THE THEORY OF SPECIATION *VIA* THE FOUNDER PRINCIPLE

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ABSTRACT

The founder principle has been used to explain many instances of rapid speciation. Advances from theoretical population genetics are incorporated into MAYR's original founder-effect genetic-revolution model to yield a newer model called the genetic transilience. The basic theoretical edifice lies upon the fact that founder event can sometimes lead to an accumulation of inbreeding and an induction of gametic disequilibrium. This, in turn, causes alleles to be selected more for their homozygous fitness effects and for their effects on a more stable genetic background. Selection occurring in multi-locus systems controlling integrated developmental, physiological, behavioral, etc., traits is particularly sensitive to these founder effects. If sufficient genetic variability exists in the founder population, such multilocus genetic systems can respond to drift and the altered selective forces by undergoing a rapid shift to a new adaptive peak known as the genetic transilience. A genetic transilience is, therefore, most likely to occur when the founder event causes a rapid accumulation of inbreeding *without* a severe reduction in genetic variability. The implications of this model are then examined for three aspects of the founder-effect genetic-transilience model: the attributes of the ancestral population, the nature of the sampling process used to generate the founders and the attributes of the founder population. The model is used to explain several features of the evolution of the Hawaiian Drosophila, and experimental designs are outlined to test the major predictions of the theory. Hence, this theory of speciation can be tested in the laboratory, using systems and techniques that already exist—a rare attribute of most models of speciation.

EVOLUTION is often divided into two basic processes: anagenesis, or change within a phyletic line, and cladogenesis, or the multiplication of phyletic lines. The bulk of population genetics, both theoretical and experimental, has been directed toward anagenesis, mainly because the very important problem of cladogenesis has been less amenable to the standard mathematical and empirical techniques of population genetics. Thus, MAYR (1978) recently stated that it is rather startling "how little population genetics has contributed to our understanding of speciation." This state of affairs is unfortunate because population genetics can be applied to the problem of speciation in such a manner as to greatly augment the explanatory and, more importantly, the predictive powers of speciation theories. I will illustrate this by focusing upon one such mechanism of speciation—the founder principle and genetic revolutions.

ERNST MAYR (1954) was the first to articulate a mechanism of rapid speciation based explicitly upon the founder effect. He envisioned a situation in which a small number of individuals (founders) are geographically isolated from their ancestral populations. The theoretical work of WRIGHT (1942) predicted that such founder populations would undergo considerable genetic differentiation from the ancestral population both in terms of a reduced level of genetic variability and random changes in allele frequencies due to genetic drift. WRIGHT (1931, 1932) also hypothesized that drift and selection would interact to allow small populations occasionally to undergo radical transitions to new adaptive complexes that selection alone (*i.e.*, selection in large populations) would prevent. Moreover, MAYR emphasized the role of another factor that WRIGHT noted was a consequence of small size—the accumulation of inbreeding in the founder population. To MAYR, this accumulation of inbreeding was perhaps the most important consequence of the founder effect since it greatly increased the chance of a founder effect leading to a fundamental shift in the adaptive complex. In the words of MAYR (1954):

> As a consequence of their increased frequency in the founder population, homozygotes will be much more exposed to selection and those genes will be favored which are specially viable in homozygous condition. . . . We come thus to the important conclusion that *the mere change of the genetic environment . . . may change the selective value of a gene considerably*. This change . . . may affect all loci at once. Indeed, it may have the character of a veritable "genetic revolution."

The explanatory power of this hypothesis is undoubted (MAYR 1970), particularly in the brilliant manner in which it has been modified and applied to the Hawaiian Drosophila (CARSON and KANESHIRO 1976; CARSON 1968; CARSON 1975; CARSON 1978). However, there still remain some major weaknesses of the founder principle as a mechanism of speciation. For example, as mentioned above, the founder principle has been used by CARSON (1975) to explain the rapid speciation of Hawaiian Drosophila. Although CARSON's theories have recently been attacked by RINGO (1977), I believe this attack has been unsuccessful, and the explanatory power of the founder effect as a speciation mechanism in Hawaiian Drosophila is far greater than the alternatives (TEMPLETON 1979a). Nevertheless, other species of Drosophila have apparently undergone many founder events without speciation; *e.g.*, *D. melanogaster* has most likely undergone hundreds, if not thosuands, of founder events during its recent history, but it has become a cosmopolitan species rather than a speciose sugroup like the Hawaiian picture wings. Why the difference? Also, laboratory studies that simulate the founder effect in Drosophila sometimes show the evolution of isolating barriers (POWELL 1978; TEMPLETON 1979b) and sometimes do not (AYALA, personal communication); once again, why the difference? Thus, although MAYR's theory can explain why the founder event can lead to speciation and explain some existing patterns of species, it fails to predict which founder events lead to speciation and which lead to trivial changes from a cladistic point of view. In this paper, I will address myself to the problem of making the founder principle more predictive.

DEFINITIONS AND SCOPE OF PROBLEM

As used by MAYR, the term "genetic revolution" implied extensive changes throughout the entire genome. CARSON (1975) modified this position by dividing the genome into an "open" and "closed" system, with "genetic revolutions" affecting only the closed system. The experimental work reported in TEMPLETON and RANKIN (1978) and TEMPLETON (1979b) indicates that CARSON's view is correct; in particular, that isozyme loci are part of the open system, whereas loci regulating fundamental developmental, physiological and life history processes are part of the closed system. The exact number of loci that underlie a "genetic revolution" is not known at this time, but it may be relatively small (*i.e.*, on the order of magnitude of 10 rather than 100's or 1000's). Hence, because of the established connotation of the term "genetic revolution," it is better to use a more neutral term at this time, such as "genetic transilience" (TEMPLETON 1979b). Although genetic transilience may involve only a few loci, it is definitely a multilocus phenomenon and involves, in part, non alleles selectively responding to each other. Based on the experimental results of TEMPLETON (1979b), genetic transilience is defined as a rapid shift in a multilocus complex influencing fitness in response to a sudden perturbation in genetic environment. As shown in TEMPLETON (1979b), genetic transilience can lead to both pre- and post-mating isolation from the ancestral population, as well as differences in morphology, life history, development, physiology, etc. Thus, the effects of genetic transilience may serve as the basis of speciation. Note that in this model, the shift in genetic environment (which can be caused by the founder effect) directly leads to the altered selective conditions that, in turn, cause the transilience and speciation. This serves to distinguish speciation *via* the founder-effect genetic-transilience model from gradual, adaptive, allopatric speciation: in the former case, the act of isolation itself directly leads to an altered genetic environment that *causes* the speciation event to occur; in the latter, the act of isolation merely *allows* speciation to occur as a by-product of ordinary microevolutionary processes.

Also, the range of biological situations for which the genetic transilience model is appropriate exceeds the strict allopatric situation to which the founder principle is usually applied. For example, some insect species have undergone host shifts that lead to reproductive isolation (BUSH 1975). Consequently, many host races, although sympatric with their ancestors, have essentially arisen from a founder effect in genetic isolation from the ancestral race. The principle of genetic transilience could apply to such host shifts. Also, if the external environment changes radically, a population may undergo an extreme bottleneck, at least in local areas. Once again, genetic transilience could occur in some areas before gene flow is reestablished with the remainder of the population. In both of the above cases, some aspect of the external environment is also changing, which could invoke selective forces on its own in addition to the altered genetic environment. A final possible example is the evolution of self-mating populations from outcrossing populations in plants. Often, such populations arise due to mutants in the outcrosser; hence, the self-mating population arises perhaps from only one founder. Once again, although this is a sympatric situation, the imposition of self-mating obviously alters the genetic environment in such a way as to lead possibly to genetic transilience.

BASIC THEORETICAL FRAMEWORK

Before developing specific predictions, I will give an overview of the theoretical framework and present the types of population genetic measures and parameters to be used.

The founder effect, as formulated by WRIGHT and used by MAYR, leads to two major changes in the genetic structure of the founder population: (a) random changes in allele frequency, including loss and fixation, which lead to genetic divergence from the ancestral population and a reduction in genetic variability; and (b) an accumulation of inbreeding and associated increases in levels of

homozygosity. Population geneticists generally measure the effect of (a) by the "variance effective size." If p is an allele frequency in the ancestral population, then sampling will cause the allele frequency to become a random variable with some nonzero variance. The variance effective size (N_{ev}) is chosen such that this variance can be expressed as:

$$\frac{p(1-p)}{2N_{ev}} . \tag{1}$$

Thus, the variance effective size measures how much change in allele frequencies (including loss of fixation) can be expected due to the founder effect. A second measure of (a) is simply the genetic distance between ancestor and founder, which, once again, reflects the degree to which allele frequencies have been changed. A final measure of effect (a) deals more directly with the reduction in genetic variability caused by the founder effect. If a random sample of $2N$ neutral alleles are drawn from the ancestral population, EWENS (1972) has shown that the expected number of alleles in the sample is:

$$E(K) = 1 + \frac{\Theta}{\Theta+1} + \ldots + \frac{\Theta}{\Theta+2N-1} , \tag{2}$$

where K is the number of alleles in the founder population and $\Theta = 4N_{ev}(A)\mu$, where $N_{ev}(A)$ is the variance effective size of the *ancestral* population and μ is the mutation rate. Although true only for neutral alleles, (2) provides a useful gauge for measuring the reduction in genetic variability caused by a founder event of size N.

The second effect of the founder event is the accumulation of inbreeding. This is most conveniently measured by the "inbreeding effective size." If there is a random mating monoecious population of size N, then the inbreeding coefficient at generation t (f_t) obeys the following recursion formula:

$$f_t = \frac{1}{2N} + \left(1 - \frac{1}{2N}\right) f_{t-1} . \tag{3}$$

When deviations from this ideal, randomly mating monoecious population exist, an inbreeding effective size, N_{ef}, is chosen so as to make a recursion relation of the form (3), *i.e.*,

$$f_t = \frac{1}{2N_{ef}} + \left(1 - \frac{1}{2N_{ef}}\right) f_{t-1} . \tag{4}$$

These parameters and measures of population size relate to the genetic transilience in two distinct fashions. First, the transilience requires that genes selectively respond to the altered genetic environment induced by the founder event. There are at least two consequences of the inbreeding associated with the founder effect that directly alter selective forces. First, alleles will be selected more for their

homozygous effects than they previously were. This is shown by noting that the average effect of an allele, i, on fitness is given by (CROW and KIMURA 1970):

$$[(p_i+q_if)(W_{ii}-\bar{W}) + \sum_{j\neq i}^{n} p_j(1-f)(W_{ij}-\bar{W})]/(1+f) \quad , \qquad (5)$$

where $p_i = 1 - q_i$ = the frequency of allele i, W_{ij} = the fitness of genotype ij, $\bar{W}$ is the average fitness, n = the number of alleles and f = the inbreeding coefficient. Whether or not selection tends to increase, decrease or not affect the frequency of allele i depends upon whether equation (5) is positive, negative or zero (from FISHER's 1958 Fundamental Theorem). Hence, if the level of inbreeding is changed, the evolutionary fate of an allele should change. Moreover, the larger the change in f, the greater the change in selective forces operating at the genic level.

The second important effect of inbreeding and homozygosity is that a given allele appears against a narrower spectrum of genetic backgrounds. Thus, due to the founder effect, an allele is selected for its effects on a more limited range of genetic backgrounds. This means that epistatic terms that could not be effectively selected for in the ancestral population can now respond to selection and play a major role in restructuring the fitness properties of the genome. For example, fixation at one critical locus could have cascading fitness effects in a strongly epistatic genetic system. This apparently occurred in a transilience observed in the laboratory in *Drosophila mercatorum* (TEMPLETON and RANKIN 1978; TEMPLETON 1979b). Fixation of an allele at the *X*-linked abnormal abdomen locus (*aa*) that had a frequency of 0.20 in the ancestral population was the focus of a radical shift in the fitness properties of the resulting founder population due to the strong epistatic fitness interactions *aa* has with other loci scattered throughout the *D. mercatorum* genome. An example in a natural speciation event is, perhaps, provided by the Hawaiian species pair *Drosophila silvestris* and *D. heteroneura*. Circumstantial evidence strongly implies that a founder event occurred in their recent history (CARSON and KANESHIRO 1976). They are virtually identical with respect to their chromosomes and isozymes (SENE and CARSON 1977), but are extremely different in head shape, a trait apparently under strong sexual selection (TEMPLETON 1977). The head-shape differences are determined by an *X*-linked locus that regulates the effects on head shape of about ten autosomal loci (TEMPLETON 1977). Thus, chance fixation of certain alleles at the *X*-linked locus would have cascading effects throughout the genome in this system subject to strong sexual selection. This pattern of an epistatic polygenic system with a few major genes is perhaps crtitical for genetic transilience. As I will argue later in this paper, founder effects do not often greatly alter the overall levels of genetic variability and heterozygosity (*e.g.*, see NEI, MARUYAMA and CHAKROBORTY 1975). Hence, if a polygenic trait is controlled by a very large number of additive loci, each with small effects, often very little change or reduction in variability in the polygenic trait would be expected after the founder event. But, if there are a few major genes, the stochastic effects of a founder event cannot be ignored. However, strong epistasis with the other loci contrib-

uting to the trait is also critical for effecting a transilience, for in this way, the impact of chance events operating upon the major loci can be greatly amplified. WRIGHT (1931, 1932) also argued that genetic systems characterized by epistasis are most sensitive to interactions with drift and inbreeding. For these reasons, genetic transilience will almost always occur in a multilocus complex with some major genes controlling some integrated developmental, physiological, behavioral, etc., trait that strongly influences fitness, a view supported by my experimental work on *Drosophila mercatorum* (TEMPLETON 1979b).

For genetic transilience to occur, these changes in the genetic selection environment must be so drastic and rapid that a "selective bottleneck" (TEMPLETON, CARSON and SING 1976; ANNEST and TEMPLETON 1978; TEMPLETON 1979b,c) is engendered. With a founder effect, the degree of change in the genetic environment can be gauged by the degree of change in the inbreeding effective number between founder and ancestor. Consequently, the first fundamental assumption of this theory is that the change in inbreeding effective sizes between ancestor and founder, $\Delta N_{ef} = N_{ef}(A) - N_{ef}(F)$, be large, so as to induce a selective bottleneck.

However, creating a selective bottleneck does not insure genetic transilience; the founder population must also be able to survive and respond genetically to this drastically altered genetic environment. The chance of the population surviving and/or responding to the selective bottleneck depends upon the level of genetic variability present in the founder population, since without genetic variability there can be no response to selection. As can be seen from equations (1) and (2), the amount of genetic variability present initially in the founder population will be influenced by the variance effective size of the ancestral population $[N_{ev}(A)]$, the sample size of the founders (N), and the variance effective size of the founders $[N_{ev}(F)]$. The fundamental theoretical framework of this paper may now be stated:

> *Genetic transilience will most likely occur when: (1)* ΔN_{ef} *is large so as to create an intense selective bottleneck, and (2)* $N_{ev}(A)$, $N_{ev}(F)$ *and* N *are as large as possible subject to the constraint* ΔN_{ef} *large so that the founder population may have sufficient genetic variability to respond to the selective bottleneck.*

The implications of this fundamental conclusion will now be examined for three basic aspects of the founder event: (1) the population structure of the ancestral population, (2) the nature of the sampling procedure used to generate the founders, and (3) the population structure and other attributes of the founder population.

Ancestral population structure

The ancestral population structure exerts an important influence both on the potential for the founder effect to yield a large change in genetic environment and on the level of genetic variability carried by the individuals drawn to form the founder population. In order to examine the effects of ancestral population structure, I will assume throughout this section that the founder population is always drawn as a random sample from a single local geographical population

or deme of the ancestral population. This assumption will be dropped in later sections.

To illustrate the role of ancestral population structure, three extreme cases will be discussed (see Figure 1): (1) a large, panmictic population, (2) WRIGHT's Island model in which the population is subdivided into small demes with little gene flow between demes, and (3) the "Yanomama," a type of population structure found in many human populations and typified by the Yanomama Indians of South America. Obviously, most real populations will lie between these extremes; *e.g.*, as deme size increases in the WRIGHT Island model, each deme approaches the state described under (1). Nevertheless, these three extreme types of population structure will illustrate the importance of the ancestral population in influencing the chances of a genetic transilience.

The large panmictic ancestral population is the type of structure usually assumed (often implicitly) in discussions of the founder principle. As I will now argue, this structure has many features that make it an optimal type for yielding a genetic transilience in response to a founder effect. First, because $N_{ef}(A)$ is large, polymorphic alleles are frequently selected for their heterozygous effects

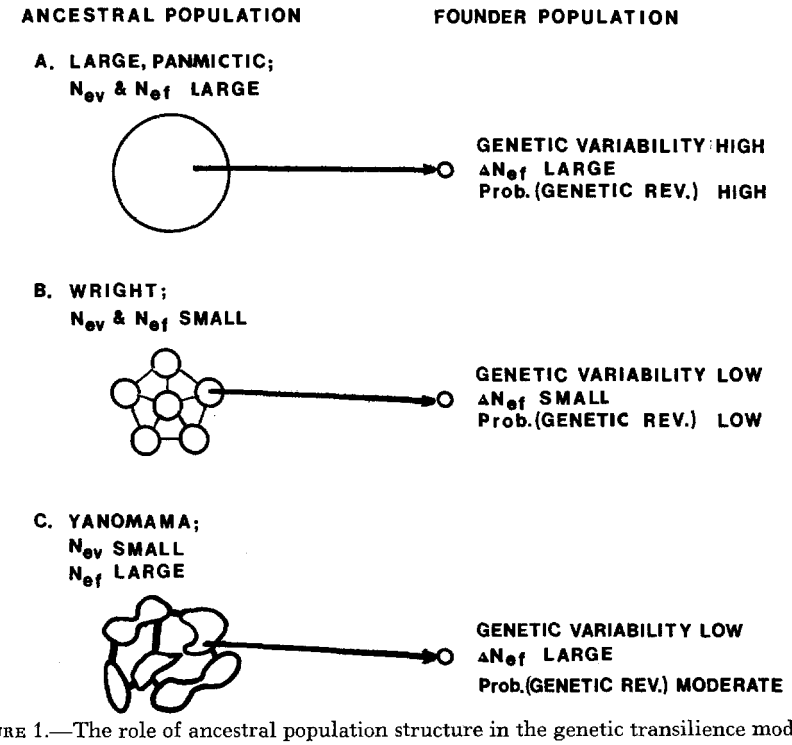

FIGURE 1.—The role of ancestral population structure in the genetic transilience model. Three types of ancestral populations are considered: (A) a large panmictic population with no subdivision and random mating; (B) a WRIGHT Island model population consisting of many small demes with little gene flow between demes, and; (C) a "Yanomama" population in which demes split off from one another along lines of kinship, but with much gene flow between demes. The relative probabilities that a founder event will induce a genetic transilience (genetic revolution) are given.

against an ever-changing genetic background. Hence, alleles are selected primarily for their additive effects; *i.e.*, for being good combiners (CROW 1957). Thus, the potential for a founder event effecting a radical change in the genetic environment is large; that is, the potential exists of ΔN_{ef} being large and of a selective bottleneck being induced. Moreover, the chance that the founder population is able to respond selectively to this bottleneck is enhanced by this type of ancestral structure. Because $N_{ev}(A)$ is large, the potential for the ancestral population bearing large amounts of genetic variability is enhanced (equation 2). More importantly, because of panmixia, this variability is evenly distributed among individuals. Thus, a substantial proportion of the population's genetic variability is carried in the form of individual heterozygosity. This is critical when discussing founder effects because the founder population initially consists of only a few individuals; hence, individual heterozygosity is the primary source of genetic variability in the founders. The selective bottleneck occurs so rapidly (if it occurs at all) that mutation is not an important source of variability; thus, the founder population's capability to respond to the bottleneck is limited by the amount of genetic variability carried by the founding individuals. In other words, when examining the genetic consequences of the founder effect, average observed heterozygosity is a more important parameter than level of polymorphism (TEMPLETON 1979b,c). This, in turn, is determined primarily by the ancestral population structure. In the case of panmixia, levels of individual heterozygosity are high for a given degree of population variability. Therefore, the primary effect of sampling from such an ancestral population is that rare alleles are lost, leading to a reduction of allele number, but overall genetic variability as measured by percent polymorphic loci or individual heterozygosity may remain quite high and be only minimally decreased by a founder event (NEI, MARUYAMA and CHAKROBORTY 1975). Hence, founder events do not necessarily lead to greatly reduced levels of genetic variability.

In summary, the large panmictic ancestral population represents an optimal structure for yielding genetic transiliences because there is a large potential for inducing a selective bottleneck and a large capacity for responding to the bottleneck by utilizing the genetic variability carried by individual founders.

Figure 1B portrays the opposite extreme from the panmictic situation—the WRIGHT Island Structure in which both $N_{ev}(A)$ and $N_{ef}(A)$ of the ancestral population are small. Individuals from such small isolated demes have already accumulated much inbreeding and are characterized by having high levels of homozygosity and restricted ranges of genetic backgrounds within demes. Hence, a founder effect caused by sampling a few individuals from a local deme will change the inbreeding effective size very little and barely alter the genetic environment in which selection takes place. Hence, the potential for creating a selective bottleneck is very low.

This low potential can be illustrated by considering a particular class of alleles that may be involved in speciation *via* the founder principle, as postulated by NEI (1975) and elaborated by WILLS (1977). Suppose there is an allele *a* at locus 1 such that homozygous *aa* males are unable to produce viable or fertile

offspring when mated with females who are homozygous *bb* at locus 2. Assuming no linkage and an infinitely large, panmictic population, WILLS (1977) showed that many such alleles could be present in such a population, with some being at very high frequencies, including fixation for one (but not both *a* and *b* together). Hence, a founder event in such a population would sometimes fix a combination of alleles that could yield post-mating isolation with other populations, including the ancestral one. However, in an ancestral population with the structure portrayed by Figure 1B, the high level of homozygosity and inbreeding would greatly enhance the effectiveness of selection in eliminating such alleles. Thus, such alleles would be absent or extremely rare in the ancestral population, and the chance of a founder effect leading to reproductive isolation by this mechanism would be negligible. Hence, the ancestral population structure is a critical determinant of the effectiveness of the founder principle as a speciation mechanism.

Besides reducing the potential for inducing a selective bottleneck, the Island model reduces the amount of genetic variability carried in individuals. Hence, the capacity to respond to any altered selective conditions is restricted. The restricted genetic variability occurs for two reasons. First, with this type of population structure, much of the population's genetic variability is distributed between demes and is not found in individuals within a deme. Second, inbreeding within a deme further reduces the effective number of genomes sampled within a deme to create the founders. Thus, equation (2), EWENS' sampling formula, is valid only for a random sample of genomes. However, because founder effects involve samples of individuals, EWENS' formula is valid only for a panmictic situation. Suppose the inbreeding coefficient within a deme is f and the founders represent a random sample from this deme. Then, the appropriate expected number of neutral alleles is:

$$E(K|f) = \Theta\int_0^1 x^{-1}(1-x)^{\Theta-1}\{1-[(1-x)^2+fx(1-x)]^N\}dx$$
$$= E(K|f=0) - \Theta\sum_{r=0}^{N-1}\frac{f^{N-r}}{N-r}\cdot\frac{\Gamma(N+1)\Gamma(N+r+\Theta)}{\Gamma(r+1)\Gamma(2N+\Theta)}, \qquad (6)$$

where $E(K|f=0)$ is equation (2), N the number of individuals sampled, $\Theta = 4N_{ev}(A)\mu$ and Γ is the gamma function. As is readily seen from equation (6), the expected number of neutral alleles in the founders decreases as $N_{ev}(A)$ decreases and as f increases. Thus, the WRIGHT model of isolated demes with small N_{ef} and small N_{ev} has little chance of leading to a genetic transilience because its potential for altering the genetic environment is very small and its capacity for genetically responding to any change is likewise small.

The final population structure considered here (Figure 1C) was inspired by studies on the Yanomama Indians of South America (NEEL 1978a,b). This type of population structure is very dynamic. There is a great deal of gene flow between villages, but, when villages split, they divide principally along lines of kinship. This causes large genetic distances to arise between villages despite the large amount of gene flow. Consequently, there is considerable genetic differ-

entiation between villages, but a small negative "f" (actually the F_{IS} of WRIGHT 1965) within villages. Such a population structure is not limited to man, since a similar structure has been recently described for the Rhesus monkey (CHEVERAD, BUETTNER-JANUSH and SADE 1978) and the Macaque (CHEPKO-SADE and OLIVIER 1979). The critical feature of this population structure is that the variance effective size tends to be smaller than the inbreeding effective size. Thus, a considerable portion of the Yanomama genetic variability is distributed between villages, but the loci that are polymorphic in a village tend to have high heterozygosities (NEEL 1978a). However, the overall levels of homozygosity are high (NEEL 1978a,b). Thus, average heterozygosity for isozyme loci in these and similar Amerindian populations is 0.049, as compared to 0.078 for civilized "Caucasians." Therefore, selection for homozygous effects of an allele are undoubtedly more important with this type of population structure as compared to a large panmictic population, but less important as compared to the WRIGHT Island model. However, because of the large intervillage differences and the large amount of gene flow between them, alleles are selected for their properties on a wide range of genetic backgrounds that constantly shift each generation. Moreover, individuals sampled from within a village would have a high probability of being rather closely related due to the lineal nature of village fission. Consequently, a small founder population established from sampling a village would accumulate inbreeding at a very fast rate. Thus, even though the ancestral $N_{ef}(A)$ may not be as large in this case as compared to an otherwise comparable panmictic population, a founder population of given size will result in a smaller $N_{ef}(F)$ from a Yanomama population than from a panmictic population. Hence, a founder event from a Yanomama type of population still has the potential of inducing a large ΔN_{ef}. Moreover, this inbreeding would greatly alter the genetic environment since the genetic environment of the polymorphic alleles in the founder population would have been characterized by high heterozygosities in the ancestral population from which they were drawn and would have been selected against a broad spectrum of constantly changing genetic backgrounds. Both of these aspects of the genetic environment could be drastically altered by a founder effect. However, the overall reduced levels of genetic variability and individual heterozygosity mean that the founder population will have considerably less genetic variability than a founder derived from a comparable large, panmictic population. In addition, the very kinship between villagers that augments ΔN_{ef} would, at the same time, reduce the genetic variability in the founder even more. Hence, the capacity of the founder population to respond to the selective bottleneck is reduced in this case. Thus, this type of ancestral population structure would yield probabilities of leading to a genetic transilience that are lower than the panmictic situation, but considerably higher than the WRIGHT Island model.

Sampling the ancestral population

In the previous section, I assumed random sampling from a local area in the ancestral population; that is, a sampling scheme that accurately reflects in the individual founders the population structure of the ancestral demes. However,

other types of sampling schemes are possible and may be more realistic under certain types of biological situations.

One type of sampling scheme would be to make a random sample of the entire population; that is, all founders are independently drawn from the entire geographical range of the species. This does not seem to be a very realistic scheme in many situations, but could occur if the species were of a sufficiently restricted geographical range and if an environmental agent dispersed over this entire range was responsible for the sampling; *e.g.*, a hurricane or strong wind blowing flies endemic to one island all in the same direction until they reached a second island. More commonly, it might be expected to obtain intermediate situations in which individuals from two or more demes constitute the founders. Viewing the problems as a continuum, it is of interest to see the implications of random sampling over all demes as one extreme to contrast with random sampling within a deme at the other extreme. Once again, the implications of this type of sampling interact with the type of ancestral population structure. If the ancestral population were large and panmictic, a random sample over the entire range would be genetically equivalent to a random sample from a local area; hence, the predictions previously made would still hold. If the ancestral population structure fit the WRIGHT Island model, sampling over all demes would increase the genetic variability in the founders since it would tap the sizable amount of between-deme genetic variability found in the ancestors. However, a genetic transilience would be, at most, only slightly more likely than in the case considered previously since inbreeding in the founders would reestablish the ancestral genetic environment. One might have an adaptive shift, but it would be more in the context of the shifting balance theory, and the resulting founder population would really represent just one more isolated deme of the ancestors. Once again, the founder population might speciate, but, if it do so, it would be more likely as an indirect by-product of adaptive evolution operating in the founders in isolation of the ancestral population, and not due to a genetic transilience. However, if the founders from different demes have gene complexes that are adaptively incompatible with one another, and if extensive hybridization occurs in the founder population, a type of genetic transilience may occur. For the present, this case will be ignored, but will be developed more extensively in another paper.

Sampling over all demes in a Yanomama-like population would likewise create large levels of genetic variability in the founders since it would tap into that variability that is described by the F_{ST} term in the ancestors. In fact, such a "pooling" was done by NEEL (1978a,b) in the computer for 12 Indian tribes, and the resulting "population" had very enhanced levels of genetic variability, as measured by individual heterozygosity that simulated "civilized" man very well. However, unlike the WRIGHT structure, an isolated founder population of Yanomama-like individuals would not simply recreate their ancestral situation. The founder effect would greatly increase the stability of the genetic background in the descendants of the founders; a situation very different from the ancestral condition. Consequently, sampling from several demes in a Yanomama-like population would create better conditions for a genetic transilience than sampling from a single deme; that is, the potential for ΔN_{ef} being large still

exists, but now the founders have sufficient genetic variability to selectively respond to the genetic consequences of a large ΔN_{ef}.

A third type of sampling occurs when genotypic correlations exist between the founders. The Yanomama provide a possible example of this. They have been expanding their territory within the last century, and, as new colonies are sent out, they consist not of randomly drawn individuals either from within or between villages, but, rather, individuals who are generally related to one another. If, by chance, such a colony became completely isolated from the rest of the Yanomama population, it would create a founder population of related individuals with no option but to inbreed among themselves. Such a sampling scheme is probably common in many mammal and other animal populations that have social structuring (*e.g.*, CHEVERAD, BUETTNER-JANUSH and SADE 1978; CHEPKO-SADE and OLIVIER 1979). Another method of generating correlated sampling is suggested by the work of BUSH (1974). Suppose the founder population is isolated by a host shift, and the host shift, in turn, is caused by a "sensory mutation" (BUSH 1974, 1975). Suppose this mutant is dominant and occurred in the germ line of one individual. Then, the founder population during the next generation that makes the host shift will all be sibs of one another. Moreover, the genotypic correlations for loci closely linked to the sensory mutation will be even larger than that expected for randomly drawn genes between sibs. Consequently, whenever the act of founding itself is caused or influenced by genes, genotypically correlated sampling may be expected. As a third example, in many plants, several seeds are contained within a single fruit or berry, which are then eaten by animals and dispersed. Hence, when the animal defecates, the seeds present in the feces are quite likely to be from the same original fruit or berry. Even when not, they might well be genetically correlated since animals will often eat several berries or fruits from the same parent plant or neighboring (and most likely related) plants.

With such correlated sampling, let ρ be the average correlation between the genotypes of two individuals drawn from the founder population. Then, using the results and techniques given in ROTHMAN, SING and TEMPLETON (1974), the $N_{ev}(F)$ of the founders will be

$$\frac{1}{N_{ev}(F)} = \frac{1+F_{IT}}{N} + \frac{2(N-1)}{N}\bar{\psi}$$

or

$$N_{ev}(F) \simeq \frac{N}{1+F_{IT}+2N\bar{\psi}},$$

where F_{IT} is WRIGHT's measure of deviation from Hardy-Weinberg proportions at the total population level, and $\bar{\psi}$, the average coefficient of kinship between randomly drawn alleles in the founders, $= 0.5(1+F_{IT})\rho(1-\psi) + \psi$, where ψ is the average coefficient of kinship within a deme of the ancestral population. Note that F_{IT} and ψ are zero for the large, panmictic ancestral structure, but both are greater than zero for the WRIGHT and Yanomama structures. Hence, the impact of correlated sampling is always to reduce the variance effective size and

level of genetic variability of the founders, but these effects are most pronounced for WRIGHT and Yanomama type ancestral structures. This aspect of correlated sampling, by itself, will tend to reduce the chances for a successful genetic transilience.

However, correlated sampling also has an impact on $N_{ef}(F)$. For example, assume the founders randomly mate. Then the inbreeding coefficient of the first generation of the founders will be

$$f_1 = \frac{1}{2N} + \left(1 - \frac{1}{2N}\right)(f + (1-f)\bar{\psi}) \ ,$$

where f is the inbreeding coefficient of the founders ($f = 0$ for the large panmictic ancestors, greater than zero for the WRIGHT's Island model and close to zero for the Yanomama type structure). Thus, the presence of $\bar{\psi} > 0$ causes inbreeding to accumulate at a faster rate, thus making it more likely for a drastic and rapid change in genetic environment to occur. By itself, this attribute of correlated sampling should increase the chances of a genetic transilience.

Gauging the impact of this type of sampling scheme on the chances for a genetic transilience is, therefore, difficult since, on one hand, it increases the potential for a large ΔN_{ef}, while, on the other hand, it reduces the likelihood of the founder population being able to respond selectively to that change by reducing the level of genetic viability. Under the WRIGHT Island model ancestral structure, the ancestors themselves are so inbred that even correlated sampling will have little impact on ΔN_{ef}; hence, the reduction in genetic variability will probably be the dominant effect and make it even more unlikely for a genetic transilience to occur. However, for a large, panmictic or Yanomama-like ancestral structure, correlated sampling could conceivably either increase or decrease the chances of a genetic transilience, depending on the relative importance of these two antagonistic effects. The empirical data of TEMPLETON (1979b,c) suggest that, if levels of individual heterozygosity are sufficiently high, even the genetic variability carried by a single individual is sufficient to allow the founder population to undergo a genetic transilience with high probability. Consequently, with the large, panmictic ancestral structure, the increase in ΔN_{ef} may well dominate over the decrease in founder $N_{ev}(F)$ with a corresponding increase in the likelihood of a genetic transilience over the random sampling case. Table 1 gives a summary of this and the other predictions made in this section.

TABLE 1

The chances for a genetic transilience as a function of the ancestral population structure and the type of sampling scheme used to generate the founders

Ancestral population structure	Random from total population	Random from local deme	Correlated from local deme
Panmictic	High	High	High → Very high
WRIGHT	Low	Low	Very low
Yanomama	High	Moderate	Low → High

The population structure and other attributes of the founder population

The potential for a large ΔN_{ef} and for the founders to carry over large amounts of genetic variability is also determined by the $N_{ef}(F)$ and $N_{ev}(F)$ of the founders themselves immediately after the act of isolation. Once again, optimal conditions for a genetic transilience will occur when $N_{ef}(F)$ of the founders is small, but $N_{ev}(F)$ is as large as possible. Fortunately there are many types of attributes that allow such seemingly opposite effects on effective size to occur simultaneously. To see this, consider the following standard formulations of the effective sizes (CROW and KIMURA 1970) of the colony after the founding event

$$N_{ef} = \frac{N\bar{k}-2}{\bar{k}-1+\frac{V}{\bar{k}}} \tag{7}$$

$$N_{ev}(F) = \frac{N\bar{k}}{1-\alpha+\frac{N(1-\alpha)V}{k(N-1)}}, \tag{8}$$

where $\bar{k}$ = the mean number of offspring per individual, V = the variance in offspring number and α = a measure of deviation from Hardy-Weinberg proportions. One attribute that has drastically different effects on the two types of effective size is $\bar{k}$, the mean number of offspring per individual. The reason for this is that the inbreeding effective size is more closely related to the number of parents, while the variance effective number is more closely related to the number of offspring (CROW and KIMURA 1970). Thus, as $\bar{k}$ goes to infinity, $N_{ef}(F) = N$ (the number of founders), but $N_{ev}(F)$ goes to infinity. On the other hand, if $\bar{k} = 1$ and $V = 0$, $N_{ef}(F)$ becomes infinite and $N_{ev}(F) = N/(1-\alpha)$. Consequently, when $\bar{k}$ is very large, stochastic loss of genetic variability is virtually limited to the initial founder sampling itself and is not compounded by further loss after the founder colony has been formed. The fact that the founder colony will not lose much genetic variability under these conditions has already been demonstrated by NEI, MARUYAMA and CHAKRABORTY (1975). Yet, despite this rapid recovery and modest diminution of genetic variability, the inbreeding effective size remains limited by the initial founder size for many generations to come; that is, inbreeding continues to accumulate as if the founder population were still small. Thus, those organisms most likely to experience a genetic transilience after a founder event are those with a large reproductive capability. This not only decreases the chance of the founder population becoming extinct, but allows the founders to retain high levels of genetic variability, while still accumulating inbreeding at a rapid rate. This also implies that a genetic transilience will most likely occur in those species in which the stage of life history corresponding to maximal liability to a founder event coincides with maximal reproductive value. Moreover, this condition increases the chance of a colony surviving (WILLIAMSON and CHARLESWORTH 1976). Lastly, these effects are probably even more pronounced when the founder population has overlapping generations.

Suppose the founding individuals are very long-lived and reproduce over much of their lifespan. This not only increases the chance that the founders are individuals with high reproductive values, but also by increasing the average generation time, the variance effective size of the colony is increased (CROW and KIMURA 1972), while the inbreeding effective size per generation is decreased (CHOY & WEIR 1978). The opportunity for inbreeding is particularly increased in a colony undergoing rapid growth in size (GIESEL 1971). Consequently, overlapping generations tend to make it more likely to have a genetic transilience than discrete generations. All of these considerations emphasize the importance of the "flush" phase in CARSON's founder-flush models (CARSON 1975, 1978).

Another important attribute affecting the decay of genetic variability and the accumulation of inbreeding in the colony is $D\sigma^2$, where D is the density of the colony and σ^2 is the variance of dispersion distance. If $D\sigma^2 < 1$, the rate of decay of genetic variability in the colony due to drift is given by $D\sigma^2/(2N)$, where N is the colony size; whereas, if $D\sigma^2 \geq 1$, the decay is given by the usual rate, $1/(2N)$ (MARUYAMA 1972). Hence, if the founders find themselves in an environment in which there is an open niche, k can be large and D small during the initial critical generations, a situation that optimizes the retention of genetic variability. Such a situation might be particularly relevant for founder events assocated with host shifts. However, $D\sigma^2$ can also be made small, if σ^2 is small in the founder population. That is, suppose that the founder population becomes subdivided into geographically isolated neighborhoods or demes, at least during the first few generations. Then MARUYAMA's (1972) model indicates that the overall loss of genetic variability will be less than if it were panmictic; yet, levels of inbreeding would be greatly increased. Moreover, such a population structure in the colony would allow effective interdemic selection to occur upon the various genetic transiliences that would occur in different isolates of the colony. Note that I previously argued that the optimal ancestral population structure for a genetic transilience was panmixia; now I add that a genetic transilience will most likely occur when the founder event is also associated with a shift in population structure from panmixia to an "Island" model situation (at least for the first 50 or so generations of the founder colony).

To summarize the points made so far, a genetic transilience is most likely when the founders have high reproductive values, find themselves in a low density environment with rapid increases in population size possible, have overlapping generations and have a hierarchical population structure, at least temporarily.

As argued above, the very act of colonization may alter the population structure. As I will now discuss, a founder event in itself may also alter the system of mating; and, depending upon the exact nature of this modification, the chance of a genetic transilience may be either enhanced or diminished to the point of impossibility.

To illustrate this, consider an ancestral population that is characterized by either assortative or disassortative mating with respect to some phenotypic traits (of course, both are simultaneously possible for different traits of the individuals involved). Then, the "inbreeding" coefficient (deviation from Hardy-Weinberg

proportions) induced at the loci underlying the trait is given by WRIGHT (1971) and CROW and KIMURA (1970).

$$f = \frac{r}{2n_e(1-r)+r}, \tag{9}$$

where r is the phenotypic correlation between mates and n_e is the effective number of loci that depends upon the number of actual loci, the number of alleles at these loci and the allele frequency distribution. In general, n_e increases as the number of loci and alleles increases and as the allele frequency distribution becomes more even; *i.e.*, all alleles being about equally frequent. If $r < 0$, mating is disassortive, and an excess of heterozygotes over Hardy-Weinberg expectations is expected. If $r > 0$, mating is assortive, and an heterozygote deficiency is expected. As is obvious from equation (9), as n_e becomes large, the impact of assortative or disassortative mating becomes minimal as a cause of deviation from Hardy-Weinberg expectations. Other systems of mating yield very similar effects to that described by equation (9), such as the rare male or similar minority advantages. Although the exact mode of sexual seelction in such cases is not truly disassortative, the genetic consequence of such systems of mating often mimic disassortative mating.

One impact of the founding event is that many alleles, particularly the rarer alleles, tend to be lost. This causes n_e to decrease, and hence the quantitative impact upon genotypic frequencies of a constant system of mating increases after the founder effect. In particular, if mating is assortative, the system of mating interacts with the founder effect to increase deficiencies in heterozygote frequencies; that is, it behaves as if inbreeding were intensified. Thus, when $r > 0$ and the ancestral population was large and outcrossing, the founder effect intensifies the potential for a large ΔN_{ef} and, thus augments the chances of a genetic transilience. In addition, $r > 0$ implies $\alpha > 0$, which increases $N_{ev}(A)$ (equation 8). Moreover, this effect is not limited to the loci underlying the assortative-mating phenotype, but to all loci in linkage disequilibrium with them. This is particularly important because the founder event in itself can generate substantial linkage disequilibrium (SVED 1968; HILL 1974; AVERY and HILL 1977, 1979), even between unlinked loci. Even though this disequilibrium may be ultimately dissipated, that is irrelevant here because the genetic transilience is not an equilibrium phenomenon. If it occurs at all, it will probably occur during the initial few generations immediately after the founder event during which the linkage disequilibrium induced by sampling will be maximal. Thus, even without a reduction in n_e, an assortative mating system will effectively increase the inbreeding at many loci through disequilibrium after a founder effect that would have been unaffected before.

When $r < 0$, mating is disassortative, and the system of mating interacts with the founder effect to decrease the amount of "inbreeding." This effect is not unknown; in fact, many disassortative mating systems have been discovered by experimentors deliberately starting with a small number of founders and then detecting deviations that were not noticeable in the large ancestral popula-

tions (*e.g.*, self-sterility alleles in many plant species; the multiple pheromone loci discovered by AVERHOFF and RICHARDSON 1974, 1976 in *Drosophila melanogaster*). Moreover, the chance of a founder event effecting a large quantitative change on deviations from Hardy-Weinberg is probably much greater for disassortative mating than for assortative mating. The reason is that disassortative mating systems (and, for the purpose of this paper, such "similar" systems of mating as minority advantage) tend to maintain large amounts of genetic variability in the form of numerous alleles, all of which are about equally frequent (and hence, individually "rare"), for example, self-sterility alleles in plants. Consequently, n_e in the ancestors tends to be very large and deviations from Hardy-Weinberg are minimal. However, as NEI, MARUYAMA and CHAKRABORTY (1975) demonstrated, the primary type of loss of genetic variability induced by a founder event is loss of rare alleles, but with this system of mating, almost all alleles are rare. Hence, the loci underlying such a disassortative-mating phenotype are particularly sensitive to a founder effect. For example, suppose there is one self-sterility locus in a plant with 50 alleles. Such figures are not unreasonable, even for populations as small as 500 individuals (WRIGHT 1939). However, if a founder event of two individuals occurred, the maximal number of self-sterility alleles would be four. Hence, the potential for a dramatic quantitative change in the genetic consequences of disassortative mating is large when a founder event occurs because this type of genetic variability is maximally sensitive to loss or fixation by small population size. However, given that at least some genetic variability survived the founder event, which is also very likely with this type of variability since individuals tend to be heterozygous at such loci and mates tend to differ in the alleles they carry, this system of mating will cause a very large heterozygote excess at the loci underlying the disassortive mating phenotype, as well as at all loci in linkage disequilibrium with them during the critical early generations. In fact, precisely this point has already been made by AVERHOFF and RICHARDSON (1976). They discovered a multiple-locus pheromone system leading to disassortative mating in *Drosophila melanogaster*. As expected, such a system is virtually not detectable in outcrossed populations, but, when the flies are forced into a population bottleneck, this system of mating coupled with linkage disequilibrium creates a strong counterforce to inbreeding. Moreover, in a species, such as *Drosophila melanogaster* that has small chromosome number and relatively little recombination, just a few pheromone loci can effectively buffer the entire genome from the inbreeding normally induced by a bottleneck effect, provided, of course, that the bottleneck effect does not persist for too long a period. Consequently, disassortative or rare-male mating systems can greatly reduce the chances of a genetic transilience, particularly when coupled with a species with a low chromosome number, little recombination, and numerous crossover suppressors (or at least crossover suppressors that are readily lost during the founder event itself).

The structural constraints of the genome are also important even in the absence of disassortative or assortative mating. FRANKLIN (1977) has recently calculated the variance in the inbreeding coefficient as a function of chromosome number

and the chromosomal map distances. For example, he calculated that the standard deviation in the inbreeding coefficient for sib matings in *D. melanogaster* would be 0.188 (the mean inbreeding coefficient = f = 0.25 for that case), or for half-sibs it would be 0.167 (f = 0.125). This means that in a founder event involving unrelated individuals of *D. melanogaster* that led to full-sib or half-sib mating in the next generation (*e.g.*, a single, multiply mated, gravid female), about 15% of the offspring from full sibs and 33% of the offspring from half sibs would have actual average inbreeding coefficients of 0.05 or less. In other words, when chromosome number is small, chromosomal map lengths small, and/or crossover suppressors are common and not lost during the founder event, a substantial proportion of the population is effectively not inbred despite what appears to be very high levels of average inbreeding. If selection favors those individuals that have the genetic environment most like their ancestors (*i.e.*, the ones with low *f*), this selection could be very efficient in a population in which the variance of *f* is large and could effectively prevent the population from becoming inbred. As the calculations of FRANKLIN (1977) show, heterotic selection at just four to five scattered loci could easily prevent the accumulation of inbreeding in the early generations of a founder population established by a single, gravid female *D. melanogaster*. Consequently, for a genetic transilience to occur, the chromosome number and chromosomal map lengths must be sufficiently large and crossover suppressors sufficiently rare (or easily lost) so as to insure that the variance in *f* is small. Hence, selection will be forced to respond to a genetic environment characterized by increased homozygosity, rather than selection favoring a substantial minority of the population that simply recreates by the chance processes of meiosis the genetic environment of the ancestors.

Another system of mating that interacts with a founder event is stabilizing sexual selection on a mate-recognition system (CARSON 1978; TEMPLETON 1979a). Such a system, when coupled with a founder event, can greatly increase the *a priori* chances of a genetic transilience occurring and can quickly and effectively stabilize a transilience once it has occurred. Indeed, the integrated morphological-behavioral complex that usually constitutes the mate-recognition system is capable of undergoing a genetic transilience itself that could directly lead to speciation. Details of these arguments will be found in TEMPLETON (1979a), who also argued that if such mate-recognition systems are selected to reinforce the reproductive isolation of a species in the presence of sympatric species, a founder event can alter the external selective environment of the founders' mate-recognition system simply by placing the founders into a new community of sympatric species. PATERSON (1980) has recently objected to this later hypothesis, noting that the existence of a mate-recognition system does not require selection for reinforcement of reproductive isolation and, moreover, that the evidence for reinforcement has some serious flaws (PATERSON 1978). However, another mechanism by which a founder event can alter the sexual selective environment in a way compatible with PATERSON's definitions is suggested by the works of SENE (1977) and PRUZAN *et al.* (1979). They have shown that the mate-recognition system in some Drosophila is only partially genetic and that

the environmental experiences of naive flies can greatly alter their sexual-recognition signals. Suppose a founder event occurs through a single gravid female entering a new territory (as is likely for the Hawaiian Drosophila). The emerging founder population will have no "experienced" models, so that the entire population is naive. Hence, males deviating from the ancestral mate-recognition system can be expected to occur for purely environmental reasons (SENE 1977), and the naive females will be receptive to them (PRUZAN *et al.* 1979). In addition, the genetic consequences of the founder event and inbreeding could also lead to major deviations in the genetic component of the mate recognition system that would be strongly selected against in the ancestral population, but tolerated in the naive founder population. However, once the deviation occurs, a new norm is established that is subject to intense stabilizing sexual selection (PRUZAN *et al.* 1979). Moreover, the variability released by both environmental and genetic factors could easily lead to different subpopulations of the founders becoming imprinted for a different mate-recognition system. This, in turn, could lead to the establishment of more than one reproductively isolated population from a single founder event *via* the population genetic mechanisms given in KALMUS and SMITH (1966). Thus, a mate-recognition system that is partially learned can, under some conditions, make a genetic transilience more likely than a totally genetic mate-recognition system.

TABLE 2

Attributes of the founder population that either increase or decrease the chance for genetic transilience

Attributes that increase the chance	Attributes that decrease the chance
Average number of offspring large	Average number of offspring small
Reproductive value of founders high	Reproductive value of founders low
Open niche allowing population flush	Population flush not possible
Initial density low	Initial density high
Initial subdivided population structure	Initial panmictic population structure
Overlapping generations	Discrete generations
Assortative mating	Disassortative mating
Sexual selection on the mate recognition system	Rare male or similar sexual selection
Imprinting, partially learned sexual behavior	Sexual behavior totally genetic
Chromosome number, large	Chromosome number, small
Total genomic map, length large	Total genomic map, length small
Crossover suppressors few or easily lost	Crossover suppressors many and not easily lost

In this section, I have shown that many attributes of the founder population can play a critical role in whether or not a genetic transilience will occur. The essential conclusions are summarized in Table 2.

DISCUSSION

As should now be evident from the arguments made in the preceding sections, not all founder events lead to speciation *via* the genetic transilience model. Indeed perhaps only a minority of founder events leads to such rapid speciation, although this mechanism of speciation might well be clustered in certain phylads because of the importance of such attributes as population structure, genome structure, system of mating, etc. Other founder events could also lead to speciation, either through fixation of chromosomal translocations or mutations that seriously decrease the fitness of heterozygotes (WRIGHT 1940, 1941; WHITE 1978; MAYR 1978) or simply by providing isolation from the ancestral population so as to allow speciation to arise as an indirect by-product of gradual adaptive divergence. Thus, there is not one founder principle in speciation, but several. Unfortunately, the distinction between these types of founder events has not generally been made in classifications of modes of speciation (*e.g.*, MAYR 1970; BUSH 1975; WHITE 1978; ENDLER 1977); yet, this distinction is essential for incorporating population genetics into both modeling and empirically analyzing the role of the founder effect in speciation. The incorporation of population genetic considerations into the model presented in this paper has, I hope, expanded both the explanatory and predictive powers of the genetic transilience (genetic revolution) model of speciation. The fact that this model makes specific predictions about what factors should be associated with genetic transilience means that this model is testable. I will now discuss some studies and observations that relate to the validity of this model.

As mentioned in the introduction, my empirical studies on "genetic revolutions" in the laboratory (TEMPLETON 1979b) helped inspire this model. As a result, my experimental results are obviously consistent with the model and do not contribute to substantiating its validity. Recently, however, there have been other experimental studies on the role of the founder principle in speciation that can be related to my model.

POWELL (1978) tested CARSON'S (1975) founder-flush theory, using populations of *Drosophila pseudoobscura* and found that premating isolating barriers evolved in some lines. The experimental design of POWELL (1978) had several features that were "optimal" for speciation under my model. First, he created an ancestral population by combining several different isofemale lines from various geographical localities. This would tend to increase the level of genetic variability in the ancestors and, in particular, the level of individual heterozygosity. Consequently, POWELL created the optimal ancestral/sampling structure under my model. Next, POWELL used only the most homokaryotypic lines as founders; thus, he effectively selected against crossover suppressors. Moreover, by the end of this experiment, almost all inversions were fixed (POWELL, personal communication), so that the few inversions initially present were lost

during the course of the experiment. This is also "optimal" under my model. Third, POWELL (1978) selected for those lines with highest $\bar{k}$ (average number of progeny) and insured that each bottleneck was followed by a "flush" of rapid population growth, once again, optimal features for inducing a genetic transilience. Fourth, chosing *D. pseudoobscura*, as opposed to a species such as *D. melanogaster*, was also optimal. *D. pseudoobscura* has a haploid chromosome number of five instead of four like *D. melanogaster* and, moreover, has generally longer map distances per chromosome, yielding a total map length of about 4.54 Morgans (STURTEVANT and NOVITSKI 1941; STURTEVANT and TAN 1937) as compared to 2.88 for *D. melanogaster*. Under my model, as chromosome number and map distance increase, the likelihood for genetic transilience also increases. Finally, and very importantly, inbreeding in *Drosophila pseudoobscura* does not cause a deviation from random mating (POWELL and MORTON 1979). This contrasts with the negative assortative mating induced by inbreeding in *D. melanogaster* (AVERHOF and RICHARDSON 1974).

These last two observations lead to an interesting prediction: if one repeated POWELL's experiments, but substituted *D. melanogaster* for *D. pseudoobscura*, one should be less successful in finding the evolution of isolating barriers, particularly in light of the way in which *D. melanogaster's* mating system responds to inbreeding (AVERHOFF and RICHARDSON 1976). One could extend this procedure even further by using a variety of Drosophila that differ in chromosome number and genomic map lengths; for example, if *D. virilis* were used instead of *D. pseudoobscura*, my theory predicts an even greater likelihood of isolating barriers than that reported by POWELL (1978), because for *D. virilis* $n = 6$, the total map length is about 7.92 Morgans (ALEXANDER 1976), and this species tends to lack inversions (DOBZHANSKY 1970). To test my model more accurately, experiments of the type performed by AVERHOFF and RICHARDSON (1976) should also be done to see how each species' mating system responds to inbreeding. Moreover, the ancestral population structure and sampling of the founders is under the experimentor's control in POWELL's design. For example, he could have simulated an Island model ancestral population by sampling his founders from within isofemale lines. Similarly, he could simulate correlated sampling by choosing relatives of known degree to be the founders. Similar kinds of extensions can also be incorporated into the design I used for testing "genetic revolutions" (*e.g.*, for the role of ancestral population structure in the *D. mercatorum* system, see TEMPLETON 1979c). The important point is that virtually all the predictions made in this paper are testable in the laboratory using current methodologies and techniques. This is an attribute rarely found in models of speciation.

Another possible experimental system for testing the genetic transilience model has been developed by WALLACE (1978). He placed individuals from several different species of Drosophila on an "artificial crab" to see if they could adapt to this radically new environment, as suggested by CARSON (1974). With this design, the flies were exposed to a selective bottleneck induced by the external environment, as well as a founder event (since all populations went through

bottlenecks of small size). Consequently, the selective bottleneck was extremely intense, and, in fact, most populations became extinct. I should point out that, although the selective bottleneck discussed in this paper was primarily induced by the genetic environment, most of the predictions made in this paper would also apply if the selective bottleneck were induced by the external environment; this is particularly so since most extreme external environments also induce bottlenecks in population size. It is also interesting to note that the only population that successfully adapted to this extreme environment—and to the extent that standard Drosophila medium was now lethal to it—was *D. virilis*, which, under my model, should be the type of Drosophila maximally liable to a genetic transilience with respect to chromosome number, genome map size and lack of crossover suppressors.

A final experimental study on the founder effect and speciation arose inadvertantly when a strain of the Hawaiian picture-wing *Drosophila adiastola* was brought into the laboratory and spontaneously underwent a crash-flush cycle (ARITA and KANESHIRO 1979). After this crash-flush cycle, mating tests revealed that strong premating isolating barriers had arisen between this laboratory strain and other strains that had not undergone such a crash-flush cycle. Similar results have been obtained in another Hawaiian Drosophila, *D. silvestris* (AHEARN 1980). This provides a laboratory demonstration of the ease with which a founder event can lead to isolating barriers in the Hawaiian Drosophila.

The Hawaiian Drosophila themselves provide an excellent natural test for the role of the founder event in speciation. The circumstantial evidence is quite strong that repeated inter-island founder events occurred in this archipelago, and these were almost always followed by speciation (CARSON *et al.* 1970; CARSON and KANESHIRO 1976). The model presented in this paper can explain much of the observed pattern of speciation in the Hawaiian Drosophila, including some of the anamolous features of this speciation. First of all, despite repeated founder events over a short period of geological time, the Hawaiian Drosophila do not show depauperate levels of isozyme polymorphism or individual heterozygosity, and local demes carry large stores of genetic variability comparable to that of continental demes (SENE and CARSON 1977; JOHNSON *et al.* 1975; CARSON and KANESHIRO 1976). Also, not only are levels of heterozygosity high in many species, but the between-population variation is usually rather modest (SENE and CARSON 1977; CARSON and KANESHIRO 1976). Consequently, based on inferences from the isozyme data, the population structure of many Hawaiian Drosophila apparently approximates a large, panmictic situation. This conclusion may seem somewhat at odds with the importance that kipuka ("islands" of rainforest isolated by lava flows) have apparently played in the evolution of Hawaiian Drosophila (CARSON and KANESHIRO 1976; RICHARDSON 1974). However, one must remember that the ancestral populations generally come from the older islands, while the founders inhabit the geologically new (and volcanically active) islands. Hence, the subdivision of the habitat into kipuka would be more pronounced for founders than for ancestors in general. Indeed, those species with the most interpopulation variability are generally found in the geologically

young areas (CARSON and KANESHIRO 1976). This transition from a more panmictic to a more subdivided population structure (at least initially) could greatly add to the chance of establishing a genetic transilience.

Another startling feature of the isozyme evolution of the Hawaiian Drosophila is the low rate of accumulation of genetic distance, even between species that have recently (less than 1,000,000 years ago) been separated by a founder event. Such a finding is not expected of founder effects in general, but is expected when coupled with the optimal attributes for genetic transilience (NEI, MARUYAMA and CHAKRABARTY 1975; TEMPLETON 1980).

The chromosome evolution of the Hawaiian Drosophila also has some peculiar features that are explained by this theory. First, the amount of inversion evolution has been rather modest when compared to that of continental Drosophila. Thus, about two-thirds of the Hawaiian species are from homosequential species groups (*i.e.*, species with identical polytene banding patterns), whereas this phenomenon is extremely rare in continental groups (CARSON *et al.* 1970; CARSON and KANESHIRO 1976). Moreover, the inversion evolution that has occurred is primarily in the form of fixed inversion differences between species. Thus, few inversion polymorphisms survive as a polymorphism through a founder event. This pattern cannot be explained by the founder event *per se* because, as already pointed out, isozyme polymorphisms are virtually unaffected by the founder event. Thus, the mode of speciation of the Hawaiian Drosophila is characterized by the retention of isozyme polymorphisms and the fixation of inversions (or, more commonly, no inversion evolution at all), which is precisely what the theory presented in this paper predicts (since inversions act as crossover suppressors in Drosophila). Moreover, the rather low rate of inversion evolution occurs despite evidence that some regions of the genome undergo frequent chromosomal breakage in the Hawaiian Drosophila (YOON and RICHARDSON 1978a,b). Thus, although new inversions may commonly occur, they apparently rarely persist for long in the population. YOON and RICHARDSON (1978a,b) attribute this to the fact that newly arisen inversions rarely acquire the "co-adaptation" (DOBZHANSKY and LEVENE 1951) that is necessary for their selective maintenance in a population. However, if a new inversion arose during the early flush phase of a founder colony, standard population genetic theory predicts that it should have a greatly increased likelihood of persisting for a long time, even if originally neutral or slightly deleterious (EWENS 1967). Given a long persistence, the inversion would then have a greatly increased likelihood of evolving "co-adaptation" (DOBZHANSKY and LEVENE 1951), perhaps through a mechanisms such as envisioned by WALLACE (1959) or HARTL (1977), and, thereby, persisting in the population as a balanced polymorphism. This hypothesis explains the fact that the third of the Hawaiian picture-winged Drosophila species that do have inversion polymorphisms are generally polymorphic for inversions not found in their ancestors (CARSON and KANESHIRO 1976). Thus, the genetic transilience model explains both the loss of ancestral inversion polymorphisms and the occasional production of new inversion polymorphisms that are associated with founder events in the Hawaiian Drosophila.

A second feature of the Hawaiian Drosophila chromosome evolution is that the haploid number is generally six, the maximum number in Drosophila, and has rarely been reduced in number by Robertsonian fusion. This is quite different from the continental pattern in which a reduction in chromosome number is a common occurrence (DOBZHANSKY 1970). However, this observation is in accordance with the prediction of this theory, since a reduction in chromosome number would lower the chance of a genetic transilience.

The Hawaiian Drosophila have many other attributes that are optimal for yielding a genetic transilience. First, a partially learned mate-recognition system (SENE 1977) greatly increases the *a priori* chance for a genetic transilience and stabilizes any transilience that occurs (CARSON 1978; TEMPLETON 1979a). Second, individual adults are very long-lived and reproduce throughout their life. Hence, the chance of getting founder females with high reproductive value is enhanced. Third, founder events are not generally associated with noticeable shifts in ecological niches (HEED 1971). Thus, the ecological significance of the founder event is in placing the population into a relatively unexploited environment that presents the same basic niches, a condition that promotes rapid population growth soon after the initial founder event. Finally, because individuals are long-lived and reproduce continuously in a near season-free environment, generations are broadly overlapping, which would intensify inbreeding effects in the expanding founder population. All of these attributes, when coupled with the previously considered geological, population structural and chromosomal factors, create a situation to insure that a founder event will have a high probability of causing a genetic transilience.

One difficulty in testing the genetic transilience model lies, perhaps, in the fact that it depends upon the ancestral population being polymorphic for loci controlling integrated developmental, physiological, behavioral, etc., traits that strongly influence fitness. Although extensive information exists concerning enzyme polymorphisms (*e.g.*, NEVO 1978), very little information exists concerning the types of polymorphisms needed for genetic transilience. This does not imply that isozyme data are totally irrelevant, for such data can reveal much about population structure that, in turn, can profoundly influence the chance for genetic transilience (TEMPLETON 1979b,c). It is also interesting to note that those enzyme loci most involved in regulating biochemical pathways (and hence, perhaps, more liable to a genetic transilience) are much more polymorphic in general than are nonregulatory enzyme loci (JOHNSON 1974). These facts indicate that the type of polymorphism required as a prerequisite for genetic transilience may be very common. More direct data on this point now exist for *Drosophila mercatorum*. The transilience in *D. mercatorum* observed in the laboratory affecting fundamental developmental, physiological and life history processes (TEMPLETON 1979b; TEMPLETON and RANKIN 1978) has been shown to depend on several loci that are polymorphic in the ancestral, natural population (TEMPLETON and RANKIN 1978; unpublished data). Similarly, in the Hawaiian Drosophila, many of the transiliences apparently occur in the integrated behavioral trait known as the mate-recognition system (CARSON 1978;

TEMPLETON 1979a). The ease with which genetic changes occur in this trait in the laboratory (AHEARN 1979; ARITA and KANESHIRO 1978) strongly implies that much genetic variability exists in natural populations. Moreover, CARSON and BRYANT (1979) have recently demonstrated the existence of intraspecific variation in natural populations of *Drosophila silvestris* for a secondary sexual character that may be involved in mate recognition. Hence, the types of polymorphisms that are prerequisite for genetic transilience definitely exist in some species, but many more studies on natural populations of the type being done on *D. mercatorum* and *D. silvestris* will be needed before an accurate evaluation of the applicability and generality of the genetic transilience model can be made.

As the above discussion has shown, the theory presented in this paper has increased the explanatory powers of the "genetic revolution" model of speciation and, more importantly, has generated testable predictions that can be examined in both the laboratory and the field, using current methodologies and systems. I hope that this theory demonstrates how population genetic theory can be applied to the problem of speciation in a more extensive and thorough fashion than it has in the past. Perhaps, some day, MAYR's (1978) lament that it is startling "how little population genetics has contributed to our understanding of speciation" will no longer be true.

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