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PERSPECTIVE:

MATERNAL KIN GROUPS AND THE ORIGINS OF ASYMMETRIC GENETIC SYSTEMS—GENOMIC IMPRINTING, HAPLODIPLOIDY, AND PARTHENOGENESIS

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Abstract.—The genetic systems of animals and plants are typically *eumendelian*. That is, an equal complement of autosomes is inherited from each of two parents, and at each locus, each parent's allele is equally likely to be expressed and equally likely to be transmitted. Genetic systems that violate any of these eumendelian symmetries are termed *asymmetric* and include parent-specific gene expression (PSGE), haplodiploidy, thelytoky, and related systems. Asymmetric genetic systems typically arise in lineages with close associations between kin (gregarious siblings, brooding, or viviparity). To date, different explanatory frameworks have been proposed to account for each of the different asymmetric genetic systems. Haig's kinship theory of genomic imprinting argues that PSGE arises when kinship asymmetries between interacting kin create conflicts between maternally and paternally derived alleles. Greater maternal than paternal relatedness within groups selects for more "abstemious" expression of maternally derived alleles and more "greedy" expression of paternally derived alleles. Here, I argue that this process may also underlie origins of haplodiploidy and many origins of thelytoky. The tendency for paternal alleles to be more "greedy" in maternal kin groups means that maternal-paternal conflict is not a zero-sum game: the maternal optimum will more closely correspond to the optimum for family groups and demes and for associated entities such as symbionts. Often in these circumstances, partial or complete suppression of paternal gene expression will evolve (haplodiploidy, thelytoky), or other features of the life cycle will evolve to minimize the conflict (monogamy, inbreeding). Maternally transmitted cytoplasmic elements and maternally imprinted nuclear alleles have a shared interest in minimizing agonistic interactions between female siblings and may cooperate to exclude the paternal genome. Eusociality is the most dramatic expression of the conflict-reducing effects of haplodiploidy, but its original and more widespread function may be suppression of intrafamilial cannibalism. In rare circumstances in which paternal gene products gain access to maternal physiology via a placenta, PSGE with greedy paternal gene expression can persist (e.g., in mammals).

Key words.—Eusociality, evolution of sex, genetic conflict, genomic imprinting, haplodiploidy, kin selection, sexual conflict.

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There are certain asymmetries between males and females, and between male and female tissues of hermaphrodites. The size of gametes is the defining asymmetry, and there are usually many others as well. But there are also, in most lineages, certain conserved genetic symmetries between the two sexes. Sex chromosomes may be highly dimorphic but typically comprise only a small fraction of the genome. Across most of the genome—the autosomes—a striking set of symmetries is maintained between the sexes: each parent contributes an equal complement of autosomes to each offspring of either sex; alleles are equally likely to be expressed regardless of their parent of origin; and alleles are equally likely to be transmitted to offspring of either sex regardless of their parent of origin. Although there are of course different patterns of gene expression in the two sexes, these

differences are decoupled from transmission genetics; that is, an autosomal allele expressed only in males is no more likely to have been inherited from the father than from the mother, and no more likely to be transmitted to a son than to a daughter.

As far as we can tell, these symmetries are primitive in every lineage in which male and female functions are separate, and apply to an absolute majority of species across such lineages (White 1973; Bell 1982; Bull 1983; Suomalainen et al. 1987; Kondrashov 1997; Normark 2003). There are, however, many lineages in which one or more of these symmetries have been broken. Asymmetry of gene expression depending on parent of origin has been discussed under the rubric of genomic imprinting, and asymmetries of gene transmission have been discussed under the rubrics of partheno-

TABLE 1. The eumendelian symmetries and the asymmetric genetic systems that violate them (Bell 1982; Normark 2003; Burt and Trivers 2006).

Symmetry	Genetic systems in which symmetry is broken
1. Equal complement of autosomes from each of two parents	parthenogenesis (thelytoky, arrhenotoky), pseudogamy (=gynogenesis), androgenesis
2. Maternal vs. paternal origin does not affect expression of autosomal alleles	parent-specific gene expression
3. Maternal vs. paternal origin does not affect transmission of autosomal alleles	paternal genome elimination, hybridogenesis

genesis, paternal genome elimination, and haplodiploidy. These terms are briefly defined in the caption to Figure 1, and are discussed in more detail under Three Asymmetric Systems, below. (Note that I am considering here asymmetries in the fates of maternal vs. paternal alleles. There are several other kinds of genetic asymmetry—e.g., meiotic drive—that do not depend on parent of origin. These are not known or expected to have the same sorts of life-history correlates, and are beyond the scope of this paper.)

There are certain parallels between the ecological situations in which these various asymmetric genetic systems arise. Parent-specific gene expression (PSGE), haplodiploidy, and parthenogenesis have all been found (or at least perceived) to arise in lineages in which there is a prolonged association between mother and offspring or between siblings. Yet, for each asymmetric genetic system, a different cause has been suggested for its association with kin gregariousness (Hamilton 1967, 1978; Lively and Johnson 1994; Haig 2000; Normark 2004).

Here, I present a synthetic review of asymmetric genetic systems and argue that a more unified theoretical approach to asymmetric gene expression and transmission may be justified. I suggest that Haig's kinship theory of genomic imprinting (Haig and Westoby 1991; Moore and Haig 1991; Haig 2000) is the most promising basis for such a unified perspective. Conflicts of interest between maternally and paternally derived alleles within kin groups may have led to the evolution of asymmetric systems of nuclear inheritance, just as, earlier in eukaryotic evolution, conflicts between biparentally inherited elements within the cytoplasm may have led to the evolution of asymmetric systems of cytoplasmic inheritance (Hurst and Hamilton 1992; Randerson and Hurst 1999, 2001).

THE EUMENDELIAN SYMMETRIES

The typical sexual eukaryotic life cycle consists of an alternation between meiosis and syngamy. In meiosis, a diploid genome is split into two recombined haploid genomes, and in syngamy a new diploid genome is formed by the union of two haploid genomes. In plants and animals, there is an extreme dimorphism (anisogamy) in the haploid phase, and each new diploid individual is formed from the syngamy of one haploid egg (ovum) and one haploid sperm (spermatozoon). Despite the extreme morphological asymmetry between these haploid gametes, their genomes typically obey the following symmetries (summarized in Table 1): (1) Each gamete contributes an equal complement of autosomes to each zygote. (2) In each diploid individual, at each locus, the allele derived from the egg and the allele derived from the sperm are equally

likely to be expressed. (3) In each diploid individual, at each locus, the allele derived from the egg and the allele derived from the sperm are equally likely to be transmitted.

There does not appear to be any general term for genetic systems that conform to all of these symmetries. Most existing terms for such typical eukaryotic genetic systems refer not to their symmetry, but to other properties such as genetic recombination (*sex*, *sexuality*, *amphimixis*), ploidy (*diploidy*), gamete dimorphism (*isogamy*, *anisogamy*), or separation of sexes (*gonochorism*, *hermaphroditism*, *dioecy*, *monoecey*). To facilitate the discussion of symmetry per se, here I introduce the term *eumendelian* to refer to the class of genetic systems that conform to all three of the cardinal symmetries listed above.

These symmetries are rarely perfect. They do not apply to cytoplasmic elements or sex chromosomes, which is why their definition is restricted to autosomes. In some lineages with multiple sex factors, the distinction between sex chromosomes and autosomes may be difficult to draw (White 1973; Bull 1983). Polymorphism for insertions and deletions means that any two gametes are unlikely to have exactly the same number of base pairs, or even genes, in their autosomal complement, which blurs symmetry 1. Almost every model organism has been found to have parent-specific chromatin marking (McGowan and Martin 1997; Bean et al. 2004) or actual parent-specific gene expression (Lloyd 2000) of at least a few loci, and it is conceivable that no eukaryotes hew to symmetry 2 at all loci. Nonetheless, classical Mendelian genetics—which assumes all three symmetries for the loci considered—works well in most cases, rather like classical Newtonian physics, implying that asymmetries are often near zero. It has been argued that natural selection will normally act to minimize transmission asymmetries (Leigh 1971).

THREE ASYMMETRIC SYSTEMS

Haplodiploidy

Perhaps the most strikingly asymmetric genetic systems are those in which a male expresses and transmits only the genes he received from his mother. In some lineages, a male hatches from an unfertilized egg and never has a father at all (*arrhenotoky*, violating all three symmetries). In other lineages, a male starts development as a diploid zygote but his paternal chromosomes become inert heterochromatin (*genomic imprinting*, violating symmetry 2), and then are excluded from sperm and never transmitted (*paternal genome elimination*, *PGE*, violating symmetry 3). In the sciarid flies (Haig 1993a), a male's paternal genome is expressed but is

nonetheless excluded from sperm (violating only symmetry 3).

Haplodiploidy is a relatively complex system that arises rarely—perhaps only 20 times in nature—and often characterizes large and ancient groups, mostly ranking as families or higher (Otto and Jarne 2001; Normark 2004). The asymmetric genetics of haplodiploidy favors females, giving them a greater share of parenthood and boosting their relatedness to F_2 grand-offspring. A haplodiploid mother's grand-offspring through her sons are related to her as $r = 0.5$, since all her son's genes came straight from her without dilution. Brown (1963, 1964) was the first to interpret this asymmetry as the cause of the initial spread of haplodiploidy in an ancestral eumendelian population, allowing females that could exclude the paternal genome from their sons to genetically outcompete females that could not. But neither Brown nor those who have followed him (Hartl and Brown 1970; Bull 1979; Haig 1993a; Herrick and Seger 1999; Smith 2000) have proposed an ongoing adaptive significance for genetic asymmetry per se. Those who perceive present-day adaptive benefits of haplodiploidy have usually cited its scope for sex-ratio control (Hamilton 1967), or for purging deleterious mutations through male haploidy (Goldstein 1994; Richerd et al. 1994; Smith 2000).

Parent-Specific Gene Expression

This system is more subtle than haplodiploidy and was only discovered late in the 20th century (Reik et al. 1987). In PSGE, an allele inherited from the mother is expressed differently than an allele inherited from the father (violating symmetry 2). Unlike haplodiploidy, it affects a small minority of loci in the genomes in which it is known and does not typically affect allele transmission (Wilkins and Haig 2003; but see Naumova et al. 2001; Croteau et al. 2002). Also unlike haplodiploidy, the paternal allele may be more highly expressed at some loci and the maternal allele at other loci. Both paternal genome elimination and parent-specific gene expression are forms of genomic imprinting: they depend on heritable yet reversible “marking” of the genetic material that differs depending on whether it has passed through sperm or egg. Chemically, the marks primarily consist of different patterns of DNA (cytosine nucleotide) methylation, often accompanied by modifications of the protein component of chromatin (Bongiorni et al. 1999; Alleman and Doctor 2000; Ferguson-Smith and Surani 2001; Reik and Walter 2001).

Parent-Specific Gene Expression appears to be particularly important in mammals and in flowering plants (Haig 2002; Dindot et al. 2004; Autran et al. 2005). Since it is difficult to detect in nonmodel systems, its pervasiveness in the biota is hard to assess. Haig (2000, 2002, 2004) argued that PSGE results from conflicts of interest between maternally and paternally derived genes in the context of viviparity and other situations in which there is intimate contact between embryonic and maternally derived tissues (as in plant seeds). This theory has been successful at accounting for patterns of expression in a range of mammalian genes (Haig 2004; Burt and Trivers 2006). Although other adaptive explanations for PSGE have been advanced (Varmuza and Mann 1994;

McGowan and Martin 1997; Beaudet and Jiang 2002; Wilkins and Haig 2003), the kinship theory is the most promising and influential (Nicholls 2000; Burt and Trivers 2006) and is the one I will focus on here.

Thelytoky

Thelytokous systems are those in which males are absent, and consequently genetic exchange between lineages and genetic recombination are limited or nonexistent. This is a radical alternative to Mendelian genetics, and one might argue that it is only notionally asymmetrical, since it is only male-ness that has been disposed of, both haploid genomes being inherited symmetrically through the maternal lineage. There is some merit to this objection, and thelytoky fits somewhat uncomfortably into a classification of asymmetric genetic systems. But I include it here because I wish to highlight those patterns that do tend to connect thelytoky to the other systems. Mechanistic interrelationships between the major asymmetric systems are diagrammed in Figure 1. Facultative and cyclic thelytoky (alteration of thelytoky with sexual systems; see Normark 2003) clearly are asymmetric systems, with a persistent but reduced role for males.

Thelytoky is usually interpreted as conferring numerous whole-organism, short-term evolutionary advantages including a twofold fecundity advantage over outcrossing sex, fertilization assurance, and preservation of successful genotypes (Bell 1982; Hamilton 2001). Recently, many cases of thelytoky have been attributed to the action of intracellular bacteria, parasitically acting in their own interest and contrary to the interests of the nuclear genome (Huigens et al. 2000; Braig et al. 2002). But the asymmetric aspect of thelytoky has not been emphasized: unlike haplodiploidy and PSGE, thelytoky has not been widely interpreted as resulting from genetic conflict between maternally and paternally derived alleles.

GREGARIOUS KIN AND THE ORIGINS OF ASYMMETRIC GENETIC SYSTEMS

All three types of asymmetric genetic systems have been noted to arise in situations in which there is close and prolonged contact between maternally related kin, either in the form of viviparity or in the form of gregarious broods (Hamilton 1967; Lively and Johnson 1994; Haig 2000; Normark 2004). The case of PSGE is the one for which the connection between asymmetric kin groups and asymmetric genetics has been most clearly drawn (Haig 2000), and yet, ironically, it may be the system with the least comparative evidence for this connection, since so few independent origins are known. Possibly PSGE is best known in mammals only because mammals are physiologically the best-known organisms. Nonetheless, the kinship theory of genomic imprinting has been notably successful in accounting for the loci targeted (typically involved in maternal-fetal interaction) and the directionality of the allele silencing (Burt and Trivers 2006), so it is reasonable to suppose that PSGE is indeed a set of adaptations to close and prolonged mother-offspring contact.

In the case of haplodiploidy, Hamilton (1967, p. 481) was the first to remark on its correlation with “gregarious development, as a group of siblings, from egg to adult,” and

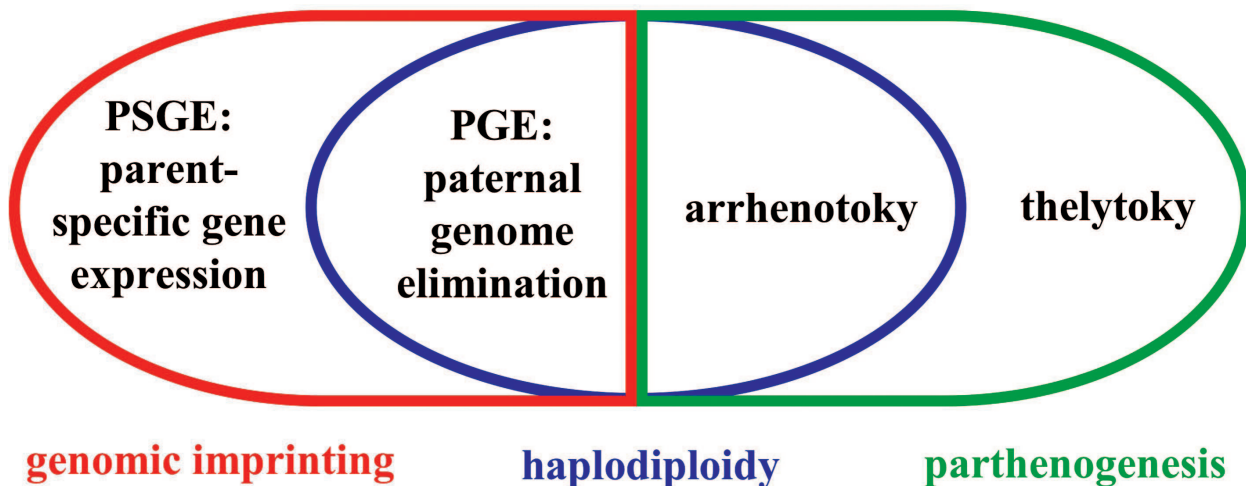


FIG. 1. A Venn diagram showing the interconnections of the main asymmetric genetic systems discussed in this Perspective. Broader terms covering more than one specific genetic system are printed in color and represented by a shape of that color. Genomic imprinting (red): differential expression or transmission of alleles, depending upon maternal versus paternal origin, at some or all loci. (This term can also refer to the parent-specific, reversible modification of the genetic material that cues the differential expression. This modification usually consists of methylation of cytosine nucleotides.) Haplodiploidy (blue): any genetic system in which males transmit only maternally derived alleles to their offspring. Parthenogenesis (green): the production of viable offspring from unfertilized eggs. More specific terms are printed in black. Parent-specific gene expression: differential expression of alleles at some loci depending upon maternal versus paternal origin. Paternal genome elimination (also called paternal genome loss or pseudoarrhenotoky): a genetic system in which males develop from diploid zygotes but in which the paternal genome of males is typically heterochromatic and unexpressed and is never transmitted to offspring. Arrhenotoky: a genetic system in which males are derived from unfertilized eggs and are haploid. Thelytoky: an effectively asexual genetic system in which unmated females produce diploid eggs that develop into daughters.

I have recently argued that close association of maternal kin in a brood chamber is a primitive condition of most haplodiploid insect clades and both nonarthropod haplodiploid clades (Normark 2003, 2004). However, this association remains conjectural pending a full comparative study, which may in turn have to rely on more complete phylogenetic and natural-history information about insects and mites than is currently available.

In the case of thelytoky, Lively and Johnson (1994) provide statistical evidence of a positive association between brooding and thelytoky across several groups of freshwater invertebrates. Their comparative study was not corrected for the influence of phylogeny, but this is unlikely to be a serious problem because of the very frequent independent origin of parthenogenesis. The association has not been tested in other groups, though freshwater invertebrates comprise a large proportion of the known thelytokous lineages (Bell 1982). Whether it holds across other groups is unclear. However, Hamilton (1967) noted the frequent occurrence of thelytoky in haplodiploid groups with gregarious kin. And I conjecture that, for insects, a comparative analysis would show an association between female winglessness and thelytoky (Bell 1982; Normark 2003; Triplehorn and Johnson 2004), and between viviparity and thelytoky (Mockford 1971; Koteja 1990). The empirical evidence for a general association between thelytoky and gregarious broods is weak, but both Lively and Johnson's argument, and the argument to be developed in this paper, imply that such an association should be expected.

For each of the three asymmetric genetic systems, different explanations have been suggested for the association with brooding. These are summarized in Table 2. Brooding, or

other prolonged associations of offspring with mothers and/or other maternal kin, leads to the evolution of an asymmetric genetic system for the following reasons. (1) There is conflict between maternally and paternally derived alleles over the rate of resource transfer from mother to offspring (Haig and Westoby 1991; Moore and Haig 1991). (2) Brooding facilitates origins of novel genetic systems by allowing replacement of offspring that die due to developmental problems (Stearns 1987; Lively and Johnson 1994). (3) Association of close kin leads to inbreeding, which leads to selection for distorted sex ratios (Hamilton 1967). (4) When siblings compete, cytoplasmic elements that reduce male survivorship by destroying the paternal genome of males can benefit themselves and their female hosts (Normark 2004).

Of these four explanations, the last two are fairly specific to different varieties of haplodiploidy (arrhenotoky and paternal genome elimination, respectively). The first two have potentially wider explanatory power. But only the first one—Haig's kinship theory of genomic imprinting—points toward a satisfying explanation for maternal/paternal asymmetry *per se*. Gregarious broods often produce predictable conflicts of interest between maternally and paternally derived alleles within the siblings. These conflicts of interest may often result in the evolution of asymmetries in the fates of maternal versus paternal alleles.

PARENT-SPECIFIC GENE EXPRESSION AND GREEDY PATERNALLY IMPRINTED ALLELES

Prolonged association of offspring with their mother creates the potential for conflicts of interest between maternally and paternally derived alleles. Some of these potential con-

TABLE 2. Explanations for the association between gregarious kin and asymmetric genetic systems.

Asymmetric system	Kin association	Taxa	Explanation	Reference
Arrhenotoky (haplodiploidy)	gregarious broods	terrestrial arthropods	Gregarious siblings mate with each other; arrhenotoky permits facultative female-biased sex ratios which are adaptively favored under inbreeding.	Hamilton (1967)
Parent-specific gene expression (genomic imprinting)	viviparity, seeds	mammals, angiosperms	Maternally and paternally derived alleles in embryo can have different optimal levels of expression due to differing kinship with mother.	Haig (2000)
Parent-specific gene expression (genomic imprinting)	eusociality	ants	Maternally and paternally derived alleles in workers can have different optimal levels of expression due to differing kinship with other colony members.	Queller (2003)
Parthenogenesis	brooding	aquatic invertebrates	Brooding allows greater investment in developing than nondeveloping embryos; this is conducive to origins of novel genetic systems in which many eggs may initially fail to develop.	Lively and Johnson (1994)
Paternal genome elimination (haplodiploidy)	gregarious broods	terrestrial arthropods	Gregarious broods are susceptible to invasion by male-killing maternally inherited bacteria; paternal genome elimination was initially a male-killing bacterial phenotype.	Normark (2004)
All of the above	all of the above	all of the above	In maternal kin groups, conflict will occur between maternally and paternally derived genomes. The evolution of asymmetric inheritance (haplodiploidy or thelytoky) can partially resolve these conflicts and hence increase overall fitness of kin groups.	this paper

flicts have been thoroughly explored by Haig (2000, 2002, 2004) in the context of the kinship theory of genomic imprinting. Most discussions of PSGE have focused on mammals, in which there is a long period of direct transfer of nutrients from the mother to the offspring, and on genes affecting the rate of nutrient transfer. Typically, paternally derived alleles favor a higher rate of nutrient transfer, and maternally derived alleles favor a lower rate. This is because, to the paternally derived allele, the mother and any half-siblings are not kin ($r = 0$); whereas, to the maternally derived allele, the mother is “self” ($r = 1$) and every member of the brood is close kin ($r = 0.5$). Thus, for any process that implies a trade-off between maternal fitness and the fitness of an individual offspring (such as the rate of resource transfer from mother to offspring), maternally and paternally derived alleles will tend to have different optima for maximizing inclusive fitness. Because the inclusive fitness of the maternally derived allele always includes the fitness of mother and siblings, whereas that of the paternal allele often does not, a paternally derived allele in an embryo is often selected to be more “greedy,” valuing embryonic fitness relatively highly compared to maternal fitness, whereas the maternally derived allele in the same embryo is selected to be more “abstemious,” giving relatively more weight to maternal fitness (Haig and Wilkins 2000).

Although viviparity of mammals is the canonical case, there are many other circumstances that lead to kinship asymmetries (average maternal relatedness of an individual to its neighbors different from average paternal relatedness of an individual to its neighbors). These asymmetries in turn lead to conflicts of interest between maternally and paternally derived alleles, which may lead to the evolution of PSGE. Although asymmetries occur in both directions, it is more com-

mon for kin groups to have higher maternal than paternal relatedness among their members. Factors that tend to boost maternal relatedness relative to paternal relatedness include: (1) brooding, viviparity, maternal care, or other long-term association of mother with offspring or siblings with each other; (2) a strong sexual dimorphism in dispersal, with high male dispersal and low female dispersal; (3) outcrossing; and (4) multiple matings by females.

The other critical factor for the evolution of PSGE is that members of the kin group compete for resources. This competition may be subtle. Even in the case of mammals, if a female rears and weans one offspring at a time, there is still a kind of competition between siblings: each offspring is essentially competing with future siblings for maternal resources. In mammals, we think of the mother trading off reproductive investment between the current offspring and future offspring, but one could also model such a female mammal as a finite resource to be consumed by a series of sequentially hatching offspring (Haig 1992; Haig and Wilkins 2000). How much of the resource should an individual offspring consume, and how much should it leave for its potential future siblings? Maternally imprinted and paternally imprinted alleles would have different optima for such a trade-off. In many animals with brood sacs, the relevant resource for which they compete may be nothing more than space in the brood sac.

Lineages characterized by all of the above factors will have a strong tendency to evolve PSGE with greedy paternal alleles and relatively abstemious maternal alleles. This theory has been developed most fully for alleles affecting fetal, placental, and maternal physiology in highly viviparous mammals (Haig 2004); the embryo/endosperm/maternal tissue mosaics of angiosperm seeds (Haig and Westoby 1991); and to a lesser

extent (and with much less empirical corroboration) social interactions among insects and primates (Nicholls 2000; Queller 2003).

It may be hard to imagine what a “greedy paternal allele” might code for in a much looser gregarious kin group. As a concrete example, let us consider invertebrates with facultative cannibalism, in which cannibalism occurs in conditions of near starvation. There may be a large number of loci affecting the threshold level of starvation at which cannibalism begins to occur; these may include olfactory receptors involved in food discrimination, any of various loci expressed under physiological stress (e.g., heat shock proteins), and others. With high kinship asymmetries such that neighbors are more closely related maternally than paternally, the optimal threshold level for a switch to cannibalism will differ between maternally and paternally derived alleles, with paternally derived alleles benefiting from cannibalism under a wider variety of circumstances. Consider a gene product historically indicative of physiological stress that might trigger a switch to cannibalism. An allele of such a gene may benefit from being more highly expressed when paternally derived than maternally derived, and the organism containing the allele will also benefit. Once PSGE evolves, with the paternal allele being more highly expressed, there is selection for the allele to have lower expression when maternally derived. The typical outcome in these circumstances is for the maternal copy of the allele to be entirely silenced, and for expression of the gene to be set entirely by the paternally derived allele—Haig’s (1997) “loudest voice prevails” principle. Thus, animals with gregarious broods and high relatedness asymmetries may be at risk of evolving high levels of cannibalism or other traits that compromise the fitness of kin groups, because of the action of greedy paternally-imprinted alleles.

I propose that the tendency to evolve greedy paternally imprinted alleles is present in many groups of organisms, particularly small terrestrial arthropods. Possibly, most lineages with very high kinship asymmetries, which give rise to full expression of such imprinted alleles, tend to suffer demographic collapse and extinction. Possibly also, selection often acts on modifier alleles that decrease the extent of asymmetry or otherwise mitigate the organism-level harmful effects of greedy paternally imprinted alleles. In either case, the evolutionary pathway leading to greedy paternally imprinted alleles might have its greatest importance as a filter or constraint, shaping the extant biota to have relatively low relatedness asymmetries or low capacity to evolve PSGE. I propose that haplodiploidy and thelytoky tend to evolve in response to genetic conflict—conflict which may often be mediated by PSGE; that they serve both to reduce the conflict and to promote maternal interests; and that this may explain their association with brooding and other aspects of their ecological distribution.

CANNIBALISM AND GENETIC SYSTEM DIVERSITY UNDER BARK

Consider the case of bark beetles (Coleoptera: Curculionidae: Scolytinae). They are among the first decomposers of woody plants worldwide. In most species, adults bore galleries into damaged or dead trunks and branches; adult fe-

males lay eggs in the galleries; and larvae consume phloem tissue (Hamilton 1978; Wood 1982; Kirkendall 1993). Even with a phoretic community of fungi and bacteria to decompose it, phloem is a nutritionally marginal resource. There are many reports of cannibalism in bark beetles (Schenk and Benjamin 1969; Beaver 1974; Weber and McPherson 1983; Norris and Chu 1985), and it would appear that individual larvae would typically benefit from cannibalism and typically have the opportunity to commit it. Although there is thus a wide scope for intrabrood genetic conflict in bark beetles, many bark beetles have life cycles that seem to minimize this conflict by minimizing kinship asymmetries: the most common and phylogenetically primitive mating system in Scolytinae is monogamy, in which a single male and single female have a prolonged association with a single gallery and clutch of eggs, and often have broken tarsi that would prevent them from moving to a new gallery system (Kirkendall 1993; Sequeira et al. 2000; Farrell et al. 2001). Also common is harem polygyny, in which males bore and defend gallery systems with a single female in each branch, such that neighboring eggs are full siblings (Kirkendall 1993; Kirkendall et al. 1997; Sequeira et al. 2000). Pseudogamy (apomictic clonal reproduction involving mating followed by complete elimination of the paternal genome from the all-female progeny) is known in bark beetles (Kirkendall 1990; Kirkendall and Stenseth 1990; Løyning 2000), and both systems of haplodiploidy (paternal genome elimination and arrhenotoky) have arisen in association with regular sibling mating (Brun et al. 1995; Normark et al. 1999).

In addition to bark beetles, there are several other groups that primitively feed in rotting wood (typically in a more advanced state of decay than the wood used by bark beetles) and that have given rise to haplodiploid lineages: primitive Diptera (two PGE lineages, Cecidomyiidae and Sciaridae), archostematan beetles (one arrhenotokous lineage, Micromalthidae), and Hymenoptera (one hyperdiverse arrhenotokous lineage) (Normark 2003, 2004). Cannibalism is not well documented in Cecidomyiidae or Sciaridae, but is known in related fly families (Srinivasan and Panicker 1992; Sherratt et al. 1999). Cannibalism is known in micromalthid beetles and in sawflies (primitive Hymenoptera that bore through wood and other plant tissues) and can be severe in both groups. Male micromalthid beetles are obligately cannibalistic, consuming their own mothers (Pollock and Normark 2002). And in some sawflies, larvae will consume any siblings they encounter (Morrill et al. 2000).

Hamilton (1978) wrote at length about the diversity of genetic systems in insects that live under bark. He supposed that the driver of this diversity was the potential for inbreeding and consequent benefit to being able to control the sex ratio. Here I suggest that a more important factor may have been the potential for facultative cannibalism, in asymmetric kin groups, driving genetic conflict mediated by PSGE. This conflict led in turn to a diversity of innovations to obviate the conflict, including suppressed expression of paternal alleles (PGE), exclusion of males from parentage of sons (arrhenotoky), deletion of males from the life cycle (thelytoky), and asymmetry-minimizing life histories (including Hamilton’s inbreeding itself).

The above scenario—broods under bark with the possi-

bility of cannibalism—characterizes six of the 10 origins of haplodiploidy in insects (the three beetle origins; the two fly origins; and Hymenoptera; see Normark 2003, 2004). It is also plausible (Saito 1990; Halmai 1994; Schausberger and Croft 2000) in the case of the eight origins in mites (Norton et al. 1993), though little is known about these. Cannibalism seems much less plausible in the case of the origins of haplodiploidy in rotifers, and nematodes, and phytophagous paraneopteran insects (three Hemiptera, one Thysanoptera), but other types of agonistic interactions between siblings are possible. In the particularly inert-seeming scale insects (a hemipteran group with two origins of haplodiploidy), greedy behavioral interactions between siblings may include burrowing—settling under another individual to use the other individual as an effective shield against parasitoids (Matsumoto et al. 2002)—and forming galls “upstream” with respect to the flow of phloem sap (L. G. Cook, pers. comm.).

ALTERNATIVE CAUSAL MODELS: NUCLEAR, CYTOPLASMIC, OR SYNERGISTIC

It is widely recognized that gregarious kin groups (construed broadly to include gravid individual females) can be subject to various sorts of genetic conflict, including conflict between maternally and paternally derived alleles (Mills and Moore 2004; Isles and Holland 2005). It has also been recognized, less widely, that evolutionary lineages with gregarious kin are prone to originate novel, asymmetric genetic systems (Hamilton 1967, 1978; Lively and Johnson 1994; Normark 2003, 2004). If we assume for the present that there is some causal connection between these patterns, there are still a number of different possible chains of causation that might connect them. Here I will briefly consider three possible approaches to modeling how genetic conflicts might give rise to asymmetric genetic systems.

Nuclear Genes Only

The kinship theory of genomic imprinting describes conflict and interaction between nuclear genes without consideration of what cytoplasmic genes may be doing. In addition, it is possible to construct a model of the origin of the other asymmetric genetic systems (haplodiploidy and thelytoky) from PSGE that considers only nuclear genes. Consider a metapopulation of demes of a facultatively cannibalistic insect in which a cue that triggers cannibalism is produced by a paternally imprinted allele (with a silent maternal copy) or a cue that inhibits cannibalism is produced by a maternally imprinted allele (with a silent paternal copy). A new mutation allowing the suppression or elimination of the paternal genome (through haplodiploidy or thelytoky) would have a non-cannibalistic phenotype, and such a phenotype could plausibly result in a higher demic population growth rate and the production of more emigrants, leading to the spread of the new genetic system.

It is probably also possible to construct such a model without invoking a metapopulation; I use a metapopulation for this verbal model only because demic fitness is a convenient proxy for overall organismal fitness irrespective of maternal versus paternal fitness. It might seem redundant to invoke such a model for the spread of haplodiploidy or thelytoky,

because both of these genetic systems are thought to have substantial intrinsic transmission-genetic advantages that cause them to spread rapidly and deterministically once they originate. There is the famous twofold cost of sex (Williams 1975), which could as easily be called the twofold advantage of thelytoky; and there is the ‘maternal transmission advantage’ of haplodiploidy (Brown 1964; Smith 2000). These advantages are no doubt real, but because they are logically inevitable properties of thelytoky and haplodiploidy they cannot by themselves explain anything about those systems’ ecological correlates. The most influential models of haplodiploidy are all based ultimately on Brown’s (1963, 1964) maternal transmission hypothesis, and they all implicitly view haplodiploidy as serving maternal interests in a maternal-paternal conflict over transmission genetics (Hartl and Brown 1970; Bull 1979; Haig 1993b; Herrick and Seger 1999). All of these models treat the maternal-paternal conflict as a zero-sum game. But in maternal kin groups with greedy paternally imprinted alleles, maternal-paternal conflict is not a zero-sum game: paternal strategies tend to depress the overall fitness of the kin group whereas maternal strategies enhance group fitness. That may be why asymmetric genetic systems favoring maternal alleles often succeed when they arise in such groups.

Cytoplasmic Genes

A serious problem for models of the origins of haplodiploidy is the expectation that haploid individuals will initially have extremely low fitness (e.g., Smith 2000). This problem is somewhat reduced when broods are gregarious and there is competition between siblings, but remains a serious problem for any model invoking only nuclear genes. A possible solution to this problem is to invoke cytoplasmic elements as the agents that initially cause haploidy in males (Normark 2004), since these can often benefit from low male fitness when siblings compete for resources (Hurst 1991; Stevens et al. 2001). Cytoplasmic elements that eliminate the paternal genome can initially benefit themselves (by male-killing) and eventually benefit their hosts (by maternal transmission advantage if nonzero haploid male survivorship evolves). Population-genetic models indicate that such a male-haploidizing cytoplasmic element could in principle invade a eumendelian population (Engelstädter and Hurst 2006; F. Úbeda and B. B. Normark, unpubl. ms.).

Synergy between Nuclear and Cytoplasmic Genes

The nuclear (maternal-paternal conflict) and cytoplasmic (cytonuclear conflict) models sketched above are separate hypotheses for the origin of haplodiploidy that may stand or fall independently of each other. But when siblings compete in asymmetric kin groups, we expect that both maternal-paternal and cytonuclear conflict will occur. In addition, the two hypotheses may be strengthened by being combined. In a combined model, haplodiploidy is seen as a kind of tactical alliance between maternally imprinted and maternally transmitted elements, which precipitates when kin-group fitness is compromised by greedy paternally imprinted alleles.

As noted above, the presence of greedy paternally imprinted alleles turns maternal-paternal conflict into a non-zero-

sum game in which the kin group (and demic) optimum is closer to the maternal optimum than the paternal optimum. The same non-zero-sum dynamic affects the cytonuclear conflict. In previous models of male-haplodiploidizing endosymbionts (Normark 2004; Engelstädter and Hurst 2006), the endosymbionts were modeled as having no stake in the maternal-paternal conflict but as benefiting from paternal genome elimination only because it initially compromised male survivorship. But in the presence of greedy paternally imprinted alleles, suppression of the paternal genome can boost the fitness of kin groups and thereby directly benefit maternally transmitted endosymbionts even if there is perfect haploid-male survivorship.

Evolution of Sex-Determining Mechanisms

The above discussion of modeling approaches is of course incomplete. Missing is consideration of a question that has preoccupied many previous students of the origins of haplodiploidy (Hartl and Brown 1970; Bull 1983; Haig 1993a,b): By what genetic mechanism do haploids develop as males? Although answers to this question will clearly be necessary for a complete understanding of origins of haplodiploidy, emphasis on this question has historically in my view been a red herring, diverting attention from the clear ecological and life-history correlates of haplodiploidy and motivating a frustrated expectation of cytogenetic correlates. Haplodiploidy generally arises from male-heterogametic systems, but beyond that there are few commonalities. For instance, it arises from XY-male systems about as often as XO-male systems (Normark 2004). Differences in the cytogenetic details of different haplodiploid systems have inspired—and probably require—different ad hoc cytogenetic models for each case (Bull 1983; Haig 1993a,b). I have no quarrel with any of these specific models. My goal here is not to suggest an alternative but to explain something that all the cytogenetic models neglect: why these cytogenetic transformations occur in some ecological situations and not others.

EGG CHORION VERSUS PLACENTA

I argue that PSGE has a destabilizing effect on the genetic systems of arthropods, leading to the evolution of other asymmetric genetic systems (haplodiploidy and thelytoky). Yet, PSGE is widely seen as having had precisely the opposite effect in mammals, stabilizing the genetic system against the emergence of any system of uniparental inheritance (Kono et al. 2004). Why such divergent pathways in these two taxa? The difference between taxa with stably virulent PSGE (like mammals) and taxa prone to reversible PSGE, haplodiploidy, and thelytoky (like arthropods) may hinge on the physiology of the mother-embryo interface. Arthropods and most other animals have an intact egg chorion around each embryo which both prevents the release of gene products from paternally derived alleles and prevents the youngest embryos from biochemically interacting with kin. The egg chorion thus prevents loci expressed very early in ontogeny from evolving PSGE. In the lineage leading to mammals, the egg chorion was breached, allowing paternal gene products access to maternal physiology. This led to the evolution of PSGE in loci expressed in very early ontogeny, which prevented

mammals from following the “normal” pathways of resistance to greedy paternal alleles, such as haplodiploidy and thelytoky. In lineages with an egg chorion, interactions with kin will begin much later in ontogeny (after hatching) and will be mediated primarily by behavior rather than biochemistry. Thus, loci essential to early embryogenesis are unlikely to be imprinted, and disruption of imprinting is unlikely to disrupt development. Therefore, although PSGE may prevent the origins of thelytokous mammals, it would not be expected to have any such effect in arthropods.

A potentially instructive case is seen in the scale insect family Stictococcidae, which has reverted from PGE to an apparently eumendelian system with diploid males. Stictococcid males lack endosymbionts, which I previously cited to argue for the involvement of endosymbionts in the maintenance of PGE (Normark 2004). Due to the lack of endosymbionts, males cannot feed, and they receive all of their nutrition from their mother via a placenta (Buchner 1965). Thus, an alternative explanation of stictococcid male diploidy is that stictococcids have evolved mammalian-style placenta-mediated PSGE. Several species of stictococcids are pests of African agriculture (Richard 1976; Ngeve 2003; Lema 2004) and this is a promising system for the study of the interaction of endosymbionts and genomic imprinting.

THE SPECIAL CASE OF THELYTOKY

Variable Benefits of Sex and the Distribution of Thelytoky

The question of the adaptive significance of sexuality has been characterized as the queen of problems in evolutionary biology (Bell 1982). Thelytokous lineages seem to be the perfect control groups—very much like their close sexual relatives, but lacking sex—and they have been scrutinized for clues to the benefits of sexuality (Glesener and Tilman 1978; Bell 1982). In particular, the ecological distribution of thelytokous lineages has been interpreted as indicating troughs in the ecological distribution in the benefits of sex: for instance, if sex is an adaptation to resist parasites, then we expect thelytokous lineages where parasites are uncommon or avirulent (Bell 1982). Implicit in this research program is the assumption that the costs of sex are relatively constant.

Variable Costs of Sex and the Distribution of Thelytoky

But there is a second possible approach: one could assume that the benefit of sex is relatively constant and independent of ecology, and interpret the ecological distribution of thelytokous lineages as indicating peaks in the ecological distribution of the costs of sex. This approach to interpreting the distribution of thelytoky is sometimes referred to as invoking theories of parthenogenesis, as opposed to theories of sex. An example is the reproductive assurance hypothesis: thelytoky occurs where the costs of finding a mate are unusually high, which in effect elevates the cost of sex (Cuellar 1994).

Here, I am suggesting that thelytoky is associated with brooding because brooding elevates the cost of sex by creating wide scope for genetic conflict. In particular, the kinship asymmetries in brood chambers are conducive to the evo-

lution of greedy paternally imprinted alleles that can depress average offspring fitness. But surely (one might argue) thelytoky is not a long-term solution to the problem, since thelytokous lineages typically do not persist. This is true, and one expects under this paradigm that the close sexual relatives of thelytokous lineages should exhibit unusually high levels of genetic conflict, and/or exhibit adaptations for conflict reduction other than thelytoky. Thus obligate thelytoky is a symptom of genetic conflict rather than a solution to it. Cyclic or facultative thelytoky, however, may be an adaptation to reduce the scope of sexual conflict by reducing the role of sex in the life cycle; similarly, apparent obligate thelytoky may mask systems of “covert sex” (Hurst et al. 1992). For instance, although we typically assume that parthenogenesis-inducing bacteria are parasites that have taken control of their hosts’ reproduction, an alternative hypothesis is that “curable parthenogenesis” may often be a system of rare sex that benefits both host and endosymbiont.

Caveats

Thelytoky typically implies the complete loss of genetic recombination and exchange. This has many dramatic consequences besides the elimination of males and of any sexual conflict. I am not suggesting that escape from sexual conflict by itself will yield a complete understanding of the distribution of thelytoky; only that it may be an important factor affecting this distribution and one that has hitherto been overlooked.

GREGARIOUS KIN, HAPLODIPLOIDY, AND EUSOCIALITY

Hamilton (1964a,b) proposed an influential theoretical explanation why eusociality tends to arise in haplodiploid lineages. He also proposed an influential hypothesis for the adaptive significance of haplodiploidy—that it is an adaptation for sex ratio control under inbreeding (Hamilton 1967)—a hypothesis which bore no particular connection to haplodiploidy’s role in preadapting lineages to eusociality. Likewise other adaptive explanations of haplodiploidy—maternal transmission advantage (Brown 1964; Hartl and Brown 1970), deleterious mutation clearance (Goldstein 1994; Richerd et al. 1994), and endosymbiont-based hypotheses (Hamilton 1993; Normark 2004)—have implicitly treated the sisterly solidarity-enforcing properties of haplodiploidy as an incidental by-product unrelated to haplodiploidy’s original adaptive significance.

The perspective on haplodiploidy advocated here is the first that sees haplodiploidy as having been, from its earliest appearance, an adaptation for the reduction of conflict among siblings. Eusociality is of course rare and recent compared to haplodiploidy; typically the altruism of haplodiploid sibs must be expressed much more subtly, for instance as the mere absence of cannibalism. Haplodiploidy reduces conflict between maternal sibs at two levels: first, starting in the first haplodiploid generation, potentially greedy paternal alleles are excluded from sons and sons harbor only maternal alleles; second, starting in the second haplodiploid generation (females that mate with haploid males), clonal sperm boosts relatedness among full sisters. By enhancing paternal relatedness among siblings this may also act to reduce the benefit

to be gained by greedy paternally imprinted alleles. Those eusocial lineages that are not haplodiploid are those whose ancestors took an alternate route to reduction of relatedness asymmetries in gregarious broods: monogamy (Kirkendall et al. 1997).

Haplodiploidy reduces conflict between sisters (Hamilton 1964a,b), but may actually enhance some kinds of conflict between sisters and brothers (Seger and Stubblefield 2002; Wilson 2005). In a nuclear-genes-only model of haplodiploidy, this may be seen as an unfortunate byproduct of the necessity of maintaining sexuality through one of the sexes: if paternal genes were excluded from females as well as males, the result would be thelytoky. But in a cytonuclear model, in which maternally transmitted endosymbionts play a role in the origin of haplodiploidy, this feature can be interpreted as giving a direct benefit to the maternally transmitted elements. In the context of greedy paternally imprinted alleles, haplodiploidy benefits maternally transmitted elements by restricting these greedy elements to females and eliminating them from males. Even without invoking greedy paternal alleles, haplodiploidy restructures relationships among siblings, boosting relatedness of sisters to each other and reducing their relatedness to brothers. Both of these features will tend to make any enhanced altruism in a haplodiploid brood tend to flow away from brothers and toward sisters, benefiting cytoplasmic elements.

SUMMARY: TOWARD A KINSHIP THEORY OF ASYMMETRIC GENETIC SYSTEMS

Three genetic systems with asymmetric gene expression or transmission are PSGE (asymmetric expression of some genes in both sexes), haplodiploidy (asymmetric expression and transmission of all genes in one sex), and thelytoky (deletion of one sex). All three of these asymmetric genetic systems arise in lineages in which there is extended contact between maternal kin. Possibly, competition among maternal kin may create genetic conflicts that can drive the evolution of asymmetric genetic systems. This hypothesis has been developed into a sophisticated theory—the kinship theory of genomic imprinting—for the case of PSGE (Haig 2000, 2004; Mills and Moore 2004). It is possible to invoke the same kinds of conflicts (resource allocation conflicts between maternally and paternally imprinted alleles in asymmetric kin) to explain the origins of haplodiploidy and some features of the distribution of origins of thelytoky. Symmetric inheritance is stable when kinship between an individual and its neighbors is symmetric (or negligible). But when there is substantial kinship asymmetry between an individual and its neighbors, with (typically) greater maternal than paternal relatedness, then conflict will occur between maternally and paternally derived genomes. The evolution of asymmetric inheritance (haplodiploidy or thelytoky) can partially resolve these conflicts and hence increase overall average organismal fitness across kin groups.

There is at least one other hypothesis that is at least as good at explaining the ecological correlates of haplodiploidy (Normark 2004), and at least one that is as good at explaining the association of brooding with parthenogenesis (Lively and Johnson 1994). There is no direct evidence in favor of the

idea that PSGE creates the conflicts that drive the evolution of the other asymmetric genetic systems. But the hypothesis that PSGE is the original asymmetry and that the other asymmetric systems evolve in response to it (a general kinship theory of asymmetric genetic systems) would have some appealing properties. It would allow a more unified understanding of genomic imprinting, encompassing both the PSGE seen in mammals and angiosperms and the PGE seen in insects. It would also allow a more unified understanding of the origins of haplodiploidy and the significance of haplodiploidy for social evolution. Such a theory would also be closely parallel to some hypotheses of the origin of the original asymmetry in cytoplasmic inheritance between the sexes (Randerson and Hurst 1999), and conceivably could lead to a more general understanding of the role of genetic conflict in the origin of genetic asymmetry, both cytoplasmic asymmetry and nuclear asymmetry.

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