

1 Indirect genetic effects clarify how traits can evolve even when 2 fitness does not

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12 Abstract

13 There are many situations in nature where we expect traits to evolve but not necessarily for
14 mean fitness to increase. However, these scenarios are hard to reconcile simultaneously
15 with Fisher's Fundamental Theorem of Natural Selection and the Price identity. The
16 consideration of indirect genetic effects on fitness reconciles these fundamental theorems
17 with the observation that traits sometimes evolve without any adaptation, by explicitly
18 considering the correlated evolution of the social environment, which is a form of
19 transmission bias. While transmission bias in the Price identity is often assumed to be
20 absent, here we show that explicitly considering indirect genetic effects as a form of
21 transmission bias for fitness has several benefits: 1) it makes clear how traits can evolve
22 while mean fitness remains stationary, 2) it reconciles the fundamental theorem of natural
23 selection with the evolution of maladaptation, 3) it explicitly includes density-dependent
24 fitness through negative social effects that depend on the number of interacting conspecifics,
25 and 4) its allows mean fitness to evolve even when direct genetic variance in fitness is zero,
26 if related individuals interact and/or if there is multilevel selection. In summary, considering
27 fitness in the context of indirect genetic effects aligns important theorems of natural selection
28 with many situations observed in nature and provides a useful lens through which we might
29 better understand evolution and adaptation.

30

31 **Key words:** adaptation, evolution, fundamental theorem of natural selection, indirect genetic
32 effects, maladaptation, natural selection

33

34 Fundamental theorems of evolution and adaptation

35 R. A. Fisher's "fundamental theorem of natural selection" (FTNS) is one of the most famous,
36 and still widely debated ideas in evolutionary biology (Fisher 1930). Following careful re-

37 evaluation by G. R. Price, it is generally understood that Fisher's FTNS should be
38 understood as: In any population at any time, the rate of change of fitness ascribable to
39 natural selection is equal to its additive genetic variance at that time (Price 1972). This is:

40

$$41 \quad \Delta \bar{W} = V_{A,W} \quad 1$$

42

43 Where $\Delta \bar{W}$ refers to the change in mean fitness from one generation to the next caused by
44 natural selection, and $V_{A,W}$ is the additive genetic variance in fitness. Fitness here is "lifetime
45 breeding success" or similar, i.e. an absolute value, as Fisher related it to population growth
46 (Fisher 1930). Recent commentators have concluded that the FTNS is essentially true, and
47 in the way Fisher meant it (Bijma 2010a; Grafen 2015; Birch 2016). Therefore, when $V_{A,W} >$
48 0, natural selection is causing mean fitness to increase. Note that mean fitness may also be
49 increased or decreased by changes in the environment, hence the change ascribable to
50 natural selection may not be equal to observed changes in fitness, but for our purposes here
51 we assume a constant abiotic environment.

52 Independently derived, but fundamentally linked (Queller 2017), is the Price identity
53 (Price 1970; hereafter the PI, note a similar expression, but lacking the second term, was
54 derived earlier by A. A. Robertson 1966):

55

$$56 \quad \Delta \bar{P} = cov_A(\omega, P) + E(\omega \Delta P) \quad 2$$

57

58 Where $\Delta \bar{P}$ refers to the change in mean phenotypic trait value from one generation to the
59 next, and $cov_A(\omega, P)$ to the additive genetic covariance between individuals' relative fitness
60 (ω , equal to $\frac{W}{\bar{W}}$) and some phenotype (P) and $E(\omega \Delta P)$ is the change in mean phenotype
61 between parents and offspring, which could be caused by a bias in meiosis or fertilisation, or
62 by changes in the environment, which is referred to as "transmission bias". This simple but
63 powerful expression for the expected change in phenotypes states that for evolution to
64 occur, there must be a genetic covariance between relative fitness and the trait in question.

65 In typical treatments of trait evolution based on the Price identity, researchers
66 assume that the transmission bias is equal to zero, which gives Robertson's expression for
67 the evolution of traits (Robertson 1966). We do not contend this is incorrect, but we highlight
68 later that a portion of the change partitioned to transmission bias will in fact often have an
69 additive genetic basis, and therefore considering it explicitly is essential to understand
70 evolutionary trajectories in some cases. Otherwise, we assume a constant abiotic
71 environment throughout. Although it is not always appreciated, the PI implies that *for any*
72 *trait to evolve there must be non-zero additive genetic variance in fitness*, otherwise the

73 genetic covariance is undefined and evolution does not proceed (Morrissey et al. 2010;
74 Shaw and Shaw 2014).

75 The PI therefore makes clear that if any trait is evolving, there must be genetic
76 variance in fitness. Further, if there is genetic variance in fitness ($V_{A,W} > 0$), then according to
77 the FTNS mean fitness must be increasing ($\Delta\bar{W} > 0$). Conversely, if mean fitness is not
78 being increased by natural selection ($\Delta\bar{W} = 0$) then genetic variance in fitness must be zero
79 ($V_{A,W} = 0$) and so no trait can evolve. The combination of Fisher's FTNS and the PI,
80 therefore, lead to the following statements:

81

82 *"If a trait is evolving by natural selection, there must be genetic variance in fitness, and so*
83 *mean fitness is evolving"*

84 and

85 *"If a population's mean fitness is not evolving, then additive genetic variance in fitness must*
86 *be zero, so no trait can evolve as a result of natural selection"*

87

88 We refer to situations where some trait is evolving in response to natural selection as
89 "evolution by natural selection", while we refer to situations where mean fitness is increasing
90 by evolution as "adaptation". Taking the FTNS and the PI together implies evolution by
91 natural selection is *always* associated with adaptation. There are, of course, many ways in
92 which changes in the environment might cause mean fitness to remain stationary or decline,
93 but here we consider scenarios where the external environment remains constant.

94 In contradiction with these statements derived from the FTNS and PI, we clearly
95 observe situations in nature where evolution occurs, but adaptation does not (Fisher 1941;
96 Cooke et al. 1990; Frank and Slatkin 1992; Wolf et al. 2008). An example of this is that
97 males with larger weapons, or preferred sexual displays, are expected to sire more offspring
98 than their less well-endowed conspecifics. If these sexually selected male traits are
99 heritable, we would expect the mean trait to change across generations; we therefore have a
100 genetic covariance between the trait and fitness that is greater than zero. If so, there must
101 be additive genetic variance in fitness, and so Fisher's FTNS predicts that mean fitness
102 ought to evolve ($\Delta\bar{W} > 0$). However, in reality there is no expectation that the *total* amount of
103 reproductive success in the population will evolve, i.e. in this situation we would not expect
104 females to start having more offspring, and so mean fitness is not expected to change.
105 Therefore, no adaptation is occurring, and following Fisher's FTNS, genetic variance in fitness
106 ought to be zero ($V_{A,W} = 0$). Following the PI, evolution should then be impossible, yet we
107 clearly expect the weapons or the display trait to evolve if they are heritable. This scenario
108 also applies to any example of "soft" selection, where selection occurs among-individuals,

109 but does not lead to the mean reproductive output increasing (as opposed to “hard”
110 selection, where selection does lead to an increase in mean fitness Wallace 1975). So how
111 can we explain the action of sexual and soft selection, given that the FTNS and the PI are
112 true? To put it another way, when mean fitness is not evolving, do we really expect all
113 evolution to cease?

114 Furthermore, we can observe situations where trait evolution (requiring non-zero
115 $V_{A,W}$) leads to reduced rather than increased fitness (“maladaptation”, distinct from situations
116 where mean fitness is reduced purely by a change in the environment; Crespi 2000;
117 Rogalski 2017). For example, *Agelenopsis aperta* spiders in riparian zones show suboptimal
118 foraging and anti-predator behaviours compared to grassland populations, despite the
119 riparian habitat being available for at least 100 years (Riechert 1993). The FTNS suggests
120 that, as $V_{A,W}$ cannot be less than zero, $\Delta\bar{W}$ cannot be negative. Therefore, the FTNS seems
121 incompatible with observations of the evolution of maladaptation.

122

123 Social interactions as part of the environment

124 This paradox can be resolved by revisiting an element of the PI that is typically set aside: the
125 transmission bias. A transmission bias occurs when the mean phenotype of offspring and
126 parents differ, but not due to evolutionary change (Frank 2012). Typical examples are when
127 meiosis or fertilisation are not random with respect to the genes of interest, or when the
128 environment has changed in some way, and organisms’ traits depend on this environment.
129 Fisher too had a term for when phenotypes differ across generations due to environmental
130 change (“environmental deterioration”), and noted that it would typically act to reduce mean
131 fitness, which otherwise would continually increase (Fisher 1930). Fisher and others
132 considered the competitiveness of conspecifics to be a key part of the environment (Fisher
133 1930; Cooke et al. 1990; Frank and Slatkin 1992). Importantly, this “social environment” is
134 partly genetic in basis (as social traits will be partly heritable like any other trait) and so can
135 evolve (Griffing 1967; Moore et al. 1997). Hence a possible source of transmission bias and
136 environmental deterioration with limitless potential to continually change is the social
137 environment. Here we contend that not only can the social environment evolve, but that with
138 respect to many situations there are strong reasons to believe that *the social environment*
139 *must evolve*. Explicitly considering the evolution of the social environment and its influence
140 on the evolution of transmission bias allows trait evolution and adaptation to become
141 dissociated.

142 As an example of how the evolution of the social environment will dissociate trait
143 evolution from adaptation, we can consider the evolution of the ability to win contests for
144 dominance in a dyadic interaction, such as when two stags square off to determine who is

145 the strongest. Winning contests generally gives fitness benefits, and the propensity to win
146 contests is also often heritable (Wilson et al. 2009, 2011), so we would expect the mean
147 tendency to win such interactions to evolve. However, following Wilson and colleagues
148 (2009, 2011; 2014), a “common-sense” approach sees this is impossible, because in every
149 dominance interaction, there must be one winner and one loser, and hence the mean
150 outcome in a dyadic contest is constrained to remain half winning and half losing in each
151 generation. This is analogous to a situation where mean reproductive output cannot evolve,
152 for instance when it is constrained at the population level by resource availability (be that
153 food, territory space, or total offspring production of females in the case of sexual selection)
154 even though increased reproductive output is always expected to be favoured by fecundity
155 selection (Cooke et al. 1990; Frank and Slatkin 1992).

156 Common sense and models for micro-evolutionary change are reconciled by
157 appreciating that individuals possess genetic effects for their *opponent's* ability to win the
158 dominance interaction (Wilson et al. 2009, 2011; Wilson 2014). In a zero-sum contest, where
159 one individual's success directly detracts from their competitor's success, genes that
160 enhance an individual's chance of winning a contest necessarily reduce their opponent's
161 chance of winning. As these genes will be selected for, the propensity to win evolves, but so
162 too does the propensity for others to lose as a correlated response. As opponents are drawn
163 from the same population, contests for dominance in the next generation are now with more
164 competitive opponents, i.e. the environment has evolved to become more competitive at the
165 same time (Wilson 2014). This leads to no change in mean phenotype overall. This has
166 been termed the evolution of environmental deterioration as the environment the trait
167 (winning contests) is being expressed in has deteriorated (i.e. it has become more difficult to
168 express the trait; Fisher 1930). Crucially, there is still direct genetic variance in the
169 population for dominance, and so breeding values for it will increase over time. As such,
170 traits correlated with direct breeding values for the ability to win contests, such as weapon
171 size, will still evolve.

172 We can consider the importance of the evolution of the social environment to trait
173 evolution and adaptation in general by considering a quantitative genetic model of trait
174 evolution that considers indirect genetic effects (IGEs). Indirect genetic effects occur when
175 the phenotype of one individual is affected by the genotype of another individual (Moore et
176 al. 1997). Examples include genes in mothers influencing offspring growth (McAdam and
177 Boutin 2004), and genes in males influencing the date their partner lays a clutch (Brommer
178 and Rattiste 2008). In general, the response to selection in the presence of IGEs is (Bijma
179 and Wade 2008):

180

$$181 \quad \Delta \bar{P} = \beta_{W_D P} [V_{AD} + ncov_A(D, I)] \quad 4$$

182

183 Where $\beta_{W_{DP}}$ is the selection gradient of an individual's direct phenotype on fitness, V_{AD} is the
184 additive direct genetic variance in the trait, n is the number of conspecifics an individual
185 interacts with (i.e. group size excluding itself, note this replaces $n-1$ used by Bijma and Wade
186 2008, as they set n as group size *including* the focal individual), and $cov_A(D, I)$ is the additive
187 genetic covariance between the direct and indirect effects on the trait. The product of $\beta_{W_{DP}}$
188 and V_{AD} is equivalent to the first term in the Price Identity in the absence of an environmental
189 covariance between the trait and fitness (Rausher 1992). The product of $\beta_{W_{DP}}$ and
190 $ncov_A(D, I)$ represents the correlated evolution of the social environment that occurs
191 because of the genetic covariance between an individual's effect on its own phenotype
192 (direct genetic effect; DGEs) and its effect on the phenotype of others (IGEs). This is the
193 correlated evolution of the social environment, or in other words a non-zero transmission
194 bias. Equation 4 makes clear that, in the presence of covariance between DGEs and IGEs,
195 transmission bias in the Price identity is non-random with respect to selection and clearly
196 cannot be ignored. While transmission bias is often ignored because of an assumption that
197 the environment remains constant, considering genetic variance in social interactions makes
198 clear that in the presence of $cov_A(D, I)$ the environment cannot remain constant; the social
199 environment will necessarily evolve as a correlated response to selection. In the extreme
200 example of contests for dominance, the resource for which individuals compete (success in
201 a dyadic contest) is absolutely limited. However, as Cooke *et al.* (1990) observed, directional
202 selection on any resource dependent trait can be counteracted by changes in the
203 competitive environment, so the same IGE-based model can be applied to any trait
204 dependent on contests for limited resources (Frank and Slatkin 1992; Wilson 2014). For
205 instance, Muir *et al.* (2013) conducted an experiment on Japanese quail (*Coturnix japonica*),
206 where they applied artificial selection for body mass, which possesses additive genetic
207 variance. They observed no response to selection over 20 generations, despite the simple
208 expectation that mean body mass would increase over time in response to artificial selection.
209 In quail, however, body mass is a proxy for competitiveness with pen-mates for access to
210 feed. The heaviest quail were, therefore, the ones that suppressed the body mass of their
211 pen-mates the most, by outcompeting them for access to feed. As such, by artificially
212 selecting the heaviest individuals, Muir *et al.* were also selecting for those that reduced the
213 body mass of their pen mates the most. As these traits possessed additive genetic variance,
214 the result was the evolution of direct breeding values for body mass, but also the evolution of
215 breeding values for increased suppression of pen-mates' body masses. Therefore, there
216 were DGEs for body mass, IGEs for the body mass of pen-mates, and a negative DGE-IGE
217 covariance, overall giving no change in mean body mass. A similarly strong negative

218 covariance between direct and indirect genetic variance in performance was found for
219 diameter at breast height in plantations of Eucalyptus trees (*Eucalyptus globulus*),
220 presumably due to competition with neighbouring trees for light or other resources (Costa e
221 Silva et al. 2013). In both these examples the competitive ability of individuals can evolve,
222 but this leads to the evolution of equally more competitive social environments, and so mean
223 of the trait under selection does not change across generations.

224

225 Indirect genetic effects on fitness

226 If we consider fitness as a trait influenced by social interactions, then conspecifics can
227 influence each other's fitness following existing IGE models (Bijma 2011):

228

$$229 \quad W_i = \mu + C_i + \sum_n S_j + E_i \quad 5$$

230

231 Where individual i 's fitness (W_i) depends on the population mean (μ), as well as i 's direct
232 competitive ability (C_i), the sum of the social effects of its n neighbours ($\sum_n S_j$) and an
233 environmental/residual component (E_i ; Bijma 2011). This is an analogous framework to the
234 one proposed by Cooke *et al.* (1990), for the evolution of clutch size in birds, subsequently
235 built upon by Frank and Slatkin (1992). This simply says that an individual's fitness will be
236 influenced by its own competitive ability (e.g. its weapon size) but also by the competitive
237 abilities of other individuals in the group/population (see also models for "social selection",
238 e.g. Goodnight et al. 1992; Eldakar et al. 2010).

239 If we wish to consider how these social effects might constrain or facilitate the
240 evolution of fitness, we need to consider the genetic basis of competitive ability and social
241 effects on others' fitness (following Cooke *et al.* (1990) and Frank and Slatkin 1992). The
242 direct competitive abilities of individuals can be partitioned to an additive genetic component
243 and a non-genetic component. Similarly, an individual's social effects can be divided into
244 genetic and non-genetic effects on its competitors' fitness. There is, therefore, additional
245 genetic variance in fitness, stemming from competitors, alongside the more traditionally
246 considered direct genetic variance stemming from the focal individual. This additional
247 genetic variance can contribute to the evolution of fitness. The expected change in mean
248 fitness in the presence of IGEs (when unrelated individuals interact and in the absence of
249 multilevel selection) is given by (note that, as fitness is always maximally selected upon,
250 while the relationship between fitness and fitness passes through zero and is linear, $\beta_{W_{DP}}$ is
251 at the maximum of 1; Hereford et al. 2004):

252

$$253 \quad \Delta \bar{W} = V_{AD,W} + ncov_A(D_W, I_W) \quad 6$$

254

255 There are two important things to note from eq. 6. First, when $cov_A(D_W, I_W)$ is 0, we recover
256 the FTNS. This would be true, however, only when there is no intra-specific competition.
257 Instead, often an individual's fitness gains will necessarily detract at least somewhat from the
258 fitness of others and $cov_A(D_W, I_W)$ will be negative. A negative $cov_A(D_W, I_W)$ will reduce the
259 rate of evolution of mean fitness, which we have seen is a result of the evolution of a
260 deteriorating environment. If $cov_A(D_W, I_W)$ is sufficiently negative, $\Delta\bar{W}$ can equal 0 despite
261 $V_{AD,W}$ being non-zero. This will occur when fitness is completely zero-sum, such that any
262 fitness accrued by one individual is equal to the fitness lost by a competitor or competitors
263 (e.g. contests over a limited resource). Therefore, $cov_A(D_W, I_W)$ represents an explicit
264 measure of the degree to which adaptation will be constrained by competition, thereby
265 counteracting the continual evolution of increased mean fitness as predicted by the FTNS
266 (c.f. Cooke et al. 1990; Frank and Slatkin 1992). $cov_A(D_W, I_W)$ also represents an explicit
267 modelling of environmental deterioration, and of a form of transmission bias, in terms of the
268 contribution of IGEs (changes in the social environment) to the change in mean fitness.
269 Direct breeding values for fitness are still expected to increase across generations, as
270 selection for fitness always occurs. The effect on fitness at the phenotypic level, however, is
271 counterbalanced by the evolution of an increasingly competitive (deteriorating) environment
272 resulting from IGEs on fitness (Cooke et al. 1990; Frank and Slatkin 1992). The degree to
273 which fitness increases are counterbalanced by a deteriorating social environment, and
274 hence the degree to which fitness is zero-sum is measured by $cov_A(D_W, I_W)$.

275

276 Evolution without adaptation

277 While fitness IGEs might constrain the evolution of mean fitness (adaptation), the continued
278 evolution of DGEs on fitness means that traits correlated with fitness DGEs can still evolve
279 (unless these traits are also subject to IGEs; see Box 1). This is analogous to the situation
280 observed by Muir *et al.* discussed above. In Muir *et al.* (2013), body mass could not evolve
281 as it was subject to IGEs, but the competitiveness of individual quail was able to evolve. This
282 commonly occurs in livestock selected for increased yields, when pecking or biting
283 behaviours increase across generations, but yields do not (Ellen et al. 2014). This occurs
284 because traits related to social competition (e.g. aggressive pecking) are correlated with the
285 *direct* additive genetic variance in the yield trait (e.g. body mass). Traits related to social
286 competition can, therefore, increase, while overall performance (e.g. yield) remains constant
287 because of the evolution of more competitive environments. In the case of fitness, traits
288 related to fitness, such as weapon size or the brightness of a sexual display trait, can evolve
289 over time even when mean fitness does not evolve (but see Box 1). This, therefore, solves

290 the apparent problem posed by the two statements we made at the start of this paper.
291 Evolution occurring in populations where mean fitness is not evolving is in fact compatible
292 with Fisher's FTNS and the PI once IGEs on fitness are considered. Furthermore, evolution
293 without adaptation is absolutely required for the evolution of environmental deterioration to
294 occur (in the form of the evolution of more competitive rivals), yet this is often not made
295 explicit. If traits related to competitive ability cannot evolve then the environment cannot
296 deteriorate in this manner.

297 Neither the general ideas, nor models that we have outlined here are new. Applying
298 these ideas and models to fitness itself, however, clarifies when evolution and adaptation are
299 expected to occur, and when they are not. Arguably, Fisher would have classified all
300 changes in indirect effects as environmental deterioration, meaning that we should not
301 model them explicitly here. However, as this change has an additive genetic basis and is
302 correlated with changes in fitness due to direct genetic effects, it seems essential to include
303 them in our models for the evolution of fitness. Furthermore, there are additional insights into
304 trait evolution and adaptation that come from considering IGEs on fitness and fitness-related
305 traits.

306

307 The evolution of maladaptation

308 An interesting outcome of models for evolution in the presence of IGEs is that traits can
309 respond in the *opposite* direction to selection if a negative $cov_A(D, I)$ outweighs the influence
310 of direct effects (Griffing 1967; Moore et al. 1997; more formally, when $-1(cov_A(D, I)) >$
311 V_{AD}/n). In these cases, selection favours individuals whose indirect effects reduce the
312 population mean more than their direct effects increase it. What this means for the evolution
313 of fitness is that, although $V_{AD,W}$ can never be less than zero, $\Delta\bar{W}$ can be negative (i.e. the
314 evolution of maladaptation), if $cov_A(D_W, I_W)$ is strong enough ($-1(cov_A(D_W, I_W)) >$
315 $V_{AD,W}/n$; note this is analogous to the possible decrease in mean fitness when selection acts on
316 linked loci (Moran 1963), just that the fitness effects of the loci are observed in different
317 individuals). This is distinct from cases where fitness decreases due to a deterioration in the
318 non-social or abiotic environment, as the change in fitness caused by evolution of IGEs is
319 the direct result of selection (effectively for individuals that suppress others the most). Such
320 an effect has been observed in populations of flour beetles (*Tribolium castaneum*), where
321 artificial selection for individuals with *increased* reproductive output caused the mean
322 reproductive output across the populations to *decrease* over time (Wade 1976). This may
323 apply more generally to populations that are approaching or above a habitat's carrying
324 capacity, and so mean fitness is expected to decline in subsequent generations. That the
325 FTNS only ever allowed for an increase in fitness (adaptation, but not maladaptation) has

326 been one of its major criticisms (Frank and Slatkin 1992). Modelling the evolution of fitness
327 in the presence of IGEs allows maladaptation to occur, reconciling the FTNS with empirical
328 observations.

329

330 Indirect genetic effects and density dependence

331 Including IGEs in the expected change in mean fitness also leads to useful links between
332 quantitative genetics and population biology. For instance, eq. 6 takes similar form to the
333 logistic model of density-dependent per capita population growth:

334

$$335 \quad \frac{dn}{N dt} = r - rNK^{-1} \quad 7$$

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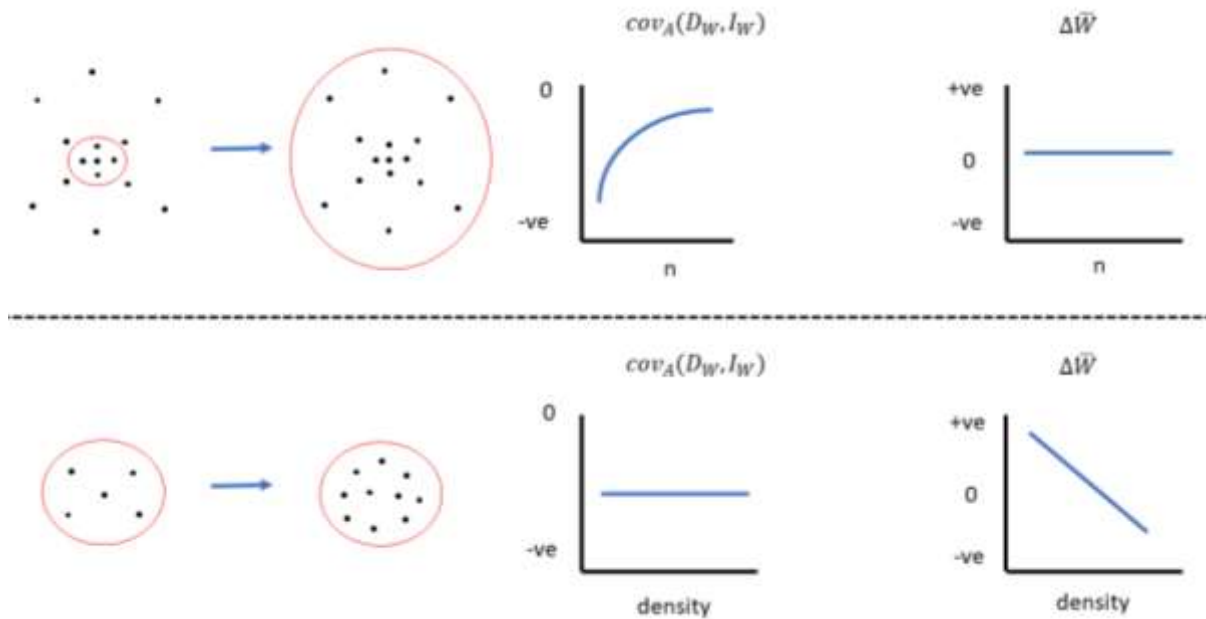
337 In the logistic model the rate of per capita population growth ($\frac{dn}{N dt}$) is positively affected by
338 the intrinsic rate of increase of the population (r), while $-rNK^{-1}$ represents the degree to which
339 per capita population growth is reduced by per capita increases in death rates and
340 decreases in birth rates as the population approaches its carrying capacity (K). Such density
341 dependence results from social interactions (such as competition for space or food) among
342 individuals that cause them to suppress the birth rate or increase the death rate of others.
343 These social effects may well have a genetic component, and hence be IGEs. When
344 populations are far below K , indirect effects on fitness are expected to be relatively weak. In
345 this scenario $V_{AD,W}$ can exceed $ncov_A(D_W, I_W)$ and mean fitness can evolve. This is
346 analogous to r-selection, as a low contribution from $ncov_A(D_W, I_W)$ due to non-limiting
347 resources allows the evolution of fitness and so rapid population growth. However, as the
348 population size approaches K , negative social effects on fitness become stronger, and
349 $ncov_A(D_W, I_W)$ will eventually be large enough to equal $V_{AD,W}$, and mean fitness can no
350 longer evolve. The change in mean fitness may even reduce below zero, causing the
351 population size to return below K .

352 Density-dependent selection has typically been modelled from a framework where
353 genotypes differ in their sensitivity to competition, which has led to the prediction of the
354 evolution of increased carrying capacity at high density (an increase in “efficiency” of
355 organisms; MacArthur 1962). The model including IGEs on fitness, however, makes an
356 additional prediction: at high density, we expect the evolution of increased ability to depress
357 the survival and reproduction of others as the population approaches carrying capacity (in
358 Fisher’s words: “life is made somewhat harder to each individual when the population is
359 larger”; Fisher 1930). This process ought to result in the evolution of *reduced* K . It is not
360 currently clear the degree to which density dependent selection in nature favours increased
361 efficiency versus enhanced ability to suppress the fitness of others.

362 It is tempting to directly relate the group size, n , in eq. 6 with the population size, N ,
363 in eq. 7, but these are not necessarily equivalent. All individuals within a population are
364 unlikely to interact with one another socially to the degree that they might depress one
365 another's fitness, so if population size (N) increases but density does not (i.e. the population
366 expands into uninhabited space) then the number of socially interacting individuals (n) will
367 not change. It is also generally expected that larger groups sizes should weaken
368 $cov_A(D_W, I_W)$, as more distant or more weakly interacting individuals who do not influence
369 each other's fitness are included within progressively larger groups (Fig. 1, top panel, see
370 also Bijma 2010b). If, however increasing population size implies greater *density*, as well as
371 simply more individuals, then social interactions may well get more intense (Fig. 1, bottom
372 panel). This would imply a greater, or at least stationary, $cov_A(D_W, I_W)$ as n increases, and
373 so the product $ncov_A(D_W, I_W)$ would contribute increasingly to $\Delta\bar{W}$. The explicit inclusion of
374 IGEs on fitness, therefore, results in the emergence of density-dependent per capita
375 reproduction through social effects.

376 The magnitude of the reduction in $\Delta\bar{W}$ caused by a negative $cov_A(D_W, I_W)$ depends
377 on how completely mean fitness in the population is constrained. Mild constraints will mean
378 a $cov_A(D_W, I_W)$ closer to zero (but still negative), and therefore a reduced, but not completely
379 eliminated, increase in mean fitness across generations. Absolute constraints mean a strong
380 negative $cov_A(D_W, I_W)$, and no change in mean fitness (no adaptation) or even a decrease
381 (maladaptation). Therefore, the difference between $V_{AD,W}$ and $ncov_A(D_W, I_W)$ is a measure
382 of the magnitude of the constraints on the evolution of mean fitness. How $cov_A(D_W, I_W)$
383 changes with n is an indication of the strength of density dependence, but cannot be
384 predicted beforehand. This instead remains an empirical question to be answered.
385 $cov_A(D_W, I_W)$ can be converted to a correlation between an individual's direct and indirect
386 genetic effects on fitness to compare across populations, with 0 indicating no constraints and
387 -1 indicating complete constraints, as found when analysing the evolution of dominance
388 contests (Wilson et al. 2009, 2011; Sartori and Mantovani 2013). Positive values would
389 indicate synergistic effects such as Allee effects (Allee 1931). In terms of hard and soft
390 selection, a correlation of 0 would indicate that selection is hard (not dependent on the traits
391 of others and leads to adaptation) while a correlation of -1 would indicate that selection is
392 completely soft (entirely dependent on the trait of an individual relative to others and does
393 not lead to adaptation).

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Figure 1. The relationship between n , density, $cov_A(D_W, I_W)$, and expectations for $\Delta\bar{W}$. Here we assume that the fitness of individuals is based on competition for limited resources, and so $cov_A(D_W, I_W)$ ranges from 0 to strongly negative. If we simply increase the number of individuals considered (top panel), then we expect $cov_A(D_W, I_W)$ to approach 0, as the additional individuals are less closely associating with each other, decreasing the mean social effect individuals have on each other. This balances the increase in n , giving a stationary $\Delta\bar{W}$. Here we have depicted $\Delta\bar{W}$ remaining at 0, assuming the population has reached a point that resources are completely preventing further evolution of increased reproduction. If, however, we increase the density of the individuals, as well as their number (bottom panel), then the $cov_A(D_W, I_W)$ may be stationary, or even become more negative, as the number of individuals increases. This reduces $\Delta\bar{W}$, in our example from an initial period of increasing fitness (below K), through no change (at K) and then to a decline (above K). This is the emergence of density dependent reproduction, only apparent through the FTNS when IGEs for fitness are considered.

Adaptation when direct genetic variance in fitness is zero

A final outcome of considering IGEs on fitness is that fitness can evolve (adaptation or maladaptation can occur) in populations where direct genetic variance in fitness is zero ($V_{AD,W} = 0$), if there are IGEs on the fitness of *related* conspecifics. When unrelated individuals interact, if $V_{AD,W}$ is zero, $cov_A(D_W, I_W)$ is then undefined and, following eq. 6, $\Delta\bar{W}$ is zero. However, if related individuals interact, the expected change in mean fitness follows (Bijma and Wade 2008):

419
$$\Delta\bar{W} = r[V_{AD,W} + 2n \text{cov}_A(D_W, I_W) + n^2V_{AI,W}] + (1 - r)[V_{AD,W} + n\text{cov}_A(D_W, I_W)] \quad 8$$

420

421 Where r is the mean coefficient of relatedness between interacting individuals, $V_{AI,W}$ is the
422 additive indirect genetic variance for fitness, and other terms are as defined for eq. 6. This
423 allows a change in mean fitness when $V_{AD,W}$ and $\text{cov}_A(D_W, I_W) = 0$, as long as $V_{AI,W} > 0$ and r
424 $\neq 0$:

425

426
$$\Delta\bar{W} = rn^2V_{AI,W} \quad 9$$

427

428 So, in contrast with a simple interpretation of FTNS, population mean fitness can evolve
429 even in the absence direct genetic variance in fitness, as long as fitness-relevant social
430 interactions are with relatives and there are IGEs for fitness. Note in these equations for the
431 response to selection in the presence of IGEs, r can be replaced without altering the
432 equations by g , the relative strength of multilevel selection (Bijma and Wade 2008). As such,
433 the presence of multilevel selection can also allow adaptation (or maladaptation) to occur
434 when $V_{AD,W}$ is zero, as long as $V_{AI,W} > 0$ and $g \neq 0$ (see Bijma and Wade 2008 for when both r
435 and g are non-zero, and see also McGlothlin et al. 2010).

436 Given that populations in equilibrium conditions are typically expected to show very
437 little $V_{AD,W}$ (Fisher 1930), this provides a mechanism for those populations to still adapt. For
438 instance, in a population of North American red squirrels (*Tamiasciurus hudsonicus*) $V_{AD,W}$
439 was found to be essentially zero, but maternal genetic effects on fitness were present
440 (McFarlane et al. 2015). Maternal genetic effects are a specific form of IGE where a mother's
441 genes (e.g. for milk production) influence the traits of her offspring. When parents interact
442 with offspring, r is non-zero. Models for evolution in the presence of maternal genetic effects
443 are then valid, which allows the population to evolve, albeit with a lag due to the cross-
444 generational effect (Kirkpatrick and Lande 1989; Mousseau and Fox 1998). Therefore,
445 fitness can change from generation to generation, despite lacking direct additive genetic
446 variance. This is not a new result, as evolution and adaptation in the presence of maternal
447 genetic effects and IGEs in general is accepted. Worth noting is that, as direct breeding
448 values for fitness are not changing across populations, the breeding values for any traits
449 genetically correlated with these will also not change. A trait may evolve, however, if it is
450 genetically correlated with indirect breeding values for fitness.

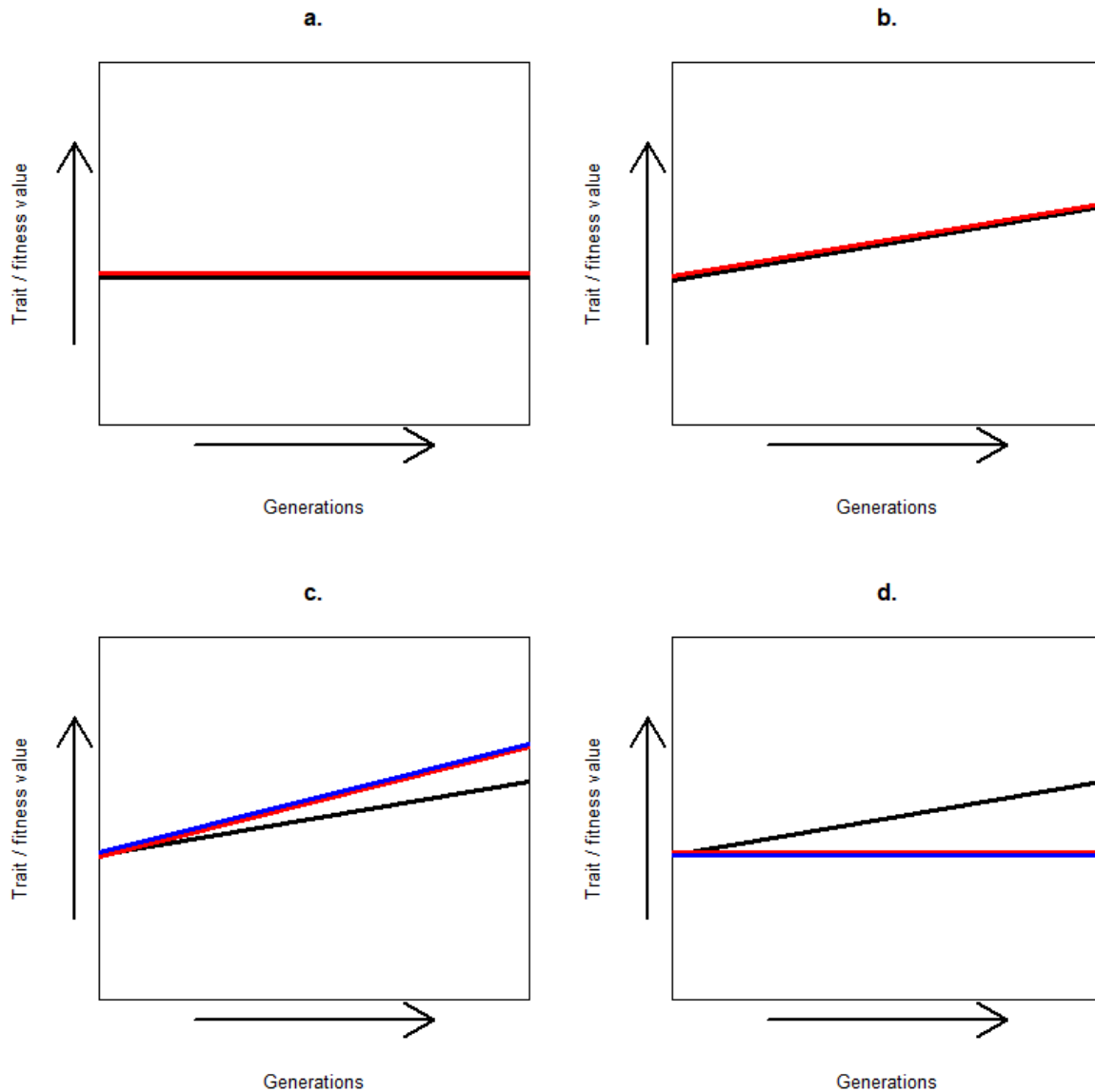
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452 Conclusions

453 Fig. 2 illustrates four situations which correspond to our formulation for the change in mean
454 fitness we have outlined above (although we do not plot the case where DGEs for fitness are

455 absent but IGEs among relatives and/or in the presence of multilevel selection do occur, see
456 the section on “Adaptation when direct genetic variance in fitness is zero”). These represent
457 a complete range of cases: when DGEs for fitness are either absent or present, when IGEs
458 are either absent or present, and if both DGEs and IGEs are present, if they positively or
459 negatively covary. We indicate the consequences each situation has for the expected
460 evolution of mean fitness (adaptation), as well as for the evolution of other traits within the
461 population (evolution by natural selection). These demonstrate that considering the evolution
462 of fitness as the response to selection in the presence of IGEs allows us to account for many
463 situations observed in nature and captive breeding. Frank and Slatkin stated that
464 “fitness...increases by an exact amount because of natural selection but simultaneously
465 increases or decreases by an unpredictable amount because of the environment”(Frank and
466 Slatkin 1992). We hope we have shown here that, by incorporating IGEs into our models, a
467 portion of this change caused by the environment is predictable.

468 In summary, considering IGEs on fitness allows us to reconcile the FTNS and the PI
469 with several observations: 1) it allows evolution even when adaptation is not occurring. This
470 was acknowledged by Fisher, and is implied by models for trait evolution in the presence of
471 IGEs, but appears impossible under conventional understandings of the FTNS and PI. 2) It
472 allows the evolution of maladaptation, reconciling the FTNS with empirical observations. 3)
473 Including n in the equation for the change in mean fitness reveals density-dependence,
474 helping to link quantitative genetics to population biology. 4) It indicates when adaptation can
475 occur even when direct genetic variance in fitness is lacking. Considering IGEs on fitness
476 explicitly models the deterioration of the social environment, a type of transmission bias, and
477 so clarifies how both the evolution of traits and the adaptation of populations is expected to
478 proceed.



479

480 Figure 2a-d. How fitness (red) and a trait (black) are expected to change across generations.
481 Note the scale for both the trait and fitness is arbitrary; we do not necessarily expect a trait
482 and fitness to increase at exactly the same rate in scenario b. for example. For simplicity we
483 assume that interactions are with non-relatives ($r = 0$) and there is no multilevel selection ($g =$
484 0). a: No DGEs for fitness, no IGEs. No genetic variance in fitness. Neither adaptation nor any
485 evolution will occur. b: DGEs for fitness, but no IGEs. Heritable variance in fitness is present,
486 and so mean fitness is expected to evolve over time in line with the FTNS. Traits genetically
487 correlated with fitness are also able to evolve. Both adaptation and evolution can occur. c:
488 DGEs and IGEs for fitness, positive DGE-IGE covariance. Heritable variance in fitness is
489 present, and so mean fitness is expected to increase over time, and rapidly as the positive
490 DGE-IGE covariance shifts the response in the same direction as selection. Traits genetically
491 correlated with fitness will evolve, although only as fast as fitness if they too are influenced by
492 IGEs (blue line). Evolution and rapid adaptation. d: DGEs and IGEs for fitness, negative DGE-

493 IGE covariance for fitness. The expected evolution of fitness will be reduced, possibly to zero
494 or even below. However, as direct breeding values for fitness will still be increasing across
495 generations, traits genetically correlated with fitness may evolve, unless they too are influence
496 by IGEs (blue line). This corresponds to situations where livestock under artificial selection for
497 increased yield have shown no evolution of yield but do show increases in aggressive
498 behaviours such as biting or pecking, as well as the instances of sexual selection described
499 in the text. Evolution but no adaptation.

500

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504

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