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Ontogeny in the Family

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When ontogeny takes place in a family, and parents provide essential resources for development, the parents become an environmental component to the development of a wide range of offspring traits. Because differences among parents may partly reflect genetic variation, this environmental component contains genes and may itself evolve. Also, when offspring play an active role in family interactions, offspring become a social environmental component to parents, affecting their behavior in turn, which potentially results in reciprocal social selection. Thus, an evolutionary process of coadaptation to family life, additionally driven by conflicts of interests, may have shaped the expression and development patterns underlying infant behaviors. The complex genetics arising from family interactions can be formalized by extending standard quantitative genetic models. These models demonstrate how the explicit consideration of the family environment can profoundly alter both the expression and evolutionary response to selection of behaviors involved in family interactions. Behavioral genetic studies have begun to unravel the complex genetics underlying infant solicitation behaviors and parental provisioning, although many focus on one side of the interaction. A genetic analysis incorporating interactions among family members explicitly may be critical because the genes underlying the expression of parental provisioning indirectly affect offspring behaviors, and *vice versa*.

KEY WORDS: Family interactions; infant behavior; ontogeny; parental care; parent–offspring conflict; quantitative genetics; sibling rivalry.

INTRODUCTION

Most multicellular organisms go through an ontogenetic period of development to the adult phenotype. Ontogeny is of particular interest not only from a developmental but also from an evolutionary perspective (West-Eberhard, 2003). Whether an individual reaches adulthood and reproductive age, and thus whether it has a chance to contribute gene copies to the next generation, depends critically on the success of the individual during ontogeny. Also, this period is often characterized by substantial mortality, and the final set of adult traits often arises through complex and dynamic interactions between the environment in which an animal develops and its own genes that regulate trait development (Hahn *et al.*, 1990; Mousseau and Fox, 1998; Roubertoux *et al.*, 1990; West-Eberhard, 2003). Variation in adult traits arising during ontogeny, therefore, results in variation in Darwinian Fitness, and thus natural selection on developmental patterns (West-Eberhard, 2003).

The susceptibility of ontogeny to external sources of variation suggests that the genetic architecture underlying trait development may be under natural selection for buffering trait expression against unfavorable environmental influences experienced early in life. Organisms often experience a wide range of environmental factors during ontogeny that should result in tremendous variation in trait expression. The observed variation is generally

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not that extreme, however. Considerable research has focused on internal mechanisms of developmental homeostasis (see West-Eberhard, 2003 and references therein). An alternative, less considered, way by which trait development may be canalized is through the evolution of a family group maintained beyond fertilization. By providing care, such as e.g., shelter, food, warmth, protection against parasites and predators, parents can at least partly counter environmental hazards to which their offspring would be fully exposed without their presence (Clutton-Brock, 1991; Mousseau and Fox, 1998; Roubertoux *et al*., 1990). A maintained family with parental care enhances offspring survival and stabilizes trait development (Clutton-Brock, 1991). Interactions among family members become a potentially important factor in trait development and evolution.

The aim of this paper, is to provide an overview over the potential implications of the family environment for the expression and evolution of traits involved in family interactions, in particular parental provisioning and offspring solicitation. To this end, I review in a first part theoretical concepts that have been developed for an understanding of the genetics and evolution of parent–offspring interactions. They include extensions to quantitative genetics theory (i.e., maternal effects and indirect genetic effects theory), and parent–offspring conflict theory. The second part is a more formal presentation of the quantitative genetic underpinning of a theory of family interactions. Finally, I review empirical research on the behavior genetics of family interactions, focusing on studies that have investigated the genetics of parental provisioning and offspring solicitation.

The Family as an Environment

One immediate consequence of the formation of family groups is that the environment in which infants develop partly consists of individuals of the same species that are genetically related. Interpreting the family as an environmental component to infant development (e.g., Roubertoux *et al*., 1990) implies that this environment contains genes, may be heritable, and can itself evolve (Cheverud, 2003; Cheverud and Moore 1994; Wolf, 2003; Wolf *et al*., 1998). Thus, the expression and development of infant traits is not only regulated by the infant's own genes and their interaction with the external environment (Fig. 1a), but also indirectly by the genes expressed in their caring parents (Fig. 1b).

Because variation in parental behavior may exert selection on offspring morphological and behavioral traits, just as an external source of environmental variation (Cheverud and Moore, 1994), offspring and parental traits are expected to become

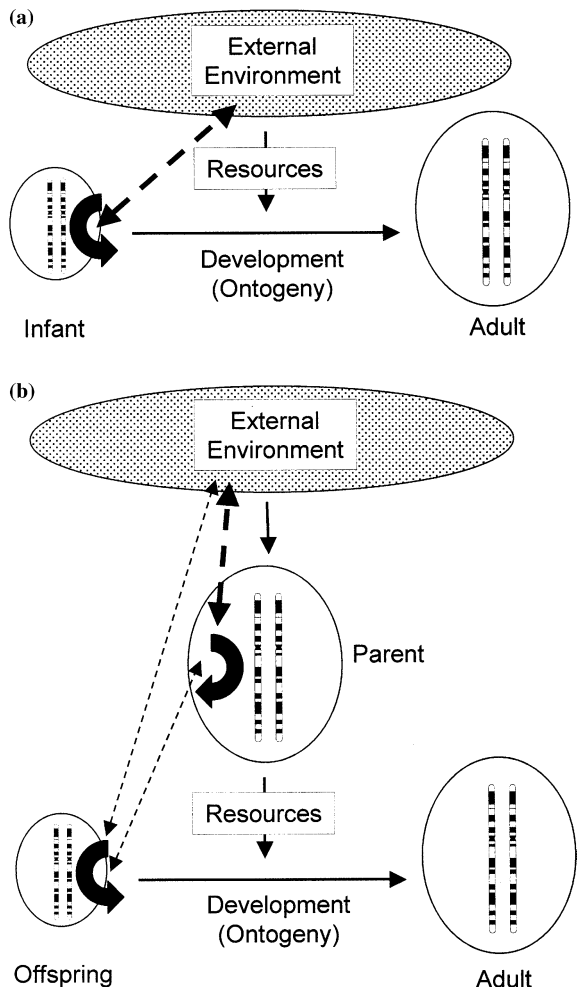

Fig. 1. Developmental scheme for systems without maintained family units and parental care (a), and for systems where infants develop within a family unit (b). The black arrows symbolize gene expression. Two chromosomes are drawn for both infants and adults/parents to represent their genomes. In (a) the infant's development will basically be guided by it's own genes' interactions with the external environment from which the resources required for development are gained. In (b), the parent provides these resources to the offspring, functioning as an environmental buffer or filter, and henceforth reduces the amount of direct interaction its offspring experiences with the external environment. Thus, the infant's development now depends to a large extent on its own genes' interaction with the environment provided by the parents. And this environment is at least partly based on the parents' genes and their interaction with the external environment.

(co-) adapted to family life. For example, morphological traits or behaviors that allow an infant to communicate effectively its "needs" or "quality" to the parents might evolve to allow parents dynamic fine-tuning of their care (God fray, 1991; Kölliker and Richner, 2001; Trivers, 1974). Explicitly taking into account that parents may vary genetically in their provisioning behaviors, a similar level of care favored by selection may be expressed by low-provisioning parents having demanding offspring or high-provisioning parents having non-demanding offspring (Wolf and Brodie, 1998). Co-adaptation to family life can therefore result in genetic correlations among offspring solicitation and parental provisioning behaviors through the generation of linkage disequilibrium (i.e., the non-random association of loci within genomes; Wolf and Brodie, 1998), or integration into at least partly overlapping genetic, physiological, and neurological pathways (West-Eberhard, 2003).

Parent–Offspring Conflict and Sibling Rivalry

A second consequence of the formation of family units on trait development and evolution is ultimately due to the fact that the evolution of parental care comes at a fitness cost to parents (Clutton-Brock, 1991). By remaining with their current offspring, expending time and energy for resource provisioning and protection, parents forego the opportunity to produce additional offspring (Clutton-Brock, 1991). The (co-) adaptation of traits to family life is therefore characterized by a trade-off between care provided to a given offspring and the parent's potential to produce additional offspring, or, in other words, by a trade-off between the number and quality of offspring (Stearns, 1992).

An additional twist arises when both the parent's and offspring's evolutionary genetic point of view in the interaction is considered (Trivers, 1974). A gene expressed in a parent (affecting its amount of parental provisioning) passes to future generations through any of its offspring by an equal chance of 50%. Conversely, a gene expressed in an offspring (affecting its parent's amount of care e.g., by expressing a solicitation behavior) has a 100% chance of being passed to the next generations if the bearer survives, *versus* a 50% chance, if a full-sibling survives. Thus, offspring (but not their parents) are expected to weigh their own survival higher than the survival of siblings. Evolutionary theory predicts the evolution of infant traits (e.g., solicitation behaviors) that allow offspring to outcompete siblings for parental resources (Godfray, 1995; Mock and Parker, 1997; Trivers, 1974), biasing the parents' quality–quantity trade-off somewhat towards the quality end (Stearns, 1992). Parents in turn may become reluctant to their offspring's demand, because the rivalry among their offspring may be against their own best evolutionary interest (Godfray, 1995; Mock and Parker, 1997).

A very general prediction derived from the theory of parent–offspring conflict (Trivers, 1974) is that infant behaviors/traits involved in the regulation of parental care (including their genetic, physiological, and neurological bases) are expected to be exaggerated beyond the minimal level that would be required for the successful communication with parents (Godfray, 1995; Maynard Smith and Harper, 1995; Mock and Parker, 1997; Trivers, 1974). Thus, the (co-) adaptation of offspring traits and parental care to family life may involve a selection component akin to a tug-of-war among genes expressed in parents and offspring, favoring exaggerated solicitation behaviors in offspring and parental countermeasures to sibling rivalry (Godfray, 1995; Mock and Parker, 1997).

In mating systems where females mate with multiple males, and females provide most care and resources to the offspring (e.g., many mammal species), conflict theory predicts that selection favors mechanisms regulating the expression of alleles depending on whether the allele was inherited through the mother or the father (i.e., genomic imprinting; see e.g., Keverne, 2001; Wilkins and Haig, 2003). For loci involved in infant growth and solicitation, the maternally inherited alleles have a greater evolutionary "interest" in the survival and future reproduction of the mother than do the paternally inherited alleles. While the former can be passed on to future generations through reproduction of the bearer female only, the latter can be passed on to future generations through reproduction with other females (Wilkins and Haig, 2003). Because the paternal allele spreads faster evolutionarily than the maternal allele if it takes more resource from the mother, the paternal allele only is expected to be expressed at such loci (Wilkins and Haig, 2003). The molecular mechanism behind this parent-of-origin specific silencing of alleles seems to largely depend on DNA-methylation (Gibbs, 2003).

Infant Traits, Parental Care, and Parental Effects

Infant traits may be categorized into: (1) traits that are specific adaptations to the external environment experienced during ontogeny (e.g., larval stages of amphibians (tadpoles)) or holometabolic insect (e.g., grubs and caterpillars), (2) traits that constitute intermediate stages towards an adult phenotype (e.g., juvenile body size), (3) evolutionarily neutral or possibly non-adaptive by-products of developmental or behavioral processes, and (4) traits that are specific adaptations to the environment provided by family members. All four trait categories may be affected in their development by the family environment. Infant traits from categories (1)–(3) potentially are passively modulated by parental provisioning. Traits from category (4) have been under selection arising from family interactions to allow offspring to play an active role in the regulation of parental provisioning.

Here I will be mostly concerned with this last category and refer to these traits as solicitation behaviors/traits. This definition also complies with the functional definition of a signal, i.e., an "action or structure which increases the fitness of an individual by altering the behavior of other organisms" (Maynard Smith and Harper, 1995, see also Dawkins and Krebs, 1978).

Infant traits from category (4) may have their evolutionary origin in any of the other categories, provided that this original trait elicited a differential response in parents in terms the provisioning of care.

For example, the begging display of altricial birds typically consists of vigorous gaping, backed-up by body stretching, wing flapping, and calling (Wright and Leonard, 2002). The structure of the gape is enhanced (as compared to the adult beak) by extended flanges (Kilner and Davies, 1998). A bird chick necessarily has to open its mouth to ingest food provisioned by the parent just as an adult needs to during food intake. Opening the beak when hungry and/or expecting food intake might therefore, be a quite general physiological–behavioral adaptation to the availability of a food source and the expectation of food intake (i.e., a trait of category 2), and thus might be considered a minimal behavioral level required for successful food transfer between parent and offspring (referred to as "ancestral threshold" by Payne and Rodríguez Gironés, 1998). The benefit to parents of discerning gaping from non-gaping chicks, and offering the food to the gaping ones, is also obvious. Parents may therefore be under strong selection, and thus predisposed, to develop a "receiver bias" (e.g., Payne and Rodríguez-Gironés, 1998) towards feeding gaping chicks. From there, a process of evolutionary (co-) adaptation of offspring solicitation and parental food provisioning preferences, additionally driven by sibling rivalry and parent–offspring conflict, might have lead to this very conspicuous solicitation display we observe today.

Quantitative genetics theory has introduced the terms "maternal effect" or "maternal inheritance" to account for effects of parental traits on offspring traits that are not inherited through Mendelian inheritance (Cheverud and Moore, 1994; Kirkpatrick and Lande, 1989; Mousseau and Fox, 1998; Roubertoux *et al*., 1990). Though the term "maternal effect" is sometimes specifically used in conjunction with pre-birth effects of the mother on her offsprings' phenotype, its definition is much broader. Maternal effects occur "when the phenotype of a mother [...], or the environment she experiences, causes phenotypic effects in her offspring" (Mousseau and Fox, 1998), which includes prezygotic maternal effects (e.g., nest-site choice, nest-building, and gametic size), postzygotic-prenatal maternal effects (e.g., nutritional/hormonal environment in the egg or womb), and postzygotic-postnatal maternal effects (all aspects of parental care after offspring birth, e.g., food and warmth provisioning, and protection) (Wade, 1998; see also Roubertoux *et al*., 1990). Parental care, which is "any form of parental behavior that appears likely to increase the fitness of a parent's offspring" (Clutton-Brock, 1991) is thus included under the definition of a maternal effect. In addition, paternal effects may also contribute to offspring phenotype if males provide resources for offspring development. I will in the following use the term "parental effect" to include both maternal and paternal effects in the discussion.

A convenient aspect when defining parental care as parental effects is that parental effects are easily formalized in quantitative genetic models as standardized regression/path coefficients (Dickerson, 1947; Willham, 1963; see next paragraph). Parental effects, therefore, can be quantified in a standardized way in empirical studies using appropriate experimental designs (Cheverud and Moore, 1994; Kirkpatrick and Lande, 1989; Lynch, 1987) and directly linked to theoretical quantitative genetic models. Parental effects can further be partitioned

into effects arising from either genetic variation among parents or variation in environmental factors experienced by the parents. The former have been termed indirect genetic effects (IGE) (Moore *et al*., 1997; Wolf *et al*., 1998) and are of particular interest for the genetics of family interactions and the coadaptation to family life.

A QUANTITATIVE FRAMEWORK FOR THE GENETICS UNDERLYING FAMILY INTERACTIONS

Under traditional quantitative genetics (in its simplest form) each trait can be defined as a sum of direct additive genetic effects and environmental deviations (Table I, point 1). The response to selection, i.e., the expected evolutionary change in the trait, depends on its heritability and the selection differential ($R = h^2S$ Falconer and Mackay, 1996). This formulation has limited applicability because it does not include effects of selection on genetically correlated traits (Lande and Arnold, 1983) or interacting phenotypes (Moore *et al*., 1997) on evolutionary change. Both these aspects are critical, however, in the study of the genetics underlying family interactions.

Parental effect models are an important step towards a solution by allowing for a one-directional effect of a parental trait on an offspring trait independent of the inherited genes (see Table I, point 2; Cheverud and Moore, 1994; Kirkpatrick and Lande, 1989; Lynch, 1987). Let z_s be the offspring solicitation behavior, and z_p be the parental provisioning behavior of interest. The parental effect of parental provisioning on offspring solicitation is here defined as the regression coefficient m_{sp} (Table I). The equation predicting the offspring solicitation behavior is now a function not only of its own genes and environment (a_s and e_s, respectively), but also of the parental provisioning genes and the environment experienced by the parents ($m_{sp}a_p$ and $m_{sp}e_p$, respectively; (Table I). There is thus an indirect genetic effect and an indirect environmental effect (Moore *et al*., 1997) of parental provisioning on offspring solicitation, as mediated by m_{sp}.

These models of trait composition provide critical insights for the evolution of the traits involved in family interactions. A response to selection in the offspring trait (here solicitation) can occur even in the absence of heritable variation in solicitation itself (Cheverud and Moore, 1994; Kirkpatrick and Lande, 1989). The selection on the expressed offspring solicitation phenotype (Table I; z_s^*) indirectly affects the distribution of additive genetic values for parental provisioning (Table I; $m_{sp}a'_p$), which alters the mean environment experienced by the offspring, which indirectly alters offspring solicitation. Also, the extent and even direction of evolutionary change in offspring solicitation now critically depends on the sign and size of m_{sp}, the genetic correlation between offspring solicitation and parental provisioning, and the genetic variance in parental provisioning (Cheverud and Moore, 1994; Kirkpatrick and Lande, 1989; Kirkpatrick and Lande, 1992). Finally, the parental effect induces momentum in the response to selection. Evolutionary change may continue even in the absence of current selection due to selection in previous generations

Table I. Mechanisms regulating the expression of an offspring trait z_s and a parental trait z_p

Mechanism offspring trait z_s	Mechanism parental trait z'_p	Offspring trait level expressed z_s^*	Parental trait level expressed $z_p^{*\prime}$
1) Traditional trait[1]			
$z_s = a_s + e_s$	$z'_p = a'_p + e'_p$	$z_s^* = a_s + e_s$	$z_p^{*\prime} = a'_p + e'_p$
2) Parental effect on offspring trait[2]			
$z_s = a_s + e_s + m_{sp}z'_p$	$z'_p = a'_p + e'_p$	$z_s^* = a_s + e_s + m_{sp}(a'_p + e'_p)$	$z_p^{*\prime} = a'_p + e'_p$
3) Reciprocal effects of offspring and parental traits[2,3]			
$z_s = a_s + e_s + m_{sp}z'_p$	$z'_p = a'_p + e'_p + o_{ps}z_s$	$z_s^* = (a_s + e_s + m_{sp}(a'_p + e'_p))/(1 - m_{sp}o_{ps})$	$z_p^{*\prime} = (a'_p + e'_s + o_{sp}(a_s + e_s))/(1 - m_{sp}o_{ps})$

[1]Falconer and Mackay (1996), [2] Kirkpatrick and Lande (1989), [3] Moore *et al*., (1997).
Including social mechanisms underlying the development of a phenotypic trait allows one to predict the expressed level of the trait through an equilibrium state reached in the social interaction. Simple direct additive genetic inheritance with an environmental deviation is assumed for simplicity. Dominance deviations, G^*E interactions and epistasis can be added to the equations. a_s and a_p; direct additive genetic values for infant and parental behavior; e_s and e_p; direct environmental deviation for the infant's and parent's behaviors; m_{sp}: maternal effect coefficient mediating the effect of parental provisioning on offspring solicitation; o_{ps}: offspring effect coefficient mediating the effect of solicitation on parental provisioning. Primes are used to assign the effects to the different generations involved. No prime = offspring, single prime = their parents. Asterisks denote trait levels at equilibrium.

(Kirkpatrick and Lande, 1989; McAdam and Boutin, 2003).

The parental effect models described above treat the expression of an infant trait as a passive outcome of a parental effect and thus applies to infant trait categories (1)–(3) as defined earlier. Part of the definition of a solicitation behavior is that, by soliciting care, offspring play an active role in the regulation of parental provisioning. Thus, by actively interacting with parents, offspring become an environmental component (containing solicitation genes) for parents affecting their provisioning. To include this effect in the model we need to define the mechanism underlying the expression of parental provisioning as a function of offspring solicitation (Table I, point 3). The offspring effect coefficient o_{ps} mediates this relationship in the definition. We have now incorporated a reciprocal feedback loop, the solution of which defines the levels of offspring solicitation (z_s^*) and parental provisioning (z_s^*) as an equilibrium state of the interaction (Table I, point 3; Kölliker, 2003; Moore *et al.*, 1997). The indirect genetic (and environmental) effects now play a role in the expression of both behaviors. The consequences for evolutionary change discussed above for the one-directional maternal effect model now apply to both behaviors, not only to offspring solicitation. An addition is that the feedback loop results in an interaction term in the denominators ($1 - m_{sp}o_{ps}$). The impact of this denominator on the predicted response to selection depends on the signs of m_{sp} and o_{ps}. If they are of the same sign (both positive or both negative), the denominator is smaller than 1 and the total genetic variances in the traits exposed to selection, and thus the response to selection itself, becomes amplified (Moore *et al.*, 1997). Conversely, if they are of opposite sign, the denominator is larger than 1, and the total genetic variance expressed becomes less than expected under traditional quantitative genetics (Moore *et al.*, 1997).

The socially responsive components of the behaviors m_{sp} and o_{ps} are in these models taken to be fixed population parameters (i.e., regression/path coefficients across families). In reality, these parameters may need to be treated as traits/behaviors (i.e., as regression/path coefficients within individuals) with underlying genetic variation (Cheverud and Moore, 1994). m_{sp} determines the effect of parental provisioning on offspring solicitation, and may therefore be considered a trait expressed in the offspring. It could, for example, relate to hunger physiology (Friedman and Halaas, 1998; Spiegelman and Flier, 1996) which in turn may affect solicitation, or to the degree of thermoregulatory independence from parental warming. Conversely, o_{ps} is the responsiveness of a parent to variation in offspring solicitation, i.e., a trait expressed in the parent. Assume genetic trait definitions with additive and dominance genetic effects, i.e., $z_s = a_s + d_s + m_{sp}z_p$ and $z_p = a_p + d_p + o_{ps}z_s$. Substituting a genetic trait definition of the form $z = a + d$ for m_{sp} and o_{ps}, respectively, and expanding the equation reveals a large number of epistatic (gene-by-gene) interactions (Wolf *et al.*, 2000; see Fig. 2). Without going into further details, it is straightforward to imagine the additional complexity arising when an environmental deviation is added to the trait definitions. Gene-by-environment (Falconer and Mackay, 1996; Lynch and Walsh, 1998) and environment-by-environment interactions would add to the equations shown in Fig. 2. These complex interaction effects are solely due to the social interaction among parents and offspring and constitute the (partially genetic) family environment for trait development and expression (Fig. 1b; Brodie, 2002).

A family often contains siblings whose behaviors may influence the solicitation behavior of an infant or the provisioning behavior of parents, complicating the picture of the genetics underlying infant trait development and expression further (Lynch, 1987). Also, parental provisioning may

(a)
$$z_s^* = \frac{a_s + d_s + a_{msp}a_p' + a_{msp}d_p' + d_{msp}a_p' + d_{msp}d_p'}{1 - (a_{msp}a_{ops}' + a_{msp}d_{ops}' + d_{msp}a_{ops}' + d_{msp}d_{ops}')}$$

(b)
$$z_p^* = \frac{a_p' + d_p' + a_s a_{ops}' + a_s d_{ops}' + d_s a_{ops}' + d_s d_{ops}'}{1 - (a_{msp}a_{ops}' + a_{msp}d_{ops}' + d_{msp}a_{ops}' + d_{msp}d_{ops}')}$$

Fig. 2. Equations for the expression of offspring solicitation z_s^* (a) and parental provisioning z_p^* (b) with reciprocal behavioral feedback (Table I, point 3). For simplicity, external environmental factors are ignored. The maternal effect coefficient m_{sp} is replaced by the genetic trait definition $m_{sp} = a_{m_{sp}} + d_{m_{sp}}$, and the solicitation effect coefficient o_{ps} is replaced by the trait definition $o_{ps} = a_{o_{ps}} + d_{o_{ps}}$. Both additive ($a$) and dominant ($d$) genetic effects are included. Highlighted in gray is the part of the equation that corresponds to the traditional quantitative genetic trait definition. The rest of the equation constitutes the genetic aspect due to the family environment. The product of two genetic effects indicates epistasis. Note that the purpose of this model is to qualitatively illustrate the importance of the family environment in the genetics underlying infant and parental behavior. It should not be taken as a quantitatively accurate genetic equation.

have a parental effect on itself in the subsequent generation (Cheverud and Moore, 1994), as has recently been reported in rats (Francis *et al*., 1999). The above model can be extended to incorporate such an effect (M. Kölliker, unpublished results).

Sibling interactions have been built into statistical models for the analysis of twin and family data in human behavior genetics (Neale and Cardon, 1992). The equations for trait definitions involving parent–offspring interactions presented here (Table I) can be formulated in matrix form, and with estimable parameters (i.e., path coefficients) to fit such models to empirical data on parental provisioning and offspring solicitation. Alternative statistical models that are defined by various levels of assumed complexities of family interactions (e.g., simple direct genetic effects, one-way parental effect, reciprocal parent–offspring effects, sibling effects, etc.) may then be tested for their relative goodness of fit to such data using model selection criteria (Neale and Cardon, 1992). This might be a promising statistical approach to elucidate the complex genetics arising from family interactions, and to estimate the parameters of theoretical evolutionary models directly.

THE GENETICS OF FAMILY INTERACTIONS: REVIEW

The theoretical framework presented above highlights the importance of a multidimensional analysis of the genetics underlying behaviors (and other traits) involved in the regulation of family interactions. When studying the development of infant solicitation behaviors, the study of the associated aspect of parental care is necessarily a partial determinant of the behavior under study and ideally should be investigated in parallel. For a review of the genetics underlying infant solicitation behaviors it is therefore natural to review simultaneously studies investigating the genetics underlying parental care.

Genetics of Solicitation Behaviors

The evidence for genetic influences on variation in infant solicitation behaviors was recently reviewed by Kölliker and Richner (2001). Such evidence came from studies on bird chick begging calls, rodent pup ultrasonic vocalizations, lamb bleating and human baby crying. New evidence for genetic variation in offspring solicitation has been reported from recent cross-fostering studies in organisms as diverse as burrower bugs (*Sehirus cinctus* Agrawal *et al*., 2001), burying beetles (*Nicrophorus vespilloides* Lock *et al*., 2004), mice (*Mus domesticus* Hager and Johnstone, 2003) and rhesus macaques (*Macacca mulatta* Maestripieri, 2004).

Some studies have measured offspring solicitation indirectly as an effect on (foster-) parents. This method provides a composite measure subsuming all solicitation behaviors into a single net "solicitation performance" trait, which partly also contains variation in the responsiveness of the (foster-) parents. This is analogous to an approach often used in quantitative genetics when parental care is measured as "parental performance" (e.g., Cheverud, 1984), an effect quantified e.g., as offspring growth or survival and attributed to the parent. For a detailed understanding of family interactions and behavioral development knowledge of the relevant behaviors and traits involved becomes important.

Rodent Infant Ultrasonic Vocalizations

Ultrasonic vocalizations emitted by rodent pups complies with the definition of a solicitation behavior used here (see above). Calling enhances parental proximity to the pup, facilitates maternal retrieval of pups and thus plays a critical role in the regulation of maternal behavior and the well-being and survival of pups (e.g., D' Amato *et al*., 2005; Hahn and Lavooy, 2005; Thornton *et al*., 2005). Many studies demonstrate an effect of body temperature and/or maternal proximity on a number of call characteristics. Pups call more intensely when the mother is absent and/or body temperature decreases, suggesting a strong socially responsive component to pup ultrasonic calling (see other papers in this issue).

The dependency of ultrasonic calling on hunger seems to be much less studied, and the evidence available is equivocal. There is some indirect evidence for a role of hunger through an effect of appetitive learning on pup ultrasonic calling in albino rats (Amsel *et al*., 1977). The only direct test I know of was carried out on gerbils (*Meriones unguiculatus* and reported a non-significant effect (McCauley and Elwood, 1984). Additional indirect evidence for such a role of USV is suggested by studies showing increased prolactin release in lactating rat dams exposed to pup USV (Hashimoto *et al*., 2001; Terkel *et al*., 1979; see also Barron and Gilbertson, 2005). Because maternal milk is critical

for pup survival and development, and is one of the major and probably most costly resources provided by mammalian mothers (Clutton-Brock, 1991), a pup solicitation trait signaling nutritional requirements may be expected. USV may be a prime candidate for such a behavior. Pup odors could play a role, too (e.g., Amsel *et al*., 1977). More studies evaluating a role of pup USV in the regulation of food provisioning (i.e., lactation) are needed.

Ultrasonic calling of rodent pups is the best studied solicitation behavior from a genetic perspective. Both cross-breeding and artificial selection experiments have repeatedly confirmed the presence of underlying additive genetic variance (Brunelli *et al*., 1997; Brunelli, 2005; Burgdorf *et al*., 2005; Hahn *et al*., 1987; Hofer *et al*., 2001; Roubertoux *et al*., 1996; Thornton *et al*., 2005), as well as dominance, epistatic, and pre-birth maternal effects (Roubertoux *et al*., 1996; Thornton *et al*., this issue). Additive genetic variance is critical for a response to selection and coadaptation to take place. Also, in the context of the genetics underlying family interactions the epistatic interactions are of particular interest (see below for more details; (Fig. 2)).

In these genetic analyses ultrasonic calling was often analyzed based on measurements from a single recording session after a fixed amount of pup isolation/cooling (see Hahn and Lavooy, 2005). An approach where USV for each pup is measured after various amounts of time in isolation (at least two; see Kölliker, 2003) and analyzed simultaneously would allow the partitioning of the total genetic variance observed into a component due to a fixed baseline calling activity (a_s; Table I, Fig. 2) and a component that socially responds to the amount of separation from the mother ($a_{m_{sp}}$; Fig. 2).

The partitioning into a fixed baseline and a socially responsive component may also be of interest in experiments investigating the effects of drugs on USV as a model for infant anxiety syndromes (see Barron and Gilbertson, 2005; Brunelli, 2005; Burgdorf *et al*., 2005; D' Amato *et al*., 2005). The preferential target of such drugs might be the baseline component. Inhibiting drug effects on the socially responsive components might have undesirable negative effects on the dynamics of family interactions (Barron and Gilbertson, 2005).

A next critical step in the study of the genetics underlying USV may be to track the observed genetic variation down to individual loci, for example using quantitative trait loci (QTL) mapping (e.g., Lynch and Walsh, 1998). An *a priori* list of candidate genes may also be established from the physiological context of USV-production. Genes involved in thermoregulation and the development of endothermy are certainly among the candidates for genes affecting USV production. If milk-intake and hunger turns out to affect pup USV, a rich literature on genes involved in the physiology of hunger and food intake would be available (Friedman and Halaas, 1998; Spiegelman and Flier, 1996).

A recent study demonstrated a role for the μ-opioid receptor gene in the expression of pup USV-intensity (Moles *et al*., 2004).

Genetics of Parental Provisioning

Studies investigating the genetics underlying parental provisioning have been reviewed by Bridges (1998), Kölliker and Richner (2001) and Peripato and Cheverud (2002). Additional evidence has been reported in Savannah Sparrows (*Passerculus snadwichensis*; Freeman-Gallant and Rothstein, 1999), long-tailed tits (*Aegithalos caudatus* MacColl and Hatchwell, 2003), burying beetles (*Nicrophorus pustulatus* and *Nicrophorus vespilloides* Lock *et al*., 2004; Rauter and Moore, 2002), dung beetles (*Onthophagus taurus* Hunt and Simmons, 2002) and mice (*Mus domesticus* Hager and Johnstone, 2003; Peripato *et al*., 2002).

Most studies have used traditional quantitative genetics designs such as parent–offspring regression, cross-fostering and cross-breeding (Boake *et al*., 2002; Falconer and Mackay, 1996; Lynch and Walsh, 1998) to reveal genetic variation underlying parental behavior. There is also a number of studies that have identified individual loci involved in the expression of maternal care behavior in mice through targeted knockout mutations (reviewed in Bridges, 1998; Kölliker and Richner, 2001; Peripato and Cheverud, 2002). Two recent studies in mice have adopted a new route based on cross-breeding among inbred mouse strains and QTL mapping (Peripato and Cheverud, 2002; Peripato *et al*., 2002; Wolf *et al*., 2002). They were able to identify QTL loci involved in maternal performance for pup growth (Wolf *et al*., 2002) and survival (Peripato *et al*., 2002), accounting for roughly 32% and 35% in variation in maternal performance, respectively.

An interesting result from the QTL study on maternal performance for pup survival (Perripato

et al., 2002) is the particularly large number of epistatic interactions underlying this trait. This is exactly what is predicted by the models presented above (Fig. 2). Epistatic interactions among genes expressed in offspring and their parents make up the genetic part of the behavioral interaction between an infant and the family environment (Brodie, 2002). Though Peripato *et al*. (2002) did not measure pup behaviors or traits involved in the regulation of maternal provisioning (e.g., USV, odors, and suckling), I would predict from the theoretical models (Fig. 2) that at least part of these epistatic interactions reflect interactions among maternal care genes and genes involved in the expression of pup solicitation behaviors. The QTL loci involved in these interactions may thus be taken as one possible starting point for looking into genetic regions affecting pup ultrasonic calling and other solicitation behaviors.

The variation in maternal performance investigated in the study by Peripato *et al*. (2002) related to ''abnormal'' variation, i.e., the complete failure of mothers to nurture pups resulting in the pup's deaths. Similar QTL studies on more subtle, natural variation, in specific maternal provisioning behaviors, focusing simultaneously on pup solicitation behaviors, will be of great interest to help unravel the complex genetics defining the family environment.

Genetic Correlations Among Solicitation and Parental Provisioning

As mentioned earlier, coadaptation between solicitation and provisioning can result in genetic correlations among the traits, and genetic correlations can critically affect the response to selection. A considerable number of studies, mostly on domesticated animals, have looked at the genetic correlation between direct genetic effects on juvenile growth and the maternal performance for this trait (this correlation is often referred to as the direct–indirect genetic correlation; e.g., Rauter and Moore, 2002), mostly reporting a negative genetic correlation (Cheverud and Moore, 1994; Lynch, 1987). Some studies have looked at the genetic relation between parental food provisioning and offspring growth (e.g., Hunt and Simmons, 2002; Rauter and Moore, 2002). I know of only four studies that have tested directly for a genetic association between parental provisioning and offspring solicitation. A positive genetic correlation was reported in a passerine bird species [i.e. great tit (*Parus major*)] between the maternal provisioning response and nestling begging call intensity (Kölliker *et al*., 2000), and in a burying beetle (*Nicrophorus vespilloides*) between maternal food provisioning and the time spent begging by larvae (Lock *et al*., 2004). On the other hand, a negative genetic correlation between maternal food provisioning and offspring elicitation performance was observed in burrower bugs (Agrawal *et al*., 2001). In rhesus macaques, a cross-fostering experiment showed that highly active (in terms of contact making and breaking) infants tend to have more rejecting biological mothers than less active infants, also suggesting a negative genetic correlation (Maestripieri, 2004). It is not yet clear why and under what circumstances coadaptation to family life may lead to either negative or positive genetic correlations (Lock *et al*., 2004).

The study in great tits involved, besides the cross-fostering, both begging playback and chick hunger experiments (Kölliker *et al*., 2000). This design allowed to roughly separate the socially responsive components from the fixed components. It was not standardized enough for quantitatively accurate estimates of the behavioral components, though (see Kölliker (2003) for a discussion of experimental difficulties involved). The results suggested a genetic correlation among the socially responsive component in mothers (i.e., $a_{o_{ps}}$, see Fig. 2) and the fixed component for begging in chicks (a_s; Kölliker *et al*., 2000).

A consequence of genetic correlations is that the evolutionary response to selection is altered (Lande and Arnold, 1983), including the possibility, in the case of a positive genetic correlation, for a runaway process driving both offspring solicitation and parental provisioning beyond the levels favored by selection alone (Kölliker *et al*., 2000; Wade, 1998; West-Eberhard, 1983, 2003).

Genomic Imprinting

The conflict hypothesis for explaining the occurrence of genomic imprinting received empirical support mostly from genes involved in physiological interactions among mammalian mothers and their fetuses through the placenta (Haig, 1993; Keverne, 2001; Wilkins and Haig, 2003). The most famous example concerns the growth enhancing insulin-like-growth-factor 2 (*Igf2*) gene and its growth-inhibiting type-2 receptor. While for *Igf2* the paternally

inherited allele only is expressed in the fetus, the maternally inherited allele only is expressed for the receptor gene (Haig and Graham, 1991). More paternally expressed genes, partly also involved in the postnatal physiological (up-) regulation of postpartum growth, have been discovered since (Itier *et al*., 1998; Keverne, 2001; Wilkins and Haig, 2003). A link to pup solicitation behavior has been proposed (i.e., suckling behavior; Keverne, 2001), and recently confirmed in mice. The targeted mutation of the gene *Peg3*, a gene where only the paternally inherited allele is expressed, resulted in reduced suckling efficiency of pups (Curley *et al*., 2004). Interestingly, this same gene simultaneously plays a critical role in the regulation of maternal behavior (see e.g., Bridges, 1998; Curley *et al*., 2004). *Peg3* thus is an example of integration of pup and parental behavior into a common genetic basis (i.e., pleiotropy; Falconer and Mackay, 1996). Additional evidence for imprinted genetic effects on pup suckling intensity comes from another recent study in mice involving both cross-breeding and cross-fostering experiments (Hager and Johnstone, 2003). Contrary to *a priori* expectation, the study showed a maternally expressed genetic effect on pup suckling efficiency. This same study also reported paternal inheritance of litter size (Hager and Johnstone, 2003).

CONCLUSIONS

The study of the genetics development and evolution of infant and parental behaviors is particularly challenging (Godfray, 1995; Roubertoux *et al*., 1990). One cannot achieve a detailed understanding of an infant behavior without studying its dependencies on and interactions with the behaviors of parents and siblings. This is true for the phenotypically plastic components of the behaviors, including e.g., learning (Kölliker, 2003), but also for the genetics underlying their expression and development in a family (Cheverud and Moore, 1994). Coadaptation of infant (solicitation) behaviors and parental provisioning is expected, with the levels of expressed behaviors additionally driven by conflicts of interests among family members.

Coadaptation of offspring solicitation and parental provisioning can result in genetic correlations among the behaviors. Few studies have investigated genetic correlations directly. Given its importance for an understanding of patterns of coadaptation to family life and the traits' responses to selection, additional research in this area will be valuable. Virtually nothing is known about the mechanistic causes for the observed genetic correlations. They could result from linkage disequilibrium among the genes involved in the expression of the two behaviors, or from genetic, physiological and/or neurological integration into similar expression pathways. It is noteworthy in this context to mention that the hypothalamus has been associated with the neural regulation of thermogenesis (Jessen, 2001), hunger physiology (Friedman and Halaas, 1998; Spiegelman and Flier, 1996) and maternal behavior (Keverne, 2001; Perripato and Cheverud, 2002) in rodents. Partially overlapping neural and physiological mechanisms for infant solicitation behaviors (e.g., USV) and maternal provisioning, therefore, seem at least possible.

Studies have started to unravel the complex genetics underlying infant solicitation behaviors and associated aspects of parental provisioning. The studies available so far come from a wide range of species encompassing mammals, birds and insects which underscores the widespread importance of genetic variation and correlation in behaviors involved in family interactions (see also Boake *et al*., 2002; Moore and Kukuk, 2002 for reviews of general behavioral genetic investigations).

I have argued that the study of the developmental behavioral genetics (Hahn *et al*., 1990) underlying infant behaviors is ideally complemented by the parallel study of the corresponding aspect of parental provisioning. In addition, a partitioning of the involved behaviors into a fixed baseline and a socially responsive component seems critical (Kölliker, 2003). Both the fixed and socially responsive component may have partly genetic bases, which can be investigated simultaneously. Depending on which component prevails, the behavioral dynamics of parent–offspring communication, the development of the involved behaviors and their evolution might be quite different.

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