Control of Meiotic Drive of B Chromosomes in the Mealybug, *Pseudococcus affinis* (*obscurus*)

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ABSTRACT

Isofemale lines of *Pseudococcus affinis* (MASKELL) differ in their ability to maintain B chromosomes (Bs) due to the presence of genotypes that affect the rate of transmission (k) of the Bs. The nature of these genotypes was analyzed by comparing ks of males carrying the same B and the same paternal genome (which is heterochromatic), but differing in their maternal genome. In males from line L-60, which maintained the B at a frequency of over 4.0 Bs per individual, the mean k varied between 0.7 and 0.95 in different experiments. Over the same period, the mean k of males with a maternal genome from one of two lines in which the B was rapidly lost (L-119), increased from 0.5 to 0.9, and that of the other line (L-230) decreased gradually from 0.6 to less than 0.1. The ks appear not to be correlated with the geographical or parental origin of the B. The observed changes in k are attributed at least in part to changes in the frequency of genotypes (alleles) which can drastically reduce the transmission of the B and, when present in high frequency, can lead to its rapid loss. The frequency distribution of the ks of sons of F_1 females from the cross L-230 × L-60 suggests that the two lines differ at two unlinked loci with additive effects on k. The genome of L-119 also caused the B to undergo nondisjunction in about 10% of the primary spermatocytes. A comparison between the ks of the males tested and those of males from a natural population suggests that in that population the B is "parasitic" and that the frequency of transmission-reducing genotypes is low.

THE supernumerary, or B chromosomes (Bs) of several plant and animal species are transmitted at a higher rate than that expected according to Mendelian inheritance (0.5) (JONES and REES 1982) and may thus be considered to exhibit meiotic drive. In several species with Bs exhibiting meiotic drive which were studied in detail, the Bs were found to reduce at least some of the components of fitness in the individuals carrying them (reviewed in JONES 1985). Thus, it has been proposed that such Bs are "parasitic" or "selfish" and that they are maintained only because they exhibit meiotic drive (KIMURA and KAYANO 1961; NUR 1966a, 1969, and 1977; THOMSON 1984; GREGG, WEBB and ANEDA 1984). This proposal led WHITE (1973) and others to ask why the "host" species have not gotten rid of the Bs. This question is intriguing, because of reports that individuals from natural populations of the same species differed in the rate of transmission of their B chromosomes (PARKER, TAYLOR and ARMSWORTH 1982) and that the differences in transmission were apparently due to the host's genome and not to the B (SHAW, HEWITT and ANDERSON 1985). Moreover, in our recent study of the Bs of the mealybug, *Pseudococcus affinis* (MASKELL) (= *P. obscurus* ESSIG) (NUR and BRETT 1985) we found genotypes which can reduce the rate of transmission (k) of the B from over 0.9 to less than 0.1 and can lead to the rapid loss of the B from the lines carrying it. These observations raise the questions why these species do not become fixed for such genotypes and what role do these genotypes play in the maintenance of the Bs. Thus, it is clear that in order to understand the evolution and population dynamics of B chromosomes, it is necessary to know the genetic nature of the genotypes affecting the Bs' rate of transmission, their frequency and the effect that they may have on the rest of the host genome and on the phenotype.

In the present study we provide information about the nature of these genotypes in *P. affinis* and discuss the bearing of this information on the question of whether the Bs reduce the fitness of individuals carrying them, and may thus be considered to be "parasitic." We also attempt to account for an apparent discrepancy between the rapid loss of the Bs from some of the lines maintained in the lab and the observed rate of transmission of the B in controlled crosses (NUR and BRETT 1985), as well as the temporal changes in the rate of transmission of the B in males from three isofemale lines.

MATERIALS AND METHODS

This report presents data on the rate of transmission of a B chromosome by males with one B (1B males) from seven new series of crosses (III–IX) as well as from those of our previous study (series I and II of NUR and BRETT 1985).

The crosses involved the same isofemale lines used earlier (Table 1 in NUR and BRETT 1985). Each of the lines used was established from one inseminated female. The lines were maintained by transferring the ovisacs of 5–10 females to a new potato each generation. Additional details are presented in NUR and BRETT (1985). The lines used in the crosses were:

Line 60 (L-60): This line originated from a female collected in 1973 at Davis, California. When the line was first examined cytologically in generation 18 (G_{18}), it had no Bs. In G_{27} a B from L-72 was introduced into a subline of L-60 [designated L-60 (B-72)] at a frequency of about one B per individual (b = 1.0). The crosses used to transfer cleanly a B from one line to another are described later. The frequency of the B in L-60 (B-72) increased rapidly to about 4.0 in 10 generations. This line provided the paternal set of chromosomes of all the males tested, and for some of the males (the E60 males) it also provided the maternal set.

L-72: This line originated from a female collected in 1973 from the same row of oleanders as L-60. When first examined in G_{18}, the frequency of the B was 5.3. This frequency remained fairly stable and in G_{49} it was about 5.5 Bs per individual. This line provided the B whose k was tested in series I, II, VII and VIII.

L-119: This line originated from a female collected on myrtle at Davis in 1975. When first examined in G_9, the frequency of the B (b) was 2.5. In G_{15} b was only 0.1 and the B was lost soon afterward.

L-230: This line originated from a female collected on yew in 1975 at Berkeley, California. When first examined in G_9, it had no Bs. In G_{17}, Bs were introduced from L-251 into a subline of L-230 at a mean frequency of about 2.0, but by G_{22} the Bs were lost. On the basis of our previous study (NUR and BRETT 1985), this line, as well as L-119 provided an E set which was assumed to carry transmission-reducing genotypes (TRGs).

L-251: This line originated from a female collected on the same yew tree as L-230. When first examined in G_9, b was 4.1. b remained fairly stable, and in G_{37}, b was 4.4. This line provided the B whose k was tested in series III, IV, V and IX.

Determining the karyotypes of males and females: In females the number of Bs present was determined in cells in interphase, by counting the number of heterochromatic (H) bodies present in large (polyploid) nuclei (NUR 1962). In adult males, however, the number of Bs cannot be determined directly because of the presence of an H set in most of the nuclei, and because most of the large nuclei lacking an H set degenerate prior to the adult stage. Thus, the karyotypes of the males used in the crosses had to be determined indirectly. However, the karyotypes of males derived from certain crosses can be inferred fairly accurately from those of the males' sibs (usually sisters) and from those of 20 or more daughters of these males and 0B females.

The males tested were produced by two crossing schemes. In the first scheme the males received their B from a 1B male from a subline of L-60, L-60(B), and males of this subline usually transmitted their B to about 90% of their offspring. This scheme was used to produce the males of series I–V and VII, except that in series V the males received their B(s) from a 2B male (Figure 1a). In the second scheme, used in series VI, VIII an d IX (Figure 1b), the males received their B from their 2B mothers, which were also expected to transmit a B to about 90% of their offspring. However, about 3% of the offspring of such females usually receive two Bs (NUR 1969). Thus, according to both crossing schemes, only about 90% of the males used in the crosses were expected to carry a B; the rest were expected to carry

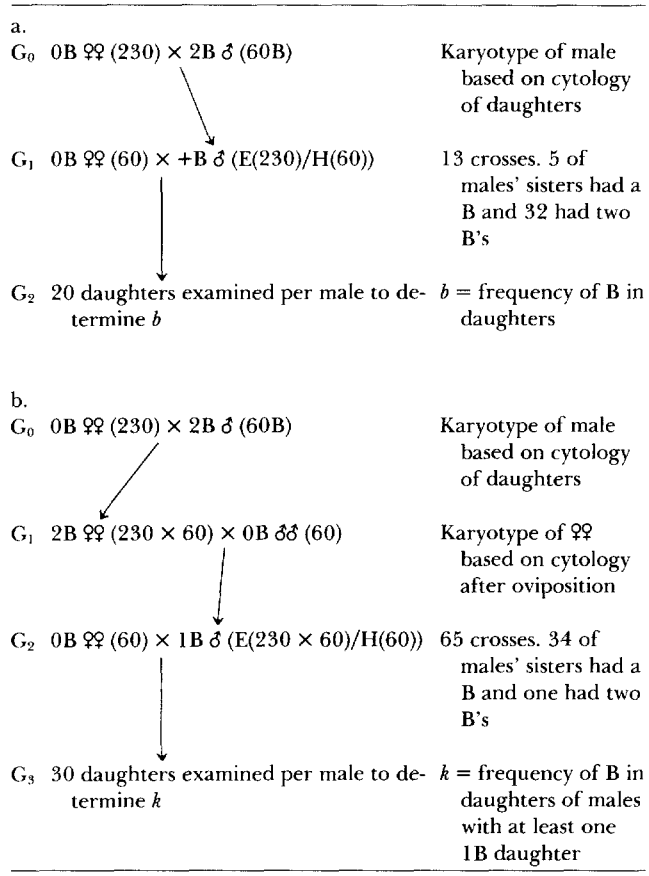

FIGURE 1.—The two crossing schemes used to test the effect of different euchromatic (E) sets of chromosomes (of maternal origin) on the rate of transmission of the B chromosome (B) by males. In all the males tested, the heterochromatic (H) set (of paternal origin) came from L-60, or from a subline of L-60 [L-60(B)]. a, The crossing scheme used to obtain the males of series V (as well as those of the other series), in which the tested males received their B from their father. In the males whose b was measured the euchromatic set of chromosomes came from line 230. On the basis of cytology of their sisters, most of the 13 +B males used in the crosses were expected to have two Bs and the rest one B. b, The crossing scheme used to obtain the males of series VI (and other series), in which the tested males received their B from their 2B mother. On the basis of cytology of their sisters, almost all the 65 males whose k was measured [the E(230 × 60)/H(60) males] were expected to have one B.

no Bs, or if they were produced by the second scheme, either no Bs or two Bs.

Terms used:

b: The mean frequency of the B in a line, a population or the offspring of a particular male (sibship).

k: The rate of transmission. In 0B × 1B or 1B × 0B crosses $k = b$.

+B males or females: Individuals with one or more Bs, or individuals that contributed at least one B to at least one offspring.

0B and +B crosses or sibships: 0B × +B or +B × 0B crosses in which none (0B), or at least one (+B) of the offspring carried a B (Bs).

E60, E119 and E230 males (and crosses): Males carrying a euchromatic (E) set of chromosomes (of maternal origin) from a particular line (*e.g.*, L-60), or the crosses involving these males.

TRGs: Transmission-reducing genotypes. Genotypes (or alleles) reducing the mean k significantly below the level in a line carrying the same B, and which exhibits the highest k.

RESULTS

Background: In populations of *P. affinis* an individual may have 0–8 B chromosomes (Bs) and the mean may vary from zero to over 2.7 Bs per individual (NUR 1969). The B is smaller than the regular chromosomes, has no obvious effect on the phenotype and is usually heterochromatic. The rate of transmission of the B (k) in females ranges from about 0.3 to 0.5, depending on the number of Bs present. In males, however, k is usually about 0.9 (NUR 1969) and thus the B exhibits a strong meiotic drive. The drive depends on the peculiar chromosome system of this and other mealybugs (the lecanoid chromosome system). In males with this system the chromosome set of paternal origin becomes heterochromatic and genetically inactive in early embryogeny (BROWN and NELSON-REES 1961). During spermatogenesis the heterochromatic (H) set and the euchromatic (E) set (of maternal origin) segregate and only the nuclei carrying the E set develop into sperm. Thus, males only transmit chromosomes of maternal origin, except for the B which is usually transmitted regardless of parental origin (NUR 1962). The transmission of the B apparently comes about as a result of a change in the B from being heterochromatic to being even less condensed than the E set (negatively heteropycnotic). The B then usually segregates with the E set in over 90% of the secondary spermatocytes.

The fact that males transmit Bs regardless of origin, but do not transmit non-B chromosomes of paternal origin, makes it possible to transfer Bs from one line to another rapidly and cleanly by crossing a male with Bs to a female from a line lacking them (a 0B line) and backcrossing a son with Bs to another female from the 0B line (NUR 1966b). This procedure makes it fairly simple to test the effect of different genetic backgrounds on k. Moreover, because in males the paternal set is heterochromatic and apparently genetically inactive, the effect of the genetic background on k was studied by comparing males which all have the same H set but differ in the origin of their E set.

The experimental series: This study presents the results of nine series of crosses which were each designed to answer questions about the possible effect of various factors on the rate of transmission of the B (k). All the males whose k was measured carried an H set of chromosomes from line 60 (L-60), but differed in the origin of their E set of chromosomes and/or in the origin of the B. The B came either from L-72 from Davis (as in NUR and BRETT 1985) or from L-251 from Berkeley, and was received by the tested males either from their 1B father or their 2B mother (see MATERIALS AND METHODS). The E set of the tested males came from one of the three lines used in the previous study (lines 60, 119 and 230, see MATERIALS AND METHODS). L-60 was assumed not to carry transmission-reducing genotypes (TRGs), because males of this line had a mean k of over 0.9 (NUR and BRETT (1985). The other two lines (L-119 and L-230) were assumed to carry TRGs, because in these two lines the Bs were rapidly eliminated (see MATERIALS AND METHODS) and because the ks of males carrying an E set from either of these lines were significantly lower than the ks of males of L-60 (NUR and BRETT 1985).

As indicated in MATERIALS AND METHODS, only about 90% of the males tested were expected to carry a B, and the karyotypes of the males could not be determined cytologically. Thus, the karyotype of each male was inferred from the frequencies of the 0B, 1B and 2B karyotypes among 20, 30 or 40 of its daughters, and from the frequency of the various karyotypes among the males' sibs. The aim of each of the series will now be described briefly.

Series I and II: These series were initiated following the observation that when isofemale lines were established from females from natural populations with Bs, the frequency of the Bs usually either increased rapidly to a mean of over 4.0, or the Bs were rapidly lost. The aim of these series was to determine whether the rapid loss of the Bs in L-119 and L-230 was the result of the presence of genotypes which suppress the high k usually observed in males. The results indicated that indeed such transmission-reducing factors or genotypes [abbreviated TRFs in NUR and BRETT (1985), and TRGs in this report] are present in these lines, because in series I and II the mean ks of males with an E set from L-119 (E119 males), and of the males with the E set of L-230 (E230 males) were significantly lower than in L-60 (Table 1), in which once the Bs were introduced they were maintained at a mean frequency of more than four Bs per individual. However, the ks of the E119 and E230 males varied greatly and were still higher than 0.5, and thus could not explain the rapid loss of the B in the two lines.

Series III and IV: The aim of these series was to determine whether the discrepancy between the observed ks of the E119 and E230 males and that which would have been necessary to bring about the rapid reduction in the frequency of the B (b) (from $b = 2.5$ to $b = 0$ in six or fewer generations in L-119, and from b of about 2.0 to $b = 0$ in five or fewer generations in L-230) was due to a greater sensitivity of the Bs in L-119 and L-230 to the effect of the TRGs. At the time that series III and IV were initiated, the B of L-119 had already been lost, but the B of L-230 came from L-251 and was still available. Thus, it was introduced to the same three types of male as in series

TABLE 1

Effect of the origin of the euchromatic (*E*) set of chromosomes (of maternal origin) and of the B chromosome on the mean rate of transmission (*k*) of the B by 1B males

E set of males	Genera-tion	Series	Origin of B	Males tested	1B males	Mean *k* of 1B ♂♂	SE of *k*	OB crosses excluded	Frequency of OB sibs (*n*)
L-60 (Davis)	27	Pre-I	Davis, M	2	2	0.870	0.044	0 (0)	
	28	I	Davis, P	23	19	0.913	0.030	4 (0.17)[a]	
	30	Pre-II	Davis, M	2	2	0.900	0.033	0 (0)	
	31	II	Davis, P	15	13	0.954	0.016	2 (0.13)[a]	
	33	Pre-III	Berkeley, P	4	4	1.000	0	0 (0)	
	34	III	Berkeley, P	20	20	0.698	0.056	0 (0)	0 (30)
	38	Pre-IV	Berkeley, P	4	4	0.930	0.047	0 (0)	
	39	IV	Berkeley, P	23	22	0.893	0.043	1 (0.04)	0.04 (23)
	44	Pre-VII	Davis, P	8	8	0.832	0.041	0 (0)	
	45	VII	Davis, P	23	21	0.776	0.040	2 (0.09)[a]	0.07 (30)
	45	VIII	Davis, M	20	19	0.905	0.023	1 (0.05)[a]	0 (10)
L-119 (Davis)	21	I	Davis, P	23	21	0.529	0.038	2 (0.09)	
	24	II	Davis, P	15	14	0.782	0.055	1 (0.07)[a]	
	27	III	Berkeley, P	19	19	0.737	0.061	0 (0)	0 (10)
	33	IV	Berkeley, P	24	23	0.920	0.029	1 (0.04)	0.20 (20)
	38	VII	Davis, P	20	18	0.856	0.028	2 (0.10)[a]	0.10 (30)
L-230 (Berkeley)	20	I	Davis, P	22	18	0.686	0.045	4 (0.18)[a]	
	23	II	Davis, P	21	9	0.556	0.086	12 (0.57)	
	23	IIC	Davis, P	14	14	0.404[b]	0.066		
	26	III	Berkeley, P	20	15	0.207	0.042	5 (0.25)[a]	0 (10)
	26	IIIA	Berkeley, P	20	15 + 4[c]	0.163	0.033	1 (0.05)[a]	
	32	IV	Berkeley, P	26	2	0.050	0.017	24 (0.92)[a]	0 (20)
	32	IVA	Berkeley, P	26	2 + 23[c]	0.004	0.003	1 (0.04)[a]	
	34	V	Berkeley, P	13	2	0.100[d]	0.005	11 (0.85)	0 (57)[e]
	34	VA	Berkeley, P	13	2 + 11[c]	0.015[d]	0.012	0 (0)	
	36	VII	Davis, P	26	12	0.161	0.036	14 (0.54)[a]	0.15 (30)
	36	VIIA	Davis, P	26	12 + 10[c]	0.088	0.027	4 (0.15)[a]	
	38	IX-1	Berkeley, M	23	17	0.322	0.045	6 (0.29)[a]	0.21 (28)
	38	IX-1A	Berkeley, M	23	17 + 1[c]	0.304	0.042	5 (0.22)[a]	
	38	IX-2	Berkeley, M	13	4	0.188	0.077	9 (0.69)[a]	0.25 (20)
	38	IX-2A	Berkeley, M	13	4 + 5[c]	0.083	0.038	4 (0.31)[a]	
L-230 × L-60	34	VI	Berkeley, M	63	57[a]	0.480	0.039	6[f] (0.10)	0 (35)

All the males carried a heterochromatic set from L-60. Gen. is the age of the line at the time that the males' mothers were isolated. P and M indicate whether the male received the B from a 1B father or from a 2B mother (Figure 1). Pre-I, Pre-II, etc., are the crosses which produced the males whose *k*s were measured in series I, II, etc. The data are based on the cytology of 20 daughters per male, except as indicated (a and f). The data for series I and II are from NUR and BRETT 1985.

[a] Based on the cytology of 30 daughters per cross.

[b] Based on the segregation of the B in spermatogenesis (see NUR and BRETT 1985).

[c] Number of crosses in which all the analyzed daughters lacked a B (OB crosses), but which on the basis of the cytology of the males' sisters were assumed to have been sired by 1B males.

[d] *k* is based on the assumption that all the males had one B.

[e] Seven 1B and 50 2B sibs.

[f] Based on the cytology of 40 daughters per cross.

I and II (NUR and BRETT 1985). The comparison between the *k* of a B from Davis (series I and II) and one from Berkeley (series III and IV) was also of interest because the frequency of the B in the populations studies from Davis was 0.1–1.0, while in those from Berkeley it was 1.1–2.0 (NUR 1969) and it was of interest to know whether this difference may have been, at least in part, due to a difference between the Bs.

The results of series III and IV indicate that while there are some differences in the mean *k* between series III and IV and series I and II (Table 1 and Figure 2), these were not greater than the differences between series III and series IV. The data also suggest that in the course of the four series *k* increased in the E119 males, decreased in the E230 males, and decreased and then increased in the E60 males. These results raised the possibility that the differences between the series were due to changes in the frequency of the TRGs in these lines and not to differences between the Bs used. This possibility was tested in series VII.

Series V: The aims of this series were: (1) to test the effect of the TRGs of L-230 on the *k* of 2B males and (2) to determine cytologically whether the low *k* of the E230 males was due to the loss of the B prior to, or during spermatogenesis.

Series VI: This series tested the *k*s of 1B males with

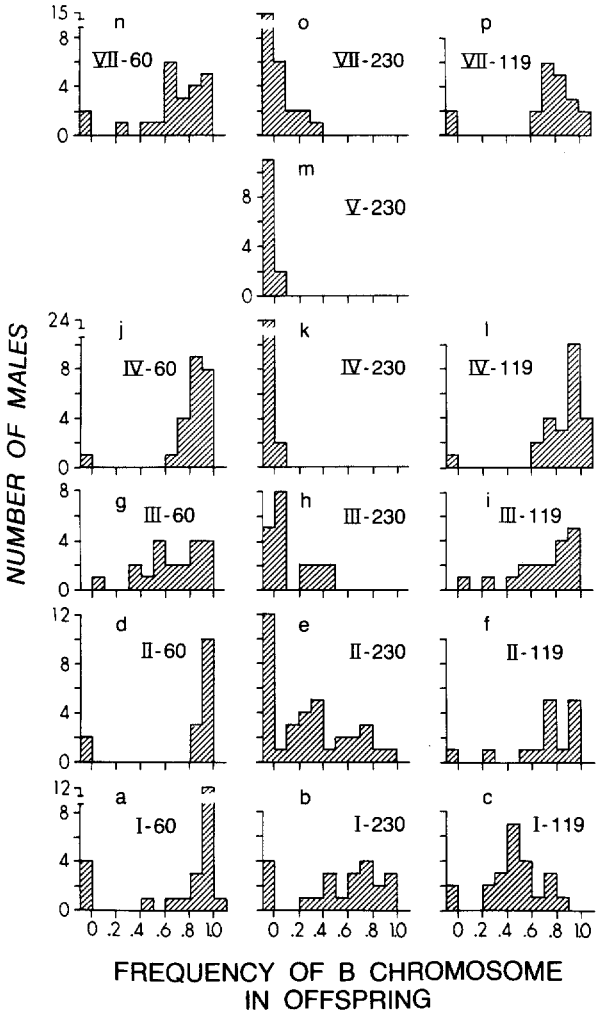

FIGURE 2.—The frequency distributions of the mean number of Bs per daughter (b) of the males from those series in which the males received their B from their father (see Figure 1a). The data are presented according to series (I–VII) and according to the line which provided the E set of chromosomes, *i.e.*, L-60, L-119 and L-230. The data for series I and II are from NUR and BRETT (1985). The bs are based on the cytology of 20 daughters (see Table 1), and are grouped into the ranges 0, 0.01–0.1, 0.11–0.2, etc. On the basis of the type of cross that produced the males and the cytology of sibs, all the males with $b > 0$ had only one B, except for the two E230 males of series V, which probably had two Bs. All the E60 and E119 males with $b = 0$ apparently did not have a B, while most of the E230 males with $b = 0$ had one B (or in series V, two Bs), but failed to transmit it to any of their analyzed daughters (see text). In series I, II and VII the B came from L-72 (from Davis), while in series III–V it came from L-251 (from Berkeley).

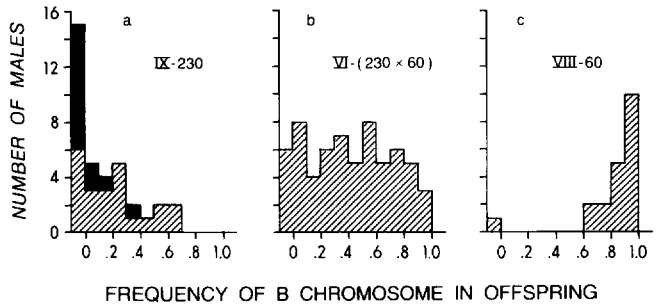

FIGURE 3.—The frequency distributions of the mean number of Bs per daughter (b) of the males from the three series in which the males received their B from their 2B mother (see Figure 1b). The bs are based on the cytology of 20 (series VIII and IX) or 30 (series VI) daughters of each male. Other details are as in Figure 2. On the basis of the cytology of the males' sisters, one or more of the E(230 × 60) males with $b = 0$ and most of the E230 males with $b = 0$ (series IX) apparently had one B but failed to transmit it. The data from the males of series IX-2 are in black.

an E set derived from F_1 females from a cross between males from L-60 (with high k) and females from L-230 (with low k). The aim of this series was to determine the number of loci involved in the difference in k between the two lines. In the course of setting up the crosses for this series it was observed that the k of the L-60 males, which were expected to contribute the B to the males to be tested, was fairly low (about 0.7). Thus, the crossing scheme was changed to the second scheme, in which the tested males receive their B from a 2B mother (Figure 1b), because in such a scheme over 90% of the offspring are expected to receive a B.

Series VII: This series used a B from L-72, as in series I and II, and its aim was to test whether the differences between the first two series and series III and IV were due to the source of the B, or to temporal genetic changes in the lines.

Series VIII and IX: These series determined the ks of E60 and E230 males which received their B from 2B mothers. Their aim was to determine the effect on k (if any) of whether a male receives the B from its mother, or its father. In an earlier study (NUR 1962) there was no evidence that the parental source of the B affected k, but the number of crosses of each type was fairly small (five), and we thought it worthwhile to examine this question again. Thus these series serve as a control for series VI.

The main conclusions from these series are that the ks of the males depended mostly on the source of the E set, but that it also depended on the age of the lines at the time that they provided the E set. In order to demonstrate these conclusions, the results of all the crosses are presented in some detail according to the origin of the E set of the males tested, and within each type, according to series.

The E60 males: The k of males with an E chromosome set from L-60 (E60 males) was determined in six of the series (I–IV, VII, VIII) and the results are presented in Figures 2 and 3 and are summarized in Table 1. As was explained earlier, the karyotypes of the males tested were determined indirectly. Thus, before calculating the mean k of the E60 males in each of the series, it was necessary to infer the karyotype of each of the males used in the crosses from the karyotypes of the daughters and of the sibs. In the crosses sired by the E60 males, the frequency of crosses in which none of the 20 or 30 analyzed daughters carried a B (0B crosses) is very similar to the frequency of 0B individuals among the males' sisters

(Table 1, last two columns). Thus, it is assumed that the 0B crosses (sibships) were sired by 0B males, and these crosses were excluded from the calculation of the mean *k* per male (which for 1B males is the same as the frequency of the Bs per daughter). All the other crosses (sibships) contained at least one 1B daughter and all but one were assumed to be the offspring of 1B males. The remaining sibship (from series VIII) consisted of 15 2B daughters and five 1B daughters, and it is assumed to have been sired by a 2B male. Thus, the data from this cross was excluded from further analysis.

Once each of the E60 males was assigned a karyotype it was possible to calculate the mean *k* per 1B male (Table 1). In the new series (III, IV, VII and VIII), the mean *k*s of the E60 males (0.7–0.9) were mostly lower than those observed in our previous study (series I and II, Table 1), in which *k* was over 0.9. In order to try to understand the cause of this unexpected variation in *k*, we also examined the *k*s of the 1B males which sired the E60 males tested in five of the series (I–IV and VII), because in these males both sets of chromosomes also came from L-60 (as in the E60 males). The data from these additional crosses are listed in Table 1 as "Pre-I, Pre-II," etc., just above the data about each series. As may be seen in Table 1, however, the combined data also do not provide a clear pattern of the variation in *k*: the *k*s appear not to be correlated with the age (in generations) of L-60 at the time of the crossing, or with whether the B came from Davis or from Berkeley. There is also no clear evidence that *k* was affected by whether the 1B males received their B from their father (series I–IV and VII) or their mother (Pre-I, Pre-II and series VIII). The origin of the variation in *k* will be discussed later. At this point it should be noted that even the lowest mean *k* observed (0.7; series III) represents a fairly strong meiotic drive, and is significantly greater than that of the E230 males of the same series (about 0.2).

The E230 males: The *k* of the E230 males was measured in seven series (I–V, VI and IX) and the results are presented in Figures 2 and 3, and are summarized in Table 1. In each series the males whose *k* was measured were the offspring of two or more females (sisters), and in most series the mean *k*s of the sons of the sisters were similar. Thus, for most series, the results from the sons of all the mothers are presented together. In series IX, however, the *k*s of the sons of the two 2B females used were significantly different ($t = 2.70$, 34 d.f., $P < 0.05$) and thus in Table 1 and in Figure 3 they are presented separately (as IX-1 and IX-2).

In Table 1, the mean *k*s of the E230 males of each of the new series are presented in two lines (*e.g.*, III and IIIA). The first is based only on the +B crosses (cross with at least one 1B daughter), and the second on the assumption that some of the 0B crosses involved +B males which failed to transmit a B to any of their 20 (or 30) analyzed daughters. The number of 0B crosses which were likely to have been fathered by +B males, and thus were included among those used to calculate the mean *k*, is based on the frequency of the males' sisters which lacked a B, and the number of sisters which were examined cytologically in each series (Table 1, last column).

The conclusion that some of the E230 males carried a B, but failed to transmit it to any of their analyzed daughters, is based on the cytology of sisters of the E230 males of the various series and especially on the cytology of the brothers and sisters of the males used in series V. Series V consisted of 11 0B crosses (sibships) and two +B sibships, one in which the frequency of the B among the daughters (*b*) was 0.05 ($b = 0.05$) and one in which *b* was 0.15. Among the sisters, five had one B and 32 had two Bs, and among the brothers two had one B and 18 had two Bs. These data suggest that most of the 13 males used in the crosses had two Bs but failed to transmit them to any of their 20 analyzed daughters. The low *k* of these males was confirmed by determining the number of Bs present in early spermatids in brothers of the 13 males used in the crosses (see Figure 4 in NUR and BRETT 1985). In the four 2B males in which 50 or more spermatids could be scored the *k*s were 0, 0.01, 0.03 and 0.06, while in the one 1B male scored, *k* was 0.02. Thus, in series V almost all the E230 males used in the crosses carried two Bs, but failed to transmit them to any of their 20 analyzed daughters. These observations suggest that the reduction in *k* in 2B males, due to the TRGs, is about as great as in 1B males. The cytological analysis of brothers of the males used in series II and V also demonstrated that the low *k* of these males is due to the failure of the B to become negatively heteropycnotic (uncondensed) during prophase I (see RESULTS, *Background*), and to segregate with the E set during anaphase II of spermatogenesis. The results of the cytologs will be described in a separate publication.

The above discussion indicates that the mean *k*s of the E230 males with at least 1B daughter (the +B males) is higher than the true mean *k*s of all the E230 males which carried Bs. Nevertheless, even the mean *k*s of the E230 males with at least 1B daughter are still consistently much lower than those of the E60 males. Thus, the new data strongly support the conclusion of NUR and BRETT (1985) that L-230 carried transmission-reducing genotypes.

Overall, the mean *b*s (and the mean *k*s) of the E230 males decreased with time, and the decrease is highly significant; the correlation between the age (in generations) of L-230 at the time that it was used to produce the E230 males of the various series, and the

TABLE 2

Effect of the source of the euchromatic (*E*) set of 1B males on the production of 2B daughters (♀♀)

Source of *E* set	Origin of B	Series	No. of 1B ♂♂	Males with 2B ♀♀	No. of 2B ♀♀	2B♀♀/+B♀♀	Frequency of +B ♀♀
Line 60	Davis	I	19	0	0	0	0.913
	Davis	II	13	0.077	1	0.004	0.950
	Berkeley	III, IV	42	0	0	0	0.800
	Davis	VII	21	0.095	3	0.009	0.767
	Totals and means		95	0.032	4	0.003	0.836
Line 119	Davis	I	21	0.333	13	0.062	0.498
	Davis	II	14	0.786	14	0.068	0.732
	Berkeley	III	19	0.263	6	0.022	0.721
	Berkeley	IV	23	0.565	25	0.063	0.865
	Davis	VII	18	0.611	16	0.055	0.811
	Totals and means		95	0.494	74	0.054	0.725
Line 230		I–IV, VII	56	0	0	0	0.346

Data are based on the cytology of 20 daughters (♀♀) per cross. A comparison between the frequency of females with either one or two Bs (frequence of +B females; last column) and the mean *k* of the various types of male in column 7 of Table 1 shows the effect of the presence of 2B daughters on *k*.

frequency of the B among the daughters (*b*) of each of the E230 males is $r = -0.475$, 176 d.f.; $P < 0.0001$. The possible origin of the variation in *k* within and between series will be discussed later. As in the data from the E60 males, there was no clear evidence that *k* was affected by the source of the B (Davis *vs.* Berkeley) or by whether the males received their B from their father or their mother.

The E119 males: The *k* of the E119 males was determined in five series (I–IV and VII), and in each of these series the frequency of the 0B crosses was 10% or lower. Thus, it was assumed that all the 0B crosses involved 0B males. Each of the series also had six or more males with one to three 2B daughters (Table 2), but none of the analyzed sisters of these males had two Bs. Thus, the *k*s of these males were calculated on the assumption that all the E119 males carried only one B. The presence of several 1B males with a few 2B daughters in five different series supports the conclusion reached earlier (NUR and BRETT 1985) that 1B males with the E set of L-119 occasionally transmit two Bs.

A cytological examination of spermatogenesis in brothers of the E119 males used in series II demonstrated that the mechanism by which these 1B males produce 2B daughters is nondisjunction of the sister chromatids of the B during anaphase I. The cytological analysis also indicated that unlike the situation in the E230 males, in the E119 males the B is negatively heteropycnotic during prophase I and metaphase I and usually segregates with the E set, and that at least some of the reduction in *k* is due to the loss of the B prior to spermatogenesis. Thus, L-119 and L-230 apparently carried different TRGs. The results of the cytological analysis will be described in a separate publication.

As may be seen in Table 1, the mean *k* of the E119 males in series I was only about 0.53 and was much lower than the *k* of the E60 males. The *k* of the E119 males then rose to 0.78 in series II, remained about the same (0.74) in series IV, rose to 0.92 in series IV and remained high (0.86) in series VII. In the last three series, the mean *k* of the E119 males was even slightly higher than that of the E60 males of the same series (Table 1). Thus, it is likely that at the time of these series, L-119 no longer had TRGs. The overall pattern is a highly significant increase in *b* (and in *k*) over time; the correlation between the age (in generations) of L-119 at the time that the various E119 males were produced and the *b*s of these males is $r = 0.371$, 99 d.f.; $P < 0.0001$.

The E(230 × 60) males: As described in the previous sections, the *k*s of the E230 males were much lower than those of the E60 males and this was attributed to the presence of TRGs. In order to try to determine the genetic nature of the difference in *k* between the two lines, *k* was measured in 64 males (series VI) whose E set came from F_1 females from a cross between a 2B male from L-60(B-251) and two 0B females from L-230 (Figure 1b). Some of the F_1 females had two Bs and these were crossed to L-60 males, and thus the males tested [designated E(230 × 60) males] received their H set from L-60 and their B from their mother.

A cytological examination of 35 sisters of the 64 males used in series VI revealed that 34 had one B and one had two Bs. Following the cytology of 30 daughters from each of the 64 crosses, one cross (sibship) was excluded from further analysis because most of the daughters had two Bs and it was apparently sired by a 2B male. The remaining 63 males had either only 0B daughters (six males), or both 0B and

1B daughters (57 males), and it is assumed that the latter were sired 1B males. The frequency distribution of the mean number of Bs per daughter (b) in these males is given in Figure 3 and summarized in Table 1. The ks of the +B (1B) males ranged from 0.067 to 0.97, and the frequency distribution of the ks was fairly flat and did not exhibit any clear peaks. An analysis of the frequency distribution of ks in these males suggests that the difference in k between L-60 and L-230 are due to the presence of different alleles at two loci affecting k (see DISCUSSION).

The mean k of the (E230 × 60) males (0.48 ± 0.04) is only slightly higher than the midpoint between the mean ks of the males with the E set of the two parental lines (the E60 and E230 males) in the series which preceded series VI (0.45; series IV) and also the midpoint (0.43) in the series which immediately follow it (series VII). Moreover, it is only slightly lower than the midpoint (0.55) between the ks of the E60 and E230 males in the only other two series in which the males received their B from their mothers (series VIII and IX). These results suggest that the genetic factors affecting k do not exhibit a strong maternal effect or epistasis.

DISCUSSION

Nature of differences in k among males of each type: As was discussed earlier, the mean rate of transmission of the B (the mean k) in the three types of male studied (E60, E119 and E230) varied from series to series, and in some of the series there were also large differences in k among males of the same type (Figures 2 and 3). Such differences in k can be caused by (1) differences in one or more uncontrolled aspects of the environment, (2) genetic differences between males of the same type, or (3) a combination of the two. At present, the only environmental factor known to affect the k of a B chromosome is temperature (SHAW and HEWITT 1984). In the present study, however, temperature may be ruled out, because it varied during the year only from about 21° to 24° and because it did so in an irregular way. Thus, it cannot account either for the intratype variation within series, or for the significant increase in the ks of the E119 males, and for the decrease in the ks of the E230 males. There may be other environmental factors that can affect k, but again there is no reason to assume that these would affect the ks of the three lines differently. It is likely, therefore, that at least part of the observed intratype variations in k within and between series is genetic.

The possibility that the intratype variation in k had a genetic basis is supported by the observation that the same B behaved differently in different lines from the same population (*e.g.*, in L-230 and L-251). This observation strongly suggests that the populations from which the lines came were polymorphic for alleles affecting k. Thus, it seems reasonable to assume that at the time that the isofemale lines were established, some were polymorphic for such alleles, and that at least some of the temporal changes in k in these lines are due to changes in the frequency of these alleles over time. One possible difficulty which must be considered in trying to invoke changes in the frequency of alleles to account for the observed variation in k is that all the lines were maintained by transferring the ovisacs of only five to ten females of each line to a new potato each generation. Thus, under this procedure the effective population size (N_e) must be small (<12) and in the absence of strong selection genetic drift is expected to lead to a fairly rapid fixation of alleles at all loci. Moreover, because of the very small N_e, the appearance of new alleles at loci affecting k is unlikely.

The possibility that in the E60 males the variation in k is due to changes in the frequency of alleles of a polymorphic locus (loci) affecting k is especially unlikely because in these males k was generally high through generation 33 (G_{33}) of L-60 (Pre-series III, Table 1), and then became variable in the sons of the G_{34} females (series III). Thus, it is necessary to explain the sudden appearance of genetic variability affecting k. Similarly, the large difference in the mean k between the E230 males of series IX-1 and series IX-2 is also unlikely to have been due to polymorphism at a locus (loci) affecting k, because at the time of series IX, L-230 was already in G_{37}, and because the variability in k of the E230 males in the three series preceding series IX (IV, V and VII) was fairly low. Thus, at present the cause of the observed variation in k in the E60 males, as well as in some of the other males such as those of series IX, remains unknown.

It is much more likely, however, that some of the variability in k in the E119 and E230 males is due to changes in the frequencies of alleles affecting k. First, because k was variable when the lines were in the lab for a shorter period (in males derived either from G_{20} or from G_{21}) and second, because, as expected from drift, the variability between males and between series tended to decrease with time (Figure 2). Moreover, the presence of polymorphism in these lines in G_{20} or G_{21} is apparently also consistent with the expected rate of fixation of neutral alleles under drift, and with the behavior of a mutation in one of the lines.

According to KIMURA and OHTA (1969) for a neutral allele (with frequency p) which will eventually be lost, the average number of generations until the loss is $t_0 = -4N_e p \ln p/(1-p)$ and the number of generations to fixation is $t_1 = -4N_e (1-p) \ln (1-p)/p$. In the isofemale lines the initial p of at least one of the alleles of a polymorphic locus is expected to have been one-third, because each line originated from one in-

seminated female and the male transmits only the *E* set. It is more difficult to estimate N_e, but because the lines were maintained by transferring parts of or whole ovisacs of five to ten females each generation, and because under laboratory conditions multiple inseminations are fairly common (U. NUR, unpublished data), it is likely that N_e is about 10. On the basis of these values the expected number of generations until a given allele is lost is about 22 and that the same allele will become fixed is about 33.

Evidence that under the present maintenance procedures a line can remain polymorphic for at least 25 generations is provided by the behavior of a body-color mutation (*pale*). It was discovered in G_{10} in one of the eight "synthetic" lines described in NUR and BRETT (1985) and the line was still polymorphic in G_{33}. Because the mutation is recessive, however, and was discovered because of the presence of *pale* females, it had to have been present in the line at least since G_8, and thus the line has remained polymorphic for at least 25 generations. Moreover, because in G_{33} the two alleles were at about equal frequencies, the line is expected to remain polymorphic for at least a few more generations. Thus, we conclude that it is likely that in G_{21} (when series I was initiated), L-119 and L-230 were still polymorphic for TRGs and that at least some of the observed variability in k within and between series was due to genetic differences between males of the same type. It is also likely that the discrepancy between the fairly high ks of the E119 and E230 males in series I and II, and the low k expected on the basis of the rapid elimination of the B in these lines, is due to changes in the frequency of the TRGs between the time that the B was being eliminated and series I, because at that time the lines were in the laboratory for only a relatively short period and were even more likely to have been polymorphic.

Do different populations of *P. affinis* carry different Bs? After the analysis of the first four series was completed (Table 1) it appeared that the B from L-251 (from Berkeley), which was used in series III and IV, was transmitted by the E60 and E230 males at a lower rate than the B from L-72 (from Davis) which was used in series I and II. However, on the basis of the results of series VII, in which (as in series I and II) the B came from L-72, but the ks of the E60 and E230 males were more similar to those of series III and IV than to those of series I and II, it now seems likely that the differences between the series are not due to the origin of the B. A comparison between the frequency of 2B daughters among the +B daughters of the E119 males with the two types of B also does not reveal any clear difference between the Bs; in the five series involved the frequencies of 2B daughters were fairly similar (about 5%), regardless of the origin of the B (Table 2).

These results are consistent with those of NUR (1966b) in which the effect of a B from Berkeley and a B from Rochester on the number of sperm cysts produced per male were compared. Both Bs were introduced into the same line from Berkeley, and in each experiment the comparison was between 0B and 1B sib males. It was found that both Bs reduced the number of sperm cysts produced per male, and by similar amounts: the B from Berkeley reduced the number of cysts by 2–8%, and the B from Rochester by 4–9%.

The number of loci affecting *k*: The best available information about the number of loci which may have a significant effect on k is provided by the frequency distribution of the bs and ks of the E(230 × 60) males (Figure 3). Because of the loss of the TRGs by L-119 it was not possible to obtain similar information from a cross between L-60 and L-119. Thus, we limit the discussion to the question of how many loci were involved in the almost complete suppression of k in the E230 males, and will try to answer it by estimating the number of loci that contributed significantly to the difference in k between the E60 and E230 males. In our analysis, we assume that in males, k is determined only by the alleles on the E set, and thus that males are functionally haploid. This assumption is based on the results of BROWN and NELSON-REES (1961) which indicate that in mealybugs the H set is genetically inactive, as well as on the present observation that the E230 males had a very low k, in spite of the fact that in these males the H set came from a line with a high k (L-60). We also assume that each of the loci affecting k has two alleles: a plus (+) allele permitting a high k and a minus (−) allele causing a reduction in k, and that at the time that L-60 and L-230 were crossed to produce series VI, the two lines were monomorphic at all the loci affecting k. The last assumption is reasonable, because at the time of crossing, L-60 was in G_{43} and L-230 was in G_{34}.

We will first consider the possibility that k was controlled by a single locus. An examination of the bs of the E60 and E230 males in the series that preceded series VI (series IV) and the one that followed it (series VII) (Figure 2) suggests that in both series the frequency distribution of the bs of each type is unimodal and indicates that the distributions of the bs of the two types of male overlap only slightly. The situation is similar when one compares the bs of the E60 and E230 males from series VIII and IX in which, as in series VI, the males received their B from their mother (Figure 3). Thus, if we assume that k is controlled by a single locus, the frequency distribution of the bs (and also of the ks) of the E(230 × 60) males should have been bimodal, with one peak at $b = 0$ and

another somewhere between $b = 0.6$ and $b = 0.9$. An examination of Figure 3 indicates, however, that although in the E(230 × 60) males the distribution of the *b*s is somewhat irregular, it clearly does not exhibit the strong bimodality expected from segregation at a single locus.

One can construct a number of multilocus models which can account for the present data, but the following two-locus model is fairly simple, and accounts for them quite well. The model assumes (1) that *k* is controlled by two unlinked loci, each with two alleles (+ and −), (2) that the alleles on the H set are genetically inactive, (3) that two (+) alleles of maternal origin are needed for a high *k*, (4) that the *k*s of the (+ +) males are similar to those of the E60 males either in series IV and VII or in series VIII and that the *k*s of the (− −) males are similar to those of the E230 males in either series IV, V and VII or in series IX, and (5) that the *k*s of the (+ −) and (− +) males are in the range of 0.11–0.8, and thus that all the males with $b < 0.11$ (the two lowest *b* classes in Figure 3) are (− −) and all the males with $k > 0.8$ (the two highest *b* classes) are (+ +).

From Figure 3 it can be calculated that in the E60 males of series VIII the combined frequencies of the two lowest *b* classes (0 and 0.01–0.1) and of the two highest *b* classes (0.81–0.9 and 0.91–1.0) is 0.75 and in the E230 males of series IX it is 0.56. Thus the mean of the combined frequencies of the four most extreme *b* classes in the two parental lines is about 0.65. With segregation at two loci the expected frequency of these classes in the E(230 × 60) males should be half that value, or about 0.33, and the observed frequency of these four classes in the E(230 × 60) males is 0.35. Similarly, on the basis of the combined data of series IV, V, and VII the frequency of the four most extreme *b* classes in the E(230 × 60) males is expected to be 0.39. Thus, there is a fairly close agreement between the frequencies expected on the basis of our two-locus model and those observed. This agreement favors our model over a one-locus model with similar assumptions, because in such a model the frequency of the sum of the four most extreme *b* classes is expected to be either 0.65 or 0.77 (the same as the mean of the parental lines), or over a similar model with three loci, for which the expected frequency of these classes is either 0.17 or 0.19, depending on the data used.

The frequency distribution of the *b*s of the E230 males (Figures 2 and 4) is also consistent with the suggestion that the complete suppression of *k* in these males is due to the presence of (−) alleles at two loci. Thus, in series II the frequency distribution of the *b* has three peaks, at about 0–0.1, 0.21–0.4 and 0.61–0.8, and the reduction in the mean *b* in the first three series appears to be due mostly to changes in the

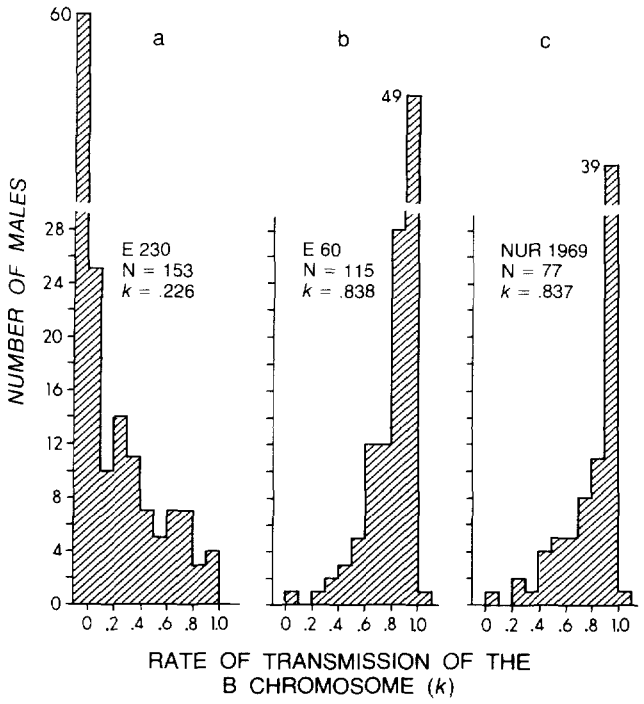

FIGURE 4.—The rate of transmission of the B chromosome (*k*) of some of the presumed 1B males from this study and from NUR (1969). a, The *k*s of the E60 males from all the series. Data from Figures 2 and 3. Ten E60 males with only 0B daughters were excluded (see text). b, The *k*s of the E230 males from all the series. Twenty-five males with only 0B daughters were excluded, because on the basis of the cytology of the males' sisters they apparently did not have a B. c, The *k*s of the presumed 1B males from NUR (1969). Data from 46 0B females whose analyzed daughters did not have a B were excluded, because it is assumed that all, or almost all, of these females were inseminated by 0B males (see DISCUSSION).

relative frequencies of males with these distinct levels of *b* (Figure 2). These observations suggest that in the first three series L-230 was polymorphic at two loci whose (−) alleles have about equal effect on *k* and that the three peaks represent the modal *k*s of the (− −) (+ − or − +) and (+ +) males.

The existence of at least one additional locus with a strong effect on *k* is suggested by the observation that in the E119 males the reduction in *k* was at least partially due to a premeiotic loss of the B, while in the E230 males the reduction in *k* was due to the failure of the B to become negatively heteropycnotic during the first meiotic division. This possibility could not be tested, however, because the TRGs of L-119 were apparently lost after series II.

Is the B of *P. affinis* "parasitic"? The results of the present study have significant bearing on the conclusion of NUR (1969) that in at least one population of *P. affinis*, the B is "parasitic," because it has little or no effect on female survival, but reduces the fitness of all the males carrying it. This conclusion is based on a comparison between the frequencies of the various karyotypes among the adult males and females in

a population in Oakland, California, and those expected among the zygotes. The frequencies of the various karyotypes among the females were estimated directly from the cytology of 224 "wild" females, and those among the zygotes were calculated from the known *k*s in females and from 4732 offspring of "wild" males and 0B females. The frequencies of the various karyotypes among the males, however, were estimated only indirectly from the analysis of about 20 daughters of each of 231 0B females which were inseminated by "wild" males, and were based on the assumption that a +B male will usually transmit all its Bs to at least one daughter in 20. The conclusion reached about the higher fitness of the 0B males relative to the +B males would be invalid, however, if a substantial number of the 1B males had a very low *k*, and were misclassified as 0B males. Thus, the discovery of genotypes which can suppress *k* below 0.1 (NUR and BRETT 1985) led us to question the validity of the conclusions of the 1969 study. However, the additional information obtained in the present study about these genotypes strongly suggests that in the Oakland population analyzed in the 1969 study, the frequency of genotypes capable of suppressing *k* sufficiently to cause some of the 1B males to be misclassified as 0B males was much too low to affect the conclusion that the B was "parasitic."

The conclusion that very few (if any) of the 1B males from the 1969 study were misclassified is based on a comparison of the frequency distributions of the *k*s of the E60 and E230 males with that of the 77 +B crosses of the 1969 study which were assumed to have been sired by 1B males (Figure 4). This comparison indicates that the frequency distribution of the *k*s of the 77 males of the 1969 study resembles fairly closely that of the +B crosses of the E60 males in both the mean *k* (0.837 *vs.* 0.838) and its shape. Moreover, because there is no evidence either that L-60 carries TRGs or that any of the E60 males tested failed to transmit a B to at least one analyzed daughter, it seems reasonable to conclude that in the Oakland population the frequency of the TRGs was low and that in the 1969 study the frequency of 1B males which were misclassified as 0B males was very low.

A similar conclusion may be reached from the observation that in the E230 males the presence of a large number of 1B males which failed to transmit a B to any of their 20 daughters (about 60) is associated with the presence of a substantial number of 1B males (25) with *k*s in the range of 0.01 to 0.1. The presence of E230 males with low *k*s is not unexpected, because with $k = 0.05$, 64% of the males would still be expected to transmit a B to at least one daughter in 20 ($1-0.95^{20} = 0.64$), and with $k = 0.02$, this value is still 33%. In the 1969 study, however, only one male had a *k* in this range. Thus, the number of 1B males of the 1969 study which may have been misclassified as 0B males is likely to have been small (0–4).

Another reason for concluding that the frequency of 1B males which were misclassified is low follows from the conclusions reached earlier that in the Oakland population the frequency of the TRGs was low and that the reduction in *k* below about 0.1 requires the presence of (−) alleles at two loci. Thus, the frequency of (− −) males that could have been misclassified is expected to be very low. This conclusion is significant because it can be shown that the ranking of the fitnesses of the 0B and 1B males would not be reversed unless at least 12 1B males have been misclassified as 0B males, and that in order to conclude that the mean fitness of all the +B males was greater than that of the 0B males, at least 22 of the 1B males had to have been misclassified as 0B males. We conclude, therefore, that the results of the present study are consistent with the conclusion of NUR (1969) that in the Oakland population studied the B is "parasitic." Moreover, this conclusion is also consistent with experimental results which demonstrated that 1B males develop more slowly and produce fewer sperm than their 0B sibs (NUR 1966b) and that +B females develop more slowly than 0B females (U. NUR and B. L. H. BRETT unpublished data).

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