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The Evolution of Hermaphroditism by an Infectious Male-Derived Cell Lineage: An Inclusive-Fitness Analysis

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abstract: There has been much recent interest in the role for genetic conflicts to drive the evolution of genetic systems. Here we consider the evolution of hermaphroditism in the scale insect tribe Iceryini and the suggestion that this has been driven by conflict between a female and an infectious male tissue derived from her father. We perform an inclusive-fitness analysis to show that, owing to genetic relatedness between father and daughter, there is scope for collaboration as well as conflict over the establishment of the infectious tissue. We also consider the evolutionary interests of a maternally inherited bacterial symbiont that has been implicated in mediating the tissue's establishment. More generally, our analysis reveals that genetic conflicts can drive the evolution of hermaphroditism.

Keywords: class structure, genetic conflict, *Icerya*, kin selection, relatedness, reproductive value.

Introduction

There exists a wide diversity of reproductive strategies among multicellular organisms, and understanding the evolutionary significance of this variation remains an important challenge for evolutionary biologists (Policansky 1982; Heller 1993; Barrett 2002; Normark 2003; de Jong and Klinkhamer 2005; Avise and Mank 2009). The first and most fundamental difference in the way that organisms reproduce is the distinction between sexual and asexual reproduction (Cuellar 1977; Judson and Normark 1996; Vrijenhoek 1998; Otto 2009). A second important difference among sexual organisms is between those species with separate sexes (gonochorism) and those in which the same individual produces both male and female gametes (hermaphroditism; Ghiselin 1969; Charnov et al. 1976). Hermaphroditism is found in a large number of taxa across a wide taxonomic range (Ghiselin 1969; Charnov et al. 1976; Barrett 2002; Jarne and Auld 2006). Although hermaphroditism is common in some taxonomic groups, it is rare or absent from others. For example, while only 5%–6% of all animal species are estimated to be hermaphroditic, the estimate rises to ~30% if insects are excluded (Schärer 2009). The reasons for the rarity of hermaphroditism among insects, a species-rich group characterized by its wide diversity of genetic systems, remain obscure.

The traditional paradigm for understanding the evolution of genetic systems has been to seek adaptive explanations at the level of the individual organism (Darlington 1958; Bull 1983). Thus, a separation of the sexes is expected when there are efficiency benefits for individuals specializing in a single reproductive mode (Charnov et al. 1976; Charnov 1982), sequential hermaphroditism is expected when one sex benefits from a size difference more than the other (Ghiselin 1969), and simultaneous hermaphroditism is expected to evolve when finding a partner or investing in a specific sexual function is expensive (Charnov et al. 1976; Puurtinen and Kaitala 2002). Such explanations have focused on ecological and demographic factors. For example, both low population density and impaired mobility have been suggested to drive the evolution of simultaneous hermaphroditism, owing to scarcity of mating opportunities (Ghiselin 1969; Puurtinen and Kaitala 2002; Eppley and Jesson 2008).

In contrast to this traditional approach, recent years have seen growing interest in the role for conflicts between genes to mediate the evolution of novel genetic, reproductive, and sex-determination systems (Haig 1993; Hurst 1995; Hurst et al. 1996; Werren and Beukeboom 1998; Hurst and Werren 2001; Normark 2004; Burt and Trivers 2006; Uller et al. 2007; Van Doorn and Kirkpatrick 2007). One source of conflict that has been especially well documented is that between nuclear genes and cytoplasmic genes (Cosmides and Tooby 1981; Hurst 1992; Werren and Beukeboom 1998; Charlat et al. 2003; Wernegreen 2004;

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Burt and Trivers 2006). Many insects harbor intracellular bacteria that are transmitted only via daughters (Buchner 1965; Moran and Telang 1998; Moran and Baumann 2000; Moran 2002) and hence have an interest in biasing their host's sex allocation toward daughters (Cosmides and Tooby 1981; Stouthamer et al. 1990; Werren et al. 2008). Another source of conflict is that between females and males in species with sex-asymmetric transmission. In haplodiploid species—where females develop from fertilized (i.e., diploid) eggs and males develop from unfertilized (i.e., haploid) eggs—males pass on their genes only through daughters, whereas females can achieve fitness through both offspring sexes, leading to a potential for conflict over sex allocation (Normark 2009; Shuker et al. 2009). Females typically control sex allocation by deciding the fraction of eggs to be fertilized. However, any adaptation on the part of their mate to increase this fraction would be strongly favored.

Such conflict over fertilization rate has been suggested to have driven the evolution of an unusual form of hermaphroditism found in three species of the scale insect tribe Iceryini (Hemiptera: Coccoidea: Monophlebidae; Nur 1980; Normark 2003)—the only known instance of hermaphroditism in insects (Hughes-Schrader 1925, 1930; Royer 1975). Scale insects are small plant-feeding insects (Gullan and Kosztarab 1997; Ross and Shuker 2009) that exhibit a remarkable variety of genetic systems—a diversity that has been suggested to reflect the operation of extensive genetic conflicts (Ross et al. 2010). Hermaphroditism in scale insects has evolved in an otherwise haplodiploid clade (Hughes-Schrader and Monahan 1966; Nur 1980; Ross et al. 2010), and molecular phylogeny suggests that it has evolved independently in each of the three species for which it has been described (Unruh and Gullan 2008).

In the hermaphroditic species of *Icerya*, males are rare, and females—who contain an ovitestis capable of producing sperm and oocytes—can internally self-fertilize and hence produce offspring in the absence of a mating partner (Hughes-Schrader 1925). What makes this system so unusual is that the sperm-producing gonads of the ovitestis are haploid (Hughes-Schrader 1963) and, in at least one species (*Icerya purchasi*), this tissue appears to derive from excess sperm that penetrated the oocyst when the female was conceived (Royer 1975). So although *I. purchasi* resembles other hermaphroditic taxa in that individuals can produce both male and female gametes, the mechanism by which male gametes are produced differs markedly. Normark (2009) has suggested that this peculiar reproductive mode has been driven by conflict between males and females over genetic transmission: by infecting his daughters with cells that form male gametes inside their bodies, a father is able to fertilize the eggs of his daughters as well as those of their mother.

Here we perform an inclusive-fitness analysis of the evolutionary origin and subsequent spread of infectious male tissue. While Normark (2009) has suggested that the infectious tissue is parasitic on the female and will spread, owing to the transmission advantage that it provides for the male, we consider the possibility for collaboration as well as conflict between the female and her infectious tissue. Some overlap of interests is possible, owing to genetic relatedness between father and daughter, with the former sometimes showing restraint and the latter sometimes showing a shared interest in allowing the infectious tissue to establish. In addition, we consider the interests of a maternally inherited bacterial symbiont that has also been implicated in facilitating the establishment of the infectious tissue (Royer 1975; Hurst 1993; Ross et al. 2010). More generally, our analysis lends support to the idea that genetic conflicts have driven the evolution of this unusual form of hermaphroditism.

Model and Analysis

We outline below the basic model on which our analysis rests, and we describe the inclusive-fitness approach that we use to determine the evolutionary dynamics of natural selection. Thereafter, we determine how females should adjust their sex allocation when infectious tissue is present in the population, and we examine the scope for conflict among the female, her infectious tissue, and her maternally transmitted symbionts over the decision as to whether the infectious tissue should be established within her body and permitted to fertilize her eggs.

Basic Model

We build on the familiar model of haplodiploidy, in which the family unit is made up of an adult female (F), an adult male (M), a juvenile daughter, and a juvenile son. Females are diploid, with one maternal genome and one paternal genome, and males are haploid, with one maternal genome. We extend this model by additionally assigning every female a haploid infectious tissue (T) that derives from her father, and we allow this tissue the possibility of fathering the female's daughters (and hence also their infectious tissues). We thus discriminate five classes of juvenile individual: α sons, regular males derived from unfertilized eggs in the usual way; β daughters, regular females fathered by regular males in the usual way; γ daughters, females that are fathered by their mother's infectious tissues; δ sons, infectious tissues that are fathered by regular males and incorporated into the bodies of β daughters; and ϵ sons, infectious tissues fathered by infectious tissues and incorporated into the bodies of γ daughters. For simplicity, we assume that the adult females

are unrelated to regular males with which they mate. An illustration of the model is given in figure 1.

The behavior of an adult female and her infectious tissue affects the allocation of reproductive resources to each of her five types of offspring. With probability $1 - x$, the infectious tissue fails to establish in the focal female's body, and in this event, the female fertilizes a proportion y of her eggs by using sperm derived from a regular male, and a proportion $1 - y$ of her eggs remain unfertilized. With probability x, the infectious tissue successfully establishes, incurring a relative fecundity cost k for the female, and in this event, the infectious tissue fertilizes all of the female's eggs. Some fecundity cost should arise as a consequence of part of the female's normal reproductive tissue being replaced by infectious tissue. Hence, if we denote the number of eggs produced by an uninfected female by n, the expected numbers of offspring of each class produced by the focal female are $n_\alpha = n(1 - x)(1 - y)$ α sons, $n_\beta = n(1 - x)y$ β daughters, $n_\gamma = nx(1 - k)$ γ daughters, $n_\delta = n(1 - x)y$ δ sons, and $n_\varepsilon = nx(1 - k)$ ε sons (table 1). Thus, the expected numbers of male, female, and tissue offspring produced by the focal female are $n_{\mathrm{m}} = n_\alpha = n(1 - x)(1 - y)$, $n_{\mathrm{f}} = n_\beta + n_\gamma = n[x(1 - k) + (1 - x)y]$, and $n_{\mathrm{t}} = n_\delta + n_\varepsilon = n[x(1 - k) + (1 - x)y]$, respectively.

We assume an infinite population of such families, and we denote population averages (e.g., of x) with an overbar (e.g., $\bar{x}$). We also denote the population sex ratio (proportion of regular individuals who are male) by $z = \bar{n}_{\mathrm{m}}/(\bar{n}_{\mathrm{m}} + \bar{n}_{\mathrm{f}}) = (1 - \bar{x})(1 - \bar{y})/(1 - \bar{x}k)$ and the proportion of females that are of type γ by $\phi = \bar{n}_\gamma/\bar{n}_{\mathrm{f}} = \bar{x}(1 - k)/[\bar{x}(1 - k) + (1 - \bar{x})\bar{y}]$, on the assumption of vanishing variation in x and y across the population. We assume that female fertilization strategy $\bar{y}$, being a simple quantitative trait under the sole control of the female, evolves relatively quickly. We assume that the probability of tissue establishment $\bar{x}$, being a complex trait requiring various innovations and involving adaptation of multiple parties, evolves relatively slowly.

Inclusive Fitness

A focal actor is expected to value her social partners according to how well they transmit copies of her genes to future generations (Hamilton 1964; Frank 1998). This is the product of two quantities: the social partners' ability to transmit copies of their own genes to future generations (reproductive value, v; Fisher 1930; Frank 1998) and the extent to which genes transmitted by the social partners are the same as those carried by the actor (relatedness, r; Hamilton 1964; Frank 1998). We assume that all genetic similarity owes to shared genealogy, such that relatedness can be computed from coefficients of consanguinity (e.g., we exclude greenbeard effects; Gardner and West 2010).

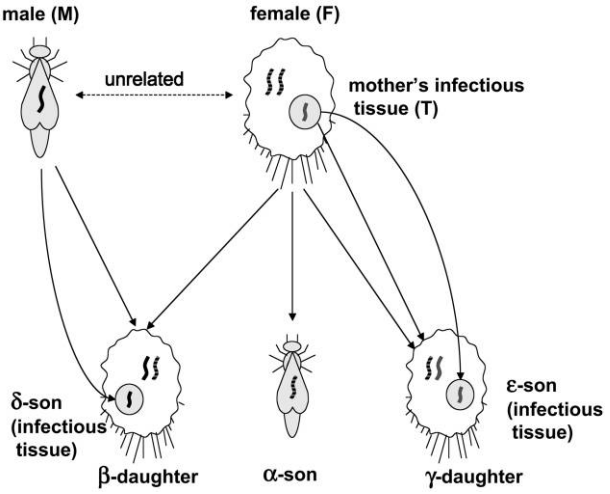

Figure 1: Family unit. Our model is based on standard haplodiploid inheritance, with only the female (F) contributing a genome to her haploid son (α son) and with the female and male (M) each contributing a genome to their diploid daughter (β daughter). In addition, the male contributes a genome to infectious tissue that grows in his daughters (δ sons), and the mother's infectious tissue (T) can fertilize her eggs to produce daughters (γ daughters) and also further infectious tissues (ϵ sons).

Thus, in the context of this model, the inclusive fitness H_A of an actor A is defined as

$$H_A = n_\alpha v_{\mathrm{m}} r_{A\alpha} + n_\beta v_{\mathrm{f}} r_{A\beta} + n_\gamma v_{\mathrm{f}} r_{A\gamma} + n_\delta v_{\mathrm{t}} r_{A\delta} + n_\varepsilon v_{\mathrm{t}} r_{A\varepsilon}, \quad (1)$$

where v_{m}, v_{p}, and v_{t} are the reproductive values of a juvenile male, a juvenile female, and an infectious tissue residing in a juvenile female, respectively (expressions for these coefficients are provided in table 1; for derivation, see appendix), and r_{AX} is the genetic relatedness of a type X offspring to the actor A from the perspective of the actor (expressions for these coefficients are provided in table 1; for derivation, see appendix). The condition for natural selection to favor an increase in any character is that this increases the inclusive fitness of the actor (Hamilton 1964).

Female Sex Allocation

We first consider the fertilization strategy of the female. In the event that the infectious tissue does not establish itself, the female fertilizes a proportion y of her eggs by using sperm derived from a regular male. The condition for natural selection to favor an increase in the value of this character is that this increases her inclusive fitness. Assuming vanishing genetic variation, this condition is $\partial H_{\mathrm{F}}/\partial y > 0$, that is,

Table 1: Offspring type, number, reproductive value, and relatedness to mother and infectious tissue

Type (X)	Number (n_X)	Reproductive value (v_X)	Relatedness to mother (r_{FX})	Relatedness to infectious tissue (r_{TX})
α	$n(1-x)(1-y)$	$(1-\phi)/\bar{n}_m$	1	$1/(2-\phi)$
β	$n(1-x)y$	$[2(1-\phi)]/\bar{n}_f$	1/2	$1/[2(2-\phi)]$
γ	$nx(1-k)$	$[2(1-\phi)]/\bar{n}_f$	1	$(3-\phi)/[2(2-\phi)]$
δ	$n(1-x)y$	$\phi/\bar{n}_t$	0	0
ϵ	$nx(1-k)$	$\phi/\bar{n}_t$	1	1

Note: The proportion of females who are γ daughters is $\phi = \bar{n}_\gamma/(\bar{n}_\beta + \bar{n}_\gamma)$, and the average number of offspring of each sex is $\bar{n}_m = \bar{n}_\alpha$ (males), $\bar{n}_f = \bar{n}_\beta + \bar{n}_\gamma$ (females), and $\bar{n}_t = \bar{n}_\delta + \bar{n}_\varepsilon$ (infectious tissues).

$$\frac{\partial n_\alpha}{\partial y} v_m r_{F\alpha} + \frac{\partial n_\beta}{\partial y} v_f r_{F\beta} + \frac{\partial n_\gamma}{\partial y} v_f r_{F\gamma} + \frac{\partial n_\delta}{\partial y} v_t r_{F\delta} + \frac{\partial n_\varepsilon}{\partial y} v_t r_{F\varepsilon} > 0, \quad (2)$$

where all derivatives are evaluated in a monomorphic population ($x = \bar{x}, y = \bar{y}$). Using the information provided in table 1, we can rewrite condition (2) as $\bar{y} < [1 - \bar{x}(2-k)]/2(1-\bar{x})$. Hence, the population is expected to converge on the strategy value y^*, given by

$$y^* = \begin{cases} \dfrac{1-\bar{x}(2-k)}{2(1-\bar{x})} & \text{if } \bar{x} < \dfrac{1}{2-k} \\ 0 & \text{if } \bar{x} \geq \dfrac{1}{2-k} \end{cases}. \quad (3)$$

Thus, the female fertilizes some or none of her eggs with sperm derived from a regular male when her infectious tissue does not establish ($y^* \geq 0$; this is $y^* = 1/2$ when $\bar{x} = 0$; fig. 2, *top*), and, by assumption, all of the female's eggs are fertilized when her infectious tissue does establish. As a consequence, the population sex ratio is given by $z = 1/2$ if $\bar{x} < 1/(2-k)$ and by $z = (1-\bar{x})/(1-\bar{x}k)$ if $\bar{x} \geq 1/(2-k)$, which decreases to $z \to 0$ as $\bar{x} \to 1$ (fig. 2, *bottom*).

Tissue Establishment

We now examine the evolution of the probability of tissue establishment, x. We begin by considering the interests of the female by assigning her full control of the probability of establishment and determining when she is favored to increase or decrease this quantity. The condition for natural selection to favor an increase in the probability of tissue establishment is that this increases her inclusive fitness. If we assume vanishing genetic variation, this condition is $\partial H_F/\partial_x > 0$, that is,

$$\frac{\partial n_\alpha}{\partial x} v_m r_{F\alpha} + \frac{\partial n_\beta}{\partial x} v_f r_{F\beta} + \frac{\partial n_\gamma}{\partial x} v_f r_{F\gamma} + \frac{\partial n_\delta}{\partial x} v_t r_{F\delta} + \frac{\partial n_\varepsilon}{\partial x} v_t r_{F\varepsilon} > 0, \quad (4)$$

where all derivatives are evaluated in a monomorphic population ($x = \bar{x}, y = \bar{y} = y^*$). If we use the information provided in table 1 and assume $\bar{x} < 1/(2-k)$ (and hence $y^* = [1 - \bar{x}(2-k)]/2(1-\bar{x})$), then condition (4) can be rewritten as $k < 1/(2-\bar{x})$. If instead $\bar{x} \geq 1/(2-k)$ (and hence $y^* = 0$), then condition (4) is always satisfied. Hence, when tissue establishment is relatively uncommon ($\bar{x} < 1/(2-k)$), the female is favored to promote the establishment of her infectious tissue when the fecundity cost of establishment is low ($k < 1/(2-\bar{x})$) and is favored to suppress the establishment of her infectious tissue when the fecundity cost is high ($k > 1/(2-\bar{x})$). In the special case of vanishingly rare establishment of tissues ($\bar{x} \to 0$), the maximum cost the female will endure without being favored to suppress tissue establishment is the loss of half of her fecundity ($k = 1/2$), and as tissue establishment becomes more common (higher $\bar{x}$), the female is favored to promote establishment for even higher fecundity costs (fig. 3).

Next, we consider the interests of the infectious tissue by assigning it full control of the probability of its own establishment and determining when it is favored to promote or suppress its own establishment. Natural selection favors an increase in the probability of establishment when $\partial H_T/\partial x > 0$, that is,

$$\frac{\partial n_\alpha}{\partial x} v_m r_{T\alpha} + \frac{\partial n_\beta}{\partial x} v_f r_{T\beta} + \frac{\partial n_\gamma}{\partial x} v_f r_{T\gamma} + \frac{\partial n_\delta}{\partial x} v_t r_{T\delta} + \frac{\partial n_\varepsilon}{\partial x} v_t r_{T\varepsilon} > 0, \quad (5)$$

where all derivatives are evaluated in a monomorphic population ($x = \bar{x}, y = \bar{y} = y^*$). If we use the information

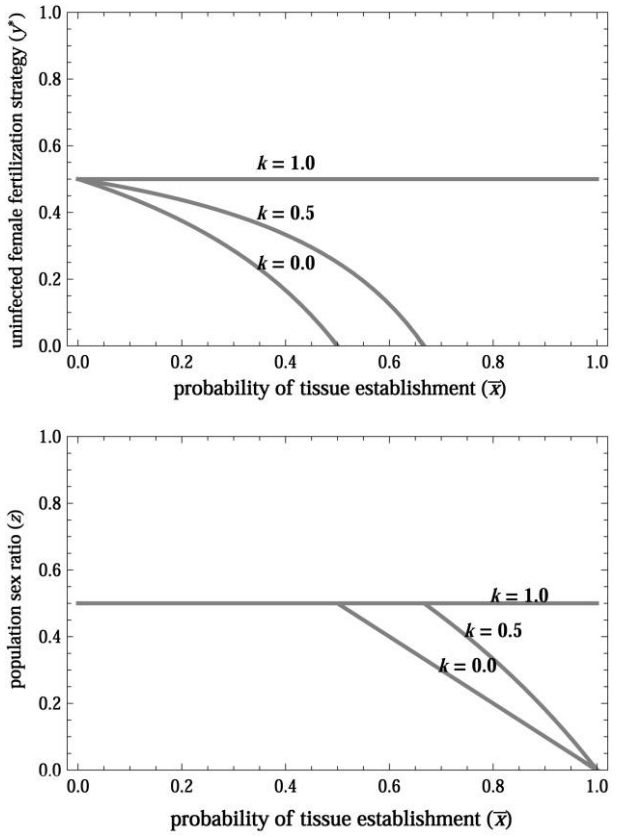

Figure 2: Sex allocation. *Top*, uninfected females are favored to fertilize a proportion of their eggs ($y^* = [1 - \bar{x}(2 - k)]/2(1 - \bar{x})$) with sperm from regular males, which decreases as the probability of tissue establishment ($\bar{x}$) increases and increases as the cost of tissue establishment (k) increases. *Bottom*, sex ratio ($z = \min[1/2, (1 - \bar{x})/(1 - \bar{x}k)]$; proportion of regular individuals who are male) remains fixed at one-half when the probability of tissue establishment is low ($\bar{x} < 1/(2 - k)$) and falls to 0 as the probability of tissue establishment approaches unity ($z \to 0$ as $\bar{x} \to 1$).

provided in table 1 and assume $\bar{x} < 1/(2 - k)$ (and hence $y^* = [1 - \bar{x}(2 - k)]/2(1 - \bar{x})$), then condition (5) can be rewritten as $\bar{x}k^2 + (3 - 4\bar{x})k - 2(1 - \bar{x}) < 0$. If instead $\bar{x} \geq 1/(2 - k)$ (and hence $y^* = 0$), then condition (5) is always satisfied. Hence, when tissue establishment is uncommon ($\bar{x} < 1/(2 - k)$), the tissue is favored to promote its establishment when the fecundity cost is low ($k < \{4\bar{x} - 3 + [9 - 8\bar{x}(2 - \bar{x})]^{1/2}\}/(2\bar{x})$) and is favored to suppress its establishment when the fecundity cost is high ($k > \{4\bar{x} - 3 + [9 - 8\bar{x}(2 - \bar{x})]^{1/2}\}/(2\bar{x})$). In the special case of vanishingly low frequency of tissue establishment ($\bar{x} \to 0$), the maximum fecundity cost to the female that the tissue will endure without being favored to suppress its own establishment corresponds to her fecundity being reduced by two-thirds ($k = 2/3$), and as the tissue establishment becomes more common, the tissue is prepared to accept even higher collateral damage to the female (fig. 3).

Notice that when the probability of tissue establishment is low ($\bar{x} < 1/(2 - k)$), both the infectious tissue and the female can be favored to promote or inhibit the establishment of the former, depending on the fecundity cost incurred by the latter. Moreover, the critical cost value from the perspective of the infectious tissue is always equal to or greater than the critical cost value from the perspective of the female ($0 < 1/(2 - \bar{x}) \leq \{4\bar{x} - 3 + [9 - 8\bar{x}(2 - \bar{x})]^{1/2}\}/(2\bar{x}) < 1$). Hence, when the fecundity cost is low ($k < 1/(2 - \bar{x})$), both parties are favored to promote the establishment of the infectious tissue (collaboration); when the fecundity cost is high ($k > \{4\bar{x} - 3 + [9 - 8\bar{x}(2 - \bar{x})]^{1/2}\}/(2\bar{x})$), both parties are favored to suppress the establishment of the infectious tissue (collaboration); and when the fecundity cost is intermediate ($1/(2 - \bar{x}) < k < \{4\bar{x} - 3 + [9 - 8\bar{x}(2 - \bar{x})]^{1/2}\}/(2\bar{x})$), the tissue is favored to promote and the female to suppress the establishment of the infectious tissue (conflict). The scope for conflict narrows as the establishment of infectious tissue becomes increasingly common in the population, with both parties becoming more inclined to promote establishment (fig. 3).

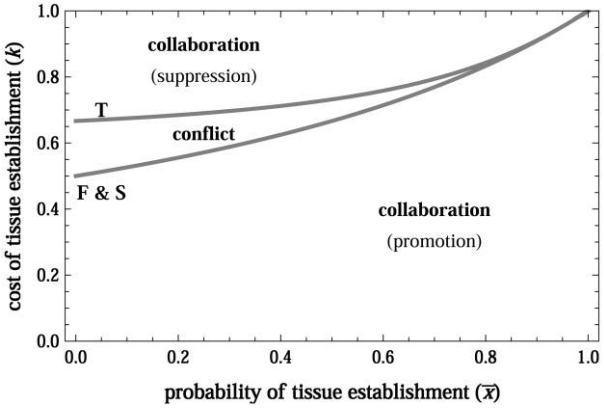

Figure 3: Evolution of infectious-tissue establishment. Females (F), infectious tissues (T), and maternally inherited symbionts (S) are all favored to promote tissue establishment when this is sufficiently common (higher $\bar{x}$) and when the cost of tissue establishment is sufficiently low (lower k). For uncommon tissue establishment (low $\bar{x}$) and intermediate cost of establishment (intermediate k), females and maternally inherited symbionts are favored to suppress establishment, while infectious tissues are favored to promote establishment, giving rise to an evolutionary conflict. Elsewhere, all parties are favored to either promote tissue establishment (when the cost is low; small k) or suppress tissue establishment (when the cost is high; large k), giving rise to an evolutionary collaboration. Note that the interests of females and maternally inherited symbionts are exactly aligned for this trait.

Finally, we consider the interests of a maternally inherited symbiont carried by the female by assigning it control of the probability of infectious-tissue establishment and seeing how it is favored to adjust this. The condition for natural selection to favor an increased probability of tissue establishment is $\partial H_S/\partial x > 0$, that is,

$$\frac{\partial n_\alpha}{\partial x} v_{m|S} r_{S\alpha} + \frac{\partial n_\beta}{\partial x} v_{f|S} r_{S\beta} + \frac{\partial n_\gamma}{\partial x} v_{f|S} r_{S\gamma} + \frac{\partial n_\delta}{\partial x} v_{t|S} r_{S\delta} + \frac{\partial n_\varepsilon}{\partial x} v_{t|S} r_{S\varepsilon} > 0, \quad (6)$$

where reproductive values are in terms of transmission of symbionts rather than autosomal genes (i.e., $v_{m|S} = v_{t|S} = 0$, $v_{f|S} = 1$), relatedness coefficients are in terms of presence or absence of a descendant symbiont (i.e., $r_{S\alpha} = r_{S\beta} = r_{S\gamma} = 1$, $r_{S\delta} = r_{S\varepsilon} = 0$), and all derivatives are evaluated in a monomorphic population ($x = \bar{x}, y = \bar{y} = y^*$). If we use the information provided in table 1 and assume $\bar{x} < 1/(2 - k)$ (and hence $y^* = [1 - \bar{x}(2 - k)]/2(1 - \bar{x})$, then condition (6) can be rewritten as $k < 1/(2 - \bar{a})$. If instead $\bar{a} \geq 1/(2 - k)$ (and hence $y^* = 0$), then condition (6) is always satisfied. Notice that these are precisely the conditions derived under the assumption of female control of tissue establishment. Hence, the interests of the maternally inherited symbiont and the female are exactly aligned in this respect (fig. 3).

Discussion

We have considered the evolution of hermaphroditism driven by genetic conflicts between the sexes in an ancestrally haplodiploid population. This hypothesis, proposed by Normark (2009), suggests that by infecting females with sperm-producing tissue, males may fertilize not only their partners but also their future daughters. We have performed an inclusive-fitness analysis of this evolutionary model, confirming the potential for a genetic conflict of interests to have driven this unusual form of hermaphroditism. However, while Normark (2009) assumed that the infectious male tissue would always be parasitic—harmful to the interests of females and favored solely on the basis of a selfish transmission advantage—we have shown that there is scope for collaboration as well as conflict between females and their infectious male tissues in the evolution of this novel reproductive system.

In particular, we have found that, owing to relatedness between father and daughter and hence between a female and her infectious male tissue, the infectious tissue can be favored to suppress its own establishment if the fecundity costs incurred by the female are too great, and, conversely, the female may be favored to promote the establishment of the tissue if the fecundity costs are sufficiently low. Thus, while each party may disagree over the critical values of these fecundity costs (the male accepting a greater collateral damage to the female's fecundity than the female is prepared to accept for herself), giving rise to a zone of conflict in the parameter space defined by the evolutionary model, there is also scope for both parties to collaborate in establishing the infectious tissue and thereby promoting the evolution of hermaphroditism (fig. 3).

When we consider the evolutionary origin of the infectious tissue, our model predicts that the tissue itself would be favored to pursue this unusual mode of transmission only when the relative fecundity cost to the infected female was less than two-thirds. Before having been honed by natural selection, to become adapted to its new environment within the female's body, the infection can be expected to have caused disruption to normal female function and hence incurred substantial fecundity costs. It seems very likely, then, that the early stages of the evolution of this reproductive mode occurred within the zone of conflict between the female and her infectious tissue (i.e., $1/2 < k < 2/3$; fig. 3). Hence, the females would initially have been favored to suppress the establishment of the infection before, eventually, their interests aligned and conflict gave way to collaboration. We might therefore expect to find remnants of this historical conflict in the biology of contemporary infections.

Although lack of adaptedness to the internal environment of the female would have presented a barrier to the initial evolution of the infectious tissue, this barrier need not have been insurmountable. Indeed, very little structural adaptation appears to have been necessary, as the ovitestis strongly resembles the original female ovaries and the testis portion serves the dual role of sperm production and sperm transport (becoming hollow as the sperm mature and forming a duct by which they reach the maturing oocytes; Hughes-Schrader 1925). Also, the male and female functions of the ovitestis are separated in space and time, with sperm developing first and in the central portions of the ovitestis and the oocytes developing later and on the periphery of the common gonad (Hughes-Schrader 1925). Hence, although our model assumes a fixed fecundity cost (k) of tissue establishment, there is scope for this cost to have been reduced during the evolutionary history of this genetic system.

Even under the assumption that the fecundity cost of tissue establishment remains fixed, our model shows that as the frequency of tissue establishment increases in the population, females are increasingly favored to promote the establishment of their infectious tissue (fig. 3). This is because females must balance the indirect fitness benefit that they gain from helping their infectious tissue (to which they are genetically related) gain reproductive success against the direct fitness cost owing to reduced fecundity.

As tissue establishment becomes more common, the reproductive value of infectious tissue increases relative to that of regular sons and daughters (table 1), so the female increasingly improves her inclusive fitness by allowing her infectious tissue to establish and to provide it with daughters that will carry the infection into subsequent generations.

An important assumption of our model is that supernumerary sperm deriving from a regular male establish a haploid sperm-producing tissue within his daughter's body. We have assumed that all females receive this supernumerary sperm and that this incurs no extra cost to the male. Royer (1975) showed, in *Icerya purchasi*, that sperm deriving from the haploid tissue are able to achieve this and, presumably, that the original infectious tissue derived from sperm from a regular male. However, it is currently not known whether regular males can readily transmit their tissue in this way. A key test of the model will be to determine whether the rare males, found in some populations of *Icerya*, are able to infect their daughters with new tissue. A further assumption of the model is that infectious tissues fertilize all of a female's eggs on successful establishment. Elsewhere, we have relaxed this assumption, allowing the fertilization strategy to evolve under the control of the infectious tissue (bypassing the usual mechanisms by which females decide which of their eggs are to be fertilized), and this does not change any of the model's predictions (Ross 2010, chap. 5). More generally, it is difficult to assess assumptions and predictions concerning the early stages of the evolution of the infectious tissue. However, there may be potential to introduce the infectious tissue into nonhermaphroditic species of *Icerya* to re-create these initial conditions.

A curious aspect of the developmental biology of the infectious male tissue is the interaction this appears to have with endosymbiotic bacteria, inherited from the mother, during early embryonic development. Although there is no conclusive evidence that the endosymbiont—which *Icerya* harbors for nutritional reasons—is involved in the establishment of the infectious tissue, Royer (1975) observed that there was a strong physical association between the developing haploid cells and the bacteria, with the bacteria surrounding the haploid cells. He also showed that when females were treated with antibiotics in order to remove the endosymbionts, they were more likely to produce sons (Royer and Delavaul 1974). Royer (1975) suggested that the bacteria may protect the haploid cells from degeneration and hence play a crucial role in the evolution of their host's hermaphroditism. In order to assess the likelihood of this suggestion, we investigated the evolutionary interests of a maternally inherited symbiont with regard to the establishment of the infectious tissue. The symbiont is expected to promote tissue establishment when this increases the expected number of daughters produced by its host, as only females transmit the symbiont to future generations. In the context of our model, we found that the interests of the symbiont are exactly aligned with those of the female host: although ultimately the inclusive fitness objectives of the two parties are not the same, they are in perfect agreement more proximately, in terms of how large a fecundity cost should be endured before suppression of the infectious tissue is favored (fig. 3). Thus, the endosymbiont does have a stake in mediating the establishment of the infectious male tissue. Endosymbiotic bacteria in other taxa have proven capable of manipulating their host's reproduction in numerous ways; if this role of endosymbionts in *Icerya* were to be confirmed, it would provide the first known example of endosymbiont-induced hermaphroditism.

Our model accounts for the rarity of males among the hermaphroditic species of *Icerya*. Although all three species can reproduce by "selfing," regular males have been observed in each of these species, where they develop from unfertilized eggs. The reported frequencies of males vary between studies and species (roughly 0%–10%; Hughes-Schrader 1925, 1930, 1963; Hughes-Schrader and Monahan 1966). We have shown that for populations in which it is the norm for infectious tissues to become established in females ($(\bar{x} > 1/(2 - k))$), those females for which the male tissue has failed to establish are predicted to fertilize none of their eggs ($y^* = 0$; fig. 2, *top*). Hence, regular males are expected to be produced whenever there is a less-than-perfect rate of infection. This prediction could be tested by experimentally disrupting the transmission of the infectious tissue to daughters, possibly via temperature effects (Royer and Delavaul 1974).

Why are uninfected females favored to invest resources into the production of sons, even when those sons have virtually no prospect of achieving mating success (i.e., when no female uses sperm from regular males to fertilize any of her eggs)? This is because while sons may struggle to find mates, daughters have similarly bleak prospects in terms of achieving longer-term reproductive value. Daughters can reproduce, but if almost all females fertilize their eggs by using sperm derived from infectious tissue, then essentially all of the genetic ancestry of the population belongs to the infectious tissues. The reproductive value of an uninfected female hinges on her producing regular sons who may have some small probability of establishing a new infectious tissue. In contrast, infected females maximize their inclusive fitness by producing daughters to serve as vessels for carrying their infectious tissue into future generations.

Our model shows interesting parallels with previous work on the evolution of self-fertilization, which has received much attention in relation to plants. All else being

equal, an individual's inclusive fitness is raised by allowing its relatives' (including its own) male gametes to fertilize its female gametes (Fisher 1941; Parker 1979). However, this selective advantage for selfing may be countered by inbreeding depression owing to the phenotypic expression of recessive deleterious genes (Lande and Schemske 1985; Porcher and Lande 2005*a*, 2005*b*, 2005*c*). Inbreeding depression is typically neglected in models of inbreeding in haplodiploids, on account of male haploidy, which exposes recessive genes to selection and hence is expected to purge recessive deleterious genes from the population (Henter 2003). We have not explicitly modeled the impact of inbreeding depression that, if in operation, would act to slow or prevent the evolution of selfing in *Icerya*. However, the qualitative effects of inbreeding depression are implicitly captured in the fecundity cost of infectious-tissue establishment (k). A more explicit analysis of inbreeding depression in this system, incorporating explicit mutation and purging rates, would be an interesting avenue for future exploration. Finally, self-fertilization can be a particularly successful strategy if mating opportunities are scarce. We have assumed that females are not sperm limited in the ancestral haplodiploid population, but allowing for this factor could provide an additional evolutionary advantage for tissue establishment.

A semantic point may be made on our use of the term "hermaphroditism" in the context of *Icerya*. While the endpoint of the evolutionary process is an integrated individual organism comprising genetically identical ($r = 1$) male and female reproductive tissues, earlier stages in the evolution of this genetic system might be better conceptualized in terms of separate sexes, with the genetically distinct ($r < 1$) infectious tissue being regarded as a separate organism and not part of the female's body (i.e., extreme male dwarfism; Haig 1997). More generally, we have used the term "hermaphroditism" mainly for consistency with the existing literature on *Icerya* (Hughes-Schrader 1925, 1963; Hughes-Schrader and Monahan 1966; Royer 1975; Nur 1980; Hurst 1993; Normark 2003, 2009; Ross et al. 2010), despite the dissimilarities with the reproductive systems of other hermaphroditic taxa. Given the apparent ease with which this system has evolved in *Icerya*, it is perhaps surprising that similar systems have not been observed elsewhere. One possibility is that, owing to the close resemblance to "classic" selfing hermaphroditism, it may be that other examples do exist but have been overlooked. We hope our model will inspire more thorough study on the origin of male gametes in other hermaphroditic taxa.

There is growing interest in the role for genetic conflicts to explain the evolutionary transitions between genetic systems, including the evolution of well-known and widespread systems, such as haplodiploidy and parthenogenesis (Bull 1979; Hurst et al. 1990; Normark 2004; Ross et al. 2010). The hypothesis considered in this article constitutes the first suggestion that the evolution of hermaphroditism can been driven by such conflicts (Normark 2009). In other taxa, genetic conflicts have been implicated in evolutionary transitions in the opposite direction, for example, cytoplasmic sterility as an adaptation of mitochondria to induce loss of male function in hermaphroditic plants to give rise to a system of gynodioecy (Saumitou-Laprade et al. 1994). More generally, while the ecological dominance of one reproductive mode over another may be determined by such factors as mate availability and the costs and benefits of specializing in different sexes, the evolutionary transitions between such systems may be driven by rather different pressures, including conflict between genes over their transmission.

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APPENDIX

Reproductive Value and Relatedness

Reproductive Value

The reproductive value of a class is the expected asymptotic contribution of genes made by individuals of that class to future generations (for an accessible account, see Taylor 1996). This can be calculated recursively: the reproductive value of a focal class is equal to the total reproductive value of all classes in the next generation, each being weighted by the proportion of its genes donated by the focal class in the current generation. We will consider three classes: males (m; comprising α males), females (f; comprising β females and γ females), and infectious tissues (t; comprising δ tissues and ϵ tissues). The reproductive value of the male class is $c_{\mathrm{m}} = \sum_{\mathrm{X}} g_{\mathrm{X}\leftarrow\mathrm{m}} c_{\mathrm{X}}$, where $g_{\mathrm{X}\leftarrow\mathrm{m}}$ is the proportion of class-X genes contributed by males (i.e., $g_{\mathrm{m}\leftarrow\mathrm{m}} = 0$, $g_{\mathrm{f}\leftarrow\mathrm{m}} = (1 - \phi)/2$, and $g_{\mathrm{t}\leftarrow\mathrm{m}} = 1 - \phi$). We can write corresponding equations for each of the three classes and summarize these in linear algebraic form:

$$(c_m \quad c_f \quad c_t) = (c_m \quad c_f \quad c_t)\begin{pmatrix} 0 & 1 & 0 \\ (1-\phi)/2 & 1/2 & \phi/2 \\ 1-\phi & 0 & \phi \end{pmatrix}, \tag{A1}$$

where $\phi = \bar{n}_\gamma/\bar{n}_f$ is the proportion of females who are of type γ (see main text) and each element of the gene-flow matrix specifies the proportion of genes in the recipient class (row) that derive from the donor class (column). The class reproductive values are found by solving equation (A1). (Formally, they are given by the left eigenvector of the gene-flow matrix; Taylor 1996.) They are $c_m = (1-\phi)/(3-2\phi)$, $c_f = 2(1-\phi)/(3-2\phi)$, and $c_t = \phi/(3-2\phi)$. Note that for the classical haplodiploidy scenario ($\phi = 0$), all reproductive value belongs to males and females ($c_m + c_f = 1$, $c_t = 0$), and the class reproductive values are in the usual ratios ($c_m = 1/3$, $c_f = 2/3$). Conversely, if all females are fathered by infectious tissue ($\phi = 1$), then all reproductive value belongs to the infectious tissues ($c_m = c_f = 0$, $c_t = 1$).

In a monomorphic population, the reproductive value of a class is shared equally over all individuals in that class. Since we may scale reproductive values by any constant of proportionality K, we can write the reproductive value of an individual male as $v_m = Kc_m/T\bar{n}_m$, where T is the total number of adult females in the population. Setting $K = T(3-2\phi)$ obtains $v_m = (1-\phi)/\bar{n}_m$; similarly, the reproductive value of an individual female is $v_f = 2(1-\phi)/\bar{n}_f$, and the reproductive value of an individual infectious tissue is $v_t = \phi/\bar{n}_t$. These expressions are listed in table 1.

Relatedness

Analysis of kin selection in our model requires calculation of probabilities for social partners to share genes that are identical by descent; these are termed "coefficients of consanguinity" (Bulmer 1994). The consanguinity between an actor A and a social partner X will be denoted p_{AX}. The actor will be either the adult female (F) who is mother to the brood, her infectious tissue (T), or a maternally inherited symbiont also carried by the mother (S). The recipient is an individual of one of the five types of offspring (α–ε).

We begin by denoting the consanguinity of an adult female to her infectious tissue by p; this is the probability that two genes picked at random from the same locus from these two individuals are identical by descent. Note that because the female's infectious tissue is genetically identical to her paternal genome (both deriving from her haploid father), the consanguinity of the female to herself is also p. This is the probability that two genes picked at random, with replacement, from any one of her loci are identical by descent and is given by $p = (1+f)/2$, where f is the consanguinity of her parents. With probability $1-\phi$, she is a β female (her father was a regular male), in which case her parents were unrelated; otherwise, with probability ϕ, she is a γ female (her father was her mother's infectious tissue), in which case the consanguinity of her parents was p. Thus, $f = \phi p$, and hence $p = 1/(2-\phi)$.

The consanguinity of the female to her α son is $p_{F\alpha} = p$ (she supplies her son's genome), to her β daughter is $p_{F\beta} = p/2$ (she supplies one of her daughter's genomes, and an unrelated male supplies the other), to her γ daughter is $p_{F\gamma} = p$ (she supplies one of her daughter's genomes, and her infectious tissue supplies the other), to her δ son is $p_{F\delta} = 0$ (an unrelated male supplies this genome), and to her ϵ son is $p_{F\epsilon} = p$ (her infectious tissue supplies this genome). The consanguinity of the female's infectious tissue to her α son is $p_{T\alpha} = p$ (the female supplies the son's genome), to her β daughter is $p_{T\beta} = p/2$ (the female supplies one of the daughter's genomes, and an unrelated male supplies the other), to her γ daughter is $p_{T\gamma} = p/2 + 1/2$ (the female supplies one of the daughter's genomes, and her infectious tissue supplies the other), to her δ son is $p_{T\delta} = 0$ (an unrelated male supplies this genome), and to her ϵ son is $p_{T\epsilon} = 1$ (the haploid tissue supplies this genome).

Coefficients of relatedness are obtained by dividing the coefficient of consanguinity between actor and social partner by the consanguinity of the actor to herself ($r_{AX} = p_{AX}/p_{AA}$; Bulmer 1994). This scaling is not necessary for a kin selection analysis but is adopted in this article simply because coefficients of relatedness are more familiar than coefficients of consanguinity. The consanguinity of the female to herself is p, so her relatedness to each of her offspring is $r_{F\alpha} = p_{F\alpha}/p = 1$ to her α son, $r_{F\beta} = p_{F\beta}/p = 1/2$ to her β daughter, $r_{F\gamma} = p_{F\gamma}/p = 1$ to her γ daughter, $r_{F\delta} = p_{F\delta}/p = 0$ to her δ son, and $r_{F\epsilon} = p_{F\epsilon}/p = 1$ to her ϵ son. The consanguinity of the tissue to itself is 1, so its relatedness to each of the female's offspring is $r_{T\alpha} = p_{T\alpha} = p$ to her α son, $r_{T\beta} = p_{T\beta} = p/2$ to her β daughter, $r_{T\gamma} = p_{T\gamma} = (1+p)/2$ to her γ daughter, $r_{T\delta} = p_{T\delta} = 0$ to her δ son, and $r_{T\epsilon} = p_{T\epsilon} = 1$ to her ϵ son. After we made the substitution $p = 1/(2-\phi)$, all coefficients of relatedness are listed in table 1.

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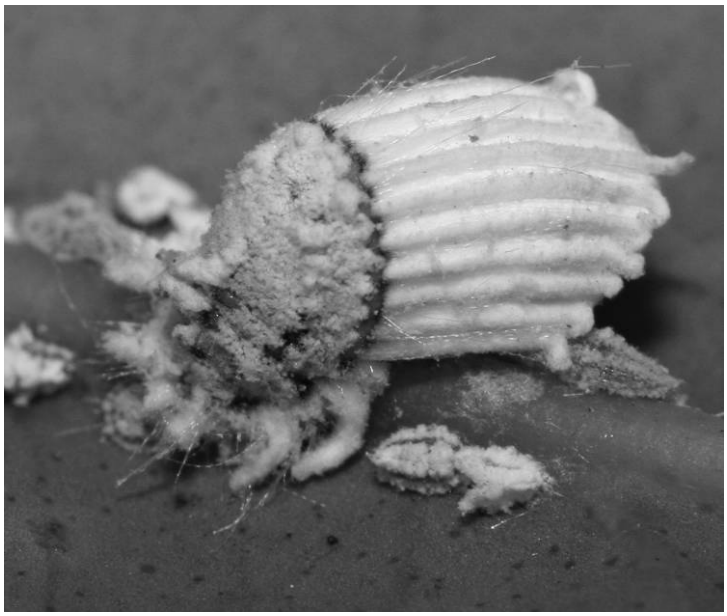

Icerya purchasi mother and babies. Photograph by P. Hollinger.