

Towards robust evolutionary inference with integral projection models

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1 **Abstract**

2 Integral projection models (IPMs) are extremely flexible tools for ecological and evolutionary infer-
3 ence. IPMs track the full joint distribution of phenotype in populations through time, using functions
4 describing phenotype-dependent development, inheritance, survival and fecundity. For evolutionary in-
5 ference, two important features of any model are the ability to (i) characterize relationships among traits
6 (including values of the same traits across age) within individuals, and (ii) characterize similarity between
7 individuals and their descendants. In IPM analyses, the former depends on regressions of observed trait
8 values at each age on values at the previous age (development functions), and the latter on regressions
9 of offspring values at birth on parent values as adults (inheritance functions). We show analytically that
10 development functions, characterized this way, will typically underestimate covariances of trait values
11 across ages, due to compounding of regression to the mean across projection steps. Similarly, we show
12 that inheritance, characterized this way, is inconsistent with a modern understanding of inheritance, and
13 underestimates the degree to which relatives are phenotypically similar. Additionally, we show that the
14 use of a constant biometric inheritance function, particularly with a constant intercept, is incompatible
15 with evolution. Consequently, we should expect current constructions of IPMs to predict little or no
16 phenotypic evolution, purely as artifacts of their construction. We present alternative approaches to
17 constructing development and inheritance functions, based on a quantitative genetic approach, and show
18 analytically and by empirical example, using a population of bighorn sheep, how they can potentially
19 recover patterns that are critical to evolutionary inference.

20
21 *Keywords:* integral projection models, regression to the mean, inheritance, development, body size,
22 evolutionary responses, bighorn sheep

23 Introduction

24 Evolutionary and ecological dynamics converge at the scale of generation-to-generation change in popula-
25 tions (Pelletier et al., 2009; Coulson et al., 2010). When traits cause fitness variation, the distributions of
26 those traits, weighted by fitness, necessarily changes within generations (Godfrey-Smith, 2007). If differences
27 among individuals have a genetic basis, then genetic changes will be concomitant with phenotypic changes.
28 Such genetic changes are the basis for the transmission of within-generation change due to selection, to ge-
29 netic change between populations, i.e. evolution (Lewontin, 1970; Endler, 1986). The fundamental nature of
30 this relationship between phenotypic change due to selection, and associated genetic and thus evolutionary
31 change, has motivated the development of various expressions relating selection to genetic variation and
32 evolution in quantitative terms (Lush, 1937; Robertson, 1966, 1968; Lande, 1979; Lande & Arnold, 1983;
33 Morrissey, 2014, 2015). Important recent advances in population demography, particularly the introduction
34 (Easterling et al., 2000) and popularization (e.g. Childs et al., 2003; Ellner & Rees, 2006; Coulson et al.,
35 2010; Ozgul et al., 2010; Coulson, 2012; Merow et al., 2014) of integral projection models (IPMs), can poten-
36 tially allow the construction of very flexible models of changes in phenotype, and of associated demographic
37 implications (Coulson et al., 2010).

38

39 IPMs are structured population models used to study the dynamic of populations when individuals' vital
40 rates (e.g. survival, growth, reproduction) depend on one or more continuous state variables (e.g. mass).
41 In principle, these models track the distribution of individual values of the state variables through time. To
42 achieve this, IPMs project from regression models that define the underlying vital rates as a function of the
43 state variables. In fact, four core sets of functions for vital rates have been defined, termed fundamental
44 functions or fundamental processes (Coulson et al., 2010): (i) survival, (ii) fertility, (iii) ontogenetic develop-
45 ment of focal trait conditional on surviving (development functions), and (iv) distribution of offspring trait
46 as a function of parent's (inheritance functions). The inclusion of the inheritance functions allows IPMs
47 to be used to make evolutionary inference, mainly by the estimation of biometric heritabilities and related
48 quantities (Coulson et al., 2010; Schindler et al., 2013; Traill et al., 2014; Bassar et al., 2016). As discussed
49 by Coulson et al. (2010), these four processes underly the high flexibility of IPMs and their ability to link
50 different aspects of population ecology, evolutionary biology and life history. The fundamental functions are
51 combined to compute a function called kernel, which represents all possible transitions between state values
52 through time (e.g. the probability density of transitions from size x_1 at time $t - 1$ to size x_2 at time t).
53 The product of the kernel by the number of individuals at time $t - 1$ is integrated over all possible sizes
54 to obtain the number of individuals with size x_2 at time t . In general, numerical implementation of IPMs

55 involves the construction of an iteration matrix to solve the integral. Empirical examples include the study
56 of monocarpic plant species (Childs et al., 2003; Rees et al., 2006; Ellner & Rees, 2006), Soay (Ozgul et al.,
57 2009; Childs et al., 2011) and bighorn (Traill et al., 2014) sheep, yellow-bellied marmots (Ozgul et al., 2010),
58 and Trinidadian guppies (Bassar et al., 2016).

59

60 A key aspect of the distribution of phenotypes is how traits covary at the level of individuals. Genetic and
61 phenotypic covariances among traits are key determinants of evolution (Lande, 1979). In the context of
62 IPMs, which often consider single traits (e.g., mass), age-specific values of a given trait can be thought of
63 as separate, age-specific traits, the covariances among which are key determinants of evolutionary processes.
64 For example, if selection acts only on juveniles, the influence of that selection on future generations can only
65 occur if there is covariance between trait values at juvenile ages and some aspect (genetic or phenotypic)
66 of the state of individuals at a stage when they reproduce. In most IPMs as parameterized to date (e.g.
67 Childs et al., 2003; Ellner & Rees, 2006; Coulson et al., 2010; Ozgul et al., 2010), estimating covariance
68 across ages depends on correctly estimating regressions of observed trait values at each age on trait values
69 at the previous age. In practice, as it is well known, such regressions will typically be underestimated due to
70 regression to the mean (Campbell & Kenny, 1999; Barnett et al., 2005; Kelly & Price, 2005). This statistical
71 phenomenon - unusually extreme measurements being followed by measurements that are closer to the mean
72 - is a manifestation of measurement error. Regression to the mean therefore occurs if phenotypic measure-
73 ments of predictor variables imperfectly reflect relevant biological quantities. This problem has begun to be
74 investigated in the context of IPMs (Chevin, 2015), and it is likely to be very general. It is very important
75 to note that IPMs do not imply the occurrence of regression to the mean, and the issues that we discuss
76 are related to the statistical models that are typically - but not necessarily - used in IPMs. In this article,
77 we present a theoretical analysis of development functions in IPMs in order to determine the scope of the
78 problem, and suggest possible solutions.

79

80 In age-size-structured IPMs, size-dependent transition functions of the fundamental demographic processes
81 are used to project size distribution from one age to the next, and across generations. The inheritance
82 function has been defined as an association between the phenotype of the offspring as newborns or juveniles
83 and that of the parents at the time the offspring was produced (Coulson et al., 2010; Schindler et al., 2013;
84 Traill et al., 2014; Bassar et al., 2016). Essentially, it is a cross-age parent-offspring regression, which is a
85 peculiar measure of resemblance due to inheritance. Outside of the IPM framework, the concept of biometric
86 heritability - the slope of the offspring trait regressed on the midparent's (Jacquard, 1983) - is defined by
87 comparing parent and offspring at the same age (e.g. Galton, 1886). In fact, no theory exists for the concept

88 of cross-age heritability as used in IPMs. Body size, commonly the focal trait in IPMs, is typically a dy-
89 namic trait (a trait that varies over the development) - in opposition to static - and therefore its value at a
90 certain age is the result of the accumulation of growth until that age, causing differences among individuals
91 to accumulate over the ontogeny due to environmental and genetic variation in size trajectories (Chevin,
92 2015). As genes are inherited, rather than phenotypes resulting from development, parental phenotype (as
93 an adult) is an imperfect predictor of the adult's (genetic) contribution to the phenotype of the offspring.
94 In effect, a phenotype at a given age is generally an imperfect measure of genetic value for phenotype at any
95 age. As a consequence of phenotype being used as a predictor, regression to the mean occurs and results in
96 the underestimation of resemblance between parents and their offspring, and therefore of the genetic contri-
97 bution to phenotypic change (Chevin, 2015).

98

99 In this article, we construct simple but realistic theoretical models of development and inheritance of a quan-
100 titative phenotypic trait. For both development and inheritance, we also construct models that correspond
101 to the functions normally implemented in IPMs. By comparing these two sets of models, we investigate how
102 the development and inheritance functions adopted to date in IPMs use data on size-at-age of relatives, and
103 what across-age and across-generation population structure in continuous traits they can recover. Aspects of
104 the distribution of traits through time, other than over single iteration steps (in size-dependent development
105 and inheritance functions), are normally not used to parameterise IPMs. Also, the way IPMs are typically
106 iterated means that once the population structure at time $t + 1$ is generated, the state of the population
107 at time t is discarded. Consequently, while IPMs' most important feature is tracking the distribution of
108 phenotype through time, they do not output aspects of the population structure (e.g. correlations in size
109 among ages) that allow their performance to be checked. Critically, aspects of the distribution of traits
110 across time for any inference, particularly evolutionary inference, requires that correlations of individual
111 trait values across ages, and of trait values of relatives across generations, are adequately reflected. Path
112 analysis can prove to be very useful in studying such correlations. In fact, the structure of both the develop-
113 ment functions - with its autoregressive structure - and of the biometric inheritance - with associations both
114 among different generations and among different ages - can be conveniently illustrated by a path diagram, a
115 diagram representing the causal relationships amongst a set of variables. Also, the path (or tracing) rules are
116 easily applied to obtain the correlations among variables that are not directly associated (e.g. mass at age
117 1 and mass at age 3). As such, we use path analysis to generate analytical expressions that isolate growth
118 and inheritance, providing insight into the degree to which the models of these processes typically used to
119 date allow IPMs to recover the structure of populations.

120

121 We demonstrate that current parameterizations of IPMs generally recover only a small fraction of the true
122 underlying similarity within individuals across ages (section *Development*), and a small fraction of the true
123 underlying similarity between relatives (section *Inheritance*). These patterns have severe consequences for
124 evolutionary inference with IPMs. After these two sections, we give an empirical example where we develop
125 a quantitative genetic analysis of developmental trajectories in a pedigreed wild population of bighorn sheep
126 (*Ovis canadensis*) using a random regression animal model of body mass. We compare the random regression
127 analysis, which not only should be robust to regression to the mean, but also uses a modern notion of inheri-
128 tance of quantitative traits, to the inheritance function based on the cross-age parent-offspring regression and
129 standard regression methods for growth functions normally implemented into IPMs. We show a large differ-
130 ence between the two parameterizations in the ability to capture similarity within individuals across ages,
131 which results in standard regression methods normally used in IPMs not capturing the across-age structure
132 in growth. Similar conclusions are reached across generations, with most similarity among relatives being
133 missed by IPMs, corresponding to a failure of the typical IPM inheritance function to predict evolution. In
134 a final section, we discuss the results from the theoretical and empirical sections and also potential solutions
135 that may prove useful in fully realizing the potential of IPMs.
136

137 Development

138 Regression to the mean is particularly relevant to IPMs due to the method by which size-dependent growth
139 coefficients are typically - although not necessarily - estimated. Transition rates between size classes for sur-
140 viving individuals are modelled by regressing observed size at age $a + 1$ on observed size at age a , observed
141 size being therefore a predictor. Either linear models (Childs et al., 2003; Coulson et al., 2010), or extensions
142 of such models, including generalized linear or additive (mixed) models and nonlinear models (Ozgul et al.,
143 2010; Rees et al., 2014; Traill et al., 2014) have been used for this purpose. All these methods assume that
144 predictors are measured without error. When this assumption is violated, downwardly biased estimates are
145 obtained (for a review on problems and proposed models to deal with measurement error see Thompson &
146 Carter, 2007). Measurements of most traits, including size, will virtually always be made with non-trivial
147 error, for two reasons. First, limitations in the measurement process caused by different measuring conditions
148 (e.g. different levels of stomach fill when measuring the mass of a sheep), or limitations of instruments used
149 for measurement, tend to occur. Second, size, or most other variables of ecological interest, is a conceptual
150 variable, not directly measurable. As such, proxy variables that do not perfectly represent size are measured
151 instead, such as mass or some aspect of skeletal size (this issue becomes more evident if we think that the

152 heaviest sheep in a group is not necessarily the one with the longest body). Importantly, the mechanics
153 underlying IPMs neither imply measurement error nor regression to the mean. Rather, the application of
154 standard regression methods that do not account for measurement error within an autoregressive structure
155 on size (subquent sizes being used as predictors) promotes the occurrence of regression to the mean due to
156 measurement error.

157

158 Since the measurement error that causes regression to the mean is random rather than systematic, this
159 problem can be modelled by thinking of true size as a latent variable (z) that cannot be measured (e.g.
160 McArdle, 2009; Little, 2013, p. 43). In such a scenario, instead of the true values z , a proxy, x , is recorded,
161 which differs from z by a measurement error, σ_ϵ^2 , and it is related to it by a repeatability, r^2 (the square
162 of the regression coefficient of observed size at age a , x_a , on true size at the same age, z_a). x can, there-
163 fore, be written as $x = z + \sigma_\epsilon^2$. In figure 1A, we illustrate such model of the ontogenetic development
164 of size, which we named *latent true size model*, using a path diagram. In this diagram, true size at age
165 1, z_1 , determines true size at age 2, z_2 . z_2 is, then, a predictor of true size at age 3, z_3 , and so on un-
166 til size at age n , z_n , is predicted. In contrast, the kinds of regression analyses implemented to date in
167 IPMs (e.g. Childs et al., 2003; Coulson et al., 2010; Ozgul et al., 2010; Rees et al., 2014; Traill et al., 2014)
168 assume that true size z is being measured when in fact the observed variable is x . This model, which
169 we termed *observed size model*, is illustrated in Figure 1B Here the autoregressive structure in this model
170 is very similar to the one in figure 1A, but is built on observed sizes rather than true ones. We use the
171 theoretical models in figure 1 to illustrate the consequences of this conceptual mismatch and to inspect how
172 regression to the mean affects inference about development. We show that the correlations, and therefore
173 the regression coefficients, estimated using IPMs do not correspond to the true latent ones. We then derive a
174 generic analytical expression for how much correlation an IPM is able to recover given a certain measurement
175 error (repeatability) and number of projection steps (number of IPM iterations).

176

177 If we consider linear size-dependent growth functions, it is possible to express the true biometric relationships
178 (i.e. true theoretical expressions) among traits (z , e.g. size at different ages), as well as the relationships
179 captured by standard regression methods typically used in IPMs to describe development, using the princi-
180 ples of path analysis (McArdle & McDonald, 1984). Developed by Wright (1921, 1934) for estimating causal
181 path coefficients, path analysis is a method that mathematically decomposes correlations (or covariances)
182 among the variables in a path diagram. For convenience, in the path diagrams that we show we assume
183 that all variables are standardized (mean 0 and variance of 1). In such circumstances, the expected cor-
184 relation between two variables is the product of the standardized path coefficients that link them. Some

185 notational details are worth summarizing: σ is used to denote several aspects of true covariation (covariance
186 in growth among ages), whereas σ^2 represents true variances. Variances estimated by IPMs are denoted
187 by s^2 . Since the models in figure 1 are antedependence models (or autoregressive, as the response variable
188 depends on itself at a previous time), σ_g^2 in figure 1A and s_g^2 in figure 1B correspond to variances in
189 growth (associated with the regressions of true size on true size at a previous time and observed size on
190 observed size at a previous time, respectively). Finally, b corresponds to regressions of size on size (path
191 coefficients). Following the principles of path analysis, we used a variance-covariance matrix with the vari-
192 ances in growth ($\sigma_{g_a}^2$) and errors associated with observed sizes ($\sigma_{\epsilon_a}^2$) for each age a , and a matrix with
193 path coefficients (b_{z_a} and r_a) matching figure 1A to obtain a variance-covariance matrix for sizes at different
194 ages (Appendix A.1). From this matrix, we then extracted the covariances among ages for both true and
195 observed sizes (Table B.1 in appendix B). As an example, according to the path rules, the correlation and
196 covariance in true size between ages 1 and 3 is given by $b_{z_1} \cdot b_{z_2}$ and $\sigma_{g_1}^2 \cdot b_{z_1} \cdot b_{z_2}$, respectively. Analogous
197 quantities were obtained similarly for IPMs (Table B.2 in appendix B). Since regressions of observed size
198 on observed size, b_{x_a} , are estimated from the data (rather than implied), these quantities are necessarily
199 recovered, and therefore the b_{x_a} estimated in IPMs (figure 1B) are equivalent to the analogous quantities
200 in figure 1A. In contrast, variances in growth estimated with observed sizes, $s_{g_a}^2$, do not correspond to
201 variances in growth estimated with true latent sizes, $\sigma_{g_a}^2$, nor to the measurement error associated with
202 observed sizes, $\sigma_{\epsilon_a}^2$. Consequently, since these quantities are crucial to estimate covariances in size among
203 ages, the across-age distribution of phenotype that occurs in a typically-constructed IPM does not gener-
204 ally recover the across-age distribution of either a measured aspect of phenotype (e.g. correlations in the
205 x variable across ages) or of an underlying quantity (e.g. correlations in the z variable across ages). An
206 across-age distribution of phenotype (which includes correlations among ages) is not typically tracked by
207 an IPM (e.g. Childs et al., 2003; Ellner & Rees, 2006; Coulson et al., 2010; Ozgul et al., 2010). However, an
208 IPM's utility for any ecological and evolutionary inference depends on its ability to track this distribution
209 through time. In a typical implementation, the distribution of phenotype at age $a - 1$ is discarded once the
210 distribution at age a is generated, so such correlations cannot easily be outputted and checked against data.
211 As such, we use path analysis to mimic basic IPM mechanics and to extract the across-age dynamics that are
212 not otherwise easily tracked. In contrast, an IPM can easily be interrogated for the distribution of phenotype
213 at any given time. These distributions generally closely match data (Ozgul et al., 2010; Childs et al., 2011,
214 also see fig3(a) in Chevin, 2015 for a simulation example), which is corroborated by our model: age-specific
215 variances of x are correctly recovered (the diagonals of matrices A1 and A2 in appendix A.1 coincide).

216

217 For tractability, we demonstrate that IPMs do not in general recover the across-age structure of phenotype

218 using a simplified case of the path diagram in figure 1A as the true model. Specifically, we focus on the case
219 of a static trait, as it renders the basic principles more clearly without loss of generality. For that, we assume
220 that all size-dependent growth coefficients are one ($b_{z_a} = 1, \forall a$), that the variance in true growth at age one
221 - which also corresponds to the variance in true size at age one - is one ($\sigma_{g_1}^2 = 1$) and that the subsequent
222 variances are zero ($\sigma_{g_a}^2 = 0, \forall a > 1$). Finally, all repeatabilities, r_a , and measurement errors, $\sigma_{\epsilon_a}^2$, take
223 the same value, r and $1 - r^2$, respectively. Applying the path rules and these assumptions results in the
224 particular case of all true phenotypic variances and covariances being 1 and variances and covariances for
225 phenotypic observed size being 1 and r^2 , respectively (see appendix A.1.2 for details). Standard regression
226 methods typically used in IPMs underestimate regressions for true growth in any instance where $r < 1$, by
227 a factor of r^2 . Whenever true and observed sizes differ, which is virtually every time there is an attempt
228 to measure size, instead of 1 (value set for all b_{z_a}), b_{x_a} take the value r^2 for any consecutive pair of ages
229 (both in figure 1A and 1B). As mentioned before, covariances in size across ages are in general not reported
230 when building an IPM. However, the implied covariances can be calculated using the principles of path
231 analysis (see appendix A.1.1 for the general case and appendix A.1.2 for this simplified example). Since that
232 according to the path rules of standardized variables correlations between two variables correspond to the
233 product of the path coefficients linking them, in this example correlations in size among two ages will be r^2
234 to a power equivalent to the number of links between them. As such, since $r < 1$, these correlations will be
235 underestimated. As for the covariances, these are obtained by multiplying the correlations by the variance
236 in size at age 1, which corresponds to the variance in growth at age one, s_{g_1} . We know that variances in
237 size are well recovered in IPMs (this occurs because these quantities are directly estimated from the data).
238 Therefore, in this example, s_{g_1} corresponds to one, resulting in covariances in size implied by the growth
239 functions normally implemented in IPMs being given by

$$cov_{IPM}(x_i, x_j) = (r^2)^{\Delta\prime}, \quad (1)$$

240 where $\Delta\prime$ is the number of projection steps (or path coefficients) connecting ages i and j ($j - i$).

241

242 The standardized conditions set in this simplified example are useful to illuminate how much correlation
243 between sizes at different ages the standard IPM formulation will miss. As true correlations (or covariances)
244 in size across ages were set to one, subtracting the correlation in equation (1) to that theoretical value
245 corresponds to the amount of correlation a standard regression fails to recover,

$$\text{missed correlation} = 1 - (r^2)^{\Delta\prime}. \quad (2)$$

246 We visualize the theoretical result of equation (2) in figure 4A to demonstrate that this quantity is far from
247 negligible, increasing rapidly with the number of projection steps and decreasing values of r . Many IPM
248 analyses to date have focused on long-lived organisms. In these systems, age differences (projection steps)
249 of 5 to 10 years may correspond to the gap between juvenile stages, which are often subject to the strongest
250 viability selection, and ages of greatest fecundity. Even for high repeatabilities (e.g. $r = 0.9$), correlations
251 over such age differences will be underestimated by more than 60% (Figure 4A). Curiously, all else being
252 equal, IPMs with narrower projection intervals (e.g. monthly, rather than yearly) will suffer more from
253 regression to the mean than models constructed with wider projection intervals.

254

255 Inheritance

256 The modern understanding of how genes contribute to similarity among relatives (Fisher, 1918, 1930; Wright,
257 1922, 1931) has a very different structure from the inheritance function included in IPMs (e.g. Coulson
258 *et al.*, 2010; Traill *et al.*, 2014; Bassar *et al.*, 2016). Fisher and Wright showed how Mendelian inheritance at
259 many loci influencing a trait generates the observed biometric relationships among relatives, including the
260 relationships of a quantitative character between parents and their offspring. The notion of breeding value,
261 or genetic merit, of an individual is central to the current theory of the inheritance of quantitative traits,
262 and has its roots in Fisher's (1918) and Bulmer's (1980) infinitesimal model (see Falconer, 1981; Lynch &
263 Walsh, 2014, Chapter 15). Each parent passes half of its genes and therefore half of its breeding value on
264 to the offspring. As such, the expected breeding value of offspring i , $\mathbb{E}[BV_i]$, corresponds to half the sum of
265 parental breeding values, as follows

$$\mathbb{E}[BV_i] = \frac{(BV_{m_i} + BV_{f_i})}{2}, \quad (3)$$

266 where BV_{m_i} and BV_{f_i} are the maternal and paternal breeding values, respectively. The true breeding value,
267 BV_i , follows a normal distribution,

$$BV_i \sim \mathcal{N}(\mathbb{E}[BV_i], \frac{\sigma_a^2}{2}), \quad (4)$$

268 with its expected value as mean and $\frac{\sigma_a^2}{2}$ as variance, corresponding to the variance in breeding values in
269 the absence of inbreeding, conditional on mid-parent breeding values, resulting from segregation (Bulmer,
270 1980). The variance in the breeding values divided by the phenotypic variance is defined as heritability, h^2 ,
271 a measure of evolutionary potential. The degree of resemblance between relatives provides the means for
272 distinguishing the different sources of phenotypic variation and therefore for estimating heritabilities and

273 other quantitative genetic parameters (Falconer, 1981). The simplest way of doing so is by using correlations
274 of close kin, for example, of parents and their offspring, as h^2 corresponds to the slope of the offspring
275 trait regressed on the midparent's (Lynch & Walsh, 1998, Chapter 7). In fact, Jacquard (1983) defines the
276 heritability estimated with a parent-offspring regression as a biometric heritability, as opposed to broad-
277 and narrow-sense heritabilities, for which the genetic and additive genetic variances are, respectively, explic-
278 itly estimated. Any genetic architecture, i.e. broad- and narrow-sense heritability, determines a biometric
279 relationship among kin (Lynch & Walsh, 1998, Table 7.2). In IPMs, heritabilities have been estimated us-
280 ing parent-offspring regressions. Specifically, inheritance has been defined as a regression of the phenotype
281 of the offspring as newborns or juveniles on that of the parents at the time the offspring was produced
282 (Coulson *et al.*, 2010; Schindler *et al.*, 2013; Traill *et al.*, 2014; Bassar *et al.*, 2016). In this section, we in-
283 vestigate whether this cross-age biometric notion of inheritance is compatible with what is known about
284 trait transmission across generations.

285

286 Regression to the mean may cause similarity among relatives to be missed in IPMs through two different
287 mechanisms. The first is related to how the biometric notion of heritability has been applied in IPMs. The
288 second is a result of the across-age structure of the inheritance function. The consequences of the first will
289 be more severe with increasing number of generations between two relatives ($\Delta_{\mathcal{G}}$). For example, similarities
290 between grandparents and their grandoffspring will be less affected than those of great-grandparents and
291 their great-grandoffspring. The consequences of the latter will be more severe with increasing parental age,
292 as a result of increased number of projection steps ($\Delta_{\mathcal{A}}$) connecting parents and offspring (Chevin, 2015).
293 We create the simplest possible theoretical models to isolate inheritance and illuminate the components of
294 an IPM that relate to inheritance of a dynamic trait.

295

296 **Inheritance across generations**

297 We start by addressing the consequences of regression to the mean related to the biometric concept of inher-
298 itance when it is applied across multiple generations. We defined a true model for trait transmission across
299 four generations of the same age, according to Fisher's and Wright's understanding of trait transmission
300 (Figure 2A), and a comparable model reflecting the biometric concept of inheritance typically used in IPMs
301 (Figure 2B). As for the development models, we used path diagrams and path analysis to compare the cor-
302 relations implied by both models. In figure 2A, breeding values, the underlying units that are inherited, are
303 passed on across generations (in the example, from great-grandparents to grandparents, from grandparents

304 to parents, and from these to the offspring). Since each parent passes on half its breeding value to the next
305 generation, the regression coefficient linking generations is $\frac{1}{2}$. The variance associated with the breeding val-
306 ues is $\frac{3}{4}$, which corresponds to $\frac{1}{2}$ from the other parent and $\frac{1}{4}$ from segregation. h corresponds to correlation
307 between the breeding values and phenotypic values (Falconer, 1981) and, in a standardized path analysis, to
308 the corresponding regression coefficient as well. If observed size is standardized (variance of 1), then accord-
309 ing to the path rules its exogenous variance corresponds to $1 - h^2$. Finally, if any regression was to be made
310 between the observed sizes, x , the coefficient would be half the heritability. There is a close analogy with the
311 path diagram in figure 2A and the one in figure 1A. Not only they share the same structure (instead of sizes
312 at different ages we have now sizes in different generations), but other analogies can be taken. For example,
313 as the regression coefficient of phenotype on breeding values, the (square root of the) heritability expresses
314 the reliability of the phenotype to represent the underlying genetics, which in figure 1A was represented by
315 the square root of the repeatability. In figure 2B we show a series of parent-offspring regressions based on
316 phenotype, rather than genetics. The slope of the parent-offspring regression for a single parent is known to
317 be $\frac{1}{2}h^2$ and in a standardized path analysis, the associated variance is $1 - \frac{1}{4}h^4$. Similarly, the path diagram
318 in figure 2B relates to the one in figure 1B.

319

320 With this single age set up, we can isolate the regression to the mean that occurs as a result of a purely
321 biometric approach to the inheritance function. As for the true regressions, parent-, grandparent-, great-
322 grandparent-offspring regressions are given by $\frac{1}{2}h^2$, $\frac{1}{4}h^2$, $\frac{1}{8}h^2$, respectively (Lynch & Walsh, 1998). The
323 extension for arbitrary ancestral regressions is given by

$$\beta_{\Delta_{\mathcal{A}}} = \frac{1}{2^{\Delta_{\mathcal{A}}}} h^2. \quad (5)$$

324 We used path analysis to obtain the analogous regressions that a biometric inheritance function would
325 imply. The structure of the path diagrams in figures 1B and 2B are equivalent and therefore the reasoning
326 for obtaining covariances and regressions for size presented in appendix A.1 also applies in this case. As
327 such, according to the path rules, IPMs, as parameterized to date, will estimate these regressions as

$$\beta_{\Delta_{\mathcal{A}} IPM} = \left(\frac{1}{2}h^2\right)^{\Delta_{\mathcal{A}}}. \quad (6)$$

328 As an example, tracing the regression of grandoffspring size (x_O) on grandparent size (x_{GP}) in this stan-
329 dardized path diagram (with all variances of 1) involves two paths with coefficient $\frac{1}{2}h^2$, resulting in $\frac{1}{4}(h^2)^2$.
330 Equation (6) implies that trait transmission between same-age relatives is not fully recovered when the gap
331 between generations ($\Delta_{\mathcal{A}}$), is greater than one. For ancestral regressions other than of offspring on parent to

332 be correctly recovered the heritability of this trait would have to be one, which tends not to happen in nature
333 for most ecologically interesting traits. The proportion of the true regressions recovered by the biometric
334 inheritance function is given by $h^{2(\Delta_z^{-1})}$, which is illustrated in figure 4B. For example, if a trait has a
335 heritability of 25%, the grandparent-grandoffspring regression will be estimated as $\frac{1}{4}h^4 = 0.015625$ rather
336 than their true values of $\frac{1}{4}h^2 = 0.0625$, which corresponds to only recovering 25% of the regression. This
337 value drops to 6.25% for great-grandparents and their offspring.
338

339 **Across-age inheritance functions**

340 Regarding the second mechanism by which regression to the mean affects inference with the inheritance
341 function, particularly resulting from its cross-age structure, it is important to note that although an indi-
342 vidual's genetic constitution is constant throughout its life, the genetic variants relevant at one life stage
343 need not affect other life stages. Genetic variants acting late in life may be latent early in development.
344 Such variants may be inherited and contribute to similarity among relatives, even if they do not contribute
345 to the similarity of parents, as adults, to their offspring, at young, or arbitrary, life stages. Consequently,
346 there is potential for the concept of inheritance applied to date in IPMs to neglect a major fraction of
347 the ways by which genetic variation can generate similarity among relatives (Hedrick et al., 2014; Chevin,
348 2015). Chevin (2015) illustrated this issue with numerical demonstrations. Here we formalize his findings
349 analytically in order to explore the generality of his conclusions. We examine what would happen to two
350 cohorts (parents and offspring) with two ontogenetic stages (juvenile, J , and adult, A , Figure 3). We choose
351 a simple model with only two ontogenetic stages, since extending it to include more age classes would cor-
352 respond exactly to what was described for development in the previous section. We explore two different
353 perspectives of trait transmission - first using basic quantitative genetic principles and second, a cross-age
354 biometric approach typical in IPMs. The first path diagram in figure 3 reflects the former and may be in-
355 terpreted as a simplification of Wright's famous guinea pig path diagram (Wright, 1920, 1921), whereas the
356 diagram in figure 3B illustrates a cross-age phenotypic transmission between parents and offspring normally
357 used in IPMs (e.g. Coulson et al., 2010; Traill et al., 2014; Bassar et al., 2016). We use the subscripts z ,
358 ε and e to distinguish between phenotypic variance, σ^2 , and covariance, σ , and their additive genetic and
359 environmental components, respectively. As before, we will consider linear size-dependent growth functions,
360 and additive genetic effects on juvenile size and subsequent growth, so that path analysis can be used to
361 obtain the biometric relationships among traits (true theoretical expressions), as well as the relationships
362 captured by the cross-age inheritance function implemented in IPMs (see appendix A.2 for details). First,

363 we defined true hypothetical additive genetic and environmental variance-covariance matrices for growth at
 364 age, as well as true path coefficients that match the path diagram in figure 3A. Subsequently, we used path
 365 analysis to obtain the true phenotypic variance-covariance matrix for size, a matrix that quantifies both
 366 direct and indirect effects of size at age. Finally, the slopes of the regressions of offspring size on parent size
 367 were obtained analytically from the model, corresponding to the true parent-offspring regressions for both
 368 juveniles,

$$\beta_{O_J, P_J} = \frac{1}{2} \frac{\sigma_{a_J}^2}{\sigma_{z_J}^2} = \frac{1}{2} h_J^2, \quad (7)$$

369 and adults,

$$\beta_{O_A, P_A} = \frac{1}{2} \frac{\sigma_{a_J}^2 b^2 + 2\sigma_{a_{J,A}} b + \sigma_{a_A}^2}{\sigma_{z_J}^2 b^2 + 2\sigma_{z_{J,A}} b + \sigma_{z_A}^2} = \frac{1}{2} h_A^2. \quad (8)$$

370 Two other expressions are required, as they are used in constructing IPMs, namely for the regression of adult
 371 offspring size on juvenile offspring size, or adult parent size on juvenile parent size,

$$\beta_{O_A, O_J} = \beta_{P_A, P_J} = \beta_{A, J} = \frac{\sigma_{z_J}^2 b + \sigma_{z_{J,A}}}{\sigma_{z_J}^2}, \quad (9)$$

372 which models the ontogenetic development of size, and for the regression of juvenile offspring size on adult
 373 parent size,

$$\beta_{O_J, P_A} = \frac{1}{2} \frac{\sigma_{a_J}^2 b + \sigma_{a_{J,A}}}{\sigma_{z_J}^2 b^2 + 2\sigma_{z_{J,A}} b + \sigma_{z_A}^2}, \quad (10)$$

374 which corresponds to the cross-age inheritance function.

375

376 As shown in figure 3B, typical IPMs adopt β_{O_J, P_A} (b_{inh}) as the inheritance function. We use the path rules
 377 to obtain the covariances among same-age parent and offspring that are implied by this quantity, and there-
 378 fore to obtain expressions for the same-age parent-offspring slopes, which are proven to correspond to half
 379 the heritability. In practice, we then compare the theoretical results presented above, in particular the true
 380 parent-offspring regressions in equations (7) and (8), to those that occur with the cross-age inheritance func-
 381 tion, allowing us to derive the conditions under which IPMs recover the population structure of continuous
 382 traits between parents and offspring. According to the path rules, IPM-based inference for parent-offspring
 383 regression at both juvenile and adult stages, β_{O_J, P_J} and β_{O_A, P_A} , respectively, corresponds to the product of
 384 $\beta_{J,A}$ (Equation 9) and β_{O_J, P_A} (Equation 10, see appendix A.2 for details), as follows

$$\frac{1}{2}h_{(IPM)}^2 = \beta_{O_J, P_J(IPM)} = \beta_{O_A, P_A(IPM)} = \frac{1}{2} \frac{\sigma_{z_J}^2 b + \sigma_{z_{J,A}}}{\sigma_{z_J}^2} \frac{\sigma_{a_J}^2 b + \sigma_{a_{J,A}}}{\sigma_{z_J}^2 b^2 + 2\sigma_{z_{J,A}} b + \sigma_{z_A}^2}. \quad (11)$$

385 As a result, in a two-stage case, an IPM implies the same value of the parent-offspring regression for both
386 stages, which is not the case for the true values (Equations 7 and 8). Also, and even more importantly, the
387 IPM-based inference corresponding to the expression in equation (11) does not correspond to the true values
388 for either age (Equations 7 and 8). Thus, IPMs do not, in general, recover parent-offspring regressions, or
389 more generally, IPMs as typically constructed do not track major portions of the distribution of phenotype
390 across generations.

391

392 The comparison between IPM-based inference and true values becomes more straightforward in the simplified
393 case of no covariances of growth across ontogenetic stages ($\sigma_{a_{J,A}}$, and more generally $\sigma_{z_{J,A}}$), which is assumed
394 in all IPM implementations to date for developing traits. In such circumstances, the IPM implies a parent-
395 offspring regression, for both juveniles and adults, of

$$\frac{1}{2}h_{(IPM)}^2 = \beta_{O_J, P_J(IPM)} = \beta_{O_A, P_A(IPM)} = \frac{1}{2} \frac{\sigma_{a_J}^2 b^2}{\sigma_{z_J}^2 b^2 + \sigma_{z_A}^2}, \quad (12)$$

396 which is always less than the corresponding true values. This is a best-case scenario for IPMs, as covariances
397 of growth across ages are in general not modelled when estimating size transitions in such models. Even
398 in such unrealistic conditions, a standard IPM can only recover the true parent-offspring regressions under
399 very specific conditions. According to equation (12), for parent-offspring regression in juveniles to be fully
400 recovered by a model using a cross-age biometric inheritance function, the phenotypic variance in growth,
401 $\sigma_{z_A}^2$, must be zero. When that is not the case, the proportion of regression recovered decreases with de-
402 creasing size-dependent size regression, b (Equation 7, Figure 4C). The same is valid for the parent-offspring
403 regression in adults (Equation 8, Figure 4D). These are quite narrow conditions that in general are not
404 guaranteed to occur. We obtained similar results for the case where covariance in growth exists (Appendix
405 B). Indeed, although IPMs were developed to model dynamic traits, the conditions for which IPMs are
406 guaranteed to recover parent-offspring regression, particularly the absence of variance in growth, essentially
407 constrain a dynamic trait to be static.

408

409 Parent-offspring regression with a constant intercept

410 The preceding analysis shows that regression to the mean prevents the inheritance function from capturing
411 all, or indeed most, aspects of covariance between individuals and their descendants. In language typically
412 used to describe properties of IPMs, a (cross-age) biometric inheritance function does not fully capture the
413 most important ways in which inheritance influences the dynamics of a population through time. Import-
414 tantly, however, as shown above, the biometric inheritance notion does capture the correct covariance of
415 parents and offspring. In itself, this may imply that a purely biometric notion of inheritance can be used, at
416 least in simple cases, to track some important features of a population. Nonetheless, the use of the concept
417 of biometric inheritance that is extensively recommended for IPMs (Coulson *et al.*, 2010; Coulson, 2012;
418 Rees *et al.*, 2014) does not correctly employ the concept. This recommendation is based on two misconcep-
419 tions about biometric inheritance, both of which lead to failures to characterize even the simplest aspects of
420 phenotype (e.g. the dynamic of mean phenotype). The first misconception, shown above, is the assumption
421 that theory underlying the biometric relations among kin can be applied to a non-static trait when parents
422 and offspring are of different ages. Also, that iteration of the purely phenotypic relations of parents and off-
423 spring across multiple generations can recover biometric relationships among more distant kin, e.g., arbitrary
424 ancestral regressions. The second misconception is that the biometric inheritance concept, and its known
425 relationships to quantitative genetic parameters (Lynch & Walsh, 1998, Chapter 7), implies that biometric
426 functions are constant. A constant genetic basis (e.g., an assumption that h^2 is constant over some period of
427 time) to a trait implies that the slope of the parent-offspring regression is constant. However, should a trait
428 evolve, with the associated change in mean phenotype, then the intercept of the parent-offspring regression
429 necessarily changes. If the intercept is assumed to not change, or a model is constructed where the intercept
430 cannot change, then the dynamic of mean phenotype will be highly restricted. This means that even the
431 simplest possible IPM constructed with a typical inheritance function will necessarily fail in describing the
432 evolution of mean phenotype.

433

434 As an example, consider a non-age structured population, with no class structure other than that associated
435 with some focal trait, z . We denote the mean trait value in generation g by \bar{z}_g and its heritability as h^2 .
436 Without loss of generality, we assume that during a period of equilibrium z is measured such that its mean
437 is 10. We also assumed that z is heritable ($h^2 = 0.5$) but, since there is no selection, no phenotypic change
438 is observed (Figure 5A). Suppose that the equilibrium is then disrupted and that both sexes experience
439 the same selection, which represents a change in mean phenotype for the first generation ($\Delta\bar{z}'_1$) of 1 unit
440 (Figure 5B). The offspring on mid-parent regression is then $\mathbb{E}[z_2] = \alpha + h^2 \frac{z_{1m} + z_{1f}}{2}$, where α is the inter-

441 cept and z_{1m} and z_{1f} denote maternal and paternal phenotypes, respectively. An IPM constructed using
442 this regression (appropriately handling the two sexes) yields a mean phenotype in the next generation of
443 $\bar{z}_2 = \int \alpha + h^2 \cdot z \cdot p_1(z) dz = \alpha + h^2 (\bar{z}_1 + \Delta \bar{z}'_1)$. The first expression corresponds to the integral that would be
444 solved (typically numerically) by an IPM corresponding to this example, and $p_1(z)$ is the probability density
445 function of phenotype after selection but before reproduction in generation 1. The second expression is the
446 analytical solution for this integral, made possible by assuming a linear function. Under the conditions set
447 for this example, this expression would be $\bar{z}_2 = 5 + 0.5 \cdot (10 + 1) = 10.5$. This change satisfies the breeder's
448 equation for the change in mean phenotype across generations $\bar{z}_{i+1} - \bar{z}_i = h^2 \Delta \bar{z}'$. The problem arises in the
449 next generation.

450

451 Let us suppose that selection is now relaxed, such that $\Delta \bar{z}'_2$ is zero. In the absence of selection (and drift, im-
452 migration and mutation) we expect no change in allele frequencies (Wright, 1937) and therefore no evolution.
453 Consequently, we expect no change in mean phenotype (Figure 5C). In a very simple non-age structured IPM,
454 we would use the current distribution of trait values ($\mathcal{Z} = 2$) and the same inheritance function to obtain
455 the mean phenotype in generation 3, and that would correspond to $\bar{z}_3 = \int \alpha + h^2 \cdot z \cdot p_2(z) dz = \alpha + h^2 (\bar{z}_2)$,
456 which in this case would be 10.25 (Figure 5D). In this example, an IPM would predict the trait moving
457 back 0.25 phenotype units, which corresponds to reverting back to half of the response to selection. If z_2
458 is any value other than 10, the static biometric inheritance function results in changes in mean phenotype
459 in the absence of selection, drift, mutation and migration. With each subsequent generation (iteration step,
460 in this simple argument), the mean phenotype regresses further toward a value determined by the nature
461 of the static biometric inheritance function (Figure 5E). If selection is sustained, then the dynamic of the
462 mean phenotype even in this very simple IPM will be wrong, representing a component associated with the
463 response to selection, and a spurious change due to the misconception of biometric inheritance associated
464 with a parent-offspring regression with an immutable intercept. This is because a biometric inheritance
465 function with a constant slope and intercept is inconsistent with evolution.

466

467 Study case: bighorn sheep

468 We used a pedigreed population of bighorn sheep from Ram Mountain, Alberta, Canada (52°N, 115°W) to
469 demonstrate the limitations of the development and inheritance functions as implemented in standard IPMs.
470 Both quantitative genetics analyses (e.g. Coltman et al., 2003; Wilson et al., 2005) and IPM analyses (Traill
471 et al., 2014) have been conducted for this study system. This isolated population has been the subject of

472 intensive individual-level monitoring since 1971. Sheep are captured and weighed multiple times per year
473 between late May and late September. For detailed information on the study system see Jorgenson *et al.*
474 (1993), Festa-Bianchet *et al.* (1996) and Coltman *et al.* (2003). We analyzed predicted individual age-specific
475 masses adjusted to September 15 (see Martin & Pelletier, 2011; Traill *et al.*, 2014) for 461 ewes captured
476 from 1975 to 2011 and aged up to 10 years (2002 ewe-year observations). We built two statistical models, one
477 reflecting how the ontogenetic development of size and inheritance have been typically modelled in IPMs, and
478 the other corresponding to a possible alternative to estimating these two key functions, a random regression
479 animal model of size (Kirkpatrick *et al.*, 1990, 1994; Meyer & Hill, 1997; Meyer, 1998; Wilson *et al.*, 2005).
480 The reason why we chose random regression was its wide use in studying the genetics of developmental
481 trajectories. This specific alternative approach satisfies a number of criteria, namely: (i) it allows across-age
482 covariance, over and above that attributable to measured values of focal traits, (ii) it incorporates the known
483 fundamentals of quantitative genetics, (iii) it is economical in terms of the number of parameters that need
484 to be estimated, and (iv) its basic structure is compatible with IPMs. Criteria (i) and (ii) results in random
485 regression analysis providing an approach for characterizing development and inheritance that should be
486 robust to regression to the mean, as imperfectly measured quantities are not used as predictor variables,
487 and as it uses a modern notion of inheritance of quantitative traits. Nonetheless, several different options
488 can be adopted to avoid regression to the mean, including a formulation of an explicit genetic autoregres-
489 sive size-dependent model that accounts for measurement error. Also, although we suggest that the random
490 regression approach (and potentially other models using quantitative genetic approaches to characterize vari-
491 ation in phenotype and its inheritance) could profitably be integrated into the broader IPM framework, for
492 simplicity we refer to the former approach as “IPM” and to the latter as “RRM”. Both models were fitted in
493 a Bayesian framework, using MCMCglmm (Hadfield, 2010), and following the suggestions of Gelman *et al.*
494 (2003) and Gelman (2006) for choosing prior distributions.

495

496 **Standard IPM approach**

497 We used a linear model to estimate the development and inheritance functions used in typical IPMs. We
498 modelled observed ewe size at each age as a function of size at the previous age, with separate intercepts
499 and slopes for each age. For lambs, we estimated a regression of lamb mass on the mass of their mother two
500 months before conception (previous September). Formally, the model is described as

$$x_{i,a} \sim \mathcal{N}(u_a + b_{dev_a} \times I_{adult_i} \times x_{i,a-1} + b_{inh} \times I_{lamb_i} \times mothermass_{i,e_{i,a}}), \quad (13)$$

501 where $x_{i,a}$ is the observed mass of individual i at age a , u_a age-specific intercepts, b_{dev_a} age-specific size
502 slopes and b_{inh} is the inheritance function coefficient. I_{lamb} and I_{adult} are indicator variables for lambs and
503 older individuals, respectively. Finally, e_a are heterogeneous residuals per age. The estimated fixed effects
504 and variance parameters are presented in table 1.

505

506 Random regression of size

507 To model the family of size-at-age functions in bighorn sheep ewes, its genetic basis, and associated pheno-
508 typic and genetic covariances of size across age, we fitted a random regression animal model (Kirkpatrick
509 et al., 1990, 1994; Meyer & Hill, 1997; Meyer, 1998; Wilson et al., 2005) of the form

$$x_{i,a} \sim \mathcal{N}(\mu_a + f_1(d_i, n_1, a) + f_2(BV_i, n_2, a), e_{i,a}), \quad (14)$$

510 where $x_{i,a}$ is the size of individual i at age a and μ_a are age specific intercepts. f_1 and f_2 are random
511 regression functions on natural polynomials of order n , for permanent environment effects, f_1 , and additive
512 genetic values, f_2 . The permanent environment effect refers to everything consistent about individuals other
513 than the additive genetic effect (see Kruuk & Hadfield, 2007). In both f_1 and f_2 , n was set to 2, allowing
514 the estimation of random intercepts, slopes, and curvatures. Polynomials were applied to mean-centred
515 and standard deviation-scaled ages to improve convergence. Finally, heterogeneous residuals across ages
516 were estimated ($e_{i,a}$). d and BV , vectors with individual and pedigree values, respectively, were assumed
517 to follow normal distributions, $d \sim \mathcal{N}(\mathbf{0}, \mathbf{D})$ and $BV \sim \mathcal{N}(0, \mathbf{G} \otimes \mathbf{A})$. Both $\mathbf{D} = \mathbf{I}\sigma_i^2$, where σ_i^2 is the
518 permanent environment effect of individual i , and the additive genetic variance-covariance matrix, \mathbf{G} , are
519 3×3 matrices, \mathbf{A} is the pedigree-derived additive genetic relatedness matrix, and \otimes denotes a Kronecker
520 product. More information on partitioning of phenotypic variance into different components of variation
521 using general pedigrees and the animal model is provided by Lynch & Walsh (1998), Kruuk (2004) and
522 Wilson et al. (2010). In order to obtain the genetic variance-covariance matrix for the 10 ages, the following
523 equation is used

$$\mathbf{G}_{10} = \mathbf{\Phi}\mathbf{G}\mathbf{\Phi}^T, \quad (15)$$

524 where \mathbf{G}_{10} is the resulting 10×10 genetic matrix, \mathbf{G} is the 3×3 genetic matrix estimated by the model and
525 $\mathbf{\Phi}$ is a 10×3 matrix with the polynomials evaluated at each age (Kirkpatrick et al., 1990; Meyer, 1998).
526 A 10×10 matrix, \mathbf{D}_{10} , for individual effects at the 10 ages can be obtained similarly. The estimated fixed

527 effects and variance parameters are presented in table 2.

528

529 **Recovering resemblance within and across-generations**

530 We compare the correlations in mass among ages implied by the development functions typically adopted
531 in IPMs and those derived from a RRM, to the observed phenotypic correlations in the data set (Figure
532 6A-C). We used the path rules, as described for the theoretical models, to obtain the correlation matrix for
533 size at different ages implied by the IPM approach. There was no need to do the same for the RRM, as
534 these correlations were recovered with equation 15. We also analyze the proportion of correlation recovered
535 for different gaps between ages (projection steps, Δt) by both models (Figure 6D). The RRM estimates a
536 phenotypic correlation matrix (Figure 6C) that is much more similar to that observed (Figure 6A) than the
537 correlation matrix implied by the IPM approach (Figure 6B). This results in across-age correlations being
538 better recovered by the RRM when compared to the IPM (Figure 6D). The proportion of correlation in
539 size among ages recovered by an IPM follows the pattern predicted in figure 4A, with higher recoveries for
540 a single projection step, and from then on rapidly decaying to almost zero (Figure 6D). As predicted by
541 our theory, typical parameterizations of the development functions severely underestimate similarity of trait
542 values within individuals across ages.

543

544 Second, we show the parent-offspring regressions recovered by the RRM and the IPM, and use the “observed”
545 regressions as reference (Figure 7). These latter values correspond to regressions of daughters’ mass on their
546 mothers’ for all matching ages available, also including random intercepts for mother ID by age, year and
547 cohort, as well as heterogeneous residuals by age. The cross-age biometric inheritance function implemented
548 in IPMs recovers parent-offspring regression for lambs (age 1), but for older ages most similarity between
549 parents and offspring is missed (Figure 7). In contrast, the patterns of parent-offspring similarity recovered
550 by the RRM are of the observed order of magnitude throughout most of the life cycle (Figure 7).

551

552 **Discussion**

553 We have shown analytically that IPMs, as typically implemented, will generally, and severely, underestimate
554 quantities that are critical to evolutionary inference. Both our theoretical results, and our empirical example,
555 show that estimated phenotypic covariances within and across individuals can be effectively zero in these
556 models, due purely to artifacts of their construction. Additionally, the static nature of the inheritance func-

557 tion artificially reverses any response to selection that may occur. Consequentially, it is a foregone conclusion
558 that IPMs, as typically constructed, will suggest that evolution is not an important aspect of the dynamics
559 of traits over time. We suggest, and demonstrate empirically, alternative approaches that could be used to
560 characterize some key functions in IPMs. IPMs in principle are extremely useful and highly flexible, and
561 their original conceptualization (Easterling *et al.*, 2000; Ellner & Rees, 2006) should be broadly compatible
562 with a variety of alternative ways of characterizing variation in growth and inheritance.

563

564 The main reason why development functions in IPMs fail to recover within-generation covariances of traits
565 is regression to the mean. This problem is well-understood in some situations that arise in evolutionary
566 and ecological studies (e.g. Kelly & Price, 2005). In IPMs, the problem is even more severe than in other
567 situations, as the multiple age-specific projection steps compound the effect of measurement error to reduce
568 covariance among predictor and response variables. Consequently, covariance between non-adjacent ages,
569 which can be substantial (Figure 6A, Wilson *et al.*, 2005), is severely underestimated (Figure 6B), even
570 when measurement error is relatively small (Equations 1 and 2).

571

572 The failure of biometric inheritance functions to predict phenotypic similarity among relatives is partially
573 also a direct manifestation of regression to the mean. Indeed, it is the canonical manifestation of regression
574 to the mean – coined in exactly this context by Galton (1886). What we now understand is that Mendelian
575 factors are inherited, and that, in terms of statistical mechanics of quantitative genetics, environmental vari-
576 ation can be regarded as measurement error obscuring the inference of breeding values. It is hard to see how
577 any model of inheritance that does not include our understanding of how inheritance drives similarity among
578 relatives in quantitative traits (Fisher, 1918, 1930; Wright, 1922, 1931) can be expected to suffice for even
579 the most basic evolutionary predictions. Another issue arises from assuming that the biometric inheritance
580 function is constant. Whenever the mean phenotype changes, the intercept of the parent-offspring regression
581 necessarily changes as well. To presume that it is constant across generations implies assuming that the
582 mean phenotype can only change transiently.

583

584 In our theoretical models we use simple but general development and inheritance functions that are specifi-
585 cally designed to isolate each of these two fundamental processes from each other. However, in practice, the
586 undesirable behaviours that we have modelled separately will interact. Importantly, in iteroparous organ-
587 isms, where multiple episodes of reproduction occur over the lifetime, regression to the mean in development
588 functions will further obscure relationships between parents and offspring, with increasing effects as parents
589 age. Additionally, our results concerning covariance of parents and offspring are compounded across mul-

590 tiple generations. The underestimation of similarity between parents and offspring will be compounded at
591 each generation, leading to increasingly severe undervaluation of the relevance of relationships among more
592 distant relatives to the evolutionary process. This interaction is very evident in the empirical example we
593 present. Parent-offspring regressions recovered with the development and inheritance functions generally
594 used in IPMs (Figure 7) could not be predicted by the two-age theoretical model presented here, and specif-
595 ically by equation 11.

596

597 IPMs with typical cross-age biometric inheritance functions have been recommended for studying evolution-
598 ary responses to selection (Coulson *et al.*, 2010; Coulson, 2012; Rees *et al.*, 2014). Some studies applying
599 this approach have concluded that non-evolutionary changes in trait distributions are the major contributors
600 to temporal changes in phenotype (Ozgul *et al.*, 2010; Traill *et al.*, 2014). Our theoretical findings do not
601 indicate that these conclusions are, in themselves, wrong. Rather, we demonstrate that these are the con-
602 clusions that this kind of model must inevitably generate when applied to any system, regardless of whether
603 evolutionary change is important or not. Since typical parameterizations of IPMs neglect the vast majority
604 of similarity between parents and offspring, they cannot attribute phenotypic change to evolution. Concern
605 about how IPMs model the transmission of dynamic traits had been previously raised (Hedrick *et al.*, 2014;
606 Chevin, 2015; Vindenes & Langangen, 2015). Particularly, Chevin (2015) identified some issues addressed
607 in this paper, presenting insightful numerical examples that illuminate the main concern with the cross-age
608 structure of the inheritance function. Besides our analytical demonstrations, and the numerical examples
609 made available by Chevin (2015), we also provide an empirical example, where we use random regression
610 analysis as a potential alternative that addresses the issues presented here. The random regression model
611 provided substantial improvement in recovering both correlations across ages within a generation (Figure
612 6D), and parent-offspring regressions (Figure 7).

613

614 Vindenes & Langangen (2015) discuss joint models of static traits (constant through life) and developing
615 traits (such as those typically handled in IPMs) in the general IPM framework. They suggest that incorpora-
616 tion of static traits could solve some of the problems that had begun to be acknowledged about evolutionary
617 inference with IPMs (Hedrick *et al.*, 2014; Chevin, 2015). The authors propose that the static trait, birth
618 mass in their example, could be modelled as influencing mass at all other ages and demographic rates, which
619 would allow covariances among birth mass and older ages to be well recovered. In a sense, the solution we
620 have begun to explore treats breeding values (as opposed to some realized phenotypic value) as a static trait,
621 but critically also models the inheritance of breeding values, not as some observed function, but according to
622 the principles of quantitative genetics. It is noteworthy to mention that a genetic notion of trait transmission

623 has already been implemented into an IPM for a single Mendelian locus (Coulson et al., 2011). The authors
624 constructed an IPM that describes the dynamics of body mass and a biallelic gene determining coat color
625 in Yellowstone wolves (*Canis lupus*). And, in contrast to biometric IPMs of quantitative traits, Coulson
626 et al. (2011) conclude that the genetic variance within the studied population is enough for natural selection
627 to cause evolution. In fact, it in principle relatively straightforward to implement an IPM that uses the
628 basic principles of inheritance of polygenic quantitative traits to define inheritance functions of breeding
629 values; such exercises have indeed begun for a single trait (Childs et al., 2016). It is easy to conceive of
630 multivariate extensions of such inheritance functions (based on multivariate versions of equations 3 and 4),
631 whereby one could treat age-specific sizes as different characters, and could estimate genetic variances and
632 covariances from data. Nonetheless, a great deal of work is still required. For long-lived organisms, genetic
633 covariance matrices of age-specific traits would be very challenging to estimate with useful precision (Wilson
634 et al., 2010). Furthermore, the dimensionality of resulting phenotypes would overwhelm typical strategies
635 for implementing IPMs (Coulson et al., 2010; Rees et al., 2014; Merow et al., 2014). In practice, a key
636 challenge, but a surmountable one, will be to develop sufficiently flexible, low-dimensional characterizations
637 of the genetic basis of development for practical estimation and subsequent modelling. The function-valued
638 trait approach we adopted with our random regression model of bighorn sheep ewe mass is just one such
639 possibility. It is possible that other approaches could be even more useful; for example, uses of various
640 autocorrelation functions (Pletcher & Geyer, 1999; Hadfield et al., 2013), or factor-analytic mixed model
641 (de los Campos & Gianola, 2007; Meyer, 2009; Walling et al., 2014).

642 Summary

643 We have shown analytically and by empirical example that standard implementations of integral projection
644 models will generally severely underestimate the likelihood of evolutionary change. IPMs to date have been
645 constructed using characterizations of development and inheritance that would not stand up to scrutiny
646 in studies focusing on development and inheritance. It is not surprising that more complex models built
647 on such functions behave poorly. In fact, insofar as the ability of IPMs to track the full joint distribution
648 of phenotype has been suggested as their main quality for ecological inference, the problems that preclude
649 their typical use for evolutionary inference should be of equal concern to ecologists. Importantly, we have
650 suggested ways in which more nuanced models of development, and a modern understanding of inheritance,
651 can be incorporated into the general IPM approach. A great deal more work is required before IPMs based
652 on adequate models of development and inheritance will be field-ready. As a next step, careful studies of the
653 performance of different approaches for characterizing the genetic basis of developmental trajectories, with

654 particular focus on approaches that could be incorporated into an IPM framework, are needed.

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803 Tables

Table 1: Coefficients for the IPM standard approach, including regressions of mass at age a on mass at age $a - 1$, and of lamb's mass on mother's mass at conception for the bighorn sheep population of Ram Mountain. The values correspond to posterior modes and 95% quantile-based credible intervals.

Age	Intercept	Slope	Residuals
1	17.37 (14.14 - 20.59)	- (- - -)	17.80 (15.97 - 20.29)
2	25.54 (22.01 - 28.78)	0.71 (0.59 - 0.85)	20.44 (17.93 - 24.46)
3	26.11 (22.13 - 29.75)	0.69 (0.61 - 0.78)	17.53 (15.34 - 20.71)
4	35.17 (30.40 - 39.87)	0.50 (0.42 - 0.59)	18.01 (15.56 - 21.13)
5	26.79 (20.87 - 32.87)	0.63 (0.54 - 0.72)	15.29 (12.95 - 18.08)
6	27.25 (20.56 - 34.82)	0.63 (0.52 - 0.73)	17.85 (15.23 - 21.62)
7	27.44 (21.09 - 34.25)	0.62 (0.53 - 0.71)	12.67 (10.69 - 15.52)
8	27.53 (18.83 - 35.34)	0.62 (0.51 - 0.75)	15.16 (12.70 - 18.69)
9	23.45 (15.12 - 30.82)	0.68 (0.58 - 0.79)	11.45 (9.54 - 14.47)
10	20.05 (9.84 - 31.18)	0.72 (0.57 - 0.86)	15.24 (12.30 - 19.92)
Posterior mode and 95% credible interval for the inheritance regression: 0.12 (0.06 - 0.17)			

rather than the effect of age within each individual

Table 2: Coefficients for the random regression model on body mass, including age-specific fixed intercepts and residuals (upper part), as well as estimates for the intercept, slope (Age) and curvature (Age^2) of the random effects on breeding values (BV) and permanent environment (d , lower part) for the bighorn sheep population of Ram Mountain. The values correspond to posterior modes and 95% quantile-based credible intervals.

Age-specific intercepts and residuals		
Age	Intercept	Residuals
1	25.77 (25.18 - 26.36)	8.19 (4.75 - 11.68)
2	44.22 (43.48 - 44.94)	14.01 (10.83 - 17.23)
3	57.05 (56.26 - 57.83)	16.21 (12.90 - 19.73)
4	63.64 (62.85 - 64.42)	11.45 (8.80 - 14.20)
5	66.76 (65.93 - 67.59)	9.78 (7.41 - 12.32)
6	69.15 (68.29 - 70.03)	10.20 (7.55 - 12.99)
7	70.32 (69.47 - 71.16)	6.84 (4.91 - 8.96)
8	71.09 (70.20 - 71.99)	8.72 (6.27 - 11.31)
9	71.36 (70.44 - 72.30)	6.90 (4.50 - 9.43)
10	71.34 (70.14 - 72.48)	10.0 (5.95 - 14.56)
Random regression on age		
Term	BV	d
<i>Intercept</i>	7.59 (1.52 - 13.29)	8.60 (2.00 - 15.93)
<i>cov(Intercept, Age)</i>	2.29 (0.24 - 4.29)	0.46 (-1.45 - 2.56)
<i>cov(Intercept, Age²)</i>	-1.44 (-3.11 - 0.19)	-1.12 (-2.94 - 0.45)
<i>Age</i>	2.07 (0.71 - 3.37)	0.53 (0.01 - 1.38)
<i>cov(Age, Age²)</i>	-1.13 (-1.90 - -0.36)	-0.01 (-0.48 - 0.39)
<i>Age²</i>	0.89 (0.20 - 1.57)	0.34 (0.02 - 0.80)

804

805 Figures

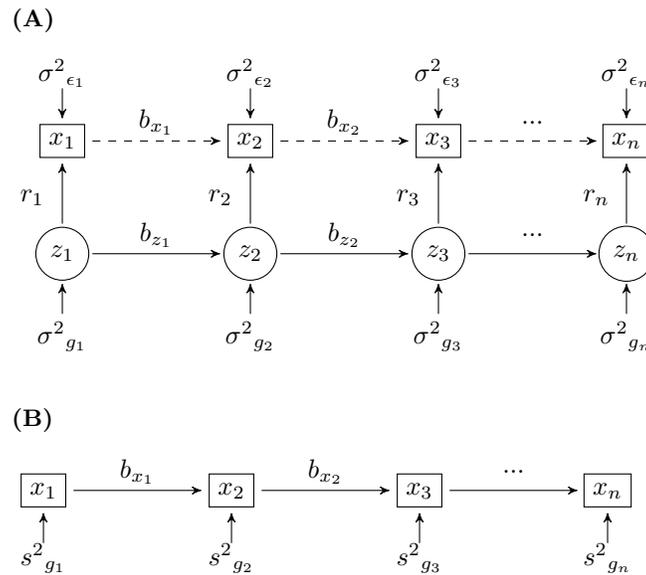


Figure 1: Path diagrams illustrating the ontogenetic development of size. **(A)** Latent true size model; **(B)** Observed size model implemented into IPMs. z_a and x_a are, respectively, the true and observed sizes at age a . r_a , linking true and observed sizes, are defined such that repeatabilities are r_a^2 . In these antedependence models, $\sigma_{g_a}^2$ and $s_{g_a}^2$ are exogenous variances in growth for true and observed values, respectively, except when they refer to $a = 1$. In this case, $\sigma_{g_1}^2$ and $s_{g_1}^2$ also correspond to variances in size. $\sigma_{\epsilon_a}^2$ are exogenous errors associated with observed sizes. b_{z_a} and b_{x_a} are growth regressions (path coefficients) for true and observed values, respectively. Dashed lines, as opposed to solid lines, do not belong in the path diagram. Although b_{x_a} correspond to the same quantities in both models, the two models result in covariance structures that are very different (see appendix A.1).

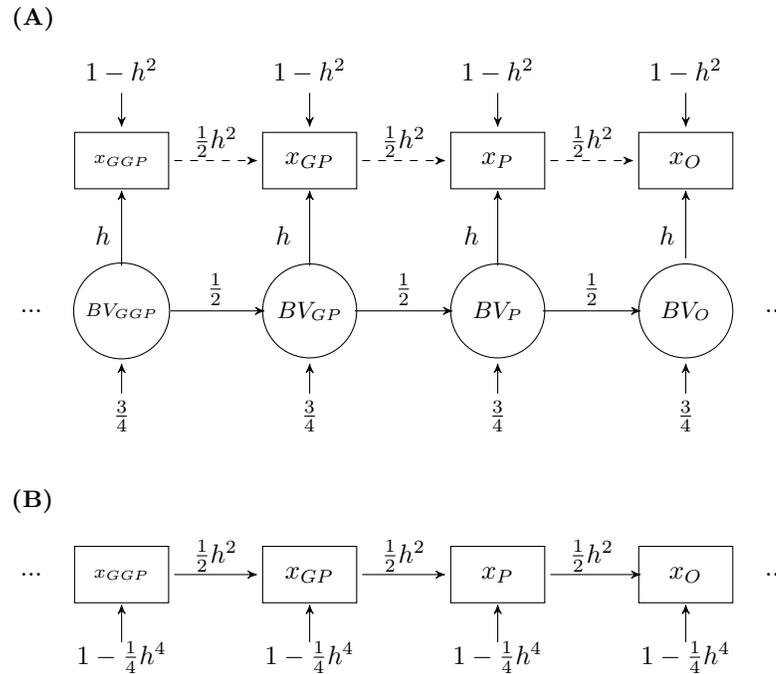
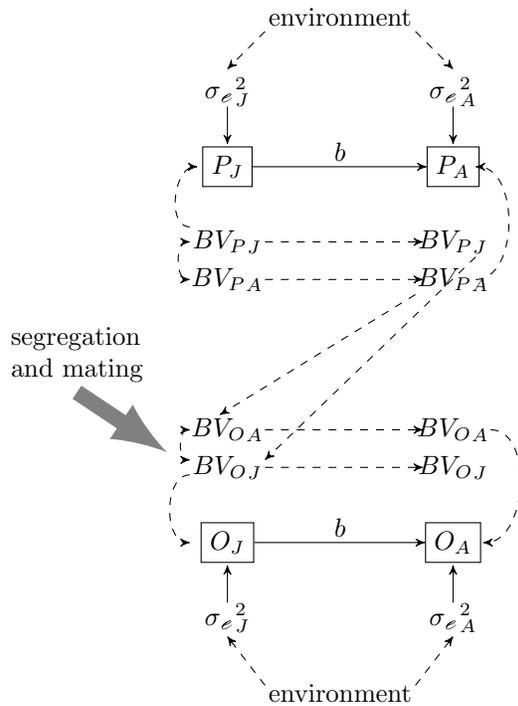


Figure 2: Path diagrams illustrating the transmission of a quantitative trait across generations of the same age. **(A)** Model based on the fundamentals of quantitative genetics; **(B)** model corresponding to a purely biometric notion of inheritance. BV and x correspond to breeding values and the observed phenotype, respectively. The exogenous inputs to BV s include contributions from the other parent and segregation. The subscripts GGP , GP , P and O denote great-grandparent, grandparent, parent and offspring, respectively. h^2 corresponds to the heritability and therefore h and h^4 to its square root and square, respectively. Dashed lines, as opposed to solid lines, do not belong in the path diagram. While the observed parts of the two models look very similar, they imply different correlation structures among relatives more than one generation apart (see main text).

(A)



(B)

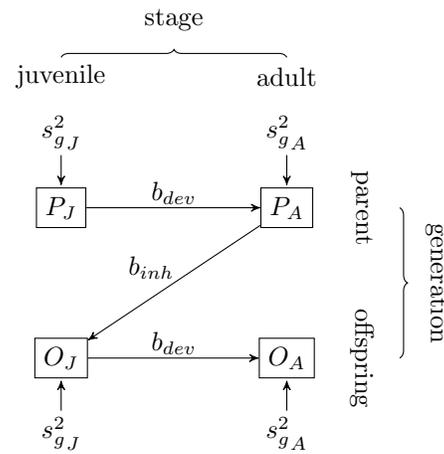


Figure 3: Path diagrams illustrating the transmission of a quantitative trait between parents and offspring with two ontogenic stages, juvenile, J , and adult, A . **(A)** Model based on the fundamentals of quantitative genetics; **(B)** model corresponding to a cross-age concept of trait transmission. P_J and P_A correspond to parental trait as juvenile and adult, respectively, and likewise for offspring (O_J and O_A). σ_e^2 and s_g^2 correspond to the exogenous variances of size at birth ($\sigma_{e_J}^2$ and $s_{g_J}^2$, and of growth until the juvenile stage) and of growth ($\sigma_{e_A}^2$ and $s_{g_A}^2$). b , b_{dev} and b_{inh} correspond to regressions, namely for development (b and b_{dev}) and inheritance (b_{inh}). Finally, BV are breeding values. Although the genetic constitution is constant over an individual's life, different genes are activated throughout life, which is denoted by distinguishing BV for both juvenile and adult stages.

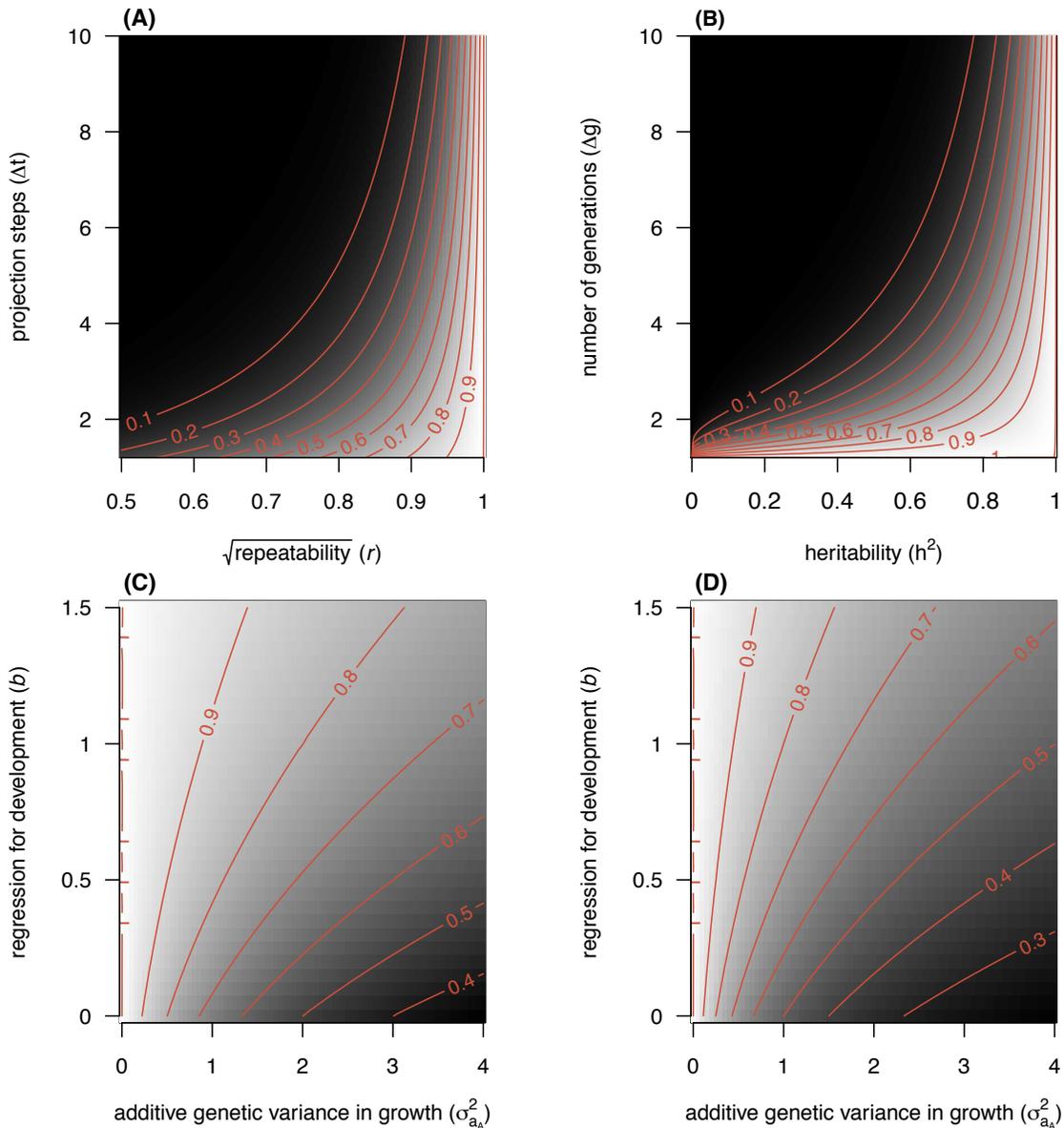


Figure 4: **(A)** Proportion of correlation in size among ages recovered by an IPM as a function of the square root of the repeatability (r) and number projection steps (Δt); **(B)** proportion of the parent-offspring regression recovered by a same-age inheritance function as a function of the heritability (h^2) and the number of generations (Δg); proportion of parent-offspring regression recovered by a cross-age parent-offspring regression, in juveniles, **(C)**, and adults, **(D)**. In **(C)** and **(D)** correlation in growth, both genetic ($\sigma_{a_{J,A}}$) and environmental ($\sigma_{e_{J,A}}$) was assumed to not exist, and the remaining parameters were set as follows $\sigma_{a_J}^2 = 1$, $\sigma_{e_J}^2 = 1$, and $\sigma_{e_A}^2 = 0$. The true values were used as reference in all plots.

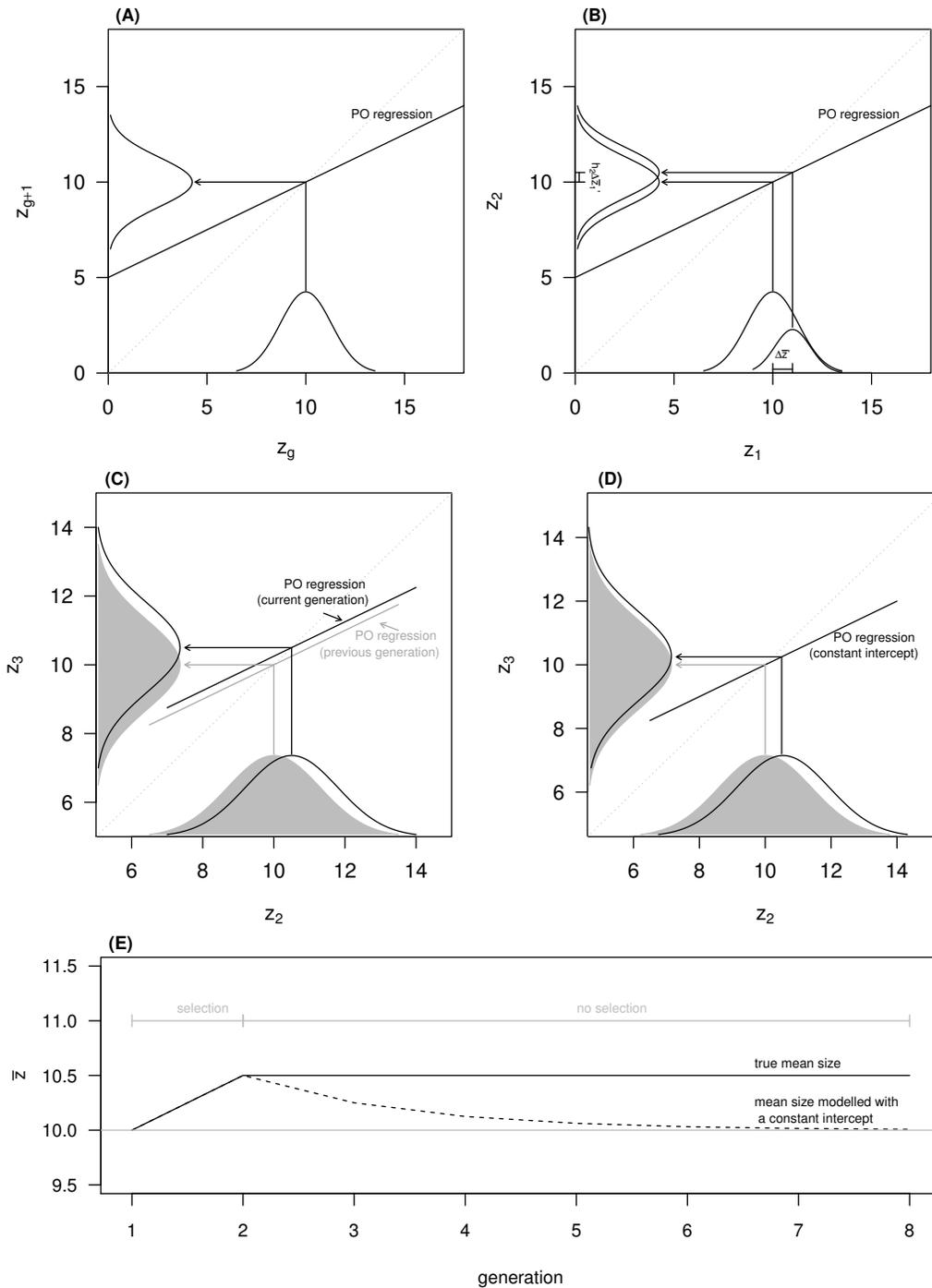


Figure 5: The consequences of assuming a constant intercept for the parent-offspring (PO) regression across generations. **(A)** Population at equilibrium, where mean phenotype is 10; **(B)** Period of selection. Selection before reproduction causes mean parental size to change from 10 to 11 ($\Delta \bar{z}'_1 = 1$). Mean offspring phenotype (z_2) is 10.5, which implies a parent-offspring regression given by $\bar{z}_2 = 5 + 0.5 \cdot (\bar{z}_1 + \Delta \bar{z}'_1)$, and therefore a $h^2 = 0.5$ and an intercept of 5; **(C)** Relaxed selection. When mean phenotype changes across generations, in this case from 10 to 10.5, the intercept of the parent-offspring regression necessarily changes as well. In a case of no selection in generation 2, the parent-offspring regression is given by $\bar{z}_3 = 5.25 + 0.5 \cdot \bar{z}_2$; **(D)** Relaxed selection with constant intercept. If the intercept is assumed to remain constant, and the first parent-offspring regression is used to estimate the mean phenotype in generation 3 (z_3), instead of the true value 10.5, 10.25 is obtained instead; **(E)** Extension of the models in **(C)** and **(D)** to subsequent generations of relaxed selection. In **(C)** and **(D)** the distribution in grey corresponds to the previous generation.

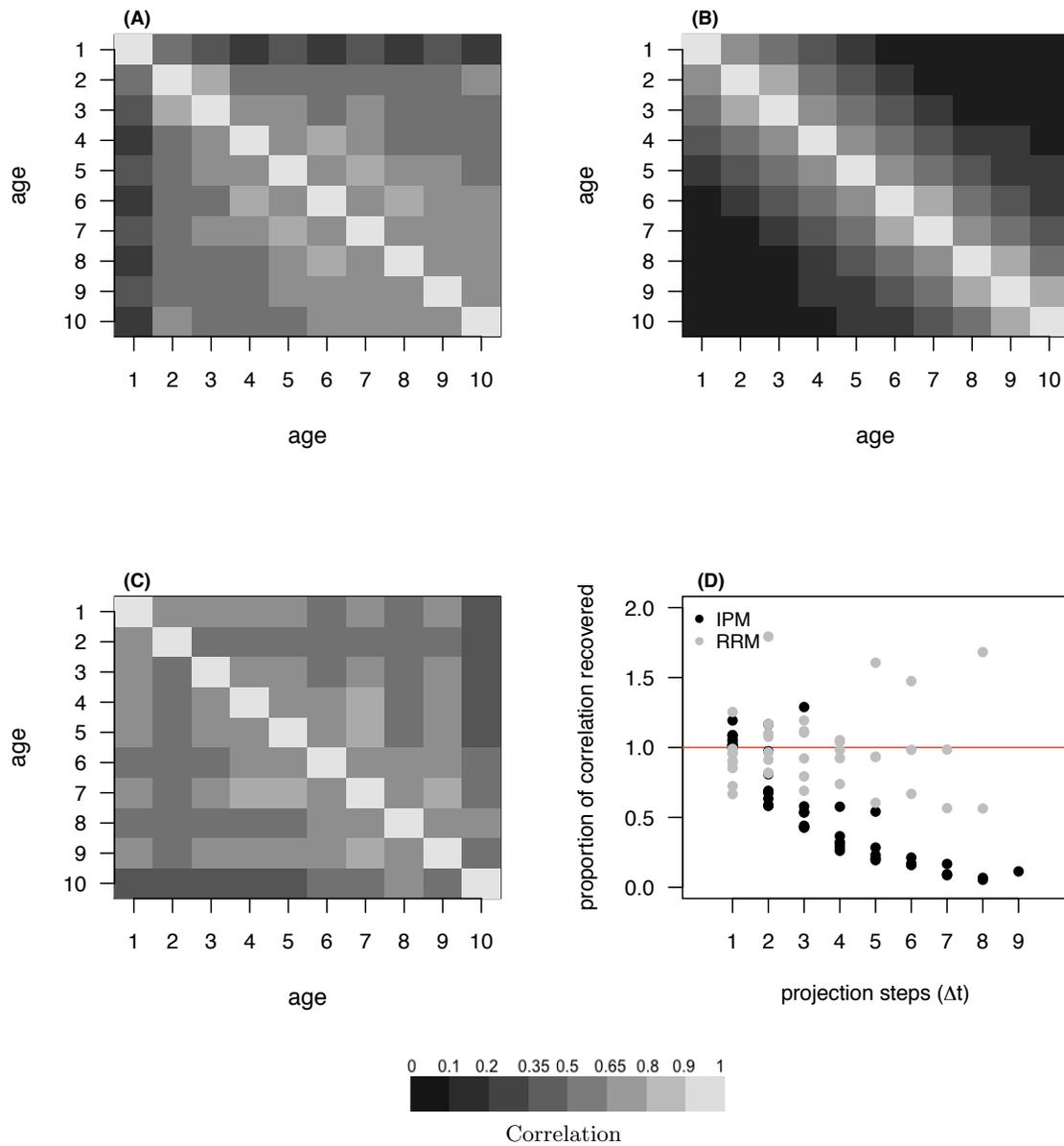


Figure 6: Observed phenotypic correlation matrix for size across ages for the bighorn sheep population of Ram Mountain (A), and analogous matrices implied by the IPM (B) and estimated by the RRM (C) approaches. Proportion of the correlations in size among ages recovered by the IPM (black dots) and RRM (grey dots) for different age gaps (projection steps, Δt), using the observed phenotypic correlations as reference (D). In (D), a proportion of 1 (horizontal line) corresponds to a perfect recovery of the observed correlation.

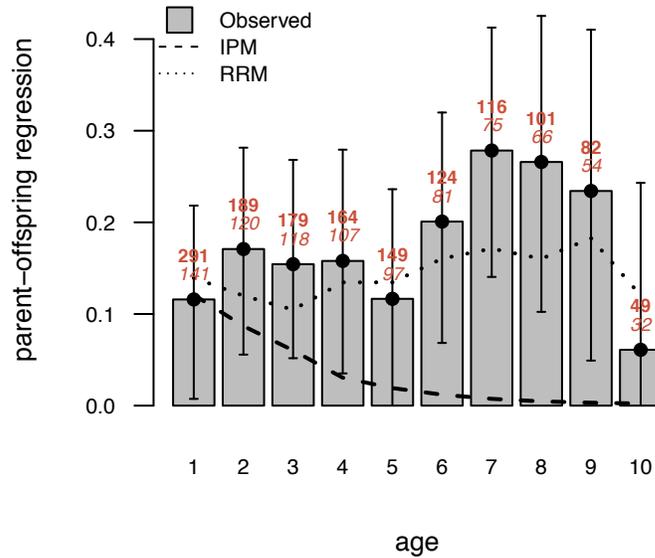


Figure 7: Parent-offspring regressions estimated for different ages for the bighorn sheep population of Ram Mountain, by the IPM and the RRM approaches. The observed values, and the corresponding 95% credible intervals, were estimated by a linear mixed model of daughters' mass on mothers' mass for matching ages, with random intercepts for the mother ID by age, year, and cohort. The values on top of the bars correspond to the number of offspring (top, bold) and mothers (bottom, italic) available for each age.