Some Host Parasite Genetic Interaction Models*

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1. INTRODUCTION

This paper studies some models of host-parasite interaction based on genetic responses. Polymorphism is maintained both in the host population and in the parasite population. The models are motivated by a simple model of Jayakar [2]. The special feature in Jayakar's model is that only a fraction $\alpha$ of the host population interact with the parasite in each generation. The remaining $1 - \alpha$ fraction of the host population do not interact with the parasite. This feature is incorporated in all our models and turns out to be crucial.

The interaction of host and parasite is based on the following simple rule. Genes in the host are matched with genes in the parasite to form pairs. For example, we can let $A, B$ be genes in the host, $A', B'$ be genes in the parasite, and let $A - A', B - B'$ be matched pairs of genes. A particular parasite is virulent on a particular host (or the host is susceptible to the parasite) if, among the genes in the host and in the parasite, there are one or more pairs of matched genes. If there are no such pairs, the parasite is avirulent on the host. To take up the above example, a parasite carrying an $A'$ gene is virulent on any host carrying an $A$ gene because there is the matched pair $A - A'$. A parasite having $A'$ gene but not $B'$ gene is avirulent on a host having $B$ gene but not $A$ gene because there are no matched pairs of genes.

The following models exhibit two interesting properties. The first is that given the rule of interaction, polymorphism can be maintained both in the host and the parasite only when $\alpha$ (the fraction of host which interact with the parasite) is small. In fact, the smaller the $\alpha$, the better the stability of the polymorphism. For $\alpha$ too large, polymorphism can no longer be maintained in both the host and the parasite. The second is that as we extend the above rule for host-parasite interaction in a natural way to involve two, three and more independent loci, the stability of the systems tend to progressively improve. Thus complexity improves stability. Here is another example of the general observa-

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tion that "... complexity appears to be the basis for the stability of predator-prey systems..." [4].

Assumptions About the Models

(1) Time is in discrete generations. Each species reproduces by random mating.

(2) With respect to the interaction property, the host population is determined at a single locus with two alleles A, a; the parasite is also determined by a single locus with two alleles A', a'.

(3) Whenever a host and a parasite meet, either the host dies and the parasite survives to reproduce for the next generation or the parasite dies and the host survives to reproduce for the next generation.

(4) In each generation, not every host individual comes into contact with a parasite.

(5) Every parasite finds a host.

(6) The rule for the host-parasite interaction is the following. A - A', a - a' are considered as matched pairs of genes. A particular parasite is virulent on a particular host if their genotypes are such that among the genes carried by them, there is one or more matched pairs of genes. Figs. 1 and 7 illustrate the rule in operation.

2. Model I: Haploid Host and Diploid Parasite

In this model, the host is haploid and the parasite is diploid. The same model applies to the case where the host is diploid, consisting of types AA, aa and reproduces by selfing; one just lets AA take over the role of A and aa take over the role of a. Let \( p_1, (1 - p_1) \) be the frequencies of A, (a) in the host population. Let \( p_2^e, 2p_2(1 - p_2), (1 - p_2)^e \) be the frequencies of \( A'A', A'a', a'a' \) in the parasite population. The scheme of interaction is indicated by the above diagram.

![Fig. 1. Scheme of host-parasite interaction for Model I. An arrow from a parasite genotype to a host genotype indicates that the parasite is virulent on the host. No arrow between a parasite genotype and a host genotype means that the parasite is avirulent on the host.](image-url)
A particular host of type A will survive to reproduce for the next generation either if it does not meet a parasite or if it meets a parasite of type a' a' which is avirulent on it. The probability of the former is \((1 - \alpha) p_1\), the probability of the latter is \(\alpha p_1(1 - p_2)^2\). So the amount of A-host which survives through interaction with the parasite to reproduce for the next generation is \((1 - \alpha) p_1 + \alpha p_1(1 - p_2)^2\). Similarly, the amount of a-host which survives through interaction with the parasite and reproduce for the next generation is \((1 - \alpha)(1 - p_1) + \alpha(1 - p_1)(1 - p_2)^2\). So the gene frequency of A-host in the next generation is

\[
p_1' = \frac{(1 - \alpha) p_1 + \alpha p_1(1 - p_2)^2}{(1 - \alpha) p_1 + \alpha p_1(1 - p_2)^2 + (1 - \alpha)(1 - p_1) + \alpha(1 - p_1)(1 - p_2)^2}.
\]

A particular A' A' parasite will survive to reproduce for the next generation only if it meets a AA host. So the amount of A' A' parasites which survive through host-parasite interaction is \(p_2^2 p_1\). An A' a' parasite will survive no matter which kind of host it meets. The amount of A' a' parasites which survive through host-parasite interaction is \(2p_2(1 - p_2)\). Similarly, the amount of a' a' parasites which survive through interaction is \((1 - p_2)^2 (1 - p_1)\). The frequency of A' gene after interaction is

\[
p_2' = \frac{p_2^2 p_1 + p_2(1 - p_2)}{p_2^2 p_1 + 2p_2(1 - p_2) + (1 - p_2)^2 (1 - p_1)}.
\]

Since random mating does not change gene frequencies, \(p_2'\) is the frequency of A' gene in the next generation before host-parasite interaction.

The above system has exactly five equilibrium points and the only internal (polymorphic) equilibrium is \((p_1, p_2) = (\frac{1}{4}, \frac{1}{2})\). To study stability we evaluate the Jacobian at \((\frac{1}{4}, \frac{1}{2})\), obtaining

\[
\begin{pmatrix}
\frac{1}{4} & -c \\
\frac{1}{2} & 0
\end{pmatrix}, \quad \text{where} \quad c = \frac{\alpha/2}{1 - 3/4\alpha}.
\]

![Fig. 2](image-url)  
**Fig. 2.** Local behavior in Model I of the successive interaction of \((p_1, p_2)\) near the internal equilibrium point \((\frac{1}{4}, \frac{1}{2})\) for various values of \(\alpha\).
Fig. 3. The trajectory traces 50 successive generations of host and parasite when $\alpha = 0.08$ in Model 1.

Fig. 4. The trajectory traces 50 successive generations of host and parasite when $\alpha = 0.5$ in Model 1.
Fig. 5. The trajectory traces 50 successive generations of host and parasite when \( \alpha = 0.8 \) in Model I.

Fig. 6. Each of the two trajectories traces 50 successive generations of host and parasite when \( \alpha = 0.9 \) in Model I. Note the clear indication of a limit-cycle.
The eigenvalues of this matrix are \( \lambda = \frac{5}{6} \pm \frac{1}{6} \sqrt{1 - 12c} \). The eigenvalues are both positive and <1 when \( 0 < c < 1/12 \) (i.e., \( 0 < \alpha < 4/27 \)). They are both complex with absolute values <1 when \( 1/12 \leq c < 1 \) (i.e., \( 4/27 \leq \alpha < 4/5 \)). They are both complex with absolute value 1 when \( 1 \leq c \) (i.e., \( 4/5 \leq \alpha \leq 1 \)). Thus, the internal equilibrium point \( (\frac{1}{2}, \frac{1}{2}) \) is stable if \( 0 < \alpha < 4/5 \).

Near \( (\frac{1}{2}, \frac{1}{2}) \) the successive iterations of \( (p_1, q_0) \) behave differently, depending on the form of the eigenvalues and hence on \( \alpha \).

Letting \( q_0 = 1 - p_0 \), successive iterations of \( (p_1, q_0) \) carried out by computer, produced Figs. 3, 4, 5, 6. Each path in these figures traces the behavior of 50 successive generations. From the computer data, it appears that for \( 4/5 < \alpha \), there exists a limit cycle. The limit cycle appears to start out as a point when \( \alpha = 4/5 \) and becomes larger and larger as \( \alpha \) increases in value.

3. Model II: Diploid Host and Diploid Parasite

\[
\text{FIG. 7. Scheme of host–parasite interaction for Model II.}
\]

Let \( p_1^2, 2p_1(1 - p_1), (1 - p_1)^2 \) be the frequencies of AA, Aa, aa in the host and \( p_2^2, 2p_2(1 - p_2), (1 - p_2)^2 \) be the frequencies of A'A', A'a', a'a' in the parasite. The scheme of host–parasite interaction is indicated in the above diagram. The gene frequencies after interaction are:

\[
p_1' = \frac{(1 - \alpha) p_1^2 + \alpha p_1^2(1 - p_2)^2 + (1 - \alpha) p_2(1 - p_2)}{(1 - \alpha) p_1^2 + \alpha p_1^2(1 - p_2)^2 + 2(1 - \alpha) p_1(1 - p_1) + (1 - \alpha)(1 - p_1)^2 + \alpha(1 - p_1)^2 p_2^2},
\]

\[
p_2' = \frac{p_2^2 p_1^2 + 2p_2^2 p_1(1 - p_1) + p_2(1 - p_2)}{p_2^2 p_1^2 + 2p_2^2 p_1(1 - p_1) + 2p_2(1 - p_2) + (1 - p_2)^2(1 - p_1)^2 + 2(1 - p_2)^2 p_2(1 - p_1)}.
\]

Again, \( (p_1, p_2) = (\frac{1}{2}, \frac{1}{2}) \) is the only internal equilibrium point. To study its stability, we evaluate the Jacobian at \( (\frac{1}{2}, \frac{1}{2}) \), obtaining

\[
\begin{pmatrix}
1 + e & -2e \\
2/7 & 6/7
\end{pmatrix},
\] where \( e = \frac{1/8}{1 - 7/8 \alpha} \).

The eigenvalues of this matrix are

\[
\lambda = [1 + (6/7 + e) \pm \sqrt{(1 - (6/7 + e))^2 - 12e/7}]/2.
\]
For $\alpha$ sufficiently small (hence $e$ small), it is easy to see that both of the
eigenvalues are positive and $<1$. As $\alpha$ becomes larger, the eigenvalues become
complex. But as long as $\alpha < 8/17$, the eigenvalues have their absolute value $<1$.
Hence the internal equilibrium point $(\frac{1}{2}, \frac{1}{2})$ is stable for $0 < \alpha < 8/17$.

4. MODEL III: HAPLOID HOST AND DIPLOID PARASITE WITH
INTERACTION BASED ON TWO INDEPENDENT LOCI

The assumptions of the previous models are extended naturally in the
following way.

The host population is determined by two independent loci with two alleles
at each locus. Let the alleles be $A$, $a$ at one locus, $B$, $b$ at the other. Since the
host is haploid, there are altogether four types of host, $(A, B)$, $(A, b)$, $(a, B)$,
$(a, b)$.

The parasite is also determined by two independent loci with alleles $A'$, $a'$ at
one locus and $B'$, $b'$ at the other. Since the parasite is diploid, there are ten
types of parasite:

<table>
<thead>
<tr>
<th>A' B'</th>
<th>A' B'</th>
<th>A' B'</th>
</tr>
</thead>
<tbody>
<tr>
<td>A' B'</td>
<td>A' b'</td>
<td>A' b'</td>
</tr>
<tr>
<td>a' B'</td>
<td>a' B'</td>
<td>a' b'</td>
</tr>
<tr>
<td>a' B'</td>
<td>a' b'</td>
<td>a' b'</td>
</tr>
</tbody>
</table>

The double heterozygotes $(A'B')$ and $(A'b')$ are considered the same.

Now, consider $\Lambda - A'$, $a - a'$, $B - B'$, $b - b'$ as matched pairs of genes. A parasite is virulent on a host only if among the genes of the host and the
parasite, there is at least one matched pair at each one of the two loci. A sample
of such interaction is shown in the following diagram.

![Diagram](image-url) **Fig. 8.** A sample indicating the scheme of host-parasite interaction for Model III.
Let $p_1, q_1, p_2, q_2$ be the gene frequencies of $A, B, A', B'$, respectively. The new gene frequencies after interaction are the following: To simplify expressions, let

$$[m; n] = m^2 + n^2 - m^2 n^2,$$

$$p_1' = f(p_1, q_1, p_2, q_2) = \frac{(1 - \alpha) p_1 + \alpha p_1 q_1[1 - p_2; 1 - q_2] + \alpha p_1 (1 - q_1)[1 - p_1; q_1]}{1 - \alpha + \alpha p_1 q_1[1 - p_2; 1 - q_2] + \alpha p_1 (1 - q_1)[1 - p_1; q_1]},$$

$$q_1' = f(q_1, p_1, q_2, p_2),$$

$$p_2' = \frac{p_1 p_2^2 + p_2 (1 - p_2)}{p_1 p_2^2 + 2 p_2 (1 - p_2) + (1 - p_1) (1 - p_2)^2},$$

$$q_2' = \frac{q_1 q_2^2 + q_2 (1 - q_2)}{q_1 q_2^2 + 2 q_2 (1 - q_2) + (1 - q_1) (1 - q_2)^2}.$$

Note that if we limit attention to only one generation, the parasite frequencies are exactly those of two independent one-locus host-parasite interactions (model I).

Once more $(p_1, q_1, p_2, q_2) = (\frac{1}{3}, \frac{1}{3}, \frac{1}{3}, \frac{1}{3})$ is the only internal equilibrium point. Evaluation of the Jacobian at $(\frac{1}{3}, \frac{1}{3}, \frac{1}{3}, \frac{1}{3})$ gives

$$\begin{pmatrix}
1 & 0 & -g & 0 \\
0 & 1 & 0 & -g \\
1/3 & 0 & 2/3 & 0 \\
0 & 1/3 & 0 & 2/3
\end{pmatrix}, \quad \text{where} \quad g = \frac{3/8 \alpha}{1 - 9/16 \alpha}.$$

The two different eigenvalues of this matrix are just the eigenvalues of

$$\begin{pmatrix}
1 & -g \\
1/3 & 2/3
\end{pmatrix},$$

and these are

$$\lambda = [(1 + 2/3) \pm \sqrt{(1/3)^2 - 4g/3}] / 2.$$

The eigenvalues are positive and $<1$ when $0 < g < 1/12$ (i.e., $0 < \alpha < 16/81$). They are complex and with absolute value $<1$ when $1/12 \leq g < 1$ (i.e., $16/81 \leq \alpha < 16/15$). So for any value of $0 < \alpha < 1$, the internal equilibrium point is stable.

This scheme of interaction can be extended to three, four, or more independent loci. The results will be similar. In fact, the segment of values of $\alpha$ for which the eigenvalues are positive and $<1$ [(0, 16/81) in the case of two independent loci] will become larger and larger and the absolute values of the
eigenvalues for fixed $\alpha$ decreases as the number of independent loci involved in interaction is increased. So that if $\alpha$ is fixed, the larger the number of loci involved, the better the quality of stability. This can be regarded as a quantitative example of the general observation that the stability of a predator-prey system improves when its complexity increases [4].

5. Generalization

So far, it is assumed that whenever a host and a parasite meet, one dies and the other survives. Instead of this, one can let $e$ be the probability of the parasite killing the host and $(1 - e)$ the probability of the host killing the parasite. For each host-parasite system, one can make a table of these virulence probabilities between various kinds of host and parasite. For example, tables for models I and II are:

<table>
<thead>
<tr>
<th>Host</th>
<th>Aa'</th>
<th>Aa</th>
<th>aa</th>
</tr>
</thead>
<tbody>
<tr>
<td>A'a'</td>
<td>1</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>A'a'</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>a'a'</td>
<td>0</td>
<td>1</td>
<td></td>
</tr>
</tbody>
</table>

Table (I) is an alternative description of the interactions given in Fig. 1.

For each one of the models considered so far, similar behavior will result if one perturbs slightly its table of virulence probabilities. For example, the system of interaction produced by the following table for small $e$'s will have similar behavior as that of Table (I).

<table>
<thead>
<tr>
<th>Aa</th>
<th>a</th>
</tr>
</thead>
<tbody>
<tr>
<td>A'A'</td>
<td>$1 - \epsilon_1$</td>
</tr>
<tr>
<td>A'a'</td>
<td>$1 - \epsilon_2$</td>
</tr>
<tr>
<td>a'a'</td>
<td>$\epsilon_3$</td>
</tr>
</tbody>
</table>

(III)
The symmetry in the Tables (I) and (II) reflects the symmetric role played by the two alleles A, a in the host and the two alleles \(A'a'\) in the parasite. Preserving this symmetry, the most general tables one can construct for the haploid–diploid case and diploid–diploid case are:

<table>
<thead>
<tr>
<th>A</th>
<th>a</th>
<th>AA</th>
<th>Aa</th>
<th>aa</th>
</tr>
</thead>
<tbody>
<tr>
<td>BB</td>
<td>e</td>
<td>f</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bb</td>
<td>g</td>
<td>g</td>
<td></td>
<td></td>
</tr>
<tr>
<td>bb</td>
<td>f</td>
<td>e</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(IV)

<table>
<thead>
<tr>
<th>BB</th>
<th>e</th>
<th>f</th>
<th>g</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bb</td>
<td>h</td>
<td>i</td>
<td>h</td>
</tr>
<tr>
<td>bb</td>
<td>g</td>
<td>f</td>
<td>e</td>
</tr>
</tbody>
</table>

(V)

where \(e, f, g, h, i\) are all values in \([0, 1]\).

\((p_1, p_2) = (\frac{1}{2}, \frac{1}{2})\) remain an internal equilibrium point for such models. One can get various sufficient conditions in terms of the probabilities in the table for stability of this internal equilibrium point. For example, in the system determined by Table (IV), \(2g > e + f\) is a sufficient condition provided \(\alpha\) is small enough.

6. DISCUSSION

The host–parasite models investigated were found to exhibit stable polymorphisms in both host and parasite for a considerable range of values of the parameter \(\alpha\). For still larger values of \(\alpha\) the numerical studies clearly indicated the presence of stable periodic behavior (limit cycles).

The only selection incorporated in the models is the selection mediated via the host–parasite interaction. Thus there is no confounding of the effects of different kinds of selection, and the existence and stability of the polymorphisms and limit cycles can be attributed to the host–parasite genetic responses alone.

On the other hand, it was noted that the stable polymorphisms which were found persist with at most small changes in the equilibrium gene frequencies when the virulence coefficients in the models are subject to small but quite general perturbations. The stable polymorphisms will persist in like manner if small selective differences are assigned to the various genotypes in the host population and the parasite population (see [3] for a general treatment of perturbations). Thus the polymorphisms are robust in the sense that they will be present even under adverse selection provided the selection is not too strong. It should be noted that the same statement is not true for the host polymorphism found in the model of Jayakar [2].
Models with both host and parasite haploid, but otherwise similar to models I and III, were investigated. These models did not yield a stable polymorphism for either the host or the parasite. These results are similar to the results for the continuous time prey–predator models of Stewart [5], where stable polymorphism for both prey and predator occurs in the diploid–diploid case but fails to occur in the haploid–haploid case.

By increasing the number of loci involved in our rule of host–parasite interaction, we complicate the system by involving more genotypes of host and parasite in a more complicated interaction. But the stability of the more complicated system improves in the sense that the absolute value of the eigenvalues associated with local stability actually decreases, the parameter \( \alpha \) being held fixed. This is a nice example of the general observation that complexity is the basis of stability for predator–prey systems.

Systems of flax and flax-rust are host–parasite systems occurring in nature where interaction is based on matching of genes [1]. However, the models in the paper do not describe those systems owing to different rules of interaction.

**Acknowledgment**

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**References**