# The quantitative genetic theory of parental effects

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## 15.1 Introduction

There are many theoretical approaches for studying the evolution of parental care and parent–offspring interactions (Chapters 2, 7, 9, and 16; Mock and Parker 1997), but here I focus on theory developed in the field of quantitative genetics. The reasons for this are twofold; first, they allow tractable dynamic models for phenotypes determined by multiple genes and the environment. Second, theory and application are so entwined in quantitative genetics that the development of theory is nearly always followed, or sometimes even preceded, by methods to estimate the relevant parameters from data—a useful resource for empiricists. However, my main aim is not to champion the quantitative genetic approach over others, but to clarify how quantitative genetic models of parent–offspring interaction work, and how the key concepts fit with more familiar ideas from behavioural ecology. Traditionally, the two approaches have often focused on fundamentally different things; with behavioural ecology focusing on parent–offspring conflict and quantitative genetics focusing on parent–offspring co-adaptation (reviewed in Smiseth et al. 2008; Hinde et al. 2010). The goal of this chapter is to dispel the perception that the tension between the interests of the individual and the interests of kin does not have a natural place in the quantitative genetic approach, and to clarify that its omission from much recent theoretical and empirical work is not warranted. Its omission seems to be inadvertent and may have arisen because those applying the quantitative genetic approach have continued to associate concepts from behavioural ecology with concepts of the same name from quantitative

genetics, particularly those pertaining to fitness and selection (Chapter 1).

Quantitative genetic models of parental effects are designed to predict evolutionary change in suites of traits that affect traits expressed in offspring and/or are affected by traits expressed in parents. In the first section I give a detailed exposition of the Kirkpatrick–Lande model (hence forth the K–L model; Kirkpatrick and Lande 1989, 1992; Lande and Kirkpatrick 1990), a model that generalized a great deal of previous theory in which the phenotype and fitness of an individual was influenced by the phenotypes of its parents (Dickerson 1947; Willham 1963, 1972; Falconer 1965; Cheverud 1984). The model is difficult to understand and so the intention is to derive and explain it in a way that is both didactic and complementary to the original work, with special emphasis on clarifying what is meant by selection. To facilitate this, I work through a simple biological example in the second section and highlight the relationship between the selection parameters of the K–L model and concepts from behavioural ecology and life-history evolution. By doing this I argue that recent theoretical and empirical work in quantitative genetics has assumed values for these selection parameters that contradict central ideas from behavioural ecology that have wide empirical support. In the third section I describe the Willham model (Willham 1963, 1972), a special case of the K–L model widely used by empiricists, and show that by changing assumptions about the form of selection we come to very different conclusions about what types of genetic architecture act as constraints to evolutionary change. Following

Cheverud (1984) I place the Willham model in the context of Hamilton's rule in order to further elucidate the meaning of selection, and relatedness, in quantitative genetic models of parent–offspring interaction.

In most quantitative genetic models, values for the genetic parameters, such as genetic correlations, are assumed and the main focus is on evolutionary change in the mean. However, explaining why certain values for the genetic parameters are more likely than others is an interesting avenue of research, particularly in the context of social interactions where they appear in the relatedness term of Hamilton's rule (see also Chapter 16). In the fourth section I discuss why we might expect the genetic parameters of traits involved in parent–offspring interactions to be different from those of other traits, but suggest that current expectations about the genetic architecture of traits involved in parent–offspring interactions (Wolf and Brodie 1998) may be challenged when we entertain more reasonable patterns of selection and mutation.

In order to understand parent–offspring interactions fully the K–L model has one important short-coming: it fails to account for the fact that offspring are not passive vessels receiving parental care, but often express traits such as begging that modify parental behaviour. A general framework for modelling a wide range of interactions is the indirect genetic effect (IGE) approach (Moore et al. 1997; Wolf et al. 1999; McGlothlin et al. 2010) which has recently been used to analyse a model in which parents affect offspring *and* offspring affect parents (Kölliker et al. 2005). Although such an approach may become the quantitative genetic method of choice for modelling these types of interactions, in the fourth section I identify a conceptual difficulty with the IGE approach that arises when it is applied to parent–offspring interactions. Although this contradiction may have few practical consequences it is hoped that more theoretical work identifying any consequences are pursued before the IGE approach is more widely used.

In order to prevent the chapter from becoming too turgid, readers can find various notes concerning the smaller and less relevant technical details in Box 15.1. These are referenced in the body of the text.

## 15.2 The K–L model

The model of Kirkpatrick and Lande in its most general form follows the evolutionary dynamics of a suite of traits using a quantitative genetic approach (Kirkpatrick and Lande 1989, 1992; Lande and Kirkpatrick 1990). However, unlike the multivariate breeders' equation (Lande 1979) an individual's trait values can be, in part, determined by the trait values expressed in that individual's parents (Box 15.1, note 1). The K–L model generalizes a great deal of previous work and remains the most comprehensive theoretical treatment of the subject. However, it is not easy to understand. In part, this is because theory is never easy, but it is also because some of the notation is ambiguous, the discussion of key concepts and terms is often cursory, and there are some confusing mistakes only some of which are corrected in a little known erratum (Kirkpatrick and Lande 1992). In order to understand how the K–L model works it will be useful to take a concrete example, and so for the majority of the chapter I will consider two traits: weight at independence (henceforth *body-size*) and the amount of food that an individual provisions each of its offspring (henceforth *provisioning*).

For clarity I will use the words *influence* and *affect* and their derivatives in a precise way throughout the chapter: if by provisioning its offspring a parent can make it larger, and by being larger that offspring has higher fitness I say that parental provisioning *affects* offspring body-size, and by this *influences* offspring fitness. This is what a quantitative geneticist means by parental effect—parental provisioning has no parental effect on fitness in this instance, because there is no direct causal link. Another source of confusion is that the word parental can refer to a generation or a role, and sometimes traits when they are specific to a role. In the context of generation I will try and use the words 'previous generation' ($t - 1$), 'current generation' ($t$), and 'future generation' ($t + 1$).

The trait values of an individual from the future generation follow the model:

$$\mathbf{z}(t + 1) = \mathbf{a}(t + 1) + \mathbf{e}(t + 1) + \mathbf{M}\bar{\mathbf{z}}(t) \qquad (15.1)$$

where $z$ denotes phenotype, $a$ additive genetic value (or breeding value), and $e$ a non-heritable

## Box 15.1  Additional notes on quantitative genetic models of parental effects

Note 1: Much of the work that I discuss actually considers maternal effects only, but here I elucidate the theory in the context of parental effects since the extension is straightforward.

Note 2: Although it will be obvious to many it is worth pointing out that $E_t [\mathbf{z}(t)] = E_t [\bar{\mathbf{z}}(\mathbf{t})]$ since each individual has two parents.

Note 3: These are given incorrectly in Kirkpatrick and Lande (1989) but are corrected in Kirkpatrick and Lande (1992).

Note 4: A special case of this equation is given incorrectly as Eq.10 in Kirkpatrick and Lande (1989) and appears corrected as Eq.5 in Kirkpatrick and Lande (1992). Nevertheless, the section dealing directly with the Willham model remains very confusing because they use the term $\beta_m$ and refer to it as a parental selection gradient before they introduce the concept of parental selection. However, $\beta_m$ is *not* a parental selection gradient in the Willham section, it is the direct selection gradient on trait $m$—in my notation $\beta_{I,2}$. Part of the difficulty with Kirkpatrick and Lande (1989) is that the subscripts $o$ and $m$ switch meaning throughout the manuscript: sometimes they refer to the role of the individual in which the trait is expressed (i.e. $o$ indicates body-size, and $m$ indicates provisioning) as in the Willham section, and sometimes they refer to the role an individual is playing (i.e. $o$ indicates a trait in an individual, and $m$ indicates a trait expressed by an individual's mother) as in the distinction between direct and parental selection.

Note 5: When the selection gradients are zero for the traits that parentally effect body-size (i.e. the Willham model), evolutionary change in the total parental effect (parental performance) caused by a correlated response to selection is correctly predicted, as is the change in body-size. However if the selection gradients are non-zero (i.e. Cheverud's extension), then evolutionary change cannot be predicted by measuring a composite selection gradient on parental performance (Kirkpatrick and Lande 1989), except under very specific assumptions. Technically then, Cheverud's extension only applies to cases where a trait is parentally affected by a *single* trait expressed in parents, thus undermining the strength of the Willham

model to empiricists studying natural selection. However, from a practical perspective, estimating the selection gradient on the composite parental performance and applying it to the genetic parameters of the Willham model may prove to be more precise and more accurate than attempting the full K–L approach. Exploring the bounds of error on Cheverud's extension to the Willham model when empiricists are challenged by modest sample sizes and the danger of a misspecified K–L model would seem like a worthwhile task (See Discussion).

Note 6: Cheverud (1984) mistakenly uses selection intensities rather than standardized selection gradients through out the paper. In addition, the derivation is not exactly equivalent to the Willham model because what is called parental performance is actually something *proportional* to parental performance such that $m_{1,2}$ is positive but not necessarily equal to one. This may sound like a small point, but the relationship between the Willham model and Hamilton's rule given by Cheverud (1984) is easier to interpret when $m_{1,2} = 1$ (see below).

Note 7: Note that in Cheverud (1984) parental performance is sex-limited (it is maternal performance) and so the LHS ($-\beta_{I,2}$) is divided by 2. If a trait is sex-limited then *direct* selection gradients associated with that trait should be halved, but parental selection gradients should remain untouched. For example, if only females provision then only half the individuals (females) will experience fitness variation caused directly by the trait. However, under parental selection all individuals will experience fitness variation caused by the trait because all individuals have a mother, even males.

Note 8: In fact, rather than setting $m_{1,2} = 1$, Cheverud (1984) has this as a free parameter and defines the relatedness term in Hamilton's rule as $m_{1,2} \left( \frac{g_{1,2}}{g_{2,2}} + 0.5 \right)$. However, I think it makes more sense to keep the relatedness term as I have done, and think of the benefit as $m_{1,2}\beta_{I,1}$. The advantage of this is that it puts the cost and benefit in the same units; the benefit is how much does a unit change in parental performance change offspring fitness (via a change in the offspring trait).

Note 9: I use the (matrix) notation **A** for the *vector* of total breeding values following McGlothlin et al. (2010).

residual (Falconer and Mackay 1996; Bulmer 1985; Lynch and Walsh 1998). They appear in bold face because they are vectors and include terms for both traits: body-size ($z_1$) and provisioning ($z_2$). **M** is the

parental effect coefficient matrix and $\bar{z}(t)$ is the average trait value expressed by an individual's parents. The product $\mathbf{M}\bar{z}(t)$ is a vector of parental effects for the traits, with the $i^{th}$ element being the total effect

of traits expressed in the individual's parents on the $i^{th}$ trait of the individual: $\sum_j m_{i,j} \bar{z}_j(t)$, where $m_{i,j}$ is the effect that a unit change in trait $j$ expressed in the individual's parents has on trait $i$.

It is important to state at which point in the future generation's life-cycle *all* quantities appearing in Equation 15.1 are measured. Conventionally, the most natural point is to measure the traits when the future generation are zygotes, because then we are measuring the future generation's traits before they have been exposed to selection. After all, quantitative geneticists are interested in predicting the mean phenotype of the future generation without having to specify the form of selection that may act on them. Of course, zygotes express neither body-size nor provisioning, so the trait values at this point are hypothetical: it would be the expected trait values if selection ceased until expression.

In order to work out the mean phenotype in the future generation we can simply sum the expectations for each term:

$$\underset{t+1}{E}[\mathbf{z}(t+1)] = \underset{t+1}{E}[\mathbf{a}(t+1)] + \underset{t+1}{E}[\mathbf{e}(t+1)] + \mathbf{M} \underset{t+1}{E}[\bar{\mathbf{z}}(t)]$$

(15.2)

where I use subscripts to denote the generation over which the expectation is taken.

This notation singles out $\underset{t+1}{E}[\bar{\mathbf{z}}(t)]$ as being something odd—we're taking the expectation of a trait expressed in the current generation, $\bar{\mathbf{z}}(t)$, but over individuals in the future generation, $\underset{t+1}{E}$. Indeed, it has been one source of confusion. Because of selection in the current generation the average trait values of individuals in the current generation when they were zygotes, $\underset{t}{E}[\bar{\mathbf{z}}(t)]$, differ from the mean trait values of the current generation that go on to become parents (Box 15.1, note 2). K–L often use the term 'individuals measured after selection' to denote $\underset{t+1}{E}[\bar{\mathbf{z}}(t)]$, which at first reading may suggest that the mean trait value of the current generation after selection is the mean trait value of those individuals that go on to be parents. This is not what is intended. More correctly, the mean of the current generation after selection is the mean trait value of the current generation that an individual of the future generation experiences. When the trait is independent of fertility then this quantity is the same as measuring the mean trait value of individ-

uals that go on to be parents, but generally 'after selection' includes both viability selection and fertility selection. To take an example, let's imagine four zygotes (two of each sex) from the current generation form two pairs, and that the provisioning values of these pairs are -1 and 1. Consequently, $\underset{t}{E}[\bar{z}_2(t)] = 0$. Let's imagine that all zygotes survive to be parents and that pair 1 contributes 1 zygote to the future generation and pair 2 contributes 2 zygotes. The three individuals of the future generation will then experience provisioning values, -1, 1, and 1 giving $\underset{t+1}{E}[\bar{z}_2(t)] = \frac{1}{3}$. Essentially, selection modifies the distribution of traits in the current generation *experienced* by individuals of the future generation because fit parents interact with more individuals of the next generation than less fit parents.

Equation 15.2 does not appear to be very predictive: terms with $t+1$ are still appearing on the right hand side and it would be nice to write them down using terms with $t$ only. We can do so using the following approximation (i), assumption (ii), and identity (iii):

i) One of the foundations of quantitative genetics developed by Fisher (1918) is the concept of the breeding value, which has the property:

$$\underset{t+1}{E}[\mathbf{a}(t+1)] \approx \underset{t+1}{E}[\mathbf{a}(t)]$$

since breeding values are transmitted without bias from parents to their offspring under weak selection and random mating (Falconer 1985).

ii) We will assume that the residual environment of the future generation at conception is the same as the residual environment that the current generation experienced at conception:

$$\underset{t+1}{E}[\mathbf{e}(t+1)] = \underset{t}{E}[\mathbf{e}(t)]$$

This may seem untenable, but the idea is that if there are differences in the average environment between generations such as temperature, parasites, or food availability, their effects on the traits could be controlled for, at least hypothetically.

iii) One of the most powerful identities in evolutionary biology, first shown by Robertson (1966) is:

$$\underset{t+1}{E}[\mathbf{z}(t)] = \underset{t}{E}[\mathbf{z}(t)] + C\underset{t}{O}V(\mathbf{z}(t), w(t))$$

$$= \underset{t}{E}[\mathbf{z}(t)] + S(\mathbf{z}(t))$$

where $w$ is the relative fitness of an individual, and fitness in our case is measured as the number of zygotes. $S()$ is known as a selection differential and is the change in the mean value measured before and 'after selection'. Although I have expressed Robertson's (1966) result in terms of trait value, $z$ could be exchanged with anything measurable (for example breeding value) and the result would still hold. It is also important to realize that this result makes no assumption about the relationship between relative fitness and $z$—it could be linear, it could be loop-the-loop.

We can substitute these three results into Equation 15.2, to give:

$$\underset{t+1}{E}[\mathbf{z}(t + 1)] = \underset{t}{E}[\mathbf{a}(t)] + S(\mathbf{a}(t))$$

$$+\underset{t}{E}[\mathbf{e}(t)] + \mathbf{M}\left(\underset{t}{E}[\bar{\mathbf{z}}(t)] + S(\bar{\mathbf{z}}(t))\right)$$

To obtain the mean phenotype in the current generation we can also replace $t$ with $t - 1$ in Equation 15.2:

$$\underset{t}{E}[\mathbf{z}(t)] = \underset{t}{E}[\mathbf{a}(t)] + \underset{t}{E}[\mathbf{e}(t)] + \mathbf{M}\underset{t}{E}[\bar{\mathbf{z}}(t - 1)]$$

and then use Robertson's (1966) identity (iii):

$$\underset{t}{E}[\mathbf{z}(t)] = \underset{t}{E}[\mathbf{a}(t)] + \underset{t}{E}[\mathbf{e}(t)]$$

$$+\mathbf{M}\left(\underset{t-1}{E}[\bar{\mathbf{z}}(t - 1)] + S(\bar{\mathbf{z}}(t - 1))\right)$$

The change in mean phenotype is therefore given as:

$$\underset{t}{\Delta}\mathbf{z} = S(\mathbf{a}(t)) + \mathbf{M}\left(\underset{t-1}{\Delta} S(\bar{\mathbf{z}}) + \underset{t-1}{\Delta} \bar{\mathbf{z}}\right) \qquad (15.3)$$

where $\underset{t}{\Delta}$ indicates a change in a quantity from generation $t$ to $t + 1$.

In words, $S(\mathbf{a}(t))$ represents evolutionary change *sensu stricto*; caused by selection altering gene frequencies (see also Bonduriansky and Day 2009). However, it is apparent that changes in the parental effects can also cause change, not because the effect of the parental traits change (**M** is fixed) but because the actual values of the parental traits experienced

by offspring are altered. This alteration can be the result of two processes; it can arise because the trait values of individuals that *may* have gone on to become parents differ between the two generations, $\underset{t-1}{\Delta} \bar{\mathbf{z}}$, but it can also arise because different patterns of selection modify the parental traits that offspring experience even when the different generations are identical as zygotes, $\underset{t-1}{\Delta} S(\bar{\mathbf{z}})$.

$S(\mathbf{a}(t))$ are the genetic covariances between traits and relative fitness, and the most widely known corollary of Robertson's (1966) result is that these are equal to evolutionary change in standard quantitative genetic models (see also Price 1972). Unfortunately, they are not always very useful for understanding the biology that underlies evolutionary processes because they substitute causation for correlation, and conflate inheritance with selection, both of which may be of interest. In many respects evolutionary biologists are not interested in what these models tell them about currently changing gene frequencies (Grafen 1988), but the insight they give into the adaptive significance of parental effects and their genetic basis. For these reasons we can rewrite these covariances in terms of inheritance (**C**) and selection ($\beta$):

$$S(\mathbf{a}(t)) = \mathbf{C}(t)\beta(t) \qquad (15.4)$$

By doing this it should be understood that we make some very strong assumptions. These assumptions have been discussed many times before, usually in the context of the multivariate breeders' equation (Lande 1979; Mitchell-Olds and Shaw 1987; Grafen 1988; Rausher 1992; Hadfield 2008; Morrissey et al. 2010) but also in the context of kin selection (Queller 1992). In essence, a sufficient condition for Equation 15.4 to be valid is that $\beta$ represents the *causal* effects of the traits on fitness and that all genetically correlated traits directly affecting fitness have been included in the analysis.

In our example $\mathbf{C}(t)$ is a matrix with two rows (because there are two traits) and an arbitrary number of columns. The element $c_{ij}$ is the covariance between the breeding values for trait $i$ in individuals in the current generation and some characteristic $j$ which selection acts upon. $\beta_j$ are selection gradients; the causal effect of characteristic $j$ on the

relative fitness of an individual in the current generation. Kirkpatrick and Lande (1989) choose two types of characteristics—the trait values of the individuals themselves, but also the trait values of the individual's parents. In this case, $\mathbf{C}$ would have four columns, two associated with traits in the individuals and two associated with traits in the individual's parents.

For clarity we can rewrite this as $\mathbf{C}(t)\beta(t) = \mathbf{C}_I(t)\beta_I(t) + \mathbf{C}_M(t)\beta_M(t)$, where terms involving the individual's own traits ($I$) are separated from those involving the traits of the individual's parents ($M$). I will call $\beta_I(t)$ *direct* selection gradients, and following K–L I will call $\beta_M(t)$ *parental* selection gradients, which are conceptually equivalent to a trait's parental effect on fitness.

The meaning of these selection gradients appears to be the single biggest source of confusion. We can think of these selection gradients as regression coefficients from a multiple regression with the response variable being the number of zygotes an individual from the current generation produces, and the predictor variables being, in this example, the four characteristics; the individual's body-size, the individual's provisioning, the individual's parent's body size and the individual's parent's provisioning. Since the regression coefficients represent the *effect* of a particular characteristic on fitness we might question whether all four gradients are likely to be non-zero, a question we will return to. For now, we will assume they could be:

$$\underset{t}{\Delta}\mathbf{z} = \mathbf{C}_I(t)\beta_I(t) + \mathbf{C}_M(t)\beta_M(t) + \mathbf{M}\left(\underset{t-1}{\Delta}S(\mathbf{z}) + \underset{t-1}{\Delta}\mathbf{z}\right) \tag{15.5}$$

The model results in complicated dynamical behaviour because change at time $t$ depends on what happened at time $t - 1$, which in turn depends on what happened at time $t - 2$. To simplify matters we can pretend that a constant pattern of selection ($\beta(t) = \beta$) has been operating on the suite of traits such that $\underset{t}{\Delta}S(\mathbf{z}) = 0$ and consequently the rate of evolutionary change has become constant $\underset{t}{\Delta}\mathbf{z} = \Delta\mathbf{z}$. If we are also willing to assume that this selection is weak then the covariances between breeding values and phenotype values will also, over time, become constant ($\mathbf{C}(t) = \mathbf{C}$), and we can derive a simplified version of Equation 15.5:

$$\Delta\mathbf{z} = \mathbf{C}_I\beta_I + \mathbf{C}_M\beta_M + \mathbf{M}\Delta\mathbf{z}$$
$$= (\mathbf{I} - \mathbf{M})^{-1}(\mathbf{C}_I\beta_I + \mathbf{C}_M\beta_M) \tag{15.6}$$

We can use this result to explore other models and the conclusions drawn from them. However, before we do so it will be useful to dig a bit deeper into what is meant by inheritance, and to do this we will work through the example of $\mathbf{C}_I$. Each element of $\mathbf{C}_I$ is the covariance between the breeding value of a trait and the phenotypic value of a trait. In the absence of parental effects $\mathbf{C}_I = \mathbf{G}$, the familiar $\mathbf{G}$ matrix that appears in the multivariate breeders equation (Lande 1979). However in the presence of parental effects complications arise. Imagine a mutation arising in a zygote that increases provisioning. This mutation will increase both the individual's breeding value and trait value for provisioning, and will contribute positively to the covariance between breeding value and phenotype. If we imagine that this mutation has no pleiotropic effect on body-size then the mutation cannot contribute to the covariance between breeding value for provisioning and phenotypic body-size, or at least not immediately. In the following generation, half the descendants of this individual are expected to carry the mutation and will therefore have a greater breeding value for provisioning. However, they will also have larger body sizes because the mutation present in their parent has had a parental effect on their body size. This will contribute positively to the covariance between breeding value for provisioning and phenotypic body-size in spite of there being no direct causal effect of the mutation on both traits (i.e $g_{1,2} = 0$ but $c_{1,2} > 0$). $\mathbf{C}_I$ must therefore capture the direct effects of genes, but also the indirect effects of genes expressed in parents. As this example shows, these covariances also take time to equilibrate, but K–L show that given certain assumptions regarding stationarity $\mathbf{C}_I$ can be expressed in terms of the genetic covariances ($\mathbf{G}$) and a geometric series of the parental effect matrix ($\mathbf{M}$) that modifies these (co)variances:

$$\mathbf{C}_I = \mathbf{G}(\mathbf{I} - \frac{1}{2}\mathbf{M}^\top)^{-1} \tag{15.7}$$

and

$$\mathbf{C}_M = \frac{1}{2}\mathbf{C}_I \tag{15.8}$$

When quantitative geneticists discuss genetic variances and genetic correlations they are referring to quantities that can be derived from $\mathbf{G}$ rather than $\mathbf{C}$.

The relationship between $\mathbf{C}_I$ and $\mathbf{C}_M$ (Equation 15.8) also allows us to substitute $\mathbf{C}_M$ in Equation 15.6 to give:

$$\Delta \mathbf{z} = (\mathbf{I} - \mathbf{M})^{-1} \, \mathbf{C}_I \left( \beta_I + \frac{1}{2} \beta_M \right)$$

$$= (\mathbf{I} - \mathbf{M})^{-1} \, \mathbf{C}_I \beta_N \qquad (15.9)$$

where K–L call $\beta_N = \beta_I + \frac{1}{2}\beta_M$ the *net* selection gradients (Box 15.1, note 3).

## 15.3   An example and its relation to behavioural ecology

The derivation given above is very general, with no restrictions placed on the form of $\mathbf{M}$ or the sign and magnitude of the selection gradients. However, the example of body-size and provisioning suggests that restrictions could be made and that certain patterns of selection and parental effect are biologically more reasonable than others. Here, I argue that in this example, and examples like it, we can often use previous experimental and theoretical work in behavioural ecology and life-history evolution in order to make *a priori* predictions about the form of selection. However, in doing so we need to be careful how ideas from these fields are translated into related concepts in quantitative genetics because mistranslations are easy.

First we will assume that the amount of provisioning by a parent positively affects offspring body-size ($m_{1,2} > 0$), but the extent to which an individual is provisioned does not have a causal effect on how much that individual goes on to provision its own offspring. Likewise we assume that parental body size has no causal effect on traits expressed in offspring, giving:

$$\mathbf{M} = \begin{bmatrix} 0 & m_{1,2} \\ 0 & 0 \end{bmatrix} \qquad (15.10)$$

In the more general derivation we allowed direct selection and parental selection on both traits. However, in many cases it would seem reasonable to set the parental selection gradients to zero. This is not equivalent to saying that parents do not influence the fitness of their offspring—they do—only that they do so by affecting ($\mathbf{M}$) some aspect of their offspring's phenotype which then has a causal effect ($\beta_I$) on their offspring's fitness (see also Chapter 16). For many aspects of parent–offspring interaction it would seem possible to posit some traits expressed in the individuals themselves that mediate the influence that parental traits have on offspring fitness (Price 1998). There are of course examples, such as brood defence or infanticidal behaviour, where it is natural to think of parental selection operating, but personally I think it is possible to distil the central features of the K–L model without it. Bearing this in mind, let us assume that being larger confers a fitness advantage ($\beta_{I,1} > 0$; Kingsolver and Pfennig 2004) but any additional fitness benefits of being provisioned, or having large parents, are absent ($\beta_{M,2} = 0$ and $\beta_{M,1} = 0$). Let's also assume that the direct selection gradient for provisioning is *negative* ($\beta_{I,2} < 0$) reflecting the fact that an individual pays a cost (in terms of current or future zygote production) by provisioning its offspring more. Although the opposing signs of these direct selection gradients are assumed, it should be emphasized that they are based on broad empirical support demonstrating the trade-off between offspring size and number (Smith and Fretwell 1974), one of the best supported of all life-history trade-offs (Stearns 1992).

Much of the recent work in quantitative genetics has been derived or interpreted under the assumption that there is no net directional component to selection on provisioning ($\beta_{N,2} = 0$). This assumption may be tenable in models in which the parents' influence on offspring fitness is solely captured through the parental selection gradients (e.g. Kölliker et al. 2010), but is unlikely to hold in models where parents are able to influence their offspring's fitness via their effect on their offspring's traits, as shown above. Changing the assumptions of such models to something more reasonable may alter the conclusions and insights drawn from such models considerably. Moreover, the assumption that there is no net directional selection on provisioning (or related traits such as litter size) also has putative empirical support (e.g. McAdam and Boutin 2004, using results from Réale et al. 2003)

although close inspection of these types of study often reveal two problems: 1) offspring survival has been included in the fitness measure and 2) an individual's traits have been dropped in favour of parental traits when calculating selection gradients such that direct selection gradients are effectively set to zero.

Although it is common in behavioural ecology to define individual fitness so that it includes the survival, and sometimes even the fecundity, of the individual's offspring (Clutton-Brock 1988), this definition of fitness—which I will call *weighted* fitness (Grafen 1982)—is not compatible with the quantitative genetic approach (Chapter 1; Cheverud and Moore 1994; Wolf and Wade 2001): individual fitness in these discrete-generation quantitative genetic models is how many zygotes an individual produces over its lifetime. The notion that at equilibrium, or conflict resolution (Godfray 1995), selection on provisioning should be stabilizing rather than directional may have arisen because these different definitions of fitness have been used out of context. Indeed, a paraphrase of Lack (1954) and related work (Charnov and Krebs 1974; Smith and Fretwell 1974): 'there is an optimal amount of provisioning that parents should engage in, and it is that which *maximises individual fitness*' certainly suggests stabilizing selection should be the norm. However, the idea that current levels of provisioning may be optimal are derived from the fact that individual fitness in this statement is the number of *surviving* offspring—weighted fitness—not fitness as a quantitative geneticist should define it.

These opposing direct selection gradients represent antagonistic selection across life-stages and at face value suggest that individuals are selected to behave like cuckoos; to take as much parental care as possible ($\beta_{I,1} > 0$) but at the same time minimize their own parental investment in order to maximize egg production ($\beta_{I,2} < 0$). Of course, in many species this selfishness is limited by kin selection, and in the behavioural ecological models described above this is dealt with, in part, by using weighted fitness. Kin selection enters into quantitative genetic models through other routes, which can most easily be understood by putting them in the context of Hamilton's (1964) rule. Before doing this however, it will be instructive to work through the Willham

model (a special case of the K–L model similar in form to this example), not only because it is an empirically tractable and well used model, but also because it was in the context of this model that Cheverud (1984) developed the key insight that direct selection on parental care would be negative.

## 15.4   The Willham model

Incorporating parental effects, or more specifically maternal effects, into quantitative genetic models has a long history in animal breeding dating at least back to Dickerson (1947). Rather than work chronologically through the developments I choose to describe directly the work of Kirkpatrick and Lande in which much of the previous work can be subsumed. In Table 15.1, using the notation of the K–L model employed above, I represent key developments in the quantitative genetic theory of parental effects. The modelling framework now known as the Willham model (Willham 1963, 1972) deserves special mention, as it has been the focus of much theoretical work and is the basis for a great deal of applied work in both plant and animal breeding (Lynch and Walsh 1998, pp. 687–714, Walsh and Lynch 2012, Chapter 21). The model has been explored from an evolutionary perspective (Cheverud 1984) and several empirical studies on wild species of plant (Platenkamp and Shaw 1993; Thiede 1998; Byers et al. 1997; Galloway et al. 2009) and animal (Wilson et al. 2005a; Kruuk and Hadfield 2007) have employed it.

Imagine a case where body-size ($z_1$) is parentally affected by a set of other traits ($z_2, z_3 \ldots z_n$) which are not themselves parentally affected by each other or body-size. In this case the parental effect coefficient matrix looks like:

$$\mathbf{M} = \begin{bmatrix} 0 & \mathbf{m}^\top \\ \mathbf{0} & \mathbf{0} \end{bmatrix} \qquad (15.11)$$

where the vector of coefficients $\mathbf{m}$ contains the effect that a unit change in traits $z_2, z_3 \ldots z_n$ expressed in an individual's parents have on that individual's body-size. In the context of the K–L model the parental effect on body-size is obtained by identifying those traits that have a parental effect (i.e $z_2, z_3 \ldots z_n$) and measuring the strength of those effects ($\mathbf{m}$) to obtain the weighted sum: $\mathbf{m}^\top \bar{z}_{2:n}$.

**Table 15.1**   A short history of maternal effects models in matrices. The K–L model places no restrictions on the number of traits, how they maternally affect each other, or whether they are under directional selection or not. However, the earlier models are special cases of the K–L model as can be seen by the size of the vector/matrices and by which maternal effect coefficients or selection gradients are zeroed out. Representing the model of Cheverud (1984) as a two-trait model may seem surprising given the text, but see Box 15.1, note 5

|   | Willham (1963, 1972) | Cheverud (1984) | Falconer (1965) | Kirkpatrick and Lande (1989) |
|---|---|---|---|---|
| $M$ | $\begin{bmatrix} 0 & m_{1,2} & \dots & m_{1,n} \\ 0 & 0 & \dots & 0 \\ \vdots & \vdots & \ddots & 0 \\ 0 & 0 & \dots & 0 \end{bmatrix}$ | $\begin{bmatrix} 0 & m_{1,2} \\ 0 & 0 \end{bmatrix}$ | $\begin{bmatrix} m_{1,1} \end{bmatrix}$ | $\begin{bmatrix} m_{1,1} & m_{1,2} & \dots & m_{1,n} \\ m_{2,1} & m_{2,2} & \dots & m_{2,n} \\ \vdots & \vdots & \ddots & \vdots \\ m_{n,1} & m_{n,2} & \dots & m_{n,n} \end{bmatrix}$ |
| $\beta_I$ | $\begin{bmatrix} \beta_1 \\ 0 \\ \vdots \\ 0 \end{bmatrix}$ | $\begin{bmatrix} \beta_1 \\ \beta_2 \end{bmatrix}$ | $\begin{bmatrix} \beta_1 \end{bmatrix}$ | $\begin{bmatrix} \beta_1 \\ \beta_2 \\ \vdots \\ \beta_n \end{bmatrix}$ |

In reality, there are likely to be many traits that have parental effects on traits such as body-size ($n$ is large), and identifying them all and measuring their effect would be a daunting task. The Willham model sidesteps this problem elegantly, although with certain limitations. Imagine a pair of individuals with multiple offspring. These offspring all have the same parental effect for body-size since $m$ is a constant and they all share the same parental phenotypes: $\bar{z}_{2:n}$. Consequently, we could imagine obtaining an estimate of the parental effect by seeing how much more similar these offspring are to each other than they are to another set of offspring from different parents. Although the offspring will also resemble each other because their breeding values are positively correlated (if there is genetic variation for body-size) it is possible to control for this source of variation if a multi-generational pedigree is available and/or manipulative reciprocal cross-fostering techniques are used (Rutledge et al. 1972). Animal breeders often use the term 'parental performance' to denote the deviation of these parental effects from the population mean, and what we can do, with some abuse of notation, is to define the second trait (i.e. $z_2$) as parental performance. This reduces the problem to a two trait model, similar in form to our assumed model for body-size and provisioning, although the parental effect coefficient is set to one:

$$\mathbf{M} = \begin{bmatrix} 0 & 1 \\ 0 & 0 \end{bmatrix} \qquad (15.12)$$

Not only does the Willham model allow a tractable empirical framework for estimating the combined effects of many parental traits, the pattern of zeros in **M** makes mathematical analysis easier. For example, evolutionary change in body size (from Equation 15.6) simplifies to (Box 15.1, note 4):

$$\Delta z_1 = \left(g_{1,1} + \frac{3}{2}g_{1,2} + \frac{1}{2}g_{2,2}\right)\beta_{I,1} + (g_{1,2} + g_{2,2})\beta_{I,2} \qquad (15.13)$$

where $g_{1,1}$ is the additive genetic variance for body-size, $g_{2,2}$ the additive genetic variance in parental performance and $g_{1,2}$ the additive genetic covariance between the two traits. Although parental selection gradients can be non-zero with certain forms of artificial selection (e.g. if a calf is allowed to breed because her mother had high milk yield) I have omitted them, and retain the direct selection gradients only. The objective of animal breeders was to select on traits such as body-size, and so naturally they set $\beta_{I,2} = 0$. Under this assumption, genetic variance in parental performance amplifies the response of body-size to selection, but a negative genetic correlation between parental performance and body-size ($g_{1,2} < 0$) constrains the response when individuals are selected

to be larger ($\beta_{I,1} > 0$) (Dickerson 1947; Willham 1972). This notion has been widely taken up in the evolutionary literature (e.g. McAdam and Boutin 2004; Wilson et al. 2005a).

In an important paper, Cheverud (1984) considered the case where selection on parental performance exists (Box 15.1, notes 5–6), and emphasized the situation where it is negative ($\beta_{I,2} < 0$) and opposite in sign to selection on body-size; a situation which, as I argue above, is much more likely to be the case than selection on body-size alone. He refers to this pattern of selection as *altruistic selection*. However, parental performance is under negative direct selection, implying selection for greater selfishness or lower provisioning, and hence, *less* altruistic values. Cheverud's (1984) choice of words appears to stem from the idea that this negative direct selection is maintained because of kin-benefits, and so the adjective *altruistic* refers to the conditions that maintain negative selection on parental provisioning rather than referring to selection itself. Rearrangement of Equation 15.13 to give:

$$\Delta z_1 = g_{1,1}\beta_{I,1} + g_{1,2}\left(\frac{3}{2}\beta_{I,1} + \beta_{I,2}\right) + g_{2,2}\left(\frac{1}{2}\beta_{I,1} + \beta_{I,2}\right)$$

$$(15.14)$$

shows that under this pattern of selection a negative genetic covariance is only a constraint on the evolution of larger body-size when the strength of selection on body-size is more than two thirds that of selection on parental performance $\frac{|\beta_1|}{|\beta_2|} > \frac{2}{3}$. Likewise, the parental genetic variance only facilitates an evolutionary response when selection on body size is more than twice that of selection on parental performance $\frac{|\beta_1|}{|\beta_2|} > 2$. It seems likely that the strength of selection on traits such as body-size will be stronger than selection on traits linked to parental performance, given that the latter often involve traits that are expressed at later life stages (Medawar 1952; Charlesworth 1994). Consequently, the notion that a negative genetic correlation acts as a constraint may have some generality, although it is unclear whether genetic variance in parental performance will always facilitate a response to selection. This may be particularly so for species with 'slow' life-histories where the relative magnitude of

the two selection gradients are likely to be more equal, and this warrants more attention.

## 15.5 Hamilton's rule

Given that the direct selection gradients appear to represent the selfish benefits afforded by a trait, it is natural to ask how kin-selection enters into the quantitative genetic framework. In the most general setting kin-selection enters through two distinct routes—directly through the effect of parental traits on offspring fitness (the parental selection gradients) and indirectly through the influence that parental traits have on offspring fitness via their effect on offspring traits. In keeping with the sections above we will initially show the relationship between the Willham model and Hamilton's rule under the second process in isolation.

Equation 15.13 describes evolutionary change in body-size in the Willham model. The equivalent equation for parental performance is:

$$\Delta z_2 = (g_{1,2} + \frac{1}{2}g_{2,2})\beta_{I,1} + g_{2,2}\beta_{I,2} \qquad (15.15)$$

from which Cheverud (1984), giving the conditions under which parental performance increases, derives a version of Hamilton's rule (Box 15.1, note 7):

$$0 < (g_{1,2} + 0.5g_{2,2})\beta_{I,1} + g_{2,2}\beta_{I,2}$$
$$-g_{2,2}\beta_{I,2} < (g_{1,2} + 0.5g_{2,2})\beta_{I,1} \qquad (15.16)$$
$$-\beta_{I,2} < \left(\frac{g_{1,2}}{g_{2,2}} + 0.5\right)\beta_{I,1}$$

where $-\beta_{I,2}$ is the cost, $\beta_{I,1}$ the benefit, and $\frac{g_{1,2}}{g_{2,2}} + 0.5$ a form of relatedness. The benefit represents the fitness advantage of increasing body-size by one unit, and the cost represents the decrease in fitness caused by increasing parental performance by one unit. Because body-size is in the same units as parental performance (and the parental coefficient is 1), the benefit can also be interpreted as the increase in fitness caused by receiving an additional unit of parental performance, thereby placing the cost and benefit on the same scale (Box 15.1, note 8). Although we may expect the relatedness term to be simply 0.5, rather than involving genetic (co)variances, this definition of relatedness is consistent with the concept in Hamilton's rule (Michod and Hamilton 1980). When the

genetic covariance between body-size and parental performance is zero Equation 15.16 reduces to a more familiar version of Hamilton's rule, $-\beta_{I,2} < 0.5\beta_{I,1}$, thus highlighting an assumption that underpins many arguments based on a simple version of Hamilton's rule (Chapter 16; Cheverud 1984).

## 15.6  The evolution of *G*

As we have seen, not only do the genetic variances and covariances play a key role in determining the rate and direction of evolutionary change, they also enter directly into the relatedness term of Hamilton's rule. However, in the models we have used above these parameters (**G**) are fixed quantities and our focus has been on the predictions regarding the direction and rate of evolutionary change ($\Delta\mathbf{z}$). In an interesting paper, Wolf and Brodie (1998) asked a slightly different question: given a certain pattern of selection how should patterns of inheritance change and what should they be at equilibrium (i.e. when evolutionary change ceases; $\Delta\mathbf{z} = 0$)? To understand why this may differ when parental effects exist, imagine two bi-allelic loci, the first affecting body-size and the second affecting provisioning. To make the verbal argument simpler we will assume complete dominance so the effects at each locus are either + or −, and we will also assume that the parental effects only depend on the maternal genotype (i.e maternal effects only). There are four possible combinations of genotypic effects: $--$, $-+$, $+-$, and $++$ where the first sign is the effect of the genotype on body-size and the second sign is the effect of the genotype on provisioning. Since the genotype at the second locus affects provisioning, it is the genotype *carried* by the mother that affects offspring body-size, rather than the genotype *transmitted* by the parents. We will denote the genotype carried by an individuals mother with an $M$, giving offspring of types $-M^-$, $-M^+$, $+M^-$, and $+M^+$ with body-sizes of -2, 0, 0, and 2 respectively. Imagine then, that there is an absence of direct selection on provisioning but direct stabilizing selection operates on offspring body-size such that a body-size of zero is optimal. In this case offspring of types $-M^+$ and $+M^-$ do better, and because mothers pass their genes to their offspring this implies that on average $-+$ and $+-$ offspring do better. This form of selec-

tion should generate a negative genetic correlation between the traits through linkage-disequilibrium.

To get a more quantitative idea of how selection generates evolutionary change in genetic (co)variances, we can obtain the difference between the genetic (co)variances 'after selection' and before selection by modifying Equations 9b and 12a from Lande (1980) and Equation 15a from Lande and Arnold (1983):

$$\underset{t+1}{COV}(\mathbf{a}(t)) - \underset{t}{COV}(\mathbf{a}(t)) = \mathbf{C}(\gamma - \beta\beta^{\top})\mathbf{C}^{\top} \quad (15.17)$$

where $\gamma$ is a matrix of quadratic selection gradients. $\underset{t}{COV}(\mathbf{a}(t)) = \mathbf{G}(t)$ and so the left-hand side of this equation is often denoted $\underset{t}{\Delta\mathbf{G}}$ (Phillips and Arnold for example, Equation 2 from 1989), but this can be misleading. Unfortunately there is no robust result which allows $\mathbf{G}(t + 1) = \underset{t+1}{COV}(\mathbf{a}(t + 1))$ to be obtained from $\underset{t+1}{COV}(\mathbf{a}(t))$ as there was for the mean breeding value (i.e. approximation i) (Walsh and Lynch see chapter 31 of 2012, for an excellent review).

Wolf and Brodie (1998) assume that pleiotropic mutations do not exist, and that changes in **G** are due to patterns of linkage-disequilibrium generated by selection. Using a multivariate extension of the Bulmer (1971) Equation derived by Tallis (Tallis and Leppard 1987, 1988; Tallis 1989) they find:

$$\mathbf{G}(t + 1) = \frac{1}{2}\left(\underset{t+1}{COV}(\mathbf{a}(t)) + \mathbf{G}(0)\right) \quad (15.18)$$

where $\mathbf{G}(0)$ is a diagonal matrix representing the fact that initially, in the absence of selection, the traits cannot be genetically correlated if mutations do not have pleiotropic effects. Equation 15.18 can be solved iteratively to find **G** at equilibrium. In accordance with the example above Wolf and Brodie (1998) assume no parental selection and direct stabilizing selection on offspring body size only:

$$(\gamma_I - \beta_I\beta_I^{\top}) = \begin{bmatrix} \gamma_{I,1} & 0 \\ 0 & 0 \end{bmatrix} = \begin{bmatrix} - & 0 \\ 0 & 0 \end{bmatrix} \quad (15.19)$$

where the right hand matrix indicates the sign of the elements. The motivation behind this choice is in part justified by the fact that *'data from humans demonstrate clear stabilizing selection for birth weight'* (Karn and Penrose 1951: see also Schluter and

Nychka 1994) (Wolf and Brodie 1998). However, Schluter and Nychka (1994) state *'Survival in human infants rises steeply with increasing birth mass ... to a broad flat dome'* thus echoing the earlier statement that *'most selection appeared to be directional'* (Schluter 1988). As discussed above, we do not expect stabilizing selection to be a dominant feature of the system, but rather positive direct selection on body-size ($\beta_{I,1} > 0$) and negative direct selection on provisioning ($\beta_{I,2} < 0$) which gives a very different pattern:

$$(\gamma_I - \beta_I \beta_I^\top) = \begin{bmatrix} -\beta_{I,1}^2 & -\beta_{I,1}\beta_{I,2} \\ -\beta_{I,2}\beta_{I,1} & -\beta_{I,2}^2 \end{bmatrix} = \begin{bmatrix} - & + \\ + & - \end{bmatrix}$$

(15.20)

Figure 15.1 shows the expected genetic correlation between body-size and provisioning under these two regimes. When offspring body-size is under direct stabilizing selection then a negative genetic correlation is expected when provisioning positively affects offspring body-size ($m_{2,1} > 0$) (Wolf and Brodie 1998). However, when directional selection is prevalent, we can see from Figure 15.1 that the sign of the genetic correlation depends critically on the relative magnitudes of selection on body-size and provisioning, with a negative genetic correlation only expected when selection on body-size is twice the magnitude of selection on provisioning. The evolution of the genetic correlation in this instance should not be seen as a result of co-adaptation, but rather the result of selection eroding 'useful' genetic variation leaving segregating variation that defines a genetic trade-off (Hazel 1943; Lande 1982; Roff 1992; Stearns 1992; Blows and Walsh 2009). This trade-off differs from the traditional concept of a trade-off in quantitative genetics because the trade-off is cross-generational, occurring between traits such as offspring number (generation $t$) and offspring size (generation $t + 1$).

The absolute magnitudes of the genetic correlation in Figure 15.1 (and Figure 1 of Kölliker et al. (2005)) are small, and indeed the effect of linkage-disequilibrium is likely to be weak and transitory in out-bred populations compared to the effect of pleiotropic mutations (Bulmer 1971; Lande 1980, 1984). Although it has been suggested that the effect of segregating pleiotropic mutations on the genetic correlation will follow the same pattern as that found for linkage-disequilibrium (Chapter 16; Wolf and Brodie 1998; Kölliker et al. 2005) this fact is far from clear (chapter 31 in Walsh and Lynch 2012) and under certain models directional selection has no impact on genetic correlations in the absence of linkage disequilibrium (Hill 1982). Consequently,
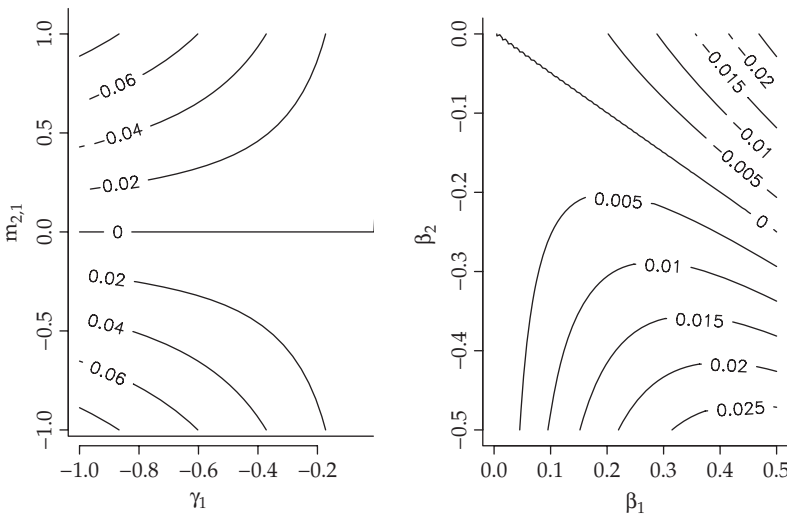


**Figure 15.1** Left: Equilibrium genetic correlation when the body-size is under stabilizing selection ($\gamma_1 < 0$), after Wolf and Brodie (1998). Right: Equilibrium genetic correlation when body-size is under positive directional selection ($\beta_1 > 0$) and provisioning under negative directional selection ($\beta_2 < 0$). $m_{2,1}$ is the effect that provisioning has on body-size, which is fixed at one in the right plot in accordance with the Willham model.

explaining any interspecific variation in the sign and magnitude of the genetic correlation needs to be done in the context of interspecific variation in patterns of selection, and even then it should be borne in mind that the genetic correlation may not be shaped by selection but may simply be the outcome of functional constraints on the types of (pleiotropic) mutations that can exist.

## 15.7   General indirect genetic effect models and parental effect models

In many taxa, offspring also affect the phenotypes of their parents, and one of the most obvious examples of this is the modulation of parental provisioning caused by offspring begging (Chapter 7). A major shortcoming of the K–L model is that it does not allow traits in offspring to affect traits in parents. However, parental effects and offspring effects can be viewed as part of a wider class of indirect effects in which an individual's phenotype may be affected by traits expressed by many different parties such as partners, competitors, predators, and parasites. A general quantitative genetic framework—the IGE (indirect genetic effect) approach—for analysing evolutionary dynamics in the presence of these interactions has been proposed (Moore et al. 1997; Wolf et al. 1999; McGlothlin et al. 2010) and was used by Kölliker et al. (2005) to explore a quantitative genetic model of offspring begging and parental provisioning. Following Wolf and Brodie (1998) they analysed a model showing that the sign of the genetic correlation between provisioning and begging would change depending on a) the parental effect that provisioning has on begging, b) the offspring effect that begging has on provisioning, and c) the relative strengths of selection on the two traits. Like Wolf and Brodie (1998) they assumed that selection on both traits was stabilizing. Rather than restate why stabilizing selection should not be the default assumption in such models and provide alternative analyses under different assumptions (e.g that begging is costly), I would like to show why the IGE approach in its current form is not obviously applicable as a general framework for modelling parent–offspring interactions. During this section, I hope that readers do

not lose sight of the fact that the IGE approach has successfully extended quantitative genetic models into new and profitable areas of research, and that richer and more realistic models such as those proposed by Kölliker et al. (2005) should be pursued. Hopefully, this short section will be a first step in delineating the types of models that can be analysed using the IGE approach and identifying the assumptions under which the interpretation given to model parameters remains valid and the models give correct predictions.

Although parent–offspring interactions are not analysed in McGlothlin et al. (2010) I will use it as the reference text for the IGE approach for two reasons. First, it is the third paper in the IGE series synthesizing results from the two earlier papers (Moore et al. 1997; Wolf et al. 1999). Second, a form of Hamilton's rule is put forward in McGlothlin et al. (2010) which is at odds with that formulated above, and is likely to cause confusion without greater clarification.

Equation 4b of McGlothlin et al. (2010) (in my notation) gives evolutionary change as:

$$\Delta \mathbf{z} = \mathbf{C}_{A_I} \beta_I + \mathbf{C}_{A_M} \beta_M \qquad (15.21)$$

where direct selection, $\beta_I$, is called non-social selection and parental selection, $\beta_M$, is called social selection. $\mathbf{C}_A$ is the covariance between *total* breeding values (**A**) (Box 15.1, note 9) and trait values, where $\mathbf{A} = (\mathbf{I} - \mathbf{M})^{-1}\mathbf{a}$ (see also Bijma et al. 2007).

Given that $\mathbf{C}_{A_I} = (\mathbf{I} - \mathbf{M})^{-1}\mathbf{C}_I$, it is apparent that Equation 15.21 cannot be a general solution to problems of this sort, because it is equivalent to K–L's result (Equation 15.6) which required quite restrictive assumptions regarding stationarity. In itself this does not seem to be a major problem, but on closer investigation the correspondence between the K–L model under stationarity and Equation 15.21 seems to be surprising given the definition of a central concept in the IGE approach: the total breeding value. Let's assume that our model conforms to the Willham model, in which case the total breeding value of an individual for body-size is simply $A_1 = a_1 + a_2$, and represents the contribution of an individual's breeding value to the average phenotype of *its* offspring. However, an individual's total breeding value should be the contribution of an individual's breeding value to the average phenotype of the off-

spring generation. Under certain types of interaction these two statements are equivalent, but when the interactions are between parent and offspring they cannot be equivalent when selection occurs. Because individuals with high fitness interact with more individuals of the offspring generation (their offspring) they contribute more to the population mean than the average of their offspring value. Consequently the change in total breeding value (as currently defined) only predicts the change in mean in the absence of selection unless one is willing to entertain hypothetical cross-fostering experiments: we would have to reallocate zygotes at random to individuals of the current generation, such that individuals that failed to breed and those that had the highest number of zygotes both raised on average equal numbers of offspring. Although the indirect genetic effect approach can accommodate group sizes greater than two (Bijma et al. 2007; McGlothlin et al. 2010), having group size as a random variable that depends on the phenotypes of the interacting individuals would require more work. I should emphasize that McGlothlin et al. (2010) do *not* consider cross-generational indirect genetic effects in their paper and that for within-generation effects the issue raised above may be a moot point given the number of interactants is not equivalent to fitness.

Not only does the IGE approach offer a general way of dealing with the quantitative genetics of social interactions, it has been suggested that the approach also extends and generalizes Hamilton's rule (McGlothlin et al. 2010, but see Gardner et al. 2011). McGlothlin et al. (2010) derive their version of Hamilton's rule (see also Queller 1992) in the univariate case which I merely state again using my own notation (Equation 22b from McGlothlin et al. 2010):

$$-\beta_I < \frac{C_{A_M}}{C_{A_I}}\beta_M \qquad (15.22)$$

where terms involving direct (non-social) selection are identified with the cost of Hamilton's rule and terms involving parental (social) selection are identified with the benefit. As McGlothlin et al. (2010) only derive their version of Hamilton's rule in a single-trait case it is hard to establish whether they proscribe equating non-social/social selection

with Hamilton's cost/benefits in a more general setting. Since McGlothlin et al. (2010) are not explicit on this matter the unwary reader may come away with the understanding that these two concepts are the same. However, earlier in this chapter I have given plausible examples where parental (social) selection gradients are zero and following Cheverud (1984) derived a version of Hamilton's rule in which both the costs and benefits are equated with different aspects of direct (non-social) selection. Consequently, it should be emphasized that social and non-social selection cannot always be identified in a straightforward way with the costs and benefits of Hamilton's rule, and treating them as such is likely to generate a great deal of confusion.

## 15.8   Discussion

Currently, the quantitative genetic approach provides a rich framework for understanding, predicting, and measuring evolutionary dynamics when parents directly affect the phenotypes of their offspring. In spite of this there is still a lot of work to do, both theoretically and empirically. In particular, the effect of offspring on parents has largely been ignored by theoretical quantitative geneticists with work by Kölliker et al. (2005) being a rare exception. Further development and refinement of these models is sorely needed. However, the assumptions of these models, and quantitative genetic models generally, need to be better aligned with ideas and beliefs held in other fields, particularly behavioural ecology (see also Chapters 7 and 16). Some of the theory outlined in this chapter needs to be re-evaluated under more reasonable assumptions regarding selection, and empiricists can play a leading role in this by estimating appropriate selection gradients from field data. Currently, much of our understanding about selection in the presence of parental effects comes from analyses that use definitions of fitness incompatible with the quantitative genetic approach and that estimate parental selection gradients while omitting direct selection gradients (e.g. McAdam and Boutin 2004; Wilson et al. 2005b). A better understanding of these processes may well give us insight into how persistent directional selection (Kingsolver and Pfennig

2004; Morrissey and Hadfield 2011) can be observed in the presence of genetic variation despite evolutionary change being small or absent (Garant et al. 2004).

The Willham model liberates empirical quantitative geneticists from having to identify and measure all the parental traits that effect the focal trait in an individual. However, when these traits are under selection the Willham model, as formulated by Cheverud (1984), fails except under the most restrictive of circumstances (Box 15.1, note 5). However, misspecification of the K–L model is a real danger in empirical studies where it may be hard or even impossible to identify and measure all traits that cause parental effects. Moreover, even if all $n$ traits that cause parental effects could be identified, estimating $n$ selection gradients, $n^2$ parental effect coefficients and $n(n + 1)/2$ genetic parameters would be a daunting task if $n$ was even moderately large. Comparing the bias and power between an (incomplete) K–L model and Cheverud's extension to the Willham model would seem like a worthwhile task, as estimating selection on parental performance would be relatively straightforward in a mixed model framework by estimating the covariance between parental performance effects and fitness (Hadfield 2008). Of particular interest is the introduction of simultaneous-recursive mixed models into quantitative genetics from econometrics (Gianola and Sorensen 2004). Not only do these methods generalize other approaches for fitting the trait-based K–L model (e.g. Lande and Price 1989; McGlothlin and Brodie 2009) they would also allow hybrid models to be fitted where parental performance can be separated into a part explained by traits measured in parents and a part explained by a Willham-like residual parental performance. All methods are data hungry and assumption-laden and empiricists need to rise to the difficult challenges of obtaining sufficient sample sizes to estimate quantitative genetic parameters with precision, having the necessary pedigree structure to estimate those parameters with minimal bias, being able to identifying and measure the relevant traits, and designing experiments to obtain estimates of causal relationships that are meaningful outside of the context in which the experiment was performed.

I have not reviewed the existing empirical literature on the quantitative genetics of parental care, as the subject has been reviewed explicitly multiple times before in both plants (Roach and Wulff 1987; Shaw and Byers 1998) and animals (Cheverud 1984; Räsänen and Kruuk 2007; Wilson and Réale 2006; Kruuk et al. 2008). Although sampling errors on genetic correlations are large it does seem like there is genuine variation across taxa in the magnitude and sign of the genetic correlation between offspring traits and traits linked to parental performance (Chapter 16). In this respect, the work of Wolf and Brodie (1998) and Kölliker et al. (2005) are welcome attempts at trying to explain this diversity, although it would seem prudent to push this work beyond the assumptions of the infinitesimal model into more realistic assumptions about the genetic basis of traits. Given that the genetic parameters also appear in the relatedness term of Hamilton's rule it would also be intriguing to see how the evolution of **G**, in the context of parent-offspring interactions, connects with ideas about the evolution of relatedness in social theory (Gardner et al. 2007).

## 15.9    Acknowledgements

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