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Multiline varieties and disease control

5. The "dirty crop" approach with complex mixtures of genotypes based on overlapping gene sets*

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Summary. A general model for the evolution of pathogen populations on mixtures or multilines is developed. This model is used to extend previous analyses of the effects of the widespread cultivation of multilines on the evolution of virulence in obligate parasites to mixtures of lines carrying different numbers of resistance genes. It is concluded that the composition of an equilibrium pathogen population growing on a multiline may vary within wide limits and the prinicipal determinant of its composition is the number of components in the multiline and the resistance genes they carry. Other factors of importance are (i) the relative contribution made by each host class (with different numbers of resistance genes) to the pathogen spore pool each generation; (ii) the levels of 'stabilizing selection' against unnecessary virulence genes; and (iii) the way in which unnecessary genes for virulence combine to reduce pathogen fitness. Conditions for the fixation of avirulent biotypes in the pathogen population and the evolution of a pathogen superrace are given for multilines of various compositions.

Key words: Multiline varieties – Disease control – "Dirty crop" approach – Evolution – Pathogens

1 Introduction

"Dirty crop" or "partially resistant" multilines, where each component of the multiline carries one or more resistance genes but none of the resistances are effective against all known races of the pathogen (Marshall 1977), have attracted considerable interest recently because of their potential in the long-term control of plant diseases (Browning and Frey 1969; Frey et al. 1973, 1975, 1977). The proponents of "dirty crop" multilines argue that because many different pathogen races have an opportunity to survive on such a mixture, and because each component carries a different gene or set of genes for resistance, stabilizing selection against unnecessary genes for virulence will prevent the development of highly complex pathogen biotypes (Van der Plank 1963, 1968). It also will ensure that simple races, with one or a few genes for virulence, dominate the pathogen population. They further argue that because each component of the multiline would be attacked by one or a few races of the stabilized pathogen population, the remaining lines would act as spore traps reducing the rate of spread of the disease. In this way multiline cultivars would have an effect similar to horizontal resistance (Van der Plank 1963) in delaying the intra-crop buildup of the pathogen.

However, the question of the ultimate utility of multiline varieties in disease control remains a matter of considerable controversy. Indeed it has been suggested that multilines will not only fail to stabilize and control pathogen populations as predicted by their proponents, but also that they will serve as a breeding ground for new and complex pathogen races, perhaps even a "superrace" which can overcome all known sources of resistance (Browning and Frey 1969; Marshall 1977). The aim of this series of studies was to examine the effects of mixed host varieties on the racial composition of pathogen populations and disease control using simple theoretical models. In the first paper (Marshall and Pryor 1978), we considered mixtures of lines each of which carried a single unique gene for resistance. More recently results were extended to mixtures of lines each carrying g ($g \geqq 2$) genes for resistance (Marshall and Pryor 1979; Marshall and Burdon 1981).

In this paper, we develop a more general procedure for the analysis of the effects of multiline varieties on

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the evolution of virulence in obligate parasites which is applicable to mixtures of lines carrying different numbers of resistance genes. We use this procedure to investigate the effects of the inclusion of a completely susceptible component (zero genes for resistance), in a multiline mixture where all other components carry g genes for resistance, on pathogen evolution and disease control. Leonard (1969) advocated the inclusion of a completely susceptible component in multiline mixtures to prevent the selection of complex pathogen races on such varieties. We also present some results for multilines where hosts may carry any of the numbers 0, 1, ..., g of resistance genes.

2 General model

We consider a mixture of diploid host genotypes that are identical except for the number of resistance genes they carry to a specified pathogen. Resistance is assumed to be dominant over susceptibility, all resistance genes are assumed to be nonallelic, and all individuals are assumed to be completely homozygous. The multiline is taken to be reconstituted annually so that its composition is stable over time and we also assume that it is grown over a sufficiently large area for it to be the major force influencing the evolution of the local pathogen population.

We limit attention to pathogens that conform to the gene-for-gene relationship between host resistance and pathogen virulence (Flor 1956), and we assume that genes for virulence in the pathogen are recessive (Person and Sidhu 1971). Pathogen biotypes with all possible combinations of virulence genes are assumed to exist, at least initially, in the pathogen population and this population is taken to be sufficiently large that a deterministic treatment of changes in biotype frequencies is adequate.

Pathogens are identified by v, the number of virulence genes they carry, and by V, the particular set of v virulence genes, while hosts are identified by r, the number of resistance genes they carry, and by R, the particular set of r resistance genes. Pathogens are dispersed at random over all host genotypes, and have constant relative reproductive rates over any particular class of hosts. There is no reproduction if a pathogen lacks virulence genes for any of the host resistance genes, so that, if ${}_{rR}w_{vV}$ is the reproductive rate for the vVth pathogen biotype on the rRth host genotype,

$$\begin{aligned} {}_{rR}w_{vV} &= 0 \quad v < r \\ &= 0 \quad v \geqq r, \quad rR \not\subset vV \\ &\neq 0 \quad v \geqq r, \quad rR \subset vV \end{aligned}$$

where $rR \subset vV$ means that all resistance genes in host class rR are matched by virulence genes in pathogen class vV.

The relative contribution from the rRth host class to the pathogen spore pool at any generation is ${}_{rR}f$, so that the frequency ${}_{rR}x_{vV}(t+1)$ of the vVth pathogen class arising from the rRth host class at the start of pathogen generation $(t+1)$ is

$${}_{rR}x_{vV}(t+1) = {}_{rR}f\,{}_{rR}w_{vV}\,x_{vV}(t)/{}_{rR}\bar{W}(t)\,. \qquad (1)$$

Here $x_{vV}(t)$ is the total frequency of pathogens of type vV at the beginning of the tth reproductive cycle, and

$${}_{rR}\bar{W}(t) = \sum_i \sum_I {}_{rR}w_{iI}\,x_{iI}(t)$$

is the mean fitness of all pathogen biotypes on the rRth host.

Summing equation (1) over host classes provides

$$x_{vV}(t+1) = x_{vV}(t) \sum_r \sum_R [{}_{rR}f\,{}_{rR}w_{vV}/\sum_i \sum_I {}_{rR}w_{iI}\,x_{iI}(t)]\,. \qquad (2)$$

We are interested in determining the equilibria of this system, and then which equilibrium a pathogen population is likely to evolve towards on a particular multiline. Further developments of the model are simplified with these following assumptions:

(i) All p_v members of the vth pathogen class (v virulence genes) are equally frequent,

$$x_{vV}(t) = x_v(t)/p_v$$

where $x_v(t)$ is the total frequency of pathogens with v virulence genes in generation (t).

(ii) All ${}_rh$ members of the rth host class (r resistance genes) make equal contributions to the pathogen spore pool,

$${}_{rR}f = {}_rf/{}_rh$$

where ${}_rf$ is the total contribution of the hosts containing r resistance genes.

(iii) Any of the ${}_rh$ members of the rth host class can be attacked by ${}_ra_v$ members of the vth pathogen class. Further, the reproductive rate of each of these attacking pathogens is the same on that host and depends only on v,

$$\begin{aligned} {}_{rR}w_{vV} &= w_v \quad rR \subset vV \\ &= 0 \quad \text{otherwise}\,. \end{aligned}$$

Substituting these values into (2), and noting that $\sum_I {}_{rR}w_{iI} = {}_ra_i\,w_i$ provides

$$x_v(t+1) = x_v(t) \sum_r [{}_rf\,{}_rc_v/{}_r\bar{W}(t)]\,, \qquad (3)$$

where

$${}_rc_v = {}_ra_v\,w_v/p_v$$

is a measure of fitness for pathogen class v on host class r, and

$${}_r\bar{W}(t) = \sum_i {}_rc_i\,x_i(t)$$

is the mean pathogen fitness on host class i.

Now equations (3) are just those for the behavior of haploids in a multiniche environment (Strobeck 1979) and have the same structure as for species (v) in a spatially heterogeneous environment (Levene 1953). Strobeck points out the apparent lack of general conditions that are necessary and sufficient for the attainment of equilibria with specified pathogen compositions. However, good progress can be made with special cases.

3 Parameters of model

3.1 Host contributions ${}_rf$

We let ${}_rq$ be the frequency of class r hosts in the multiline. This quantity is under direct control of the breeder. He may choose to omit some classes r, and then ${}_rq = 0$, or he may choose a single class r, so that ${}_rq = 1$. We phrase results in terms of ${}_rq$ to allow our theory to accommodate any arrangement, subject to the requirements of symmetry. We require every resistance gene to be equally represented in a particular class.

In this paper we limit our attention to the case where the classes, r, of hosts are developed by taking

all possible sets of r resistance genes, that is, the breeder uses "overlapping gene sets" (Marshall and Pryor 1979; Marshall and Burdon 1981). It follows that the number of host types within a class is

$$_rh = \binom{n}{r}.$$

The host contributions $_rf$ are set to be

$$_rf = C\,{}_rq\,{}_rS,$$

where $_rS$ is a measure of the "susceptibility" of the rth host to the pathogen population, and C is to ensure that the $_rf$ sum to one. There is little information in the literature on which to assess "susceptibility" in this context, and consequently we consider two cases that we believe cover the range of likely possibilities. In the first place, we assume that the relative contribution of each host genotype to the total spore pool is proportional to the mean fitness of the pathogen races to which it is susceptible. This situation would be encountered in practice if the crop was lightly diseased each generation so that spore production was a function of the number and relative fitnesses of the pathogen races virulent on each host genotype. Under this "lightly diseased" situation

$$_rS = {}_r\bar{W}, \quad {}_rf = {}_rq\,{}_r\bar{W}/\sum_r {}_rq\,{}_r\bar{W}$$

and, from (3),

$$x_v(t+1) = x_v(t)\left(\sum_r {}_rq\,{}_rc_v\right)/\bar{W}(t) \tag{4}$$

with overall mean fitness

$$\bar{W}(t) = \sum_r \sum_i {}_rq\,{}_rc_i\,x_i(t).$$

The second extreme assumption we make about host contributions is that all hosts, regardless of the number of resistance genes they carry, contribute equally to the spore pool. This situation would be encountered in practice if the crop was severely and continuously diseased so that each host genotype produces the maximum number of spores possible, regardless of the number of pathogen races to which it is susceptible. In this "heavily diseased" situation

$$_rS = 1, \quad {}_rf = {}_rq$$

and, from (3),

$$x_v(t+1) = x_v(t)\sum_r [{}_rq\,{}_rc_v/{}_r\bar{W}(t)]. \tag{5}$$

3.2 Selection values $_rc_v$

We suppose that the fitness of a pathogen, on any host upon which it can survive, decreases as the number of virulence genes increases. That is, there is stabilizing selection against unnecessary genes for virulence in the pathogen (Van der Plank 1963, 1968). We assume that each additional virulence gene decreases fitness additively

$$w_v = 1 - vs$$

or multiplicatively

$$w_v = (1-s)^v,$$

where s is a positive selection coefficient.

For multiplicative selection, the requirement that fitness not be negative implies that $0 \leqq s \leqq 1$, but for additive selection, the requirement is more restrictive. There are no problems if $s \leqq 1/n$, where n is the maximum number of resistance genes used. For larger s values, we just assign a fitness of zero to those pathogen classes v for which $s > 1/v$, still with the restriction $0 \leqq s \leqq 1$.

For populations with hosts carrying different numbers of resistance genes, we assume that all pathogen biotypes have equal fitness on all hosts on which they can grow. In Table 1, we display the additive selection values $_rR w_{vV}$ in the situation where hosts have 0, 1 or 2 resistance genes from a total of 4 available genes, and pathogens have up to 4 virulence genes. The table emphasizes that all fitness are relative to that of a pathogen with no virulence genes growing on a host with no resistance genes, and that a host with no resistance genes can be attacked by all pathogen biotypes.

We suppose the breeder has n resistance genes at his disposal and that he can develop and use any combination of r, $0 \leqq r \leqq g$, of such genes. Therefore, we can restrict consideration to those pathogen biotypes with no more than n virulence genes, $0 \leqq v \leqq n$. The fitnesses in Table 1 suggest that pathogens with virulence genes for which the corresponding resistance genes are absent from the host population are less fit than those with only effective virulence genes. It is easy to show that these pathogens with redundant genes are invariably eliminated from the population (Marshall and Pryor 1978).

The number of types in pathogen class v is

$$p_v = \binom{n}{v},$$

while the number in that class that can attack a particular host with $r \leqq v$ resistance genes is

$$_ra_v = \binom{n-r}{v-r}$$

so that

$$_rc_v = w_v \binom{v}{r} \Big/ \binom{n}{r}.$$

Table 1. Relative fitnesses ${}_{r}R w_{vV}$ of Vth member of pathogen class v on Rth member of host class r when n = 4 and g = 2 under the additive fitness model

Resistance genes (R) present			Virulence genes (V) present															
			None	V_1	V_2	V_3	V_4	V_1V_2	V_1V_3	V_1V_4	V_2V_3	V_2V_4	V_3V_4	$V_1V_2V_3$	$V_1V_2V_4$	$V_1V_3V_4$	$V_2V_3V_4$	$V_1V_2V_3V_4$
		v	0	1	1	1	1	2	2	2	2	2	2	3	3	3	3	4
	r	R	1	1	2	3	4	1	2	3	4	5	6	1	2	3	4	1
None	0	1	1	$1-s$	$1-s$	$1-s$	$1-s$	$1-2s$	$1-2s$	$1-2s$	$1-2s$	$1-2s$	$1-2s$	$1-3s$	$1-3s$	$1-3s$	$1-3s$	$1-4s$
R_1	1	1	0	$1-s$	0	0	0	$1-2s$	$1-2s$	$1-2s$	0	0	0	$1-3s$	$1-3s$	$1-3s$	0	$1-4s$
R_2	1	2	0	0	$1-s$	0	0	$1-2s$	0	0	$1-2s$	$1-2s$	0	$1-3s$	$1-3s$	0	$1-3s$	$1-4s$
R_3	1	3	0	0	0	$1-s$	0	0	$1-2s$	0	$1-2s$	0	$1-2s$	$1-3s$	0	$1-3s$	$1-3s$	$1-4s$
R_4	1	4	0	0	0	0	$1-s$	0	0	$1-2s$	0	$1-2s$	$1-2s$	0	$1-3s$	$1-3s$	$1-3s$	$1-4s$
R_1R_2	2	1	0	0	0	0	0	$1-2s$	0	0	0	0	0	$1-3s$	$1-3s$	0	0	$1-4s$
R_1R_3	2	2	0	0	0	0	0	0	$1-2s$	0	0	0	0	$1-3s$	0	$1-3s$	0	$1-4s$
R_1R_4	2	3	0	0	0	0	0	0	0	$1-2s$	0	0	0	0	$1-3s$	$1-3s$	0	$1-4s$
R_2R_3	2	4	0	0	0	0	0	0	0	0	$1-2s$	0	0	$1-3s$	0	0	$1-3s$	$1-4s$
R_2R_4	2	5	0	0	0	0	0	0	0	0	0	$1-2s$	0	0	$1-3s$	0	$1-3s$	$1-4s$
R_3R_4	2	6	0	0	0	0	0	0	0	0	0	0	$1-2s$	0	0	$1-3s$	$1-3s$	$1-4s$

4 Equilibrium populations

4.1 Lightly diseased case

The lightly diseased case allows an immediate discussion of equilibrium populations. From (4)

$$x_v(t) \propto \left(\sum_r {}_rq\ {}_rc_v\right)^t x_v(0)$$

with the result that the final population contains only one class of pathogen, and that is the class for which

$$\sum_r {}_rq\ {}_rc_v = X_v$$

is a maximum. The only exception occurs when two v values give the same maximum value and those two classes coexist in a neutral equilibrium.

Our approach is to divide the range of the selection parameter s into disjoint regions identified by the X_v values maximized there. In other words, the unit interval can be partitioned into sets, upon each of which one class of pathogens becomes fixed. The point values of s between each pair of adjacent regions are those values which allow the coexistence of two classes, but these are unlikely to be of practical importance. In general, smaller values of s lead to fixation of pathogens with larger numbers of virulence genes.

While algebraic approaches are possible, it is simpler to use numerical values to compare X_v values and thus determine the nature of equilibrium populations.

4.2 Heavily diseased case

The heavily diseased case corresponds exactly to the elegant treatment of Strobeck (1979). He showed that the transition equations (5) may be written as

$$x_v(t+1) = \frac{x_v(t)}{U(t)} \frac{\partial U(t)}{\partial x_v(t)},$$

where

$$U(t) = \prod_r [{}_r\bar{W}(t)]^{rq}$$

and that U (t) increases over time (Baum and Eagon 1967). Equilibrium, therefore, corresponds to the maximum value of this fitness function U (t), as also shown by Cannings (1973) and, in less general form, by Li (1955).

At equilibrium, for those pathogen classes v with $\hat{x}_v \neq 0$ (a caret denotes the limiting value as time increases),

$$1 = \sum_r ({}_rc_v\ {}_rq / {}_r\hat{\bar{W}}) . \tag{6}$$

Strobeck points out that (6) may be regarded as a set of equations in the quantities

$${}_ry = {}_rq / {}_r\hat{\bar{W}} .$$

If the multiline contains R* host classes, and V* pathogen classes coexist at equilibrium, then (6) is a system of V* equations in R* unknowns, and a solution exists only if $R^* \leqq V^*$. The number of pathogens at equilibrium is bounded above by the number of classes of hosts.

If matrix C is defined to have ${}_rc_v$ as the element in row v and column r (possibly renumbered to avoid problems of missing classes), Strobeck points out that (6) leads to

$$\hat{x}_v = \sum_r \left(\frac{{}_rq\ {}_rA_v}{\sum_i {}_rA_i} \right), \tag{7}$$

where ${}_rA_v$ is the cofactor of ${}_rc_v$ in matrix C [i.e., $(-1)^{r+v}$ times the determinant of C with row v and column r omitted]. Valid equilibria require $0 \leqq \hat{x}_v \leqq 1$.

Algebraic treatments tend to be quite complex, so again numerical studies are preferred. Equation (7) determines the equilibria (there may be several), and the system moves towards that equilibrium with maximum fitness U.

5 Specific cases

We consider three classes of multilines: those in which each line carries g genes for resistance, those composed of lines with 0 and g genes for resistance, and those composed of lines with $0, 1, \ldots, g$ genes for resistance. In each case, we assume that selection against unnecessary genes for virulence is additive or multiplicative, while for complex mixtures containing lines with different numbers of resistance genes, we also assume that the level of disease is either "heavy" or "light".

5.1 Mixtures of lines carrying g genes for resistance

In this case, ${}_rq = 1$ if $r = g$, and is zero otherwise. Pathogens with $v < g$ cannot survive on this multiline. With the mean fitness of each class of pathogen biotype now being the same in all hosts, we need not distinguish between lightly and heavily diseased situations. Following our previous discussion, we predict that only class v pathogens will exist at equilibrium, where v maximizes the quantities $X_v = {}_gc_v$. We can write ${}_gc_v$ as c_v without ambiguity.

For additive fitness values then, we maximize the selection values

$$c_v = (1 - vs)\binom{v}{g} \quad g \leqq v \leqq \min\left(\frac{1}{s},\ n\right)$$

$$= 0 \qquad\qquad v < g \text{ or } v > \min\left(\frac{1}{s},\ n\right)$$

so that the range of s values is partitioned as follows:

$$I_n:\ 0 \leqq s \leqq J(n)$$

$$I_v:\ J(v+1) \leqq s \leqq J(v)$$

$$I_g:\ J(g+1) \leqq s \leqq J(g) = \frac{1}{g}.$$

Here

$$J(v) = \frac{g}{v(g+1) - g}$$

and we have the result that class v will become fixed when s is an interior point of interval I_v, while classes v and $v \pm 1$ will coexist in a neutral equilibrium for s values on the boundary of intervals I_v and $I_{v\pm1}$. No pathogens survive when s exceeds $1/g$.

For the multiplicative case, the partitions are:

$$I_n:\ 0 \leqq s \leqq J(n)$$

$$I_v:\ J(v+1) \leqq s \leqq J(v)$$

$$I_g:\ J(g+1) \leqq s \leqq J(g) = 1$$

with

$$J(v) = \frac{g}{v}$$

and the same general statement about the effect of s on the equilibrium population can be made as in the additive case.

In all cases, weaker selection against unnecessary genes for virulence favors the evolution of more complex pathogen biotypes. The partitionings of s for the case of $n = 12$ and $g = 1, 2, 3, 4$ are shown in Table 2. These results confirm those of Groth (1976) and Marshall and Pryor (1978) for $g = 1$, and of Marshall and Pryor (1979) for $g \geqq 2$ for the multiplicative model.

The results differ slightly for the additive model because here we assume that fitnesses of all biotypes are measured relative to a biotype with no genes for virulence growing on a host with no genes for resistance; whereas, earlier authors have used as their standard a biotype with a single gene for virulence growing on a host with a single gene for resistance.

5.2 Mixtures of lines carrying 0 and g genes for resistance

We now consider the inclusion of a completely susceptible host in the mixture so that the multiline consists of class 0 and class g hosts. Such mixtures were advocated by Leonard (1969) on the grounds that the completely susceptible component would increase selection against complex pathogen races, improving the effectiveness of the multiline in disease control. Such a multiline can be parasitized by pathogens carrying v genes for virulence with $0 \leqq v < g$ attacking

Table 2. Subdivisions or range of selection parameter s to show which classes, v, of pathogens are fixed at equilibrium when every host has exactly g resistance genes chosen from n = 12 genes

v	Additive selection				
	g = 1	g = 2	g = 3	g = 4	g = 6
0	–	–	–	–	–
1	0.333–1.000	–	–	–	–
2	0.200–0.333	0.286–0.500	–	–	–
3	0.143–0.200	0.200–0.286	0.231–0.333	–	–
4	0.111–0.143	0.154–0.200	0.176–0.231	0.190–0.250	–
5	0.091–0.111	0.125–0.154	0.143–0.176	0.154–0.190	–
6	0.077–0.091	0.105–0.125	0.120–0.143	0.129–0.154	0.140–0.167
7	0.067–0.077	0.091–0.105	0.103–0.120	0.111–0.129	0.120–0.140
8	0.059–0.067	0.080–0.091	0.091–0.103	0.098–0.111	0.105–0.120
9	0.053–0.059	0.071–0.080	0.081–0.091	0.087–0.098	0.094–0.105
10	0.048–0.053	0.065–0.071	0.073–0.081	0.078–0.087	0.085–0.094
11	0.043–0.048	0.059–0.065	0.067–0.073	0.071–0.078	0.077–0.085
12	0.000–0.043	0.000–0.059	0.000–0.067	0.000–0.071	0.000–0.077
	Multiplicative selection				
0	–	–	–	–	–
1	0.500–1.000	–	–	–	–
2	0.333–0.500	0.667–1.000	–	–	–
3	0.250–0.333	0.500–0.677	0.750–1.000	–	–
4	0.200–0.250	0.400–0.500	0.600–0.750	0.800–1.000	–
5	0.167–0.200	0.333–0.400	0.500–0.600	0.667–0.800	–
6	0.143–0.167	0.286–0.333	0.429–0.500	0.571–0.667	0.857–1.000
7	0.125–0.143	0.250–0.286	0.375–0.429	0.500–0.571	0.750–0.857
8	0.111–0.125	0.222–0.250	0.333–0.375	0.444–0.500	0.667–0.750
9	0.100–0.111	0.200–0.222	0.300–0.333	0.400–0.444	0.600–0.667
10	0.091–0.100	0.182–0.200	0.273–0.300	0.364–0.400	0.545–0.600
11	0.083–0.091	0.167–0.182	0.250–0.273	0.333–0.364	0.500–0.545
12	0.000–0.083	0.000–0.167	0.000–0.250	0.000–0.333	0.000–0.500

the completely susceptible host, and classes $g \leqq v \leqq n$ attacking all hosts.

It is not difficult to see that classes v with $0 < v < g$ must be eliminated from the pathogen population since they are less fit than the class 0 pathogens on the completely susceptible host, and cannot grow on the other hosts. We may therefore remove them from further consideration.

A central question here is that of determining the conditions, if any, under which the pathogen class with zero genes for virulence becomes fixed in the pathogen population so that only the completely susceptible component of the multiline is diseasd. For pathogen biotypes with no genes for virulence ${}_0c_0 = w_0 = 1$, and (3) shows that $x_0(t+1) > x_0(t)$ with subsequent fixation of this class whenever ${}_0f > {}_0\bar{W}(t)$. In other words, fixation results when the relative contributions of hosts with no genes for resistance to the pathogen spore pool exceeds the mean fitness of the pathogens on that host class. Now the heavily diseased model is inappropriate in this context because, if only class 0 pathogens exist, class g hosts would make no contributions to the pathogen spore pool contrary to the requirement of equal contributions from all hosts.

In the lightly diseased case, the condition for fixation of the simplest biotype is that

$$X_0 > X_v \quad \text{for all } g \leqq v \leqq n$$

or that

$${}_0q > {}_0q\,{}_0c_v + {}_gq\,{}_gc_v \quad \text{for all } g \leqq v \leqq n .$$

For additive selection then, fixation of the zero class will occur when

$$1 \geqq s > J(0) = \max_{g \leqq v \leqq n} (A_v), \quad A_v = \frac{1}{v} \frac{{}_gq\binom{v}{g}}{{}_0q\binom{n}{g} + {}_gq\binom{v}{g}}$$

and, for multiplicative selection, when

$$1 \geqq s > J(0) = \max_{g \leqq v \leqq n} (M_v),$$

$$M_v = 1 - \left[\frac{{}_0q\binom{n}{g}}{{}_0q\binom{n}{g} + {}_gq\binom{v}{g}}\right]^{1/v}.$$

For s values less than J (0), class v pathogens will become fixed when

$$J(v) < s < J(v-1)$$

and, from a consideration of X_v values,

$$J(v)=\frac{{}_gq\left[\binom{v+1}{g}-\binom{v}{g}\right]}{(v+1)\left[{}_0q\binom{n}{g}+{}_gq\binom{v+1}{g}\right]-v\left[{}_0q\binom{n}{g}+{}_gq\binom{v}{g}\right]}$$

for additive selection, and

$$J(v)=\frac{{}_gq\left[\binom{v+1}{g}-\binom{v}{g}\right]}{{}_0q\binom{n}{g}+{}_gq\binom{v+1}{g}}$$

for multiplicative selection. As before, two pathogen classes may coexist in neutral equilibrium on the boundary of the regions in which each becomes fixed.

In the heavily diseased case, the equilibrium population may contain either one or two pathogen classes, except that classes $v<g$ cannot become fixed. Two situations are possible. Either class 0 and class $v \geqq g$ coexist, or classes $v<g$ are completely absent. In the latter case, class $v \geqq g$ may be fixed or two adjacent such classes may coexist. The choice between the two situations is made on the basis of the fitness function U, and numerical methods must be used because we cannot derive algebraic expressions to determine when class 0 is lost or retained with some other class.

For situations in which class 0 is lost, however, we can show that classes v and v + 1 coexist when

$$\frac{g\,{}_gq}{K(v)}<s<\frac{g\,{}_gq}{K(v)-g\,{}_0q}$$

and class v becomes fixed when

$$\frac{g\,{}_gq}{K(v)-g\,{}_0q}<s<\frac{g\,{}_gq}{K(v-1)}$$

where, for additive selection,

$$K(v)=(v+1)\,{}_0q+(v+1+vg)\,{}_gq$$

and, for multiplicative selection,

$$K(v)=v+1\,.$$

For situations in which class 0 persists, we can show that classes 0 and v coexist when, for the additive case,

$$\frac{v\binom{v+1}{g}-(v+1)\binom{v}{g}}{v(v+1)\left[\binom{v+1}{g}-\binom{v}{g}\right]}<s$$

$$<\frac{(v-1)\binom{v}{g}-v\binom{v-1}{g}}{v(v-1)\left[\binom{v}{g}-\binom{v-1}{g}\right]}$$

and, for the multiplicative case, when

$$\frac{\left[\binom{v+1}{g}-\binom{v}{g}\right][1-(1-s)^{v+1}]}{\binom{v+1}{g}}<s$$

$$<\frac{\left[\binom{v}{g}-\binom{v-1}{g}\right][1-(1-s)^{v}]}{\binom{v}{g}}.$$

Numerical results are shown in Tables 3 and 4. In Table 3, $g=1$ and the frequencies of the two host classes are either made equal ($_0q={}_1q=1/2$) or made proportional to the number of host types within each class ($_0q=1/13$, $_1q=12/13$). Similarly, for $g=2$ in Table 4, we impose $_0q=1/2$ or $_0q=1/67$. Under additive selection, we must restrict attention to $s \leqq 1/g$ to ensure class g hosts can support pathogens.

5.3 Mixtures of lines carrying 0, 1, ..., g genes for resistance

The final case we consider allows all host classes $0 \leqq r \leqq g$ to be present in the multiline, and employs the same methodology as above.

For the lightly diseased case, a consideration of the X_v functions shows that class 0 pathogens will become fixed whenever

$$1 \geqq s > J(0) = \max_{1 \leqq v \leqq n} (A_v)$$

with

$$A_v=\frac{1}{v}\frac{\sum_{r=1}^{g}\left[{}_rq\dfrac{\binom{v}{r}}{\binom{n}{r}}\right]}{\sum_{r=0}^{g}\left[{}_rq\dfrac{\binom{v}{r}}{\binom{n}{r}}\right]}$$

for additive selection, and

$$1 \geqq s > J(0) = \max_{1 \leqq v \leqq n} (M_v)$$

with

$$M_v=1-\left[\frac{{}_0q}{\sum_{r=0}^{g}{}_rq\dfrac{\binom{v}{r}}{\binom{n}{r}}}\right]^{1/v}$$

for multiplicative selection.

Table 3. Subdivisions of range of selection parameter s according to which classes, v, of pathogens are fixed at equilibrium when hosts have 0 or g = 1 resistance genes chosen from n = 12 genes

v	A. Lightly diseased case			
	Additive selection		Multiplicative selection	
	$_0q = \frac{1}{13}$	$_0q = \frac{1}{2}$	$_0q = \frac{1}{13}$	$_0q = \frac{1}{2}$
0	0.500–1.000	0.077–1.000	0.500–1.000	0.077–1.000
1	0.250–0.500	0.067–0.077	0.333–0.500	0.071–0.077
2	0.167–0.250	0.059–0.067	0.250–0.333	0.067–0.071
3	0.125–0.167	0.053–0.059	0.200–0.250	0.063–0.067
4	0.100–0.125	0.048–0.053	0.167–0.200	0.059–0.063
5	0.083–0.100	0.044–0.048	0.143–0.167	0.056–0.059
6	0.071–0.083	0.040–0.044	0.125–0.143	0.053–0.056
7	0.063–0.071	0.037–0.040	0.111–0.125	0.050–0.053
8	0.055–0.063	0.035–0.037	0.100–0.111	0.048–0.050
9	0.050–0.055	0.032–0.035	0.091–0.100	0.046–0.048
10	0.046–0.050	0.030–0.032	0.083–0.091	0.044–0.046
11	0.042–0.046	0.029–0.030	0.077–0.083	0.042–0.044
12	0.000–0.042	0.000–0.029	0.000–0.077	0.000–0.042

v	B. Heavily diseased case			
	Additive selection		Multiplicative selection	
	$_0q = \frac{1}{13}$	$_0q = \frac{1}{2}$	$_0q = \frac{1}{13}$	$_0q = \frac{1}{2}$
0, 1	0.927–1.000	0.500–1.000	0.927–1.000	0.500–1.000
1	0.324–0.927	0.250–0.500	0.480–0.927	0.333–0.500
1, 2	0.315–0.324	0.200–0.250	0.462–0.480	0.250–0.333
2	0.194–0.315	0.143–0.200	0.316–0.462	0.200–0.250
2, 3	0.190–0.194	0.125–0.143	0.307–0.316	0.167–0.200
3	0.138–0.190	0.100–0.124	0.235–0.307	0.143–0.167
3, 4	0.135–0.138	0.091–0.100	0.230–0.235	0.125–0.143
4	0.107–0.135	0.077–0.091	0.188–0.230	0.111–0.125
4, 5	0.105–0.107	0.071–0.077	0.184–0.188	0.100–0.111
5	0.088–0.105	0.063–0.071	0.156–0.184	0.091–0.100
5, 6	0.083–0.088	0.059–0.063	0.154–0.156	0.083–0.091
6	0.074–0.083	0.053–0.059	0.133–0.154	0.077–0.083
6, 7	0.074	0.050–0.053	0.131–0.133	0.071–0.077
7	0.064–0.073	0.046–0.050	0.118–0.131	0.067–0.071
7, 8	0.064	0.044–0.046	0.115–0.118	0.063–0.067
8	0.057–0.064	0.040–0.044	0.105–0.115	0.059–0.063
8, 9	0.056–0.057	0.039–0.040	0.103–0.105	0.056–0.059
9	0.051–0.056	0.036–0.039	0.093–0.103	0.053–0.056
9, 10	0.050–0.051	0.051–0.051	0.092–0.093	0.050–0.053
10	0.046–0.050	0.032–0.035	0.085–0.092	0.048–0.050
10, 11	0.046	0.031–0.032	0.083–0.085	0.046–0.048
11	0.042–0.046	0.030–0.031	0.077–0.083	0.044–0.046
11, 12	0.041–0.042	0.029–0.030	0.077	0.042–0.044
12	0.000–0.041	0.000–0.029	0.000–0.077	0.000–0.042

For s values less than J (0), class v pathogens will become fixed when

$$J(v) < s < J(v-1)$$

where, for additive selection

$$J(v) = \frac{\sum_{r=0}^{g} \frac{{}_rq}{\binom{n}{r}} \left[\binom{v+1}{r} - \binom{v}{r} \right]}{\sum_{r=0}^{g} \frac{{}_rq}{\binom{n}{r}} \left[(v+1)\binom{v+1}{r} - v\binom{v}{r} \right]}$$

and, for multiplicative selection,

$$J(v) = \frac{\sum_{r=0}^{g} \frac{{}_rq}{\binom{n}{r}} \left[\binom{v+1}{r} - \binom{v}{r} \right]}{\sum_{r=0}^{g} \frac{{}_rq}{\binom{n}{r}} \binom{v+1}{r}}.$$

We do not provide any algebraic details for the heavily diseased case, but show some numerical results in Table 5. Host proportions for the three classes are either made equal ($_0q = {}_1q = {}_2q = 1/3$) or made proportional to the numbers of host types within each class ($_0q = 1/79$, $_1q = 12/79$, $_2q = 66/79$).

6 Discussion

This study extends previous analyses of the effects of the widespread cultivation of dirty crop multilines on the evolution of virulence in obligate parasites (e.g. Groth 1976; Marshall and Burdon 1981) to mixtures of lines carrying different numbers of resistance genes. It is assumed throughout that where hosts carry multigenic resistances, they involve "overlapping gene sets" (Marshall and Pryor 1979), and every resistance gene is represented equally in each host class. However, apart from this simplifying assumption, the models used allowed for great generality in the makeup of a multiline. The overall conclusion from these studies is that the composition of the pathogen population at equilibrium on a multiline may vary within wide limits and the principal determinant of its composition is the number of lines in the mixture and the resistance genes they carry.

Several variations of the model were analyzed to assess the effects of specific parameters on pathogen evolution. The first dichotomy allowed in the model was that relating to host contribution to the pathogen spore pool. Under the "lightly diseased" alternative, it was assumed that the relative contribution of each host genotype to the total pathogen spore pool is proportional to the mean fitness of the pathogen races to which it is susceptible. In contrast, under the "heavily diseased" alternative, it was assumed that all hosts, regardless of the number of resistance genes they carry, contribute equal-

Table 4. Subdivisions of range of selection parameter s according to classes, v, of pathogens fixed in equilibrium populations when hosts have 0 or g = 2 resistance genes chosen from n = 12 genes

A. Lightly diseased case

Additive selection				Multiplicative selection			
$_0q = \frac{1}{67}$		$_0q = \frac{1}{2}$		$_0q = \frac{1}{67}$		$_0q = \frac{1}{2}$	
s	v	s	v	s	v	s	v
0.250–1.000	0	0.042–1.000	0	0.385–1.000	0	0.008–1.000	0
0.188–0.250	3	0.000–0.042	12	0.364–0.385	4	0.000–0.008	12
0.148–0.188	4			0.313–0.364	5		
0.122–0.148	5			0.273–0.313	6		
0.103–0.122	6			0.241–0.273	7		
0.090–0.103	7			0.216–0.241	8		
0.079–0.090	8			0.196–0.216	9		
0.071–0.079	9			0.179–0.196	10		
0.064–0.071	10			0.164–0.179	11		
0.059–0.064	11			0.000–0.164	12		
0.000–0.059	12						

B. Heavily diseased case

Additive selection				Multiplicative selection			
$_0q = \frac{1}{67}$		$_0q = \frac{1}{2}$		$_0q = \frac{1}{67}$		$_0q = \frac{1}{2}$	
s	v	s	v	s	v	s	v
0.495–0.500	0, 2	0.250–0.500	0, 2	0.900–1.000	0, 2	0.456–1.000	0, 3
0.285–0.495	2	0.167–0.250	0, 3	0.660–0.900	2	0.356–0.456	0, 4
0.283–0.285	2, 3	0.125–0.167	0, 4	0.657–0.660	2, 3	0.291–0.356	0, 5
0.200–0.283	3	0.100–0.125	0, 5	0.494–0.657	3	0.246–0.291	0, 6
0.198–0.200	3, 4	0.083–0.100	0, 6	0.493–0.494	3, 4	0.213–0.246	0, 7
0.154–0.198	4	0.071–0.083	0, 7	0.395–0.493	4	0.188–0.213	0, 8
0.153–0.154	4, 5	0.062–0.071	0, 8	0.394–0.395	4, 5	0.168–0.188	0, 9
0.125–0.153	5	0.056–0.062	0, 9	0.329–0.394	5	0.152–0.168	0, 10
0.124–0.125	5, 6	0.050–0.056	0, 10	0.328–0.329	5, 6	0.139–0.152	0, 11
0.106–0.124	6	0.045–0.050	0, 11	0.282–0.328	6	0.057–0.139	0, 12
0.104–0.106	6, 7	0.042–0.045	0, 12	0.281–0.282	6, 7	0.000–0.057	12
0.091–0.104	7	0.000–0.042	12	0.247–0.281	7		
0.090–0.091	7, 8			0.246–0.247	7, 8		
0.080–0.090	8			0.219–0.246	8		
0.079–0.080	8, 9			0.219	8, 9		
0.071–0.079	9			0.197–0.219	9		
0.071	9, 10			0.197	9, 10		
0.064–0.071	10			0.179–0.197	10		
0.064	10, 11			0.179	11, 11		
0.059–0.064	11			0.164–0.179	11		
0.058–0.059	11, 12			0.164	11, 12		
0.000–0.058	12			0.000–0.164	12		

ly to the pathogen spore pool. These different assumptions led to markedly different conclusions. A lightly diseased multiline was shown to lead generally to the fixation of a single pathogen class, while a heavily diseased multiline could accommodate as many pathogen classes as there were host classes in the mixture. Under either assumption, the particular pathogen classes comprising the equilibrium pathogen population depended on all other variables of the model. In practice, the level of disease on a crop will vary over both locations and years, as well as throughout the season at any one location/year, depending on a wide range of biotic and climatic variables. Therefore, we might expect the situation in the field to fluctuate between the extremes considered in our models.

The second dichotomy allowed in the model was that relating to the way unnecessary genes for virulence combine to reduce pathogen fitness. Little qualitative difference resulted

Table 5. Subdivisions of range of selection parameter s according to which classes, v, of pathogens are fixed at equilibrium when hosts have 0, 1, 2, resistance genes chosen from n = 12 genes

A. Lightly diseased case

	Additive selection		Multiplicative selection	
v	${}_0q = \frac{1}{79}, {}_1q = \frac{12}{79}$	${}_0q = {}_1q = \frac{1}{3}$	${}_0q = \frac{1}{79}, {}_1q = \frac{12}{79}$	${}_0q = {}_1q = \frac{1}{3}$
0	0.500–1.000	0.077–1.000	0.500–1.000	0.088–1.000
1	0.334–0.500	–	–	–
2	0.231–0.334	0.075–0.077	0.429–0.500	–
3	0.174–0.231	0.071–0.075	0.364–0.429	–
4	0.139–0.174	0.067–0.071	0.313–0.364	–
5	0.116–0.139	0.063–0.067	0.273–0.313	–
6	0.099–0.116	0.059–0.063	0.241–0.273	–
7	0.087–0.099	0.055–0.059	0.216–0.241	–
8	0.076–0.087	0.052–0.055	0.196–0.216	–
9	0.069–0.076	0.049–0.052	0.179–0.196	0.087–0.088
10	0.062–0.069	0.046–0.049	0.164–0.179	0.085–0.087
11	0.057–0.062	0.044–0.046	0.152–0.164	0.083–0.085
12	0.000–0.057	0.000–0.044	0.000–0.152	0.000–0.083

B. Heavily diseased case

Additive selection				Multiplicative selection			
${}_0q = \frac{1}{79}, {}_1q = \frac{12}{79}$		${}_0q = {}_1q = \frac{1}{3}$		${}_0q = \frac{1}{79}, {}_1q = \frac{12}{79}$		${}_0q = {}_1q = \frac{1}{3}$	
s	v	s	v	s	v	s	v
0.478–0.500	1, 2	0.335–0.500	1, 2	0.919–1.000	0, 1, 2	0.669–1.000	0, 1, 2
0.280–0.478	2	0.238–0.335	2	0.913–0.919	0, 2	0.475–0.669	0, 2
0.275–0.280	2, 3	0.206–0.238	2, 3	0.636–0.913	2	0.383–0.475	0, 2, 3
0.195–0.275	3	0.158–0.206	3	0.608–0.636	2, 3	0.309–0.383	0, 3
0.193–0.195	3, 4	0.144–0.158	3, 4	0.470–0.608	3	0.266–0.309	0, 3, 4
0.150–0.193	4	0.121–0.144	4	0.456–0.470	3, 4	0.243–0.266	0, 4
0.149–0.150	4, 5	0.113–0.121	4, 5	0.374–0.456	4	0.235–0.243	4
0.122–0.149	5	0.097–0.113	5	0.365–0.374	4, 5	0.211–0.235	4, 5
0.121–0.122	5, 6	0.093–0.097	5, 6	0.308–0.365	5	0.207–0.211	0, 4, 5
0.103–0.121	6	0.082–0.093	6	0.305–0.308	5, 6	0.199–0.207	0, 5
0.102–0.103	6, 7	0.078–0.082	6, 7	0.264–0.305	6	0.189–0.199	5
0.089–0.102	7	0.070–0.078	7	0.262–0.264	6, 7	0.168–0.189	5, 6
0.088–0.089	7, 8	0.067–0.070	7, 8	0.231–0.262	7	0.158–0.168	6
0.078–0.088	8	0.062–0.067	8	0.229–0.231	7, 8	0.144–0.158	6, 7
0.070–0.078	9	0.060–0.062	8, 9	0.204–0.229	8	0.136–0.144	7
0.063–0.070	10	0.055–0.060	9	0.203–0.204	8, 9	0.127–0.136	7, 8
0.058–0.063	11	0.053–0.055	9, 10	0.184–0.203	9	0.120–0.127	8
0.057–0.058	11, 12	0.049–0.053	10	0.183–0.184	9, 10	0.113–0.120	8, 9
0.000–0.057	12	0.048–0.049	10, 11	0.167–0.183	10	0.106–0.113	9
		0.045–0.048	11	0.166–0.167	10, 11	0.102–0.106	9, 10
		0.044–0.045	11, 12	0.154–0.166	11	0.098–0.102	10
		0.000–0.044	12	0.153–0.154	11, 12	0.093–0.096	10, 11
				0.000–0.153	12	0.088–0.093	11
						0.084–0.088	11, 12
						0.000–0.084	12

from allowing selection against unnecessary virulence genes to be additive or multiplicative. There is also little quantitative difference with slight selection pressure (Barrett and Wolf 1978) between the two systems. However, for larger values of s, there are important quantitative differences between these models. In particular, higher levels of selection are required under the multiplicative model than the additive model to prevent the development of complex races on multilines composed of overlapping gene sets. This result confirms the previous findings of Marshall and Burdon (1981).

The third dichotomy allowed in our model which we have not considered previously, but which is worthy of some com-

ment here, relates to the relative fitness of different pathogen classes on different host classes. Under the model considered above, it was assumed that selection operated as soon as a pathogen carried a virulence gene and, as a result, the relative fitness of a pathogen class was the same on all host classes. As an alternative, it may be assumed that selection acts only against unnecessary virulence genes on each host class, so that the fitness of the same pathogen class will vary among host classes. Under the latter assumption, the non-zero fitness values of the vth pathogen class on the rth host class are, for the additive model,

$${}_rw_v = \{1 - (v - r)\, s\}$$

instead of (section 3.2)

$${}_rw_v = (1 - vs)$$

and, for the multiplicative model,

$${}_rw_v = (1 - s)^{(v-r)}$$

instead of

$${}_rw_v = (1 - s)^v$$

as before.

The relative fitnesses of a number of combinations of host/pathogen classes are given in Table 6 to illustrate the difference in those assumptions. Once again, no qualitative difference results from this change in the model. However, it does lead to important quantitative differences and significantly increases the levels of stabilizing selection required to ensure simple races dominate the pathogen population when the multiline contains more than one host class. We do not know which of these assumptions more closely approaches the practical situation in the field. This lack of knowledge underlines the need for experimental data on selection against unnecessary genes for virulence-how often it occurs, how strong it is, and how the selective forces at two or more loci interact to reduce pathogen fitness.

An important feature of our model is that it allows a rigorous and detailed examination of the effects of the inclusion of a completely susceptible component in a multiline on pathogen evolution. Leonard (1969) suggested that a completely susceptible component in a multiline would help stabilize the equilibrium racial composition of a pathogen population with simple races predominant. On the basis of estimates of the relative fitnesses of virulent versus avirulent strains of stem rust fungus for five common resistance genes in oats, Leonard (1969) calculated 35–40% of a completely susceptible component was required in the host population to ensure maintenance of simple races in the pathogen population. The present results confirm that the inclusion of a susceptible component in the multiline will materially assist the maintenance of simple races in the equilibrium pathogen population. Moreover, they indicated, as we might expect, that the levels of stabilizing selection against unnecessary genes for virulence required to ensure the survival of simple races in the pathogen population decreased as the proportional of susceptible components included in the mixture increased (Table 7). However, it is not until the level of completely susceptible components reaches 30–40% or more that the levels of stabilizing selection required to ensure the survival of simple races fall to biologically realistic values of 10% or less (Leonard 1977) where the resistant components carry either one or two genes for resistance. This figure is in remarkably

Table 6. Relative fitnesses ${}_rw_v$ of various pathogen classes on different host classes assuming unnecessary genes for virulence combine additively to reduce pathogen fitness, for two contrasting models

1. Relative fitness of each pathogen class indentical on each compatible host class

Pathogen class	Host class (r)[a]			
(r)[a]	0	1	2	3
0	1	0	0	0
1	1–s	1–s	0	0
2	1–2s	1–2s	1–2s	0
3	1–3s	1–3s	1–3s	1–3s
4	1–4s	1–4s	1–4s	1–4s

2. Relative fitness of each pathogen class varies on each compatible host class

Pathogen class	Host class (r)			
(v)	0	1	2	3
0	1	0	0	0
1	1–s	1	0	0
2	1–2s	1–s	1	0
3	1–3s	1–2s	1–s	1
4	1–4s	1–3s	1–2s	1–s

[a] r and v represent the number of resistance and virulence genes, respectively, carried by the r-th host class and the v-th pathogen class

Table 7. Minimum strength of selection, s, needed to ensure maintenance of class 0 pathogens on multiline containing classes 0 and g hosts

0^q	Additive selection		Multiplicative selection	
	g=1	g=2	g=1	g=2
0.1	0.429	0.115	0.429	0.179
0.2	0.250	0.080	0.250	0.126
0.3	0.163	0.062	0.163	0.096
0.4	0.111	0.051	0.111	0.074
0.5	0.077	0.042	0.077	0.056
0.6	0.053	0.033	0.053	0.042
0.7	0.034	0.025	0.034	0.029
0.8	0.020	0.017	0.020	0.018
0.9	0.009	0.008	0.009	0.009

good agreement with Leonard's (1969) original estimate. It is clear that because the completely susceptible component is open to attack by all races, the increased selection against complex races which results from its inclusion in the multiline, will be achieved only at considerable cost in terms of the levels of disease on the crop. Whether the benefits obtained are likely to outweigh this cost will be revealed only by careful experimental studies on the effects of the inclusion of various levels of completely susceptible plants in multiline mixtures on subsequent disease levels and damage in the crop.

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