Genetic conflict and sex allocation in scale insects
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Document Version
Publisher's PDF, also known as Version of record

Publication date:
2010

Link to publication in University of Groningen/UMCG research database

Citation for published version (APA):

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The evolution of hermaphroditism by an infectious male-derived cell lineage: an inclusive fitness analysis

Andy Gardner & Laura Ross

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.
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 & Klinkhamer 2005; Avise & 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 & 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; Charnov, Smith & Bull, 1976; Ghiselin, 1969). Hermaphroditism is found in a large number of taxa, across a wide taxonomic range (Ghiselin 1969; Charnov et al. 1976; Barrett 2002; Jarne & Auld 2006). Although hermaphroditism is very common in some taxonomic groups, it is rare or absent from others. For example, whilst 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 speciose 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 (Bull, 1983; Darlington, 1958). Thus: a separation of the sexes is expected when there are efficiency benefits for individuals specializing in a single reproductive mode (Charnov, 1982; Charnov et al., 1976); 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 specific sexual function is expensive (Charnov et al. 1976; Puurtinen & Kaitala 2002). Such explanations have focused upon ecological and demographic factors. For example, both low population density and impaired mobility has been suggested to drive the evolution of simultaneous hermaphroditism, owing to scarcity of mating opportunities (Ghiselin 1969; Puurtinen & Kaitala 2002; Eppley & 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 & Beukeboom 1998; Hurst & Werren 2001; Normark 2004; Burt & Trivers 2006; Uller et al. 2007; Van Doorn & Kirkpatrick 2007). One source of conflict that has been especially well documented is that between nuclear and cytoplasmic genes (Cosmides & Tooby 1981; Hurst 1992; Werren & Beukeboom 1998; Charlat et al. 2003; Wernegreen 2004; Burt & Trivers 2006). Many insects harbour intracellular bacteria that are transmitted only via daughters (Buchner, 1965); Moran & Telang 1998; Moran & Baumann 2000; Moran 2002), and hence have an interest in biasing their host’s sex allocation towards females (Cosmides & 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, and any male adaptation to increase this fraction would be strongly favoured.

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; (Normark, 2003; Nur, 1980) – the only known instance of hermaphroditism in insects (Hughes-Schrader 1925; Hughes-Schrader 1930; Royer 1975). Scale insects are small, plant-feeding insects (Gullan & Kosztarab, 1997; Ross & 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 & 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 & 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). The sperm-producing gonads of the ovitestis are haploid (Hughes-Schrader, 1963), and this tissue appears to derive from excess sperm that penetrated the oocyst when the female was conceived (Royer, 1975). 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 to examine the evolutionary origin and subsequent spread of infectious male tissue. Whilst Normark (2009) has suggested that the infectious tissue is always parasitic upon the female, and will always 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; the former perhaps showing some restraint, and the latter perhaps showing some shared interest in allowing the infectious tissue to establish. In addition, we consider the interests of a maternally-inherited bacterial symbiont, which has been implicated in facilitating the establishment of the infectious tissue (Ross et al., 2010b; Royer, 1975). More generally, our analysis confirms that genetic conflicts may have driven the evolution of this unusual form of hermaphroditism.
MODEL AND ANALYSIS

Basic model
We build upon 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 (D) and a juvenile son (S). Females are diploid, with one maternal 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), and we allow this tissue to father some of 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 females are unrelated to regular males with which they mate. An illustration of the model is given in Figure 5.1.

The behaviour of an adult female and her infectious tissue impacts upon the allocation of reproductive resources to each of her five types of offspring. With probability 1-a the infectious tissue fails to establish in the focal female’s body, and in this event the female fertilizes a proportion x of her eggs using sperm derived from a regular male and a proportion 1-x of her eggs remain unfertilized. With probability a the infectious tissue successfully establishes, which incurs a relative fecundity cost k for the female, and in this event she fertilizes a proportion x’ of her eggs using sperm derived from a regular male, and her infectious tissue fertilizes a proportion y of the remaining eggs. Hence, denoting 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: \(n_\alpha = n((1-a)(1-x)+a(1-k)(1-x')(1-y))\) α-sons; \(n_\beta = n((1-a)x+a(1-k)x')\) β-daughters; \(n_\gamma = na(1-k)(1-x')y\) γ-daughters; \(n_\delta = n((1-a)x+a(1-k)x')\) δ-sons; and \(n_\varepsilon = na(1-k)(1-x')y\) ε-sons (Table 5.1). Thus, the expected numbers of male, female and tissue offspring produced by the focal female are \(n_m = n_\alpha, n_f = n_\beta + n_\gamma\) and \(n_t = n_\delta + n_\varepsilon\), respectively. We denote population averages (for example, of x) with an overbar (for example, \(\bar{x}\)). We also denote the sex ratio (proportion of regular individuals who are male) by \(z = \bar{n}_m/(\bar{n}_m + \bar{n}_f)\) and the proportion of females that are of type β by \(\phi = \bar{n}_\beta / \bar{n}_f\).

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 (Frank, 1998; Hamilton, 1964). This is product of two quantities: first, the social partners’ ability to transmit copies of their own genes to future generations (reproductive value, v: Fisher, 1930; Frank, 1998); and second, the extent to which genes transmitted by the social partners are the same as those carried by the actor (relatedness, r: Frank, 1998; Hamilton, 1964).
Figure 5.1 THE FAMILY UNIT. Our model is based upon standard haplodiploid inheritance, with only the mother (M) contributing a genome to her haploid son (α-son) and with the mother and father (F) each contributing a genome to their diploid daughter (β-daughter). In addition, the father contributes a genome to infectious tissue that grows in his daughters (δ-son), and the mother’s infectious tissue (T) can fertilize her eggs to produce daughters (γ-daughter) and also further infectious tissues (ε-son).

Table 5.1 Offspring type, number, reproductive value and relatedness to mother and infectious tissue. The proportion of females who are β-daughters is \( \phi = \frac{n_β}{n_β + n_γ} \), and the average number of offspring of each sex is \( n_α = n_α \) males, \( n_β = n_β + n_γ \) females and \( n_β = n_δ + n_ε \) infectious tissues.

<table>
<thead>
<tr>
<th>Type (X)</th>
<th>Number (n_X)</th>
<th>Reproductive value (ν_X)</th>
<th>Relatedness to mother (r_XM)</th>
<th>Relatedness to infectious tissue (r_XT)</th>
</tr>
</thead>
<tbody>
<tr>
<td>α</td>
<td>((1 - x)(1 - a(1 - (1 - k)(1 - y))))</td>
<td>(\frac{\phi}{n_α})</td>
<td>1</td>
<td>(\frac{1}{1 + \phi})</td>
</tr>
<tr>
<td>β</td>
<td>(x(1 - ak))</td>
<td>(\frac{2\phi}{n_β})</td>
<td>(1/2)</td>
<td>(\frac{1}{2 + 2\phi})</td>
</tr>
<tr>
<td>γ</td>
<td>(a(1 - k)(1 - x)y)</td>
<td>(\frac{2\phi}{n_γ})</td>
<td>1</td>
<td>(\frac{2 + \phi}{2 + 2\phi})</td>
</tr>
<tr>
<td>δ</td>
<td>(x(1 - ak))</td>
<td>(\frac{1 - \phi}{n_δ})</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>ε</td>
<td>(a(1 - k)(1 - x)y)</td>
<td>(\frac{1 - \phi}{n_ε})</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>
We assume all genetic similarity owes to shared genealogy, e.g. we exclude green-beard effects (Gardner & West, 2010). Thus, in the context of the present model, the inclusive fitness \( H_A \) of an actor A is defined as:

\[
H_A = n_\alpha v_m r_{A\alpha} + n_\beta v_f r_{A\beta} + n_\gamma v_f r_{A\gamma} + n_\delta v_r r_{A\delta} + n_\epsilon v_r r_{A\epsilon},
\]

(1)

where: \( v_m \), \( v_f \) and \( v_r \) 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 5.1; see Appendix for derivation); 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 5.1; see Appendix for derivation). The condition for natural selection to favour an increase in any character is that this increases the inclusive fitness of the actor (Hamilton, 1964).

**Female fertilization strategies**

We first consider the fertilization strategies of the female. In the event that her infectious tissue does establish, she fertilizes a proportion \( x' \) of her eggs using sperm derived from a regular male. The condition for natural selection to favour an increase in the value of this character is that this increases her inclusive fitness. Assuming vanishing genetic variation, this condition is \( \frac{\partial H_F}{\partial x'} > 0 \), i.e.:

\[
\frac{\partial n_\alpha}{\partial x'} v_m r_{F\alpha} + \frac{\partial n_\beta}{\partial x'} v_f r_{F\beta} + \frac{\partial n_\gamma}{\partial x'} v_f r_{F\gamma} + \frac{\partial n_\delta}{\partial x'} v_r r_{F\delta} + \frac{\partial n_\epsilon}{\partial x'} v_r r_{F\epsilon} > 0,
\]

(2)

where all derivatives are evaluated in a monomorphic population \( (x = \bar{x}, x' = \bar{x}', y = \bar{y}, a = \bar{a}) \). Using the information provided in Table 5.1, and assuming that \( \bar{y} = 1 \) (justified in the next section), we find that condition (2) is never satisfied, hence the population is expected to converge upon the strategy value \( x'^* = 0 \).

In the event that the infectious tissue does not establish itself, the female fertilizes a proportion \( x \) of her eggs using sperm derived from a regular male. The condition for natural selection to favour an increase in this character is \( \frac{\partial H_F}{\partial x} > 0 \), i.e.:

\[
\frac{\partial n_\alpha}{\partial x} v_m r_{F\alpha} + \frac{\partial n_\beta}{\partial x} v_f r_{F\beta} + \frac{\partial n_\gamma}{\partial x} v_f r_{F\gamma} + \frac{\partial n_\delta}{\partial x} v_r r_{F\delta} + \frac{\partial n_\epsilon}{\partial x} v_r r_{F\epsilon} > 0,
\]

(3)

where all derivatives are evaluated in a monomorphic population \( (x = \bar{x}, x' = \bar{x}' = 0, y = \bar{y} = 1, a = \bar{a}) \). Using the information provided in Table 5.1, condition (3) can be rewritten as \( \bar{x} < (1 - \bar{a}(2 - k))/2(1 - \bar{a}) \). Hence, the population is expected to converge upon the strategy value \( x^* \), given by:

\[
x^* = \begin{cases} 
\frac{(1 - \bar{a}(2 - k))}{2(1 - \bar{a})} & \text{if } \bar{a} < 1/(2 - k) \\
0 & \text{if } \bar{a} \geq 1/(2 - k)
\end{cases}
\]
Thus, the female fertilizes some or none of her eggs with sperm derived from a regular male when her infectious tissue does not establish \((x^* \geq 0; \text{this is } x^* = 1/2 \text{ when } \bar{a} = 0; \text{ Figure 5.2A})\), and she fertilizes none of her eggs with sperm derived from a regular male when her infectious tissue does establish \((x^* = 0; \text{Figure 5.2B})\). As a consequence, the population sex ratio is given by \(z = \min(1/2,(1 - \bar{a})/(1 - \bar{a}k))\); proportion of regular individuals who are male) remains fixed at one half when the probability of tissue establishment is low \((\bar{a} < 1/(2 - k))\) and falls to zero as the probability of tissue establishment approaches unity \((z \to 0 \text{ as } \bar{a} \to 1)\).

**Tissue fertilization strategy**
Next, we consider the fertilization strategy of the infectious tissue. In the event that the tissue does establish in the body of a female, it fertilizes a proportion \(y\) of any eggs that she has failed to fertilize using sperm from a regular male. Above, we assumed \(\bar{y} = 1;\) i.e. a successfully establishing tissue fertilizes all of the female’s eggs.
Here we will show that this fertilization strategy is indeed the one that maximizes the tissue's inclusive fitness. The condition for natural selection to favour an increase in the tissue's fertilization strategy is $\frac{\partial H_T}{\partial y} > 0$, i.e.:

$$\frac{\partial n_\alpha}{\partial y} v_m r_{Ta} + \frac{\partial n_\beta}{\partial y} v_f r_{T\beta} + \frac{\partial n_\gamma}{\partial y} v_f r_{T\gamma} + \frac{\partial n_\delta}{\partial y} v_t r_{T\delta} + \frac{\partial n_\epsilon}{\partial y} v_t r_{T\epsilon} > 0,$$  \hspace{1cm} (5)

where all derivatives are evaluated in a monomorphic population ($x = \bar{x}$, $x' = \bar{x}' = 0$, $y = \bar{y}$, $a = \bar{a}$). Using the information provided in Table 5.1, condition (5) can be rewritten as $(r_{T\gamma} - r_{T\beta}) v_f + r_{T\gamma} v_t > 0$ which (owing to $r_{T\gamma} > r_{T\beta}$) is always satisfied, hence the population will converge upon the strategy value $y^* = 1$. Thus, the infectious tissue is always favoured to fertilize all of the female’s eggs (Figure 5.2C).

**Tissue establishment**

We now examine the evolution of the probability of tissue establishment, $a$. We begin by considering the interests of the female, by assigning her full control of the probability of establishment, and determining when she is favoured to increase or decrease this quantity. The condition for natural selection to favour an increase in the probability of tissue establishment is that this increases her inclusive fitness. Assuming vanishing genetic variation, this condition is $\frac{\partial H_F}{\partial a} > 0$, i.e.:

$$\frac{\partial n_\alpha}{\partial a} v_m r_{Fa} + \frac{\partial n_\beta}{\partial a} v_f r_{F\beta} + \frac{\partial n_\gamma}{\partial a} v_f r_{F\gamma} + \frac{\partial n_\delta}{\partial a} v_t r_{F\delta} + \frac{\partial n_\epsilon}{\partial a} v_t r_{F\epsilon} > 0,$$  \hspace{1cm} (6)

where all derivatives are evaluated in a monomorphic population ($x = \bar{x} = x^* = \bar{x}' = 0$, $y = \bar{y} = 1$, $a = \bar{a}$). Using the information provided in Table 5.1, and assuming $\bar{a} < 1/(2 - k)$ (and hence $x^* = (1 - \bar{a})(2 - k))/2(1 - \bar{a})$), condition (6) can be rewritten as $k < 1/((2 - \bar{a}))$. If instead $\bar{a} > 1/(2 - k)$ (and hence $x^* = 0$), then condition (6) is always satisfied. Hence, when tissue establishment is relatively uncommon ($\bar{a} < 1/(2 - k)$) the female is favoured to promote the establishment of her infectious tissue when the fecundity cost of establishment is low ($k < 1/(2 - \bar{a})$) and is favoured to suppress the establishment of her infectious tissue when the fecundity cost is high ($k > 1/(2 - \bar{a})$). In the special case of vanishingly rare establishment of tissues ($\bar{a} \rightarrow 0$) the maximum cost the female will endure without being favoured to suppress tissue establishment is the loss of half of her fecundity ($k = 1/2$), and as tissue establishment becomes more common (higher $\bar{a}$) the female is favoured to promote establishment for even higher fecundity costs (Figure 5.3).

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 favoured to promote or suppress its own establishment. Natural selection favours an increase in the probability of establishment when $\frac{\partial H_T}{\partial a} > 0$, i.e.:

$$\frac{\partial n_\alpha}{\partial a} v_m r_{Ta} + \frac{\partial n_\beta}{\partial a} v_f r_{T\beta} + \frac{\partial n_\gamma}{\partial a} v_f r_{T\gamma} + \frac{\partial n_\delta}{\partial a} v_t r_{T\delta} + \frac{\partial n_\epsilon}{\partial a} v_t r_{T\epsilon} > 0,$$  \hspace{1cm} (7)
where all derivatives are evaluated in a monomorphic population ($x = \bar{x} = x^*$, $x' = \bar{x}' = 0$, $y = \bar{y} = 1$, $a = \bar{a}$). Using the information provided in Table 5.1, and assuming $\bar{a} < 1/(2 - k)$ (and hence $x^* = (1 - \bar{a})(2 - k)/2(1 - \bar{a})$), condition (7) can be rewritten as $\bar{a}k^2 + (3 - 4 \bar{a})k - 2(1 - \bar{a}) < 0$. If instead $\bar{a} > 1/(2 - k)$ (and hence $x^* = 0$), then condition (7) is always satisfied. Hence, when tissue establishment is uncommon ($\bar{a} < 1/(2 - k)$) the tissue is favoured to promote its establishment when the fecundity cost is low ($k < (4 \bar{a} - 3 + \sqrt{9 - 8 \bar{a}(2 - \bar{a})})/(2 \bar{a})$) and is favoured to suppress its establishment when the fecundity cost is high ($k > (4 \bar{a} - 3 + \sqrt{9 - 8 \bar{a}(2 - \bar{a})})/(2 \bar{a})$). In the special case of vanishingly low frequency of tissue establishment ($\bar{a} \to 0$), the maximum fecundity cost to the female that the tissue will endure without being favoured to suppress its own establishment is 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 (Figure 5.3).

Notice that, when the probability of tissue establishment is low ($\bar{a} < 1/(2 - k)$), both the infectious tissue and the female can be favoured to promote or inhibit the establishment of the former, depending upon 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 ($0 < 1/(2 - \bar{a}) < (4 \bar{a} - 3 + \sqrt{9 - 8 \bar{a}(2 - \bar{a})})/(2 \bar{a}) < 1$). Hence: when the fecundity cost is low ($k < 1/(2 - \bar{a})$) both parties are favoured to promote the establishment of
the infectious tissue (collaboration); when the fecundity cost is high \((k > (4 \tilde{a} - 3 + \sqrt{9 - 8 \tilde{a}(2 - \tilde{a}))/(2 \tilde{a}))\) both parties are favoured to suppress the establishment of the infectious tissue (collaboration); and when the fecundity cost is intermediate \((1/(2 - \tilde{a}) < k < (4 \tilde{a} - 3 + \sqrt{9 - 8 \tilde{a}(2 - \tilde{a}))/(2 \tilde{a}))\), the tissue is favoured 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 (Figure 5.3).

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 favoured to adjust this. The condition for natural selection to favour an increased probability of tissue establishment is \(\frac{\partial H_S}{\partial a} > 0\), i.e.:

\[
\frac{\partial n_\alpha}{\partial a} v_m|S r_{S\alpha} + \frac{\partial n_\beta}{\partial a} v_f|S r_{S\beta} + \frac{\partial n_\gamma}{\partial a} v_f|S r_{S\gamma} + \frac{\partial n_\delta}{\partial a} v_f|S r_{S\delta} + \frac{\partial n_\epsilon}{\partial a} v_f|S r_{S\epsilon} > 0 ,
\]

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. \(r_{S\alpha} = r_{S\beta} = r_{S\gamma} = 1, r_{S\delta} = r_{S\epsilon} = 0\)); and where all derivatives are evaluated in a monomorphic population \((x = \tilde{x} = x^*, x' = \tilde{x}' = 0, y = \tilde{y} = 1, a = \tilde{a})\). Using the information provided in Table 5.1, and assuming \(\tilde{a} < 1/(2 - k)\) (and hence \(x^* = (1 - \tilde{a})(2 - k)/2(1 - \tilde{a})\)), condition (8) can be rewritten as \(k < 1/(2 - \tilde{a})\). If instead \(\tilde{a} > 1/(2 - k)\) (and hence \(x^* = 0\), then condition (8) 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 (Figure 5.3).

**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, whilst Normark (2009) assumed that the infectious male tissue would always be parasitic – harmful to the interests of females, and favoured 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 favoured to suppress its own establishment if the fecundity costs incurred by the host female are too great and, conversely the female may be favoured to promote the establishment of the tissue if the fecundity costs are sufficiently low. Thus, whilst 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 (Figure 5.3).

Considering the evolutionary origin of the infectious tissue, our model predicts that the tissue itself would be favoured 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. $1/2 < k < 2/3$; Figure 5.3). Hence, the females would initially have been favoured 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 ovitests 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 function of the ovitests is separated in space and time, with sperm developing first and in the central portions of the ovitests and the oocytes developing later and on the periphery of the common gonad (Hughes-Schrader, 1925).

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 harbours 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. 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 regards 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. 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 favoured (Figure 5.3). 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 & Monahan 1966). We have shown that, for populations in which it is the norm for females to carry the infectious tissue ($\bar{a} < 1/(2 - k)$), those females for which the male tissue has failed to establish are predicted to fertilize none of their eggs ($x^* = 0$; Figure 5.2A). Hence, regular males are expected to be produced whenever there is a less than perfect rate of infection, even though they have essentially zero reproductive value: the behaviour of the females that leads to regular males being produced (failure to fertilize eggs with sperm from regular males; Figure 5.2A) means that there is essentially no prospect for regular males to achieve reproductive success!

Why are uninfected females favoured to invest resources into the production of sons, when they could use sperm from such regular males to fertilize their eggs, and hence produce daughters who could go on to have their own offspring? The reason is that daughters have similarly bleak prospects, in terms of longer-term reproductive value. Whilst these daughters can reproduce, essentially all of the genetic ancestry of the population belongs to the infectious tissues. As the proportion of daughters fathered by regular males falls to zero, so to does the reproductive value of females, and hence so too does the inclination of the female to fertilize her eggs with sperm from regular males. Factoring in the higher relatedness to sons than to daughters, uninfected females maximize their miniscule inclusive fitness by fertilizing none of their eggs. More generally, once all daughters are fathered by infectious tissues, the only prospect for a female to achieve inclusive fitness is by producing daughters to serve as vehicles that carry the male infection into future generations.

There is growing interest in the role for genetic conflicts to explain the evolutionary transitions between several genetic systems, including the evolution of well-known and widespread systems such as haplodiploidy and parthenogenesis (Bull 1979; Hurst et al. 1990; Normark 2004). The hypothesis considered in this paper 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: e.g. 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, whilst 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.

Acknowledgements
We thank: B. Normark, I. Pen and D. Shuker for discussion and comments on the manuscript; the Royal Society (AG) and the University of Groningen (LR) for funding.
APPENDIX

Reproductive value

The reproductive value of a class is the expected asymptotic contribution of genes made by individuals of that class to future generations (see Taylor & Frank, 1996 for an accessible account). 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 $\alpha$-males), females ($f$; comprising $\beta$-females and $\gamma$-females), and infectious tissues ($t$; comprising $d$-tissue and $e$-tissues). The reproductive value of the male class is

$$cm = \sum_X X g_{X-m} c_X,$$

where $g_{X-m}$ is the proportion of class-$X$ genes contributed by males (i.e. $g_{m-m} = 0$, $g_{f-m} = \phi/2$ and $g_{t-m} = \phi$). We can write corresponding equations for each of the three classes, and summarize these in linear algebraic form:

$$
\begin{pmatrix}
0 & 1 & 0 \\
\phi/2 & 1/2(1-\phi)/2 \\
\phi & 0 & 1-\phi
\end{pmatrix}
\begin{pmatrix}
cm \\
cf \\
c_t
\end{pmatrix}
= \begin{pmatrix}
cm \\
cf \\
c_t
\end{pmatrix}
\phi/2 1/2(1-\phi)/2 ,
$$

(A1)

where: $\phi = \bar{n}_\beta / \bar{n}_f$ is the proportion of females who are of type $\beta$ (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 $cm = \phi/(1+2\phi)$, $cf = 2\phi/(1+2\phi)$ and $ct = (1-\phi)/(1+2\phi)$. Note that, for the classical haplodiploidy scenario ($\bar{\phi} = 0$), all reproductive value belongs to males and females ($cm + cf = 1, ct = 0$), and the class reproductive values are in the usual ratios ($cm = 1/3, cf = 2/3$). Conversely, if all females are fathered by infectious tissue ($\bar{\phi} = 1$) then all reproductive value belongs to the infectious tissues ($cm + cf = 0, ct = 1$).

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 $v_m = K cm / T \bar{n}_m$, where $T$ is the total number of adult females in the population. Setting $K = T(1+2\phi)$ obtains $v_m = \phi / \bar{n}_m$, and similarly the reproductive value of an individual female is $v_f = 2\phi / \bar{n}_f$, and the reproductive value of an individual infectious tissue is $v_t = (1-\phi) / \bar{n}_t$. These expressions are listed in Table 5.1.

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, or 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 $\phi$ she is a β-female (her father was a regular male), in which case her parents were unrelated; otherwise, with probability $1–\phi$, she is a γ-female (her father was her mother’s infectious tissue), in which case the consanguinity of her parents is $p$. Thus $f = (1–\phi)p$, and hence $p = 1/(1+\phi)$.

The consanguinity of the female to her: α-son is $p_{F\alpha} = p$ (she supplies her son’s genome); β-daughter is $p_{F\beta} = p/2$ (she supplies one of her daughter’s genomes, and an unrelated male supplies the other); γ-daughter is $p_{F\gamma} = p$ (she supplies one of her daughter’s genomes, and her infectious tissue supplies the other); δ-tissue is $p_{F\delta} = 0$ (an unrelated male supplies this genome); ε-tissue is $p_{F\varepsilon} = p$ (her infectious tissue supplies this genome). The consanguinity of the female’s infectious tissue to the: α-son is $p_{T\alpha} = p$ (the female supplies the son’s genome); β-daughter is $p_{T\beta} = p/2$ (the female supplies one of the daughter’s genomes, and an unrelated male supplies the other); 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); δ-son is $p_{T\delta} = 0$ (an unrelated male supplies this genome); ε-son is $p_{T\varepsilon} = 1$ (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 $r_{F\varepsilon} = p_{F\varepsilon}/p = 1$ 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 $r_{T\varepsilon} = p_{T\varepsilon} = 1$ to her ε-son. Making the substitution $p = 1/(1+\phi)$, all coefficients of relatedness are listed in Table 5.1.