependent trait expression can fail to reflect the average, latent within-individual ageing trajectory due to selective disappearance- the biased removal or death of individuals with certain phenotypes.
2. Commonly used methods to assess, and account for, selective disappearance have been developed for relatively simple scenarios, such as those with linear trajectories of ageing, age-independent selective disappearance, and complete sampling of data. We currently lack a clear understanding of how well these methods recapitulate the true ageing pattern when selective disappearance itself is age-dependent, ageing trajectories are non-linear, or under realistic study designs and ecological systems.
3. Here, we use simulations to quantify and compare how well existing methods used in ecology (which we term: “additive covariate”, “mean-centring”, “decomposition”, “random slopes”, and “interaction” models) recapitulate the true ageing trajectory under a range of sampling designs and life-histories. We analytically derive the mechanisms mediating selective disappearance and explain why some methods used in ecology are biased. Using two empirical datasets, we also illustrate how the inferred ageing pattern depends on the specific statistical model used.
4. We find that when selective disappearance results from the removal of individuals with distinct ageing trajectories, the decomposition method is inaccurate in recovering the latent, average within-individual ageing trajectory of the entire cohort. Here, the additive covariate and mean-centring methods as typically used are also inaccurate and only account for age-independent selective disappearance (based on an individual’s average trait quality). Furthermore, mean-centring based models and the decomposition method perform poorly when sampling is incomplete, partly due to mean age being a poor proxy of latent lifespan, and the decomposition method relying

on insufficient consecutively sampled individuals, respectively. Importantly, random slopes alone only account for individual-differences in ageing, but do not remove age-dependent selective disappearance bias.

5. Generally, a model that fits age terms in interaction with age at last record recovers the true ageing trajectory better than other methods across simulated life-histories and data structures where either age-dependent selective disappearance is present, or among-individual heterogeneity in ageing trajectories is recoverable.
6. Despite several modelling assumptions, our specific recommendations for empiricists provide a general and comprehensive guide to diagnose longitudinal ageing data.

Keywords: aging, censoring, frailty, heterogeneity, individual quality, longitudinal vs cross-sectional, senescence, truncation bias

64 Introduction

65 *What is selective disappearance?*

66 Individuals differ in their resource acquisition (Cam and Monnat, 2000; Reznick et al, 2000)
67 and allocation (Roff and Fairbairn, 2007; van Noordwijk and de Jong, 1986), which can lead
68 to heterogeneity among individuals in the average expression of their life-history and
69 physiological traits, as well as in the ageing trajectories of these traits (Cam et al, 2002;
70 Maklakov and Chapman, 2019). Patterns of ageing can vary within- and across-individuals,
71 traits, and taxa (Jones et al, 2014; Lemaitre et al, 2024; Sanghvi et al, 2024a). Researchers
72 generally derive information from populations of individuals followed over time in order to
73 infer the latent (true) average within-individual ageing trajectory of the entire sampled cohort
74 (Baudisch and Stott, 2019; Gaillard and Lemaitre, 2020; Lemaitre and Gaillard, 2017; Nussey
75 et al, 2013; Lemaitre et al, 2020). To do so, they commonly account for heterogeneity by
76 modelling consistent among-individual differences (Tuljapurkar et al 2009; Vindenes et al
77 2008), for instance, by using random effects (Bichet et al, 2022; Mohring et al, 2026; Pasztor
78 et al, 2022). However, unobserved effects of individual frailty on measured traits can still bias
79 estimates of ageing through ‘selective disappearance’ (Bhat and Kokko, 2026).

80 Selective disappearance arises when individuals’ phenotypes co-vary with their
81 likelihood of dying, removal or disappearance from the sampled population (Forslund and
82 Part, 1995; Lailvaux and Kasumovic, 2011; van de Pol and Verhulst, 2006; Vaupel et al,
83 1979; Vaupel and Yashin, 1985; Wilson and Nussey, 2010; Figure 1Aii, iii). This can cause
84 the observed or among-individual (cross-sectional) age-specificity of a trait to diverge
85 substantially from the true but latent, average within-individual (longitudinal) ageing
86 trajectory (van de Pol and Verhulst, 2006; Rebke et al, 2010). Negative covariances between a
87 trait and lifespan, such as when improvements in a trait comes at the cost of reduced survival,

can lead to the selective disappearance of high-quality individuals. In contrast, positive covariances can lead to individuals of a lower average quality, disappearing. This might occur when individuals with greater resource acquisition improve multiple traits and their lifespans while individuals with reduced resource acquisition have shorter lifespans as well as poor-quality phenotypes (Sanghvi et al, 2025a; van Noordwijk and de Jong, 1986).

Selective disappearance is likely pervasive across traits and taxa. Among birds, for example, 46% of trait-species combinations show evidence of selective disappearance (e.g. Bouwhuis & Vedder 2017). In other species, individuals with: shorter telomeres (Bichet et al, 2020; Haussmann and Mauch, 2007; Pepke et al, 2023; Power et al, 2025; Salmon et al, 2017); poorer immune function (Beirne et al, 2016; Peters et al 2019; Power et al, 2023); smaller body size (Hamalainen et al, 2014; Hayward et al, 2013; Nussey et al, 2008; Sanghvi et al, 2022); and lower reproductive performance (Lopez-Idiaquez et al, 2016; McLean et al, 2019; Sanghvi et al, 2025b; Sharp and Clutton-Brock, 2010; Thorley et al, 2020), have been found to selectively disappear. Selective disappearance also impacts traits related to social behaviour (Albery et al, 2022; Sadoughi et al, 2024; Moiron & Bouwhuis 2024; Siracusa et al, 2022) and migratory efficiency (Sergio et al, 2014; Wynn et al, 2025), with more social individuals or those better at navigating, having lower mortality. Finally, selective disappearance of low-quality parents can also buffer deleterious parental age effects via associations between parental survival and offspring phenotypes (Cansse et al, 2024; Ivimey-Cook and Moorad, 2018; McLean et al, 2019; Moullec et al, 2025; Sanghvi et al, 2024b).

Commonly used methods to assess, and account for, selective disappearance, in ecology

Primarily, ecological studies commonly use three methods (henceforth: ‘additive covariate’, ‘mean-centring’, and ‘decomposition’) to quantify ageing and account for/measure selective disappearance (Figure 1). However, these methods have been developed in the last two decades, such that we are only now beginning to appreciate their strengths and limitations. The ‘additive covariate’ method (Table 1) includes lifespan as an additive term alongside age in a mixed model, to account for age-independent associations between individual removal/mortality and average trait expression (Van de Pol and Verhulst, 2006; Figure 1A). This method is widely used due to its simplicity and because it directly fits the variable which mediates selective disappearance (e.g. Bouwhuis et al, 2010a, 2010b; Cope et al, 2022; Froy et al, 2013; Ivimey-Cook and Moorad, 2018; Moullec et al, 2025; Sanghvi et al, 2022). However, it requires lifespan data from a sufficient number of known-aged individuals, which can limit its application. In the absence of such data, the age at which an individual was last observed or sampled, henceforth ‘ALR’, is often used as a proxy.

The ‘mean-centring’ method (Table 1) partitions the overall, observed, population-level effect of age into an among-individual (the mean sampled age of an individual), and a within-individual component (Δage , i.e. the difference between the sampled age and mean age) (Fay et al, 2022; van de Pol and Wright, 2009; see Figure 1A). The strength of this method lies in it being able to partition the overall age effect into two processes without requiring data on lifespan (e.g. Bichet et al, 2025; Bouwhuis et al, 2015; Evans et al, 2011; Fay et al, 2021; Sparks et al, 2022; Sun et al, 2025). However, this method has less power to detect within-individual than among-individual age effects when sampling intervals are few (Westneat et al, 2020). Additionally, this method was originally developed for linear patterns (van de Pol and Wright, 2009), and only recently, nuances of its use when evaluating non-

linear ageing trajectories have been discussed- such as whether to model the quadratic within-individual term under an absolute or a relative ageing process (Fay et al, 2022).

The ‘decomposition’ method (Table 1) estimates the average, within-individual change in a trait between two successive age classes by comparing the mean values of a trait at those ages, but only of those individuals that are sampled at both ages (Rebke et al, 2010; Figure 1B). Here, the difference in the trait value between the two successive ages directly represents the within-individual change, but only so for that subset of survivor-restricted individuals (Martinig et al, 2021). This method is the least used out of the three because it: (i) relies on complete sampling, i.e. nearly all individuals alive being measured at each age, to obtain a representative set of data (Hayward et al, 2013; Hamel et al, 2018; Rebke et al, 2010); and (ii) does not provide statistical testing for the overall effect of age (Hamel et al, 2018; Hayward et al, 2013). Thus, it is sometimes used in conjunction with the mean-centring or additive covariate methods (e.g. Hamel et al, 2018; Murgatroyd et al, 2018; Nussey et al, 2011; Zhang et al, 2015).

Empirical studies that have compared the covariate and mean-centring methods find their conclusions to closely align with each other (Bichet et al, 2022; Gonzalez-Bernardo et al, 2025; Kauzalova et al, 2022). However, their agreement with the decomposition method is mixed, with some studies finding similar results (Hamel et al, 2018; Hayward et al, 2013; Nussey et al, 2011) while others reporting discrepancies (Zhang et al, 2015).

Limitations of current methods

An implicit assumption made in all three methods as presented here and as commonly used, is that individuals are disappearing from the population based on their average quality. Specifically, they assume that the bias caused by selective disappearance is *age-independent*

and the covariance between trait expression and lifespan (or removal) does not change with age. However, in populations where longer-lived individuals have slower rates of improvement and senescence compared to shorter-lived individuals (Fay et al, 2018; Smallegange and Guenther, 2025; van de Walle et al, 2023), or vice-versa, this assumption can be violated (Hayward et al, 2013; Hamel et al, 2018; Nussey et al, 2008; Zhang et al, 2015). This assumption might also be violated when the direction and magnitude of phenotypic correlations/trade-offs between life-history traits changes with age (Janeiro et al, 2025; Maklakov et al, 2017; Spagopoulou et al, 2020; Torres et al, 2011; Figure 1Aii vs Aiii). Most studies in ecology do not account for age-dependence of selective disappearance, with only few to our knowledge doing so by including an ‘age * ALR’ ‘interaction model’ (see Amininasab et al, 2017; Bouwhuis et al, 2009; Dugdale et al, 2011; Hamel et al, 2018; Kervinen et al, 2015; McCleery et al, 2008; Moiron and Bouwhuis, 2024; Robinson et al, 2012; Sanghvi et al, 2024b; Torres et al, 2011; Wynn et al, 2025). It therefore remains largely unknown whether the three methods **as typically used by other ecological studies, and developed for simpler scenarios of selective disappearance,** accurately quantify the latent ageing trajectory or are subject to potential bias.

To our knowledge, a theoretical study in ecology is lacking that (i) benchmarks the three methods on the same dataset, (ii) compares them to known ageing trajectories under age-independent and age-dependent selective disappearance scenarios, and (iii) considers **realistic sampling designs, data structures,** and a range of life-histories. Here, we fill this gap using simulations, analytical solutions, and empirical data, to achieve three aims. First, we examine how age-dependent and age-independent selective disappearance cause the observed age-specificity of traits to differ from the latent, average within-individual ageing trajectory. Second, we compare results from different quantitative methods to test which best recapitulates the true ageing trajectory in these scenarios. Third, we quantify the accuracy of

184 these methods across various biological systems (e.g. short lifespans, varying rates and onsets
185 of ageing), study designs (e.g. missingness of data) and data structures.

Glossary

- CS: complete sampling
- MCAR: missing completely at random
- MWY: missing when young
- MWO: missing when old
- LS: lifespan
- ALR: age at last reproduction or observation (leaving the dataset)
- AFR: age at first reproduction or observation (entering the dataset)
- Δ age: centred age i.e. difference between chronological age and mean age

Methods

Parameterization of scenarios

We simulated 27 different scenarios of selective disappearance. Within each scenario, we simulated quadratic ageing trajectories of 250 individuals per replicate, across 50 replicates of simulated data (unless mentioned otherwise). We sampled four random variables to calculate individual-level age-dependent fecundity ($F_{i,\text{age}}$) using the data generating equation:

$$F_{i,\text{age}} = \beta_{0i} + \beta_{1i} \cdot \text{age} + \beta_{2i} \cdot \text{age}^2 \quad \text{for } 1 \leq \text{age} \leq LS_i \quad (1)$$

where LS_i was the individual's lifespan, while the other three variables defined the quadratic ageing trajectory: the intercept (β_{0i}), the linear coefficient (henceforth, 'slope': β_{1i}), and the quadratic coefficient (henceforth, 'shape': β_{2i}). Biologically, the intercept represents the initial (or average, when age is grand-mean centred) fecundity for each individual, while the shape and slope collectively determine the rate of improvement or senescence in early- and late-life (Figure S1).

While we refer to 'fecundity' as the simulated age-dependent trait of interest, this can represent any non-survival, longitudinally measured trait. All individuals were assumed to begin expressing the trait at age one and were followed on a regularly spaced grid such that age and sampling occasion (time-step) corresponded one-to-one, and time was unitless. For each $F_{i,\text{age}}$, we added a small observation-level residual error ($\varepsilon_{i,\text{age}}$) sampled from a zero-mean normal distribution, $\mathcal{N}(0, \sigma_\varepsilon^2)$ where $\sigma_\varepsilon = 5$.

From a specified mean vector and a multivariate (VCV matrix) normal distribution, we jointly sampled four variables for every individual as:

$$\theta_i \sim \mathcal{N}_4(\boldsymbol{\mu}, \boldsymbol{\Sigma}) \quad (2)$$

201 where

$$\mu = \begin{pmatrix} \mu_{LS} \\ \mu_{\beta_0} \\ \mu_{\beta_1} \\ \mu_{\beta_2} \end{pmatrix} \quad (3)$$

$$\Sigma = \begin{pmatrix} \sigma_{LS}^2 & \rho_{LS,\beta_0}\sigma_{LS}\sigma_{\beta_0} & \rho_{LS,\beta_1}\sigma_{LS}\sigma_{\beta_1} & \rho_{LS,\beta_2}\sigma_{LS}\sigma_{\beta_2} \\ \rho_{LS,\beta_0}\sigma_{LS}\sigma_{\beta_0} & \sigma_{\beta_0}^2 & 0 & 0 \\ \rho_{LS,\beta_1}\sigma_{LS}\sigma_{\beta_1} & 0 & \sigma_{\beta_1}^2 & 0 \\ \rho_{LS,\beta_2}\sigma_{LS}\sigma_{\beta_2} & 0 & 0 & \sigma_{\beta_2}^2 \end{pmatrix} \quad (4)$$

202 We parametrized $\mu_{LS} = 25$; $\mu_{\beta_0} = 600$; $\mu_{\beta_1} = 9$; $\mu_{\beta_2} = -0.3$.

Here, note that LS did not enter the generating equation (Eq. 1), i.e. an individual's fecundity was never directly computed as a function of its lifespan. LS entered only through Σ and via truncation at $\text{age} \leq LS_i$. For our main analysis, we set the covariances between the slope, shape, and intercepts to zero because we were only interested in isolating the effects of selective disappearance. Our results were invariant to the specific, arbitrary parameterizations of the variable or coefficient-covariances (see sensitivity analyses below, and Supplementary section S1).

203

204 The standard deviation of each variable's unique distribution was set to 0.2 of its respective
 205 absolute mean, and minimum lifespan was truncated at one (Figures S2-S5). To manipulate
 206 the covariance between LS and any coefficient ($\rho_{LS,\beta_x}\sigma_{LS}\sigma_{\beta_x}$), we independently iterated the
 207 correlation between lifespan (LS) and that coefficient (ρ_{LS,β_x}). This gave us three types of
 208 selective disappearance associated with: (i) the intercept, (ii) the slope, and (iii) the shape. For
 209 each type, we varied three magnitudes of correlations: -0.5, 0, or +0.5, to simulate 27 (3^3)
 210 different scenarios (Figure S6). Here, non-zero correlations between LS-slope or LS-shape
 211 allowed us to simulate age-dependent selective disappearance.

212 Simulations were performed using R v4.6 (R core team, 2026) and MASS::mvrnorm (Ven-
 213 ables and Ripley, 2002) and matrix were used for Eq. 1-4. The complete simulation code and
 214 associated data are available at Open science framework [https://osf.io/kevnmm/overview?](https://osf.io/kevnmm/overview?view_only=93eba71a58444d19b5fb5287ffbc61e4)
 215 [view_only=93eba71a58444d19b5fb5287ffbc61e4](https://osf.io/kevnmm/overview?view_only=93eba71a58444d19b5fb5287ffbc61e4).

217 **Calculating ageing trajectories**

218 In our first simulation, all individuals alive at a given age were sampled for their fecundity at that
 219 age (henceforth, complete sampling-‘CS’ simulation). Here, after simulating individual-level
 220 ageing trajectories, we calculated three additional types of **population-level** ageing trajectories:
 221 (i) ‘true’, (ii) ‘observed’, and (iii) ‘decomposition’.

222 ***True trajectory***

223 We defined the ‘true’ trajectory as the expected, latent ageing trajectory that represented the
 224 within-individual pattern averaged across the entire population- this is typically what ecologists
 225 wish to estimate. It was a deterministic function generated by directly using the means of the
 226 three fecundity variables and was identical across all 27 scenarios. The fecundity at a given age
 227 for this trajectory was calculated as:

$$F_{\text{true,age}} = \mu_{\beta_0} + \mu_{\beta_1} \cdot \text{age} + \mu_{\beta_2} \cdot \text{age}^2 \quad \text{for } 1 \leq \text{age} \leq LS_{\text{max}} \quad (5)$$

228 ***Observed trajectory***

229 Separately for each replicate within every scenario, we also calculated the observed, population-
 230 level ageing trajectory across sampled individuals (**Aim 1**). At each age, the observed fecundity
 231 ($\bar{F}_{\text{obs,age}}$) of living individuals, was calculated as:

$$\bar{F}_{\text{obs,rep,age}} = \frac{1}{N_{\text{rep,age}}} \sum_{i|LS_i \geq \text{age}} F_{i,\text{rep,age}} \quad (6)$$

232 where $N_{\text{rep,age}}$ was the number of sampled individuals. For a given age, this value was then
 233 averaged across 50 replicates within each scenario and the observed trajectory was constructed
 234 by calculating $\bar{F}_{\text{obs,age}}$ across all sampled ages. Mortality and sampling design altered the
 235 composition of the population over time, thus, the observed trajectory reflected consequences
 236 of selective disappearance (**analytically shown** in Supplementary section S1, S2) and data or

237 sampling structure (see below).

238 ***Decomposition trajectory***

We also estimated age-dependent change in fecundity using the decomposition method (Rebke et al, 2010; Table 1, Aim 2). For each discrete age interval, we identified the subset of individuals alive and sampled at both ages. We then calculated a ‘decomposed’ mean within-individual change in fecundity across these two consecutive ages by restricting the calculation of change in the trait strictly to individuals sampled at both ages. To reconstruct the full trajectory across each age, we cumulated these interval changes from a starting baseline, $\hat{F}_{\text{start}}$. $\hat{F}_{\text{start}}$ was the mean fecundity of the subset of individuals observed at the first utilizable age who were also sampled at the next age (Table 1).

240 **Sparse sampling**

241 In addition to the CS simulation, we created three distinct types of simulations to separately test
242 the impact of three types of missingness in data (Aim 3).

243 ***Missing completely at random***

244 We simulated individuals to be missing completely at random (henceforth, ‘MCAR’ simulation)
245 at each age, which in empirical studies might be due to stochastic capture/recapture rates.
246 To do this, we first generated a complete sampling dataset as described above. We then
247 subsampled this dataset to create missingness by subjecting each time step of every individual,
248 independently, to a Bernoulli trial with a retention probability of 0.5. We doubled the number of
249 simulated individuals in each replicate of the MCAR simulation to 500 to maintain sample sizes
250 comparable to the CS simulation at each age. The MCAR simulation created stochastic among-
251 individual heterogeneity in the proportion of reproductive events sampled, while producing an
252 average retention rate of 50% across all individuals’ sampling events.

Age-dependent missingness

We simulated a dataset where older individuals were less likely to be sampled (henceforth, missing when old-‘MWO’ simulation). Biologically, this could represent lower sampling effort towards the end of a study, conducting of analysis before all individuals die, low recapture rates of old individuals, or higher rates of skipped breeding and reproductive failure in old individuals (see Bouwhuis et al, 2012; Zhang et al, 2015).

We also simulated a dataset where younger individuals were less likely to be sampled (henceforth missing when young-‘MWY’ simulation). Biologically, this could represent low capture rates of young individuals caused by their temporary dispersal/migration or reduced boldness, or the study commencing later than the birth/sexual maturity of many individuals. For the MWO and MWY simulations, we modeled 500 individuals per replicate. To create age-dependent missingness, we first generated a complete dataset as described above, which was then subsampled with the probability of retaining data at each time step for every individual, ‘ $P(\text{keep})_{\text{age}}$ ’, determined by a logistic function:

$$P(\text{keep})_{\text{age,MWO}} = \frac{1}{1 + \exp[k(\text{age} - a_m)]} \quad (7)$$

and,

$$P(\text{keep})_{\text{age,MWY}} = 1 - \frac{1}{1 + \exp[k(\text{age} - a_m)]} \quad (8)$$

where a_m represented the age at which the sampling probability was 50%; and k (set to 0.25) was the steepness of the sampling probability around the midpoint. A data point was only retained if a random number drawn from a uniform distribution between 0 and 1 was less than $P(\text{keep})_{\text{age}}$ for that observation. We set $a_m = 40$ for the MWO simulation, and $a_m = 10$ for the MWY simulation (Figure S7).

For the MCAR, MWO, and MWY simulations, the constructed ‘decomposition’ and ‘observed’ trajectories only used the respective sub-sampled data.

Calculating deviation from the true trajectory

For every simulated age, we estimated the relativised (as %) deviation between: (i) the observed ($\bar{F}$) and the true fecundity (F_{true}) as ' $D_{\text{Obs, age}}$ ' (Aim 1); and (ii) the decomposition ($\hat{F}$) and the true fecundity as ' $D_{\text{Deco, age}}$ ' (Aim 2); also see Figure 1Aiv):

$$D_{\text{Obs, age}} = \frac{1}{N_{\text{rep}}} \sum_{\text{rep}=1}^{50} \frac{\bar{F}_{\text{rep, age}} - F_{\text{true, age}}}{F_{\text{true, age}}} \times 100 \quad (9)$$

and:

$$D_{\text{Deco, age}} = \frac{1}{N_{\text{rep}}} \sum_{\text{rep}=1}^{50} \frac{\hat{F}_{\text{rep, age}} - F_{\text{true, age}}}{F_{\text{true, age}}} \times 100 \quad (10)$$

This was done independently for every replicate in each of the 27 scenarios, and separately for the four simulations (i.e. CS, MCAR, MWO, MWY). For each simulated age, this value was then averaged across the 50 replicates within each scenario. While the decomposition method was originally developed to estimate the survivor-restricted trajectory, our study investigated how well it recovered the latent average within-individual ageing trajectory. Importantly, we analytically solved how the observed and decomposition trajectory deflected from the true trajectory (see Supplementary section S1).

Comparison of statistical models

We compared the fit of six Linear Mixed-Effects Models (LMMs) (Aim 2) on the sampled data using the package `lme4::lmer` (Bates et al, 2015) with a Gaussian error distribution. In these, we modeled the fecundity F_{it} of individual i at measurement occasion t as our dependent variable. Each model was fit separately on data within each replicate and scenario, and separately for each simulation, using maximum likelihood to allow comparisons of different fixed effect structures (Table 1).

Table 1: Description of the models and methods we compared. Models 1-6 included a random intercept for individual ID (u_{0i}) and a residual error term (ϵ_{it}). $\overline{\text{age}}_i$ and $\overline{\text{age}}^2_i$ denote the individual means of linear age and quadratic age; $\Delta\text{age}_{it} = \text{age}_{it} - \overline{\text{age}}_i$; $\Delta\text{age}^2_{it} = \text{age}^2_{it} - \overline{\text{age}}^2_i$; ALR is the age at which the individual was last recorded; LS is the (true) latent lifespan.

Model	Description	Fixed effects
Model 1	Negative control not accounting for selective disappearance; matches the functional form of the generating equation	$\beta_0 + \beta_1 \text{age}_{it} + \beta_2 \text{age}^2_{it}$
Model 2	‘Additive covariate’ model with ALR (van de Pol & Verhulst, 2006).	$\beta_0 + \beta_1 \text{age}_{it} + \beta_2 \text{age}^2_{it} + \beta_3 \text{ALR}_i$
Model 3	‘Mean-centring’: separates among- ($\overline{\text{age}}_i$) and within-individual ($\text{age}_{it} - \overline{\text{age}}_i$; $\text{age}^2_{it} - \overline{\text{age}}^2_i$) components (van de Pol & Wright, 2009; Fay et al, 2022); see Supplementary section S2.2.1.	$\beta_0 + \beta_1 (\text{age}_{it} - \overline{\text{age}}_i) + \beta_2 (\text{age}^2_{it} - \overline{\text{age}}^2_i) + \beta_3 \overline{\text{age}}_i$
Model 4	Extension of Model 2, with an interaction between the age terms and ALR.	$(\beta_0 + \beta_1 \text{age}_{it} + \beta_2 \text{age}^2_{it}) + (\beta_3 + \beta_4 \text{age}_{it} + \beta_5 \text{age}^2_{it}) \cdot \text{ALR}_i$
Model 5	Extension of Model 3, with $\overline{\text{age}}_i$ interacting with the Δage terms.	$\beta_0 + \beta_1 (\text{age}_{it} - \overline{\text{age}}_i) + \beta_2 (\text{age}^2_{it} - \overline{\text{age}}^2_i) + [\beta_3 + \beta_4 (\text{age}_{it} - \overline{\text{age}}_i) + \beta_5 (\text{age}^2_{it} - \overline{\text{age}}^2_i)] \cdot \overline{\text{age}}_i$
Model 6	Positive control fitting latent lifespan as the interactive covariate.	$(\beta_0 + \beta_1 \text{age}_{it} + \beta_2 \text{age}^2_{it}) + (\beta_3 + \beta_4 \text{age}_{it} + \beta_5 \text{age}^2_{it}) \cdot \text{LS}_i$
Method	Description	Calculation
Decomposition (Rebke et al, 2010)	Survivor-restricted reconstruction of the within-individual trajectory. For each interval, the mean change is computed over individuals sampled at both ages, then cumulated from a baseline taken over that set of individuals.	$\hat{F}_{\text{age}} = \hat{F}_{\text{age}_{\text{start}}} + \sum_{t=\text{age}_{\text{start}}}^{\text{age}-1} I_t,$ $I_t = \frac{1}{ S_t } \sum_{i \in S_t} (F_{i,t+1} - F_{it})$ is the mean within-individual change between ages t and $t + 1$; S_t is the set of individuals sampled at both t and $t + 1$; $\text{age}_{\text{start}}$ is the earliest age for which S_t is non-empty; $\hat{F}_{\text{age}_{\text{start}}}$ is the mean trait value for individuals at $\text{age}_{\text{start}}$

Note that model 6 represents an idealized scenario in which each individual's true lifespan is known and can therefore be modelled directly. In reality, lifespan is frequently unknown. As selective disappearance is mediated by lifespan, however, the among-individual term in model 5 is what accounts for the bias, so mean age and ALR can be viewed as competing observable proxies of latent lifespan. The difference between the centring (3, 5) versus the ALR models (2, 4) is then partly a function of which proxy recovers lifespan better. Note that because in our simulations the trait was assumed to be expressed continuously or over regularly spaced intervals, under complete sampling, ALR was the same as discrete lifespan (see Supplementary section S2.2, S2.3). The age at first observation was the same across individuals, under complete lifespan, but differed in the MCAR and MWY simulations.

Model fit, accuracy and precision

We estimated the Akaike information criteria (AIC) for each model and used the average AIC across 50 replicates to compare models within each scenario and simulation type (Aim 2). A ΔAIC of ≥ 2 was interpreted as a worse model support.

To compare the accuracy of different models, we used the 'predict.merMod' function (*lme4* package) to predict the population-level age-effect by each statistical model's fixed effects, with ALR, mean age, and LS terms held at their sample means. Then, at each age and separately for each model in every scenario and simulation dataset, we calculated the relativized difference between the mean predicted fecundity value (across 50 replicates) of the model and the true trajectory as:

$$D_{\text{model, age}} = \frac{1}{N_{\text{rep}}} \sum_{\text{rep}=1}^{50} \frac{F_{\text{model, rep, age}} - F_{\text{true, age}}}{F_{\text{true, age}}} \times 100 \quad (11)$$

We also calculated the variance in each model's prediction and in the decomposition method's estimated fecundity, to compare the precision of these models and methods (Supplementary section S3).

Random slopes

Random slopes are sometimes included in statistical models to account for among-individual heterogeneity in ageing trajectories. To demonstrate that random slope terms do not account for age-dependent selective disappearance (Aim 2), we created a separate simulation. Here, we compared models 1-6 containing only a random intercept term, ' u_{0i} ', against models containing the same fixed effects but additionally containing a random slope term ' $u_{1i}age_{it}$ ' (representing the product of individual i 's constant deviation from the mean slope u_{1i} and the individual's changing age at each sampling point, t). For simplicity and due to sufficiency, we simulated ageing trajectories as linear. Consequently, the 12 models fit to these data did not contain a quadratic age term. As proof of concept, we only simulated three scenarios under complete sampling: (i) no selective disappearance, (ii) selective disappearance linked with intercept only, (iii) selective disappearance linked with slopes only. Lifespans, intercepts, and slopes were sampled from a multivariate distribution as described in Eq. 1-4 (however, excluding the shape variable). Here, μ_{β_1} was parameterized as -6 and σ_{β_1} as 1.2, to represent a life-history where senescence onsets in early-life without improvements in fecundity.

Sensitivity analyses

We further tested how various life-histories and data structures impacted our results (Aim 3). These included: (i) a faster or slower rate of ageing; (ii) small sample sizes; (iii) Gamma distribution of lifespan; (iv) a species with short mean lifespan; (v) a VCV matrix where $\beta_0 - \beta_1 - \beta_2$ also co-varied with each other (e.g. Maklakov et al, 2015; Spagopoulou et al, 2020); (vi) large residual error ($\varepsilon_{i,age}$) for each fecundity observation, such that $\sigma_{\varepsilon} = 0.3 * F_{i,age}$, which allowed the measured fecundity at each age to deviate substantially and stochastically from the underlying individual-specific quadratic function (Eq. 1).

Detailed descriptions of these sensitivity analyses can be found in Supplementary section S4 (Figures S8-S11).

Empirical demonstration

To test whether the commonly used additive models 2 and 3 lead to distinct inferences about ageing compared to the seldom used models 4 and 5 in empirical systems, we re-analyzed data from two studies (Aim 2). One contained data on the fecundity of lab-adapted female fruit flies across their lifetime (Sanghvi et al, 2025b). The other dataset was obtained from a long-term study on a wild population of great tits (Bouwhuis et al, 2009) and contained data on a phenological trait (egg laying date) that was not analyzed in the original study. We fit models 1-5 to both datasets, compared model AICs, and visualized their predictions for the effects of age. We also included relevant environmental and treatment variables as suggested within each corresponding study. More details can be found in Supplementary section S5.

A summary of our entire workflow can be found in Figure S11.

Results

Processes that cause deviation between trajectories

In our simulation with complete sampling of individuals and a normally distributed average lifespan of 25 time steps (i.e. ‘CS’ simulation), the ‘observed’ population-level ageing trajectory of the trait closely matched the ‘true’ within-individual trajectory in the absence of selective disappearance (Figure 2A). As expected, when selective disappearance was linked with the intercept (Figure 2B-C), slope (Figure 2D), and/or shape (Figure 2E-F) of the trait, this led to substantial deviations between the observed and true trajectories especially in mid- and late-life (Figure S12-S14). Here, the true ageing pattern was either over- or underestimated, depending on the sign of each correlation with lifespan (Aim 1). Importantly, even when selective disappearance was not linked with the trait’s intercept, but with its slope and/or shape, the observed ageing trajectory deviated substantially from the true trajectory (often by >50%- Figure 2D-F). The decomposition trajectory matched the true trajectory under no selective disappearance as well as under selective disappearance linked with the trait’s intercept (Aim 2, Figure 2A-C). However, when selective disappearance was linked with trait’s slope and/or shape, the decomposition method produced biased results that were particularly substantial in late-life (up to 40%; Figure 2D-I, S12, S13; see analytical solutions for an explanation).

How well do statistical models perform?

In our CS simulation, model 4 (containing a two-way interaction between ALR and age) and 5 (an interaction between the $\overline{\text{age}}$ and Δage terms) had lower AICs than other models across all scenarios (Figure 3, Figure S15). This included scenarios where there was no selective disappearance (Figure 3A), or only selective disappearance linked with the intercept (Figure

3B-C)- which was because the interaction terms partly recovered among individual differences in ageing. Importantly, under complete sampling, models 4 and 5 had near-identical AICs because mean age and ALR were perfect proxies of LS (Pearson's $r = 1$; also see Fay et al, 2022) (Aim 2).

Whenever selective disappearance was linked with the slope and/or shape (Figure 4), the decomposition method provided a biased estimate of the latent, average within-individual ageing trajectory of the entire cohort (deviating up to 40%) compared to predictions from models 4 and 5 (< 5% deviation) (Figure S16). There was no substantial penalty on the precision of models 4 and 5 despite them having more parameters than models 1-3 (Figure S17). Our sensitivity analyses (summarised in Figure S11, Aim 3) parameterized for: faster or slower rates of ageing (Figure S18-S25), short-lived species (or fewer time steps) (Figure S26 to S28), or a reduced sample size (Figures S29, S30), produced qualitatively similar results as above. Moreover, even when the data-generating process contained covariances among the intercept-slope-shape, the results remained the same (Figure S31). One exception to the robustness of these results occurred when observed fecundity values stochastically deviated from the underlying quadratic trajectory of each individual due to large sampling error. Here, among-individual differences in ageing patterns were likely not recoverable by the interaction terms in models 4, 5, and 6 (which fit $LS \times \text{age}$ terms). Consequently, in the absence of selective disappearance linked with slope or shape, models 4, 5, and 6 provided a worse fit to the data than models 1-3 (Figure S32-S35).

We also simulated linear trajectories of ageing to represent an early onset of senescence without initial improvements in trait (Aim 3), and here, compared models with or without random slopes (Aim 2). As expected, the six models that only contained random intercepts (Figure 5A-C) performed worse than their counterparts that additionally included random slopes (Figure 5D-F). Under age-dependent selective disappearance (i.e. linked to the

slope), models 4-6 outperformed models 1-3 (Table 1), but simply modelling a random slope term was insufficient to account for age-dependent selective disappearance bias (Figure 5C vs 5F). Even under the absence of selective disappearance, the interaction models (4 and 5) provided a better fit to the data when neither of the models included a random slope term (Figure 5B). However, when random slopes were included in a scenario without selective disappearance, all six models performed equally well (Figure 5E). These results demonstrate that a random slope term does not account for age-dependent selective disappearance. However, the interaction terms in models 4-6 account for some among-individual heterogeneity in ageing when a random slope term is not modelled. This further corroborates results from the CS simulation with large sampling error (Figures S32-S35).

The influence of missingness of data

In the MCAR simulation where 50% of data was missing completely at random, deviations between the observed or decomposition, versus the true trajectory were qualitatively similar those obtained from the CS simulation (Aim 3; Figure S36, S37). However, in contrast to findings from the CS simulation, in the MCAR simulation, model 4 not only fit the data better than models 1, 2, and 3, but also model 5 across all scenarios (Figure 6, Figure S38). Here, mean age (model 5) was a poorer proxy of LS ($r = 0.852$) than ALR (model 4) was ($r = 0.962$; Figure S39, see Supplementary Section S2.2). Comparisons of model predictions or the decomposition trajectory to the true trajectory corroborated these results. Specifically, across all scenarios model 4 better recovered the true latent trajectory, compared to models 1, 2, 3, 5 and the decomposition method (which deviated up to 40% in several scenarios) (Figure S40). These results held true even for the simulated shorter-lived species ($\mu_{LS} = 5$) (Figures S42-S44).

Similarly, when individuals were more likely to have missing data at either old or young ages (i.e. MWO or MWY simulation), results closely paralleled those from the MCAR simulation, with model 4 fitting the data and recapitulating the true trajectory, better, compared to models 1, 2, 3, 5, and the decomposition method across all scenarios (Aim 3; Figure S45-S52).

Alternative lifespan distributions

The sampling of lifespan from right-skewed Gamma distributions (to represent high early-life mortality and a long tail), simulated separately for complete and MCAR designs, produced similar results to those described above, irrespective of the number of mean time steps (i.e. species' lifespan) simulated (Figure S53-S56). A general mechanism of selective disappearance free from assumptions about lifespan distributions or ageing functions, can further be found in Supplementary section S2.1.

Empirical implementation

We re-analysed data from two published studies (Bouwhuis et al, 2009; Sanghvi et al, 2025b) to show how our theoretical results apply to real datasets. Our reanalysis of the fruit fly dataset containing data on female fecundity, showed that model 4 provided the best fit to the data (ΔAIC compared to model 4, of models 1, 2, 3, and 5 = 38.4, 24.1, 28.0, and 2.7, respectively; Figure 7A). Here, individuals that lived longer not only had higher average fecundity but also had slower rates of senescence (consistent with age-dependent selective disappearance) (Table S1). Consequently, model 4 predicted steeper senescence compared to other models (Figure 7B). Our analysis of female egg laying date and its ageing in the great tit dataset similarly revealed that model 4 outperformed other models (ΔAIC compared to

models 1, 2, 3, and 5 = 11.8, 9.5, 53.7, 48.7 respectively; Table S2; Figure 7C). Here, model 4 predicted a steeper advance in laying date at older ages compared to other models, whereby individuals with different ALR had distinct age-dependent changes in laying date- a pattern consistent with age-dependent selective disappearance (Figure 7D). Models 4 and 5 had more similar predictions in the fly compared to the tit dataset. This possibly reflects the former being a lab study and containing lower levels of missingness.

Analytical solutions

Our analytical solutions provided several important insights specific to our simulation findings (fully presented in Supplementary section S1, S2). First, the deviation between the observed and true pattern at a given age due to selective disappearance at an age, $D(t)$, depends on the product of two processes (Eq 12): the mortality hazard and the overall covariance between lifespan and fecundity at that age:

$$D(t) = \frac{\lambda(\alpha_t)}{\sigma_{LS}} \text{Cov}(LS, F_t) \quad \text{Eq. 12}$$

Here, the observed trajectory deviates from the average within-individual trajectory because individuals that survive either, (i) do not have the same age-independent quality (i.e. differ in intercept), or (ii) do not have the same ageing pattern (i.e. differ in their slope or shape) (Figures S57-S59). Due to LS-shape and LS-slope correlations being scaled by age, age-dependent selective disappearance produces a greater bias in later-life. Importantly, selective disappearance of the intercept can lead to a bias that is entirely independent of individual-level ageing (e.g. observing population-level ageing despite individuals themselves not ageing). Further, the magnitude of selective disappearance at a given age depends on the variance in intercept, slope, and shape (Eq. 13), but not variance in lifespan when age is

unstandardised (Figure S60-S62). However, the rate of change of the deviation over time depends inversely on the variance in lifespan (Figure S60-S62; Supplementary section S1.4).

$$D(t) = \lambda(\alpha_t) \cdot (\rho_{LS,\beta_0} \cdot \sigma_{\beta_0} + t \cdot \rho_{LS,\beta_1} \cdot \sigma_{\beta_1} + t^2 \cdot \rho_{LS,\beta_2} \cdot \sigma_{\beta_2}) \quad \text{Eq. 13}$$

Second, there is an inaccuracy associated with the decomposition method, which evaluates the survivor-restricted trait change across two time-steps (ΔI_t), when it is used to infer the latent average trajectory. It successfully removes bias caused due to intercept-LS correlations (i.e. age-independent selective disappearance) and due to changing mortality rates (a bias that the observed population-level trajectory contains). However, the decomposition method mathematically cannot remove bias caused by age-dependent selective disappearance linked to the slope or shape (or any age-dependent coefficient in any ageing function) (Figure S63; Supplementary section S1.3, Eq. 14). Therefore, at older ages, this produces an inaccurate estimate of the latent average within-individual ageing trajectory:

$$\Delta I_t = \underbrace{(\mathbb{E}[F_{t+1}] - \mathbb{E}[F_t])}_{\text{True change (latent cohort average)}} + \underbrace{\frac{\lambda(\alpha_{t+1})}{\sigma_{LS}} [\text{Cov}(LS, F_{t+1}) - \text{Cov}(LS, F_t)]}_{\text{Decomposition bias (survivor restricted)}} \quad \text{Eq. 14}$$

Finally, we produced general solutions for describing: (i) the latent, 'true' trajectory as the average of individual ageing trajectories over the original cohort: $\mathbb{E}[F_t] = \int g(t, \theta) f(\theta) d\theta$; (ii) the observed trajectory as this same average but after reweighting the distribution of individuals by their probability of surviving to each age: $\mathbb{E}[F_t | T > t] = \int g(t, \theta) f(\theta | T > t) d\theta$; and (iii) selective disappearance as a survival-induced reweighting of the individuals contributing to the population mean: $D(t) = \text{Cov}_\theta(g(t, \theta), w_t(\theta))$. Here, $g(t, \theta)$ describes an individual's trait value at age t given the vector of individual-specific parameters θ ; $f(\theta)$ describes the distribution of these parameters across the original cohort; and $w_t(\theta) = P(T > t | \theta) / S(t)$ is the relative survival weight, with $P(T > t | \theta)$ being the

parameter-dependent probability of surviving to age t , and $S(t) = P(T > t)$ being the proportion of the cohort surviving to that age.

Discussion

Aim 1: Age-dependent selective disappearance

For a quadratic ageing function, the covariance between lifespan and slope or shape at a given age determines the age-dependent component of selective disappearance, whereas the covariance between lifespan and intercept determines the age-independent component. For a non-quadratic ageing function such as $F_{\text{age}} = \beta_0 + \beta_1 \ln(\text{age})$, this just becomes the age-independent (β_0) versus age-dependent coefficients (β_1). Our study shows that age-dependent selective disappearance driven by the removal of individuals with distinct ageing trajectories in a trait, can bias estimates of the true underlying ageing pattern. This occurs even when selective disappearance linked with the trait's age-independent coefficient is absent or accounted for, and becomes especially problematic at later ages.

There are several non-mutually exclusive reasons for why age-dependent selective disappearance can manifest (Figure 1Aiii). First, the ageing trajectory may co-vary with lifespan if individuals in a population differ in their pace-of-life (Hayward et al, 2013; Hamel et al, 2018; Healy et al, 2019; Reale et al, 2010; Torres et al, 2011). Specifically, if individuals that die earlier also display steeper rates and early onset of senescence in other traits, older age classes will be increasingly composed of organisms with shallower rates and later onset of senescence. Second, age-dependent selective disappearance could manifest due to condition-dependence. For example, if the survival cost of expressing/improving a trait at a young age is low for all individuals, everyone might be able to express the trait equally well, leading to a shallow covariance between the trait and lifespan in early-life. However, at old

ages, if the cost is low only for individuals in better condition, longer lived individuals might express the trait more than soon-to-die individuals (Johnson and Gemmell, 2012). This could cause the association between this condition-dependent trait and lifespan to be age-dependent (Brooks and Kemp, 2001; Hansen and Price, 1995; Kokko, 1998; Sanghvi et al, 2024b). Such a covariance can also arise if younger individuals acquire resources more efficiently, causing the trait-survival trade-off to be shallow early in life and steeper as acquisition declines with age. Third, age-dependence of selective disappearance could also occur due to the force of selection weakening with age (Donertas and Partridge, 2025; Hamilton, 1966; Lemaitre et al, 2024; Maklakov and Chapman, 2019; Wensink et al, 2017; Williams, 1957; see Figure S64, S65). Fourth, trade-offs between somatic repair and the trait of interest, might generate age-dependent selective disappearance (Chen et al, 2020; Lemaitre et al, 2015, 2024; Maklakov and Chapman, 2019). Specifically, at young ages, accumulated damage is lower, which could lead to the lifespan-costs of maintaining or expressing a trait being buffered. However, at old ages, such costs might be greater because somatic damage has accumulated (Kirkwood and Rose, 1991) causing LS-trait covariances to become more negative with age. Fifth, terminal investment, where individuals allocate greater resources to reproduction at the expense of survival at later, but not at earlier ages, due to residual lifespans reducing with age (Duffield et al, 2017; Reed et al, 2008), might cause lifespan-reproduction correlation to become more negative with advancing age (Figures S64, S65).

Aims 2 and 3: Comparison of methods across a variety of systems and data structures

Additive vs. interactive, mean-centring vs. covariate models

We compared the performance of two commonly used models (i.e. the ‘additive covariate’ model 2, and the ‘mean-centring’ model 3) to that of seldom used ‘interaction’ models (4 and

5). These models differed in whether they were able to account for age-dependence of selective disappearance. Unsurprisingly, the additive models produce biased estimates of the ageing pattern when selective disappearance is age-dependent (i.e. due to LS-slope or LS-shape correlations). In such scenarios and under no missingness of data, both interaction models (4 and 5) produce identical estimates of the true trajectory (also see Fay et al, 2022) and outperform other models. Interestingly, the interaction models provided a better fit to the data even under the absence of selective disappearance, because the interaction term allowed partial recovery of among-individual heterogeneity in ageing trajectories. This, however, was limited only to instances where either heterogeneity was not masked by sampling error/noise and could be recovered, or when heterogeneity was not directly modelled using random slope terms.

Under incomplete sampling (MCAR, MWO, MWY), model 4 (included ALR x age term interactions) consistently outperformed other models including model 5 (contained $\bar{\text{age}}$ x Δage term interactions). Our reanalyses of two empirical datasets exemplified this pattern, where model 4 provided the best fit to the data and a substantially different prediction of the late-life ageing trajectory compared to other methods. Our analytical solutions pinpoint the reasons for differences between models 4 and 5 (see Supplementary section S2.2, S2.3). Selective disappearance occurs when the mean phenotype of truncated individuals differs from that of the untruncated population, and models that better capture this covariance better explain the data. ALR in Model 4, and mean age in model 5, adjust for selective disappearance bias. When AFR (age of entry) is fixed, these two terms can be viewed as different proxies of latent lifespan. Here, under missingness, the error associated with ALR is lower than the error associated with mean age, in how well they approximate latent lifespan (Figure S39, S66). Furthermore, in model 5, this error permeates into not only the among-individual term, but also the within-individual term's calculation. On the other hand, in model

4, ALR's error is restricted to a single term. When AFR is variable, mean age is penalised further, making it a worse proxy of lifespan even under complete sampling.

Model 4 vs. decomposition method

Across most scenarios and simulations, in mid- and late-life, the decomposition method performs substantially worse than model 4, and especially so under age-dependent selective disappearance. There are two explanations for this. First, in early life and under complete sampling, sufficient numbers of individuals are sampled at successive time-steps, such that cohort averages provide a precise estimate of within-individual change. However, once mortality accelerates in later-life, sample size for successively sampled individuals necessarily gets lower, causing imprecision of the decomposition method. Missingness of data further exacerbates this (Hamel et al, 2018; Hayward et al, 2013; Nussey et al, 2011)- for example, if detection probability is $0 < p < 1$, the likelihood of sampling an individual at two successive ages reduces to p^2 . Second, our analytical solutions confirm a bias (Zhang et al, 2015) in the decomposition method- it implicitly assumes that the rate of ageing is the same for individuals that survive and those who disappear (latent). When selective disappearance is age-dependent, this survivor-restricted calculation becomes more inaccurate with age if used to infer ageing of the entire cohort (Figure S63; Supplementary section S1.3, S1.4).

Suggestions for empiricists

We suggest several recommendations for empiricists to diagnose selective disappearance (Figure 8A) and choose the most appropriate model (Figure 8B). Currently, when lifespan data are incomplete, researchers tend to use the mean-centring method by calculating the mean and delta age variables from the sampled time steps. However, we demonstrate that

ALR provides a less error-prone approximation of latent lifespan than mean age does under missingness of data, allowing model 4 (uses ALR) to better account for among-individual processes than model 5. We thus suggest that model 4 and 5's performance be compared when analysing datasets with missingness, rather than choosing model 5 indiscriminately, as is common practise in ecology. Here, empiricists could further report: (i) whether missingness is random, age- or trait-dependent (e.g. Bouwhuis et al, 2012; Mittell et al, 2025); and (ii) the strength of the ALR-mean age correlation. Importantly, we recommend that studies first test for age-dependent selective disappearance in their data using the interaction model (model 4) whenever possible, and use the additive model (model 2) when the interaction terms are not significant or not of biological interest. We recommend that empiricists refrain from using the decomposition method if their aim is to recover the latent ageing trajectory, unless there is no age-dependent selective disappearance, no missingness, and an availability of large number of consecutively sampled individuals across all sampled ages. Importantly, empiricists often use the incorrect algebra to partition the among- and within-individual age effect when using the mean-centring method. We urge that when using models 3 or 5, researchers be aware of the mathematical underpinnings and biological interpretation of the mean-centring method before applying it to their data (explained in Supplementary section S2.2.1, 2.2.2).

If the data do not allow using the interaction models due to overfitting or convergence issues, we recommend several approaches to qualitatively infer the presence and type of selective disappearance. First, the influence of age on a specific trait could be visualised by grouping individuals into different ALR bins (e.g. Figure 1A, 7B, 7D, 8; Figure S6). Here, under no selective disappearance, the different bins should overlap substantially or fluctuate randomly. Under age-independent selective disappearance (linked with the intercept), the shape of each bin's trajectory will be similar, but its average value will be consistently different. However, under age-dependent selective disappearance (linked with slope or

shape), each bin will display a distinct trajectory of ageing with a systematic increase or decrease between the bins as age progresses. Similarly, we recommend visualising the correlation between lifespan (or ALR) and the sampled trait at each age. If the regression coefficients of these associations systematically change with age, this likely indicates that selective disappearance is age-dependent (e.g. Figure S64, S65). Furthermore, for data with large sample sizes, continuous traits sampled over many time-steps, and low stochasticity in individual trajectories, parametric ageing functions can be fit for each individual (e.g. quadratic, logarithmic). The function of average coefficients, $E[f(\theta)]$, or the average function, $f(E[\theta])$, can provide a parametric approximation of the within-individual ageing trajectory free of selective disappearance. All of these recommendations and diagnostics can be undertaken in our supplementary shiny app, *disappR* (<https://github.com/ElvimeyCook/disappR>).

Ultimately, a model is just a hypothesis about the underlying data generating process (Westneat et al, 2020) and the model that most closely resembles this process will best recover the true trajectory. Our simulation's data generating process contained several assumptions: individuals followed quadratic functions; missingness was independent of condition; latent environmental influences were not included (see Reale et al, 2026); the ageing function was linear in parameters; traits were sampled from continuous distributions; lifespan and the sampled trait were linearly related at a given age; $LS-\beta_x$ correlations were large; and the average, within-individual ageing trajectory was known. We also assumed that the trait (e.g. fecundity) was regularly or continuously expressed, such that 'time-steps', which tracked individuals from birth to death, matched the ages at which the trait was expressed. However, violation of some of these assumptions could impact how well different methods recover the true trajectory (see Supplementary section S2 for further discussion).

These modelling choices were made to enable clear interpretation of results and model biases, to pinpoint the mechanism of selective disappearance, and to facilitate implementable recommendations. We addressed several of these limitations by: (i) demonstrating that insights from our simulation are applicable to empirical datasets; (ii) independently deriving analytical solutions that generalise our results beyond the specific assumptions of our simulations (Supplementary Section S1, S2); and (iii) conducting several sensitivity analyses to show that our results are generally consistent even when certain assumptions are violated. However, not all data structures can be addressed in a theoretical study. We suggest some alternate models that empiricists could utilize when the data generating processes violates these assumptions. For example, ALR could be included as a non-linear (e.g. quadratic) term if individuals with medium lifespans are of a higher quality than individuals with the most extreme lifespans (Balbontin et al, 2007; Nussey et al, 2006, 2009, 2011; Reid et al, 2003; Simons et al, 2016). Generalized additive mixed-effects models containing a tensor product smooth between ALR and age (e.g. in R's *mgcv* package), rather than ALR being included as a parametric linear term (e.g. Cooper et al, 2021; Hamalainen et al, 2014; Sanghvi et al, 2021), could also be fit on large datasets where ageing trajectories cannot be described by any parametric function. Similarly, if quadratic functions are not the appropriate ageing trajectory, any other function (e.g. logarithmic, cubic) can be fit instead with analogous age-independent and age-dependent terms (e.g. $\text{trait} \sim \log(\text{age}) * \text{ALR} + (1 | \text{id})$). Furthermore, if the survival process is known, this variable instead of the absolute lifespan, can be jointly modelled with the longitudinal data and linked through shared random effects- although such data are rarely available in ecology, making joint models impractical (see Li and Su, 2018; Reinke et al, 2020; Wu et al, 2012). In our study, AFR (minimum age/age at first observation) only varied among individuals in the MWY and MCAR simulations, but our simulations did not contain selective appearance (see Figure S67, S68).

If selective appearance is present, i.e. the entering of individuals being associated with their phenotype, we recommend that empiricists can diagnose it in a similar way to selective appearance, and model AFR in addition to ALR (Dingemanse et al, 2020; van de Pol and Verhulst, 2006). Importantly, we define the true trajectory as the latent, average within-individual trajectory, because this is what ecologists often wish to estimate. However, this definition carries several counterfactuals that we urge ecologists to be aware of when interpreting their data (Supplementary section S2.4).

We caution researchers that selective disappearance can also manifest in systems or at levels of biological organisation that are not typically considered. For instance, sperm cells that are sampled later within stored ejaculates could exhibit a biased sperm phenotype due to selective death of poor-quality or rapidly senescing sperm (Alavioon et al, 2017; Marcu et al, 2024; Sanghvi et al, 2025c). Furthermore, reaction norms might selectively disappear across an environmental gradient, such that in more extreme environments, sampled individuals are those with steeper or shallower reaction norms (Fay et al, 2022). Moreover, in studies where individuals mate sequentially with several partners, those with higher mating success might also have a higher reproductive output with each mate, compared to individuals that only mate with few partners (e.g. Sanghvi et al, 2025d). This would lead to later matings containing a biased sampling of reproductive phenotypes (Sanghvi et al, 2025e). Quantifying and accounting for such biases thus is crucial when analysing any longitudinal data.

Figures

Figure 1: Conceptual diagram explaining three methods commonly used in ecology to assess ageing and selective disappearance. (A) Heterogeneity among individuals (here represented as hypothetical individual-level age-dependent changes in fecundity) can occur in their intercepts or their trajectories of ageing. Furthermore, individuals can differ in their lifespans, which can co-vary with individual intercepts and/or ageing trajectories. This can lead to age-independent (A.ii) and age-dependent (A.iii) selective disappearance, respectively. The among-and within-individual ageing terms, and their additive and interactive influences, can be used to evaluate and account for different forms of selective disappearance. In our study, we evaluate these, and compare the true trajectory against the observed ageing trajectory to quantify the influence of different forms of selective disappearance, and the ability of different methods to account for this. (B) The decomposition method considers only the subset of individuals sampled at two successive time steps to estimate the average within-individual change over that time step.

Figure 2: The relativised deviation caused by selective disappearance in a simulation of ageing inferred under complete sampling (CS) scenarios. Orange curves shows the relativised deviation between the observed (cross-sectional) versus true (within-individual) trajectory; purple curves, between the decomposition and true trajectory, averaged across 50 replicates. Black (dashed) curve shows the analytical calculation (Supplementary section S1) of the relativised deviation between the observed versus true trajectory. The vertical dotted line shows the true age of onset of senescence in fecundity. For simplicity, only 9 scenarios are shown, but all 27 considered scenarios are presented in Figure S15. '-', '0', and '+' in the facet labels represent whether the correlation between LS and that fecundity-coefficient is -0.5, 0 or +0.5, respectively; here, the first symbol corresponds to LS-intercept (β_0), the second to LS-slope (β_1), and the third to LS-shape (β_2), correlations. The top-left panel shows no selective disappearance. Extreme deviation at ages 42+ is due to small sample size causing imprecision. Note that models 4, 5 and 6 have the same degrees of freedom.

Figure 3: Support by the data for models 1-6 in the CS simulation. Points show ΔAIC (mean and ΔSE across 50 replicates) compared to the best fitting model. For simplicity, only 9 scenarios are shown but all 27 considered scenarios presented in Figure S15. '-', '0', and '+' in the facet labels represent whether correlation between LS and that fecundity-coefficient is -0.5, 0 or +0.5, respectively; here, the first symbol corresponds to LS-intercept, the second to LS-slope, and the third to LS-shape, correlations.

Figure 4: Comparison of model/method predictions for the effect of age on fecundity reveals that 'interaction model' 4 outperforms other models. Shown is the relativised difference between predictions from the statistical models 1-5 and the true trajectory, as well as between the decomposition and the true trajectory, for data generated in a simulation with complete sampling. '-', '0', and '+' in the facet labels represent whether the correlation between LS and that fecundity-coefficient is -0.5, 0 or +0.5, respectively; here, the first symbol corresponds to LS-intercept, the second to LS-slope, and the third to LS-shape, correlations.

Figure 5: Mixed-effects models containing random slopes fit the data better than models with only random intercepts. Three scenarios of selective disappearance shown as facets. The Y axis shows $\Delta AICs$ (mean and ΔSE) compared to the best fitting model that contains the same random effects within each scenario. Within each scenario, models in the top and bottom panels are fit to the same data and identical in their fixed effect structures. However, models in the top row contain only a random intercept term while models in the bottom row contain a random intercept and random slope term. Numeric text printed on top of each bar represent: (First text) Absolute AIC values of a model,

which can be used to compare models with different random effects but within the same scenario (e.g. A vs D); (Second text) the exact ΔAIC of each model compared to the best fitting model with the same random effect within that scenario/panel.

Figure 6: Interaction model 4 outperforms interaction model 5 when data are missing.

Comparison of $\Delta AICs$ (mean and ΔSE) of models 1-6 to the best fitting model in each scenario, fitted on data from simulation with data missing completely at random (MCAR). For simplicity, only 9 scenarios shown but all 27 considered scenarios presented in Figures S38. Unlike the CS simulation where data are completely sampled (Figure 3), in MCAR, models 4 outperforms model 5. '-', '0', and '+' in the facet labels represent whether correlation between LS and that fecundity-coefficient is -0.5, 0 or +0.5, respectively; here, the first symbol corresponds to LS-intercept, the second to LS-slope, and the third to LS-shape, correlations. Model 6 acts as a positive control where lifespan is known, and thus modelled instead of ALR or mean age.

Figure 7: Re-analysis of two empirical studies shows that age-dependent selective disappearance manifests in real-world datasets, where model 4 provides the best fit too. (A) Model predictions for the effect of age from the five statistical models fit to a fruit fly dataset. Model 4 predicts a steeper senescence in late-life compared to other models. **(B)** Age-dependent selective disappearance can be inferred by the difference in fecundity between lifespan bins increasing with age. **(C)** In a great tit dataset, the best fitting model 4 shows that **(D)** longer- and shorter-lived individuals have distinct trajectories of age-dependent changes in their laying date.

Figure 8: A flowchart of recommendations for empiricists to (A) visually diagnose the selective disappearance pattern and (B) decide on the most appropriate model. A1 and A2 provide complementary approaches to assess the presence and type of selective disappearance. A3 provides a way to quantify the parametric approximation of the latent average within-individual ageing trajectory for any function. B1-B5 guide empiricists through modelling the random effect structure, functional ageing equation, choosing the appropriate among-individual/selective-disappearance term, and deciding which model/method to use. An identical workflow can be used to diagnose and model selective *appearance*. Researchers can implement these diagnostics on their own data using our shiny app, '*disappR*', to analyse longitudinal ageing data.

Data and code accessibility statement

The entire simulation code and HTML output, along with all associated empirical data are available at Open science framework

https://osf.io/kevnrm/overview?view_only=93eba71a58444d19b5fb5287ffbc61e4 under an anonymized link. Data and code are also available on DRYAD:

http://datadryad.org/share/LINK_NOT_FOR_PUBLICATION/VbBelPplfMn05nDG-HvCanKeMro8e3yN1guaCkP5Vpc.

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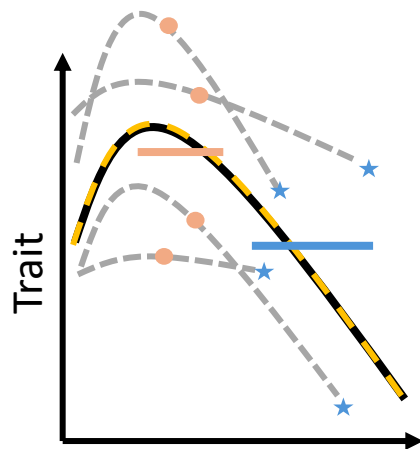
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A

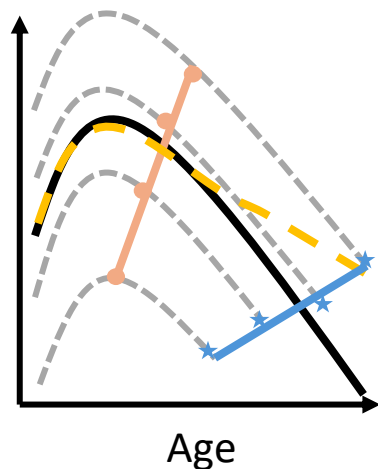
(i) Heterogeneity in intercept & ageing trajectories; no selective disappearance

No effect of mean age; effect of Δ age & age



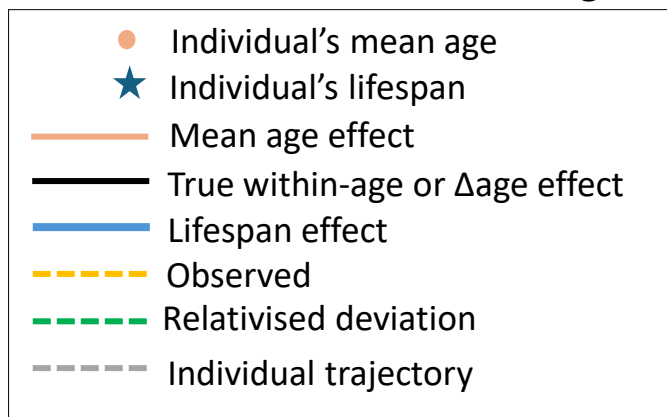
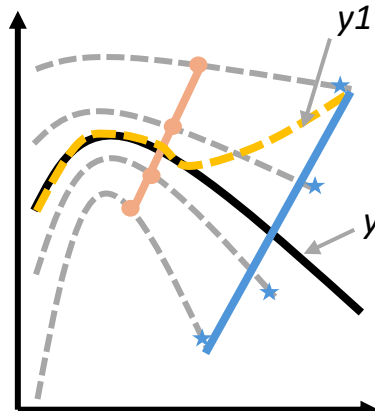
(ii) Heterogeneity in intercept but not in ageing trajectories; age-independent selective disappearance

Additive effects of mean age & Δ age; of LS & age

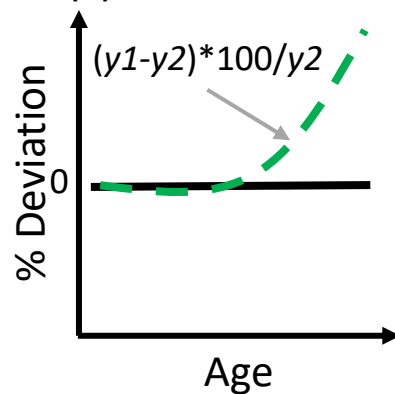


(iii) Heterogeneity in intercept & in ageing trajectories; age-dependent selective disappearance

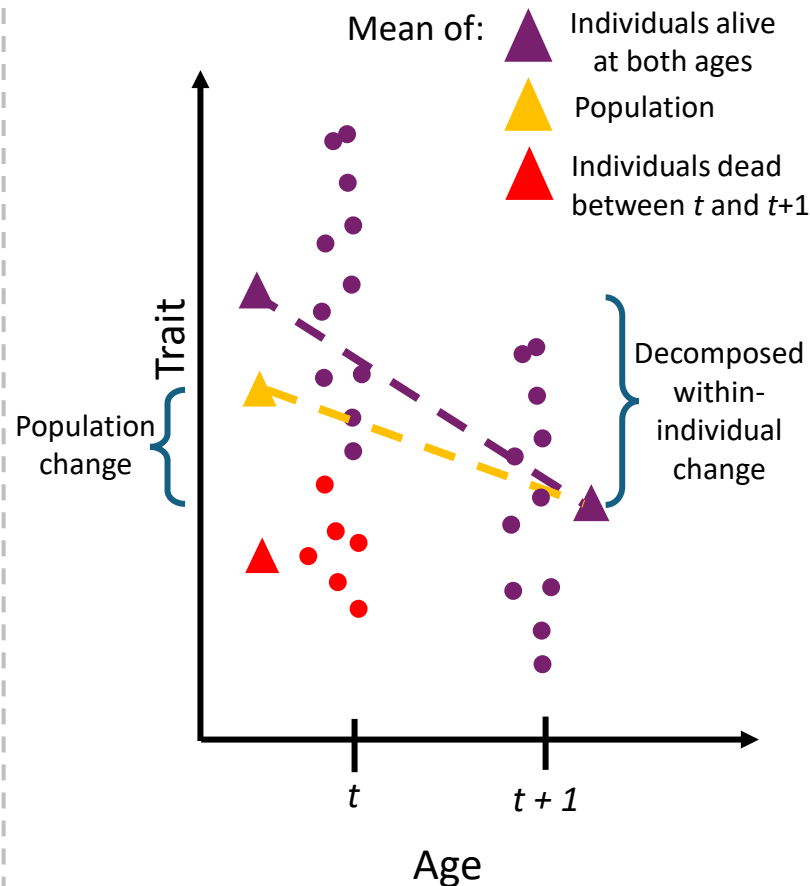
Interactive effects of mean age & Δ age; of LS & age



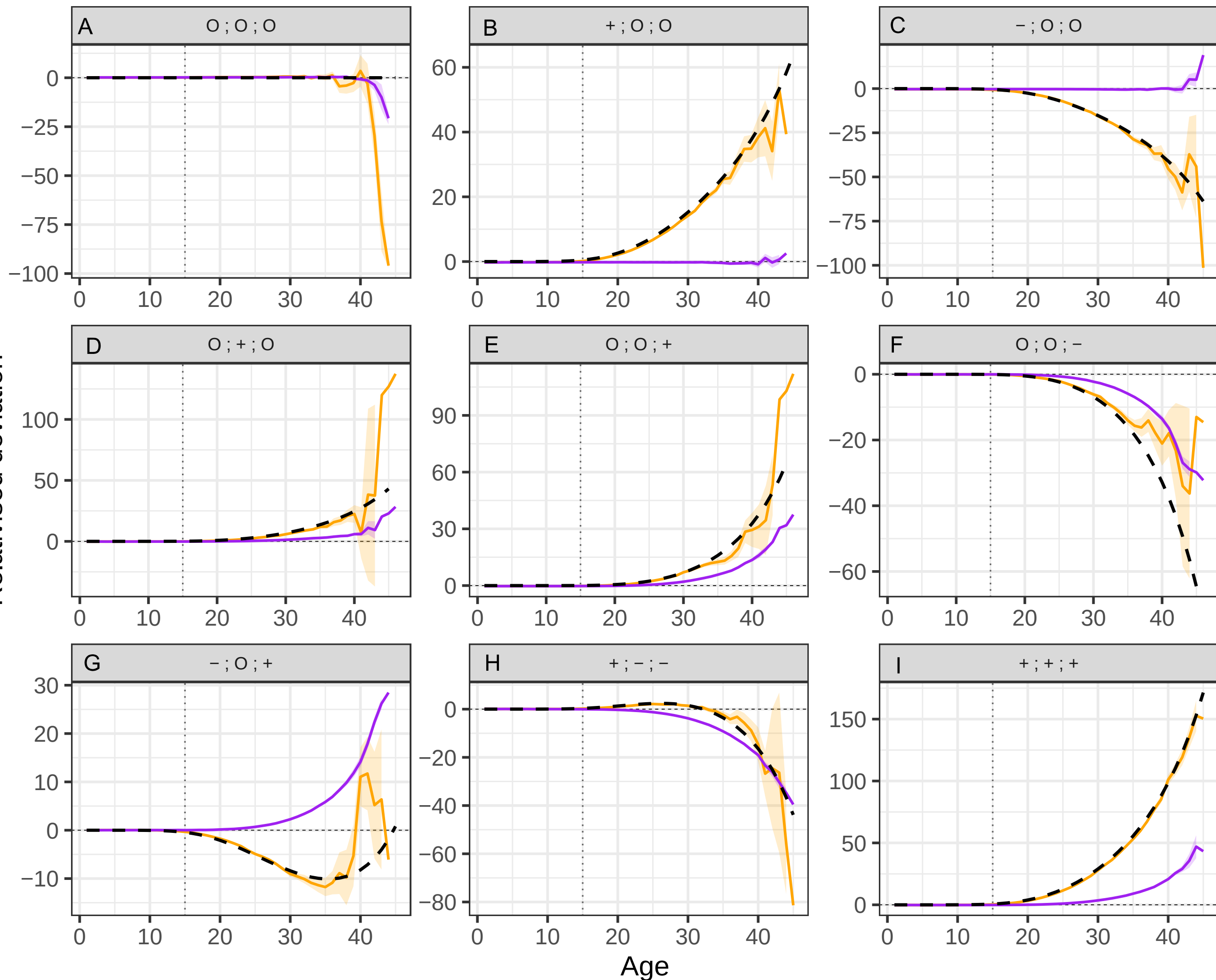
(iv) Relativised deviation



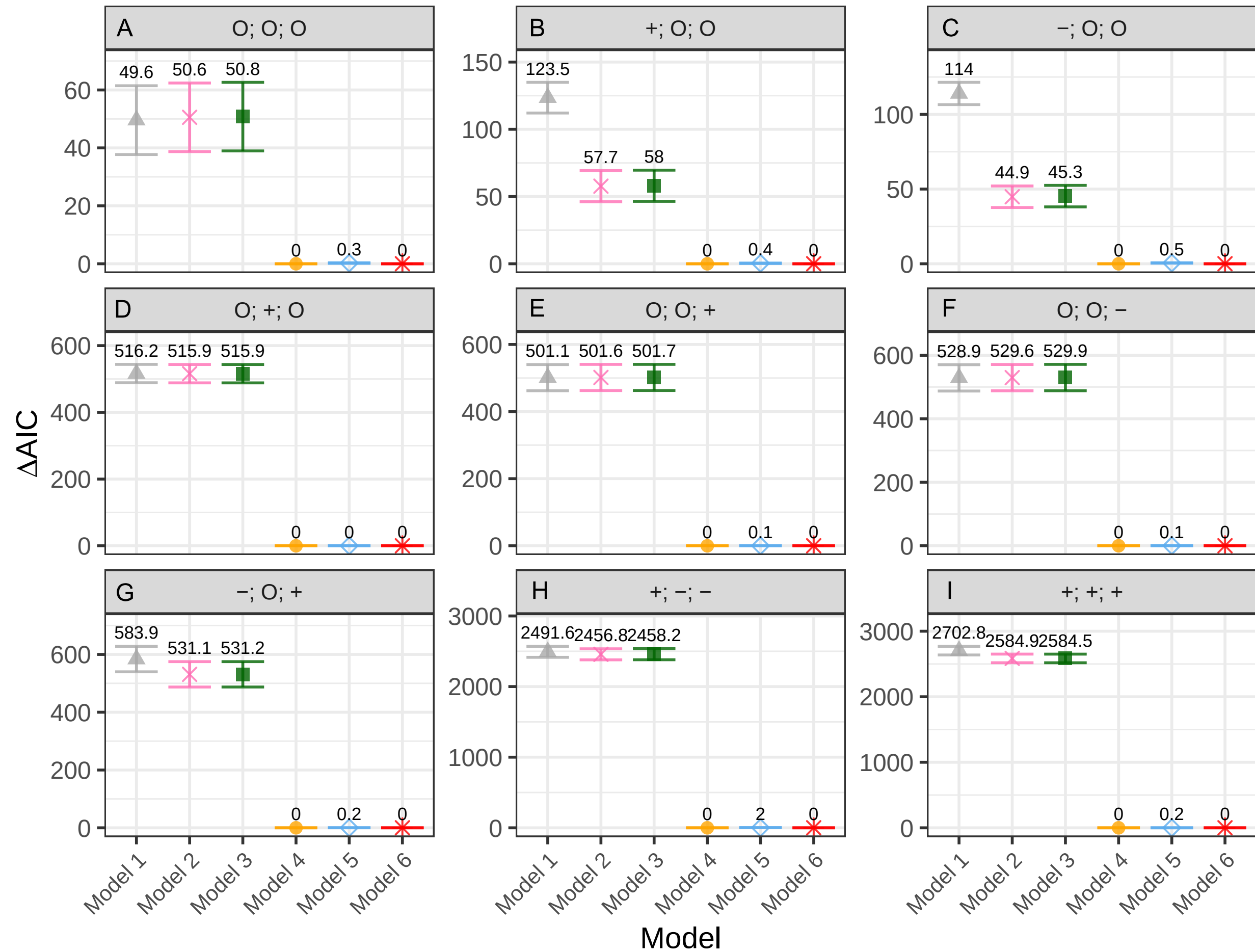
B



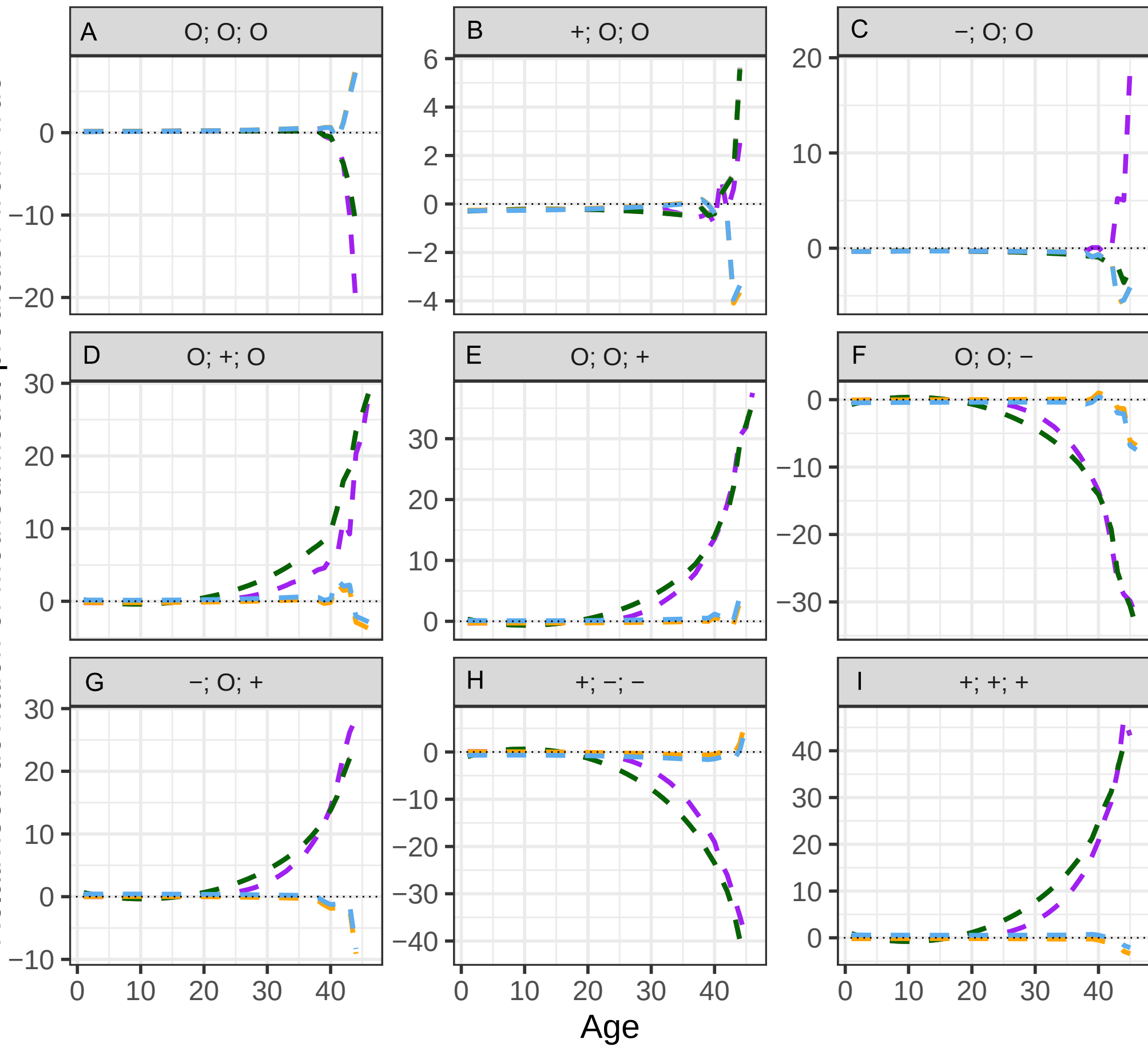
Relativised deviation



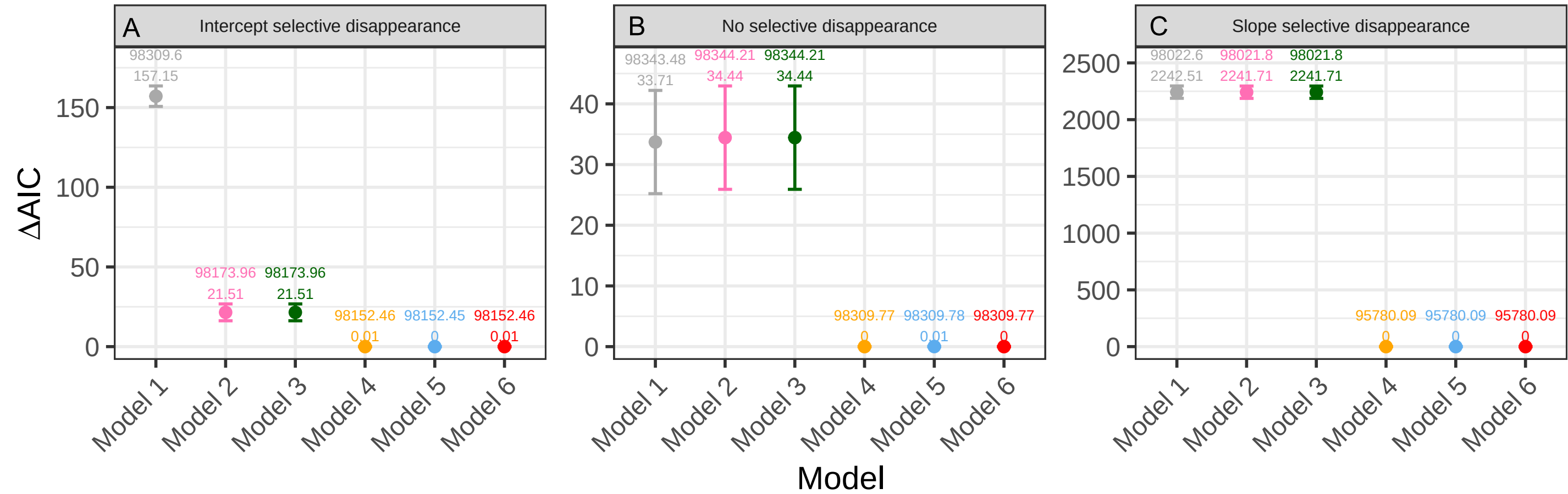
Trajectory type — Observed (complete data) — Decomposition — Analytical solution



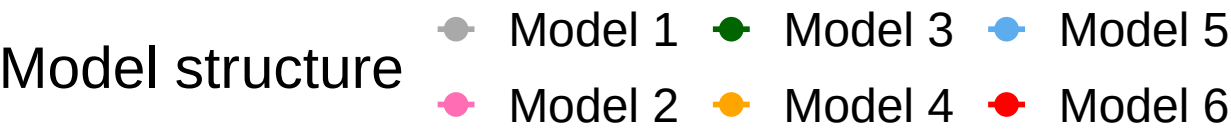
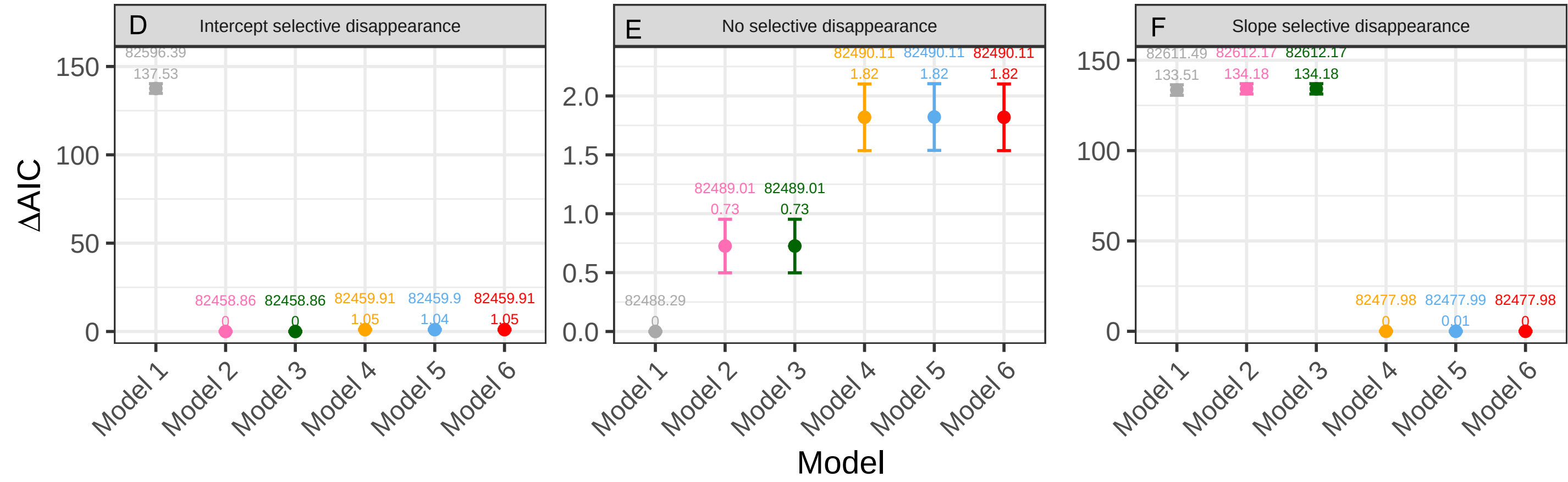
Relativised deviation of method/model prediction from True



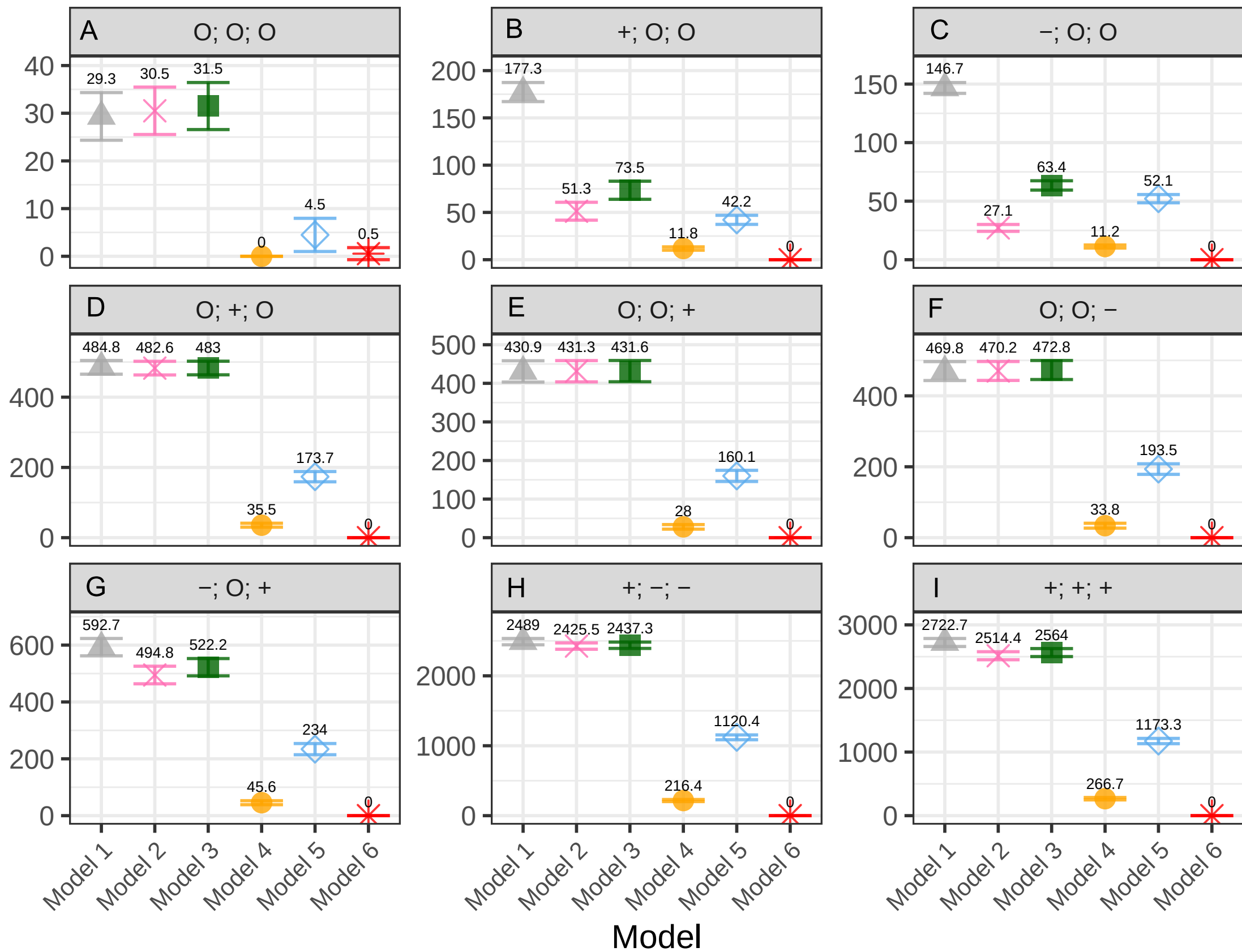
Random intercept (1|ID)



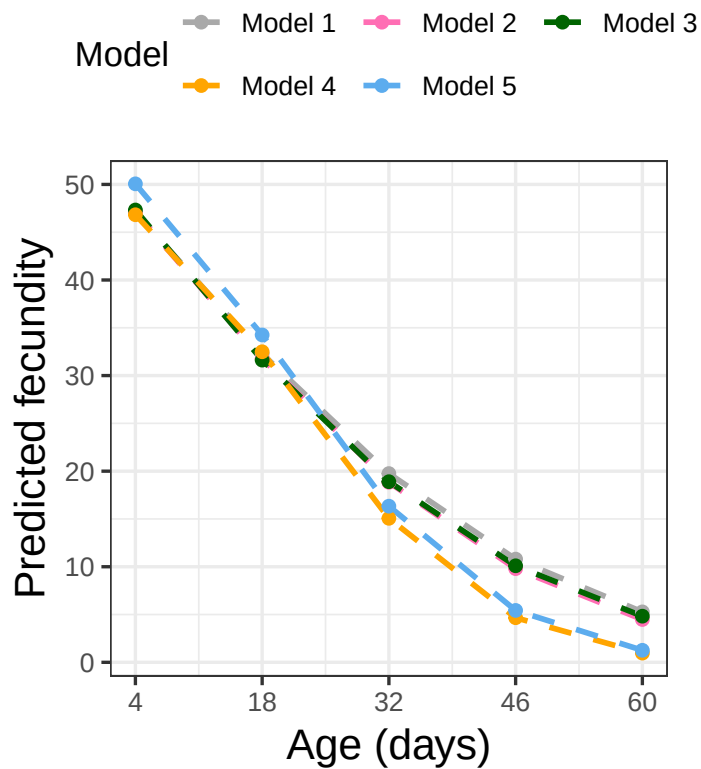
Random intercept and random slope (1|ID) + (0 + age|ID)



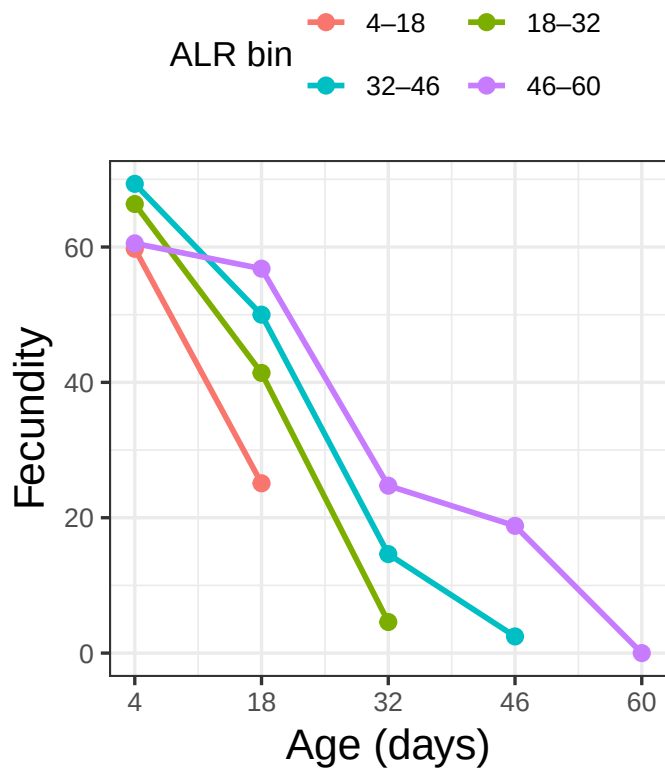
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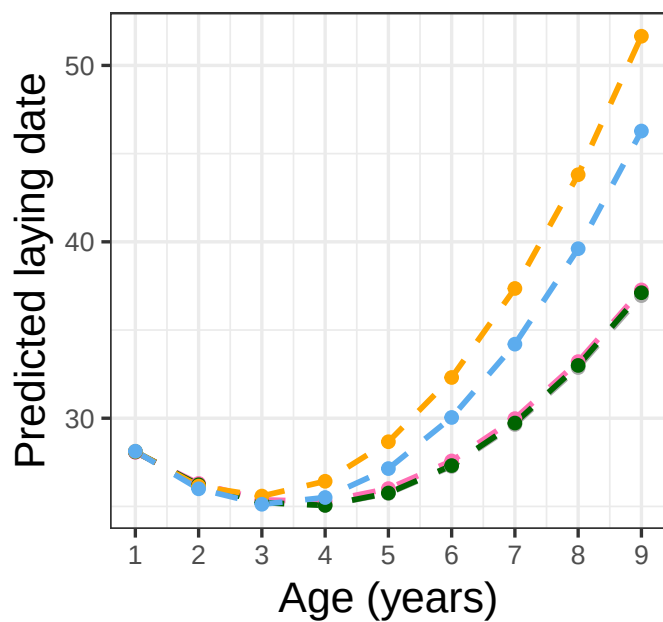
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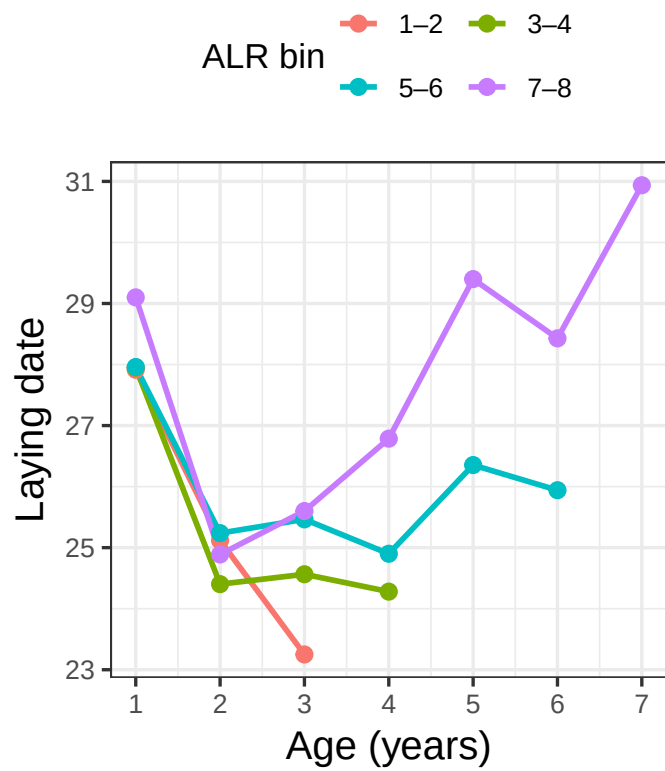
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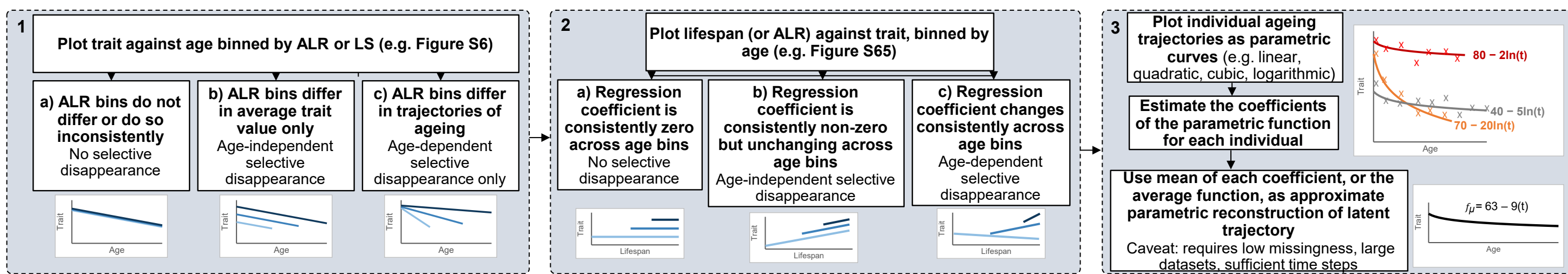
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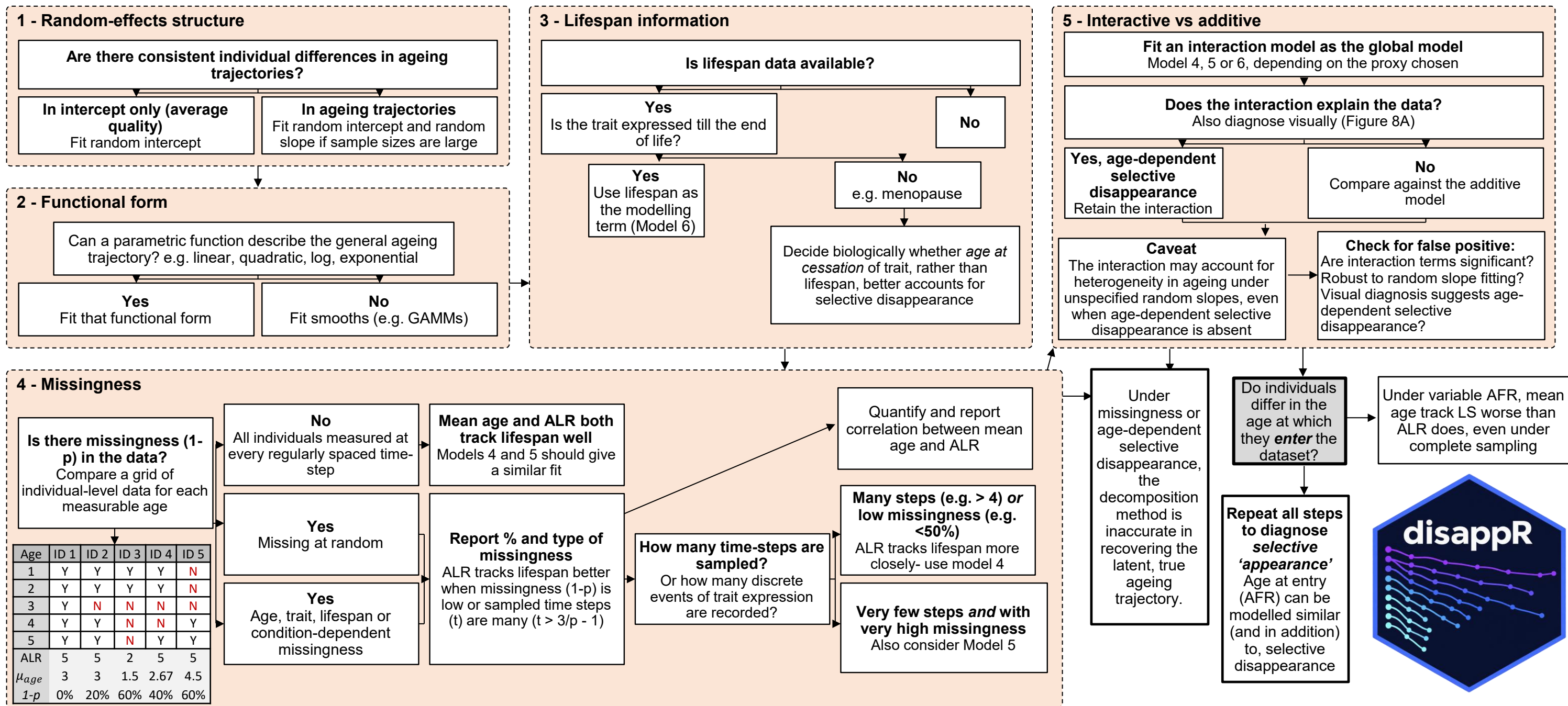
D



A



B



Supplementary sections S1-S5

1 Supplementary section S1- Simulation-specific solutions

Younger age classes often contain a mix of long- and short-lived individuals. However, old age classes, by definition, contain longer-lived individuals only. Therefore, as populations age, there is generally a bias in the longevity of the sampled population (Sanghvi et al, 2024b; Vaupel and Yashin, 1985). When researchers are interested in quantifying ageing in a trait, the observed pattern emerges not only due to effects of age on that trait directly, but also due to selection on lifespan and its association with the trait of interest- which is the mechanism that drives selective disappearance. Ecologists wish to estimate the latent, average within-individual trajectory, however only have access to the surviving cohorts age-related trait data. Throughout our study, what we imply by the ‘true’ trajectory is what ecologists wish to estimate. This ageing pattern can thus be rephrased as: “what would the population-average age-dependent trajectory look like, if the trait of all individuals were to be measured across all ages and *even beyond death*, until the maximum age in the dataset”. The true trajectory therefore utilizes an ‘immortal’ cohort (also see Li and Su, 2018; Wu et al, 2012).

Here, we derive analytical solutions to quantify: (i) how selective disappearance causes the latent within-individual (true) trajectory and the observed among-individual ageing trajectory (due to survival-dependent truncation) to diverge; (ii) how selective disappearance impacts the within-individual change calculation of the survivor-restricted decomposition method (Rebke et al, 2010); and (iii) what variables impact the rate of change of the bias caused by selective disappearance. Like in our main text, we use ‘fecundity’ as the hypothetical age-dependent trait, but our solutions are valid for any longitudinally sampled, non-survival trait.

In supplementary section S1, we provide solutions specific to our simulation under assumptions of a quadratic ageing function and normally distributed lifespans. However, in Supplementary section S2, we show how these can be generalized.

1.1 Fecundity-determining variables

For simplicity, we assume that individuals follow a quadratic trajectory of ageing, where lifespan (LS) and the three fecundity-determining random variables ($\beta_0, \beta_1, \beta_2$) are drawn from a multivariate normal distribution. Let the discrete ages at which reproduction occurs be t , such that the fecundity value at a specific discrete age, F_t , is a time-dependent linear combination of:

$$F_t = \beta_0 + \beta_1 t + \beta_2 t^2 \quad (1)$$

Because covariance is a linear operator (i.e. $Cov(X, aY + bZ) = aCov(X, Y) + bCov(X, Z)$), the covariance between LS and fecundity at age t is the sum of the covariances of the three individual random variables that collectively determine fecundity (β_0, β_1 , and β_2), and LS . This can be written as:

$$Cov(LS, F_t) = Cov(LS, \beta_0) + t \cdot Cov(LS, \beta_1) + t^2 \cdot Cov(LS, \beta_2) \quad (2)$$

The covariance between lifespan and fecundity has an age-independent component and an age-dependent component. Here, if only the $Cov(LS, \beta_0)$ term is non-zero, the association between lifespan and fecundity will remain

the same across all ages. However, if $Cov(LS, \beta_1)$ or $Cov(LS, \beta_2)$ are non-zero, the relationship between LS and the expressed trait (e.g. fecundity, F_t) will change systematically and dynamically as individuals age. Expanding, the covariance between LS and any of the other three random variables is:

$$Cov(LS, \beta_x) = \sigma_{LS, \beta_x} = \rho_{LS, \beta_x} \cdot \sigma_{LS} \cdot \sigma_{\beta_x} \quad (3)$$

1.2 Derivation of the deviation

At a given age t , we are interested in the difference between the population-level expected fecundity, $\mathbb{E}[F_t]$, and the fecundity observed in the surviving individuals. To quantify the mechanism of selective disappearance, we can model the fecundity of an individual (F_t) by adjusting for how its lifespan deviates from the population average. In a linear regression framework where both LS and F_t are treated as random variables, this relationship is expressed as:

$$F_t = \mathbb{E}[F_t] + \beta_{LS}(LS - \mu_{LS}) + \epsilon \quad (4)$$

Here, the population's baseline fecundity ($\mathbb{E}[F_t]$) is adjusted by the individual's longevity deviation ($LS - \mu_{LS}$), scaled by the regression coefficient β_{LS} . The term ϵ represents the residual variation in fecundity. When data are truncated, the conditional expectation of the outcome shifts as a function of selection on the truncated variable (see Kurland et al, 2009; Rouanet et al, 2019). Because observing fecundity at age t for an individual is conditional on the individual surviving to that age ($LS \geq t$), applying this conditional expectation gives:

$$\mathbb{E}[F_t | LS \geq t] = \mathbb{E}[F_t] + \beta_{LS} \cdot \mathbb{E}[LS - \mu_{LS} | LS \geq t] + \mathbb{E}[\epsilon | LS \geq t] \quad (5)$$

where $\mathbb{E}[F_t]$ represents the true population mean at age t ; the term $\beta_{LS} \cdot \mathbb{E}[LS - \mu_{LS} | LS \geq t]$ represents the bias introduced by the covariance between lifespan and fecundity, scaled by the shift in mean lifespan among survivors; and $\mathbb{E}[\epsilon | LS \geq t]$ is the expected value of the residuals among survivors.

Under the assumption of a multivariate normal distribution, the residuals (ϵ) are independent of the predictor (LS); consequently, $\mathbb{E}[\epsilon | LS \geq t] = \mathbb{E}[\epsilon] = 0$. For a normal distribution, the expected shift of a truncated variable can be expressed as:

$$\mathbb{E}[LS - \mu_{LS} | LS \geq t] = \sigma_{LS} \cdot \lambda(\alpha_t) \quad (6)$$

where $\lambda(\alpha_t)$ is the Inverse Mills ratio (Heckman, 1979) at the truncation point α_t . Here, $\alpha_t = (t - \mu_{LS})/\sigma_{LS}$ represents the standardized age (or Z-score- see Figure S62). Biologically, this expresses the number of standard deviations that the chronological age t is from the mean lifespan. This normalizes the mortality trajectory so that the intensity of selective disappearance depends on the cohort's relative position within the lifespan distribution rather than absolute (chronological) time. The Inverse Mills ratio at t is calculated as $\lambda(\alpha_t) = \frac{\phi(\alpha_t)}{1 - \Phi(\alpha_t)}$. Here, ϕ represents the standard-normal density evaluated at the standardized lifespan threshold, while Φ is the corresponding cumulative distribution function, representing the proportion of the population that has died by that age (Figure S62). Our assumption of normally distributed lifespans when substituting the Inverse Mills ratio has only been done for convenience and simplicity (see Supplementary section S2). For an ordinary least squares regression, the regression coefficient is expressed as $Cov(X, Y)/\sigma_X^2$. Thus we can substitute for β_{LS} as $Cov(LS, F_t)/\sigma_{LS}^2$, and further substituting Eq. 6 into Eq. 5 gives:

$$\mathbb{E}[F_t | LS \geq t] - \mathbb{E}[F_t] = \frac{Cov(LS, F_t)}{\sigma_{LS}^2} \cdot \sigma_{LS} \cdot \lambda(\alpha_t) \quad (7)$$

Here, $\mathbb{E}[F_t | LS \geq t] - \mathbb{E}[F_t]$ represents the deviation between the observed fecundity of the surviving population and the true expected fecundity, at a given age, henceforth referred to as the deflection (or deviation)- ' $D(t)$ '. By

canceling the σ_{LS} terms, we obtain:

$$D(t) = \frac{\lambda(\alpha_t)}{\sigma_{LS}} \text{Cov}(LS, F_t) \quad (8)$$

Note that in our simulation, we relativize $D(t)$ as a percentage deviation from the true trajectory. Here, $\frac{\lambda(\alpha_t)}{\sigma_{LS}}$ represents the mortality selection intensity at age t . This equation can be further rewritten by substituting for $\text{Cov}(LS, F_t)$ (from Eq. 2) as: $D(t) = \frac{\lambda(\alpha_t)}{\sigma_{LS}} (\text{Cov}(LS, \beta_0) + t \cdot \text{Cov}(LS, \beta_1) + t^2 \cdot \text{Cov}(LS, \beta_2))$. Substituting for covariances from Eq. 3, and canceling out the common σ_{LS} , we get the final solution:

$$D(t) = \lambda(\alpha_t) \cdot (\rho_{LS, \beta_0} \cdot \sigma_{\beta_0} + t \cdot \rho_{LS, \beta_1} \cdot \sigma_{\beta_1} + t^2 \cdot \rho_{LS, \beta_2} \cdot \sigma_{\beta_2}) \quad (9)$$

Equation 8 provides a crucial biological insight- that the magnitude of the deviation at age t depends on the product of two processes: (i) the selection on mortality at that age; and (ii) the total covariance between lifespan and fecundity at that age. Equation 9 further shows that: First, observed changes in fecundity with age can arise entirely independent of within-individual changes with age, i.e. when β_1 and β_2 are zero. Second, even under the absence of correlations between LS and the intercept, we can get selective disappearance due to correlations between LS , and slope or shape (also see Wu and Carroll, 1988; Wu and Bailey, 1989). Third, the bias caused by selective disappearance increases with age for two reasons: (i) because the selection on lifespan, $\lambda(\alpha_t)$, increases with age; and (ii) at later ages, the effects of $\rho_{LS, \beta_1} \cdot \sigma_{\beta_1}$ and $\rho_{LS, \beta_2} \cdot \sigma_{\beta_2}$ get multiplied by t and t^2 respectively. Due to this, even weak correlations between LS and slope or shape can lead to dramatic selective disappearance effects at later ages. Fourth, the magnitude of selective disappearance at age t scales with the among-individual variability of the fecundity parameters (σ_{β_x}). However, σ_{LS} does not appear as a multiplicative scaling factor and it cancels between the covariance and the regression coefficient when age is *chronological*. Its influence is instead entirely mediated by the standardised age $\alpha_t = (t - \mu_{LS})/\sigma_{LS}$. Consequently, two populations differing only in σ_{LS} show identical deviations at the same standardised age when selective disappearance acts through the intercept alone; when it acts through slope or shape, the population with greater lifespan variance reaches a given standardised age at a later chronological age for $\alpha_t > 0$, and therefore shows a larger deviation (Figure S60-62).

1.3 Why the decomposition method is biased

We can quantify how the decomposition method estimates within-individual change, and why this is different from the ‘true’, underlying, within-individual change across individuals. For this, let's assess the change in the trait over two discrete time steps (i.e. from t to $t + 1$). The ‘true change’ ($\Delta \mathbb{E}[F_t]$) is:

$$\Delta \mathbb{E}[F_t] = \mathbb{E}[F_{t+1}] - \mathbb{E}[F_t] \quad (10)$$

$$\begin{aligned} &= [\mu_{\beta_0} + \mu_{\beta_1}(t+1) + \mu_{\beta_2}(t+1)^2] - [\mu_{\beta_0} + \mu_{\beta_1}t + \mu_{\beta_2}t^2] \\ &= \mu_{\beta_1} + \mu_{\beta_2}(2t+1) \end{aligned} \quad (11)$$

Note that the change over one time step is independent of the mean intercept (β_0) and depends only on the mean slope (β_1) and shape (β_2). Now consider the change over this time step as estimated by the decomposition method (ΔI_t). The decomposition method estimates the within-individual change by restricting the analysis only to the cohort of individuals that survive the interval (i.e. those alive at both t and $t + 1$, conditional on $LS \geq t + 1$). Therefore:

$$\Delta I_t = \mathbb{E}[F_{t+1} \mid LS \geq t+1] - \mathbb{E}[F_t \mid LS \geq t+1] \quad (12)$$

Using the conditional expectation formula (see Eq. 5, 6, 7), we can expand both terms:

$$\begin{aligned}\Delta I_t &= \left(\mathbb{E}[F_{t+1}] + \frac{\lambda(\alpha_{t+1})}{\sigma_{LS}} \text{Cov}(LS, F_{t+1}) \right) - \left(\mathbb{E}[F_t] + \frac{\lambda(\alpha_{t+1})}{\sigma_{LS}} \text{Cov}(LS, F_t) \right) \\ &= \underbrace{(\mathbb{E}[F_{t+1}] - \mathbb{E}[F_t])}_{\text{True change}} + \underbrace{\frac{\lambda(\alpha_{t+1})}{\sigma_{LS}} [\text{Cov}(LS, F_{t+1}) - \text{Cov}(LS, F_t)]}_{\text{Bias of decomposition method}}\end{aligned}\quad (13)$$

Note that in the decomposition method, because the same set of survivors is analysed at both time points, a single selection intensity, $\lambda(\alpha_{t+1})$ applies to both terms and factors out of the difference. We can further expand and simplify the difference between the covariance terms in ‘[]’ by substituting Eq. 2, which gives us:

$$\text{Cov}(LS, F_{t+1}) - \text{Cov}(LS, F_t) = \sigma_{LS} (\rho_{LS, \beta_1} \sigma_{\beta_1} + (2t + 1) \rho_{LS, \beta_2} \sigma_{\beta_2}) \quad (14)$$

Substituting Eq. 14 back into Eq. 13 and simplifying gives us:

$$\Delta I_t = \text{‘True change’} + \underbrace{\lambda(\alpha_{t+1}) (\rho_{LS, \beta_1} \sigma_{\beta_1} + (2t + 1) \rho_{LS, \beta_2} \sigma_{\beta_2})}_{\text{Bias of decomposition method}} \quad (15)$$

Equation 15 gives us two important insights (Figure S63). First, the decomposition method’s bias is independent of the intercept (i.e. of ρ_{LS, β_0} and σ_{β_0}). This allows the decomposition method to recover an accurate estimate of the true trajectory when selective disappearance is age-independent. Second, the decomposition bias is specifically associated with selective disappearance linked to the trait’s slope and/or shape, i.e. ρ_{LS, β_1} and ρ_{LS, β_2} , and their variability (σ_{β_1} and σ_{β_2}). Here, the decomposition bias becomes exacerbated with age for two reasons: (i) selection intensity increases with age; (ii) the correlation between lifespan and shape, $\rho_{LS, \beta_2} \sigma_{\beta_2}$, is multiplied with time t . These analytical solutions provide the exact mechanism for our simulation results where under selective disappearance linked with the trait’s slope or shape, the decomposition method is inaccurate especially at mid-late ages (Figure 2D-2I); however, under selective disappearance only linked with the intercept (or no selective disappearance), it is accurate (Figure 2A-2C).

Note that the decomposition method was originally intended for only quantifying the survivor-restricted change in a trait, and for this purpose it is appropriate. However, using it to infer the latent, average within-individual ageing trajectory of the entire population leads to inaccuracy.

1.4 Observed versus decomposition

For two discrete time steps, we can calculate the observed population-level change in the trait’s value (ΔP_t) over this time step. The observed change compares the mean of *all* survivors at $t + 1$ to the mean of *all* survivors at t . Thus, unlike the decomposition method, here the pool of individuals can change between the two time steps if some individuals die:

$$\Delta P_t = \underbrace{\mathbb{E}[F_{t+1} \mid LS \geq t + 1]}_{P_{t+1}} - \underbrace{\mathbb{E}[F_t \mid LS \geq t]}_{P_t} \quad (16)$$

Note that the term, $P_{t+1} = \mathbb{E}[F_{t+1} \mid LS \geq t + 1]$, can be related to the decomposition method by rearranging and substituting Eq. 12 as:

$$P_{t+1} = \mathbb{E}[F_{t+1} \mid LS \geq t + 1] = \Delta I_t + \mathbb{E}[F_t \mid LS \geq t + 1] \quad (17)$$

Substituting this back into Eq. 16:

$$\Delta P_t = \Delta I_t + \mathbb{E}[F_t \mid LS \geq t + 1] - \mathbb{E}[F_t \mid LS \geq t] \quad (18)$$

We can further expand the two conditional expectation terms (see Eq. 5, 6, 7) as:

$$\mathbb{E}[F_t \mid LS \geq t+1] - \mathbb{E}[F_t \mid LS \geq t] = \left(\mathbb{E}[F_t] + \frac{\lambda(\alpha_{t+1})}{\sigma_{LS}} \text{Cov}(LS, F_t) \right) - \left(\mathbb{E}[F_t] + \frac{\lambda(\alpha_t)}{\sigma_{LS}} \text{Cov}(LS, F_t) \right) \quad (19)$$

Substituting this in Eq. 18 and simplifying gives us:

$$\Delta P_t = \Delta I_t + \underbrace{(\lambda(\alpha_{t+1}) - \lambda(\alpha_t))}_{\Delta\lambda \text{ (Selection acceleration)}} \cdot \frac{\text{Cov}(LS, F_t)}{\sigma_{LS}} \quad (20)$$

Eq. 20 shows that the observed population-level change in the trait's value across two discrete time steps not only includes the change as estimated by the decomposition method (i.e. ΔI_t), but also additionally includes a 'Selection acceleration' ($\Delta\lambda$) term. This term quantifies how mortality selection is changing over the time step. Under the assumed normal lifespan distribution, $\lambda(\alpha)$ increases with the truncation threshold, so $\lambda(\alpha_{t+1}) > \lambda(\alpha_t)$, which creates a 'drift' in the observed population trajectory (Figure 2).

This equation provides another crucial insight regarding the role of lifespan variability (σ_{LS}). When the term $\frac{\text{Cov}(LS, F_t)}{\sigma_{LS}}$ is expanded and simplified, σ_{LS} cancels out (as seen in Eq. 8 and 9), confirming that variability in LS does not impact the magnitude of the bias. However, σ_{LS} impacts the *rate of change* of the deviation via the selection acceleration term. This is because the standardized age is defined as $\alpha_t = (t - \mu_{LS})/\sigma_{LS}$. Consequently, the difference in the standardized age between two consecutive time steps is:

$$\alpha_{t+1} - \alpha_t = \frac{t+1 - \mu_{LS}}{\sigma_{LS}} - \frac{t - \mu_{LS}}{\sigma_{LS}} = \frac{1}{\sigma_{LS}} \quad (21)$$

Therefore, σ_{LS} contributes towards the selection acceleration term and inversely scales with the "step size" along the mortality curve (Figure S60-S62) at a given standardised age. In populations with low lifespan variability (small σ_{LS}), the step size $1/\sigma_{LS}$ will be large, thus the difference between $\lambda(\alpha_{t+1})$ and $\lambda(\alpha_t)$ will be high. This will cause a rapid acceleration in the bias (high rate of change in deviation) with age when variability in lifespan in a population is low. In contrast, populations with high variability in lifespans will experience a gradual change in the deviation over time.

1.5 Comparison of analytical and simulation results

To show that our simulations are robust and supported by our analytical solutions, we calculate how selective disappearance of intercept, slope, and/or shape, impact the deviation between the observed and the true pattern, using Eq. 8 and 9 above (Figure S13, S57-S59; Figure 2). For the entire range of simulated lifespans, these calculations can be found in the 'Analytical solution' section of our R code (and by hand in the Supplementary data: 'Analytical calculations.xlsx' uploaded on OSF https://osf.io/kevmn/overview?view_only=93eba71a58444d19b5fb5287ffbc61e4).

1.6 Relative model performance

Our data generating process (Eq. 1 in Methods) generates individual-level age-dependent fecundity. On the other hand, the data that the models evaluate is the population-level conditional expectation induced by the joint distribution of lifespan and the ageing coefficients. In our generating equation, $LS_i, \beta_{0i}, \beta_{1i}, \beta_{2i}$ are jointly sampled from the multivariate normal distribution. However, LS_i does not enter the equation used to calculate $F_{i,t}$, i.e. there is no $LS_i, t \times LS_i$, or $t^2 \times LS_i$ term in the individual-level generating equation.

Because LS covaries with the ageing coefficients, conditioning on lifespan changes the trait's expected val-

ues. Thus, terms equivalent to LS , $t \times LS$, and $t^2 \times LS$ **emerge** in the *conditional expectation* simply as a ***derived*** consequence of the truncation process, despite being absent from the individual-level data-generating equation.

The superior performance of Model 6 is because it reproduces the individual-level generating equation. Rather, the interaction terms recover a population-level conditional structure that is *induced* by the covariance between lifespan and the ageing coefficients. Lifespan truncation then determines which individuals remain observable (alive) at age t , which manifests as selective disappearance.

2 Supplementary section S2- Extended analytical solutions

2.1 General mechanism of selective disappearance

In section S1, we derived selective-disappearance's bias under some specific assumptions to match our simulations: (i) quadratic ageing functions and (ii) normally distributed lifespans. Here we provide a generalized mechanism of selective disappearance without assumptions about the trait-ageing functions or mortality distributions. We start with the premise that empiricists observe survivors, so the population-level trajectory they estimate is the average of individual trajectories weighted by the probability of still being alive.

Let individual i have its trait's (e.g. fecundity's) ageing trajectory be described as the function:

$$F_i(t) = g(t, \theta_i), \quad (22)$$

where g is any function of age (t) and θ_i is a vector of individual parameters describing the ageing function, drawn for each individual from a density $f(\theta)$. For example, when ageing is quadratic, $\theta_i = (\beta_{0i}, \beta_{1i}, \beta_{2i})$ and g is a second-degree polynomial; when ageing is a logarithmic function, $\theta_i = (\beta_{0i}, \beta_{1i})$ and $g(t, \theta_i) = \beta_{0i} + \beta_{1i} \log(t)$.

At the cohort level, we can write $F_t = g(t, \theta)$ with $\theta \sim f(\theta)$, so the true trajectory is:

$$\mathbb{E}[F_t] = \int g(t, \theta) f(\theta) d\theta. \quad (23)$$

This is the average ageing trajectory across individuals in the population in the absence of selective disappearance, i.e. the trajectory that would be estimated if survival were unrelated to the coefficients of any ageing function (θ). Now, let survival to age t be determined by any mortality process, and let an individual's probability of being alive at age t be conditioned on the vector of individual parameters that describe the trait-ageing function: $P(\text{alive at } t \mid \theta)$. Thus, the fraction of survivors is the individual survival probability averaged over the distribution of parameters in the cohort: $S(t) = \int P(\text{alive at } t \mid \theta) f(\theta) d\theta$, i.e. the marginal probability that a randomly drawn individual is still alive at age t .

The trajectory empiricists estimate is the trait average among individuals still alive, which is g weighted by survival and renormalised by the survivor fraction:

$$\mathbb{E}[F_t \mid \text{alive at } t] = \frac{\int g(t, \theta) P(\text{alive at } t \mid \theta) f(\theta) d\theta}{\int P(\text{alive at } t \mid \theta') f(\theta') d\theta'} = \frac{1}{S(t)} \int g(t, \theta) P(\text{alive at } t \mid \theta) f(\theta) d\theta. \quad (24)$$

Here, the numerator integrates each individual's trait value at age t , $g(t, \theta)$, but counts each individual only in proportion to how common its ageing coefficients are in the cohort, $f(\theta)$, and in proportion to its chance of still being alive at that age, $P(\text{alive at } t \mid \theta)$. Individuals less likely to survive to age t therefore contribute less to the integrated sum. The denominator, $S(t)$, is the total surviving fraction of individuals, and dividing by it converts the survival-weighted sum of trait values back into an average over the individuals alive at t .

The weighting in Eq. (24) can also be viewed as acting on the distribution of the ageing parameters rather than on individuals. Because $P(\text{alive at } t \mid \theta) f(\theta)/S(t)$ is non-negative and, by the definition of $S(t)$, integrates to one, it is itself a probability density in θ , obtained from the cohort density $f(\theta)$ such that:

$$f(\theta \mid \text{alive at } t) = \frac{P(\text{alive at } t \mid \theta) f(\theta)}{S(t)}. \quad (25)$$

Equation (25) represents the distribution, among the individuals still alive at age t , of the parameters that describe the ageing function.

The observed trajectory can therefore be written as g averaged over this survivor-reweighted density,

$$\mathbb{E}[F_t \mid \text{alive at } t] = \int g(t, \theta) f(\theta \mid \text{alive at } t) d\theta \quad (26)$$

This equation represents the general mechanism of selective disappearance: the true trajectory (Eq. (23)) is g averaged over the original cohort density $f(\theta)$; the observed trajectory (Eq. (26)) is g averaged over the conditional survivor-reweighted density $f(\theta \mid \text{alive at } t)$; and selective disappearance is the difference between the two, i.e. $D(t) \equiv \mathbb{E}[F_t \mid \text{alive at } t] - \mathbb{E}[F_t]$

For any ageing function, we can write the reweighting as a multiplicative weight on the original density, normalised by survival:

$$w_t(\theta) = \frac{P(\text{alive at } t \mid \theta)}{S(t)}, \quad \mathbb{E}_\theta[w_t(\theta)] = 1, \quad (27)$$

where $w_t(\theta)$ measures how over- or under-represented an individual with parameters θ is among survivors relative to the cohort: $w_t > 1$ for parameter combinations that survive better than average, $w_t < 1$ for those that survive worse, and $w_t = 1$ throughout when survival is unrelated to θ . Substituting $f(\theta \mid \text{alive at } t) = w_t(\theta) f(\theta)$ into (26), the observed trajectory becomes the cohort average of the product $g w_t$:

$$\mathbb{E}[F_t \mid \text{alive at } t] = \int g(t, \theta) w_t(\theta) f(\theta) d\theta = \mathbb{E}_\theta[g(t, \theta) w_t(\theta)].$$

The observed and the true trajectory (Eq. (23), i.e. $\mathbb{E}[F_t] = \mathbb{E}_\theta[g]$) are now both expectations taken over the same cohort density $f(\theta)$, so their difference can be taken directly. Because the weights average to one across the cohort ($\mathbb{E}_\theta[w_t] = 1$), we can write $\mathbb{E}_\theta[g] = \mathbb{E}_\theta[g] \mathbb{E}_\theta[w_t]$ without changing its value. The deviation between the observed and true ageing trajectory at any age t (i.e. the magnitude of selective disappearance) is therefore:

$$\begin{aligned} D(t) &\equiv \mathbb{E}[F_t \mid \text{alive at } t] - \mathbb{E}[F_t] = \mathbb{E}_\theta[g w_t] - \mathbb{E}_\theta[g] \\ &= \mathbb{E}_\theta[g w_t] - \mathbb{E}_\theta[g] \mathbb{E}_\theta[w_t] \end{aligned}$$

and given $\text{Cov}(X, Y) = \mathbb{E}[XY] - \mathbb{E}[X]\mathbb{E}[Y]$:

$$D(t) = \text{Cov}_\theta(g(t, \theta), w_t(\theta)) \quad (28)$$

Eq. (28) shows that selective disappearance occurs whenever individuals' trait values at age t covary with their relative probability of having survived to t . Individuals whose parameter combinations confer better survival are over-represented among those still being measured, so if their trait values differ from those of individuals that have already died, the mean trait value of the survivors will differ from the mean of the cohort. This can be interpreted akin to a selection differential, i.e. the trait mean of individuals present at t minus the mean of the entire cohort. The sign and magnitude of the bias at any age are set entirely by the sign and strength of that covariance. Because Eq. (28) is a covariance between two arbitrary functions of θ , it does not depend on the shape of the ageing function g or on the distribution of ageing coefficients $f(\theta)$.

Importantly, Equation (28) holds for any mortality function (e.g. Gompertz, Weibull). Mortality enters the mechanism only through the individual survival probability $P(\text{alive at } t \mid \theta) = \exp\{-\int_0^t h(u \mid \theta) du\}$, with $h(t \mid \theta)$ being the hazard and $S(t) = \mathbb{E}_\theta[P(\text{alive at } t \mid \theta)]$ being the surviving fraction of the cohort. Changing the

functional form of h changes $S(t)$, the weights $w_t(\theta)$, and hence the magnitude and age-profile of $D(t)$, but not the form of (28). What matters for selective disappearance is only whether the mortality hazard depends on θ (the coefficients that determine the trait-ageing function). If $h(u | \theta) = h(u)$ for every individual, then $w_t(\theta) \equiv 1$, thus the covariance vanishes and there is no selective disappearance at any age irrespective of the age-specific hazard.

One assumption that constrains our simulations is that the ageing function is linear in its parameters. Linear, quadratic, cubic, and logarithmic ageing functions all satisfy this assumption, which allows the covariance in Eq. 2 to be split into a sum of age-weighted covariances between lifespan and each coefficient. However, this condition fails whenever one coefficient enters as an exponent or multiplier of another. When the linear-in-parameters condition is not satisfied, two things change. First, the trajectory obtained by evaluating the ageing function at the mean coefficients no longer equals the average of individual trajectories. Second, Eq. 2, 8, 9 and 15 no longer apply, because $\text{Cov}(LS, F_t)$ cannot be expressed as a sum of age-weighted covariances with individual coefficients. Equation 28, however, continues to hold because it is a covariance between two arbitrary functions of θ and makes no assumption about how the coefficients combine. Under non-linearity in parameters, empiricists can either (i) linearize their data via a transformation, or (ii) instead of averaging *individual-level coefficients* to construct the ageing trajectory, *individual trajectories* can be averaged to reconstruct the true pattern (see Figure 8A3).

2.2 ALR vs mean age; Model 4 vs 5; centring versus not centring

Selective disappearance is a consequence of an individual's likelihood of disappearing (i.e. dying- measured as lifespan) from a population being associated with its phenotype. Model 5 separates the age effect into among- and within-individual components. Because selective disappearance is mediated by lifespan, however, the among-individual term is what carries the correction, therefore mean age and ALR can be compared directly as competing observable summaries of the same latent variable of lifespan. Specifically, both the additive/interaction models that use ALR (Models 2 and 4) and those that use an individual's mean observed age (Models 3 and 5), can be seen as models using an observable/measurable variable (either ALR or mean age respectively) as a proxy for the individual's unobserved, latent lifespan, LS - which is the underlying variable that is the phenotypic-expression and consequence of the survival function.

Under complete, regularly-spaced and sufficiently frequent sampling of a trait, mean age is the centroid of all the ages at which the individual was alive, while ALR equals lifespan because individuals are recorded until death (also see Supplementary section S2.3). Both proxies are then deterministic functions of LS ($ALR = LS$, $\overline{\text{age}} = (LS + 1)/2$), and models 4 and 5 are near-equivalent parameterizations of model 6. However, under incomplete sampling, each proxy (ALR vs mean age) carries an error. We therefore expect that the model built using the more reliable proxy better captures the lifespan-trait covariance (Eqs. (26) and (28)) and removes more of the selective-disappearance bias.

Let the sampling occasions be regularly spaced and frequent across time-steps (e.g. $\{1, 2, \dots, LS\}$) to represent the ages at which a trait is expressed and could be recorded. Here, let 'missingness' imply the failure to record the trait on an occasion at which the individual was alive and the trait is expressed. For traits that are expressed at discrete events (e.g. annual egg laying), individuals that are measured will contain data, while those not measured will have missingness (a zero trait value is treated as data, not as missingness). For continuously expressed traits (e.g. body size), a missing value implies that the alive individual was not measured on that occasion compared to other individuals who were (e.g. across the population, the sampling for body size was conducted across the lifetime of individuals and at the discrete intervals: 0.25, 0.5, 0.75, 1, 1.25, ..., 99.5, 99.75, 100 days of age- i.e. sampled every 6 hours, but not all individuals could be sampled at each of these sampling events).

Now consider a missing-completely-at-random (MCAR) sampling with per-occasion detection probability p . For an individual of lifespan LS , ALR- the last age at which the individual is observed, falls short of its true lifespan by the number of consecutive undetected occasions at the end of life, $G = LS - ALR$. Given that each occasion is undetected with probability $1 - p$, ignoring the truncation (i.e. treating G as geometric on $j = 0, 1, \dots$), gives

$$V_{ALR} = \text{Var}(LS - ALR) = \frac{1 - p}{p^2}, \quad (29)$$

The variance in the error associated with ALR as a proxy for LS is therefore independent of LS and is set by the detection rate alone. Also note that in a finite population, G cannot exceed LS , so the untruncated geometric form in Eq. (29) actually over-estimates how far ALR can fall short of lifespan and therefore over-estimates ALR's error- this is negligible for systems with many time-steps but potentially substantial for those with few (see below).

Now consider 'mean age'- the mean age at which the individual is observed. Because the individual's full set of possible, measurable, ages (e.g. $\{1, \dots, LS\}$ or $\{0.25, 0.5, 0.75, 1, \dots, LS\}$), can be treated as a discrete uniform distribution (each discrete age a equally likely to be sampled), thus $\mu_a = \frac{1}{LS} \sum_{a=1}^{LS} a = \frac{LS+1}{2}$. Because $\sum_{a=1}^{LS} a = \frac{LS(LS+1)}{2}$ and $\sum_{a=1}^{LS} a^2 = \frac{LS(LS+1)(2LS+1)}{6}$, the variance of the ages, $V(a)$, is:

$$V(a) = \frac{1}{LS} \sum_{a=1}^{LS} a^2 - \mu_a^2 = \frac{(LS+1)(2LS+1)}{6} - \left(\frac{LS+1}{2}\right)^2 = \frac{(LS+1)(LS-1)}{12} = \frac{LS^2 - 1}{12}. \quad (30)$$

where $V(a)$ is the dispersion of an individual's ages around its centroid of ages $(LS+1)/2$. To obtain the error in mean age caused by missingness, we can treat the expected number of retained ages as a sample of size $n \approx p \cdot LS$, i.e. the number of retained ages equals the probability of detection multiplied by the number of possible discrete sampling events (or the length of the lifespan when traits are sampled continuously or at regular, frequent intervals across life). Let this be drawn without replacement from the set of LS time-steps, whose variance is given by Eq. (30). The sampling variance is the population variance divided by n and multiplied by the finite-population correction $\frac{LS-n}{LS-1}$ (Cochran, 1977). With $n \approx p \cdot LS$ this correction is $\frac{LS-pLS}{LS-1} = \frac{LS(1-p)}{LS-1}$, giving:

$$V(\bar{a}) \approx \frac{(LS^2 - 1)/12}{pLS} \cdot \frac{LS(1-p)}{LS-1} = \frac{(LS-1)(LS+1)}{12pLS} \cdot \frac{LS(1-p)}{LS-1} = \frac{(LS+1)(1-p)}{12p} \quad (31)$$

where $\text{Var}(\bar{a})$ is the sampling variance in the estimated mean age under missingness with probability p , for an individual of lifespan (or measurable time-steps) LS . Note that Eq. (31) is only an approximation because the number of occasions actually retained is a random variable, $n \sim \text{Binomial}(LS, p)$. Here, for simplicity we have substituted its expectation, $\mathbb{E}[n] = p \cdot LS$, rather than averaging the conditional sampling variance over the distribution of n . Eq. (31) shows that in contrast to ALR, whose error variance is independent of LS , the error in mean age scales linearly with lifespan (or the number of possible sampling events of a trait). Biologically, missingness leads to sampling heterogeneity thus re-weights the distribution of ages such that the *sampled* mean age does not represent the *true* mean age of an individual.

Using these two equations, we can derive the conditions under which mean age is a better or worse proxy of LS than ALR. ALR tracks lifespan with slope 1 (since $ALR = LS$ under complete sampling), whereas mean age tracks it with slope $\frac{1}{2}$ (since its centroid, $(LS+1)/2$, is linear in LS with slope $\frac{1}{2}$). To make the proxies comparable, we can calculate the coefficient of determination between the proxy and latent lifespan, $R_{LS,X}^2$, to represent the proxy's reliability. Because R^2 is a ratio of variances, the proxy's slope enters squared. Writing $X = \lambda_X LS + \varepsilon_X$,

$\text{Var}(\varepsilon_X) = V_X$ and assuming ε_X is uncorrelated with LS :

$$R_{LS,X}^2 = \frac{\lambda_X^2 \sigma_{LS}^2}{\lambda_X^2 \sigma_{LS}^2 + V_X} = \frac{1}{1 + V_X / (\lambda_X^2 \sigma_{LS}^2)}. \quad (32)$$

where λ_X is the slope with which the proxy tracks latent lifespan. Because $\lambda_{ALR} = 1$ and $\lambda_{\bar{a}} = \frac{1}{2}$, evaluating Eq. (32) for each proxy gives:

$$R_{LS,ALR}^2 = \frac{\sigma_{LS}^2}{\sigma_{LS}^2 + V_{ALR}}, \quad (33)$$

and

$$R_{LS,\bar{a}}^2 = \frac{\frac{1}{4} \sigma_{LS}^2}{\frac{1}{4} \sigma_{LS}^2 + V_{\bar{a}}}. \quad (34)$$

Whenever $R_{LS,ALR}^2 > R_{LS,\bar{a}}^2$, ALR is the more reliable proxy; using Eqs. (33)–(34) this reduces to

$$V_{\bar{a}} > \frac{V_{ALR}}{4}, \quad (35)$$

the factor of four being the slope penalty ($1/\lambda_{\bar{a}}^2 = 4$). Because $V_{\bar{a}}$ depends on lifespan, we evaluate the crossover at the cohort mean lifespan $\overline{LS}$. Substituting $V_{ALR} = (1-p)/p^2$ and $V_{\bar{a}} = (\overline{LS} + 1)(1-p)/(12p)$ gives us the condition when ALR becomes a more reliable proxy of latent lifespan than mean age:

$$\boxed{\overline{LS} > \frac{3}{p} - 1} \quad (36)$$

i.e. whenever the average number of discrete time-steps over which a trait is expressed (here, $\overline{LS}$) exceeds $3/p - 1$. For instance, when, on average, 50% of time-steps have missing completely at random data ($p = 0.5$), $\overline{LS} > 5$. Similarly, for 10% missingness ($p = 0.9$), $\overline{LS} > 2.33$ - i.e. here, ALR tracks LS better than mean age when the average number of time-steps (or discrete ages where the trait is expressed or measurable) exceeds 2.33. Importantly, both variance expressions are approximations- treating G as untruncated makes Eq. (29) over-estimate ALR's error. Conversely, because the conditional sampling variance $V(\bar{a} | n) = \frac{LS+1}{12} \left(\frac{LS}{n} - 1 \right)$ is convex in n , substituting $\mathbb{E}[n] = p \cdot LS$ makes Eq. (31) a lower bound on the expected sampling variance of mean age (Jensen's inequality- see Denny 2017; Needham, 1993). Accounting for either approximation would therefore lower the threshold in Eq. (36), favouring ALR over mean age as a proxy for lifespan at still shorter average lifespans- this is something our simulations show. Specifically, even for the short-lived species with 5 time-steps average lifespan, ALR outperforms mean age as a proxy of LS.

When the condition in Eq. (36) is in favour of ALR, there are two reasons why model 5 performs worse than model 4 under missingness: (i) mean age is a noisier proxy of LS than ALR; (ii) mean age enters model 5 in two terms: the among-individual ($\overline{\text{age}}_i$) and the within-individual ($\text{age}_{it}^2 - \overline{\text{age}}_i^2$, $\text{age}_{it} - \overline{\text{age}}_i$) terms. Thus compared to model 4 where the proxy of LS (ALR) only appears in the *single* selective disappearance term, in centring models like model 5, *two* terms are impacted by the proxy's (mean age) error.

One assumption made in several of our simulations is that the age at which individuals enter the dataset (AFR) is fixed. In our study, AFR only varies in the MCAR and MWY simulations- i.e. different individuals are first recorded in the dataset at different times (here, note that we do not simulate selective appearance, i.e. AFR is uncorrelated with phenotype). Assuming a constant AFR removes this source of error entirely, because with AFR fixed at any value c , mean age is $(c + LS)/2$, an exact linear function of LS . However, if AFR varied among individuals, mean age would be further penalized. Importantly, under variable AFR, mean age would become a

worse proxy of lifespan compared to ALR *even* under complete sampling and no missingness. To demonstrate this, let individual i be observable on the window $\{AFR_i, \dots, LS_i\}$. Under complete sampling the centroid of that window is

$$\bar{a}_i = \frac{AFR_i + LS_i}{2}, \quad \text{while} \quad ALR_i = LS_i, \quad (37)$$

so ALR remains an exact function of lifespan whereas $\bar{a}$ is the midpoint of the *observation window*. Writing each proxy as $X = \lambda_X LS + \varepsilon_X$ and letting $\rho = \text{Cor}(AFR, LS)$, regression of (37) on LS gives

$$\lambda_{\bar{a}} = \frac{1}{2} \left(1 + \rho \frac{\sigma_{AFR}}{\sigma_{LS}} \right), \quad \text{Var}(\varepsilon_{\bar{a}}) = \frac{1}{4} (1 - \rho^2) \sigma_{AFR}^2, \quad (38)$$

while $\lambda_{ALR} = 1$ and $\text{Var}(\varepsilon_{ALR}) = 0$. To compare these proxies under variable AFR, we can express each error variance on the lifespan scale as $\tilde{V}_X = V_X / \lambda_X^2$. Substituting (29) and (31) gives:

$$\tilde{V}_{ALR} = \frac{1 - p}{p^2}, \quad \tilde{V}_{\bar{a}} = \frac{4V_{\bar{a}} + (1 - \rho^2) \sigma_{AFR}^2}{(1 + \rho \sigma_{AFR} / \sigma_{LS})^2}, \quad (39)$$

where $\tilde{V}_{\bar{a}}$ is now evaluated over the observation window $\{AFR_i, \dots, LS_i\}$ rather than $\{1, \dots, LS_i\}$. ALR is the more reliable proxy whenever $\tilde{V}_{ALR} < \tilde{V}_{\bar{a}}$. Under complete detection of $p = 1$, $\tilde{V}_{ALR} = 0$ but $\tilde{V}_{\bar{a}} = (1 - \rho^2) \sigma_{AFR}^2 / (1 + \rho \sigma_{AFR} / \sigma_{LS})^2 > 0$ unless $\sigma_{AFR} = 0$ (i.e. no variation in AFR) or $\rho = +1$ (i.e. AFR and LS are perfectly positively collinear). Thus, if AFR is variable, even when sampling is complete, mean age is penalized as a proxy of LS and is thus worse at accounting for the among-individual effect, compared to ALR which is not penalised under variable AFR.

2.2.1 What does the centring method do and how to correctly write it?

The centring method is simply a rewriting of ‘age_{it}’ as ‘age_{it} + ($\overline{\text{age}}_i - \overline{\text{age}}_i$)’, because the age_{it} terms algebraically sum to zero. The centring method then rearranges this expression into a within- and an among-term as: age_{it} = (age_{it} - $\overline{\text{age}}_i$) + $\overline{\text{age}}_i$.

The same partitioning can be done for the quadratic term, i.e. splitting $\text{age}_{it}^2 = \text{age}_{it}^2 + (\overline{\text{age}}_i^2 - \overline{\text{age}}_i^2)$ and then rearranging to: $\text{age}_{it}^2 = (\text{age}_{it}^2 - \overline{\text{age}}_i^2) + \overline{\text{age}}_i^2$. Here too, the quadratic $\overline{\text{age}}_i^2$ terms cancel out. There is an alternative variant as used in Eq.3 of Fay et al (2022): $\text{age}_{it}^2 = (\text{age}_{it}^2 - \overline{\text{age}}_i^2) + \overline{\text{age}}_i^2$. This centres the squared age on the square of the individual’s mean age (i.e. $\overline{\text{age}}_i^2$) rather than on its mean squared age (i.e. $\overline{\text{age}}_i^2$). Because $\overline{\text{age}}_i^2 = \overline{\text{age}}_i^2 + s_i^2$, where $s_i^2 = \frac{1}{n_i} \sum_t (\text{age}_{it} - \overline{\text{age}}_i)^2$ is the variance of individual i ’s sampled ages, its within-individual term sums to:

$$\sum_{t=1}^{n_i} (\text{age}_{it}^2 - \overline{\text{age}}_i^2) = n_i s_i^2, \quad (40)$$

Thus, averaging *then* squaring age sums exactly to zero **only** when $s_i^2 = 0$, that is, when the individual is sampled at a single age. Otherwise the within-individual term carries an among-individual component, s_i^2 , which increases with the number and spread of an individual’s sampled ages, and hence with its lifespan. To ensure that this does not occur, the **correct** zero-sum way to write the quadratic centring equation is therefore:

$$F_{it} = \beta_0 + \beta_{W1} (\text{age}_{it} - \overline{\text{age}}_i) + \beta_{W2} (\text{age}_{it}^2 - \overline{\text{age}}_i^2) + \beta_{B1} \overline{\text{age}}_i + \beta_{B2} \overline{\text{age}}_i^2 + u_{0i} + \epsilon_{it}, \quad (41)$$

and **not**:

$$F_{it} = \beta_0 + \beta_{W1} (\text{age}_{it} - \overline{\text{age}}_i) + \beta_{W2} (\text{age}_{it}^2 - \overline{\text{age}}_i^2) + \beta_{B1} \overline{\text{age}}_i + \beta_{B2} \overline{\text{age}}_i^2 + u_{0i} + \epsilon_{it}. \quad (42)$$

The first form’s method of averaging (Eq. 41) is the one we use in our study for Models 3 and 5 (although with a caveat- see below), i.e. squaring age first and *then* averaging it; rather than averaging age and *then* squaring it (as

done in Eq. 3 of Fay et al. (2022)). In the first form, each age term is centred on its own individual mean, so both within-individual terms sum to zero over every individual's records and carry *only* within-individual information, while all among-individual variation enters only through $\overline{\text{age}}_i$ and $\overline{\text{age}^2}_i$ terms. When within- and among-individual effects are equal ($\beta_{W1} = \beta_{B1}$, $\beta_{W2} = \beta_{B2}$), both the first (Eq 41) and second (Eq 42) form reduce exactly to the uncentred model, $\beta_0 + \beta_1 \text{age}_{it} + \beta_2 \text{age}_{it}^2$. But when the within and among-individual effects are different, this is not the case. Here, in the second form (Eq. 42), the quadratic within-individual term contains s_i^2 , so part of the estimated within-individual shape is identified from differences *among* individuals in how long and how often they were sampled. But this is exactly the among-individual signal that selective disappearance creates and that the method attempts to account for in the first place. Therefore using the second form (Eq. 42) of squaring the average age (rather than averaging the squared age), causes the among-individual effect to partly “leak” into the within individual term.

Biologically, however, the two forms predict very similar outcomes about how individuals are ageing. This is because their within-individual quadratic terms differ only by s_i^2 . This term is constant within each individual, thus it shifts an individual's predicted trait value by the same amount, $\beta_{W2} s_i^2$, at every age without changing the shape of its trajectory. Further, much of this is absorbed by the individual random intercept, u_{0i} . So despite Eq. 41 being the correct (zero-sum) partitioning of age, both Eq. 41, and 42 theoretically predict similar ageing trajectories.

One place where the quadratic partitioning does matter is in how it is typically used by empiricists (and in our study's model 3 and 5). In the linear case, $F_{it} = \beta_0 + \beta_W(\text{age}_{it} - \overline{\text{age}}_i) + \beta_B \overline{\text{age}}_i$, thus the partitioning is complete and zero-sum for the mean age terms because they cancel out (assuming that $\beta_B = \beta_W$). The cancellation of the $\overline{\text{age}}_i$ terms thus reduces the model to $F_{it} = \beta_0 + \beta_1 \text{age}_{it}$. However, for the quadratic partitioning, the within-individual term $\beta_{W2}(\text{age}_{it}^2 - \overline{\text{age}^2}_i)$ needs to be balanced by $\beta_{B2} \overline{\text{age}^2}_i$, so that they cancel and sum to age_{it}^2 . Biologically, the $\beta_{B2} \overline{\text{age}^2}_i$ term specifies that the among-individual effect is itself non-linear. For example, individuals with intermediate lifespans having a higher trait value than those with the shortest- and the longest lifespans. Its equivalent in Model 2 would be $\beta_4 \text{ALR}_i^2$. The alternative, linear among-term ($\overline{\text{age}}_i$), allows the trait to be only a linear function of lifespan (or mean age), for example, the longest lived individuals having a higher trait value than the intermediate-lifespan individual, who have a higher trait value than the shortest-lifespan individuals. Typically, empiricists often specify the within-term as quadratic, but then specify the among-term only as linear without including a quadratic among-individual term ($\beta_{B2} \overline{\text{age}^2}_i$), i.e. as:

$$F_{it} = \beta_0 + \beta_{W1}(\text{age}_{it} - \overline{\text{age}}_i) + \beta_{W2}(\text{age}_{it}^2 - \overline{\text{age}^2}_i) + \beta_{B1} \overline{\text{age}}_i. \quad (43)$$

Here, even though the correct way to average the term (Eq.41) is used (i.e. square age then average, rather than average age then square), because an among-individual quadratic term is not specified, the equation does not sum back to $\beta_0 + \beta_1 \text{age}_{it} + \beta_2 \text{age}_{it}^2$. While algebraically, the partitioning is therefore incomplete, this is not necessarily an incorrect way to model the data. Here (Eq. 43), because the within-individual terms still sum to zero within each individual, the missing among-individual quadratic component is constant within individuals and is absorbed by the random intercept, u_{0i} . It therefore leaves the within-individual estimates, and hence the estimated ageing trajectory, unchanged, and marginally penalizes only some model fit. Moreover, the among-individual quadratic term is rarely ever included by empiricists (Nussey et al, 2008; also reviewed in Fay et al, 2022) because adding it can over-parameterise models to recover a non-linear relationship that is often biologically absent. Our study uses averaging in Eq. 41, but *without* the quadratic among-individual term (Eq. 43). This is to prevent over-parameterization due to our simulated lifespan effects being linear, and because this is the more commonly fit, more pragmatic, and biologically appropriate centring model. However, our app, *disappR*, allows the quadratic among-individual term to be additionally added.

2.2.2 Relative ageing:

There is another conceptually, biologically, and mathematically distinct form to write the mean-centring equation (see Eq. 4 of Fay et al, 2022), as:

$$F_{it} = \beta_0 + \beta_{W1}(\text{age}_{it} - \overline{\text{age}}_i) + \beta_{W2}(\text{age}_{it} - \overline{\text{age}}_i)^2 + \beta_{B1}\overline{\text{age}}_i + \beta_{B2}\overline{\text{age}}_i^2 + u_{0i} + \epsilon_{it}. \quad (44)$$

In contrast to the two forms (Eq. 41, 42) above, in which age and $\overline{\text{age}}$ are squared *separately* and then subtracted, Eq. 44 instead first subtracts the individual's mean age from its age and then squares the difference, i.e. $(\text{age}_{it} - \overline{\text{age}}_i)^2$. Expanding the squared term gives: ' $\text{age}_{it}^2 - 2\text{age}_{it}\overline{\text{age}}_i + \overline{\text{age}}_i^2$ ', which contains an extra term, $-2\text{age}_{it}\overline{\text{age}}_i$. This term implicitly specifies an interaction between the within- and among-individual processes, but its coefficient is not estimated freely. It is fixed at $-2\beta_{W2}$ but also tied to the within-individual quadratic effect. As shown in 2.2.1, Eq. 41 cancels out into the additive model: $\beta_0 + \beta_1\text{age}_{it} + \beta_2\text{age}_{it}^2$, and serves a proxy of the omnibus model $\beta_0 + \beta_1\text{age}_{it} + \beta_2\text{age}_{it}^2 + \beta_3\text{LS}_i + \beta_4\text{LS}_i^2$ with mean age standing in for lifespan (see above). However, Eq. 44 is *not* a re-parameterisation nor a biologically equivalent model because the extra ' $-2\text{age}_{it}\overline{\text{age}}_i$ ' is not canceled out.

Biologically, Eq. 44 assumes that ageing is a *relative* rather than an *absolute* (chronological) process, i.e. that an individual's trait changes with its age relative to the ages at which it happened to be sampled. It further assumes that the among-individual process shifts the timing of the within-individual process. Because the within-individual part, $\beta_{W1}(\text{age}_{it} - \overline{\text{age}}_i) + \beta_{W2}(\text{age}_{it} - \overline{\text{age}}_i)^2$, depends on age only through $\text{age}_{it} - \overline{\text{age}}_i$, every individual is assumed to follow the *same* ageing trajectory when centred on its own mean age. This seems biologically implausible for most systems, especially because mean age is not just a consequence of lifespan, but also of *sampling* schedule. Eq. 44 is therefore provides a completely different model and interpretation of ageing from Eqs. 41-43, and is not their interchangeable variant. We do not use Eq. 44 in our study and, like Fay et al (2022), suggest it only be used when ageing is expected to be relative to the timing of sampling, rather than determined by chronological age.

2.3 A note on lifespan and missingness

Because death happens at an instant and in continuous time, the *exact* true lifespan for any individual in any empirical study, is *always* latent. This is because all empirical studies measure traits in discrete time, therefore however small the time-step size between two measurements is, the trait of interest and importantly, lifespan, will always ever only be recorded/rounded to the nearest time-step. For instance, a fruit fly whose survival and fecundity are recorded every day, and which dies exactly at age ' $64d-3hr-22min-4.5466...sec$ ' will have its LS recorded simply as 65 days because the discrete measurement interval is one day. Even if the measurement/sampling interval was made finer, for instance, recorded every hour, the measured LS would be ' $64d-4hr$ '. While the continuous-time lifespan is the 'true' LS of any individual, this is not a practical definition for empiricists, and it is *not* the definition we imply in our study.

Instead, the definition and meaning of LS implied throughout our study is the more pragmatic and empirically measurable, 'discrete-time lifespan'. This is the closest approximation to true continuous-time lifespan given the size of the time-step of measurement, and is quantified on the same grid of time steps, e.g. $\{1, 2, 3, \dots, \text{LS}\}$ or $\{0.05, 0.1, 0.15, 0.2, \dots, \text{LS}\}$ or any equally spaced time-interval (see Li and Su, 2018). While time-steps in empirical studies are often *regularly spaced*, this need not be the case, e.g. a sampling grid of: $\{1, 2, 2.3, 2.7, 4.8, 7, 8.2\}$.

Discrete time lifespan is distinct from ALR (the last discrete age at which the individual was observed/ sampled/ measured/ recorded). Missingness in a discrete-time setting means a discrete *time step* at which the individual was alive (at or before discrete *LS*) but was not recorded. For example, take a great tit population whose body size is measured every 6 months. Here, some individual birds will not be recorded even at the discrete time-steps

where measurement/sampling was conducted, for instance, because they were dispersing, or abandoned their known nest box, or sampling that year itself could not be conducted because of logistic reasons. In our study, this is what we mean by missingness. Under missingness, ALR (as well as mean age) diverge from tracking discrete-LS.

Here, note that finer-grained sampling in discrete time does not protect against missingness: even if body size of great tits was measured every month instead of every 6 months, individuals absent from the monthly measurement would be considered as missing. The only thing that finer-sampling allows doing in the context of missingness, is that it (i) penalizes any single missing value relatively lesser when calculating the proxies of ALR and mean age, compared to if sampling was courser-grained in time (Table S1); and (ii) it provides a larger number of points to fit a more precise ageing function.

Where missing values occur in the lifetime of individuals determine how the proxies are impacted. Missingness at the end of life pulls *ALR* below *LS*; at the start raises the age at first record (*AFR*) but leaves *ALR* unchanged; missingness in between affects neither *ALR* or *AFR*. Mean age on the other hand, is impacted by all types of missingness- random (see S2.2 above), early-life, late-life, middle-life (Table S1). This is because mean age's accuracy depends on every time-step being equally weighted.

Typically, studies on wild animals monitor/record survival along with the trait, i.e. on the same time-grid (e.g. Soay sheep being sampled every year, and individuals who are found dead that year are recorded as dying the preceding year). When sampling of the trait and of *LS* is on the same time-grid, under complete sampling/no missingness (i.e. all individuals recorded at every time step), *ALR* and *LS* track each other exactly. However, in laboratory settings or human cohorts, survival is often monitored separately from the trait. In such cases, *LS* is measured on a finer time-grid (e.g. fecundity monitored weekly, *LS* monitored daily). Here, even when sampling is complete- i.e. no missingness for any trait data (e.g. every living individual's fecundity is recorded every week), *LS* and *ALR* are not perfectly correlated, and similarly, neither are mean age and *LS*. In such cases where *LS* data is available but not on the same time grid as trait measurement, we strongly recommend that empiricists simply use **MODEL 6** (i.e. *LS* as the interaction term), even though the age of measurement of the trait (i.e. time-step interval) is on a coarser, different time-scale. One assumption we make in our study is that time-steps track age

Table 1: Example record of a great tit alive in breeding seasons (year) 1–5 (discrete *LS* = 5) under different patterns of missingness

Missing seasons	Seasons recorded	Records	AFR	ALR	Mean age
None (complete)	1, 2, 3, 4, 5	5	1	5	3.00
Middle (season 2)	1, 3, 4, 5	4	1	5	3.25
End of life (seasons 4–5)	1, 2, 3	3	1	3	2.00
Start of life (seasons 1–2)	3, 4, 5	3	3	5	4.00
Start and end (seasons 1, 4–5)	2, 3	2	2	3	2.50

linearly and exactly. However, this might not always be the case. For instance, in post-reproductive lifespan (e.g. menopausal) species, the age at last measurement of a trait like reproductive output, would be much shorter than lifespan. Here, the value of $G = LS - ALR$ would be large. Similarly, in such a species, mean age at which the trait is expressed would not track lifespan accurately. Here, if selective disappearance is caused due to the covariance between *lifespan* and a given trait (e.g. longer-lived individuals having higher trait values), instead of the *age at which cessation of reproduction occurs*, both proxies of lifespan, i.e. *ALR* and mean age, would be biased.

2.4 What is a ‘true’ trajectory?

Throughout we define the target, or ‘true’, trajectory as the latent average within-individual trajectory of the entire cohort, i.e. the average age-dependent pattern that would be seen if every individual were followed across the entire sampled age range. This is the trajectory that ecologists typically wish to estimate; it asks: ‘What would the typical ageing pattern of the sampled population look like in the absence of any truncation bias?’. However, defining the true trajectory this way carries several implicit assumptions that empiricists are often unaware of.

First, this definition extends individuals beyond their last and before their first observed age, to ask: “if these individuals had not disappeared or appeared when they did, and all individuals’ trajectories spanned the same age range, what would the average trajectory be?”. This results in a counterfactual, immortal and omnipresent cohort. The trajectory extension further assumes that an individual’s trajectory beyond the ages at which it was observed behaves exactly as it did while it was observed. For example, consider a population whose true trajectory is to be recovered between ages 1 and 20. For an individual observed between ages 4 (entry) and 10 (death), with $f(x) = 5 + 10x - 0.6x^2$ for $x \in \{4, \dots, 10\}$, this definition assumes that the same individual function holds for $x \in \{1, 2, 3\}$ and $x \in \{11, \dots, 20\}$, which need not be true, and importantly, is latent thus unfalsifiable.

Second, the average of individual trajectories (or the average trajectory of the cohort)- which is what the ecologist wishes to recover, need not actually describe any specific individual. Where individuals vary quantitatively around a common ageing process, the mean (expected) trajectory is a good approximation of the average individual. However, where individuals differ qualitatively, for instance if some improve strongly while others deteriorate, the mean trajectory can actually produce a flat, linear function of ageing, even though no individual actually aged that way.

Third, beyond the mean lifespan, the recovered trajectory is mostly an average over individual x age combinations that were never observable. The estimand removes survival-dependent changes in cohort composition, but presents it (or is interpreted) as the *expected* phenotype of an organism that is alive at that age, which is misleading.

Fourth, extrapolation beyond observed ages can produce values that are biologically nonsensical (negatives or infinities). For instance, continuing the quadratic equation $f(x) = 5 + 10x - 0.6x^2$ that describes an individual’s ageing trajectory between ages 1 (birth) and 10 (death), predicts a negative trait value beyond age 18. In a study, if the population’s average within-individual trajectory is to be estimated beyond age 18 (e.g. if the maximum sampled age in the population is 25), this specific individual’s ageing trajectory will contribute biologically impossible trait values to the calculation of the expected, latent, trajectory.

Ecologists who account for selective processes in order to recover/infer the latent within-individual ageing trajectory should recognize that the counterfactuals these assumptions generate need to be critiqued as they are sometimes biologically meaningless. The trajectory recovered by the age terms after accounting for selective processes is **not** the only valid definition of a ‘true’ trajectory. The survivor-restricted (decomposition) or cross-sectional population ageing pattern (which does not account for selective [dis]appearance) are equally valid estimands, just answering a different question. We therefore recommend that empiricists do not treat longitudinal sampling approaches that statistically account for selective processes as the only way to infer, define, or recover the “true” ageing or senescence pattern.

2.5 References

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3 Supplementary section S3- Model, method precision

We compared the precision of the different statistical models and the decomposition method. This was done to test whether the more complex models, 4 and 5, were less precise (i.e. higher variance in predicted fecundity between replicates) due to overfitting or having a low ‘sample size:fixed effects’ ratio. To quantify this, we calculated a ‘relativized imprecision’ metric for each model and the decomposition method, separately for each scenario. First, for every age within a specific scenario, we calculated the standard deviation of the predicted fecundity values across the $N = 50$ replicates as:

$$\sigma_{\text{prediction,age}} = \sqrt{\frac{\sum_{r=1}^N (\hat{P}_{\text{rep,age}} - \bar{F}_{\text{age}})^2}{N - 1}} \quad (45)$$

where $\hat{P}_{\text{rep,age}}$ is the predicted value of fecundity for a replicate at a given age, by a model. To make the $\sigma_{\text{prediction,age}}$ comparable across models, we relativized it by the magnitude of the age effect predicted by the model. Specifically, for each replicate we calculated the range of the predicted trajectory:

$$\text{Range}_{\text{rep}} = \max(\hat{P}_{\text{rep}}) - \min(\hat{P}_{\text{rep}}) \quad (46)$$

We then averaged these ranges across the 50 replicates to obtain the mean range for that model and that scenario ($\overline{\text{Range}}$). Finally, we calculated the relativized imprecision by dividing the age-specific $\sigma_{\text{prediction,age}}$ by the mean prediction range. This provided a noise-to-signal ratio such that higher values indicated lower precision relative to the strength of the predicted biological signal. This was done as:

$$\text{Relativized imprecision}_{\text{age}} = \left(\frac{\sigma_{\text{prediction,age}}}{\overline{\text{Range}}} \right) \times 100 \quad (47)$$

We calculate relativized imprecision for the decomposition method too however, replacing the ‘‘predicted’’ fecundity value at each age by the fecundity as estimated by the decomposition method for that age and replicate. Note that these calculations were done separately for each scenario.

4 Supplementary section S4- Sensitivity analyses

We conducted various sensitivity analyses (Figure S11). All sensitivity analyses included calculations of deviation between the observed and decomposition trajectories, comparisons of model AICs and model/method predictions. Sensitivity analyses were conducted under complete sampling (i.e. no missingness) and parameterized for lifespans being sampled from normal distributions with a μ_{LS} of 25 time steps, unless mentioned otherwise.

4.1 Onset of ageing

To test whether our simulations were sensitive to how different traits were parametrized, and the pace and shape of ageing, we changed the mean values of β_1 and β_2 to reflect slow ($\mu_{\beta_1} = 9$; $\mu_{\beta_2} = -0.2$) and fast ($\mu_{\beta_1} = 4$; $\mu_{\beta_2} = -0.3$) ageing. Note that because the intercept is a constant and does not scale with age, parameterizing the intercept to any other number only rescales the trajectory.

4.2 Sample size

Larger sample sizes could have levied a lower relative penalty on the AIC calculation of more complex models. Furthermore, smaller sample sizes could lead to greater imprecision of interaction terms. To test this, instead of simulating 250 individuals, we conducted a sensitivity analysis simulating only 50 individuals, per replicate.

4.3 Few time steps (short-lived species)

To test whether our results held true when a species with only few time steps or short average, instead of parameterizing μ_{LS} as 25 time steps, we simulated this value as $\mu_{LS} = 5$. Compared to the longer-lived species that could represent an annually reproducing species that lives on average 25 years (or an hourly reproducing one that lives for 25 hours- or any other time-step size), the short lifespan species represents a relatively smaller number of time-steps over which a trait is expressed (e.g. a weekly sampled species that lives for 5 weeks on average and so on).

To keep the curvature of the ageing trajectory the same as our complete sampling simulation, we scaled the linear and quadratic coefficients by 5 and 5^2 respectively. This sensitivity analysis of five simulated time steps was done for both, the complete sampling as well as the missing completely at random (MCAR) simulations. It was also repeated with a simulation where lifespan was sampled from a Gamma distribution (see section 4.4 below).

4.4 Gamma distribution of lifespan

Instead of sampling lifespan from a normal distribution, we sampled it from a right skewed Gamma distribution. This was done to reflect high mortality in early life but few individuals surviving to extreme ages, as is often the case in wild populations. For the Gamma distributed simulation, we employed both, the complete sampling as well as MCAR (50% missing) simulation structure. Additionally, we conducted this simulation for both, the longer-lived species ($\mu_{LS} = 25$ time steps) and for the shorter-lived species ($\mu_{LS} = 5$ time steps).

To create correlations between normally distributed fecundity variables and Gamma distributed LS, we employed a Gaussian copula method, which separates the simulation of the correlation structure from the simulation of the marginal distributions. The Gamma distribution is parameterized by a shape (k) and a scale (s). We first defined these variables as we would for a normal distribution, i.e. based on the mean (μ_{LS}) and standard deviation (σ_{LS}). We then converted these parameters to scale and shape using the method of moments:

$$k = \frac{\mu_{LS}^2}{\sigma_{LS}^2} \quad \text{and} \quad s = \frac{\sigma_{LS}^2}{\mu_{LS}} \quad (48)$$

We simulated an intermediate set of four traits from a multivariate normal distribution using the same mean vector μ and variance-covariance matrix Σ as described in our methods. Next, we transformed each of the four correlated normal variates into uniformly distributed variates on the interval [0, 1]. This was done using the Probability Integral Transform (PIT), by applying the standard normal cumulative distribution function (`pnorm` in R) to each simulated value. This preserved the rank correlation (copula) from the previous step while removing the specific marginal properties of the normal distribution.

Finally, we used the inverse transform sampling method to convert the correlated uniform variates into our target distributions. For lifespan, we applied the inverse cumulative distribution function (CDF) of the Gamma distribution (`qgamma` in R) to the first uniform variate, using the shape and scale variables. For the three fecundity variables, we applied the inverse CDF of the Normal distribution (`qnorm` in R) to the remaining three uniform variates. This final step imparted the desired marginal distribution to each trait (Gamma for lifespan, Normal for the others) while retaining the underlying rank-correlation structure.

We conducted this sensitivity analysis for two parameterizations of μ_{LS} as either 25 (many time-steps) or 5 (few time-steps) and to create a long tail and right-skew, σ_{LS} was set to 0.4 of the mean. Importantly, to prevent negative values for fecundity at extreme ages, the μ_{β_0} was parametrized to be 2500 instead of 600.

4.5 Full covariance matrix

In nature, the coefficients that define an ageing function can themselves co-vary with one another. For instance, consider a quadratic ageing function. Here, individuals with higher intercepts (i.e. higher average quality when age is grand mean centered) might also have more positive slopes (i.e. show greater improvement in that trait in early-life), but consequently suffer a faster decline in that trait in late-life (i.e. more negative shape coefficient) (e.g. Spagopoulou et al, 2020). In our main simulations, we set the covariances among the intercept, slope, and shape to zero to isolate the effect of selective disappearance. To show that our results were invariant to this data structure, we conducted a sensitivity analysis where we relaxed this assumption. Here, we retained the entire structure of our complete sampling (CS) simulation, but additionally specified non-zero covariances within the variance-covariance matrix, such that:

$$\Sigma = \begin{pmatrix} \sigma_{LS}^2 & \rho_{LS,\beta_0}\sigma_{LS}\sigma_{\beta_0} & \rho_{LS,\beta_1}\sigma_{LS}\sigma_{\beta_1} & \rho_{LS,\beta_2}\sigma_{LS}\sigma_{\beta_2} \\ \rho_{LS,\beta_0}\sigma_{LS}\sigma_{\beta_0} & \sigma_{\beta_0}^2 & \rho_{\beta_0,\beta_1}\sigma_{\beta_0}\sigma_{\beta_1} & \rho_{\beta_0,\beta_2}\sigma_{\beta_0}\sigma_{\beta_2} \\ \rho_{LS,\beta_1}\sigma_{LS}\sigma_{\beta_1} & \rho_{\beta_0,\beta_1}\sigma_{\beta_0}\sigma_{\beta_1} & \sigma_{\beta_1}^2 & \rho_{\beta_1,\beta_2}\sigma_{\beta_1}\sigma_{\beta_2} \\ \rho_{LS,\beta_2}\sigma_{LS}\sigma_{\beta_2} & \rho_{\beta_0,\beta_2}\sigma_{\beta_0}\sigma_{\beta_2} & \rho_{\beta_1,\beta_2}\sigma_{\beta_1}\sigma_{\beta_2} & \sigma_{\beta_2}^2 \end{pmatrix} \quad (49)$$

We compared the CS (complete sampling control- 'none') simulation, to two variance-covariance (VCV) structures with among-coefficient covariances. In the 'none' structure, all three coefficient-coefficient correlations, i.e.: $\rho_{\beta_0,\beta_1}\sigma_{\beta_0}\sigma_{\beta_1}$, $\rho_{\beta_1,\beta_2}\sigma_{\beta_1}\sigma_{\beta_2}$, $\rho_{\beta_0,\beta_2}\sigma_{\beta_0}\sigma_{\beta_2}$, were set to zero. However, in the 'moderate' VCV structure, we set $\rho_{\beta_0,\beta_1} = 0.25$, $\rho_{\beta_0,\beta_2} = 0.15$, and $\rho_{\beta_1,\beta_2} = -0.20$, representing a biologically plausible scenario in which individuals with higher initial average fecundity also improve faster in early life, but this faster early-life improvement trades off against a steeper late-life decline. In the 'strong' VCV structure, we doubled these values ($\rho_{\beta_0,\beta_1} = 0.50$, $\rho_{\beta_0,\beta_2} = 0.30$, $\rho_{\beta_1,\beta_2} = -0.40$) to represent a more extreme case. For each of the three distinct VCV matrices, we simulated all 27 scenarios of selective disappearance as described in our main methods by further iterating ρ_{LS,β_x} as either -0.5, 0, or 0.5.

We checked whether each specified correlation matrix was positive semi-definite. Matrices that were not were projected to the nearest positive-semi-definite correlation matrix using `Matrix::nearPD`. Despite this, all input and output correlation values in the VCV matrix corresponded well (see HTML R code).

4.6 Increased sampling error

The measured fecundity of an individual at a given age is determined partly by its intrinsic ageing trajectory and partly by extrinsic factors, such as stochastic environmental variation and measurement error. Because of this observation-level variation, among-individual (population) ageing trajectories often appear as parametric functions (e.g. linear, log, sigmoid, quadratic) (Jones et al, 2014), even though the ageing trajectory of any single individual's trait might be irregular and stochastic. In our simulations, we assumed that stochastic error in the trait (i.e. deviation of an individual's observed fecundity from the individual's underlying quadratic equation using the sampled random variables, that describes its ageing function) was low. We thus performed a sensitivity analysis where we allowed individual's sampled/realised trait (fecundity) value to substantially deviate from the individual's underlying quadratic ageing function (see Eq. 1 in Methods).

For this, we simulated a large error around the individual's fecundity-generating equation, such that the observed fecundity of each individual substantially deviated from the individual's fecundity-generating quadratic trajectory at any age. This allowed further separating the functional form of the modelling equation (quadratic) from the generating equation (quadratic + stochastic). Instead of adding residual error with a fixed standard deviation of 5 like in our main simulations, we scaled the error to each individual's expected fecundity such that:

$$F_{i,\text{age}}^{\text{sampled}} = \hat{F}_{i,\text{age}} + \varepsilon_{i,\text{age}}; \quad \varepsilon_{i,\text{age}} \sim \mathcal{N}\left(0, (0.3 * \hat{F}_{i,\text{age}})^2\right) \quad (50)$$

We implemented this noisier-sampling sensitivity analysis for the CS simulation as well as for the MCAR simulation.

5 Supplementary section S5- Empirical data

We [re]analysed data from two studies, to show that insights gained from our simulations were valid for empirical, real-world systems, and that the assumptions made in our simulations about the underlying data did not hinder their applicability.

5.1 *Sanghvi et al, 2025b*

We re-analysed data published by Sanghvi et al, 2025b (Am. Nat). This dataset represents an experiment conducted on a laboratory population of fruit flies, *Drosophila melanogaster*. Their aim was to test how paternal age and paternal sperm storage duration, impact the ageing trajectories of longitudinally sampled offspring. The study contained longitudinally sampled daughters' reproductive output data, collected once every 2 weeks until the offspring died. It also contained lifespans of nearly all individuals (except some that were left-censored). In their original analysis, the authors used 'model 2' to analyse the ageing of fecundity of offspring born to fathers belonging to one of four treatments. For offspring i born to a father j and at age t , they fit the model:

$$F_{tij} = \beta_0 + \beta_1 \text{age}_{tij} + \beta_2 \text{age}_{tij}^2 + \beta_3 \text{LS}_{ij} + \beta_4 \text{Paternal age}_{ij} + \beta_5 \text{Paternal storage}_{ij} + \beta_6 \text{Replicate}_{ij} + u_{0j} + u_{0ij} + \epsilon_{tij} \quad (51)$$

where F_{tij} was modeled with zero-inflated negative binomial error distribution, and LS_{ij} represented the lifespan of each offspring. The study found selective disappearance linked with the intercept in daughters, i.e. longer-lived daughters having higher fecundity on average, than shorter-lived daughters.

Our goal here was to show that even for studies that account for selective disappearance linked with the intercept using model 2, the within-individual ageing trajectory might be mis-estimated due to these studies not accounting for selective disappearance linked with the trait's shape or slope. We thus re-analysed the data on daughters' reproductive output using GLMMs (glmmTMB package) with a zero-inflated negative binomial error distribution (as in their original study). We implemented models 1-5 as described in our simulation, replacing the age terms with 'offspring age', and replacing the LS term with 'Offspring ALR', to make their models equivalent to models 1-5 in our simulation. In all 5 models, we also included the treatment and environmental variables that were originally used in their study: replicate, paternal age, and paternal sperm storage. We then compared these 5 models using AIC, and compared model predictions for the effects of age. These data can be found at open science framework https://osf.io/kevnw/overview?view_only=93eba71a58444d19b5fb5287ffbc61e4 and as Supplementary data in the file- 'Fruit fly data'. The associated analysis can be found in our code under the 'Empirical data' section.

5.2 *Bouwhuis et al, 2009*

In Bouwhuis et al, 2009, the researchers analyzed how the age of females impacts a suite of traits, such as number of recruits and clutch size. However, in their study, while they had data available on another trait- egg laying date, they did not analyse this. We obtained these data (personal communication between KS, SB, and Ben Sheldon on 18/11/2025) and analyzed them using the 5 models as in our simulation. In each model, we additionally included the four environmental variables that they included in their models for the other reported traits: immigrant status of females, nest predation pressure, year's resource quality, and density of individuals. The phenological trait of laying date was annually recorded, continuous and normally distributed, thus our models assumed a Gaussian error distribution. These data can be found at open science framework https://osf.io/kevnw/overview?view_only=93eba71a58444d19b5fb5287ffbc61e4 and as Supplementary data in the file- 'Great tits data'. The associated analysis can be found in our code under the 'Empirical data' section.

5.3 *disappR*

Our shiny app, *disappR*, accessible from the data repository, contains several other empirical datasets that users can analyse/reanalyse and explore by fitting a variety of models/methods. The app also provides comprehensive and flexible options for researchers to analyse and diagnose their own data, along with several useful diagnostics.