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Theoretical Population Biology 70 (2006) 511–526
Theoretical Population Biology
www.elsevier.com/locate/tpb

Male killers and the origins of paternal genome elimination

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Received 11 January 2006
Available online 22 July 2006

Abstract

The haploidizing male killer hypothesis suggests an evolutionary origin for paternal genome elimination (PGE) that is consistent with the ecological correlates of ancestral haplodiploid insect clades. We make use of population genetics models to test the logic and assumptions of this hypothesis with particular emphasis on the co-evolution between bacteria and host. We derive simple invasion conditions for rare modifiers of bacteria transmission and rare modifiers of host survivorship after haploidization. We also study the evolutionary dynamics of both these modifiers. We conclude that PGE shows evolutionary genetic stability and present a comprehensive analysis of the probability that such genetic system evolves due to the action of cytoplasmic genes.

Keywords: Haplodiploidy; Paternal genome elimination; Insects; Endosymbionts; Cytoplasmic genes; Intragenomic conflict; Evolutionary genetic stability; Fitness compensation; Sibling Rivalry

1. Introduction

Paternal genome elimination (PGE) is an unusual genetic system; males develop from fertilized eggs but do not transmit their paternally inherited genome. Some authors consider this system as the precursor of the second type of haplodiploidy, arrhenotoky (Schrader and Hughes-Schrader, 1931; Cruickshank and Thomas, 1999). In arrhenotoky, males develop from unfertilized eggs lacking a paternally inherited genome altogether. The evolutionary biology of these haploid-male genetic systems is interesting for several reasons. They have evolved repeatedly—20 times or more—and appear to be stable and adaptable alternatives to the usual diploid-male genetic systems (Normark, 2003). They inspired some of the earliest models of maternal–paternal genetic conflict (Brown, 1963, 1964) and have continued to attract the interest of evolutionary theorists (Hamilton, 1993; Haig, 1993a, b; Herrick and Seger, 1999; Burt and Trivers, 2006). Male-haploid systems have been thought to be critical to the origins of the most sophisticated forms of eusociality (Hamilton, 1964a,b; Wilson, 1971), though this is becoming more controversial (Wilson, 2005).

Several hypotheses seeking to account for the adaptive significance of haplodiploidy in general have been proposed. Two have been particularly influential. The maternal transmission hypothesis holds that haplodiploidy confers an advantage to females that produce fatherless sons because such sons always transmit the genome inherited from their mothers (Brown, 1963, 1964; Hartl and Brown, 1970; Smith, 2000). The deleterious mutation hypothesis claims that the advantage of haplodiploidy lies in its greater efficiency in purging deleterious recessive mutations (Richerd et al., 1994; Smith, 2000).

Other hypotheses account for the adaptive significance of PGE in particular. The feminizing endosymbiont hypothesis holds that PGE is the phenotype of maternally transmitted cytoplasmic genes acting in the germline of adult male hosts (Hamilton, 1993). These genes prevent the transmission of their host's male determining gene, thereby causing the progeny of the infected male to develop as daughters. This phenotype is advantageous to cytoplasmic genes if the host is inbreeding, but potentially deleterious if the host is outcrossing (Normark, 2004). Hamilton (1993) argued that this process can ultimately result in the

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doi:10.1016/j.tpb.2006.06.011

elimination of the paternally inherited genome. This model of Hamilton's was the first to provide an adaptive explanation for the role of cytoplasmic genes in the evolution of PGE but he was not the only one to suggest a causal connection between maternally transmitted bacteria and haplodiploidy (Peleg and Norris, 1972a, b; Norris, 1993; Behura et al., 2001; Vega et al., 2002; Harris et al., 2003).

Normark (2004) argued the need for a hypothesis to explain the apparent ecological correlates of ancestral clades of haplodiploidy insects: presence of maternally transmitted bacteria, male heterogamety, sibling competition and frequent outcrossing. Following Hamilton (1993) the haploidizing male killer hypothesis (Normark, 2004) claims that PGE is the phenotype of cytoplasmic genes acting in male zygotes. However, in contrast to Hamilton's hypothesis Normark hypothesized that the original function of maternally transmitted bacteria was to kill male zygotes by reacting against the genome of incoming male-determining sperm. Since the cytoplasmic genes considered turn male zygotes haploid, we refer to these genes as male haploidizers (MHs). The advantage to maternally transmitted bacteria comes from the fitness compensation that sisters of non-viable zygotes experience. Initially there is no advantage to the female host genome (though the fitness loss is mitigated by the fitness compensation of surviving sisters). If, however, some haploid males survive, then female hosts will derive a fitness advantage due to enhanced transmission of the maternal genome through haploid males, as suggested by the maternal transmission hypothesis.

In its original formulation the haploidizing male killer hypothesis suggests that PGE is the outcome of a co-evolutionary process between cytoplasmic genes with a given rate of vertical transmission and nuclear genes that allow survivorship of haploidized males. Normark (2004) modelled the conditions under which MHs are advantageous to their host when rare. Recently, it has been pointed out that Normark's (2004) analysis failed to consider the invasion condition for the MH in first place (Engelstädter and Hurst, 2005). Engelstädter and Hurst (2005) modelled the conditions that allow MH to invade and provided the conditions under which MH are advantageous to their host when abundant. The consideration of both these conditions led Engelstädter and Hurst (2005) to conclude that while PGE as the outcome of a co-evolutionary process between MH and its host is possible, it is unlikely.

In the present work we go one step further in analyzing the co-evolution between MH and its host. We revisit the population genetics model proposed by Engelstädter and Hurst (2005) and extend it in two ways: first we allow for variable transmission of MHs and second we allow for variable survivorship of haploidized males. We also explore the evolutionary dynamics of these models. We use the insight gained from our analysis to discuss the possible outcomes of a co-evolutionary process. We show that perfect transmission of MHs and perfect survivorship of haploidized males is the only outcome of a co-evolutionary process between both parameter values showing evolutionary genetic stability (EGS) (Eshel and Feldman, 1982). We also show how the plausibility of this co-evolutionary process greatly depends on the parametrization of the model and argue that the particular one chosen by Engelstädter and Hurst (2005) to illustrate their discussion of the hypothesis is the least favorable of many parametrizations relevant from an empirical perspective.

2. Model

Consider an infinite population of insects, some of which are infected with maternally transmitted bacteria that disable the paternally inherited genome of male hosts. Let b denote the haploid genotype of the MH bacteria in an infected host and let o denote the absence of this genotype (in an uninfected host). An infected female transmits MH vertically with probability t ($0 \leqslant t \leqslant 1$). Whenever an ovum receives at least one MH from its mother, that ovum reacts to any sperm carrying male-determining elements by inactivating its genome. Therefore, the zygote is rendered haploid at an early stage in its development. Such dramatic change in genome configuration results in the death of a fraction $1-s$ of haploidized zygotes while a fraction s ($0 \leqslant s \leqslant 1$) of them survive initiating development as haploid males (see Fig. 1).

Assume that each insect female produces the same number of zygotes, on average half male and half female, and that there is a limited amount of resources to be distributed equally among the clutch. The death of a zygote as a consequence of haploidization sets free a fraction r ($0 \leqslant r \leqslant 1$) of resources originally committed to that zygote's development. Assume that these resources are evenly re-distributed among all zygotes in the same clutch of the dead one, thereby increasing their viability. This occurrence is known in the biological literature as *fitness compensation* and is justified by a reduction in competition for scarce resources between offspring. How fitness compensation may happen in the kind of biological systems we are considering, and the range of r values that is realistic, will be discussed latter in this text.

Following previous work (Hurst, 1991; Randerson et al., 2000; Normark, 2004; Engelstädter and Hurst, 2005) let us define the viability of offspring (that survive haploidization) from infected females v_b relative to the viability of offspring from uninfected females v_o:

$$v_n = \begin{cases} 1, & n = o, \\ 1 + rp(1-p)^{-1}, & n = b, \end{cases} \quad 1 \leqslant v_n \leqslant 1 + r, \tag{1}$$

where $p = \frac{1}{2}t(1-s)$ is the fraction of zygotes killed in each clutch.

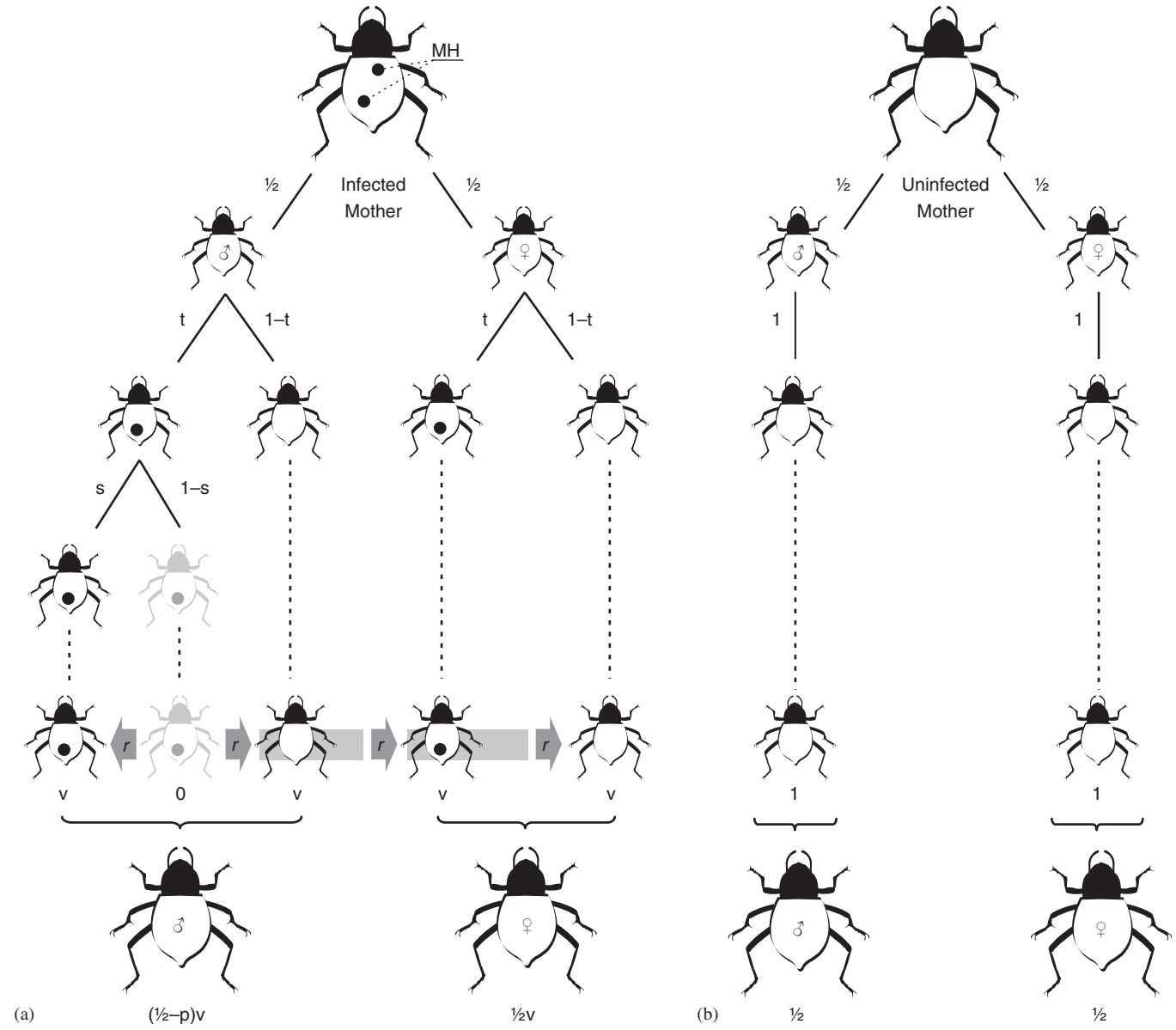


Fig. 1. Life cycle. Life cycle of an infected mother (a) and an uninfected mother (b). Both mothers produce equal number of male and female zygotes. An infected mother transmits male haploidizing bacteria to her zygotes with probability t. A proportion s of the infected male zygotes survive haplodization while the rest die. The death of a zygote results in the increment of the fitness other zygotes in the same clutch.

2.1. MH invasion condition

The model specified in this section follows the one proposed by Engelstädter and Hurst (2005) which in turns relates to a whole class of models derived within the male killer literature (Hurst, 1991; Freeland and McCabe, 1997; Randerson et al., 2000). Let the frequency of uninfected and infected females be x_o and x_b, and the frequency of uninfected and infected males be y_o and y_b ($0 \leqslant x_n, y_n \leqslant 1$ and $\sum_n x_n = \sum_n y_n = 1$). Their frequency one generation later is

$$\overline{m} y'_o = \overline{f} x'_o = x_o + (1-t) v_b x_b, \tag{2a}$$

$$\frac{\overline{m}}{s} y'_b = \overline{f} x'_b = t v_b x_b, \tag{2b}$$

where

$$\overline{f} = x_o + v_b x_b, \tag{3a}$$

$$\overline{m} = x_o + (1-2p) v_b x_b \tag{3b}$$

is the population mean fitness in female and male classes.

The above system has two equilibria, one trivial and one non-trivial. The trivial equilibrium $\widehat{\mathbf{0}} = (0,0)$ corresponds to a population without MH while the non-trivial one $\widehat{\mathbf{z}} = (\widehat{x}_b, \widehat{y}_b)$ corresponds to a polymorphic population in which the frequency of infected insects is

$$\widehat{x}_b = \frac{t v_b - 1}{v_b - 1}, \tag{4a}$$

$$\widehat{y}_b = s \frac{t v_b - 1}{(1-2p) v_b - s}. \tag{4b}$$

The only instance in which $\widehat{\mathbf{z}}$ corresponds to a population fixed for MH, $\widehat{\mathbf{z}} = (1, 1)$, is under perfect vertical transmission of these bacteria. However, this is true only if some zygotes carry on development after haploidization; otherwise the population becomes extinct after all males have been killed.

The invasion condition for MH is

$$\left.\frac{\partial x_b'}{\partial x_b}\right|_{\widehat{\mathbf{0}}} = tv_b > 1. \tag{5}$$

In particular, for viability function (1) we can plot the region of the parameter space (t, s, r) in which the invasion condition is satisfied and the equilibrium values (see Fig. 2).

The above analysis concludes that a rare MH invades when $tv_b > 1$ is satisfied. The satisfaction of this simple condition also guarantees the existence and stability of equilibrium $\widehat{\mathbf{z}}$ (not shown). Once MH has entered the population we assume that the frequency between infected and uninfected hosts adjusts to equilibrium $\widehat{\mathbf{z}}$ before any other mutant arise as it is customary when studying the long-term evolution of genetic systems (Eshel, 1996).

2.2. Variable transmission

We start by extending the previous model to allow variable transmission. There are two possibilities: either the bacterium determines its own vertical transmission or the host is the one determining the transmission of bacteria.

2.2.1. Bacteria control

This section models bacteria that can influence their own vertical transmission. Let b_{t1} be an allele in bacteria (coding for transmission rate t_1) present in the population when another allele b_{t2} (coding for transmission rate t_2) arises.

Variability in the transmission ratio brings along variability in the relative viability of offspring from infected females:

$$v_n = \begin{cases} 1, & n = o, \\ 1 + rp_i(1 - p_i)^{-1}, & n = b_i, \end{cases} \quad i = \{1, 2\}, \tag{6}$$

where $p_i = \frac{1}{2}t_i(1 - s)$ is the fraction of male zygotes killed in clutches of b_{ti}-infected mothers.

Let the frequency of uninfected and b_{ti}-infected females be x_o and x_{bi}, while the frequency of uninfected and

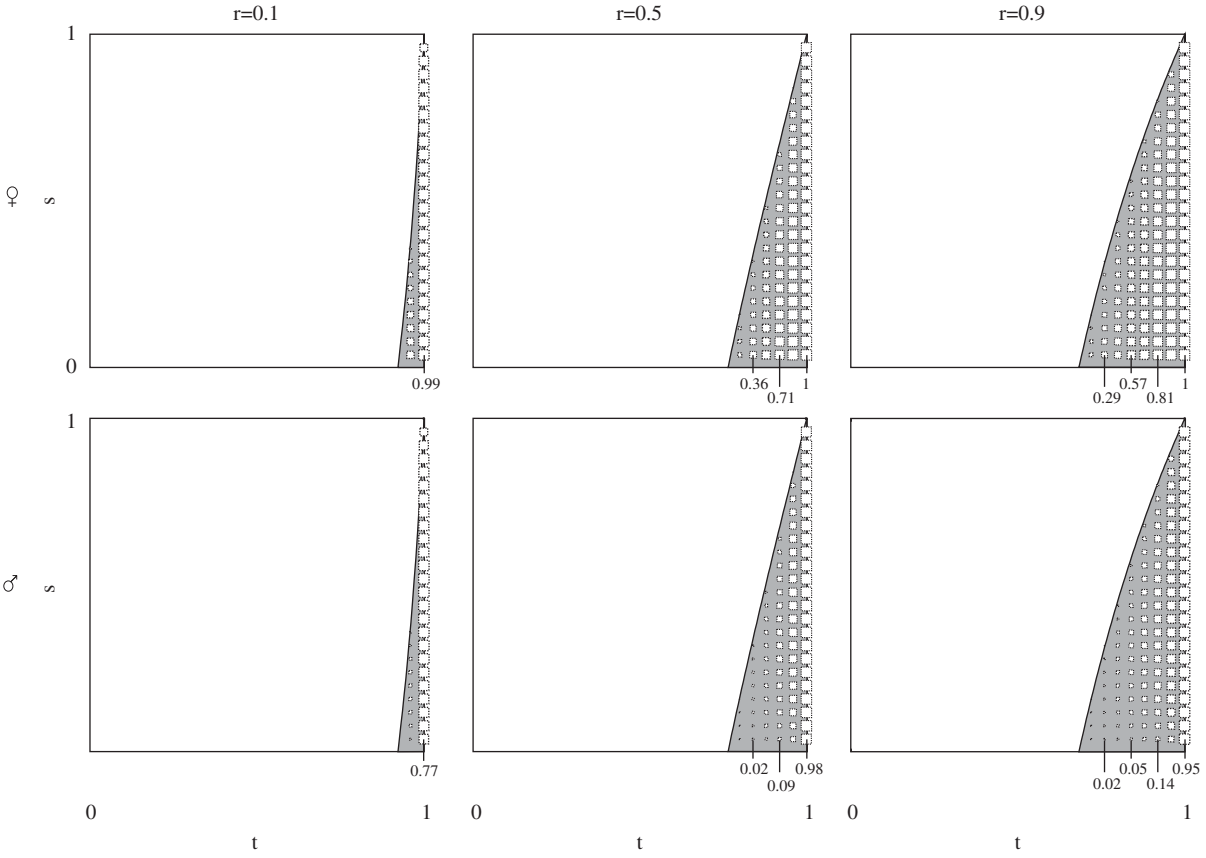


Fig. 2. Male haploidizer invasion condition. The shadowed area represents the pairs of values (t, s) that allow the invasion of a male haploidizing bacteria when r takes the values 0.1, 0.5 and 0.9. The area of each square represents the equilibrium frequency of infected females (top row) and infected males (bottom row). To illustrate the correspondence between area and equilibrium frequency the numeric value of the later has been provided for the bottom row in each graphic.

b_{ti} -infected males is y_o and y_{bi}, respectively ($0 \leqslant x_n, y_n \leqslant 1$ and $\sum_{n,i} x_n = \sum_{n,i} y_n = 1$). Their frequency one generation later is

$$\overline{m} y'_o = \overline{f} x'_o = x_o + \sum_i (1 - t_i) v_{bi} x_{bi}, \tag{7a}$$

$$\frac{\overline{m}}{s} y'_{bi} = \overline{f} x'_{bi} = t_i v_{bi} x_{bi}, \tag{7b}$$

where

$$\overline{f} = x_o + \sum_i v_{bi} x_{bi}, \tag{8a}$$

$$\overline{m} = x_o + \sum_i (1 - 2p_i) v_{bi} x_{bi} \tag{8b}$$

is the population mean fitness in females and males respectively.

Consider a population at equilibrium $\hat{\mathbf{z}}$ between uninfected and b_{t1}-infected hosts. Our interest lies in the fate of a rare allele b_{t2} that modifies the transmission ratio. The invasion condition for b_{t2} is

$$\left.\frac{\partial x'_{b2}}{\partial x_{b2}}\right|_{\hat{\mathbf{z}}} \equiv \frac{t_2 v_{b2}}{t_1 v_{b1}} > 1, \tag{9}$$

$$t_2 v_{b2} > t_1 v_{b1}. \tag{10}$$

This result relates to the one derived by Randerson et al. (2000) in the context of male killers.

Condition (10) implies that any mutant bacteria able to increase tv_b by modifying t, can enter the population. Since $\partial v_b/\partial t$ is positive by definition, $\partial t v_b/\partial t$ is also positive

$$\frac{\partial t v_b}{\partial t} = v_b + t \frac{\partial v_b}{\partial t} > 0 \tag{11}$$

and the only mutants that thrive are those incrementing t. Unless there is perfect transmission ($t = 1$), there will always be a mutant MH able to invade the population.

2.2.2. Insect control

This section models insects that can influence the vertical transmission of the bacteria they host. Let h_{t1} be an allele in insects (coding for transmission rate t_1) present in the population when a codominant allele h_{t2} (coding for survivorship rate t_2) arises. We require three letters to characterize the combination of genotypes within a host. Let the first letter correspond to the cytoplasmic genotype while the following two letters correspond to the nuclear genotype; in particular let the second letter refer to the maternally-inherited allele while the third letter refer to the paternally-inherited allele. For the particular case of infected males the first two letters suffice as the paternally-inherited genome is lost after haploidization.

Variability in the transmission rate brings along variability in the relative viability of offspring from infected females:

$$v_{nij} = \begin{cases} 1, & n = o, \\ 1 + r\frac{1}{2}(p_i + p_j)\left(1 - \frac{1}{2}(p_i + p_j)\right)^{-1}, & n = b, \end{cases} \quad i,j = \{1,2\}, \tag{12}$$

where $p_i = \frac{1}{2} t_i (1 - s)$ is the fraction of male zygotes with genotype h_{ti} killed in clutches of infected mothers.

Let the frequency of uninfected and infected females with genotype $h_{ti}h_{tj}$ be x_{oij} and x_{bij}, while the frequency of uninfected males with genotype $h_{ti}h_{tj}$ is y_{oij} and the frequency of infected males with genotype h_{ti} is y_{bi} ($0 \leqslant x_{nij}, y_{nij} \leqslant 1$ and $\sum_{n,i,j} x_{nij} = \sum_{n,i,j} y_{nij} = 1$). Their frequency one generation later is

$$2\overline{f} x'_{oij} = [\overline{o}(i) + \overline{b}(i) - \overline{bt}(i)] y(j) + [\overline{o}(j) + \overline{b}(j) - \overline{bt}(j)] y(i), \tag{13a}$$

$$2\overline{f} x'_{bij} = \overline{bt}(i) y(j) + \overline{bt}(j) y(i), \tag{13b}$$

$$2\overline{m} y'_{oij} = [\overline{o}(i) + \overline{b}(i) - \overline{bt}(i)] y(j) + [\overline{o}(j) + \overline{b}(j) - \overline{bt}(j)] y(i), \tag{13c}$$

$$\overline{m} y'_{bi} = s\overline{bt}(i), \tag{13d}$$

where

$$y(i) = \sum_{n,j} y_{nij} \tag{14}$$

is the frequency of allele h_{ti} in male insects,

$$\overline{o}(i) = \sum_j x_{oij} \tag{15a}$$

is the mean viability of allele h_{ti} in uninfected insects,

$$\overline{b}(i) = \sum_j v_{bij} x_{bij} \tag{16a}$$

is the mean viability of allele h_{ti} in infected insects,

$$\overline{bt}(1) = t_1 v_{b11} x_{b11} + \tfrac{1}{2}(t_1 + t_2) v_{b12} x_{b12} \tag{17a}$$

$$\overline{bt}(2) = t_2 v_{b22} x_{b22} + \tfrac{1}{2}(t_1 + t_2) v_{b12} x_{b12} \tag{17b}$$

is the mean viability of alleles h_{t1} and h_{t2} in infected hosts when transmitted together with bacteria, and

$$\overline{f} = \sum_i [\overline{o}(i) + \overline{b}(i)], \tag{18a}$$

$$\overline{m} = \sum_i [\overline{o}(i) + (1 - 2p_i)\overline{b}(i)] \tag{18b}$$

is the population mean fitness in female and male insects, respectively.

Consider a population at equilibrium $\hat{\mathbf{z}}$ between uninfected and infected hosts homozygous for allele h_{t1}. Our interest lies in the fate of a rare allele h_{t2} that modifies the transmission rate. Let $\mathbf{G}$ be the gradient matrix (see

Appendix for definition) of system (13):

$$\mathbf{G}=\begin{bmatrix} \frac{1}{2t_1v_{b11}} & \frac{(1-t_1)v_{b11}}{2(v_{b11}-1)} & \frac{(2-t_1-t_2)v_{b12}}{4t_1v_{b11}} & \frac{(1-t_1)v_{b11}}{2(v_{b11}-1)} \\ \frac{v_{b11}-1}{2\varphi t_1v_{b11}} & \frac{(1-t_1)v_{b11}}{2\varphi} & \frac{(2-t_1-t_2)(v_{b11}-1)v_{b12}}{4\varphi t_1v_{b11}} & \frac{(1-t_1)v_{b11}}{2\varphi} \\ 0 & \frac{t_1v_{b11}-1}{2(v_{b11}-1)} & \frac{(t_1+t_2)v_{b12}}{4t_1v_{b11}} & \frac{t_1v_{b11}-1}{2(v_{b11}-1)} \\ 0 & 0 & \frac{s(t_1+t_2)(v_{b11}-1)v_{b12}}{2\varphi t_1v_{b11}} & 0 \end{bmatrix}, \tag{19}$$

where $\varphi=(1-t_1)v_{b11}+(t_1v_{b11}-1)s$.

The invasion condition for h_{t2} requires that the leading eigenvalue of matrix $\mathbf{G}$, $\lambda_{\max}$, is greater than one, that is $\lambda_{\max}>1$. Because the analytic expression of $\lambda_{\max}$ (see Appendix) provides little insight we chose to present the results graphically. For viability function (1) we can plot the region of the parameter space (s,r,t_1,t_2) in which the invasion condition is satisfied (see Fig. 3). Fig. 3 shows that both increments ($t_2>t_1$) and decrements ($t_1>t_2$) of t may satisfy the invasion condition. Furthermore it can be inferred that a mutant allele coding for increments in t is most likely to invade when both s and r values are high but a mutant allele coding for decrements in t is most likely to invade when s and r values are low. This inference is

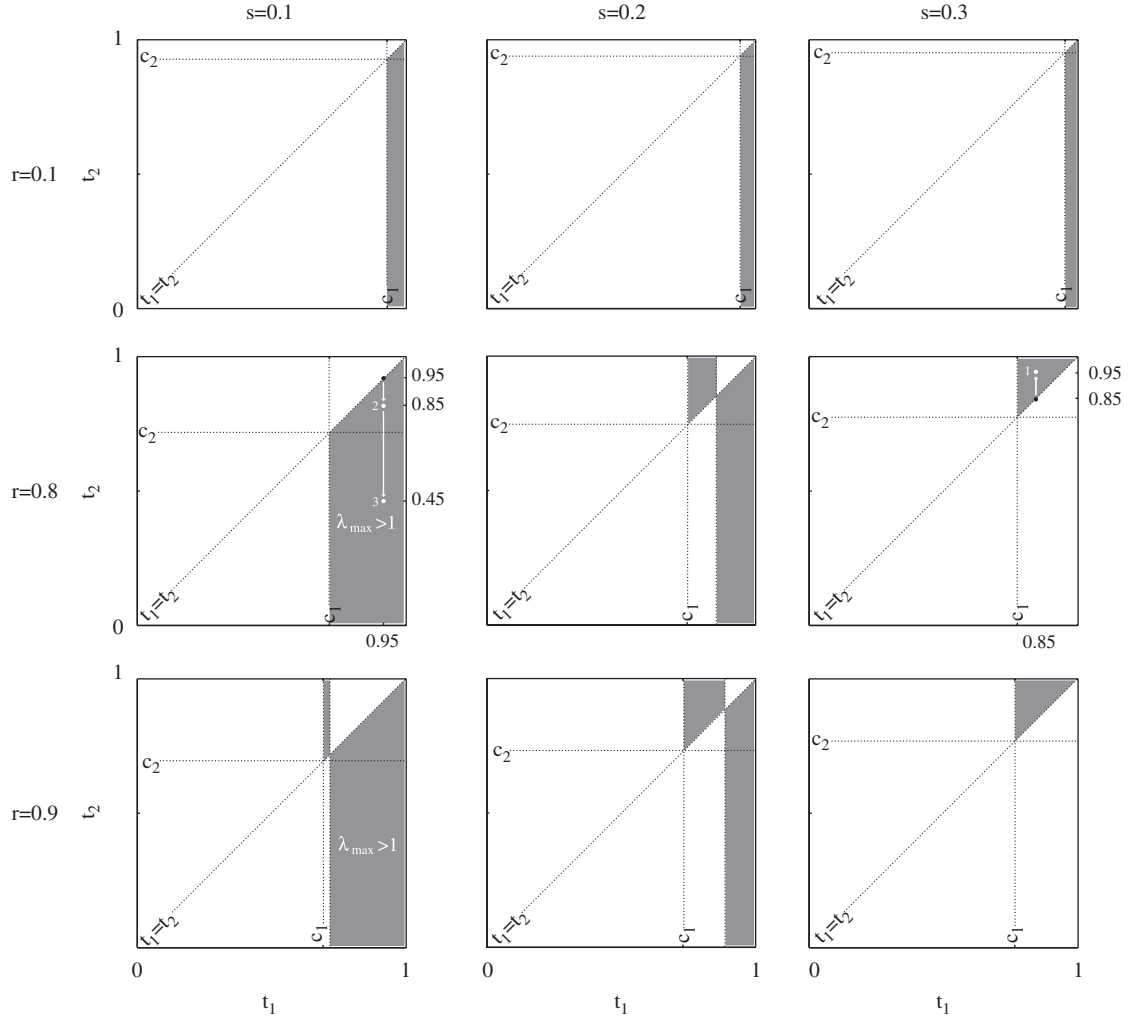


Fig. 3. Invasion conditions for modifiers of transmission. The shadowed area represents the values of t_2 such that a rare allele h_{t2} outcompetes a common allele h_{t1} coding for t_1. All combinations of $s=\{0.1,0.2,0.3\}$ and $r=\{0.1,0.8,0.9\}$ are explored. Lines representing MH invasion condition for t_1 (ic_1), t_2 (ic_2) and equal transmission $t_2=t_1$ are provided to illustrate that in all cases the shadowed area is perfectly limited by ic_1 and $t_2\gtrless t_1$.

supported by extensive numerical analysis; in particular we explored all combinations of t_1 and t_2 values in the range $[0.6, 1]$ and r and s values in the range $[0, 1]$ at intervals 0.05.

2.3. Variable survivorship

We follow by extending model (2) to allow variable survivorship. There are two possibilities: either the bacterium determines the survivorship of its insect host, or the host is the one determining its own survivorship.

2.3.1. Bacteria control

This section models bacteria that can influence the survivorship of haploidized insects hosting them. Let b_{s1} be an allele in bacteria (coding for survivorship rate s_1) that is present in the population when another allele b_{s2} (coding for survivorship rate s_2) arise. Let the frequency of uninfected and b_{si}-infected females be x_o and x_{bi}, while the frequency of uninfected and b_{si}-infected males is y_o and y_{bi}, respectively ($0 \leqslant x_n, y_n \leqslant 1$ and $\sum_{n,i} x_n = \sum_{n,i} y_n = 1$). Their frequency one generation later is

$$\overline{m} y'_o = \overline{f} x'_o = x_o + (1-t) \sum_i v_{bi} x_{bi}, \tag{20a}$$

$$\frac{\overline{m}}{s_i} y'_{bi} = \overline{f} x'_{bi} = t v_{bi} x_{bi}, \tag{20b}$$

where v_{bi}, $\overline{f}$ and $\overline{m}$ are given by Eqs. (6), (8a) and (8b) after p_i is redefined as $p_i = \frac{1}{2} t(1 - s_i)$.

The invasion condition for b_{s2} is

$$\left. \frac{\partial x'_{b2}}{\partial x_{b2}} \right|_{\widehat{\mathbf{z}}} = \frac{v_{b2}}{v_{b1}} > 1, \tag{21}$$

$$v_{b2} > v_{b1}. \tag{22}$$

This condition implies that any mutant bacteria able to increase v_b by modifying s can enter the population. Since $\partial v_b / \partial s$ is negative by definition, the only mutants that thrive are those reducing s. Therefore, unless MH causes the death of all infected males, there will always be a mutant bacteria more virulent than previous ones that can invade the population.

2.3.2. Insect control

This section models hosts that can influence their own survivorship after haploidization. Let h_{s1} be an allele in insects (coding for survivorship rate s_1) that is present in the population when an allele h_{s2} (coding for survivorship rate s_2) arise. Let the frequency of uninfected and infected females with genotype $h_{si}h_{sj}$ be x_{oij} and x_{bij}, while the frequency of uninfected males with genotype $h_{si}h_{sj}$ is y_{oij} and the frequency of infected males with genotype h_{si} is y_{bi} ($0 \leqslant x_{nij}, y_{nij} \leqslant 1$ and $\sum_{n,i,j} x_{nij} = \sum_{n,i,j} y_{nij} = 1$). Their frequency one generation later is

$$2\overline{f} x'_{oij} = \overline{o}(i)y(j) + \overline{o}(j)y(i) + (1-t)[\overline{b}(i)y(j) + \overline{b}(j)y(i)], \tag{23a}$$

$$2\overline{f} x'_{bij} = t[\overline{b}(i)y(j) + \overline{b}(j)y(i)], \tag{23b}$$

$$2\overline{m} y'_{oij} = \overline{o}(i)y(j) + \overline{o}(j)y(i) + (1-t)[\overline{b}(i)y(j) + \overline{b}(j)y(i)], \tag{23c}$$

$$\overline{m} y'_{bi} = s_i t \overline{b}(i), \tag{23d}$$

where $y(i)$, $\overline{o}(i)$, $\overline{b}(i)$, $\overline{f}$, $\overline{m}$ and v_{bi} are given by Eqs. (14), (15a), (16a), (18a), (18b) and (12) after p_i is redefined as $p_i = \frac{1}{2} t(1 - s_i)$.

Let $\mathbf{G}$ be the gradient matrix of system (23):

$$\mathbf{G} = \begin{bmatrix} \frac{1}{2tv_{b11}} & \frac{(1-t)v_{b11}}{2(v_{b11}-1)} & \frac{(1-t)v_{b12}}{2tv_{b11}} & \frac{(1-t)v_{b11}}{2(v_{b11}-1)} \\ \frac{v_{b11}-1}{2\varphi t v_{b11}} & \frac{(1-t)v_{b11}}{2\varphi} & \frac{(1-t)(v_{b11}-1)v_{b12}}{2\varphi t v_{b11}} & \frac{(1-t)v_{b11}}{2\varphi} \\ 0 & \frac{tv_{b11}-1}{2(v_{b11}-1)} & \frac{v_{b12}}{2v_{b11}} & \frac{tv_{b11}-1}{2(v_{b11}-1)} \\ 0 & 0 & \frac{s_2(v_{b11}-1)v_{b12}}{\varphi v_{b11}} & 0 \end{bmatrix}, \tag{24}$$

where $\varphi = (1-t)v_{b11} + (tv_{b11} - 1)s_1$. The invasion condition for h_{s2} requires that the leading eigenvalue of matrix $\mathbf{G}$, $\lambda_{\max}$, is greater than one, that is $\lambda_{\max} > 1$. Once more the analytic expression of $\lambda_{\max}$ (see Appendix) provides little insight and we chose to present the results graphically. For viability function (1) we can plot the region of the parameter space (t, r, s_1, s_2) in which the invasion condition is satisfied (see Fig. 4). Fig. 4 allows us to infer a simple invasion condition, namely:

$$s_2 > s_1. \tag{25}$$

This inference is supported by extensive numerical analysis; in particular we explored all combinations of t values in the range $[0.6, 1]$ and r, s_1 and s_2 values in the range $[0, 1]$ at intervals 0.05. Therefore, unless there is perfect survivorship there will always be a mutant allele coding for greater survivorship that can invade the population.

3. Evolutionary dynamics

So far, we have derived the conditions that allow a rare allele to invade a population. These conditions, however, say little about the behavior of the system once the allele in question becomes abundant. To explore the behavior of the models presented in this research we iterate the set of recursive equations that describe each of them using a script written in Mathematica (Wolfram Research, 2004). In particular, we explored all combinations of t_1 and t_2 in the range $[0.6, 1]$ and r, s_1 and s_2 in the range $[0, 1]$ at intervals 0.1.

We present an example for each kind of evolutionary dynamics we found (see Fig. 5). Starting with the model that allows the evolution of transmission when this

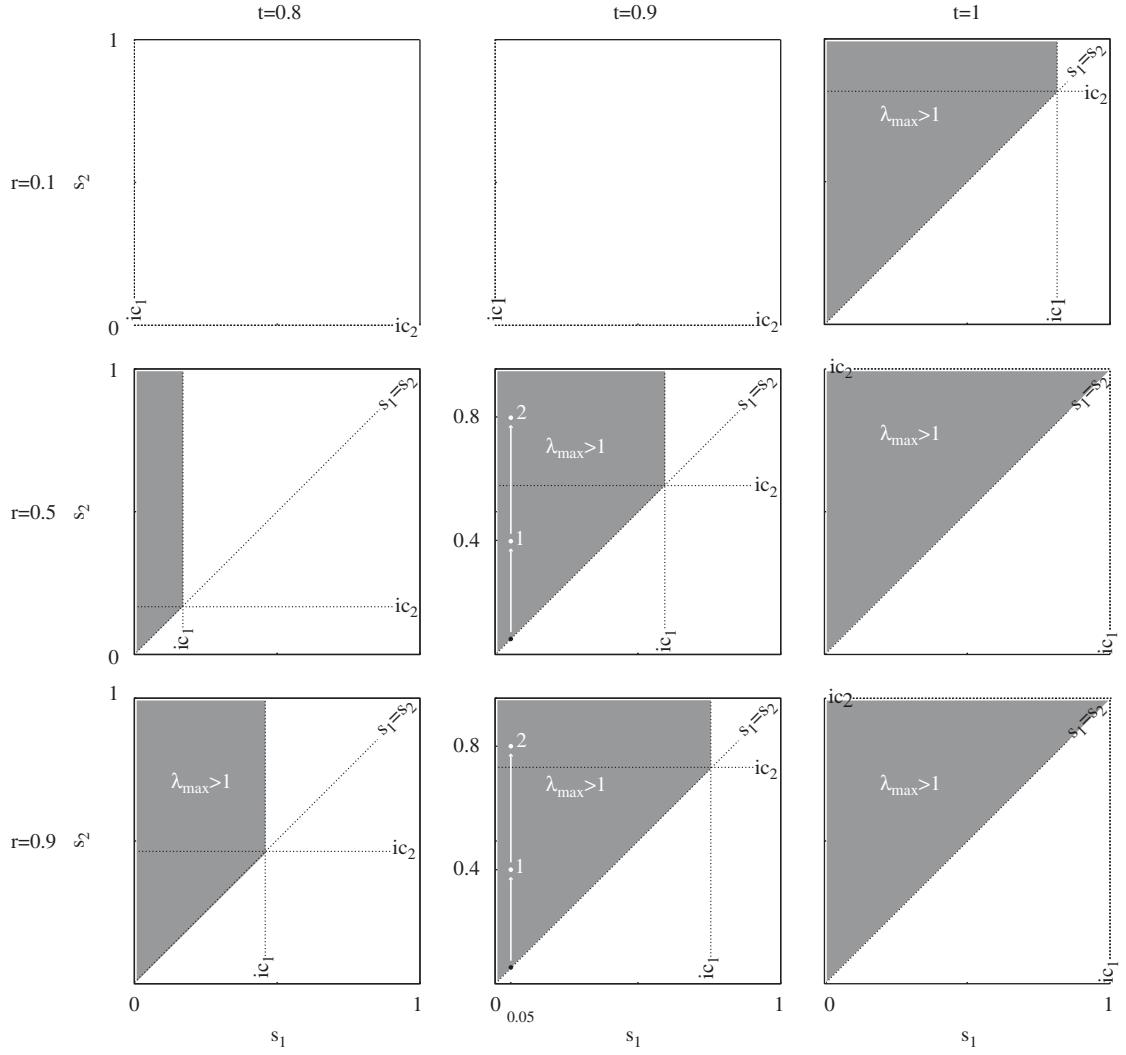
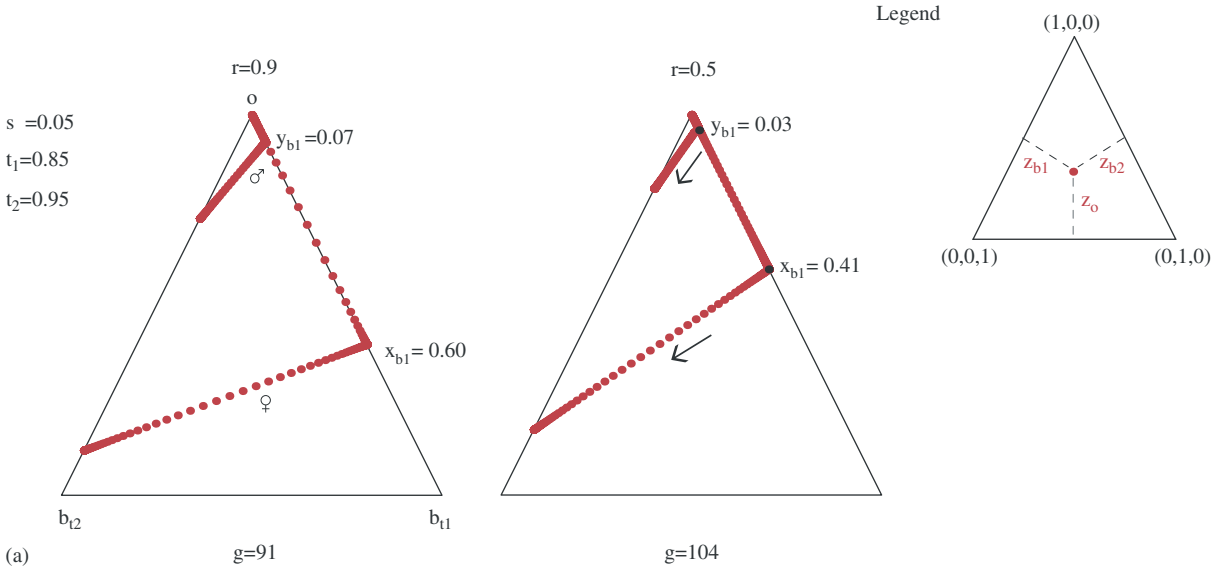
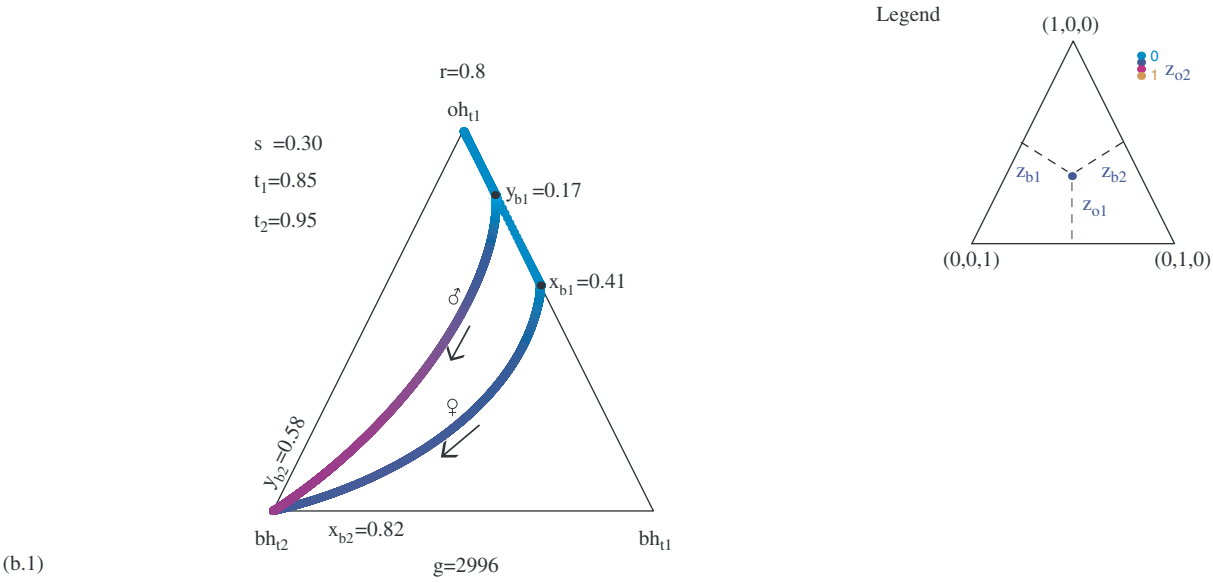


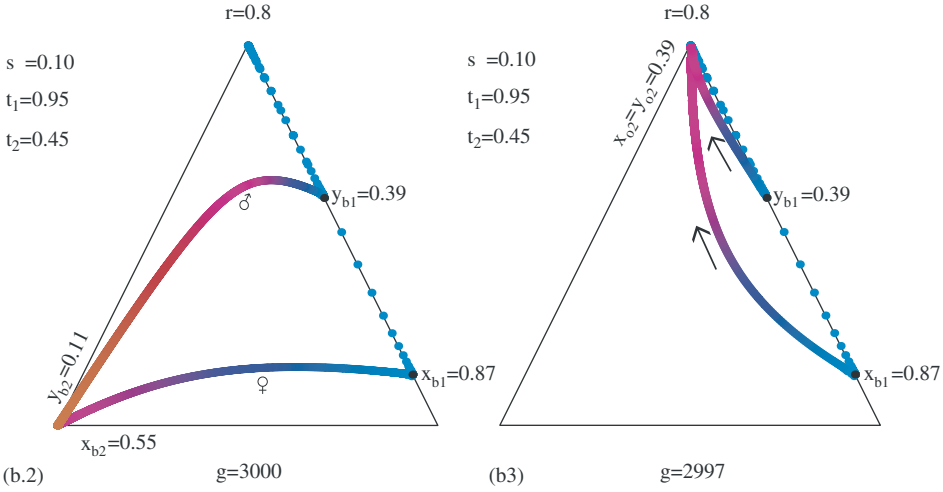
Fig. 4. Invasion conditions for modifiers of survivorship. The shadowed area represents the values of s_2 such that a rare allele h_{s2} outcompetes a common allele h_{s1} coding for s_1. All combinations of $t = \{0.8, 0.9, 1\}$ and $r = \{0.1, 0.5, 0.9\}$ are explored. Lines representing MH invasion condition for s_1 (ic_1), s_2 (ic_2) and equal survivorship $s_2 = s_1$ are provided to illustrate that in all cases the shadowed area is perfectly described by ic_1 and $s_2 > s_1$.

phenotype is controlled by bacterial genotype, in all cases in which a mutant bacteria meets the invasion condition $t_2 > t_1$, allele b_{t2} increases steadily and becomes fixed in the population (Fig. 5a). When transmission is under host control, however, there are multiple alternatives. Depending on the values of s and r, the invasion condition may require

Fig. 5. Evolutionary dynamics. Each point inside a triangle represents a trio of haplotype frequencies given by the perpendicular distance to bottom, left and right sides of the triangle. In addition a color code has been assigned to each point in b and d representing a fourth frequency (see the legends to identify the correspondence between frequency and distance or color). A point corresponds to the frequency of the different classes of male or female insects (as indicated by the male and female symbols). A sequence of points depicts the change in frequency over time starting with the absence of infected insects in all cases (top vertex of the triangle) and progressing in the direction indicated by the arrows. Variable transmission. (a) Each figure represents: 1. the change in frequency over time of a rare allele b_{t1} in a population without bacteria; 2. the change in frequency over time of alleles b_{t1} and b_{t2} in a population at equilibrium $(\widehat{x}_b, \widehat{y}_b)$ between uninfected and infected insects. The number of generations between $(\widehat{x}_b, \widehat{y}_b)$ and the new equilibrium is provided at the base of the triangle. (b) Each figure represents: 1. the change in frequency over time of a rare allele bh_{t1} in a population without bacteria; 2. the change in frequency over time of haplotypes oh_{t1}, oh_{t2}, bh_{t1} and bh_{t2} in a population at equilibrium $(\widehat{x}_{b1}, \widehat{y}_{b1})$ between uninfected and infected insects. (b.1) t increments. Parametrization corresponds to point 1 in Fig. 3. (b.2) t decrements and condition $t_2 v_{b2} > 1$ satisfied. Parametrization corresponds to point 2 in Fig. 3. (b.3) t decrements and condition $t_2 v_{b2} > 1$ not satisfied. Parametrization corresponds to point 3 in Fig. 3. In (b.1) and (b.2) the system converges towards a polymorphism between uninfected and infected insects while host allele h_{t2} goes to fixation. In (b.3) infected insects become extinct while host alleles h_{t1} and h_{t2} reach a polymorphic equilibrium. Variable survivorship. (c) Each figure represents: 1. the change in frequency over time of a rare allele b_{s1} in a population without bacteria; 2. the change in frequency over time of alleles b_{s1} and b_{s2} in a population at equilibrium $(\widehat{x}_b, \widehat{y}_b)$ between uninfected and infected insects. (d) Each figure represents: 1. the change in frequency over time of a rare allele bh_{s1} in a population without bacteria; 2. the change in frequency over time of haplotypes oh_{s1}, oh_{s2}, bh_{s1} and bh_{s2} in a population at equilibrium $(\widehat{x}_{b1}, \widehat{y}_{b1})$ between uninfected and infected insects. Condition $tv_{b2} > 1$ is satisfied by the parametrization in (d.1) but not in (d.2). In (d.1), the new equilibrium corresponds to a polymorphism between uninfected and infected insects while host allele h_{s2} goes to fixation. In (b.2) infected insects become extinct while host alleles h_{s1} and h_{s2} reach a polymorphic equilibrium.


Legend
(1,0,0)
z_{b1}
z_{b2}
z_o
(0,0,1)
(0,1,0)
r=0.9
o
s =0.05
t_1=0.85
t_2=0.95
y_{b1}=0.07
♂
x_{b1}= 0.60
♀
b_{t2}
b_{t1}
(a)
g=91
r=0.5
y_{b1}= 0.03
x_{b1}= 0.41
g=104


Legend
(1,0,0)
0
1
z_{o2}
z_{b1}
z_{b2}
z_{o1}
(0,0,1)
(0,1,0)
r=0.8
oh_{t1}
s =0.30
t_1=0.85
t_2=0.95
y_{b1}=0.17
x_{b1}=0.41
♂
♀
y_{b2}=0.58
bh_{t2}
x_{b2}=0.82
bh_{t1}
(b.1)
g=2996


r=0.8
s =0.10
t_1=0.95
t_2=0.45
y_{b1}=0.39
♂
y_{b2}=0.11
♀
x_{b1}=0.87
x_{b2}=0.55
(b.2)
g=3000
r=0.8
s =0.10
t_1=0.95
t_2=0.45
x_{o2}=y_{o2}=0.39
y_{b1}=0.39
x_{b1}=0.87
(b3)
g=2997

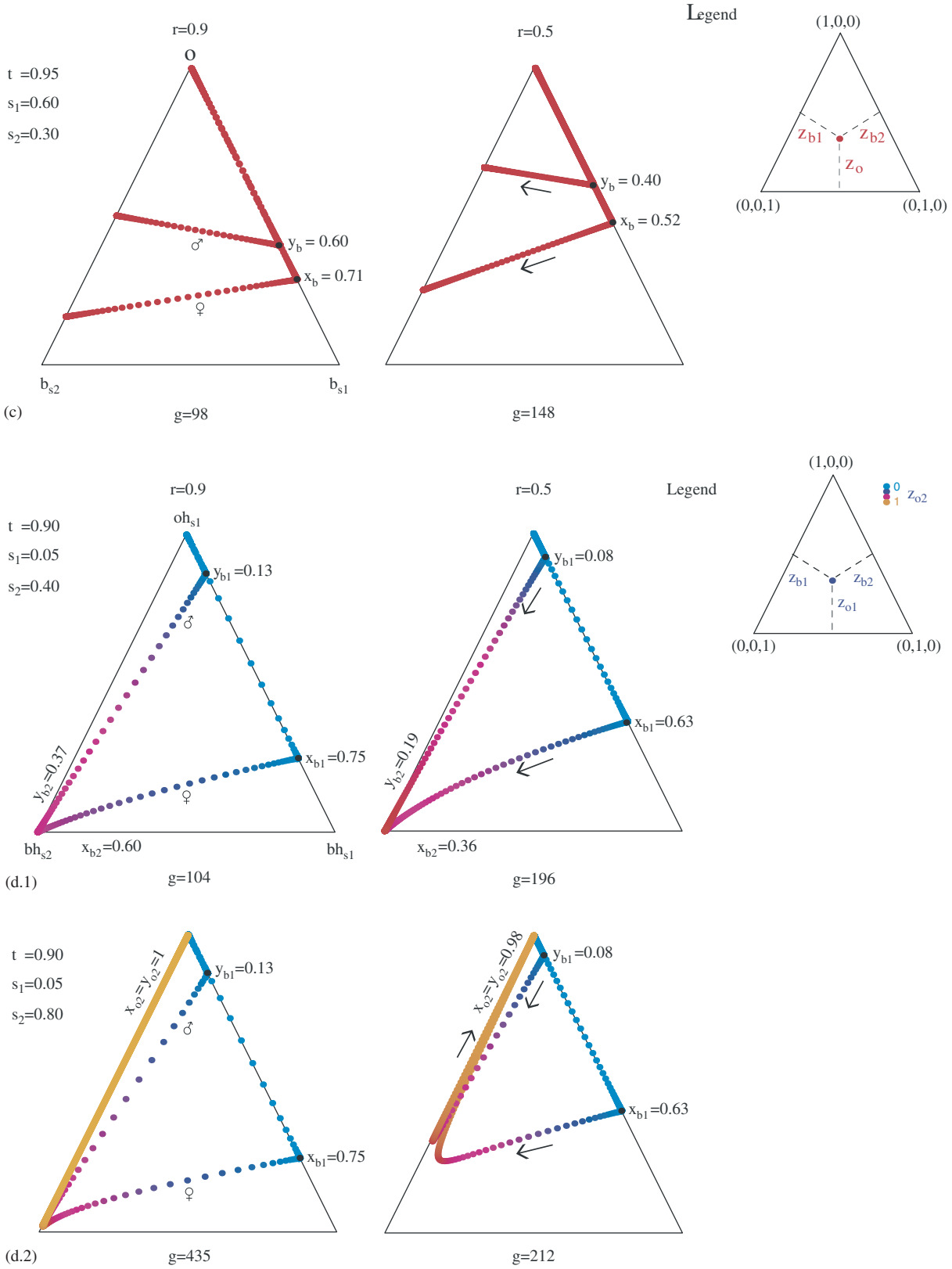


Fig. 5. (*Continued*)

increments of t ($t_2 > t_1$) or decrements of t ($t_2 < t_1$). In both these cases allele h_{t2} increases steadily, becoming fixed in the population (Fig. 5b.1 and b.2). If the reduction in t such that the invasion condition for bacteria ($t_2 v_{b22} > 1$) is now violated, allele h_{t2} drives the bacterial population to extinction before it can become fixed in the host population (Fig. 5b.3).

For the model with variable survivorship under bacterial control, in all cases in which a mutant bacteria meets the invasion condition $s_1 > s_2$, allele b_{s2} increases steadily and becomes fixed in the population (Fig. 5c). When survivorship is under host control, the satisfaction of invasion condition $s_2 > s_1$ guarantees that allele h_{s2} increases in frequency when rare. If survivorship rate s_2 is such that the invasion condition for bacteria ($tv_{b22} > 1$) is still satisfied, allele h_{s2} becomes fixed in the population (Fig. 5d.1). If, however, survivorship rate s_2 is such that the invasion condition for bacteria is now, violated allele h_{s2} drives the bacterial population to extinction before it can become fixed in the host population (Fig. 5d.2).

4. Expected outcome from co-evolution

After MHs have entered a population, bacterial transmission and host survivorship are under selective pressures that depend on which of these characters are under control of cytoplasmic as opposed to nuclear genes. In previous sections we have shown that cytoplasmic genes are selected to increase their own transmission but to decrease male host survivorship. Nuclear genes are selected to either increase or decrease bacterial transmission (depending on the initial conditions) and increase host survivorship. When considering the co-evolution of transmission and survivorship there are four possibilities, namely: (1) bacterial control of transmission and survivorship, (2) bacterial control of transmission and host control of survivorship, (3) host control of transmission and bacterial control of survivorship, and (4) host control of transmission and survivorship.

Bacterial control of survivorship leads to either the extinction of the population due to the death of all males (Fig. 6a) or the extinction of MH bacteria (Fig. 6c). Because cytoplasmic genes controlling the survivorship of haploidized males would be self-destructive we expect that survivorship is mainly under control of nuclear genes. This supports the assumption made in the formulation of the haploidizing male killer hypothesis (Normark, 2004). Assuming that nuclear genes control survivorship, whether transmission is controlled by nuclear or cytoplasmic genes does not change significantly the outcome of the co-evolutionary process; either MH bacteria become extinct or fixed (Fig. 6b and d). In the next paragraphs we will focus on the particular case of nuclear genes controlling survivorship and cytoplasmic genes controlling transmission.

There are two routes to PGE. The first scenario is one in which condition $tv_b(t,s) > 1$ is not violated along the co-evolutionary path (Figs. 6b.1). Any MH for which $t_0 v_b(t_0, s_0) > 1$, can invade a wild-type host population. In due course, this invasion opens up the possibility that a mutant host for which $s_1 > s_0$ and $t_0 v_b(t_0, s_1) > 1$ enters the population. This is followed by a second invasion of a mutant MH for which $t_1 > t_0$ and $t_1 v_b(t_1, s_0) > 1$ (the order in which mutants arise is arbitrary and does not make any difference to our arguments). This process repeats until perfect transmission and survivorship is reached, which is phenotypically equivalent to PGE. Since PGE shows EGS, the evolutionary process stops here unless a mutation in parameters other than the ones considered in this research occurs.

The second scenario occurs when condition $tv_b(t,s) > 1$ is violated along the co-evolutionary path (Fig. 6b.2). Consider the same process as above but this time at some point, i, a mutant host for which $s_{i+1} > s_i$ and $t_i v_b(t_i, s_{i+1}) < 1$ invades, driving MH to extinction. The outcome is a population of diploid insects at a polymorphic equilibrium between s_i and s_{i+1}. This population does not show EGS because can be invaded by some MH as long as $t_{i+1} v_b(t_{i+1}, s_{i+1}) > 1$. This process repeats until PGE is reached and evolution stops. From Fig. 6b.2 it becomes evident that the greater s_{i+1} is, the more restrictive the condition $t_{i+1} v_b(t_{i+1}, s_{i+1}) > 1$ is, and the less likely the right MH mutant arises. Ultimately, if s_{i+1} corresponds to perfect survivorship no MH can invade the population and the evolutionary process stops.

Excluding the very special case $s_{i+1} = 1$ in the absence of MHs, PGE is the only pair of (t,s) values that shows EGS. Therefore, if hosts are in control of their survivorship PGE will eventually evolve. How fast PGE will evolve depends on whether the host also controls transmission, and on the speed at which bacteria and host evolve. If nuclear genes control transmission, or cytoplasmic genes control transmission but the bacteria evolve faster than their hosts, the evolution of PGE may slow down.

5. Discussion

Our work advances the critical analysis of the haploidizing male killer hypothesis. What is novel about our research is that it formally models the selective forces acting on both MH transmission and host survivorship. We conclude that once MHs enter a population, selection may act to maximize MH transmission and host survivorship, thereby originating PGE. Once PGE has been reached it shows EGS and the population remains at this equilibrium.

The evolution of PGE due to the action of MHs is conditional upon the invasion of MHs in first place. The invasion of MHs requires that inequality $tv_b > 1$ is satisfied. The probability that this condition will be satisfied by some mutant MH greatly depends on the value assigned to r. While Normark (2004) fails to consider condition $tv_b > 1$ altogether, Engelstädter and Hurst (2005) focus their discussion in the lower end of the range of values that r may take (in particular $r = 0.1$ and 0.2). When such values are used, the probability that MHs can invade appears to be low, though that may not be the case in reality. Below we discuss the plausibility of sibling competition in the insect lineages in which PGE arose, and how insect ecology allows (if not favors) considering high values of r. But first we need to emphasize that in this research, we assume that

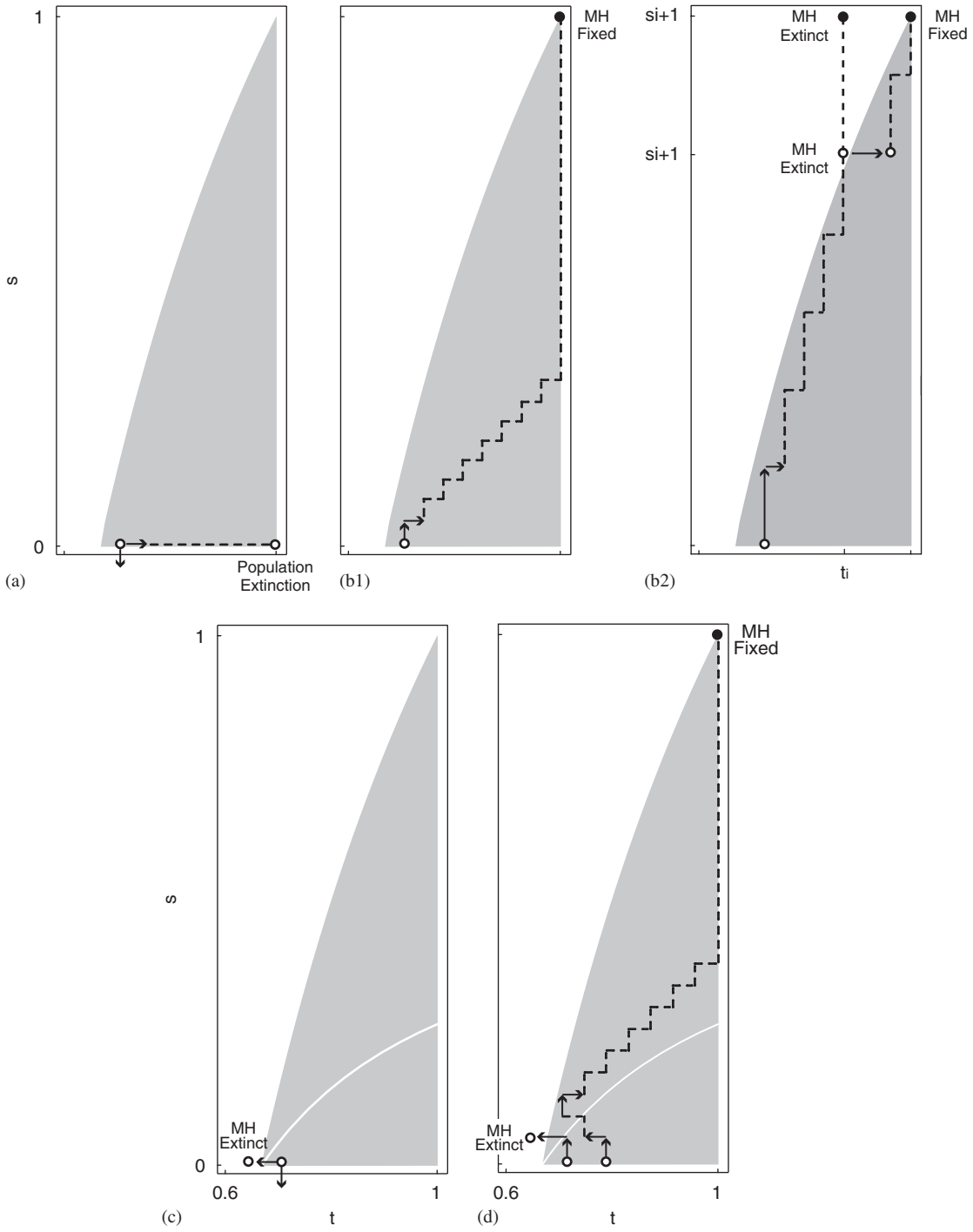


Fig. 6. Co-evolutionary paths. The shadowed area represents the pairs of values (t, s) that allow the invasion of a male haploidizing bacteria when r is 1. Solid arrows indicate the direction of selection acting on transmission and survivorship. Solid white line indicates the limit of the area where selection acting on nuclear genes controlling transmission swaps from favoring increments to favoring decrements. Open and filled circles represent combinations of s and t showing evolutionary genetic stability or not, respectively. There are four possibilities: (a) bacteria control transmission and survivorship, (b) bacteria control transmission and host controls survivorship, (c) host controls transmission and bacteria control survivorship, (d) host controls transmission and survivorship. The possible outcomes of a co-evolutionary process between cytoplasmic and nuclear genes in the second case are as follows. (b.1) (t, s) do not enter the grey area along their co-evolutionary path (dashed line); they co-evolve until perfect transmission and survivorship, that is PGE, is reached. (b.2) (t, s) do enter the grey area along their co-evolutionary path (dashed line); when they enter the grey area (t_i, s_{i+1}) MH bacteria become extinct. However (t_i, s_{i+1}) does not show evolutionary stability (except in the rare case in which $s_{i+1} = 1$). A MH bacteria with transmission rate sufficiently greater than t_i may arise and invade the population until perfect transmission and survivorship is reached.

the death of a zygote results in a compensatory increase in viability of other zygotes in the same clutch (following Engelstädter and Hurst, 2005; Hurst, 1991). This assumption differs from the original formulation of the MH hypothesis, which assumed that the death of a zygote results in a compensatory increase in maternal production of zygotes (Normark, 2004). These two ideas are conceptually different and the viability functions describing each of them ($v_b = 1 + rp(1-p)^{-1}$ and $v_b = 1 + rp\sum_{n=0}^{k} p^n$), respectively, are equivalent only when the number of times a mother can produce new zygotes to replace the dead ones (k) tends to infinity. Given a compensatory increase in viability of zygotes, the compensation can have its biological origin in a reduction of sibling competition for finite resources or in the cannibalism of dead zygotes (Freeland and McCabe, 1997).

In the ancestral ecological conditions of all four PGE insect lineages—to the extent that these can be reconstructed—there is a strong tendency for eggs to be laid in clutches and for siblings to develop in proximity to each other. In three of the four clades (*Hypothenemus* beetles, Cecidomyiidae, and Sciaridae) fitness compensation may take place through a reduction in sibling competition for a limited amount of food. The likely ancestral habit of Cecidomyiidae and Sciaridae is to lay eggs in cavities in rotting wood where larvae feed on fungi (Hamilton, 1978; see references in Normark, 2003, 2004). *Hypothenemus* beetles and some other PGE beetles in the same subfamily lay eggs in clutches that develop gregariously in small plant parts such as seeds (Kirkendall, 1993). However, competition for food might not be the only source of fitness compensation in these clades. Many beetles feeding in rotting wood—including many close relatives of *Hypothenemus* in the bark beetle subfamily Scolytinae—are facultatively cannibalistic, consuming unhatched eggs (Beaver, 1974; Norris and Chu, 1985; Pollock and Normark, 2002; Schenk and Benjamin, 1969; Weber and McPherson, 1983).

In the other PGE insect clade—the neococcoid scale insects—fitness compensation may take place through a reduction in sibling competition not for a limited amount of food but for space and shelter. In contrast to the three other clades, for scale insects the source of food is typically phloem sap, which is unlikely to be a limiting resource. However, scale insect embryos typically develop in a brood chamber—in or under the mother's body, in a gall, or in a wax enclosure (Koteja, 1990). Most adult female scale insects are completely sedentary; therefore, all of a female's eggs are deposited in one spot and progeny must develop there until they can walk away. The limiting resources driving competition are, probably, space inside the brood chamber and protection in bark crevices. The highly specialized sap-sucking mouthparts of many scale insects make cannibalism unlikely.

Sibling competition for food, space, or shelter may result in values of r close to one. For example consider the offspring of bark beetles competing with each other to eat a fraction of the seed the clutch was laid in. The death of a zygote frees entirely the fraction of seed this zygote would have eaten had it not been killed, i.e. $r \approx 1$. If for any reason the offspring dies not as a zygote but at a later stage, the fraction of seed made available to its siblings would be reduced, i.e. $r < 1$. However, it is reasonable to think that MH would act early in development, as soon as the male genome enters the cytoplasm. On the other hand, cannibalism of dead zygotes may result in low values of r. For example the cannibalization of a dead zygote by other bark beetles in the same clutch allows to recover through direct ingest some of the resources the mother of that zygote could have deposited in the eggs of its siblings straight away. Due to nutritional inefficiency the fraction of resources thereby recovered might be low. However, this is not necessarily true if proteins are deficient in the diet of juveniles and necessary at some point in their development. Since parameter r summarizes all these possibilities, natural history justifies the consideration of the full range of values r can take. Not wanting to favor one range of r values over the other we think it is worth considering the invasion condition for a representative set of values (see Fig. 2) and not for the low range of values in Engelstädter and Hurst (2005). While the probability of invasion of MHs is more restricted than suggested by Normark (2004) it is not necessarily as low as suggested in the graphics provided by Engelstädter and Hurst (2005).

Once a MH that satisfies condition (5) arises, coevolution between bacteria and insects leads either to PGE or to extinction of MHs. While in the first case PGE shows EGS and evolution stops there, in the second case evolution carries on. Eventually, a second MH such that condition (5) is satisfied may evolve. The greater the survivorship of males in the rare events in which they are haploidized, the lower the probability a second MH evolves. In our model survivorship will not be under selective pressure once the first wave of MHs becomes extinct; in nature, however, it is possible that the expression of such gene is costly both before and after the extinction of MHs. If this is the case survivorship of haploidized males would decrease once the first wave of MHs has gone to extinction thereby facilitating the invasion of a second wave of MHs. In future research it would be worth modelling costly survivorship genes.

Another facet of the system that needs to be explored in future modelling is the genetics of sex determination. Many previous discussions of the origins of PGE have postulated genetic mechanisms in ancestral diploid-male populations that would cause haploid individuals to develop as males (Bull, 1979; Haig, 1993a; Burt and Trivers 2006). The presence of such predisposing genetic mechanisms for maleness of haploids may be less important than previously thought, if MH bacteria are involved in the origins of PGE. This is because MH bacteria would benefit from either non-viability or femaleness of haploids, and maleness as well as viability of haploids could thus be interpreted as be a late countermove by the host, rather than as a necessary

predisposing condition. Thus, our intuition is that MH elements could invade under a wide range of possible ancestral sex determination mechanisms and that in this sense our model is complementary to many previous genetic models of the origins of PGE. But more extensive modelling, involving explicit genetic models of sex determination,would be a necessary part of testing this hypothesis.

Another important area for future work is the potential interaction between MH cytoplasmic elements and genomic imprinting in the origin and evolution of PGE lineages. Our model does not address the mechanism whereby the paternally-derived chromosomes are identified and destroyed by the endosymbiont. The most plausible class of mechanisms might be those in which male-determining sperm are targeted as they pass through the cytoplasm en route to the nucleus. And yet some (if not all) extant PGE lineages have a much more subtle intra-nuclear mechanism of genomic imprinting in which maternally-derived and paternally-derived chromosome regions are marked by different patterns of DNA methylation and/or histone acetylation (Bongiorni et al., 1999; Bongiorni and Prantera, 2003; Ferraro et al., 2001; Goday and Ruiz, 2002). Perhaps this genomic imprinting is one of the late-arising "redundant mechanisms to ensure the elimination of the paternal genome" postulated above. An alternative hypothesis was suggested recently by Normark (2006), who argued that the sibling competition and cannibalism seen in many arthropods could lead to the evolution of parent-specific gene expression similar to that seen in mammals and angiosperms (Haig, 2000; Burt and Trivers, 2006). The evolutionary dynamics of maternally vs. paternally-derived alleles would thus be similar to those seen in mammalian pregnancy, with "greedier" paternally-derived alleles and more "abstemious" maternally-derived alleles. In arthropods, these interactions would be mediated behaviorally between immature siblings (e.g. by larval sib cannibalism) rather than mediated biochemically between mother and embryo, as in mammals and angiosperms. In the models presented in this paper, we have assumed that maternal and paternal alleles have equivalent fitness consequences for endosymbionts and equivalent viability consequences for hosts. But if paternal alleles are " greedier" than maternal alleles, e.g. if there is a paternally-imprinted tendency to be cannibalistic, then cytoplasmic bacteria may still benefit from stable PGE even if haploid-male viability is 1. Such an effect could considerably alter the evolutionary dynamics in ways favorable to the invasion of MH elements.

Our model has focused on the origin, spread, and establishment of PGE; we have not considered the future evolution of this system. In extant lineages characterized by PGE, many evolutionary events may have occurred between the events modelled here and the present day. For instance, once PGE is established, it is likely to become an integral part of the host's system of sex determination and development, such that any disruption of the system would be deleterious or lethal. Thus, the loss of endosymbionts for any reason (such as the consumption of antibiotic-secreting fungi, high temperature, or any other environmental accident) could be highly deleterious. Such episodes would place other loci in the host's nuclear genome under selection to buffer the PGE system against loss of the symbionts by evolving redundant mechanisms to ensure the elimination of the paternal genome (including, perhaps, genomic imprinting).

The tendency of maternally-transmitted endosymbionts to lose most of their genes—by simple deletion or by transfer to the host's genome (Moran, 2003; Wernegreen, 2002)—should make it more likely that the mechanics of PGE eventually be taken over by the host's genome in ancient associations. Thus, this model can best be tested in (a) the most recent origins of PGE, such as that in Hypothenemus, and possibly in other poorly-investigated scolytine beetles with highly biased sex ratios, such as occur in the genera *Cryptocarenus*, *Trischidias*, *Periocryphalus*, *Sueus*, *Bothrosternus*, and *Araptus* (Kirkendall, 1993), and in (b) the most evolutionarily dynamic PGE systems, such as those in scale-insects in which expression and transmission of the paternal genome has re-evolved (Normark, 2003). Another possible route to stabilization of the male-haploidy system would be the evolution of PGE into arrhenotoky, with no paternal contribution to males at all and the development of males directly from unfertilized eggs. The ecological distribution of arrhenotoky is broadly similar to that of PGE (Normark, 2003, 2004); that and the cytogenetic similarity make a common origin for the two systems seem likely (Schrader and Hughes-Schrader, 1931).

Acknowledgments

We thank A. Grafen, J. Werren, J. Engelstädter, G.D.D. Hurst, M.E. Gruwell, R. Gwiazdowski and D.N. Tran for comments on the manuscript. F.U. Thanks Peter Donnelly for support. This work was supported by a Junior Research Fellowship from St. John's College at Oxford, by the USDA Cooperative State Research, Education, and Extension Service (2002-35302-12479), and by the N.S.F. (DEB-0447880).

Appendix

Let matrix **G** be:

$$\mathbf{G}=\left[\begin{array}{cccc}\frac{\partial x'_{o12}}{\partial x_{o12}} & \frac{\partial x'_{o12}}{\partial y_{o12}} & \frac{\partial x'_{o12}}{\partial x_{b12}} & \frac{\partial x'_{o12}}{\partial y_{b2}}\\ \frac{\partial y'_{o12}}{\partial x_{o12}} & \frac{\partial y'_{o12}}{\partial y_{o12}} & \frac{\partial y'_{o12}}{\partial x_{b12}} & \frac{\partial y'_{o12}}{\partial y_{b2}}\\ \frac{\partial x'_{b12}}{\partial x_{o12}} & \frac{\partial x'_{b12}}{\partial y_{o12}} & \frac{\partial x'_{b12}}{\partial x_{b12}} & \frac{\partial x'_{b12}}{\partial y_{b2}}\\ \frac{\partial y'_{b2}}{\partial x_{o12}} & \frac{\partial y'_{b2}}{\partial y_{o12}} & \frac{\partial y'_{b2}}{\partial x_{b12}} & \frac{\partial y'_{b2}}{\partial y_{b2}}\end{array}\right]\Bigg|_{\hat{\mathbf{z}}}.$$

Matrix **G** has the following structure:

$$\mathbf{G} = \begin{bmatrix} g_{11} & g_{12} & g_{13} & g_{12} \\ g_{21} & g_{22} & g_{23} & g_{22} \\ 0 & g_{32} & g_{33} & g_{32} \\ 0 & 0 & g_{43} & 0 \end{bmatrix}. \tag{26}$$

Its characteristic polynomial is given by the expression:

$$\begin{vmatrix} g_{11}-\lambda & g_{12} & g_{13} & g_{12} \\ g_{21} & g_{22}-\lambda & g_{23} & g_{22} \\ 0 & g_{32} & g_{33}-\lambda & g_{32} \\ 0 & 0 & g_{43} & -\lambda \end{vmatrix} = 0, \tag{27}$$

which is a quadratic polynomial in λ.

Substituting the second column by the second column minus the fourth column

$$\begin{vmatrix} g_{11}-\lambda & 0 & g_{13} & g_{12} \\ g_{21} & -\lambda & g_{23} & g_{22} \\ 0 & 0 & g_{33}-\lambda & g_{32} \\ 0 & \lambda & g_{43} & -\lambda \end{vmatrix} = 0. \tag{28}$$

We can expand the above determinant using the second column. It becomes obvious that $\lambda_4 = 0$ is the first eigenvalue while the other three come from solving

$$\begin{vmatrix} g_{11}-\lambda & g_{13} & g_{12} \\ g_{21} & g_{23} & g_{22} \\ 0 & g_{33}-\lambda & g_{32} \end{vmatrix} - \begin{vmatrix} g_{11}-\lambda & g_{13} & g_{12} \\ 0 & g_{33}-\lambda & g_{32} \\ 0 & g_{43} & -\lambda \end{vmatrix} = 0, \tag{29}$$

which is a cubic polynomial in λ. In particular:

$$\lambda^3 + c_2\lambda^2 + c_1\lambda + c_0 = 0, \tag{30}$$

where

$$c_2 = -g_{11} - g_{22} - g_{33}, \tag{31a}$$

$$c_1 = g_{11}g_{22} + g_{11}g_{33} + g_{22}g_{33} - g_{12}g_{21} - g_{32}(g_{23} + g_{43}), \tag{31b}$$

$$c_0 = g_{11}g_{32}(g_{23} + g_{43}) + g_{12}g_{21}g_{33} - g_{11}g_{22}g_{33} - g_{13}g_{21}g_{32}. \tag{31c}$$

Hence the other three eigenvalues are:

$$\lambda_1 = -\tfrac{1}{3}c_2 + r_1 + r_2, \tag{32a}$$

$$\lambda_2 = -\tfrac{1}{3}c_2 - \tfrac{1}{2}(r_1 + r_2) + \tfrac{1}{2}i\sqrt{3}(r_1 - r_2), \tag{32b}$$

$$\lambda_3 = -\tfrac{1}{3}c_2 - \tfrac{1}{2}(r_1 + r_2) - \tfrac{1}{2}i\sqrt{3}(r_1 - r_2), \tag{32c}$$

where $r_1 = \sqrt[3]{t + \sqrt{\Delta}}$ and $r_2 = \sqrt[3]{t - \sqrt{\Delta}}$.

Δ represents the discriminant of the cubic polynomial:

$$\Delta = s^3 + t^2, \tag{33}$$

where $s = \frac{1}{9}(3c_1 - c_2^2)$ and $t = \frac{1}{54}(9c_1c_2 - 27c_0 - 2c_2^3)$.

The leading eigenvalue of matrix **G** corresponds to the greater value of λ_i in absolute terms $\lambda_{\max} = \max\{|\lambda_1|, |\lambda_2|, |\lambda_3|\}$ where $|\lambda_i|$ stands for the absolute value of λ_i. The algebraic expression of the eigenvalues of **G** are sufficiently complicated as to convey little biological insight.

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