# Evolutionary consequences of indirect genetic effects

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**Indirect genetic effects (IGEs) are environmental influences on the phenotype of one individual that are due to the expression of genes in a different, conspecific, individual. Historically, work has focused on the influence of parents on offspring but recent advances have extended this perspective to interactions among other relatives and even unrelated individuals. IGEs lead to complicated pathways of inheritance, where environmental sources of variation can be transmitted across generations and therefore contribute to evolutionary change. The existence of IGEs alters the genotype–phenotype relationship, changing the evolutionary process in some dramatic and non-intuitive ways.**

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The various factors that influence the expression of traits are traditionally partitioned into those effects reflecting the actions of genes and those attributed to environmental causes (see Boxes 1–3). Although appealingly simple, this partitioning may be expanded and complicated by subdividing the genetic or environmental influences. For example, epistatic, dominant and additive effects of genes can be modeled by subdividing genetic influences while special or common environmental influences can be incorporated by subdividing the environmental influence[1]. One particular phenotypic influence, the effect of social or biotic environments provided by conspecifics, has recently attracted the interest of quantitative geneticists[2,3]. Unlike most environmental effects, social influences are unique because they are both environmental and genetic. If there is variation in the quality of the environments provided by others, and if that variation reflects (at least in part) genetic differences among the individuals, then 'indirect genetic effects' (IGEs) exist and the environment is heritable (Boxes 1–3). Such influences are defined as indirect genetic effects because the genes influencing the trait act indirectly; that is, they are expressed in an interacting individual (Boxes 1–3), not in the individual whose phenotype is measured[4–7]. This contrasts with the direct effects of genes acting on the phenotype of the focal individual (Boxes 1–3). The evolutionary consequences of these indirect genetic effects are more complicated than the simple model of phenotypic influences. Environments reflecting the indirect effects of genes are potentially subject to selection and subsequent evolution. Indirect environmental effects (IEEs) occur when non-genetic (i.e. environmental) influences on the phenotype of one individual have indirect effects on the phenotype of another individual[8]. While IEEs have important ecological and evolutionary consequences[8], they are not discussed here because they do not contribute directly to the evolutionary response to selection.

Perhaps reflecting our mammalian bias, most research has focused on the seemingly ubiquitous effects of environments provided by mothers to their offspring[2,3,9]. However, interactions with any relative[10], or even unrelated individuals[4–7], can result in IGEs. Whenever social environments are important influences on phenotypes, heritable environments will play an important evolutionary role. Nonetheless, the evolutionary consequences of IGEs have traditionally been ignored, beginning with Darwin's[11] concern about distinguishing heritable from non-heritable causes of individual variation. Darwin argued that individuals sharing identical environments, such as members of the same family, nevertheless exhibit heritable differences in characters: '[s]eedlings from the same fruit, and the young of the same litter, sometimes differ considerably from each other, though both the young and the parents have been exposed to exactly the same conditions of life; this shows how unimportant the direct effects of the conditions of life are in comparison with the laws of reproduction, and of growth, and of inheritance' (Ref. 11, p. 10). Darwin emphasized the primacy of heritable diversity at the expense of the homogenizing effects of common environment within families because he wanted to establish a non-environmental (i.e. heritable) basis for resemblance. In doing so, he set forth a bias against the possibility of inheritance of the family environment itself through indirect genetic effects.

Darwin's goal of separating environmental from genetic causes of phenotypic resemblance has been shared by animal and plant breeders. In reconciling mendelian and biometric perspectives on inheritance, Fisher[12] showed how various genetic and environmental contributions to phenotypic variation could be partitioned; however, his work focused primarily on the role of direct additive genetic effects in evolution[12,13]. This primary focus on direct genetic effects has continued in quantitative genetics. For example, consider Falconer and Mackay's (Ref. 1, p. 156) discussion of the genetic causes of resemblance between relatives: '[t]he young are subject to a maternal environment during the first stages of their life, and this influences the phenotypic values of many metric characters even when measured on the adult, causing offspring of the same mother to resemble each other. The common maternal environment of full sibs is often the most troublesome source of environmental resemblance to overcome by experimental design.'

The experimental bias against the study of indirect genetic effects, especially those associated with maternal environments, is clear. This bias persists in recent texts that either mention maternal effects only once (e.g. Ref. 14, in the

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context of cytoplasmic inheritance; Ref. 15, maternal nutritional effects; Ref. 16, fecundity selection) or not at all (e.g. Refs 17,18). Despite the relative lack of studies explicitly looking at indirect genetic effects, studies that document the effects of environments provided by others are common. Interactions among individuals often influence traits as diverse as development and behavior[9]. In species with parental care or delayed dispersal, the environment provided by relatives can influence nearly every life-history trait in the offspring[2]. Somatic contributions provisioned by the mother in her eggs can have profound influences on developing embryos, and are particularly well-studied in plants[2,3]. The effects of social environments on behavior such as communication, mating, aggression or foraging form a large portion of behavioral ecology[19]. Despite this wealth of studies showing that social, parental or other biotic environments are ecologically important, there has been little attention paid to causes of variation in these environments[9] and thus to their genetic influences (e.g. Boxes 1–3). Theoretical work in quantitative genetics suggests that this neglect has resulted in our overlooking potentially wide-ranging effects in evolution. Recent applications of IGE models have provided new insight into several problems in behavioral ecology and evolution such as kin selection and parental investment (Ref. 20; see Box 4), mate choice (see Refs 7,21), and the evolution of social interactions (see Refs 6,7).

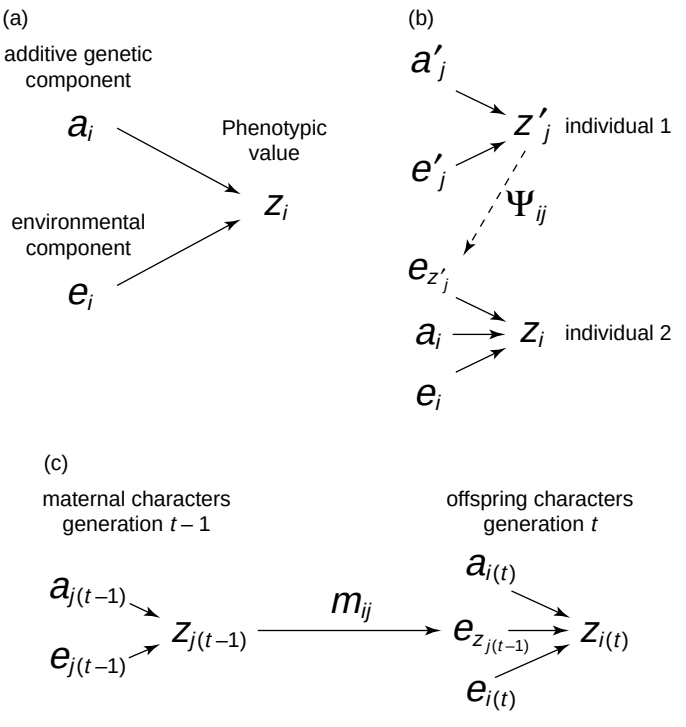
**Box 1. Performance and individual trait models of indirect genetic effects**

Theoretical and empirical studies of IGEs can be divided into those that measure performance characters and those that directly measure individual traits. Performance is a composite trait that includes all characteristics of one individual that affect the expression of a trait in another individual[9]. Performance is not a trait that is measured directly, but rather is a component of the phenotype of one individual attributable to the environment provided by another individual. Defined like this, as the total effect, performance is likely to be a composite trait. If the actual traits that contribute to performance are known, then the independent contribution of each of the multiple traits to the expression of the interacting individuals' phenotype can be measured directly using partial regression (see Refs 6,23,36). The differences between the two approaches can be seen in Boxes 2 and 3. Box 2 illustrates the partitioning of direct and indirect genetic effects on individual traits. Box 3 illustrates the performance character approach using the special case of maternal performance. In this example, three maternal traits all contribute to the expression of a single offspring trait but are not themselves measured. Analogous approaches can be applied to the study of non-maternal IGEs[6,7,10].

In general, the choice of one approach over the other depends on the question being asked and the logistics of a particular system[9]. For example, in the case of maternal and offspring characters, if one is interested in the evolution of a particular offspring trait (e.g. body mass) and not necessarily concerned with the evolution of particular maternal traits then the maternal performance approach may be favored[9]. This is because, while maternal performance may include the effects of a large number of maternal characters, these individual characters may be difficult to measure directly (e.g. characteristics of the uterine environment). One could understand the evolution of the offspring trait without having to measure all maternal characters by simply accounting for their net affect, which is maternal performance.

If one is interested in understanding the evolution of particular maternal traits, or in multivariate evolution in general, then the individual trait approach is favored[23,37]. This is because the partitioning of independent effects to individual maternal traits allows one to use a multivariate model to predict the evolutionary response to selection of multiple maternal and offspring traits[23]. However, because the individual trait approach relies on the use of partial regression to estimate the effect of individual maternal traits it assumes that either all maternal traits contributing to the expression of the offspring trait have been measured or are independent (i.e. not correlated[37]). The failure to measure all traits may result in the incorrect attribution of a maternal effect to a particular maternal trait or the inability to generate a predictive evolutionary model. This problem is avoided in the performance trait approach because all possible maternal traits are accounted for when the composite trait, maternal performance, is estimated.

**Box 2. The contribution of direct genetic effects, indirect genetic effects and environmental effects to the phenotype**




(a) The standard quantitative genetic partitioning of the phenotype ($z_i$) into additive genetic ($a_i$) and environmental values ($e_i$). (b) The indirect genetic effect of phenotype $z'_j$ in individual 1 on the phenotype $z_i$ of individual 2. Primes indicate values for characteristics of individuals other than the focal individual. The effect of the environment provided by individual 1 on the expression of trait $i$ in the focal individual, 2, is denoted $e'_{z_j}$. $\Psi_{ij}$ is a coefficient that measures the effect that $z'_j$ has on the expression of $z_i$ (see Refs 6,7 for details). (c) The special case for maternal effects. Trait $z_j$ expressed by the mother in the previous generation, $t-1$, affects the expression of trait $z_i$ in the offspring in the current generation, $t$. The coefficient $m_{ij}$ describes the degree to which the maternal trait $z_j$ contributes to the expression of the offspring trait $z_i$ (see Ref. 23).

## Modes of effect on evolution

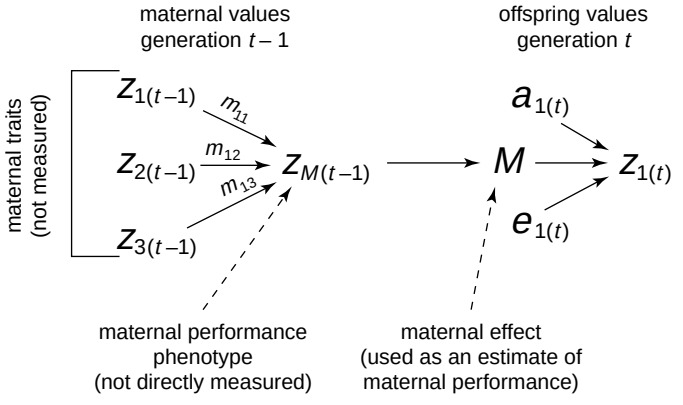
Most of the effects of IGEs on evolutionary processes can be accounted for by two phenomena: (1) IGEs alter the expected genotype–phenotype relationship, and (2) as environmental components of the phenotype, IGEs represent a component of the environment that itself can evolve[6]. While these two are clearly related phenomena, in that the genotype–phenotype relationship is altered by the environmental contribution of IGEs to the phenotype, they have distinct evolutionary consequences. An understanding of these two phenomena is central to understanding how IGEs influence or alter evolutionary predictions. Here we use a quantitative genetic perspective to illustrate the role of IGEs in evolution.

### *The genotype–phenotype relationship*

In quantitative genetics the genotype–phenotype relationship is described by the covariance between the additive genetic value and the phenotypic value of individuals [denoted $C_{az}$ (see Ref. 22)]. This relationship is important because it determines the genetic response to phenotypic selection. That is, the covariance between phenotype and genotype is what allows us to translate phenotypic change (selection) into changes in gene frequencies (evolution[7]). Models that consider only direct genetic contributions to the phenotype assume that the covariance between the genotype and phenotype is equivalent to the additive

### Box 3. Performance characters (illustrated by maternal performance)

Shown are the three components contributing to the offspring phenotype: the direct genetic effect $a_{1(t)}$, the environmental effect $e_{1(t)}$ and the maternal effect $M$. The maternal effect is caused by maternal performance of the mother that is itself composed of the contributions of three maternal traits. The effect of the maternal trait on the expression of the offspring trait is given by $m_{ij}$ where $i$ is a trait in the offspring (in this case $i$=1) and $j$ is the maternal trait ($j$=1, 2 or 3). Additional details are explained in Box 2.




### Box 4. Indirect genetic effects and Hamilton's Rule

The evolution of characters that produce indirect genetic effects in relatives is determined by the balance of direct selection on the phenotype and kin selection acting via the effect of that phenotype on the fitness of relatives (i.e. kin effects)[9,20]. Hamilton's rule[38], specifying the conditions under which a mutation resulting in an altruistic phenotype is expected to increase in frequency, provides a means to understand how traits affecting the fitness of kin are expected to evolve.

Hamilton's rule states that alleles resulting in altruistic phenotypes will increase in frequency when the ratio of the fitness costs to the 'altruist' ($c$) to the fitness benefits of recipients ($b$) is less than the coefficient of relationship between altruists and recipients, or

$$r > |c|/b \quad (1)$$

By convention, costs are considered as negative and benefits as positive. General quantitative genetic kin effects models based on maternal effects approaches (reviewed in Ref. 9) identify an unstated assumption of Hamilton's rule, that the loci are assumed to have no pleiotropic direct effect on the recipient's fitness, only the indirect genetic or kin effect. When this assumption is relaxed, Hamilton's rule becomes

$$[r + \{\mathrm{cov}(A_o,A_q)/V_{Aq}\}] > |c|/b \quad (2)$$

where '$r$' is the coefficient of relationship, $\{\mathrm{cov}(A_o,A_q)/V_{Aq}\}$ is the genetic regression of the direct effects of genes on their kin effects due to pleiotropy at individual loci, '$c$' is the cost or selection gradient against the altruistic phenotype, and '$b$' is the benefit or selection gradient for the trait in the recipient[9,20]. Interestingly, the new genetic regression term can dominate the inequality in eqn 2. The coefficient of relationship ($r$) is typically bounded by zero and one while the genetic regression is bounded by negative and positive infinity. Thus, when the regression term is negative and less than '$-r$', altruism cannot evolve regardless of minimal costs and enormous benefits. Likewise, when the regression term is positive and '$r$' plus the regression term is greater than one, costs can exceed benefits and altruism can still evolve. The system of genetic relationships between direct and kin effects measured by the genetic regression of direct on kin effects can be much more important in determining whether altruism evolves than the degree of kinship among the actors or the relative strengths of the fitness costs and benefits of altruism.

Cheverud[20] used this quantitative genetic version of Hamilton's rule to examine the evolution of 'altruistic' maternal traits (e.g. lactation and other forms of parental care). This approach provides a useful alternative to the classic method of simply considering offspring fitness (e.g. offspring survival) as a component of maternal fitness. In this approach fitness belongs to the individual but can be affected by relatives. This avoids the problem of assigning one individual fitness to another individual.

genetic variance of the trait ($G$, see Ref. 7). However, when IGEs exist, $C_{az}$ is not equivalent to $G$ (Refs 6,23,24).

When maternal effects exist, $C_{az}$ and $G$ differ because offspring receive both environments and genes from their parents, creating a covariance between the genes that they inherit and the environment that they experience. Depending on the sign of the covariance, this relationship between the genes that are inherited and the environment that is experienced can either accentuate or diminish the covariance between individuals' genes and their phenotype. For example, when a maternal trait affects the same trait in the offspring the covariance between the offspring's additive genetic value for the maternal trait and its phenotypic value for that trait ($C_{az}$) is, at equilibrium in the absence of selection, $[2G/(2-m)]$, where $G$ is the additive genetic variance for the trait and $m$ is the maternal effect coefficient that measures the degree to which the offspring phenotype is determined by the maternal phenotype beyond the direct maternal genetic contribution (Ref. 23; see also Boxes 1–3). From this equation it is clear that when the maternal effect is positive (i.e. $m > 0$; the trait positively affects the expression of the same trait in the offspring), the covariance between the additive genetic value and the phenotypic value will be greater than the additive genetic variance for that trait. Likewise, when the maternal effect is negative ($m < 0$), the covariance will be less than the additive variance. Maternal effects on any type of offspring trait (for example, the maternal trait affects a different offspring character) have a similar result. Thus, when there are positive maternal effects, selection will result in larger changes in the mean additive genetic value for the maternal trait than when they are absent (and smaller changes when $m$ is negative; see Fig. 1). Maternal effects therefore can alter the expected rate of evolution[20,23–26], especially for traits expressed early in life when maternal effects have been found to be especially important (Box 5).

When unrelated individuals contribute environmental components to the phenotype the covariance between the genes inherited and the environment experienced does not exist. However, a covariance between the genes of an individual and the social environment that it experiences may exist because individuals both experience a particular social environment and also contribute to that environment[6,7]. For example, if an individual's level of aggression is positively affected by the aggression of its social partners, and vice versa, then the covariance between additive genetic values for aggression and phenotypic values will be increased by a feedback loop[6,7]. In this case the covariance ($C_{az}$) is $G/(1-\psi^2)$ where $\psi$ is an interaction effect coefficient that describes the degree to which an individual's phenotype is affected by the phenotype of its social partners (Refs 6,7; see also Boxes 1–3). Thus, due to the feedback that a trait has on its own expression, the covariance between the additive genetic value and phenotypic value is accentuated even when the feedback is negative ($\psi < 0$). Similar effects occur whenever a trait is part of a feedback loop and affects its own expression by altering the social environment, but does not directly affect the expression of the same trait in conspecifics[6,7].

### *The evolving environment*

Evolution, or in a quantitative genetic terminology, cross-generational changes in the mean phenotype, is predicted by changes in all of the components contributing to the phenotype. When IGEs exist, three components contribute to the phenotype: (1) direct genetic effects (i.e. the additive genetic value), (2) environmental effects, and (3) indirect genetic effects[6–9,23]. Traditional quantitative genetic models of evolution consider only a change in the direct genetic component, assuming that the average contribution of the environment is always zero (that is, is random and thus cannot evolve). This assumption is reasonable for abiotic influences such as temperature. However, when IGEs occur, altering the mean

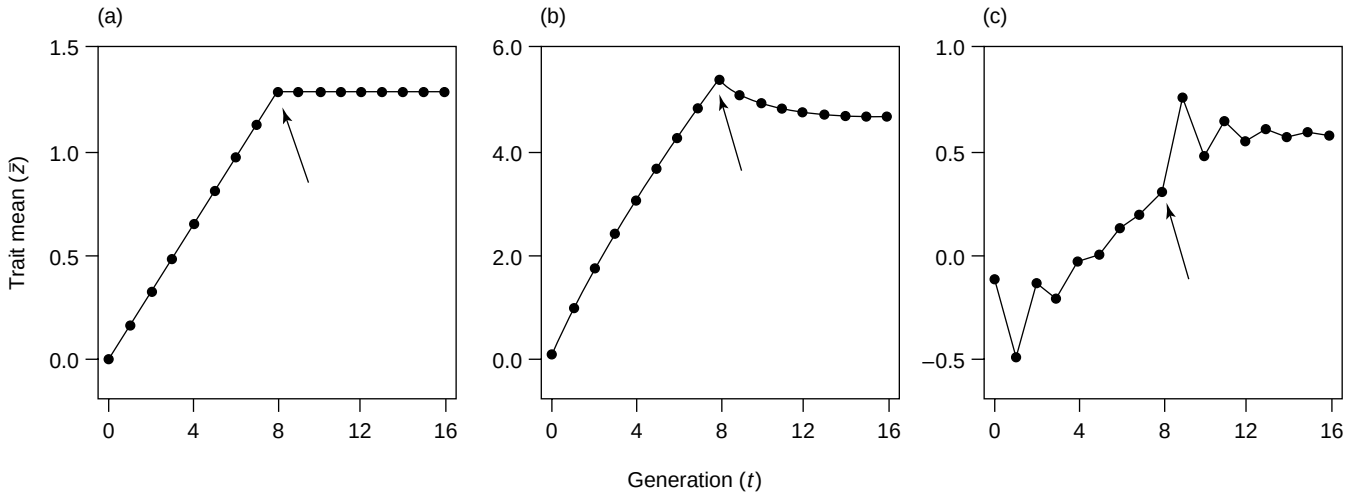



**Fig. 1.** Evolutionary trajectories showing the mean value of a maternal trait that affects the expression of the same trait in the offspring. Trajectories are calculated using the equation for the change in the mean of the character across one generation; $\Delta\bar{z}(t) = [C_{az} + mP]\beta_{(t)} + m\Delta\bar{z}_{(t-1)} - mP\beta_{(t-1)}$ (cf. Ref. 23, eqn 3), where $P$ is the phenotypic variance of the trait, β is a directional selection gradient[22], and $t$ refers to the current generation. All other symbols are given in the text. For all trajectories, $\beta = 0.4$, $G = 0.4$ and $E = 1.0$ (where $E$ is the environmental variance); using these values, $C_{az}$ is calculated as in the text. $P$ can be calculated using eqn A14 in Ref. 23. Directional selection was applied for the first 8 generations. The generation where selection ceased is indicated by an arrow. (a) Shows the case for no maternal effect ($m = 0$), (b) shows the case for a positive maternal effect ($m = 0.6$), and (c) shows the case for a negative maternal effect ($m = -0.6$).

indirect genetic contribution will also contribute to changes in the mean phenotype. This can be thought of as an evolving environment, where a genetically-based environment contributes to phenotypic changes.

The effect of this evolving environmental effect is particularly clear in maternal effects models where changes in the mean maternal phenotype shift the mean phenotypic value of the offspring in the next generation[6–9,23]. For example, if higher milk production produces bigger babies, a change in the mean milk production will result in an evolutionary change in offspring size[8,23,25]. Offspring size therefore shows a response to selection on milk production[25]. A similar effect is seen in cases where unrelated individuals provide environmental contributions to the phenotype[6,7]. If a ritualized display reduces the expression of physical aggression in a social partner, then an evolutionary change in the display will lead to a change in the mean level of physical aggression, even when there is no change in the direct genetic effect on aggression[6,7]. When a trait affects the expression of that same trait in conspecifics (e.g. aggression or competitive performance), small changes in the direct genetic component can result in larger changes in the expression of the trait due to the synergistic feedback that occurs when a trait affects its own expression[6,7].

## Response to selection

The rate of evolutionary change in response to selection is generally thought to be proportional to the amount of additive genetic variance[13]. Multivariate re-formulations of this rule show that it is actually the pattern of covariances among direct genetic effects and phenotypes that determines the rate of change in additive genetic values[22]. For traits with mendelian inheritance, this pattern is captured in the $G$-matrix that describes the additive genetic variances and covariances for traits[22]. Covariances among characters can either accelerate or impede the rate of evolutionary response, depending on their sign[27]. Under traditional mendelian inheritance, selection (often represented by the selection gradient, β; Ref. 22) acts on phenotypes, and through the genotype–phenotype relationship ($C_{az}$) alters the distribution of direct genetic effects. The change in the average trait ($\Delta\bar{z}$) in the next generation is traditionally predicted by the change in the direct genetic component, $\Delta\bar{z} = G\beta$ or more generally $\Delta\bar{z} = C_{az}\beta$ (which, in the univariate case, is equivalent to the familiar breeder's equation $R = h^2S$; Ref. 1). IGEs alter not only the genotype–phenotype relationship ($C_{az} \neq G$), but also alter the translation of genetic effects into the phenotype. When IGEs are present, some of the assumptions of the common predictive models of evolutionary change are violated and the dynamics of response to selection take on some non-intuitive properties. Most of these phenomena occur with any IGE, but a few are specific consequences of cross-generational IGEs (i.e. maternal and paternal genetic effects)[6–9,23].

### Box 5. Development and persistence of maternal effects

Most of our data on maternal effects come from studies of mammalian growth in body size. Both maternal uterine environment and post-natal care and feeding can have a profound affect on offspring phenotype. In many mammals, approximately 50% of the variance in pre-weaning body weight is due to the maternal effect[8]. In the rare instances in which the heritability of maternal performance for offspring weight has been estimated, estimates center on 40% (see Ref. 20). Thus, there is substantial heritable variance in early post-natal body size due to maternal effects (~20% of the variance). This is also a time in which there is a great opportunity for selection in natural populations in that it is not unusual for feral mammals to experience a 20–50% mortality rate in the period before and just after weaning[39]. Therefore, one expects strong selection on maternal performance for weanling size. After offspring are removed from the maternal environment, typically after weaning, the magnitude of variance due to maternal effects declines to about 10–20% of the variance in adult rodents[9,20]. It is possible that – for animals that have a long growing period outside of maternal influence – this proportion of variance would decline to zero in adulthood. However, animals that are dependent on their mothers for a greater proportion of their growth period would display higher percentages of variance due to maternal effects even in adulthood.

The genetic covariance between direct and maternal effects for offspring weight also varies dramatically with age[20]. In mammals, the covariance is usually low and positive at the earliest ages, becomes strongly negative around the period of weaning, and becomes positive at later ages. This indicates that at weaning, genes that result in high offspring weight through their direct effects in the offspring also result in low offspring weight through their indirect, maternal effects.

### *General consequences of IGE for evolutionary response*

The most dramatic consequence of IGEs is that direct additive genetic variance (often expressed as heritability) is not necessary for predictable evolutionary response to

selection. This result is possible because a portion of the environmental variance is effectively inherited (i.e. the environmental effects of both related and unrelated conspecifics). Traits that may have no direct genetic basis can therefore change across generations because the environment itself can evolve.

When IGEs exist, the genotype–phenotype relationship (as represented by the $C_{az}$ matrix) is usually a function of the *G*-matrix directly proportional to the strength of indirect effect coefficient [e.g. the strength of maternal effect, *m*, paternal effect, *f* (Ref. 24), or interaction effect, $\psi$ (Refs 6,7)]. Thus, when interactions or maternal effects have a large positive effect, per-generation evolutionary change can be greater than for traits with simple mendelian inheritance (Fig. 1). Likewise, negative effects often act to reduce the rate of change per generation (Fig. 1).

When IGEs occur, both the direct and indirect genetic components of the phenotype may respond to selection. Since IGEs alter our usual predictions about changes in genetic values associated with phenotypic selection, they modify the expected response to selection of both these components. Thus, the effect of evolving environments serves to exaggerate the effect of the altered genotype–phenotype relationship. The importance of this process is obvious for traits like aggressive behavior that are expected to have positive reciprocal effects on themselves (that is, alter the expression of the same trait in social partners). Aggression in one individual is likely to be increased by aggression in another and vice versa, leading to positively synergistic IGEs. If the influence of aggression on aggression is very strong, this trait might evolve faster than expected under mendelian inheritance[6,7,23]. IGEs may therefore help explain the perception that social behavior evolves faster than 'ordinary' traits (e.g. morphological traits; Refs 6,7,28; but see Ref. 29).

The evolving environment can also effect changes in short-term direction of evolution (Fig. 1). Patterns of genetic correlation can cause short-term 'maladaptive' evolution because the response to selection on negatively correlated traits can outweigh the response to selection directly targeting a trait[6,9,20,23]. The pattern of IGEs can exaggerate the pattern of covariances between direct genetic effects and phenotypes depending on the strength of the indirect effect. Coupled with the evolving environmental component, the net effect is that some traits will actually change in the direction opposite to the selection targeting them (e.g. Refs 20, 23,24,30,31). While most models predict that long term evolution will eventually drive a population to a local adaptive peak (if selection remains constant), short term evolutionary digressions can sometimes move a population into the attractive realm of new peaks, generating peak shifts[32].

Because IGEs represent an environmental effect on a trait that has a predictable value, they can increase the phenotypic resemblance among individuals sharing environments (IGEs) beyond their genetic similarity (i.e. relatedness). This makes it difficult to partition additive genetic effects and thus to estimate heritabilities or additive genetic variances[1]. By increasing the phenotypic resemblance of sibs, IGEs alter the distribution of phenotypic variance within and among families. Because maternal effects usually act to reduce the within-family variance (and thus increase the among-family component of variance) they increase the effectiveness of among-family selection[33]. Selection acting at the level above the individual has many evolutionary implications[33]. For example, selection acting at the family level makes several genetic variance components available for selection (e.g. epistatic variances) that are not available for the evolution of ordinary traits[33].

### *Consequences of cross-generational IGEs*

Some additional non-intuitive evolutionary dynamics can arise when IGEs cross generations, such as when parents influence traits in their offspring. The evolutionary processes are fundamentally the same as those described for other traits experiencing IGEs, except that in this case the IGEs exist in the previous generation. The primary result is that evolutionary change in one generation is actually determined in part by the evolution that occurred in the traits expressed in the previous generation, resulting in evolutionary 'time-lags' or momentum[23–25,34] (Fig. 1). Thus, the evolutionary response to selection in one generation cannot easily be partitioned from the response to selection in previous generations[23,33].

The evolutionary 'momentum' due to maternal effects causes the population to continue evolving after selection ceases[23]. Evolution continues because changes in the parental trait result in changes in the offspring trait, and thus the response to selection shown in the previous generation contributes to the response in the current generation[23,24,34]. This momentum continues indefinitely, but the magnitude of the effect dampens geometrically so that the most dramatic effect occurs in the first generation after selection ceases (Fig. 1). Traits that have cyclic effects or complicated inheritance may experience longer time lags[23,24].

Maternal and Paternal inheritance introduces time lags into the evolutionary response to selection[23]. This may result in temporary reversals in the direction of evolution when the net correlation between parents and offspring is negative[9,20,23–25] (Fig. 1c). When reversals occur, the response to selection changes sign several times (i.e. oscillates), with the magnitude of the oscillations damping in each generation (Fig. 1c). After several cycles of these oscillations, the population evolves in the direction favored by selection (Fig. 1). Regardless of the covariance between parents and offspring, time lags cause the response to selection to change every generation, even under a constant strength of directional selection[23,34]. A constant rate of response to selection is approached asymptotically (Fig. 1).

The classic example of evolutionary reversals caused by selection on a maternally effected trait is litter size in mice[25], where large females have many offspring, but offspring from these litters are small. As a result, their own litters are small in number, but the offspring grow to be large. A negative correlation between mothers and daughters arises that leads to a maladaptive reversal in the response to selection because of the process described above. This negative covariance is common (reviewed in Refs 9,34) and results in a limited response to selection.

## Conclusions and future directions

Because IGEs alter our predictions about many important evolutionary processes, an understanding of their role is essential to paint a complete picture of the evolutionary process. While there are a number of theoretical treatments of the role of indirect genetic effects in evolution, and an increasing body of evidence that indirect genetic effects occur in natural populations, there are fewer empirical studies that have directly tested these evolutionary models. Most of the empirical data on IGEs in evolution come from agricultural sciences where studies of maternal effects, and in some cases paternal effects, are relatively common[2,3,9,34]. However, nearly all of these agricultural studies use an approach that measures maternal performance (Boxes 1–3) so that there is a dearth of studies that have directly measured individual maternal traits and attempted to make predictions about multivariate evolution. There are also very few

studies that have directly demonstrated the importance of IGEs in the evolution of interactions other than those between parent and offspring (but see Ref. 35). Again, most of these have been agricultural studies concerned with associate effects resulting from competition among genotypes of plants (e.g. Refs 4,5). Clearly, there is a need for explicit studies of the evolutionary consequences of IGEs in a diversity of interactions in natural populations. In addition, the wider application of these genetic models may provide insight into related phenomena such as cultural evolution[36] and social behavior. These studies will only come, however, when empiricists resist treating IGEs as annoyances to be statistically controlled.

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