ON THE THEORY OF SPECIATION INDUCED BY TRANSPOSABLE ELEMENTS

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ABSTRACT

A simple methematical model describes the invasion of panmictic, sexually reproducing populations by a newly introduced transposon. The model places important constraints on the properties that transposons must have to successfully invade a population and describes the kinetics with which such an invasion will occur. Invasibility conditions serve as a basis for new, detailed scenarios whereby transposon-mediated depression in fitness produces reproductive isolation of populations. These scenarios, in turn, lead to several speculations concerning the role of transposons in evolution.

A mechanism was recently suggested (BINGHAM, KIDWELL and RUBIN 1982) whereby transposable elements causing various degrees of hybrid sterility could be responsible for speciation (see ROSE and DOOLITTLE 1983, for a general review). The proposed scenario in its simplest form consists of two steps:

1. The genomes of two geographically isolated populations are independently "contaminated" by different transposable elements. These "infections" spread in their respective populations in spite of the loss in fitness shown by the appropriate individuals carrying the transposon. (This loss of fitness in the well-characterized cases of the *P-M* and *I-R* dysgenic systems consists of substantially reduced fertility among some individuals carrying the transposons in question.)

2. Effective reproductive isolation of these two populations (speciation) follows even if they become subsequently sympatric as a result of a loss of fitness of hybrids due to the cumulative effects of a combination of unrelated transposable elements.

We consider here a simple mathematical model of the spread of a transposable element in a large sexually reproducing, panmictic population. Under the conditions of the model, we find that a transposable element will invade a population even if it reduces the fitness of hybrid individuals carrying it provided that the fitness loss is not greater than a certain critical value. This critical value is, in turn, determined by the "infectivity" of the transposon. Our model also predicts the kinetics of the invasion of a natural population by a

transposon. We discuss the implications of the results of our analysis to models of reproductive isolation of populations induced by transposable elements.

THE MODEL

The model is a conventional deterministic one-locus, two-allele system describing the evolution of a panmictic population and amended by a description of the transposition process. A gamete is assumed to be either "infected" by the transposable element or not infected. (Notice that transposition is probably duplicative in the cases of many, if not all, transposons. The assumption that the transposon-bearing gamete can infect the nontransposon-bearing gamete without undergoing alteration itself is thus justified.) This approximation of a population as having only two types of gametes does not, of course, take into account the dynamics of the copy number within a genome, nor does it take into account recombination and the multilocus character of the process. We discuss, however, reasons for believing that the model is a valuable and predictive approximation.

We assume that a noninfected gamete becomes infected with the probability (infection or transposition rate), $(0 < \beta < 1)$, when paired in a zygote with the infected gamete. Two alternative assumptions will be made for the loss of fitness in hybrids. In one case, the loss [reduction of fitness from 1 to $(1 - S)$] occurs only in the female dysgenic individual (analogous to the *I-R* system; see BREGLIANO and KIDWELL 1982 for a review). In the other case, the same loss occurs independently of the sex of the dysgenic individual (analogous to the *P-M* system, BREGLIANO AND KIDWELL 1982). The *P-M*-motivated model is composed of the following set of recurrent equations:

$$\begin{aligned} P_f' &= \frac{2P_fP_m + M_fP_m(1 - S)(1 + \beta) + P_fM_m}{2[P_fP_m + M_fP_m(1 - S) + P_fM_m + M_fM_m]} \\ P_m' &= P_f' \\ M_f' &= \frac{2M_fM_m + M_fP_m(1 - S)(1 + \beta) + P_fP_m}{2[P_fP_m + M_fP_m(1 - S) + P_fM_m + M_fM_m]} \\ M_m &= M_f' \end{aligned} \tag{1}$$

where P_f and P_m are frequencies of the *P*-carrying gametes within the female and male subpopulations and M_f and M_m are complementary frequencies of noninfected gametes within two sexes. Frequencies are normalized within sexes so that

$$P_f + M_f = 1$$

$$P_m + M_m = 1$$

Unprimed symbols represent generation k, whereas primed symbols represent generation $k + 1$.

The *I-R*-motivated model is a bit more complex; it consists of four recurrent equations for the frequencies of *I* element within two sexes.

$$I_f' = \frac{2I_fI_m + R_fI_m(1 - S)(1 + \beta) + I_fR_m}{2[I_fI_m + R_fI_m(1 - S) + I_fR_m + R_fR_m]}$$

$$I_m' = \frac{2I_fI_m + R_fI_m + I_fR_m}{2[I_fI_m + R_fI_m + I_fR_m + R_fR_m]}$$

$$R_f' = \frac{2R_fR_m + R_fI_m(1 - S)(1 + \beta) + I_fR_m}{2[I_fI_m + R_fI_m(1 - S) + I_fR_m + R_fR_m]}$$

$$R_m' = \frac{2R_fR_m + R_fI_m + I_fR_m}{2[I_fI_m + R_fI_m + I_fR_m + R_fR_m]}$$

Only two of the equations are independent since

$$I_f + R_f = 1$$

$$I_m + R_m = 1$$

RESULTS

We first consider invasibility conditions. It is easy to show (APPENDIX I) that in the first model, the condition for the spread of the transposable element is

$$\beta > \frac{S}{1 - S} \tag{3}$$

For the second model, the similar condition is (see APPENDIX II)

$$\beta > \frac{S}{2 - S} \tag{4}$$

The shaded area in Figure 1 shows the combinations of the transposition rate, β, and the fitness loss, S, for which the spread of the new element is possible. A typical curve of the spread of the new element in a population is shown in Figure 2.

Table 1 gives the time of the spread of a new element as a function of two parameters, β and S, evaluated from the *P-M* model. Asterisks denote combinations when spread is impossible. The same kind of results for the *I-R* model can be seen in Table 2. The process is somewhat faster in the case of the *I-R* model, but it is extremely fast in both cases. For parameters very close to the noninvasibility boundary, a typical curve shows a very low frequency for about 90% of the time with noticeable growth over a very short period of the order of 50–70 generations.

When the combination of parameters, β and S, tends to the borderline of allowable combinations set by inequalities (3) and (4), the time of fixation tends to infinity. Practically, as Tables 1 and 2 show, except for a very narrow region immediately surrounding the noninvasibility boundary, it takes typically of the order of 100 generations for a new transposable element to be fixed in a population of 1,000,000 individuals. The process is clearly very rapid.

It should be noted that our model is deterministic. The conditions for in-

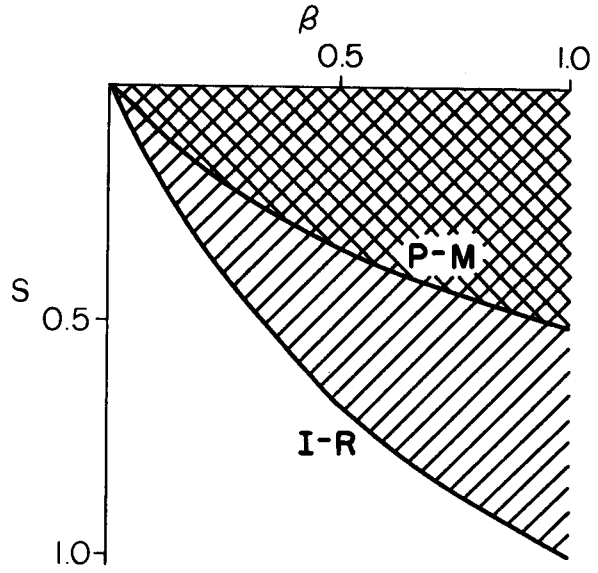

FIGURE 1.—Conditions of invasibility of a transposable element for the *P-M* and *I-R* models. Shaded area corresponds to the combination of parameters β and s that allow invasion. The conditions are clearly more strict for the *P-M* system. Note that independently of β, the *P* element cannot invade if the loss of fitness is greater than 50% ($s = 0.5$). This is intuitively reasonable since, with maximal infectivity ($\beta = 1$), the number of element-carrying gametes doubles at each generation. With an overall loss of more than 50% of offspring, heterozygote disadvantage overrides infectivity and the element is lost.

vasion, (3) and (4), are, therefore, to be viewed as necessary but not sufficient for invasion of a real, finite population. This results from the fact that our model assumes infinite population size, whereas in finite populations it is possible for a newly invading element to be lost as a result of purely stochastic processes.

A much more complex model involving drift in finite populations developed by WALSH (1982), in which meiotic drive works as a formal equivalent of our transposition process, produces a probable rapid fixation in cases of extreme meiotic drive.

The model that we have presented is clearly simplified. However, the results are, to a large extent, insensitive to the parameters β and S as long as they are well within invasibility conditions. This relative insensitivity leads us to believe that the qualitative features of the process will be preserved in a more complex and realistic model. Our treatment is most likely to be inappropriate when segregation of transposon copies is a major factor determining distribution of transposon copies during an invasion. Such a case clearly exists when the transposition rate of a transposon is relatively low. However, a transposon with a relatively low transposition rate (relatively low infectivity) must also be associated with a relatively small depression in hybrid fitness if it is to successfully invade the population. Although such transposons may, of course, exist, they are less likely (due to their small effects on hybrid fitness) to play a central role in reproductively isolating populations and are, therefore, of less interest

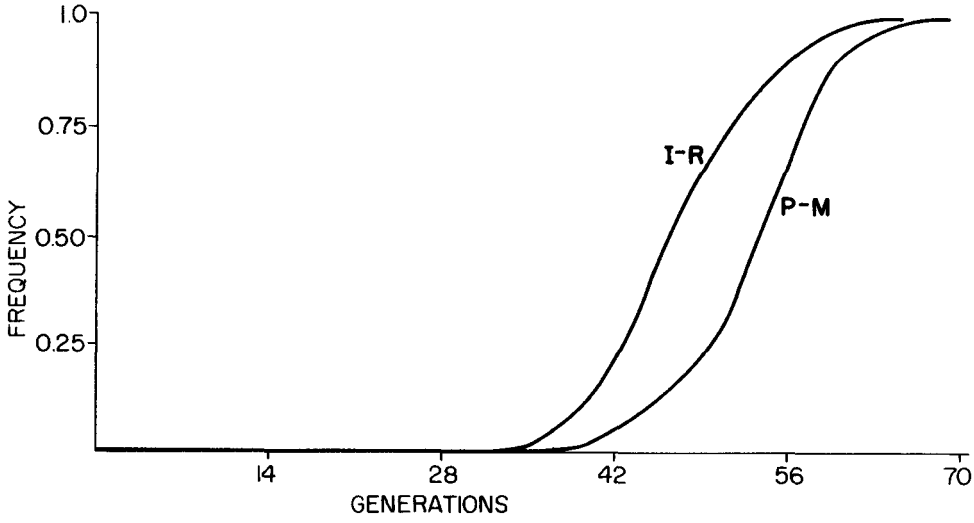

FIGURE 2.—Typical curves for the spread of transposable elements through a population. Two curves for the *P-M* and *I-R* models are shown for the same values of parameters $s = 0.1$ and $\beta = 0.9$ within invasibility conditions for both models. If an initial frequency of 10^{-6} is the starting point, less than 70 generations are required on each model (68 for the *P-M* case and 64 for *I-R*) for the element to reach a frequency of 99.9999% ($1-10^{-6}$). Note that the process can be practically invisible for approximately half of this time period. When parameters and s are chosen closer to the borderline of invasibility conditions, the "practically invisible" portions of the invasion curve can become quite long. The "visible" portion of the curve appears very similar for a variety of combinations of parameters.

TABLE 1

Time of the spread of a new transposable element evaluated from the P-M *model (in generations)*

	β									
S	0.1	0.2	0.3	0.4	0.5	0.6	0.7	0.8	0.9	1.0
0.0	551	276	184	139	110	92	79	69	61	55
0.1	*	421	232	164	128	105	89	78	68	61
0.2	*	*	644	264	176	135	110	93	81	72
0.3	*	*	*	*	499	243	168	131	109	93
0.4	*	*	*	*	*	*	1051	321	202	152
0.5	*	*	*	*	*	*	*	*	*	*
0.6	*	*	*	*	*	*	*	*	*	*
0.7	*	*	*	*	*	*	*	*	*	*
0.8	*	*	*	*	*	*	*	*	*	*
0.9	*	*	*	*	*	*	*	*	*	*
1.0	*	*	*	*	*	*	*	*	*	*

Initial frequency, $P_0 = 10^{-6}$; final frequency, $P_{final} = (1-10^{-6})$.

in this context than are those that produce more substantial degrees of hybrid fitness loss (and that, consequently, must be associated with higher levels of infectiousness to exist).

The data of KIDWELL (1983) describing the apparent spread of transposable

TABLE 2

Time of the spread of a new transposable element evaluated from the I-R *model*

	β									
S	0.1	0.2	0.3	0.4	0.5	0.6	0.7	0.8	0.9	1.0
0.0	551	276	184	139	110	92	79	69	61	55
0.1	758	306	198	145	116	96	82	71	64	57
0.2	*	408	226	160	124	102	87	75	66	59
0.3	*	1121	295	186	138	111	93	79	70	62
0.4	*	*	581	242	163	125	102	86	74	66
0.5	*	*	*	447	215	148	116	95	81	71
0.6	*	*	*	*	412	203	142	111	91	78
0.7	*	*	*	*	*	456	207	141	109	89
0.8	*	*	*	*	*	*	753	233	148	111
0.9	*	*	*	*	*	*	*	*	336	172
1.0	*	*	*	*	*	*	*	*	*	*

Initial frequency, $I_0 = 10^{-6}$; final frequency $I_{final} = (1-10^{-6})$.

elements in natural *Drosophila melanogaster* populations suggests a period of invasion of the order of 20–30 yr. Moreover, the work of BERG (1982) on the mutability peaks in the natural populations of *D. melanogaster* support the retrospective studies of KIDWELL (1983). In particular, the two periods when mutation rates in natural fly populations were an order of magnitude higher than at other times (first in the 1930s and second in the 1960s) coincide with the periods of apparent invasion of the *P* element and *I* element reported by KIDWELL. Strong increases in mutability associated with the hybridization of some strains differentiated with respect to a transposable element are well established (see BINGHAM, KIDWELL and RUBIN 1982; RUBIN, KIDWELL and BINGHAM 1982 and references therein). The reasons for believing that the *P* element transposon has recently invaded the wild *D. melanogaster* population have been recently reviewed by BINGHAM, KIDWELL and RUBIN (1982) and by KIDWELL (1983).

The kinetics of apparent invasion of *D. melanogaster* by the *P* and *I* element transposons (KIDWELL 1983) are in good agreement with those that our model predicts.

THE MODEL AND SPECIATION

The model for transposon-mediated reproductive isolation of populations proposed by BINGHAM, KIDWELL and RUBIN (1982) supposes that each of two geographically isolated populations are invaded by several separate transposon families whose *collective* effects on hybrid fitness are so severe (complete or very nearly complete sterility) as to effectively isolate the gene pools of the two populations from one another should they again become sympatric. Under the conditions of this model, a *single* transposon that can isolate two otherwise identical populations is presumed to be incapable of initial invasion. That this last, intuitive argument is likely to be correct in the simplest case is strongly supported by our results (see preceding data).

Although this model is conceptually simple, it may be unnecessarily restrictive in requiring numerous independent invasions (or losses). The requirement for numerous invasions can be eliminated in at least two new scenarios by allowing simple alternatives to assumptions implicit in this earlier model.

The following general considerations are important as background to these two new scenarios. A number of cases are well characterized in which interspecies hybrids are partially fertile. Thus, actual initial isolating mechanisms associated with speciation may give rise to incomplete hybrid sterility in some cases. The scenarios we describe allow transposon-mediated sterility to produce stable, partial reproductive isolation. Furthermore, we note that gene flow between two populations can be prevented even if hybrids are formed and are partially fertile if the hybrid is sufficiently less fit than either of the parental populations. Under these circumstances, alleles at loci relatively tightly linked to alleles responsible for depression in hybrid fitness (transposon copies in the cases to be described) cannot flow from one population to the other. [The reader is referred to BAZYKIN (1969) and SLATKIN (1973) for discussions of formation of stable hybrid zones resulting from heterozygote inferiority.]

The first implicit assumption leading to a requirement for numerous invasions for reproductive isolation was that the fitness depression (S) in hybrids is not affected by environmental factors and is constant throughout the range of the population. However, the behavior of the relatively well-characterized *P-M* and *I-R* systems in Drosophila suggests that this assumption may not be generally correct in the case of ectothermic organisms. In particular, *P-M* dysgenic hybrids are often completely sterile at relatively high temperatures (greater than ca. 27°) but usually show very little sterility below ca 22°. On the other hand, *I-R* dysgenic hybrids are most severely sterile at relatively low temperatures (ca. 18°) but show relatively little sterility above ca. 22°.

Based on these properties of the *P-M* and *I-R* systems, we imagine a possible scenario for division of a population into two reproductively isolated populations. Specifically, imagine that a "cold-tolerant" element (such as *P*) initiates an invasion near the colder extreme of the range of an ectothermic species and a "heat-tolerant" element (such as *I*) initiates an invasion near the warmer extreme of the range. The cold-tolerant element invades that portion of the range of the host species that is cold enough to allow less than complete (or very nearly complete) sterility in hybrids dysgenic for the element, and the heat-tolerant element invades the corresponding, warm portion of the range of the species. If the ranges of the two elements partially overlap or precisely abut so that no portion of the species range fails to be populated by at least one of the two elements, the species is divided into two isolated subpopulations corresponding to that portion of the species range uniquely occupied by one of the two elements but not the other. That portion of the species range occupied by both elements will behave as a hybrid zone between them; gene flow from each isolated subpopulation into the hybrid zone will occur, but gene flow out of this hybrid zone will not occur. If this detailed mechanism does, in fact, frequently participate in ectotherm speciation one might frequently find hybrid zones involving these species to be isotherms.

A second assumption that led BINGHAM, KIDWELL and RUBIN (1982) to require numerous invasions to produce reproductive isolation supposes that transposon families act independently of one another, both with respect to transposition frequency and to depression in fitness in the appropriate hybrids. A new class of mechanisms allowing pairs (or small numbers) of newly invading transposon families to reproductively isolate populations without any one of them alone causing full sterility ($S = 1$) results from relaxing this assumption. One can imagine that two transposons interact such that, when one is present in a hybrid dysgenic for the other, the severity of the associated fitness depression (S) is greater than or the infectivity (β) for either of the elements is less than in a comparable dysgenic hybrid that lacks the other element. No detailed precedent for this kind of epistatic interaction is known to us, but cross-reactions involving transposases or regulatory proteins of two transposon families with a relatively recent common ancestor is easy to imagine. Under this set of circumstances, any member of such an interacting set of transposons might invade a noninfected population while being unable to invade a population in which another member of the interacting set is already present. In the case of epistatic interactions of this sort, if invasions of a population by each of two such interacting elements are initiated effectively simultaneously, the invaded population will ultimately be divided into two isolated subpopulations corresponding to the portions of the population occupied by one or the other element. It is noteworthy that this scenario can produce fully sympatric speciation.

It has been frequently suggested that two subpopulations isolated by postmating barriers will sometimes diverge from one another as a result of character displacement, thus producing fully isolated species pairs. (This tendency is sometimes, though apparently inappropriately, referred to as the "Wallace effect"; see LITTLEJOHN 1981 for a review.) Although views on the importance of this kind of reinforcement process in real populations differ, such a mechanism seems plausible. RAPOPORT (1982, pp. 118–123) reports that about one-half of mammalian subspecies boundaries (apparently stable hybrid zones) in North America occur in the absence of a discernible environmental rationale. Perhaps mechanisms of the sort discussed earlier can account for some fraction of these cases of stable hybrid zones. We further note that, although the idea that stable boundaries between subspecies may be preserved by hybrid inferiority is not new, our suggestion that hybrid inferiority resulting from the existence of subspecies-specific transposon families may be responsible for such stable hybrid zones is apparently new.

SPECULATIONS ON A GENERAL VIEW OF EVOLUTION

As discussed earlier there is substantial circumstantial evidence that transposon families invade natural populations and that such invasions, when they occur, are very rapid. Moreover, such invasions may be quite common as suggested by the apparent invasion of natural *D. melanogaster* populations by two different transposon families (the *P* and *I* element families, see preceding data) in this century. These common, rapid invasions are apparently frequently

associated with hybrid sterility and, if they are responsible for at least some speciation events, those speciation events are likely to be frequent.

We wish to note several specific implications of this hypothesis that have apparently not been pointed out previously.

First, the history of a particular line of descent may consist not of very rare speciation events, as has traditionally been supposed, but rather extremely frequent speciation events. We might be faced with the problem of why there are so few species rather that of why there are so many (FELSENSTEIN 1981). Most events of the sort discussed earlier will produce two reproductively isolated populations that are not otherwise diversified. This, in turn, is expected to lead either to differentiation of the two populations motivated by intense competition (character displacement) or to the elimination of one of the two now isolated lines of descent by such competition (competitive exclusion). If, as may be expected, the latter outcome is more common than the former, then the history of a line of descent should be thought of as consisting of very frequent cycles of speciation followed by competitive exclusion.

Second, it has been frequently noted that transposon insertion may be an important source of mutation in various populations (BINGHAM, KIDWELL and RUBIN 1982; RUBIN, KIDWELL and BINGHAM 1982; KARESS and RUBIN 1982; ZACHAR and BINGHAM 1982; BENDER *et al.* 1983; KIDD, LOCKETT and YOUNG 1983). If, as expected, rates of mutation are substantially elevated as a result of invasion of the very same transposon families that will ultimately lead to the division of the invaded population into two distinct lines of descent, then transposons may be responsible both for the creation of new, reproductively isolated lines of descent and for the creation of the variability that occasionally allows both of those isolated lines to survive as a result of character displacement. If this, in fact, occurs then transposons can be accurately said to be central actors in the generation of potential species.

Third, we note that an additional potentially important feature of evolution induced by transposable elements is their ability to lower population fitness. An element that substantially lowers fitness can nonetheless invade a population if it is sufficiently infectious (see preceding data). Accumulation of such deleterious but infectious elements would certainly reduce the overall fitness of the population and might render a population more vulnerable to extinction.

In summary, transposable elements may be the central biological agent controlling much of the evolutionary process in sexually reproducing organisms including the generation of the mutational diversity and reproductively isolated lines of descent involved in speciation. It is conceivable that such elements may be involved in the extinction processes as well. Compelling evidence for any central role of transposable elements in evolution may come from direct study of transposons in contemporary populations. Our view of the processes of speciation predicts that closely related species should carry transposon familes that distinguish them and that such families should be causally related to the partial or full sterility in hybrids between the two species. Although conventional genetic divergence subsequent to initial reproductive isolation may, of course, complicate an experimental analysis, this prediction may be directly testable among members of the *melanogaster* subgroup of Drosophila species,

which form viable but sterile hybrids and in which germline genetic transformation is now possible.

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APPENDIX I

Consider the first equation of system (1) and assume that the initial frequencies of the P element are low in both sexes. Since $P'_m = P'_f$, we can also assume that they are equal to each other:

$$P_f = P_m = \epsilon.$$

Disregarding the second order terms in ϵ we obtain approximately

$$P_f' \approx \frac{\epsilon + \epsilon(1-s)(1+\beta)}{2(\epsilon + \epsilon(1-s) + 1 - 2\epsilon)} = \frac{\epsilon(1 + (1-s)(1+\beta))}{2(1-\epsilon s)}$$

We then obtain the invasibility condition from the inequality

$$\Delta P_f = P_f' - P_f > 0$$

or

$$\epsilon(1 + (1-s)(1+\beta)) > 2\epsilon(1-\epsilon s) \approx 2\epsilon$$

$$1 + (1-s)(1+\beta) > 2$$

$$(1-s)(1+\beta) > 1$$

$$\beta > \frac{s}{1-s}$$

APPENDIX II

Consider the system of two recurrent equations (2) and assume that the initial frequencies are low in both sexes. Since $I_f \neq I_m$, we cannot assume that these two frequencies are equal:

$$I_f = \epsilon_f, \qquad I_m = \epsilon_m.$$

Disregarding second-order terms in ϵ_f and ϵ_m, we can obtain the linearized system around the point, $I_f = 0$, $I_m = 0$:

$$I_f' = \frac{\epsilon_m(1-s)(1+\beta) + \epsilon_f}{2}$$

$$I_m' = \frac{\epsilon_m(1+\beta) + \epsilon_f}{2}$$

The condition of invasibility is simply the condition of instability of the zero equilibrium. In other words, we require that at least one of the roots of the characteristic equation

$$\det \begin{pmatrix} \frac{1}{2} - \lambda & \frac{(1-s)(1+\beta)}{2} \\ \frac{1}{2} & \frac{1+\beta}{2} - \lambda \end{pmatrix} = 0$$

is greater than 1. We have

$$\left(\frac{1}{2} - \lambda\right)\left(\frac{1+\beta}{2} - \lambda\right) - \frac{(1-s)(1+\beta)}{4} = 0$$

$$\lambda_{1,2} = \frac{\beta + 2 \pm \sqrt{\beta^2 + 4(1-s)\beta + 4(1-s)}}{4}$$

The larger root,

$$\lambda = \frac{\beta + 2 + \sqrt{\beta^2 + 4(1-s)\beta + 4(1-s)}}{4}$$

should be greater than 1. We have, therefore,

$$\beta + 2 + \sqrt{\beta^2 + 4(1-s)\beta + 4(1-s)} > 4$$

$$\beta^2 + 4(1-s)\beta + 4(1-s) > 4 - 4\beta + \beta^2$$

$$\beta > \frac{s}{2-s}$$