Chapter 11: Genetic Cooperation and Conflicts in Mating and Social Systems

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The source of life history trade-offs in social animals

Behavioral ecology (Krebs 1987) has a rich tradition of calling trade-offs a cost-benefit. We can synonymize **cost-benefit** analysis with the notion of a life history **trade-off** introduced in chapter 3, because in some cases even **social system trade-offs** have been linked to the pleiotropic effects of one gene on two or more traits. As we will see below, in **greenbeard dynamics**, which arise from three greenbeard traits envisioned by Hamilton (1964) (the signal, signal recognition of self [or non-self], signal recognition elicits donation to self [or nasty to non-self]), the evolution of a beneficial self-cooperative strategy allows alternative strategies to invade and exact a social cost to cooperation that we call **altruism**. A consideration of social trade-offs allows us to come up with a synthetic view of the genetic sources of constraints on behavioral systems. In particular, other trade-offs, besides the primary **life history trade-offs** (Chapter 3) arise in the context of mating system dynamics and social system dynamics. Trade-offs in behavioral systems impact and interact with life history trade-offs. These many axes of constraint keep behavioral systems bounded. However, trade-offs also create the opportunity for novel social adaptations to arise, referred to as alternative behavioral strategies. Thus, the goal in this chapter is to not only elaborate on adaptation and constraint in life history and behavior, but also the sources of evolutionary novelty in social systems.

As we have seen in the section on life history trade-offs, the pathways in life history traits can arise through multiple trade-offs (egg size vs. egg number, reproduction early vs. reproduction late). Evolution can however, act on the intensity and even the geometry of these trade-offs (Phillips and Arnold 1989, Chevrud 1984), shaping the evolution of the genetic correlations that serve to keep traits coupled together (Sinervo and Svensson 2002). The force of correlational selection shapes genetic correlations. Selection can also impose new environmental conditions that favor the addition of new stages to a life cycle, resulting in the emergence of new transitions, metamorphoses, and by consequence new sets of trade-offs associated with new metamorphic events (Shaffer et al. 1989). Such complexity is epitomized by the trimetamorphic Eastern Newt with a larval form, a dispersive red eft form, and a form with a terrestrial foraging stage, and stream breeding aquatic stage (Pope 1928, Chadwick 1950, See Chapter 14). Some life history transitions might also disappear, as trade-offs are reorganized, such as in cave or murky

water forms with vestigial eyes that evolve to rely on other senses. For example, novel use and elaboration of the sixth sense of electromagnetic detection is common in such murky water forms, which are used in both prey detection and intraspecific communication (MacGregor and Westby 1992) (Chapter 8). Thus, a study of the sensory mechanisms and evolutionary forces shaping trade-offs might help us better understand how they are likely to evolve, become organized, then elaborated or conversely collapse. Throughout this book I have attempted to introduce as many examples of proximate mechanism as possible for it is only through understanding the details of physiology, endocrinology and neurophysiology, can we discover how behavioral systems are constrained, or alternatively free to evolve along **genetic lines of least resistance**, i.e., trade-offs (Schluter 1993) into new social systems.

Genetic methods for assessing genetic bases of trade-offs

As noted in chapters 2 and 3, the standard view is that phenotypic trade-offs arise from genetic trade-offs, which can only be due to pleiotropic effects of single genes on the expression of two or more traits. A genetic correlation between traits that both positively affect fitness but are negatively related to each other is also referred to as a negative genetic correlation. However, genetic correlations can arise from pleiotropy, linkage disequilibrium (LD) of closely linked genes, or from strong selection that generates LD among unlinked genes. It is often assumed that because LD will rapidly decay under the action of recombination and segregation, negative genetic correlations associated with LD cannot be responsible for life history trade-offs. In Chapter 8, we introduced the reason why LD will not decay in the context of social system dynamics: frequency dependent selection (FDS) can sustain correlational selection (CS). In cyclical dynamics, CS is a permanent feature of social systems.

Life history theories are expressed in terms of pleiotropy, genes that simultaneously affect 2 traits like **egg size and number**, which is called the **offspring quantity-quality trade-off** (Zera and Harshmann 2001) (Chapter 3). Other life history trade-offs include **costs of reproduction**, which is a trade-off between current vs. future reproductive effort (Reznick et al. 2000, Sinervo 1999). The cost of reproduction trade-off is also related to genetic theories on the **evolution of senescence** (Rose and Charleworth 1980, Charmantier et al. 2006). In this genetic theory, senescence is thought to arise from a selective premium placed on alleles for early reproduction that pleiotropically shorten life span.

Four methods can be used to verify the pleiotropic basis of trade-offs: 1) artificial selection experiments, 2) estimating negative genetic correlations in a pedigree coupled with physiological manipulations to verify the effect of endocrine regulatory systems on the expression of two or more traits, 3) deleting genes (*fosB* for maternal care, Chapter 2) or augmenting levels of gene expression (vasopressin receptor that is discussed below in the context of pair bond formation), and studying its ramifying effects on two or more traits, and 4) mapping the expression of 2 or more traits to single (or many) location(s) on the chromosome(s).

Artificial selection experiments

The action of pleiotropy can be resolved in artificial selection experiments as associated genetic changes in other traits that are themselves not the direct target of artificial selection (Lynch and Walsh 1998). For example, artificial selection on early reproduction in *Drosophila* can concomitantly reduce lifespan in only a few generations (Rose and Charlesworth 1981). While such approaches are informative in a laboratory context, care must be made in interpreting the results of such experiments because transient or chronic LD of unlinked genes in a natural context can also contribute to the strength of genetic correlations (Lynch and Walsh 1998) and arise from strong FDS (Sinervo and Calsbeek 2006). A situation in which this arises is in the context of sexual selection (Chapter 3, 10), which is predicted to build LD among even unlinked signaler and receiver traits (Houde 1994), each of which might be pleiotropically related to a life history trade-off (e.g., sexually selected survival trade-offs in males, perceptual biases in females that influence both foraging and mate choice). Rather than arising from pleiotropy, a negative genetic correlation can in principle arise from an underlying cause of FDS, which couples traits in the evolutionary long term because mating or social dynamics (e.g., cyclical dynamics like the RPS, see Side Box 9.2) generate a stable force that builds LD and thus genetic correlations (e.g., runaway sexual selection builds a genetic correlation between a male signaling trait with unlinked (or linked) genes for female preference).

Measuring genetic correlations

Another method to indirectly assess pleiotropy is to measure a genetic correlation between traits in the context of a pedigree (e.g., Svensson et al. 2001a). If two traits have a strong genetic correlation that promotes a
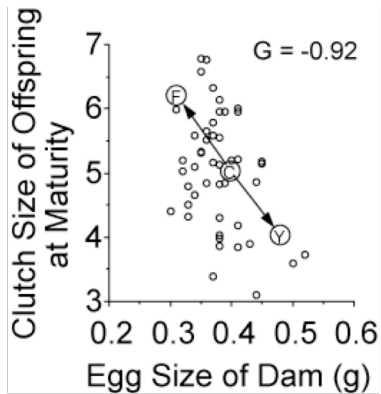
Figure 11.1. Negative genetic correlation of daughter's clutch size and dam's egg mass. The sign and magnitude of the genetic correlation, G = –0.92, is indicative of a strong negative genetic correlation (Sinervo et al. 2000a), which affects female fecundity and progeny survival, a classic offspring-size quality trade-off (see Chapter 3). Endocrine and surgical experiments (follicle stimulating hormone [F], follicle ablation [X], relative to controls [C]) elucidate the physiological basis of this negative genetic correlation.

negative relationship between fitness (e.g., cost in one trait enhances another trait, behavioral traits included), the action of pleiotropy is implicated. For example, a classic trade-off that arises in the context of reproduction is the offspring quality and quantity trade-off (Figure 11.1).

As noted above, negative genetic correlations can also arise from LD owing to CS that correlates trait combinations (Sinervo and Svensson 2003). Therefore, it is not sufficient to measure a negative genetic correlation to demonstrate a pleiotropic cause to a life history trade-off. Manipulations of hormones that govern expression of a trade-off are also necessary. In the case of the progeny quality trade-off, augmenting levels of follicle stimulating hormone cause a reduction of progeny size. Conversely ablating follicles on the ovary decreases clutch size and at the same time increases egg mass (Fig. 11.1). These endocrine manipulations get at the mechanistic basis of the hormones that control the expression of a trade-off. In this chapter, we will explore in more detail selfish genes associated with genomic imprinting that cause some progeny in a litter (or clutch) to take more investment at the expense of other progeny or female parent. In summary, while genetic correlations among parents and progeny are useful in elucidating a negative genetic correlation definitive proof requires manipulation of gene products like hormones. Of course one can also directly manipulate the genes.

Gene manipulation or gene mapping

A third and direct method to identify pleiotropy is by gene manipulation (deletion) in the lab (recall the example of the greenbeard in the social amoeba, Chapter 4). A fourth and related method is to use gene mapping to map multiple traits to single genes, or invalidate pleiotropy by

mapping traits to unlinked loci (recall the example of the genome-wide greenbeard in the side-blotched lizard, Side Box 4.3). Pleiotropy need not merely govern life history traits but can also couple behavioral traits. When genes express costs and benefits on social behaviors like cooperation (Foster et al. 2004), we identify the pleiotropic action of single genes on behavior. Conversely, when many unlinked genes are involved in the costs and benefits of cooperation (Sinervo et al. 2006a) we implicate **epistasis or gene interactions** in the social trade-offs. In this case, interactions of many genes (i.e. 3 traits of Hamiltonian greenbeards that constitute a supergene of cooperation) can be cemented by the action of correlational selection. Only by studying the **genomic architecture** of traits, by mapping genes that control behavior and life history and by in-depth analyses of selection, can we decompose the role of gene pleiotropy (single-gene effects on trade-offs) relative to gene epistasis (multiple-gene effects on trade-offs). Gene interactions among unlinked loci, which are cemented into permanent and stable negative genetic correlations, can be as permanent as pleiotropy in generating trade-offs. If mating or social system dynamics sustain correlational selection in the evolutionary long term, owing to FDS (such as cycles or a stable attractor), CS will build genetic correlations and sustain chronic life history trade-offs, even among unlinked genes.

The forms of epistasis: genetic, physiological and fitness

In the case of life history traits, many pleiotropies have seemingly been identified with genetic correlation analysis, such as the impact of egg size on clutch size (Fig. 11.1) and cascading effects on offspring survival (Chapter 3). However, this genetic perspective ignores the emergent properties of many interacting genes that underlie behavior that are difficult to resolve as single vs. multilocus effects without gene mapping or gene alteration technology to confirm or refute the action of pleiotropy (e.g., pathway for reproductive hormones in males and females, see Fig. 8.12, Chapter 8). Suites of interacting genes that have cascading effects on other genes are referred to as **gene epistasis** or **gene interactions** (Wright 1968). Epistasis can create trade-offs.

Genetic epistasis is closely related to **physiological epistasis** (Sinervo and Svensson 2003; Sinervo and Calsbeek 2003). Physiological epistasis can be defined as highly non-linear interactions (as opposed to purely additive interaction) between two or more behavioral traits, among behavioral and physiological traits, or among physiological traits.

Sewall Wright (1969) considered physiological epistasis to be universal in genetic systems (Wade 2002, Sinervo and Svensson 2003). However, he theorized that genetic variation in epistatic networks destabilized organismal function. He suggested that epistatic genetic variation is fixed in most species owing to such negative effects on fitness. However, this need not be the case in which alternative allelomorphs exist at key control loci in a physiological cascade, which creates alternative physiological pathways and cascading effects on organismal function such social behavior. It is precisely for this reason that alternative morphologies or morphs are of interest to life history theory. A study of alternative forms provides a window on the role of genetic and physiological epistasis in generating trade-offs in behavior.

In most monomorphic organisms there is little epistatic genetic variance since the physiological epistatic networks are fixed. However in species with alternative morphs, there is abundant genetic variation that arises from the interactive or nonlinear effects of genes on phenotype, as well as the typical linear or additive genetic variation that is of interest in life history studies. In addition, the expression of the same genes in the two sexes can generate either pleiotropy or epistasis (discussed below).

An analogous situation in nature, where there exists abundant epistatic genetic variation, occurs at species contact zones (Chapter 5). Species are thought to contain coadapted genes, which are fixed for most genetic epistasis. However, when species hybridize, genetic networks are mixed, generating gene epistasis. Hybrid unfitness arises from such genetic and physiological epistasis (Wade 2002). At species contact zones, hybrids and unfavorable epistases, which arise from mixing coadapted species genomes, are constantly being purged by selection. In the same way, morphs within a species contain coadapted morph complexes (e.g., garter snakes, Fig. 3.17, male lizards Side Box 4.3) that contribute to epistatic variance, when morph complexes are freely recombined.

Thus concepts of genetic and physiological epistasis are closely related to the third type of epistasis, **fitness epistasis** (Whitlock et al. 1995; Kelly 2000) in which non-linearity among traits and fitness is so extreme that they create many alternative optima on a fitness landscape. The fitness epistasis generated at a species contact zone is resolved as hybrid unfitness relative to pure parental species. Speciation and hybrid unfitness provides a useful model for visualizing the action of key control loci for alternative morphs within a species (Calsbeek and

Sinervo 2003). A given morph allele may require interactions with many other loci to create ideal combinations of alleles that yield high fitness. Complementary combinations are favored in alternative allelomorphs that reside in the same population. The constant mixing during sexual reproduction of underlying multilocus genotypes that control alternative morphologies generates fitness epistasis. CS constantly refines the genetic combinations that work best in the context of suites of multivariate traits, which comprise a given strategy (Sinervo and Svensson 2002). Crosses between optimal genotypes that reside at different fitness optima generate admixtures (e.g., fitness saddles) that do not work well and are thus are purged from the population. In species that are polymorphic, the advantage of each alternative morph within the species must be great enough (relative to a monomorphic solution that is fixed for these genetic epistases) to overcome what is referred to as a segregational or recombinational genetic load (Wallace 1975, Lynch and Walsh 1998). In essence, morphs experience a form of **outbreeding depression** (Chapter 13) due to mixing of coadapted morph complexes.

Ontogenetic conflict: Trade-offs in the design of two sexes

The easiest way to visualize the process of selection on alternative forms within a single sex is to consider the selective processes that are constantly refining the two sexes, male and female, into their respective alternative morphologies. Males and females reflect the fundamental morphs of sexual species. Recent advances in our understanding of life history trade-offs have identified different patterns of selection on the sexes as an important source of additive genetic (and epistatic) variation (Rice and Chippindale, 2001). The genetic trade-offs that promote functional trade-offs in organismal design between the sexes are referred to as **intersexual ontogenetic conflict** (Rice and Chippindale 2001). Many alleles that are favored under the force of sexual selection in the male morphology and physiology are of limited value during natural selection on the female morphology and physiology, and vice versa.

Rice and Chippindale (1998) used a hypothetical example of the human pelvic girdle, which is thought to form a strong allometric constraint on evolution of large brain size in primates (Luetenegger 1979), and is thus under strong selection for large size. However, male morphology is not subject to this functional trade-off. Alleles favored in males should reach a pelvic girdle width that is optimal for foraging behavior or perhaps mate competition (locomotion), under the optimizing force of selection.

Side Box 11.1. Mating system tradeoffs as elucidated by experiments: Ontogenetic conflict between the sexes

i) Natural variation: Large male side-blotched lizards get large rock piles (⬤), and many females

The Polygyny Threshold (Orians 1969) is a key concept governing the decision making of females. Should a female settle with a highly polygynous male and compete with other females, or settle monogamously on a lower quality territory. This concept is closely related to the **Ideal Free Distribution** (see Chapter 6).
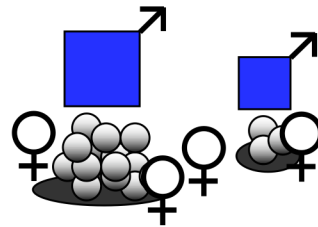
Female conflict and **density dependent competition** in *Uta (see Chapter 2)*: crowding creates density competition in female morphs (Sinervo *et al* 2000, 2001, Comendant *et al.* 2003, Svensson *et al.* 2002a, b, 2003).
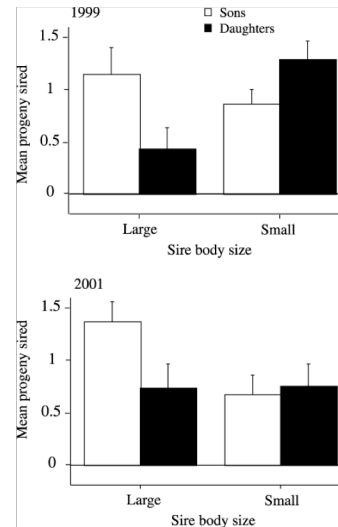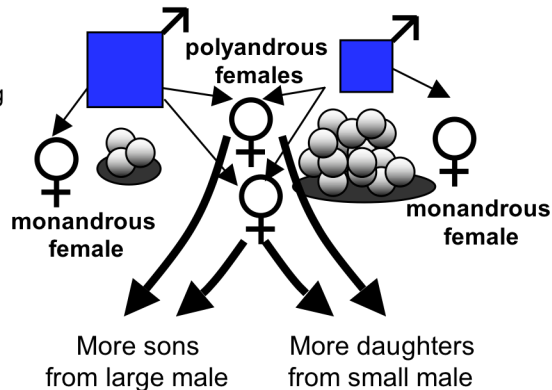
ii) Experiment: Reverse rockpile size (⬅➡) (Calsbeek & Sinervo 2002ab, 2004). Females move to high quality pile, but must mate with the small male.

Females that move produce much larger egg size (Calsbeek and Sinervo 2002), which confirms a **direct benefit** of large rock piles (thermal quality, Calsbeek & Sinervo 2007).

Females move

Move rocks
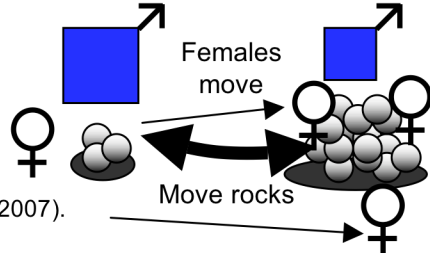
iii) Insemination (➡) and Fertilization (➡):

Some ontogenetic conflict is resolved by sperm sorting (Fig. 11.2), which is a form of cryptic female choice. The female obtains an **indirect genetic benefits** from mating with a small sire and a large sire, which produce high surviving daughters and sons respectivley (Fig. 11.3).

polyandrous females

monandrous female

monandrous female

More sons from large male

More daughters from small male

Figure 11.2. Sperm sorting in the female side-blotched lizard. The mean number of progeny (sons versus daughters) sired by large vs. small males within a female's clutch during 1999 and 2001. In both years, the production of sons and daughters depended on relative sire body size. Females were more likely to produce sons with the sperm from relatively larger sires in both years. During 1999, females were also significantly more likely to produce daughters with the sperm from relatively smaller sires. (from Calsbeek and Sinervo 2004).
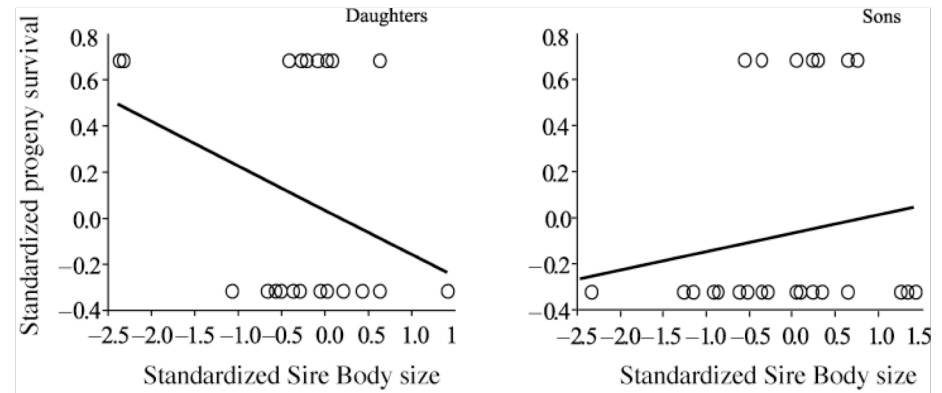
Figure 11.3. Progeny sex allocation had significant effects on survival. From 1999-2000 and 2001-2002 significant differences in viability selection (survival to maturity) favored daughters that were sired by relatively smaller males and sons that were sired by relatively larger males. Considering only progeny of **polyandrous females**, we recaptured four of 36 progeny during 2000 and 17 of 39 progeny during 2002 that had been released during the previous summer, and to whom we could accurately assign sires with paternity analyses. Sire body size and survival have been standardized to mean zero with unit variance.

Summary. Female side-blotched lizards obtain an **indirect genetic benefit** from mating with sires that vary in body size. This form of **cryptic mate choice** involving sperm sorting is likely to be common in animals with sexually dimorphic size at maturity. Males must carry genes for large size to survive well to maturity while daughters must carry genes for smaller size. The optimal body size is thus under **ontogenetic conflict** between the sexes.

Alleles should reach an optimum in each sex were it not for the fact that males and females share nearly all of their genes in a common genome (aside from sex chromosomes). Pelvic girdles in females are under the dual selective forces generated by locomotor and reproductive function, while males are under a single force of selection for locomotor function. This ontogenetic conflict can be resolved in most species via the sex-limiting action of steroid hormones that govern naturally selected traits of females or sexually selected traits in males (Sinervo and Calsbeek 2003). Gene promoters called **Estrogen Response Elements** (ERE) differentially control gene transcription and translation in the sexes (Freedman and Luisi 1993; Zajac and Chilco 1995; Sanchez et al. 2002).

The sex-specificity of endocrine systems is, however, not perfect. Not every gene for male and female traits, which are under ontogenetic conflict, has an ERE. Thus, there is always ongoing selection on female traits, and counter selection on male traits. Any secondary sexual trait is likely to be under ontogenetic conflict, given that most key regulatory genes in male and female reproduction are shared between the sexes (e.g., the gonadotropins, etc.). Even the classic sex hormones estrogen and testosterone are expressed in both sexes, albeit are reduced levels in each respective sex. For example, testosterone secretion, which is required for sex drive in females, might also trigger the action of gene expression for aggression, which is not optimal from the perspective of female life history (Sinervo and Calsbeek 2003). Similarly, genetic variation in testosterone expression in males, commonly associated with alternative male strategies in vertebrates (Brantley et al. 1993), might have a permeable expression in daughters of sexually selected males, thereby influencing female behavior, and also life history trade-offs.

Sex chromosomes, which initiate the cascade of sex determination via single genes (e.g., testis determining factor), are clearly unlinked from autosomal genes where many EREs reside. Thus, ontogenetic conflict, the fundamental life history trade-off between the sexes, must arise from the interactions among many genes or epistasis, not pleiotropy.

Empirical demonstrations of ontogenetic conflict are restricted to studies of *Drosophila melanogaster* in the lab (Pichedda and Chippindale 2006), or natural systems with pedigree on both sexes (see Side Box 4.1 for *Uta*, red deer: Foerster et al. 2006). Nevertheless ontogenetic conflict reflects a fundamental trade-off that affects all sexual organisms and it arises from the universal physiological epistasis associated with the production of the two sexes. Most life history analyses have been restricted to one sex, and thus, action of ontogenetic conflict is rarely studied, despite its primary importance to behavior. Studies on genetic correlations between traits expressed in both sexes, including behavioral and life history traits, will be required to elucidate this important trade-off (Sinervo and Calsbeek 2004, Calsbeek and Sinervo 2004).

Antagonistic sexual selection: mating system trade-offs

An important step in this direction is an analysis of the genetic bases of mating systems. Mating system trade-offs (Wiley 2000) can be partitioned into those acting on females, those acting among males, and those impacting both sexes. Given that females are more often the care-giving sex, males are under sexual selection to increase mating number, referred to as Bateman's (1948) principle. Mating systems with sex-role reversal (Gwynne 1981, Gwynne and Simmons 1990) provide a useful counterpoint to this generalization, which reflect exceptions that prove the rule (Chapter 10). The optimal number of mates for males is thought to be much higher than the optimal number for females, due to **Bateman's principle**. However, females in some mating systems are under selection to be promiscuous owing to factors like fertility assurance (Madsen et al. 1992). Other forces driving female promiscuity are related to more complex issues like ontogenetic conflict, discussed above. A female might mate with more than a single male, to produce both sons and daughters of high genetic quality (See Side Box 11.1). Nevertheless, males are typically under selection to mate with many more mates, especially given asymmetries in male and female care due to Bateman's principle.

Figure 11.4. Male versus female optima in reproductive traits. In the absence of monogamy, males and females can differ in their optima for many reproductive traits. The differences between the sexes can generate intense forms of antagonistic sexual selection. (Holland and Rice 1998)

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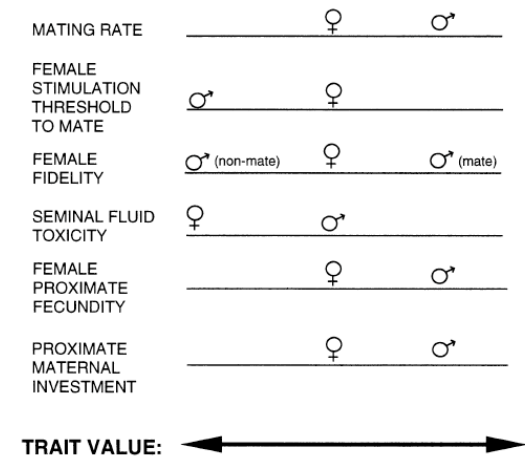
My goal is not to review all factors favoring polygyny or polyandry. These ideas are deeply rooted in traditions of behavioral ecology (Orians 1969; Emlen and Oring 1981) and mating system concepts are distributed across this book (e.g., Lek, Chapter 8). I wish to point out that mating system tradeoffs are not due to single gene effects, but rather epistasis, given that mating systems entail communication in signalers and receivers. Fitness epistases between a signaler's trait and receiver's trait in a mating system are inextricably linked to life history trade-offs.
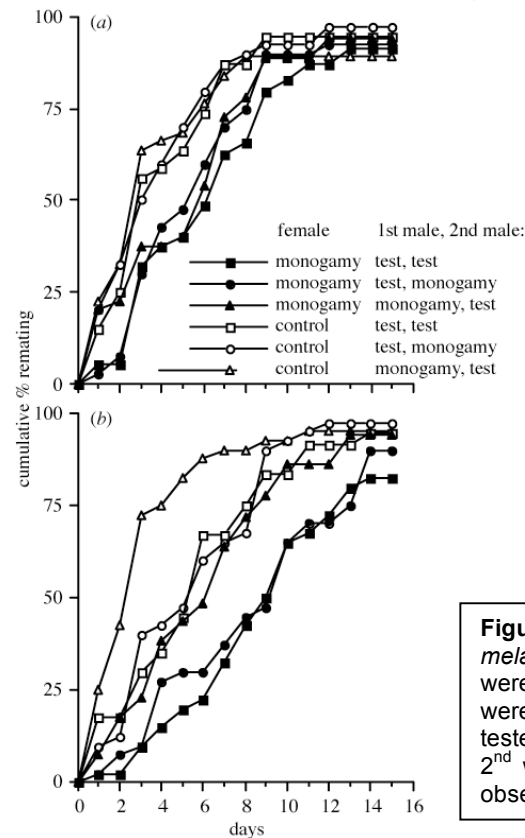
For example, trade-offs of polyandry (and polygyny) are clear in birds, which exhibit **biparental care** (Reynolds et al. 2002). In such mating systems, polyandrous females might risk detection by a mate that subsequently abandons a female in a situation of compromised paternity. Conversely, polygamous males, owing high plasma testosterone, might be less effective parents than a monogamous mate (Ketterson and Nolan 2004). Sexually selected strategies, via asymmetries in relative workload experienced by each parent, have cascading impacts on the **offspring quality trade-off** and the **cost of reproduction trade-off**, which affects parental survival as a function of workload.

The difference in mate number that is optimal in each sex (males generally with more mates) promotes **antagonistic sexual conflict**, another kind of mating system trade-off. For example, in *Drosophila*, seminal fluid proteins are hypothesized to reduce female remating rates and enhance paternity assurance (Rice 2000). This constitutes a form of **partner manipulation** that enhances his current success; even it has deleterious effects on costs of reproduction of females in subsequent reproductive episodes, which mate with different males. Physiologically naïve females, which have been selected under monogyny and thus not previously exposed to male strains that have evolved under polygyny, suffer higher rates of mortality when mated to polygamous strains compared with polyandrous females that have evolved under polygyny (Holland and Rice 1999).

Pitnick et al. (2000) followed up with similar experiments and demonstrated that female lines, which were selected under monogamy, evolved less resistance to seminal fluids (i.e., remated sooner: Fig. 11.5). Evolved female resistance to male proteins may be countered by increased efficiency of seminal fluid proteins. Similar antagonistic selection has been identified as a driving force in water striders (Rowe et al. 1994), but the selective factors acting on females relate to the burdening effect of a male that maintains amplexus for long periods of time, rather than physiological action of hormones that induce costs.

Allohormones. In the case of hormones transferred from one partner to another, Koene and Maat (2001) advocate the use of the term **allohormone** (allos – arousal by another), which refers to a substance that induces a direct behavioral response, bypassing sensory structures. Allohormones are distinct from pheromones (Chapter 13, Gk. *pherein* – to transfer), which are signaling compounds that are detected by sensory structures and then transferred to salient integrating centers of the central nervous system. Allohormones alter target tissues such as the reproductive system, just like hormones, and in many cases allohormones are derived from the same biosynthetic pathways as hormones produced by the targeted individual.

An allohormone is found on the love dart that is injected by the garden snail *Helix aspersa* (Koene and Chase 1998), which enhances male fertility (Landolfa et al. 2001). In the dusky salamander, *Desmognathus fuscus* (Arnold and Houck 1982), the male secretes a substance from the mental gland and then transfers it to the females back, only after having scraped the female's back raw with specialized maxillary teeth. The substance from the mental gland is directly transferred to the female's bloodstream and it appears to make the female more receptive to picking up the male's

Figure legend (within plot):

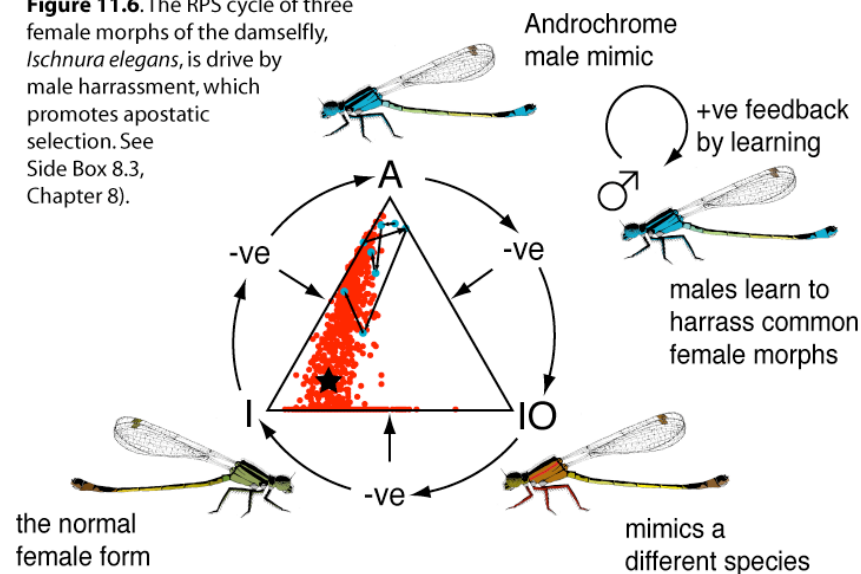| female	1st male, 2nd male:
monogamy	test, test
monogamy	test, monogamy
monogamy	monogamy, test
control	test, test
control	test, monogamy
control	monogamy, test

Figure 11.5. Cumulative percent remating of *Drosophila melanogaster* females over time as a function of two lines that were artificially selected for monogany relative to control lines that were maintained under polygyny. Females in each line were tested 1st with one male line and 2nd the other male line (or 1st and 2nd were the same). A significant difference in remate rate was observed in the replicate B but not A (from Pitnick et al. 2000).

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spermatophore packet. Whether or not such allohormones directly impacts future reproductive success of females and thus presents a cost of reproduction has not been directly demonstrated for any compound, or indeed any species, although indirect evidence suggests that seminal fluids of male *Drosophila melanogaster* have such effects.

In extreme cases, remating pressure by males favors the evolution of alternative female morphology, such as in damselflies, in which females are differentially cryptic to males. A novel androchrome female morph evolves to resemble the male form, becoming more cryptic to males. Other rare female forms avoid detection, via apostatic or rare advantage, relative to a common female form that is encountered by males at a high rate (see Chapter 8 and Fig. 11.6). Thus, trimorphisms comprising all 3 strategies are common in damselflies of Europe and North America (Svensson et al. 2005). Female trimorphism is thought to arise from search image formation in males for common female morphs (Fincke 2004). Given intense sexual conflict arising from male harassment, rare cryptic female morphs gain higher fecundity, thus invoking previously noted trade-offs of costs of reproduction, fecundity, polyandry, and sexually antagonistic selection owing to polygyny as well as trade-offs in perceptual systems, which are discussed in Chapter 13 and 14.

Figure 11.6. The RPS cycle of three female morphs of the damselfly, *Ischnura elegans*, is drive by male harrassment, which promotes apostatic selection. See Side Box 8.3, Chapter 8).

Androchrome male mimic

+ve feedback by learning

males learn to harrass common female morphs

the normal female form

mimics a different species

Sexually antagonistic selection may also explain a large number of sexually dimorphic traits, if dimorphism has evolved to limit the sex-specific costs of sexually antagonistic alleles (Rice 1984). Thus, mating system trade-offs are closely related to the trade-offs involving alternative optima in the sexes or ontogenetic conflict (noted above). This is because sexual conflict between the sexes operates on the same genes and perhaps even the same traits in the sexes (e.g., parental care in males vs. females). This generates a vicious cycle of adaptation in which traits that enhance male fitness, which conflict with female interests (e.g., lower levels of care, higher polygyny), result in counteradaptions in females (higher care, perhaps higher polyandry) that may exacerbate costs of reproduction in females, or via sexually antagonistic selection and mate harming. Thus, counteradaptations arising from sexually antagonistic selection, can generate as well integrate many primary trade-offs and secondary trade-offs mentioned above (life history trade-offs due to pleiotropy, exacerbated by trade-offs of physiology, performance, signaling and mating system, Chapter 8). Given the possibility of cyclical evolutionary change (e.g., evolutionary arms race involving **Red Queen** dynamics, Chapter 3) in life history and mating system adaptation, optimality approaches are unlikely to capture the dynamic that is theoretically possible. ESS approaches (described in Chapter 9) are essential. Moreover, FD cycles can generate LD.

A simple way to visualize why mating system trade-offs invariably arise from epistasis is to note that many existing theories of sexual selection suggest that runaway will couple female preference for exaggerated phenotypes (Lande 1991, Fisher 1930), or pre-existing biases make females prone to prefer a mutant signal trait (Ryan 1997). Male trait loci are likely to be unlinked from female preference loci. In situations where male display traits are also linked to territory quality and resource holding power (Side Box 11.1), female preferences for males and preference for high quality territories become instantly linked (and linked to physiological trade-offs in males). However, this has cascading effects on intrasexual female conflict owing to the high density of females expected on these male territories. Thus, Orians (1969) idea of **polygyny threshold**, which posits that females should equilibrate and move to lower quality territories based on ideal free principles, may not be possible if high quality son production requires residence on dense high quality territories for both **direct** and **indirect benefits** (Side Box 11.1). The genes will become correlated, but sexual selection drives the

correlation among signal traits, signal and territory quality preferences of females, and cascading effects on life history trade-offs.

A polygamous mating system, where one of the sexes has many mating partners is contrasted by a monogamous mating system, where each sex only has a single partner. Truly monogamous mating systems may in fact be very rare in nature, however, it represents an ideal form of mating system in which each partner is **mutualistically** interested in the other partners well-being and longevity. This contrasts directly with a mating system in which one partner, usually the male, obtains high fitness from obtaining additional mates. In such situations males evolve strategies that benefit only the male. Such strategies can have negative impacts on female lifespan and future reproductive success of females.

The role of ecology in governing monogamy and pair bonds

In polygynous mating systems extreme sexual dimorphism can evolve under the force of sexual selection. For example, the greatest degree of sexual size dimorphism in mammals is observed between male and female elephant seals that breed in rookeries in the western Pacific Ocean. An α male elephant seal can weigh up to 2275 kg, while females weigh 513 kg (Lindenfors et al. 2002). Most mammals are polygynous and size sexual dimorphism is typical, including humans in which males are 20% larger on average than females. Fewer than 3% of all mammals are monogamous (Klimann 1977). Therefore, when monogamous mating systems evolve in mammals it is of great interest to determine the mechanisms that keep males bonded to females.

Emlen and Oring (1981) suggested that the distribution of resources constrains defensible territory area (Chapter 6), which sets an upper limit on number of females controlled by males. Under conditions where males can only defend one mate, the adaptive mating system would be monogamous and a mate guarding strategy would be the only ESS. Males with alternative strategies or males that lacked a mate would cuckold a male that strayed too far from his single social mate. The mating system of seahorses, introduced in Chapter 10, presents a case of reverse sexual dimorphism in which female sea horses should not stray too far from their male sea horse consorts because the density of seahorses is typically extremely low on reefs.

Ecological constraints therefore impose a monogamous mating system on many organisms. A monogamous mating system is diagnosed by the
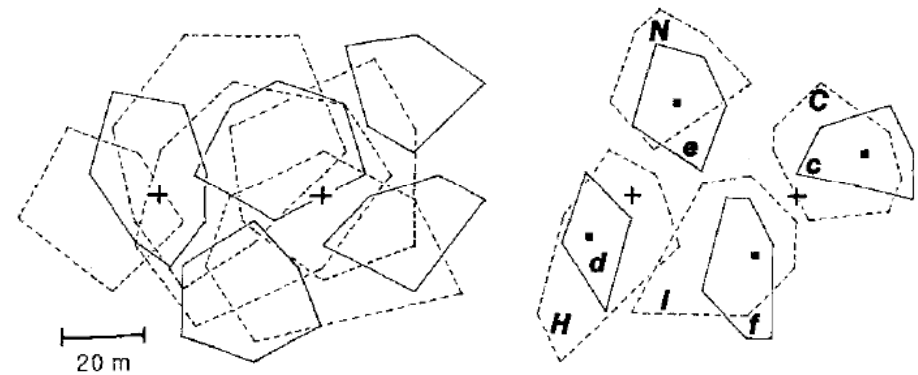
Figure 11.7. Home range overlap among males (dashed line) and females (solid lines) in a polygamous mouse, *Apodemus speciosus* (left), contrasts markedly from a socially monogamous species, *Apodemus argenteus* (right). (Oka 1992).

territorial spacing of the organism. In rodents, monogamous mating systems evolve in those species, which are found at low density. For example, two species of field mice, *Apodemus speciosus* and *A. argenteus*, prefer different habitat. *Apodemus speciosus* prefers more open habitat, while *A. argenteus* prefers a montane woodland habitat on the island of Honshu in Japan (Oka 1992). The home ranges of male *A. speciosus* are significantly larger and overlap with many more females than that of *A. argenteus*, suggesting that *A. speciosus* is polygynous and promiscuous. Conversely the home ranges of male *A. argenteus* overlap with only a single female (Figure 11.7). Oka (1992) studied these two *Apodemus* species in an area of sympatry, thereby ruling out the effects of confounding habitat on the differences between species. Thus, these species differences must arise from an evolved difference in pair bonds.

Molecular methods of DNA paternity are required to establish whether a mating system that is **socially polygamous** or **monogamous** is actually **genetically polygamous** or **monogamous**. Additional parternity studies on another socially polygynous species, the wood mice, *A. sylvaticus*, indicated that 85% of all litters are genetically polygynous and thus female wood mice are promiscuous and that the larger males typically sire more progeny than the smaller males (Bartmann and Gerlach 2001).

The pattern of monogamous and polygynous **sister taxa** is repeated across many genera of rodents. For example, territories are more widely distributed in both prairie voles and pine voles (*Microtus ochragaster* and *M. pinetorum* respectively*)* compared to two congeners, meadow voles and montane voles (*M. pennsylvanicus* and *M. montanus*, which

are both found at much higher densities. A corollary of a socially monogamous system is the evolution of bi-parental care in which males assume a role in taking care of young. Endocrine regulation of care (Chapter 13) is independent of the regulation of pair bond formation.

The neuroendocrine bases of monogamy and pair bond formation

Recent studies have elucidated neuroendocrine bases of pair bond formation in prairie voles relative to absence of pair bond in meadow voles. To understand the anatomical regions of the mammalian brain that give rise to these differences you first need to dust off your copy of the rat brain atlas and learn about a several important control centers that govern behaviors (Fig. 11.8). The **Nucleus Accumbens** (NAcc), which along with the pre-limbic cortex, are key control areas for the **reward reinforcing pathway** for natural stimuli like **appetitive behaviors** that involve food reward as well as aberrant behaviors like drug abuse

(MacBride et al. 1999). The receptor for **dopamine**, the neurotransmitter in this pathway, is concentrated in the NAcc (Fig. 11.9ef). This pathway plays a direct role in **conditioned learning** (Chapter 17).
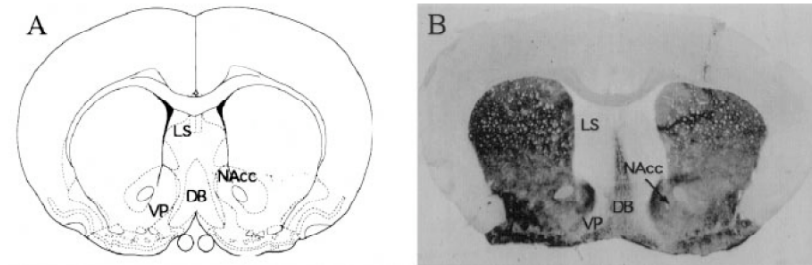
Figure 11.8. (A) Rat brain atlas illustrating the neuroanatomical boundaries of the Nucleus Accumbens (NAcc) diagonal band (DB), ventral pallidum (VP), and lateral septum (LS) (from Young et al. 2002 after Paxinos and Watson, 1998).
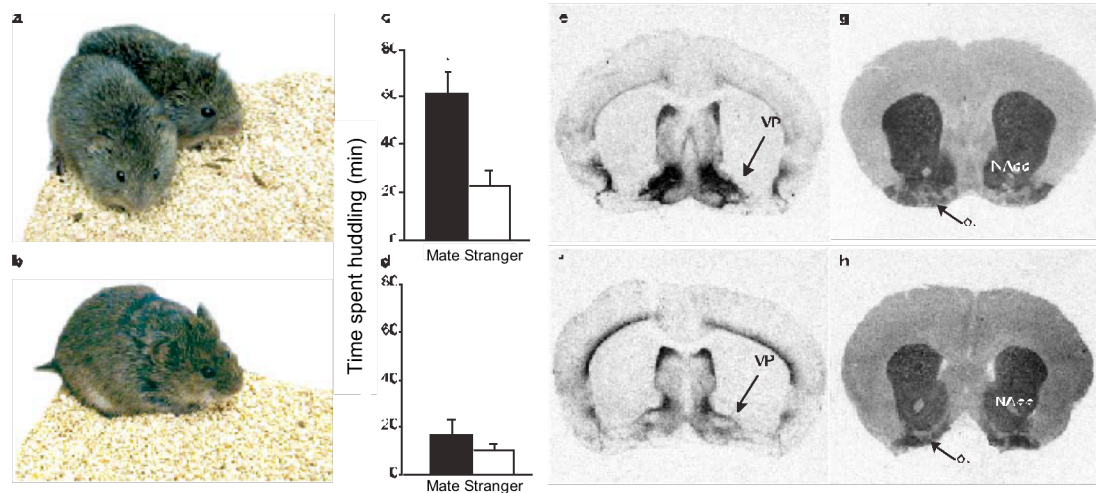
Figure 11.9. Comparison of brain neurochemistry and behavior in prairie and meadow voles. (a) Prairie voles are highly affiliative as depicted here in 'huddling' side by side, (b) whereas meadow voles are solitary. (c) After mating and cohabitating with a female, a male prairie vole tends to spend significantly more time in contact with the partner (filled columns) than the stranger (open columns). (c) Meadow voles do not form partner preferences and spend less time huddling with either the mated or novel female. Autoradiograms of the ventral forebrain illustrating the typical prairie vole (e) and meadow vole (f) expression pattern of the Vasopressin 1a receptor (V1aR) as shown by autoradiography. g, h, Despite considerable species differences in V1aR pattern, the Dopamine 2 (D2) receptor distribution was broadly similar in prairie voles (g) and meadow voles (h), as shown by D2 receptor binding in the nucleus accumbens (NAcc) and the olfactory tubercle (ot). Scale bar, 1 mm. (from Lim et al. 2004).
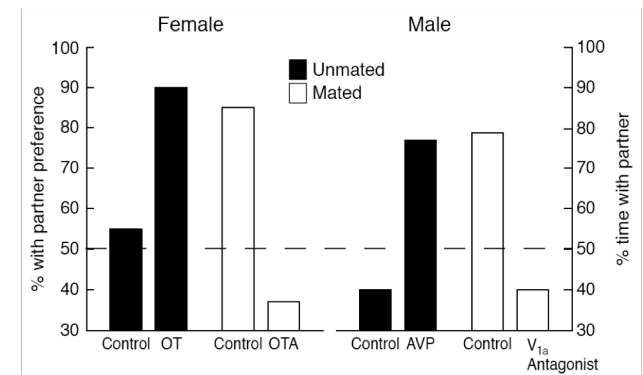
Figure 11.10. Effects of intracerebroventricular oxytocin (OT) and vasopressin (AVP) on partner preference formation in prairie voles. Oxytocin facilitates the formation of partner preferences in unmated females towards male cagemates when compared with controls, whereas the oxytocin receptor antagonist (OTA) blocks the formation of preferences in mated females. Similar results are found in male prairie voles after administration of AVP or an antagonist of the AVP receptor subtype V1a. These effects are gender-specific: OT has little effect in males and vasopressin does not facilitate partner preference formation in females (from Young et al. 1998).

10

In the monogamous prairie vole, pair bonds are established during mating, a rewarding act that triggers circuits of the NAcc and pre-limbic system. The prairie vole (*Microtus ochrogaster*), which is highly social, forms lasting pair bonds after mating. Pair bonded males prefer the company of the mate and exhibit 'selective' aggression towards other members of the species. The breeding pair nests together: both parents provide extensive, prolonged parental care, and the offspring remain in the parental nest for several weeks beyond weaning. By contrast, the montane vole (*Microtus montanus*), which is relatively asocial, nests typically in isolated burrows and breeds promiscuously. Breeding partners do not form a pair bond after mating, males are not parental, and females abandon the offspring in the second or third postnatal week.

If each species is brought into the laboratory environment, they exhibit similar differences in behavior as observed in the wild (Lim et al. 2004). A key behavior relates to the degree to which pair bonded individuals will huddle together in a cage (Figure 11.9ab). Prairie voles exhibit **filiative behaviors** and prefer to huddle next to familiar females relative to an unfamiliar female (Fig. 11.9c). In contrast, montane voles do not exhibit partner bonds (Fig. 10.3d). These differences in behavior appear to be linked to the distribution of vasopressin receptors in the brain, and prairie voles have a much higher concentration of V1aR in the NAcc compared to montane voles. Differences are not tightly associated with Dopamine receptors (DA D2), which bind in the ventral palladium (VP).

Experiments using exogenous hormone injections of OT and AVP, as well as OT and AVP antagonists that are applied to female and male prairie voles, indicate that OT and AVP govern partner bonds (Fig 11.10). The critical test of the hypothesis that partner filiative behavior in males is mediated by a simple gene difference in V1aR is to perform a transgenic experiment and express the AVP receptor from the prairie vole, V1aR-vp in the brain of a male montane vole. This can be done by surgical injection of a viral vector that contains an insert of V1aR-vp attached to a LacZ promoter (Fig. 11.11). When the V1aR-vp gene is expressed in montane voles (via the LacZ promotor), polygamous males express the pattern of a pair bond prairie vole (Fig. 11.12-13).

Neuroendocrine regulation of social behaviors in mammals is thus, regulated by OT and AVP secretion by the hypothalmus (Side Box 11.2). Key brain regions like the amygdala and the Nucleus accumbens integrate such bonds, however, males and females differ in the degree to which OT and AVP establish filiative behaviors. The effect of hormones and their antagonists on the sexes is illustrated in Fig. 11.9 and discussed in Side Box 11. While V1aR-vp in the VP elicits pair ponds in males, these transgenic mice still lack parental behavior. As we have seen in Chapter 2, genes like *fosB* affect parenting behavior. In Chapter 13, we will also explore the role of the hormone prolactin in mediating migratory and parenting behaviors. How do such linkages among disparate gene networks become integrated (e.g., no parenting in male montane voles and elaborate bi-parental care in prairie voles)? The answer is correlational selection; however, no one has established this connection, although it must have played a role in the evolution of care.
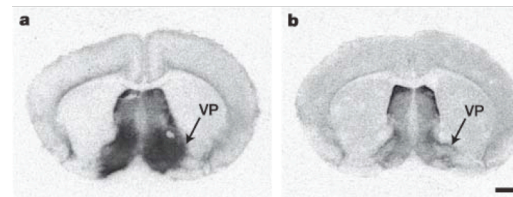
Figure 11.11. Autoradiography of V1aR in the ventral palladium (VP). a) Meadow vole overexpressing V1aR-vp in the VP by gene transfer. b) Meadow vole infused with viral vector in the VP expressing only the lacZ gene (Ctrl-vp) (Lim et al. 2004).
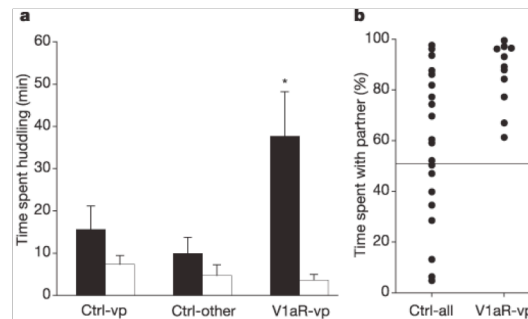
Figure 11.12. Partner preference test in transgenic mice. A) V1aR-vp meadow voles spent significantly more time huddling with the partner (filled column) than the stranger (open column), whereas control animals (Ctrl-vp) and stereotactic misses (injection site of the VP missed) (Ctrl-other) did not. b) A plot of the percentage of time spent with the partner for each subject indicates a shift from randomly distributed preferences in control groups to 100% of animals preferring the partner in the V1aR-vp group (from Lim et al. 2004)
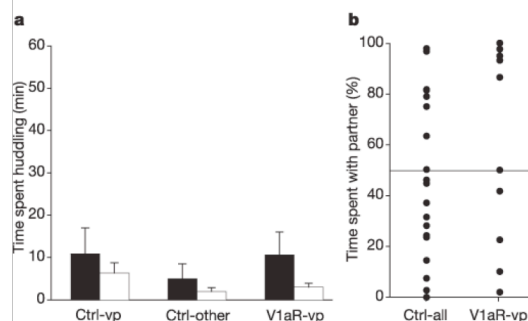
Figure 11.13. Partner preference test after eticlopride pretreatment. a) Eticlopride blocked partner preference in the V1aR-vp group. V1aR-vp, Ctrl-vp and Ctrl-other animals did not spend significantly more time with either the partner (filled columns) or stranger (open columns) b) Plot of the percentage of time spent with partners for each subject shows a similar distribution between control and V1aR-vp groups, with no significant difference from that expected by random chance (from Lim et al. 2004).

Side Box 11.2 Pair bonds, Oxytocin, and Vasopressin

Oxytocin (OT) is released within specific cells of the hypothalamus referred to as **oxytocinergic neurons**. Upon appropriate stimulation OT regulates neuroendocrine and autonomic functions related to reproduction and prosocial behaviors (supported by its cognitive effects) and emotional responses contributing to relaxation, trust, and psychological stability (from Neumann 2007). OT mainly acts in females. Vasopressin (AVP) acts to establish pair bonds in males. AVP is secreted by hypothalamic **vassopressinergic neurons**.
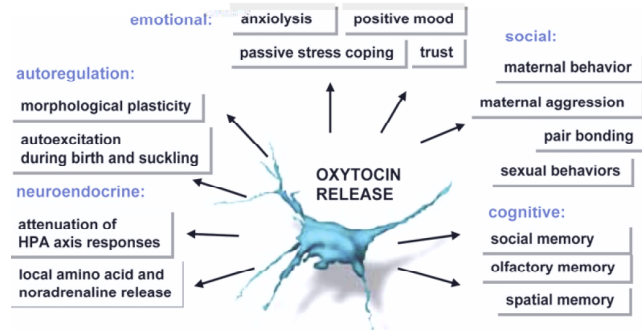
Figure 11.14. Vasopressin receptors (V1aR) and oxytocin receptors (OTR) are located in key control regions of the brain and serve to integrate sensory inputs via the **amygdala**, to the **nucleus accumbens** (Nacc) thereby generating the **primary partner imprint** during the reinforcement process of mating. Activation of Dopamine (DA) receptors (D2) and OT receptors in females and DA D2 and V1a Receptors in males in the ventral palladium underpins these forms of **associative learning** (see Learning, Chapter 17). Keverne and Curley (2004) suggest that **partner imprints** could be extended by further associations to incorporate vision, hearing and update odor changes induced by diet as well as endocrine states of each sex. (Abbreviations AMY amygdala, Me medial, BL basolateral C central; AOB accessory olfactory bulb, BNST bed nucleus of the stria terminals, OB olfactory bulbs, PC pyriform cortex, VMN ventromedial nucleus of the hypothalmus, VNO volmeral nasal organ, VS Ventral subiculum, VTA, ventral tegmental area) (from Keverne and Curley 2004).
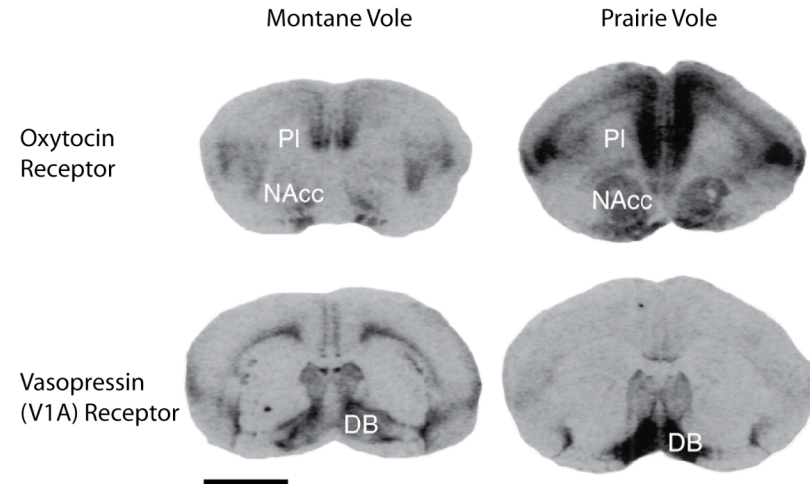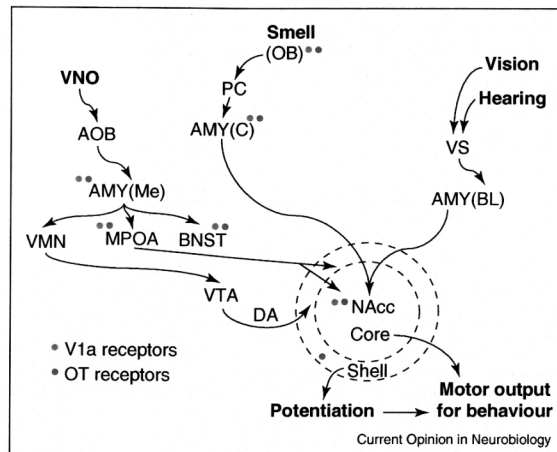
Figure 11.15. Autoradiographical localization of oxytocin receptor (OTR) and vasopressin-receptor subtype V1a (V1aR) binding in brains of montane and prairie vole. Oxytocin (OT) and V1aR autoradiographical studies (top and bottom rows, respectively). Compared with OTR binding in montane vole brains, binding in prairie vole brains is high in the prelimbic cortex (PI) and the nucleus accumbens (NAcc), whereas V1aR binding is intense in the diagonal band (DB). Similar species differences are found throughout the brain. Scale bar, 2.5 mm (from Young et al. 1998).

Female filiative behavior. The brain's oxytocinergic system along with olfactory recognition (e.g., smell, VNO and MHC recognition -- see Chapter 10) underpins the formation of female social relationships be they with mates, offspring or kin. Familiarity brings about this relationship through prolonged contact and grooming behavior. Pregnancy and oestrus provide the endocrine environment for OT and OT receptors. Oestrogen acts through several receptors including ERα, which is required for synthesis of OT receptors in the Amygdala and ERβ, which is required for synthesis of OT receptors in the hypothalmic neurons that actually synthesize OT. OT is also instrumental in milk let down post-parturition.
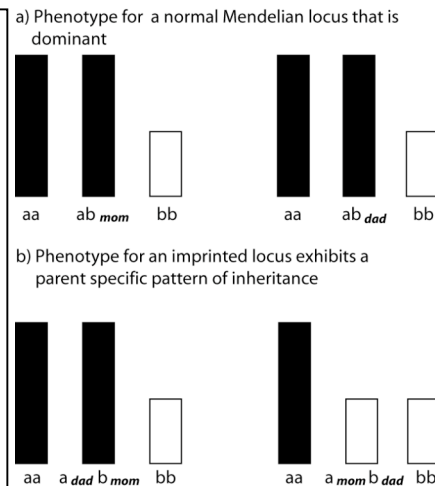
Male filiative behavior. The brain's vassopressinergic system has diverged in male prairie voles with regards to the distribution of the V1a receptor and release of AVP. AVP is released centrally following either cohabitation or mating with a female leading to the development of a pair bond, increased aggression towards strange males, but not paternal care. Differences between montane and prairie voles in the social expression of behavior are associated with variation in neural expression of V1aRs. The V1aR gene has >99% sequence homology between prairie and montane voles. They mainly differ in the 5' regulatory region in which the prairie vole has a microsatellite insert. A simple mutation in this regulatory region thus yields a profound change in behavior. AVP-mediated circuits seem to be involved in other pair bonding species like the California deer mouse (Bester-Meredith et al. 1999) and rhesus monkey (Young et al. 1999).

Genomic imprinting and parent-offspring conflict

Differences between the sexes in parental investment have consequences for genetic conflicts that invoke the **progeny quality trade-off** (Chapter 3, Fig. 11.1). In sexually reproducing organisms, offspring inherit alleles from both parents. Since the beginning of the Neo-Darwinian Synthesis (Fisher 1918), both maternal and paternal alleles were assumed to contribute equally to expression of offspring traits. However, recent molecular studies have revealed that in some species expression of either the maternal or paternal allele is silenced or reduced in offspring (Reik & Walter 2001). Parent-specific allelic expression is referred to as genomic imprinting (Fig. 11.16). To date over 70 imprinted genes have been identified in mammals (Murphy & Jirtle 2003) (see Side Box 11.3, Fig. 11.19). Genomic imprinting results from DNA methylation during gametogenesis (Fig. 11.17), that is often maintained until gametogenesis in the next generation when previous methylations are removed and are reapplied in the imprinting sex (See Side Box 11.3, Fig 11.18).

Diploid gene expression is thought to function as a valuable defense against expression of deleterious recessive mutations (Orr 1995, Otto & Goldstein 1992), so the functional hemizygosity resulting from genomic imprinting is thought to decrease fitness. As a result, there has been a great deal of interest in the benefits that may outweigh potential costs of genomic imprinting. Many hypotheses have been presented to explain the evolution of genomic imprinting (Wilkins & Haig 2003), but the most widely accepted is based on genetic conflicts between dams and sires arising from relatedness asymmetry.

Figure 11.16. Parent-specific gene expression. a) Normal Mendelian dominant locus. The heterozygote is dominant regardless of whether **b** alleles are of maternal or paternal origin. b) In the case of paternally imprinted alleles, heterozygote $a_{mom}b_{dad}$ (right panel) where **b** is of paternal origin, yields a **hemizygotic effect** in which level of phenotype expression is similar to a **bb** homozygote. Conversely, when mom contributes **b**, phenotype expression (left panel) is again similar to the situation in which allele **a** from the sire yields the same phenotype as a dominant **aa**. In this case the allele is dominant when it is derived from the sire, while the allele is recessive whenever it is derived from the dam.

a) Phenotype for a normal Mendelian locus that is dominant

aa   ab $_{mom}$ bb   aa   ab $_{dad}$ bb

b) Phenotype for an imprinted locus exhibits a parent specific pattern of inheritance

aa   a $_{dad}$ b $_{mom}$ bb   aa   a $_{mom}$ b $_{dad}$ bb

The parent-offspring conflict hypothesis.

The conflict hypothesis for genomic imprinting is an extension of parent-offspring conflict theory (Trivers 1974) based on Hamilton's ideas of inclusive fitness (Hamilton 1964). Hamilton (1964) showed that the frequency of a gene will increase in a population if direct fitness costs are exceeded by indirect fitness benefits, weighted by the coefficient of relatedness between individuals (r) (see Equation 4.1-4.3, Chapter 4). Haig & Westoby (1989), however, recognized that r is a combination of relatedness through both maternal and paternal lineages, which often differ among progeny depending on levels of polyandry. This asymmetry in the maternal and paternal relatedness introduces conflicts between the interests of each parent (Figure 11.17).

Such interparental conflict resulting from relatedness asymmetries can arise over resource allocation to current investment in progeny vs. future reproduction. For example, sires and dams benefit from increased juvenile survival due to greater maternal investment. Increased current investment, however, incurs reproductive costs to females but not males. In strictly monogamous mating systems, siblings share the dam and sire, so genetic interests of a sire are congruent with genetic interests of the dam. However, in polyandrous mating systems, paternal alleles favor greater maternal investment in current offspring for which paternal relatedness is greater, at the expense of investment in future broods, for which paternal relatedness may be lower. In addition, polyandrous broods contain progeny unrelated to the sire, but r = ½ to the dam.

Figure 11.17
relatedness of alleles ➝   0   1/2   1/2   1/2
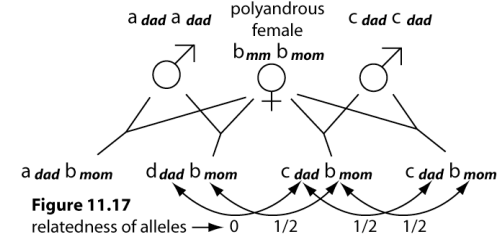
Interparental conflict over maternal investment is manifested in growth rate of offspring; paternal alleles favor increased offspring growth rates and increased maternal investment in current reproduction. Interparental conflict is predicted to increase expression of paternal growth alleles and silence maternal regulators for growth (see Side Box 11.3, Fig 11.20, for an example involving genes expressed in the placenta). The conflict hypothesis, therefore, makes two predictions about the occurrence and direction of parental asymmetries in gene expression. First, conflict-based genomic imprinting is predicted to occur when there are asymmetries in both relatedness and parental investment.

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Side Box 11.3. Molecular mechanisms of genomic imprinting
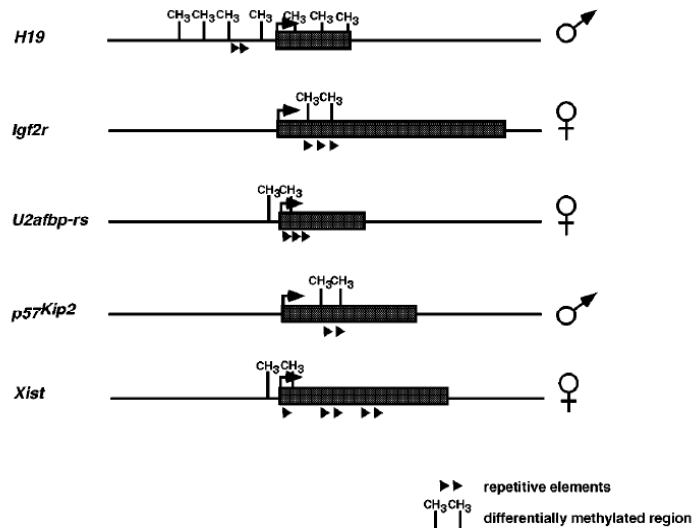
**Figure 11.18. Associations of allele-specific methylation with repetitive elements.** Five imprinted genes with allele specific methylation are depicted along with parent-specific chromosome that are methylated. Methylation is upstream of transcription initiation.
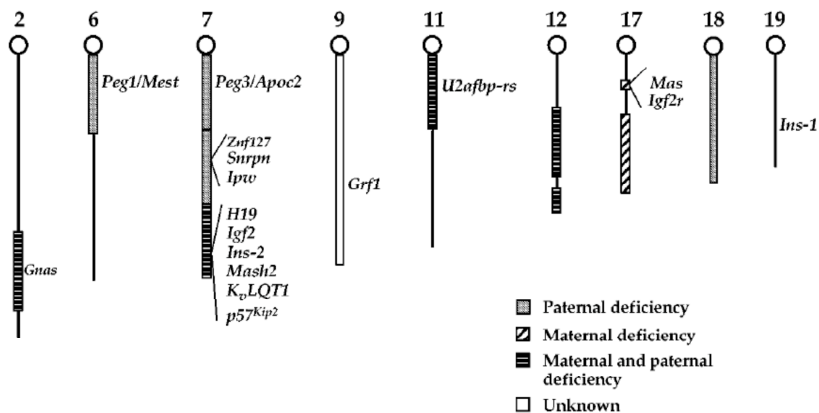
Figure 11.19. Imprinting regions of the mouse genome. The location of various imprinting genes (identified by **genetic complementation tests** involving maternal duplication/paternal deficiency or vice versa). Thin thin lines indicate no imprinting. Imprinting is widely distributed across the genome.
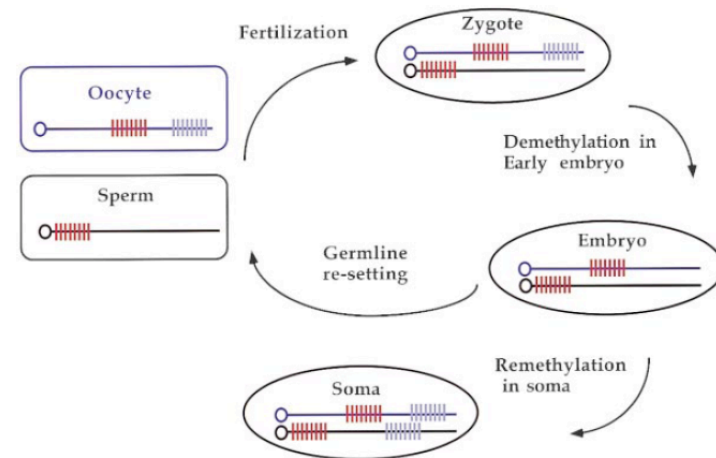
Figure 11.20. The ontogeny of genomic imprinting. At fertilization the zygote inherits one parental chromosome with a gametic methylation imprint (red bars) as well as other methylated DNA that is not sustained in the early embryo (grey bars). The other chromosome is not methylated at the gametic imprint. The embryonic cells are distributed between the soma, which is methylated, and the germ line, where methylation is erased and reset in an allele-specific manner, depending on progeny gender (e.g., female vs. male-imprinted genes).
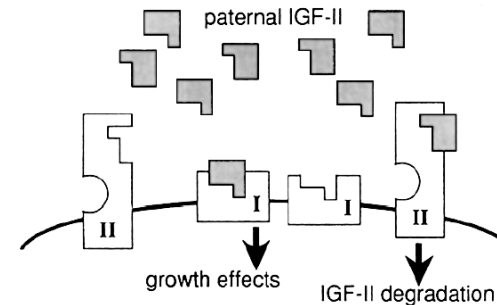
Figure 11.21. The molecular mode of action of imprinted genes. IGF-II (Insulin-like growth factor II) interactions with two receptors on the growing embryo, but only receptor IGF-1, is maternally-imprinted. IGF-II is paternally imprinted. In the absence of the maternally imprinted receptor 1, progeny carrying the paternally imprinted IGF-II extract more energy, which exacts a metabolic cost on the mother. Other progeny in the same litter, derived from a different sire that lacks imprinting at IGF-II, suffer lower growth. Notice that IGF-1R, IGF-2R, and IGF2 are found on different linkage groups (Fig. 11.19) and thus are an example of an epistatic interaction called a trans-acting factor, rather than a cis-acting factor [trans – at distance vs. cis – next to or upstream regulator]. These are fixed in most species, but species with both monogamous and polygamous males, may harbor alternative imprint alleles because a monogamous mate is mutalistically interested his partner's longevity.

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Genomic imprinting is thought to be common in polyandrous mammals (in which females are primarily or solely responsible for investment in offspring). The distribution of genomic imprints in species with alternative mating strategies remains unresolved (see Side Box 11.3). The conflict hypothesis makes explicit predictions about which parental gene should be silenced and which should be expressed. For example, mammalian growth enhancing genes should be maternally silenced while growth inhibitors should be paternally silenced. In general, the conflict hypothesis predicts that genes affecting maternal investment will be imprinted. After weaning there is no benefit to imprinting.

Empirical evidence of genomic imprinting has largely supported the conflict hypothesis both in occurrence and direction of imprinting effects (Side Box 11.3, Fig. 11.21). For example, insulin-like growth factor (*Igf2*), a growth promoter, exhibits paternally biased expression and maternal silencing in humans (Giannoukakis et al. 1993), mice (DeChiara et al. 1991), and pigs (Nezer et al. 1999), but not chickens (O'Neill et al. 2000), where maternal investment in eggs is thought to be independent of the embryo's genotype and thus not subject to interparental conflicts (Murphy & Jirtle 2003). In contrast, insulin-like growth factor receptor (*Igf2R*), a growth inhibitor, exhibits maternally biased expression and paternal silencing (Barlow et al. 1991). Despite such evidence for the conflict hypothesis from laboratory crosses and genetic analyses, the prevalence of imprinting in nature is unknown.

Speciation and the outcome of intraspecies imprinting

The classic example of genomic imprinting arises in the context of interspecific crosses involving the genus of deer mouse, *Peromyscus*. This example is salient for three reasons:

1) within a species, genomic imprinting in males and females is expected to come to an equilibrium where each paternal imprint that arises in males should ultimately be countered by a maternal imprint.

2) A corollary is that imprints should arise rapidly in an evolutionary arms race in which new genes are constantly evolving imprints in males to manipulate female effort in polygamous species, where imprinting should generate a strong form of genetic conflict between the sexes.

3) Monogamous species, lacking this evolutionary arms race between the sexes and offspring, should lack protective maternal imprinting, but may be vulnerable to paternal imprints from a polygamous species.

The pattern of imprinting should be fixed in most polygynous species, where the superior females that possess a counterstrategy will eliminate females that lack a counterstrategy to the male. However, at a hybrid zone between species the imprints of the polygynous males can act in an unprotected monogamous female background and exert their influence.

Thus, interspecific crosses between a monogamous species × polygynous species should result in imprints that affect the cross between $mom_{monogyny} \times sire_{polygyny}$ and the cross $mom_{polygyny} \times sire_{monogyny}$. The growth enhancing genes of $mom_{polygyny}$ will lack a counterstrategy from the $sire_{monogyny}$, while the converse is true for $mom_{monogyny}$.

Dawson (1965) carried out a cross between *Peromyscus polionotus*, a more monogamous species, and *P. maniculatus*, a polygynous species and observed asymmetries in the crosses; however, at the time genomic imprinting had not yet been discovered. Subsequent work by Rogers and Dawson (1970) (Figure 11.22) isolated the changes in fœtal weight and enlarged placenta to a cross with polygynous sire and monogamous dam (PM). The cross in the other direction (MP) resulted in a smaller placenta and fœtus.

Subsequent theory suggested that Dawson's experiments on crosses between species of mice and fœtal-placental size asymmetry were generated by genomic imprinting (Haig and Westoby 1989, Haig 1993). Polygynous males were hypothesized to carry imprinting alleles that extracted more energy from dams, via enlarged placentas (MP). In converse crosses, females subject to polygyny (PM) carried counterstrategic imprints, which acted unimpeded by sire imprints, thereby limiting placental size.
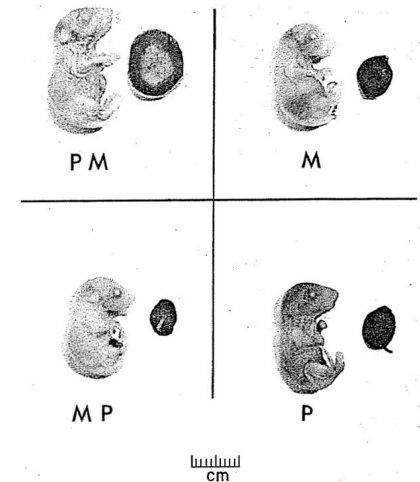
**Figure 11.22.** Results of crosses within *Peromyscus polionotos* (P) and *P. maniculatus* (M). Hybrid crosses (PM, MP) have the female of each species listed first. Notice the greatly enlarged placenta (PM) when female is the monogamous *M. polionotos* and the male is the polygynous *M. maniculatus*. Notice that in the converse cross the placenta is greatly reduced in size. (from Rogers and Dawson 1970).

Researchers have attempted to get at the mechanistic source of the size asymmetry. Haig's (1993) simple hypothesis regarding placenta relative to fœtal size has been refuted in interspecific crosses between the house mouse, *Mus musculus* × *M. spretus* (Side Box 11.4). Fœtal weight is not driven by placental size. Kurz et al. (1999) did show that retarded fœtal growth and size arose because of an imbalance in the proportion of a key set of cells in the placenta, glycogen cells that are in fact regulated by IGF2. As Kurz et al. (1999) point out, this does not invalidate Haig's (1993) hypothesis about fœtal and placenta size in being generated by imprints, rather simple relations are not observed, but imprints might act via the proportion of glycogen cells in the placenta. The experiments did confirm that IGF2 and IGF2R are responsible for the imprinting effects.

Side Box 11.4: Imprinting and interspecies crosses

Figure 11.23. Interspecies crosses that generate variation in relative size of fetus and placenta than control within species crosses. Placental size is unrelated to fetal size, a result inconsistent with a simple imprinting hypothesis (Haig 1993) in which imbalances between placenta and fetal size, generated by imprinted genes, drive fetal size in crosses (From Kurz et al. 1999).
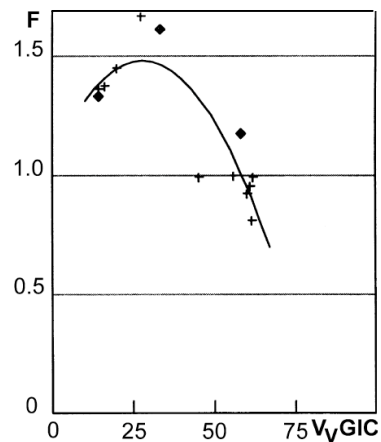
Figure 11.24. Fetal weight (F in g) vs. glycogen cell fraction (VvGIC). Optimum % of glycogen cells (30% GIC) is destabilized by imprinted IGF2, which control development of glycogen cells (GIC). Results from intra- and intraspecific crosses indicated by diamonds and pluses.

A summary of mating system trade-offs

The mating system trade-offs noted above arise because of signaler and receiver interactions among individuals (i.e., male-male, Chapter 8-9, male-female, Chapter 10) or gene interactions like greenbeards (Chapter 4). Sender and receiver co-evolution forms the basis of communication. Communication is defined to be behavior(s) (signals) in an individual that impacts behavior of another (or self) (Wilson 1975). At least some loci that govern signals differ from the loci that govern signal reception (e.g., not pleiotropic in effect). Moreover, the fitness outcomes of behaviors and traits that are elicited in receivers due to communication are due to loci other than just sender and receiver loci.

Social interactions generate 3 levels of CS: i) within sender CS which couples signal traits and other traits (and loci) that enhance or diminish activity of signals, ii) within receiver CS, between its receiver traits and behaviors or fitness traits or loci that are invoked as a consequence of signal reception, and 3) *between sender and receiver CS,* involving their different traits/loci and coevolutionary outcomes.

Trade-offs can occur at any of these three levels of CS, invoking a complex web of sender-receiver co-evolutionary process that functionally integrates the social system. Much theory assumes that CS is a property of selection at an individual level, but CS is actually a property of both inter and intra-individual selection in the case of communication. Only in the case of sexual selection, a special form of sender-receiver communication between mating partners, has this coupling been explored in any detail. However, mate choice is a special form of CS, under the umbrella of general communication theory (Kokko et al. 2006). The CS on sender traits and receiver responses are inherently FD (Sinervo & Calsbeek 2006). To understand the action of FDS we must distinguish between positive *vs.* negative FDS. For example, runaway sexual selection reflects positive FDS that fuels conditions for genetic correlations to build between the signal in one sex (often male) and preference loci in the other (often female).

Negative FDS is common in male competition where rare types have an advantage or common types are at a disadvantage (self-poisoning or self-limiting). Social systems with cooperation should generate positive FDS. For example, mating is a form of evolutionary cooperation in which each sex divides its genome and each passes on half to progeny.

The cooperation of sex is invadable by selfish genes, such as in **hybridogenetic mating systems** in which one genome ejects the partner's genome from the zygote (female genome usually ejects male: fishes, insects and amphibians [Simon et al. 2003; Normark 2003], but male ejects female genome in fire ants [Fournier et al. 2005]). Cooperation is also invadable by selfish genes such as genomic imprints that extract resources for some progeny at the expense of others, or from female parents, thereby invoking classic life history trade-offs.

West-Eberhard (1982, 1983) coined the term **sensory trap** in which individuals must engage in signal reception that lays that individual vulnerable to cheat strategies that take advantage of an open *receiver*. Genomic imprinting is a potent sensory trap in which females engage in signal reception to produce well-provisioned progeny. However, asymmetries in relatedness of progeny, due to polyandry and male polygyny, generate selection that favors the evolution of genes that take for themselves, even if it has detrimental effects on half-sib progeny or even the female's health on subsequent reproductive episodes.

Social system trade-offs

Social system trade-offs can also have a powerful synergizing action on primary life history trade-offs. Mating system trade-offs are best viewed as a special case of a social system trade-off, because mating systems impact juveniles. The social system consists of juveniles, males, and females and all their interactions. While juveniles of some species rarely interact with adults during the reproductive season, most juveniles eventually interact with adults if they visit breeding sites, or they must initially disperse to avoid adult interactions (see Chapter 13).

The forces shaping mating systems of adults therefore have ramifying effects during ontogeny. The trade-offs between traits beneficial for juvenile survival, dispersal and social behavior, promote different trait optima than those that enhance adult survival and reproduction (see above). Thus, ontogenetic conflict impacts males, females, and juvenile phases of the life history.

A formal discussion of social system trade-offs requires an explicit discussion of the root cause of behavioral trade-offs in social systems: signaler and receiver networks.

The extent to which mating or social system trade-offs act depend on the degree of information transfer during communication (Wiley 2000).

Signaler-receiver systems generate a trade-off with intended and unintended receivers that limit signal design (e.g., signal to noise ratio, Chapter 14) (Wiley 1994). Unintended receivers can be conspecifics or predators. In the case of conspecifics, a male that signals its resource holding potential to a conspecific male via a physiologically costly badge of status risks detection by alternative male strategies of crypsis that can intercept such signals. Honesty in signaler-intended receiver interactions (Chapter 8) forms a core component of the modern theory of communication; cheats either pay a cost of retaliation (Enquist and Liemar 1983, Enquist 1985) or individual recognition limits deceptive signals (van Rhijn and Vodegel 1980). As signals evolve to become more and more honest, which enhances the reliability of information transfer between signalers and receivers, they become more physiological costly, such as survival costs of testosterone production (Marler and Moore 1991) (Chapter 8), which are implicit in aggressive sexually selected male strategies (Figure 11.25).

In addition to signaler-receiver trade-offs and unintended receivers, other trade-offs arise from the cost vs. benefit of alternative social behaviors in social networks. Usurpation and cooperation reflect alternative social solutions to resource acquisition and territory defense
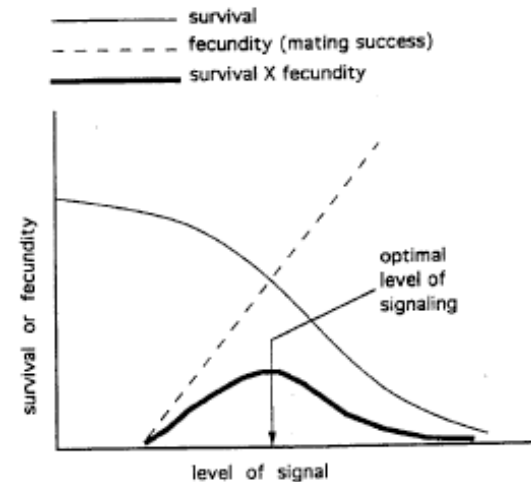
Figure 11.25. The level of signaling that optimizes survival × fecundity. In this general case male survival steadily decreases as his level of signaling increases, and thus signals are costly. A male's success in mating increases with the level of signaling. At an optimum level of signaling the highest fitness is given by a survival × fecundity trade-off (Wiley 2000).

(Calsbeek et al. 2000, Sinervo and Calsbeek 2002b). Cooperation is also common in foraging systems. Such cooperation is vulnerable to invasion by strategies that take by force or as noted above acquire information by guile without the costs of information collection (Zoltán and Szép, 1995). Thus cooperative strategies are invadable by strategies that take by force or usurp, which is in turn vulnerable to deceptive strategies.

Cooperation can be broken down into two forms (Hamilton 1964): a **mutualism** or an **altruism** (Levels of Selection, Chapter 4). Mutualisms evolve because both partners can achieve something, which they could not do without partner(s), and the reward benefits both. Altruisms evolve when the benefits of cooperation outweigh the potential risks (costs) in a social context. Both partners incur risks that ultimately only benefit one party, a recipient. Systems of genetically related altruist-recipient pairs are referred to as **kin altruism** (Hamilton 1964). Systems of genetically unrelated altruist-recipient pairs are referred to as **true altruism** (Sinervo et al. 2006a). Altruism can be viewed as a cost of mutualism, which arises in the context of alternative non-cooperative strategies (e.g., usurp vs. deceive). Here, I outline three interesting situations in which social trade-offs generate novel behavioral adaptations and counteradaptations: 1) kin altruism and sex ratio, 2) kin nepotism and queen killing behavior, and 3) greenbeard alleles for true altruism and mutualism in the context of alternative social behaviors.

Relatedness and eusocial systems

Eusocial systems involve a kin altruistic act in which one or a few individuals take the role of reproductive queen, while others adopt a sterile worker form. This situation often evolves in the context of a peculiar form of hymenopteran sex determination, referred to as haplodiploidy, in which males develop from unfertilized eggs. Males are a perfect hemiclone that reflects half of the queen's recombined genome. Sterile female workers and fertile queens develop from fertilized eggs. Depending on levels of promiscuity in the queen (e.g., number of sires), average relatedness in a hymenopteran colony can vary from 0.5 (different sire, same dam) to 0.75 (share sire and dam) (Trivers and Hare, 1976). [N.B., inbreeding in eusocial systems can push relatedness close to 1.0, as in the case of termites or naked mole rats (Sherman and Lacey 1997)]. In the case of colonies with multiple queens, a situation called polygyne, relatedness can vary from 0 (different dam, different sire) to 0.75. Single-queen colonies are referred to as monogyne.

Costs of eusocial behavior

Consider sex-ratio costs of eusociality (other than the cost of non-reproduction paid by workers). The optimum sex ratio for the queen is quite different than the optimal sex ratio for the sterile workers. The queen benefits from both daughter production (either workers or new queens) and son production (drone). However, daughters are only ½ related to the queen while sons are a perfect hemiclone, This generates an optimal queen sex ratio that is male biased for queens (Trivers and Hare 1976; Nonacs 1986, Boomsma and Grafen 1990). Workers benefit from a sex ratio that is proportional to their genetic similarity to each sex. Workers are only 0.5 related to male progeny (e.g., the recombined fraction of the queens genome). In the case of daughters, if they share the same sire, workers share a genetic similarity of 0.75. Thus, workers are favored to produce a skewed sex ratio in favor of daughters, while the queen is favored to produce an equitable sex ratio (Fig. 11.26). This, social trade-off of eusociality is nearly always resolved in favor of workers. Workers can police the reproductive output of queens, killing
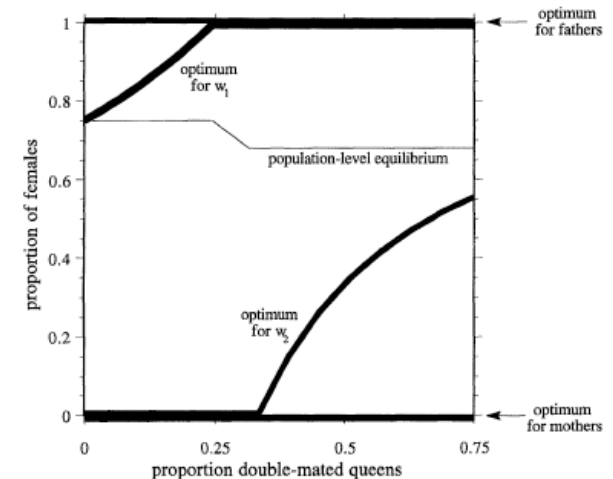
Figure 11.26. Optimal colony-level sex ratios for workers, mother queens (assuming that workers have caused overall female bias), and fathers according to the split sex ratio model by Boomsma and Grafen (1991) for monogyny, sterile workers, and single- or double-mating of queens. The worker optima (w1 and w2 for colonies with a single-mated and double-mated queen, respectively) are dependent on quantitative details of the variation in contributions by each male (the curve assumes the males contribute equally). The optima for mother queens and fathers are horizontal lines at sex ratios of 0 (all males) and 1 (all males) and independent of the frequency of single versus double mating in the population. The thin step function in the middle of the figure is the equilibrium sex ratio, which is 0.75 (3 :1) when single-mated colonies are the balancing class, and 0.67 (2 :1) when double-mated colonies are the balancing class (Boomsma and Grafen 1991).

excess males (as zygotes or unfertilized eggs) and restoring a female-biased sex ratio. The situation is only slightly more complicated in the case of colonies that form either by fission versus dispersal of the reproductive gyne forms (Pamilo 1991). Viewed from the perspective of workers, the Trivers-Hare (1976) sex ratio is seemingly cost free for the colony of workers and only the cost of sterile eggs laid by the queen is paid. The queen is cheating on workers, but workers evolve a solution because they control progeny rearing. Viewed from queen's perspective, the cost of worker behavior is enormous, given that it limits her adaptive optimum relative to the ideal trait in which she manipulates a colony for their reproductive strategy. Moreover, sex-ratio conflict is also a property of male-female conflicts over the use of sperm (Boomsma 1996) thereby invoking sexually antagonistic conflicts (noted above). Male hymenoptera should manipulate queen sex ratio via seminal fluid proteins, however, this prediction has yet to be demonstrated.

Other aspects of colony organization amplify the Trivers-Hare (1976) sex ratio conflict. In the case of polygne, mating system dynamics reduce genetic similarity among queens and among workers. With polygyne, colony members can be unrelated (different dam and sire, 0 relatedness) or as related as the maximum observed in monogynous colonies (same dam, same sire, 0.75 relatedness). Levels of polygyne vs. monogyne influence intensity of queen-worker conflict given its effect on genetic relatedness (Nonacs 1986, Boomsma and Grafen 1990).

Queen assassination and nepostic greenbeard alleles

Polygyne and monogyne in eusocial insects can also generate other social system trade-offs. The greater variation or skew in genetic relatedness found in such situations (e.g., 0-0.75) favors the invasion of **nepotistic alleles**, which enhance gene propagation of self at the expense of other related colony members. This is because nepotism can more easily invade social situations in which genetic similarity is more highly skewed among the social actors. If individuals in the kin group are more likely to be unrelated to other kin pairs, selection favors the evolution of behaviors that allow sub-groups to exploit other sub-groups. For example, in the red fire ant, *Solenopsis invicta*, a nepotistic allele has evolved that has pleiotropic effects on nonself recognition and queen-killing behavior. Female workers that carry a single copy of the *B* allele at this nepotistic locus will kill any maturing queen that is homozygous for the *b* allele. The *bb* workers lack such queen killing

behavior (Keller and Ross 1998). Experiments in which odors, extracted from *B* genotypes and applied to *bb* queens, confirms that a cuticular compound confers protection to *BB* or *Bb* queens from the queen killing behavior of *BB* or *Bb* workers. Thus *BB* and *Bb* workers identify and kill newly maturing *bb* queens that lack this novel compound. This reflects a greenbeard locus that indirectly enhances fitness of *BB* and *Bb* workers by removing competition experienced by their closely related (at the *B* locus) *Bb* queens, a form of **kin nepotism** (Alexander 1974). One reason why this social system trade-off generates costs is because the *BB* homozygous form is lethal for reproductive queens. Moreover, queen-killing behavior may impact colony reproductive output and fitness since *bb* queens are removed from the colony, unless new *Bb* queens can be quickly produced by the colony to return the colony to the optimal number of queens. Costs may also arise due to conflict among worker genotypes. Finally, *bb* queens, lacking the odor of *B* genotypes, pay the ultimate cost at maturity when *BB* and *Bb* workers assassinate them.

Queen killing is an example of a greenbeard allele (see Chapter 4). A greenbeard is a term coined by Dawkins (1976) to explain models of genic selection first proposed by Hamilton (1964). Greenbeard loci exhibit 3 behaviors: a signal, self- or nonself recognition of the signal, and signal recognition elicits social acts, which benefit others that share self signals or detriment others with nonself signals. Such greenbeard loci can be the source of many social system trade-offs, such as the noted greenbeard nepotism observed in kin-related ant colonies.

The social system of polygyne may exact many as yet unmeasured costs, when compared to the social system of monogyne. If a monogynous social system has higher colony output (because it has less relatedness asymmetry and thus less greenbeard nepotism) than a polygynous one, then the life history trade-offs are exacted at the level of colony reproductive output and survival, not merely among individuals. Such fitness relations that determine the full stability of the system have yet to be estimated, but it is central to the maintenance of alternative strategies of polygyne and monogyne and alternative greenbeard alleles.

The social costs of cooperation

Social trade-offs might arise because selection that favors mutualism that generates invasion conditions for alternative social strategies, which convert relations between mutualists into altruistic ones (Side Box 11.4).

One of the few social systems in which the fitness relations among all social actors have been estimated involves the genes controlling social behaviors of side-blotched lizards. Genetic crosses (Sinervo *et al.* 2001), gene mapping studies (Sinervo *et al.* 2006a) and theory (Sinervo 2001) confirm that color transmission in *Uta* behaves like a single-locus factor with 3 alleles (*o*, *b*, *y*). This yields 6 color phenotypes that reflect 6 genotypes (*oo*, *bo*, *yo*, *bb*, *by*, *yy*) of the OBY locus, named for the 3 color strategies of males (see Side Box 4.3, Chapter 4). Alleles at the OBY locus have co-dominant effects on color expression (two different alleles yields a throat with both colors), but dominant effects on male strategy and behavior. In males, the *o* allele is genetically dominant to *b* and *y*, and *y* is genetically dominant to *b* (i.e., the O phenotype = *oo*, *bo* or *yo*; B phenotype = *bb*, Y phenotype = *by* or *yy*). Male color morphs exhibit physiologies correlated with mating behavior, plasma testosterone, and territoriality (Sinervo *et al.* 2000a), and dispersal and settlement (Sinervo *et al.* 2006b; Sinervo and Clobert 2003). O males have high stamina, low survival, and patrol large territories with large female harems (Calsbeek and Sinervo 2002ab). O can be invaded by the sneaker strategy of Y males, which have low stamina, no territoriality, mimic female behavior, and cuckold harems of O males at high rates (Zamudio and Sinervo 2000). Y males are beaten in turn by pairs of B males that cooperate. The OBY locus appears to be a supergene that pleiotropically controls many male traits.

The male mating system of *Uta* is referred to as a rock-paper-scissors system because each strategy beats one strategy, but is each is beaten in turn by another, leading to an evolutionary cycle among three players: rock beats scissors, paper beats rock, and scissors beat paper. The highly cooperative blue strategy (**mutualism**) is invadable by O. When the competitive strategy of O (**usurpation**) becomes common, Y beats O by crypsis and female mimicry (**deception**). When Y becomes common, the cooperation of two genetically similar B males is mutualistic as these B males have much higher fitness than blue males, which lack a genetically similar partner to help it to chase away Y males (e.g., we refer to these B males as loners, because they lack a genetically similar partner to help them defend their territories, but these B loners live in similar crowded conditions as all male types). The genetic similarity of B male cooperators does not arise from kin philopatry. A deep pedigree for *Uta* (20 generations) indicates t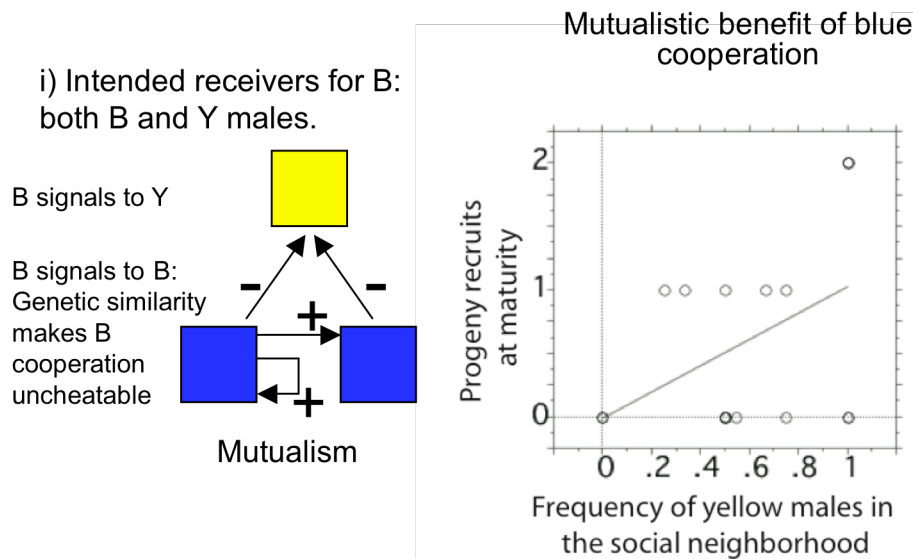hat completely unrelated B males find one another using a gene complex for self-recognition of blue color and of genetic similarity (Side Box 4.3). Thus, cooperative B males exhibit the three greenbeard traits: they carry signal genes that recognize self, and self-recognition elicits social acts that enhance fitness.

In order to cooperate, pairs of B neighbors must also share alleles at many self-recognition loci, including *b* alleles of the OBY locus (Side Box 4.3). When they find a genetically similar male partner their fitness is greatly increased. Thus, loci for self-recognition and settlement next to genetically similar males will become coupled to *b* alleles by the effects of correlational selection. In essence blue males have a social habitat preference for genetically similar blue neighbors, which enhances cooperation greatly and enhances fitness. This social habitat preference is highly heritable in the wild ($h^2 = 0.89$). Moreover the genes have analogous effects on female mating preferences for genetically similar mates ($h^2 = 1.05$) (Sinervo *et al.* 2006a). Conversely, alleles for self-repulsion become coupled to *o* alleles given that settlement next to genetically similar individuals generates zero fitness for O males, but high fitness if they settle with genetically dissimilar neighbors (Side Box 4.3). Finally, the *y* allele is relatively neutral with respect to settlement next to genetically similar neighbors (Side Box 4.3). Correlational selection on settlement behavior and the OBY color locus is an example of **fitness epistasis**. B males only obtain high fitness if they settle next to genetically similar partners. O males get no fitness in such situations.

Gene mapping studies in the deep field pedigree of *Uta* indicate that self-recognition genes are all unlinked (on different linkage groups) and are also unlinked from the OBY locus (Side Box 4.3), but yet under profoundly strong correlational selection. This correlational selection generates sender-receiver epistasis and very strong fitness epistasis (Side Box 4.3). The fact that all these genes are shared between the sexes also generates a mating preference in females for self similarity (Bleay and Sinervo 2006; Sinervo *et al.* 2006), but this preference is not a pleiotropic consequence of OBY, rather it is due to unlinked loci that govern social and mating partner preference (Sinervo *et al.* 2006a).

Social system trade-offs arise because selection that favors cooperation in B males (Side Box 4.3) generates social conditions for invasion of alternative social strategies (O beats B), which converts relations between B mutualists (B beats Y) into altruistic ones (Side Box 11.3).

Box 11.4. Social trade-offs from intended and unintended receivers in 3-way interactions:

i) Intended receivers for B: both B and Y males.

B signals to Y

B signals to B: Genetic similarity makes B cooperation uncheatable

Mutualism

Mutualistic benefit of blue cooperation

Progeny recruits at maturity

Frequency of yellow males in the social neighborhood

ii) O the Unintended receiver

O is attracted to B and settles near a B male

Altruism

Altruistic cost of blue cooperation

$\Delta W_R = -0.13 - 0.88 \quad \Delta N_O$

ΔProgeny Recruits at Maturity (ΔW_R)

Altruist B $\qquad \Delta N_O \qquad$ Recipient B

Figure 11.26. In situations with 3-way interactions between intended and unintended receivers strong tradeoffs exist in social systems. For example, blue males are involved in an honest signaling relationship that is enforced by the requirement of high genetic similarity at not just the OBY color locus, but also across the genome (Sinervo and Clobert 2003). This honest and cooperative signaling relationship is intended to thwart the much weaker yellow male strategy. However, owing to constraints placed on aggression in a cooperative relationship (asymmetries in RHP can destabilize relations, see Chapter 8), the cooperative strategy is susceptible to invasion by an ultradominant strategy that specializes in high RHP, which can beat cooperation (see Figure 11.27).
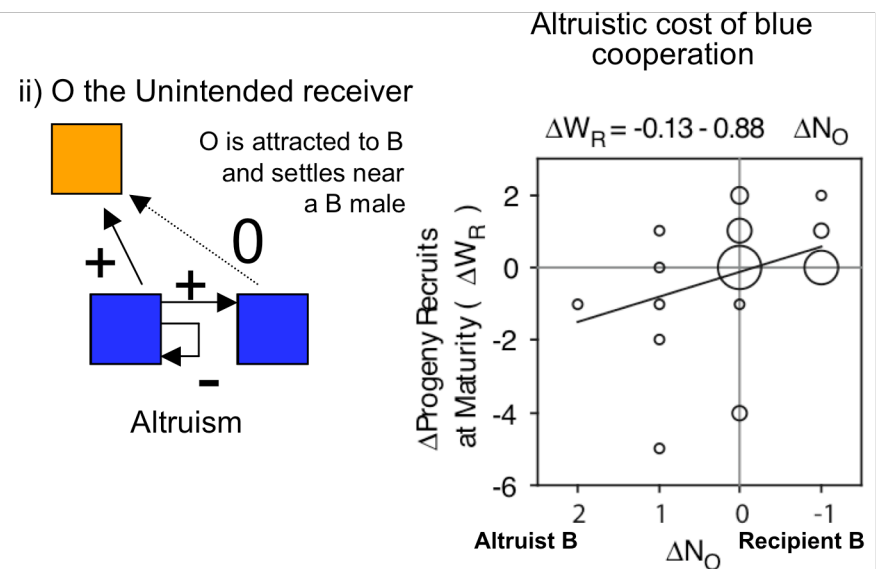
Figure 11.27. The orange male is an unintended receiver that is attracted to the mutual B signal (Sinervo et al. 2006a). When O invades B, genic selection favors altruistic behaviors in blue males that restrict the loss of fitness to only one of the males in cooperative dyads. One male buffers his territorial partner and receives no fitness, while the partner's fitness remains unaffected (e.g., This B still gains W from Y neighbors). This social situation is a pleiotropic consequence of the OBY signal being linked to testosterone regulation, and thus variation in both stamina and resource holding power (Sinervo et al. 2000a), thereby invoking life history survival tradeoffs among the males. O males have much lower survival across years compared to Y or B males.

Summary of social trade-offs. Rather than show hypothetical fitness relationships the actual fitness payoffs for B males against Y neighbors is shown in Fig. 11.26. In addition, the fitness asymmetry between B neighbors with asymmetry in number of O neighbors is shown in Fig. 11.27. Payoffs of a non-cooperative loner blue male strategy are not shown, but loner blue loses in Y neighborhoods, but beats the altruist strategy in O neighborhoods. (from Sinervo et al. 2006a)

Intended receivers:

1) Badge of status in male-male conflict: O beats B by virtue of the high RHP of O.

2) Color badges of males are also used in female choice (Bleay and Sinervo 2007).

3) Blue is a badge of cooperation to self genotype neighbors. B females that are interested in producing cooperative B sons also prefer B cooperators compared to loner B males that lack partners.
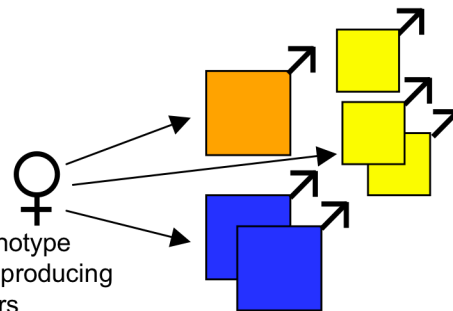
iii) Unintended receivers

Figure 11.28. Sneaker males can also cue in on the honest O badge, but rather than use cooperation like B, they exploit O with cryptic behaviors and leverage the mating system tradeoffs of monogamy vs. polygamy to their advantage, targeting vulnerable polygamous males (Zamudio and Sinervo 2000). Strategies of mate-guarding are resistant to sneakers, but require cooperation from neighbors. This does not preclude females from preferring Y males (Bleay and Sinervo 2007), which are favored for highly cryptic dorsal patterns that can benefit either sneaker sons, or daughters (Lancaster et al. 2007).

The greenbeard loci associated with B ('blue-beard' loci really) also result in a cost of cooperation that generates altruism on the part of one B male in a partnership. The B male whose territory is next to more O males ends up receiving no fitness, but this male buffers his B partner from the aggressive O male strategy. When O is rare and Y is common (Fig. 11.27), both B males enjoy high fitness and social relations are thus mutualistic between B males (Figure 11.26). Two cooperating and genetically similar B males can more efficiently defend females from Y sneakers. Therefore the social trade-offs in many social systems with cooperation are likely to arise from stable alternative strategies that can exact fitness costs (**usurp**), relative to other social strategies that yield fitness benefits (**deceit**). Social trade-offs arise from unintended receivers (Figure 11.28) that form the core fabric of every social system, and are so engrained that they will never disappear. The RPS reflects such a stable system of strategies that generate strong social trade-offs.

B males cycle between altruism and mutualism according to O frequency in the population (Fig. 11.29). These cyclical systems are **Evolutionarily Stable Social Systems** (ESSS), which by their very nature will last for long periods of time and keep social trade-offs that arise from epistasis (i.e. many genes), in chronic linkage disequilibrium.

There is reason to believe that the RPS of lizards has been cycling for over 175 million years. A new RPS has recently been discovered in the European common lizard (Sinervo et al. 2007) Lacertid lizard, that last share a common ancestor with the side-blotched, a Phrynosomatid

lizard, around the time of the dinosaurs. In fact snakes are more closely related to Lacertids, than Lacertids are to Phrynosomatids, and yet the RPS of both species share similar colors and one strategy exhibits evolutionary cooperation, which is in perpetual conflict with usurp and deceit. This type of social trade-off may be common in animals.

Biological mutualisms, may involve costs that are reflected as altruism or parasitism (Sinervo and Calsbeek 2006). Social trade-offs need not merely reside within a species, but may be fundamental to coevolutionary interactions that arise between species, which appear to be mutualistic, but involve costly parasitism (Thompson 2005). For example, the moth *Greya pollitella* pollinates flowers of the woodland star, *Lithophragma,* as an incidental byproduct of oviposition behavior. *Greya* larvae feed on some seeds, imposing a reproductive cost. A geographic mosaic across both species' ranges arises from parasitic costs vs. mutualistic benefits of *Greya* pollinating *Lithophragma,* relative to neutral pollinators like flies (Thompson and Pellmyr 1992).

Figure 11.29. Cycles of altruism and and mutualism in cooperative B male *Uta* are driven by cycles in the number of aggressive O male neighbours. a. Population cycle in the number of orange male neighbours experienced by blue males. b) Blue male fitness in three social contexts during the RPS cycle. The cycle of O neighbours drives an evolutionary cycle of altruism and mutualism in cooperative B males. The black line shows the fitness curve of the recipient of altruistic actions in a male partnership of genetically similar B male neighbours, while the grey line depicts the fitness of altruist, relative to a loner B male strategy (Dotted curve), which does not engage in group behaviours (i.e. loner B males lacks genetically similar neighbours). When O is common (labelled A for altruism) fitness of the B altruist (Grey curve), who has more O neighbours, dips below fitness of the loner B strategy (Dotted curve). At these times B altruists should defect, and adopt the loner strategy, but this egoistic behaviour has never been observed (Sinervo *et al.* 2006a). When O is rare (labelled M for mutualism) Y is common and both genetically similar B male neighbours obtain higher fitness than the loner B strategy and thus engage in evolutionary mutualism (figure from Sinervo *et al.* 2006a).
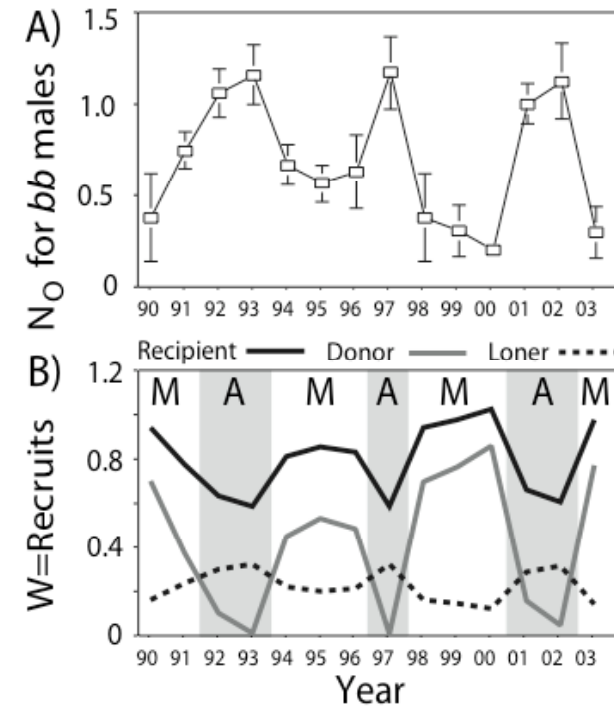
Conclusions about life history trade-offs and social systems

A consideration of these diverse trade-offs allows us to come up with a more synthetic view of the sources of constraints on behavioral systems with modification of Zera and Harshmann's (2001) diagram for the ramifying effect of life history trade-offs (Fig. 11.31).

First, demographic constraints will set up limits on what and which type of trade-offs between primary demographic traits are possible (Figure 11.31a). By primary demographic traits, I refer to traits that have direct effects on fitness (age at maturity, stage-dependent survivals and fecundities). Under the hypothesis that constraints (either internal, or external) set limits on population growth rate (population sizes are confined to some density or frequency attractor), then all primary demographic parameters cannot vary independently from one another (because population growth rate is fixed), with the consequence that they should covary. An increase in a trait that positively influences fitness will negatively influence other traits that positively influence fitness. However, positive covariation between traits can also occur if they each negatively influence a third. Multivariate trade-offs organize themselves among demographic traits, resulting in equal fitness.

Even greater multi-dimensionality of trade-offs might be the rule if they arise from epistatic interactions with physiological (Figure 11.31b) and/or behavioral machinery (Figure 11.31c). In some cases however, demographic constraints might be relaxed and species might escape certain trade-offs. For example, when species are expanding their range (e.g., at the northern margin of many species due to global warming), or when a species is expanding its niche (because of a new mutation or by modification of competition with other species) or when some alternative type replaces others within a population (mutants which replace residents, frequency-dependent polymorphism, etc.), one might find situations where the above predictions will fail and other types of constraint will prevail ending up possibly in a different organization of the trade-offs or even positive traits correlation (Reznick et al. 2000).

Physiological tradeoffs

Physiological trade-offs (Costa and Sinervo 2004) express internal constraints such those arising from energetic constraints (food processing, foraging time) or from deeper physiological organization (basal and activity metabolism, etc.). From the same energy intake, individuals might be favored to invest in testosterone production (given its influence in social system trade-offs) in place of immune products (in the absence of trade-offs arising from interspecific parasitism).

Variation in individual resource availability might however turn negative correlations among physiological traits (i.e., between hormonal and immune function, Figure 11.31c) into positive associations (Zera and Harshman 2001), rendering the study of such trade-offs difficult and the sign of the correlations environment-dependent (e.g., Ernande et al. 2004). To gain a better understanding, one needs to develop studies on the way trade-offs control and are controlled by hormones (Sinervo 1999, Sinervo and Calsbeek 2003) as well as the interaction among important physiological functions such as immunity, reserve storage and maintenance, or the social stress response (Zera and Harshman 2001; Costa and Sinervo 2004, Dufty et al. 2002).

Physiological trade-offs may or may not result in demographic trade-offs. Behavioral trade-offs may or may not result in demographic or physiological trade-offs, and vice versa.

For example, if a trade-off between vigilance and foraging results in trade-offs between survival and reproduction, a trade-off between sit-and-wait strategy and widely foraging might not result in any differences in survival and reproduction because both incur survival risk: parasitism for sit-and-wait and predation for widely foragers (Clobert et al. 2000). Sit-and-wait and widely foraging tactics also involve behavioral trade-offs of signal detection in the predator: cryptic patterns are often favored in sit-and-wait strategies, versus alternative escape patterns in the widely forage strategy. Such selection on morphology (and behavior) may amplify physiological performance trade-offs since widely foraging must adopt more costly antipredator escape behaviors involving speed, not just those involving high stamina.

Behavioral, mating system and social system trade-offs

Behavior can impart more dimensionality to trade-offs via ontogenetic conflict or mating system trade-offs (Figure 11.31d) of antagonistic sexual selection in the case of polyandry, polygyny, and parental care (if present), paternity assurance in males, or fertility assurance in females. Alternative strategies dimensionalize the trade-offs to a higher degree.

Social system dynamics further dimensionalize life history trade-offs (Figure 11.31d). While social trade-offs have usually been expressed in terms of cost-benefit equations [i.e., Hamilton's (1964) Eqn 4.1-3] these constraints can also be viewed as genetically based trade-offs that arise from pleiotropy (e.g., action of the OBY locus on *Uta* male life history traits, behavior, endocrine, and physiological systems) or from epistasis such as in signaler-receiver loci [e.g., self recognition loci that interact with OBY to generate conditions for cooperation (Side Box 4.3)].

A simple way to visualize social trade-offs is to view the benefits of mutualism as generating conditions for invasion of alternative strategies that convert mutualism to altruism (and vice versa) (Side Box 11.4). Conversely, highly aggressive strategies may be subject to invasion by highly deceptive social strategies. Highly aggressive strategies are incompatible with cooperation because of their egoist tendencies. Social trade-offs arise because of the fundamental requirement of communication in social systems, and because the benefits of communication with intended receivers come with attendant risks of exploitation by unintended receivers (conspecifics or predators).

Epistasis or gene interaction is universal in social behavioral interactions (Sinervo and Calsbeek 2006) because social behavior requires signalers

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Figure 11.31. Allocation graphs for visualizing life history trade-offs.

a. Demographic trade-offs. Bi-dimensional or multi-dimensional trade-offs (1 vs. 2). Star- or hierarchical trade-offs (3 vs. 4). The basal line represents some environmental constraints, which are similar for a given set of potential trade-offs. A change in the thickness of the basal line mimics a change in the strength of the environmental constraint, such as distribution of resources thought to govern monogamy vs. polygamy (S: survival, R: reproduction, A: age at maturity).

b. Physiological trade-offs. Potential trade-offs between the hormonal and immune system. α and β are two genotypes investing differently between testosterone and immune function. The correlation between investment in testosterone and immunity across genotype is then negative. However, if individuals vary in their resource availability (thickness of the root 15 versus 30), genotype γ can invest more in both testosterone and immunity when compared to β, leading to a positive correlation between testosterone and immunity across genotypes. However, the omnipotence of hormonal actions (Dufty *et al*. 2002) are likely to results in more complex trade-offs organisations (for examples δ and ε involve hormones corticosterone and leptin). Trade-offs might even change during development since both immune and hormonal systems have organizational effects (e.g., see Chapter 15) and might set up different forms of trade-offs late in life versus early in life (e.g., the role of prolactin in amphibian metamorphosis, migration or care, see Chapter 13). (T: testosterone, C: corticosterone, L: leptin, Is: specific immunity, In: non-specific immunity).
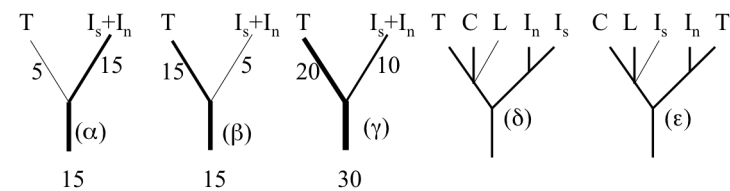
c. Behavioral trade-offs. Behavioral trade-offs involve foraging patterns, travel time, and vigilance. (Fo: foraging, V: vigilance, Tr: travelling) (see Chapters 5-6).

d. Allocation among demography, physiology, behavior, mating and social systems. Life history trade-offs involve physiological and behavioral systems, as well as mating systems and social system dynamics. This figure regroups trade-offs presented in previous graphs into a single multidimensional trade-off (same legend). The size of the circle in the centre of one possible system of trade-offs indicates the total amount of resources that is divided up into a complex pie, with the conflicting needs of each system receiving the optimal level of investment.
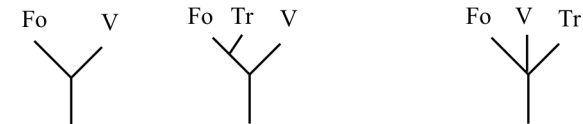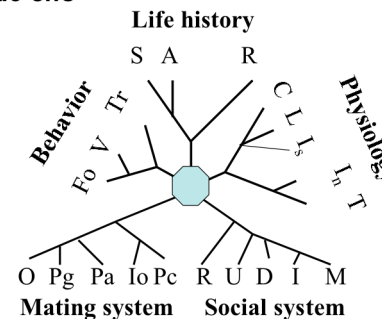

a. Life history trade-offs
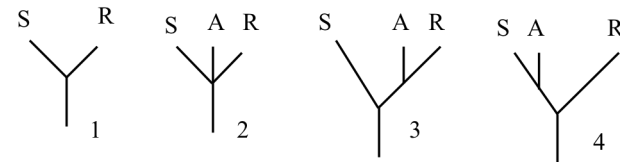
b. Physiological trade-offs

c. Behavioral trade-offs

d. Mating and social system trade-offs

Mating System:
O-ontogenetic conflict
Pg - polyandry
Pa - polgamy
Pc - parental care
Io - inbreeding vs. outbreeding

Social System:
R - relatedness asymmetry
U-unintended and
I - intended receivers
D - dispersal/philopatry
M - migration

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and receivers. The loci that govern signals will rarely be the same loci that govern signal reception and social acts. There may be exceptions to this generalizations such as greenbeard nepotism observed in fire ants (Keller and Ross 1998), where a simple cuticular compound elicits greenbeard interactions (signal, reception, social acts). However, olfaction of the cuticular compounds must invariably involve many more receiver loci than a cuticular signal, thus even this example is likely to involve hidden epistatic interactions of other signaler and receiver loci, which have yet to be identified and which build by correlational selection. For example, the three greenbeard traits (and/or loci) that comprise a Hamiltonian greenbeard can become bundled together by the force of correlational selection (Sinervo and Clobert 2003; Sinervo et al. 2006a) into social gene complexes involving signal, signal recognition, and social donations. Thus, Hamiltonian greenbeards need not arise from a pleiotropic supergene.

Given that mating behavior is ubiquitous in sexual organisms, sexual selection likewise generates strong fitness epistasis between loci for male signals that are unlinked from female preference loci. These signaler-receiver loci will become coupled by runaway sexual selection (Fisher 1931; Lande 1981). When female mate preferences or territory preferences exert ramifying trade-offs via costs of reproduction (e.g., via mating system, polygyny threshold), systems of trade-offs become multidimensional and are a property of genetic interactions, not just pleiotropy. In this regard, behaviors make the study of life history trade-offs far more difficult, but also far more interesting given the higher trade-off dimensionality that is invoked as mating or social system evolve. In chapter 13-14, we study signaler-receiver systems in detail.

Correlational selection, evolution of pair bonds and parental care

A final point is noteworthy regarding the evolution of monogamy and parental care. It is clear that the genes for paternal care are not due to V1aR, even though V1aR determines male filiative behaviors. Polygyny is ancestral for mammals. Male care and monogyny invaded polygyny. A pair-bonded female and her progeny would benefit from male care, particularly when such a monogynous strategy invaded polygyny. A care-giving and filiative male would also be more strongly favored if it directed care to its own progeny (just like in genomic imprinting). Thus, it is a logical conclusion that monogamy, pair bonds and paternal care, 3 derived traits, would arise in a polygynous-monogynous mating system.

Mongynous males that formed filiative bonds with their progeny would have enhanced survival of their progeny relative to polygynous males that provided no care. In this sense, the mutation in V1aR of prairie voles may have arisen first as a greenbeard that enhanced both male-female bonds and male-progeny bonds, but due to CS, became coupled to independent genetic loci for care, such as *fosB* genes or genes for prolactin regulation. FDS and CS on both the endocrine regulation of care and the endocrine regulation of filiative behaviors in males would have driven evolution of care-giving, filiative and monogynous behaviors, during the invasion of polygyny.

Correlational selection would have bundled filiative behaviors with care giving behaviors. However, the role of male-male filiative behaviors in the evolution of pair bonds has been largely ignored in behavioral ecology (except for the example of male *Uta* and Lacerta, both of which exhibit a form of care directed to self genotypes). The filiative behaviors in mammalian female-female bonds arise from oxytocin (Side Box 4.2). In males, V1aR governs the male-female bond. I suspect that the role of the V1aR mutation has a much more profound greenbeard role in the evolution of male-male bonds, between both progeny and adult males.

When they mature, progeny that received filiative relations from their monogynous sire would naturally want to establish filiative relations with neighboring territory holders, given the sire-progeny filative behaviors established during their youth. Therefore, the V1aR mutation of prairie voles may reflect a potent greenbeard locus, parallel to the blue-throat locus of lizards, but one that also brings in loci governing paternal-filiative care due the process of correlational selection.

The profoundly conserved neighbor-neighbor relations in monogynous voles (Fig. 11.7), even in sympatry with polygynous voles (e.g. absence of habitat differences), reflect the action of a locus that enforces territorial stability. The spacing of mongynous voles is not merely due to ecology. I am sure mixed polygyny-greenbeard monogyny strategies co-exist somewhere in the world, and a full dynamic unfolds in the modern day. I also believe that rodent populations harbor the full RPS dynamic in which non-territorial males excrete cryptic female-like urine signals. These cryptic males should co-exist with polygyny and monogyny, and obtain high fitness from the polygynous strategy. This mating system would constitute a mammalian RPS. If only we were Labrador retrievers, then we could sniff out such alternative strategies in rodents.

Study Questions.

1. Outline three methods for uncovering the genetic basis of life history tradeoffs.

2. What is ontogenetic conflict? What kind of life history tradeoff arises in the case of ontogenetic conflict?

3. What is antagonistic sexual selection? Give an example.

4. Outline the hormonal bases of pair bonding in a mammalian system. What hormones generate filiative behavior in females? What kind of filiative behavior do females exhibit? What hormones generate filiative behavior in males?

4. What kind of asymmetry gives rise to genomic imprinting?

5. Briefly sketch the genetic mechanisms that give rise to genomic imprinting. Give a developmental example of genomic imprinting.

6. What happens when you cross a monomagous species to a polygamous species and why?

7. What is the source of social system trade-offs?

8. What is the benefit of mutualism, what is the potential social cost of mutualism? What generates the costs of mutualism. (What physiological tradeoffs are involved in signals of the three basic alternative male strategies?)

9. Consider eusociality in ants. What is the optimal sex ratio for males. What is the optimal sex ratio for workers. What is the optimal sex ratio for queens. What generates these differences in sex ratio?

10. What is the source of epistasis (gene interaction) in mating system tradeoffs? What is the source of epistasis in social systems? (Hint: Think in terms of signaler-receiver loci. In mating systems consider runaway. In social systems consider the signals of cooperators and alternative strategies)