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# Population Genetics in the American Tropics XVI. Data on Partial Dominance of Recessives in *Drosophila willistoni*

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**Summary.** Through a series of genetic load studies made on 1) samples of *Drosophila willistoni* from two sites in Mesitas, Colombia, it was found that the relative contributions to the total, subvital and lethal loads reflect lethal equivalences (B/A) ratios which support more the balancing theory of population structure than the neutralist theory. Moreover, measurements of population size have revealed the existance of very small demes in local populations. Under such conditions we have calculated extremely small lethal equivalence ratios in demes where probably a great deal of consanguinity takes place. We are aware that under these conditions B/A ratios cannot be very good monitors of random load measurements and, therefore, suggest a change in the mathematical formulation that take into consideration the existance of small populations.

Furthermore, it appears plausible that the degree of penetrance in the heterozygous condition changes as the population structure changes. We speculate that natural populations may have unknown selective mechanisms capable of guiding unknown dominance modifiers according to the intensity of selection.

**Key words:** Mutation – Normalizing-selection – Lethal-equivalence – Genetic-load – Neutral-theory

## Introduction

The classical theory of modern evolution with respect to the genetic load controversy makes the following assumptions: (1) that populations are infinite in size, (2) that environments are constant and uniform, (3) that most mutations are irremediably deleterious, (4) that all recessives have considerable partial dominance in heterozygous conditions, and (5) that populations are largely homogeneous.

However, demes are increasingly found to be the inmediate units of selection. The works of Dobzhansky and Wright (1943), Wallace (1968a, and others) and Hoenigsberg et al. (1977a, b, c, d) attest to the error of pooling several samples of different populations as though they resulted from one panmictic population. Indeed Wallace (1966a), Hoenigsberg (1968 and unpublished results) and Hoenigsberg et al. (1973) find that in their natural habitats (Bogotá and Mesitas) 80% of the flies (*Drosophila willistoni* for Hoenigsberg's unpublished work) from one spot do not move beyond 10 meters and that doubtless considerable inbreeding takes place (Hoenigsberg 1968). Moreover, Band (1972) and Hoenigsberg et al. (1965, 1973, 1977a, and this paper) find that there is neither temporal nor spatial uniformity for the genetic load. Climates (tropical in Hoenigsberg's work, temperate in Band's) which by most mathematical models are regarded as static have proven to be variable, and therefore instrumental to changing selective values. Haldane and Jayakar (1963a, b) found that under certain conditions stable polymorphisms are possible if selection favors first one and then another allele in successive generations in a cyclic fashion.

The most logical argument in favor of mutation pressure (rather that normalizing selection) as the most important agent responsible for maintaining deleterious recessives in natural populations is that the B/A ratio (i.e., the inbreeding load/random load) is generally large (around 15–20). However, even this type of analysis is not without its critics (Wallace 1968a, p. 274; Lewontin 1974, p. 78).

Our present work, carried out within natural populations of *Drosophila willistoni* in carefully selected sites no more than 10 meters in diameter reveal extremely low lethal equivalent ratios. Moreover, data from the same populations yield low (sometimes negative) values for partial dominance of deleterious recessive alleles affecting viability. We also discuss reasons why others (for example, Stern et al. 1952; Crow and Temin 1964) have found much larger partial dominance for deleterious recessives.

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## Materials and Methods

Samples of third chromosomes from two neighboring populations (10 kms apart) of *Drosophila willistoni* from Mesitas were studied. In all, eleven samples taken at different times from the two neighboring sites were analyzed for genetic load. While 1–4 of site 2 (Table 1) represent collections taken at different times in an artificial orchard, the others (1–7) are samples from several different generations of demes that originated only from site 1. Some of the demes have been discussed in a previous study (Hoenigsberg et al. 1977a) on the load characteristic of a marginal population of *Drosophila willistoni.* The techniques used by Dobzhansky et al. (1942), Dobzhansky and Spassky (1944), Pavan et al. (1951), and Hoenigsberg et al. (1977a) are similar to those used in this work. For the analysis of the third chromosome, sons of wild females were crossed singly to females homozygous for pink (p). Since all samples were made up of only a few individuals because population size was very small, we had to allow that individuals caught in nature reproduce in the laboratory. The resulting male progeny was then used to cross singly to marked pink flies. Single males from the $F_1$ were outcrossed to several heterozygous females for the third dominant chromosome Delta ($\Delta$), the recessive pink (p) and inversion N°133. This inversion induced by X-rays (Spassky and Dobzhansky 1950) virtually eliminates recombination within that chromosome. From the resulting $F_2$, ten males and females showing Delta but not pink (i.e., $\Delta$ p. Inv. 133/+) were selected as parents for the $F_3$ generation. These individuals were allowed to oviposit for two days following each of two successive transfers; three replicas of each cross were thus produced. The identical procedure was used for both experimental cultures (homozygotes) and their controls (heterozygotes). Experimental cultures are those in which the parental $F_2$ flies were from the same culture; the controls were those in which the parental ($F_2$) males and females came from different $F_1$ cultures. The mating of males to unrelated females was carried out following a randomization process. The expected $F_3$ progeny should consist of $\frac{1}{3}$ +/+ wild type individuals and $\frac{2}{3}$ $\Delta$ p. Inv. 133/+ because homozygous $\Delta/\Delta$ flies die during the egg stage. Of course, if wild chromosomes contain genes which decrease the viability of the homozygotes then a proportional decrease in the frequency of +/+ flies reflects the degree of depressed viability. The cross between unrelated individuals $\Delta$/+ from the $F_2$ generation produces the same genotypes but with nonidentical wild type chromosome +i/+j. The cultures were incubated at 24 °C and three counts were made, one every third day after the first imagoes appeared. Our statistical analysis for computing concealed variation follows that of Wallace and Maden (1953) and Hoenigsberg et al (1977a). The estimated and the calculated values for the elimination of lethal genes through allelism, homozygous and heterozygous genotypes were made utilizing for allelism (i) and mutation rate (u) our own calculations, 0.05 and 0.003 respectively. These values were found for other samples collected in Mesitas site 1. Since $q^2$ represents the homozygote frequency, elimination through allelism is $iq^2$. Then, since pq stands for heterozygote frequency, hpq represents the fraction of heterozygotes eliminated by virtue of the penetrance (h = deleterious penetrance of recessives in that combination); and $2hq^2$ is the homozygous elimination. Therefore:

$$u - iq^2 = hpq + 2\,hq^2$$

which says that mutation rate minus allelic elimination is equal to the total genotypic elimination (Table 6). From above equation we can calculate h (= dominance).

Our mating scheme permits us to identify each chromosome so that every surviving class can be studied for its effect in both homozygous and heterozygous conditions. Thus, heterozygous combinations of lethals and semilethals (chromosomes with drastic effects on viability) with normal chromosomes can be separated from drastic/drastic and normal/normal combinations. The experimental method eases the calculation of covariance and lethal equivalents. The classification of each chromosome according to its viability performance is based on the frequencies of wild type flies in homozygous cultures. Since relative adaptive values are necessary to compute average contributions to the total, subvital, and lethal loads, we classified chromosomes as "quasinormal" (N) and drastic (D). On the other hand, wild type offspring from control crosses (heterozygotes) carry combinations of chromosomes chosen at random. These crosses (control cultures) enable us to identify each heterozygote combination N/N (without lethals), N/D (with 1 lethal) and D/D (with 2 lethals). Occasionally, such D/D combinations include two drastics that produce lethal or semilethal heterozygotes; these are instances of allelism. These cultures have been excluded from calculations.

All flies were grown in a standard banana-agar medium to which dry yeast was added. One or two months before sampling was undertaken, the mark and release method was used to estimate the population size (Table 7) at the collection sites. We are aware that the method for estimating population size is not precise. However, previous estimates with the same method show an average error among repeated estimates of about 15% in census data smaller than 100 individuals and of about 10% for data over 200 individuals. There is a possible error in our mark-released method (Andrewartha 1971): the same investigator did the experiments without having anyone else check for possible systematic errors. Moreover, by releasing marked flies at a single point, overcrowding might have increased migration; such errors could lead to an underestimate of population size. To what extent these errors affect our estimates cannot be estimated. Another error may occur in release – recapture experiments: the calculations assume that marked and unmarked individuals have the same chance of being recaptured.

Obviously, there are difficulties in these studies, but our experiments on population size are just beginning; we hope to identify possible errors and to improve our techniques as we gain experience. Careful attention was paid to the number of generations (indicated in Table 7) that elapsed between samples. However, since there were generational differences during the study the mean generation time (15 days) was considered adequate to represent each time unit. The botanical and the climatological characteristics for each sample were recorded; nevertheless Table 7 presents only a simple summary of the most relevant conditions.

## Results

For a description of the seven samples (site 1), the ecological setting, hours of collection, and the prevalent nearby fruiting trees, see Table 2 of Hoenigsberg et al. (1977c). Each population number constitutes a temporal sample. The difference in the number (N) of chromosomes analysed between Table 2 of Hoenigsberg et al. (1977c) and our present Table 1 is the result of having eliminated some chromosomes for which the $F_3$ culture sizes are now considered to be inadequate, and having added others that were inadvertenly omitted from the

**Table 1.** Number of homozygous chromosomes in Mesitas. Lethals, semilethals and quasinormals are shown according to the percent viability of the homozygous chromosomes sampled; lethals are those from 0 – 10%; wild type semilethals (from 10 . . . 50%) of the heterozygous viability and quasinormals (from 50% . . . up) follow as indicated

| | | | ←Lethals→ | ←——— Semilethals ———→ | | | | | ←——— Quasinormals ———→ | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Sites | Mesitas | N | 0% | 10 | 20 | 30 | 40 | 50 | 60 | 70 | 80 | 90 | 100 | 110 | 120 | 130+ | |
| 1. | 1. | 585 | 28 | 10 | 18 | 14 | 16 | 15 | 32 | 45 | 79 | 94 | 79 | 69 | 38 | 21 | 27 |
| | 2. | 439 | 28 | 20 | 17 | 14 | 18 | 15 | 21 | 36 | 46 | 73 | 72 | 42 | 14 | 16 | 7 |
| | 3. | 496 | 53 | 13 | 12 | 14 | 17 | 19 | 32 | 53 | 59 | 82 | 62 | 42 | 20 | 10 | 8 |
| | 4. | 361 | 28 | 5 | 12 | 12 | 21 | 16 | 18 | 29 | 44 | 56 | 56 | 33 | 13 | 8 | 10 |
| | 5. | 404 | 1 | 0 | 2 | 2 | 4 | 3 | 8 | 15 | 52 | 64 | 77 | 63 | 50 | 34 | 29 |
| | 6. | 158 | 1 | 0 | 3 | 0 | 6 | 1 | 0 | 3 | 13 | 25 | 24 | 35 | 20 | 10 | 17 |
| | 7. | 299 | 19 | 9 | 4 | 3 | 3 | 7 | 13 | 20 | 33 | 42 | 63 | 36 | 23 | 11 | 13 |
| 2. | 1. | 438 | 29 | 9 | 17 | 22 | 21 | 12 | 25 | 42 | 50 | 50 | 50 | 49 | 22 | 20 | 20 |
| | 2. | 241 | 27 | 7 | 12 | 20 | 12 | 10 | 21 | 29 | 32 | 23 | 20 | 14 | 9 | 2 | 3 |
| | 3. | 171 | 5 | 3 | 4 | 4 | 1 | 3 | 3 | 17 | 28 | 39 | 32 | 15 | 9 | 6 | 2 |
| | 4. | 237 | 10 | 5 | 7 | 4 | 7 | 11 | 23 | 40 | 41 | 27 | 22 | 20 | 7 | 7 | 6 |
| | Σ | 3829 | 229 | 81 | 108 | 109 | 126 | 112 | 196 | 329 | 447 | 575 | 557 | 418 | 225 | 145 | 142 |
| | X̄ | 348 | 21 | 7 | 10 | 10 | 11 | 10 | 18 | 30 | 43 | 53 | 51 | 38 | 20 | 13 | 13 |

earlier report. Moreover, the following types of cultures have been removed from the control data: all cases in which the number of heterozygous wild types flies is less than half the expected number (such combinations may or may not carry one lethal or semilethal chromosome). The present manipulation alters the results in Table 2 of Hoenigsberg et al. (1977c). Table 1 presents the distribution of chromosomes in their homozygous condition; N refers to the number of chromosomes tested. The remainder of the Table 1 indicates the number of chromosomes for each viability class. Complete lethals (0.0% wild type) appear in column three. The number of chromosomes with 0–10% of the wild type (= the lethal class) relative to the average viability of the random heterozygote combination for that sample constitute the chromosome listed next to the 0% column: for instance Mesitas 1 has 10 such chromosomes. For the same population, there were 18 chromosomes that belong to the 10–20% viability class, and so on. Chromosomes producing fewer than 50% of the average frequency of the wild class of the controls are called semilethals. Table 2 presents the proportion of lethals, semilethals and total detrimentals found in each sample. Lethals are defined as those chromosomes which in the homozygous condition produce from 0–10% of the average viability of the heterozygotes ("normals"). Semilethals

**Table 2.** Percent lethals and semilethals in the 3$^d$ chromosomes of *Drosophila willistoni* from Mesitas, Colombia. N is number of chromosomes analysed; in parenthesis, the number of lethal and semilethals found

| Sites | Mesitas | N | % Lethals | % Semilethals | % Detrimentals |
|---|---|---|---|---|---|
| 1. | 1. | 585 | 6.50 (38) | 10.94 (64) | 17.44 (102) |
| | 2. | 439 | 10.93 (48) | 14.58 (64) | 25.51 (112)** |
| | 3. | 496 | 13.31 (66) | 12.70 (63) | 26.01 (129)** |
| | 4. | 361 | 9.14 (33) | 16.90 (61) | 26.04 ( 94)** |
| | 5. | 404 | 0.25 ( 1) | 2.72 (11) | 2.97 ( 12)*** |
| | 6. | 158 | 0.63 ( 1) | 6.33 (10) | 6.96 ( 11)** |
| | 7. | 299 | 9.36 (28) | 5.69 (17) | 15.05 ( 45) |
| 2. | 1. | 438 | 8.90 (39) | 16.67 (73) | 25.57 (112)** |
| | 2. | 241 | 13.69 (33) | 19.50 (47) | 33.19 ( 80)*** |
| | 3. | 171 | 4.68 ( 8) | 7.02 (12) | 11.70 ( 20)* |
| | 4. | 237 | 6.33 (15) | 11.39 (27) | 17.72 ( 42) |
| | | 3829 | 8.09 (310) | 11.73 (449) | 19.82 (759) |

* $p>0.05$; ** $p>0.01$; *** $p>0.001$

produce from 10–50% of normal viability for that population. Chromosomes producing 50% or more wild type are considered "quasinormal". The frequencies of "drastic" chromosomes (lethal and semilethal) in the various samples differ significanctly ($X^2 = 135.22$; 10 d. f., $p < 0.001$) in a homogeneity $X^2$ test where equal proportions weighted by their number of chromosomes are expected. This phenomenon was already discovered for *Drosophila melanogaster* (Hoenigsberg and de Navas 1965), for *Drosophila pseudoobscura* (Hoenigsberg et al. unpublished results) and for *Drosophila willistoni's* peripheral populations (Hoenigsberg et al. 1977 c). In Table 3 the total, environmental, binomial or sampling, and genetic variances are shown. The procedure has been described by Wallace and Madden (1953).

The frequency distribution of homozygous and heterozygous chromosomes permits the calculation of lethal-equivalents (Crow and Temin 1964). The average frequency of wild-type in all cultures is the frequency in the non-lethal class. The Poisson probability of the frequency of lethal loci is the negative natural logarithm of the percentage of non-lethal chromosomes. This value represents the number of non-lethal loci with some detrimental effect distributed along the chromo-

**Table 3.** Estimates of total, environmental, sampling, and genetic variances of natural populations of *Drosophila willistoni's* third chromosomes $St^2$, $Se^2$, $Ss^2$ and $Sg^2$ are to be multiplied by $10^{-6}$

| Sites | Populations | Homozygous | | | |
|---|---|---|---|---|---|
| | | $St^2$ | $Se^2$ | $Ss^2$ | $Sg^2$ |
| 1. | 1. | 17,690.0 | 270.0 | 0.0105 | 117,419.98 |
| | 2. | 18,030.0 | 55.0 | 0.0308 | 17,974.97 |
| | 3. | 20,710.0 | 261.0 | 0.0455 | 20,448.95 |
| | 4. | 18,100.0 | 70.0 | 0.0884 | 18,029.91 |
| | 5. | 9,430.0 | 60.0 | 0.0440 | 9,369.96 |
| | 6. | 9,420.0 | 447.0 | 0.4614 | 8,972.54 |
| | 7. | 15,730.0 | 15.0 | 0.1177 | 15,714.88 |
| Average | | 17,507.52 | 234.165 | 0.0438 | 17,273.31 |
| 2. | 1. | 21,400.0 | 299.0 | 0.0672 | 21,100.93 |
| | 2. | 26,120.0 | 44.0 | 0.1833 | 26,075.82 |
| | 3. | 17,010.0 | 212.0 | 0.3701 | 16,797.63 |
| | 4. | 18,460.0 | 119.0 | 0.1585 | 18,340.84 |
| Average | | 21,540.55 | 111.15 | 0.1489 | 21,429.25 |
| Totals | $\bar{X}$ | 18,655.84 | 180.47 | 0.0623 | 18,475.37 |
| Sites | Populations | Heterozygous | | | |
| | | $St^2$ | $Se^2$ | $Ss^2$ | $Sg^2$ |
| 1. | 1. | 4,830.0 | 105.0 | 0.0078 | 4,120.0 |
| | 2. | 4,890.0 | 171.0 | 0.0247 | 4,720.0 |
| | 3. | 7,010.0 | 488.0 | 0.0466 | 6,520.0 |
| | 4. | 8,740.0 | 685.0 | 0.0775 | 8,050.0 |
| | 5. | 6,720.0 | 80.0 | 0.0494 | 6,710.0 |
| | 6. | 7,400.0 | 91.0 | 1.3993 | 7,310.0 |
| | 7. | 6,640.0 | 308.0 | 0.1845 | 6,630.0 |
| Average | | 6,081.95 | 69.97 | 0.0416 | 6,011.94 |
| 2. | 1. | 7,590.0 | 296.0 | 0.0771 | 7,290.0 |
| | 2. | 9,270.0 | 705.0 | 0.2061 | 8,560.0 |
| | 3. | 6,230.0 | 310.0 | 0.2280 | 6,190.0 |
| | 4. | 2,360.0 | 587.0 | 0.1407 | 1,770.0 |
| Average | | 8,766.16 | 184.95 | 0.1423 | 8,581.07 |
| Totals | $\bar{X}$ | 6,886.91 | 98.05 | 0.0595 | 6,788.81 |

**Table 4.** Lethal-equivalents in inbred and random heterozygotes in *Drosophila willistoni* from Colombia. N stands for the number of homozygous chromosomes, homo (B) is the inbred load (B), leth-eq. (B) is the inbred lethal equivalence, hetero (A) is the random load, leth-eq. (A) is the corresponding lethal equivalence of random outbred crosses, and finally, B/A stands for the ratio of the inbreeding load over the random load in lethal equivalence

| Sites | Mesitas | N | Homo (B) | Leth-eq. (B) | Hetero (A) | Leth-eq. (A) | B/A |
|---|---|---|---|---|---|---|---|
| 1. | 1. | 585 | 0.8274 | 0.1895 | 1.1518 | 0.1413 | 1.341 |
| | 2. | 439 | 0.7449 | 0.2945 | 1.1482 | 0.1382 | 2.131 |
| | 3. | 496 | 0.7420 | 0.2984 | 1.1815 | 0.1668 | 1.789 |
| | 4. | 361 | 0.7396 | 0.3016 | 1.2133 | 0.1933 | 1.560 |
| | 5. | 404 | 0.9734 | 0.0270 | 1.1338 | 0.1256 | 0.215 |
| | 6. | 158 | 0.9304 | 0.0721 | 1.0738 | 0.0712 | 1.012 |
| | 7. | 299 | 0.8495 | 0.1631 | 1.1212 | 0.1144 | 1.425 |
| 2. | 1. | 438 | 0.7489 | 0.2891 | 1.1755 | 0.1617 | 1.787 |
| | 2. | 241 | 0.6681 | 0.4033 | 1.2457 | 0.2197 | 1.835 |
| | 3. | 171 | 0.8830 | 0.1244 | 1.1920 | 0.1756 | 0.708 |
| | 4. | 237 | 0.8228 | 0.1950 | 1.3171 | 0.2754 | 0.708 |
| | $\Sigma$ | 3829 | | | | | |
| | $\bar{X}$ | 348 | 0.8028 | 0.2196 | 1.1776 | 0.1635 | 1.343 |

somes. Therefore, lethal-equivalence represents the average degree of subvitality. The deleterious effects of nonlethals are classified as fractions of the lethal genes; Table 4 presents, therefore, the average degree of subvitality in terms of lethal-equivalents. The percentage of subvitals registered in Table 4 tells us how many of those loci there are, hence the last column of Table 6. Lethal equivalents over the proportion of subvital loci per chromosome give the average degree of subvitality per subvital chromosome (Wallace 1968). Evidently, our results do not appear anywhere near the expected amount of lethal equivalence of Morton et al. (1956) and Morton (1960 and others). Their results, deriving from the ratio of the inbreeding over the random breeding load, were taken as evidence that genetic loads are primarily maintained by recurrent mutations. In fact, lethal equivalents, of the order of aproximately 8 to 24 cannot result from heterotic balancing selection. Nevertheless, our results do not support the mutation pressure theory.

From the average viability of the wild type class in all homozygous chromosomes ("All homo") and from the quasinormal combinations ("Norm homo"), we calculate the adaptive values for those categories. These relative estimates permitted us to calculate from the total contributions the subvital and lethal loads. Table 5

**Table 5.** Average viability of the wild type class of homozygotes relative to the average viability of the wild type class of heterozygotes. Relative, average contributions to the total, subvital and lethal loads are presented

| | | | | ← Load → | | |
|---|---|---|---|---|---|---|
| Sites | Populations | "All homo" | "Norm homo" | Total | Subvital | Lethals |
| 1. | 1. | 0.8184 | 0.9134 | 0.2004 | 0.0906 | 0.1098 |
| | 2. | 0.7367 | 0.8884 | 0.3056 | 0.1184 | 0.1872 |
| | 3. | 0.7529 | 0.8773 | 0.2839 | 0.1310 | 0.1529 |
| | 4. | 0.7438 | 0.8885 | 0.2960 | 0.1183 | 0.1777 |
| | 5. | 1.0067 | 1.0212 | 0.0000 | 0.0000 | 0.0000 |
| | 6. | 0.9818 | 1.0122 | 0.0184 | 0.0000 | 0.0184 |
| | 7. | 0.8435 | 0.9208 | 0.1702 | 0.0826 | 0.0876 |
| 2. | 1. | 0.7836 | 0.9398 | 0.2439 | 0.0621 | 0.1818 |
| | 2. | 0.6768 | 0.8566 | 0.3904 | 0.1544 | 0.2360 |
| | 3. | 0.8482 | 0.8921 | 0.1647 | 0.1142 | 0.0505 |
| | 4. | 0.7662 | 0.8493 | 0.2664 | 0.1634 | 0.1030 |

**Table 6.** Estimated and calculated values for the elimination of lethal genes through allelism, homozygous and heterozygous genotypes. The rate of allelism (i) calculated for *Drosophila willistoni* and used in these studies is 0.05 (39 out of 800 trials). Mutation rate is said to be 0.003

| Sites | Mesitas | $iq^2$ | PQ | $Q^2$ | $hpq + 2\,hq_2$ | h | % dominance |
|---|---|---|---|---|---|---|---|
| 1. | 1. | 0.001489 | 0.1428 | 0.0298 | 0.2024 h | 0.007411 | 0.7 |
| | 2. | 0.003254 | 0.1900 | 0.0651 | 0.3202 h | −0.000793 | −0.07 |
| | 3. | 0.003331 | 0.1915 | 0.0666 | 0.3247 h | −0.001019 | −0.10 |
| | 4. | 0.003390 | 0.1926 | 0.0678 | 0.3282 h | −0.001188 | −0.1 |
| | 5. | 0.000044 | 0.0288 | 0.0009 | 0.0306 h | 0.096601 | 9.7 |
| | 6. | 0.001132 | 0.1278 | 0.0226 | 0.1730 h | 0.010797 | 1.1 |
| | 7. | 0.001945 | 0.1583 | 0.0389 | 0.2361 h | 0.004468 | 0.4 |
| 2. | 1. | 0.003155 | 0.1881 | 0.0631 | 0.3143 h | −0.000477 | −0.05 |
| | 2. | 0.005508 | 0.2217 | 0.1102 | 0.4421 h | −0.005655 | −0.5 |
| | 3. | 0.000684 | 0.1033 | 0.0137 | 0.1307 h | 0.017719 | 1.8 |
| | 4. | 0.001570 | 0.1458 | 0.0314 | 0.2086 h | 0.006855 | 0.7 |
| Average | | 0.001945 | 0.1583 | 0.0389 | 0.2361 h | 0.004468 | 0.4 |

summarizes those results. The data are expressed relative to the mean frequency of wild type flies of the heterozygotes. Then, using natural logarithms, one can calculate the homozygous load (Crow 1968a) due to all deleterious loci and to detrimental loci alone. In other words, on one hand the homozygous load, attributable to all loci, which decreases the mean fitness of the population by virtue of its deleteriousness, is the total load; and on the other hand, the subvital load includes only the effects of the detrimental loci occuring on the chromosomes. There are, here, some amazing results that indicate the presence of an ecological component in populational-loads. Moreover, it appears that the local circumstances affect loads. The lethal load in Mesitas decreased to ¼ the value it had 8 generations before in Mesitas 5. It should be remembered that Mesitas 1, 2, 3, and 4 of site 2 are temporally different samples from the same population a few kilometers from which Mesitas 1, 2, 3, 4, 5, 6, and 7 of site 1 came. These last ones are also just temporal samples taken at intervals a few generations from one another (Table 7). The load pattern presented in Table 5 follows very closely the lethal-equivalence pattern described in Table 4. The drop in the lethal load found in "guayabas", 5 (site 1), and the slow recovery of "mangoes load" in the next 4 generations, 6 (site 1), and still more when the mango harvest began with 7 (site 1), is also clear in the lethal equivalent units. The drop in 3 (site 2) as compared to 2 also shows itself in lethal equivalence.

Table 6 presents the pertinent data that indicates the rate of elimination that lethal alleles may have through their heterozygous and homozygous effect according to mutation theory. The sum of the homozygous and of the heterozygous eliminations give values greater than the 0.005, a value usually regarded as the average mutation rate for second chromosome lethals of *Drosophila melanogaster*. Even if *Drosophila willistoni's* 0.003, considered by us as a reasonable mutation rate for III[rd] lethals (unpublished results), was an underestimate, we should still end up, in this species, with a very large fraction of loci that retain detrimental alleles (by virtue heterosis?). Our datum for allelism showed in 1966 and 1974 (unpublished data of *Drosophila willistoni)* 39 alleles out of 800 combinations. On the basis of this rate of allelism, elimination was calculated for each population. With the estimate on the average heterozygous effect we can estimate the degree of partial dominance, h. The results are startling: in most populations there is a very low penetrance in the heterozygous condition and in five populations there is clear indication of overdominance. Only Mesitas 5, characteristically a small

**Table 7.** Populations sizes and generations elapsed since first collections in Mesitas 1, 2, 3, 4, 5, 6, and 7 (site 1) and 1, 2, 3, 4, (site 2). The last column indicates prevalent fruiting trees in production, and the season (rainy or dry) under which collections were made

| Sites | Mesitas | Size | Generations (days) | Fruiting trees in production |
|---|---|---|---|---|
| 1. | 1. | 200 – 250 | 1 (0) | Mangoes – dry |
| | 2. | 60 – 80 | 10 (150) | Mangoes – wet |
| | 3. | 60 – 80 | 11 (165) | Mangoes – wet |
| | 4. | 60 – 80 | 27 (405) | Mangoes – wet |
| | 5. | 10 – 20 | 35 (525) | Guayabas – dry |
| | 6. | 20 – 40 | 39 (585) | Mangoes – wet |
| | 7. | 80 – 100 | 40 (600) | Mangoes – dry |
| 2. | 1. | 220 – 260 | 1 (0) | Guayabas – wet |
| | 2. | 220 – 260 | 4 (60) | Guayabas – wet |
| | 3. | 220 – 250 | 12 (180) | Guayabas – dry |
| | 4. | 210 – 250 | 18 (270) | Oranges – dry |

population which had previously indicated peculiar characteristics (such as no subvital chromosomes, nearly 96% normal chromosomes, practically no decrease in the population fitness because of 0.0% in the total, subvital and lethal loads, and a very low proportion of lethal and semilethal loci), now reveals the reason for this tremendous decrease in load: it had recessives in loci where practically a 10% pentrance was possible in heterozygous combinations.

## Discussion

Our results are: (1) populations of *Drosophila willistoni* are small and sometimes even very small (Table 7); (2) from Table 1, 2, 3, 4, and 5 it appears that populations are not genetically homogeneous since every sample presents different frequencies of lethal, semilethal, normal, subvital and supervital chromosomes within the same environment; (3) the environment is constantly changing as we have seen in Band's work and in Hoenigsberg et al. (1977a, 1977b) among others; (4) there is a small (about 0.0 in this work) decrease in the average fitness of heterozygotes for deleterious genes, but there are samples that yield all the range of dominance effects from complete recessives, to those that give a 10% decrease in the heterozygous fitness, to those that enhance viability. Therefore, there is a finite probability that some alleles can be retained even when lethal in homozygous conditions (see Table 6).

When wild chromosomes collected in Mesitas are made homozygous several important differences between the various temporal samples emerge. In site 1 the distribution of some viability classes present considerable differences. As far as detrimentals (Table 1 and 2) and $X^2$ homogeneity test which assumes equal proportions of drastic chromosomes as the expected number for each sample, shows significant departures from expectations. Mesitas 1 and 7 (site 1) and 3 and 4 (site 2) have the expected proportions of drastic bearing chromosomes while the other samples (2–6 in site 1; and 1 and 2 of site 2) do not. While Mesitas 1, 2, 3, and 4 (site 1), which represent the first 27 generations (see Table 7), have more or less about the same proportion of detrimental chromosomes, Mesitas 5, 6 and 7 show, in the next 15 generations, profound genetic changes that accompany drastic reductions in population size (see Table 7). On the other hand, such profound genetic changes do not appear in site 2; although a slight drop of the detrimental load does appear when the guayaba harvest ended and the oranges bloomed (Tables 1, 2, and 7). The drop in the load pattern shown (see also Table 5) in Mesitas 5 site 1 appears to be concomitant with a guayaba production in the orchard. This fact appears again but much less in site 2. We believe there is a direct correlation between the ecological variable (guayabas in production) and the dissappearance on the lethal load in site 1. There appears an interesting relationship that may be instrumental in effecting the kind of population shrinkage suggested in Table 7. This is the first time we have in a natural species such as *Drosophila willistoni* an example of an ecological incidence in the genetic structure of a population with such clear results. Although the guayaba harvest is the most likely candidate to produce both the population shrinkage and the drop in the genetic load, we feel that we cannot leave out the possible effect of the dry season in site 1, especially in virtue of the fact that when the same ecological phenomenon appeared in site 2, a similar drop in load, although not in size – and never as profound as in site 1 appeared (Tables 2, 5, and 7). Hoenigsberg et al. (1973) reported cyclical changes in load in *Drosophila melanogaster* from Caracolisito. We are tempted to speculate that this relatively small population of *Drosophila willistoni* took about 10 generations to stabilize its amount of detrimental load (see Table 5) but about two to five generations were sufficient to destabilize it through a rapid reduction in size. These results permit us to assume that the reduction in size and load was a "genetically healthy period" for the local population, since it was able to free itself of the relatively large load accumulated. We may, therefore, add to the already known forms of selection the presently discussed demographic one.

As far as the lethal equivalence ratios being a means to choose between the neutralist (=mutational in this paper) and the balance selection theory, we do not think it can unambiguously decide between one or the other theory. We think, however, that our results add more in favor of the balance selection theory than to the mutational theory. We are aware that in small populations, such as the ones reported here, there cannot be random assortment of chromosomes and, therefore, a true random load. Therefore, our B/A ratios are likely to be an underestimate of what they really could be if the population was large. That biological restriction is a serious drawback on the B/A ratio as it stands now. However, if a modification is introduced in its algebra to remove the restriction imposed by small sizes maybe it could have wider application than what it has now.

There is another widely held hypothesis – corollary that stems from genetic analysis: that if the chromosomal mutation rate $U^{L+SL} > iq^2$, then deleterious genes in the heterozygous conditions should be on the average, disadvantageous. For natural populations of *Drosophila willistoni,* (see Table 6), it is clear that in some samples (2, 3, 4 site 1) within the general Mesitas population, the mutations rate (0.003 in our case) is

smaller than the rate of elimination through homozygosis and allelism, but in other samples (1, 5, 6, 7, site 1 and 3, 4 site 2) it is larger.

In fact, when the mutation rate is smaller than the rate of elimination we get, as expected, no partial dominance on the part of deleterious recessives (column 6 of Table 6); on the other hand, when the rate of elimination is smaller than the mutation rate, we compute a slight penetrance of the ill effects of deleterious alleles. Moreover, we also get a very interesting result: there is only a small average degree of dominance, except for population 5 (site 1) and 3 (site 2). Several authors (Stern et al. 1952; Hiraizumi and Crow 1960; Crow and Temin 1964; Temin 1966; Watanabe 1969) have found for artificial populations of *Drosophila melanogaster,* which were examined for the viability effects in the heterozygous condition, an average of 2.5–3.0 percent in partial dominance. Temin (1966) did get in her data negative *h* values which meant that there were lethal genes that enhanced the viability of heterozygotes. However her overall average partial dominance turns out to be around 3 percent. We are not surprised at the large percentage of dominance found in *Drosophila melanogaster* because selection in that species cannot be strong even against newly arisen mutants. After all, *Drosophila melanogaster* is always found as artificially large populations in man-made environments. Therefore, whatever selective mechanism devised to marshall dominance modifiers in *Drosophila willistoni's* populations such as ours, in order to gain total or partial camouflage in the heterozygous condition, will pass undetected and unfelt in *Drosophila melanogaster's* large population.

Therefore, it should not be surprising to find in natural populations of *Drosophila willistoni* that there is not only a much smaller average degree of dominance (0.4% in our calculations) of heterozygotes for detrimentals, but also that there are populational circumstances that may be cyclical in producing enhancement of the viability of heterozygotes (heterosis). We hypothesize that the cyclical nature of such physiological effects of dominance results from the control exerted by modifiers which would be either strongly selected to serve biological requirements or not as strongly selected in larger and more relaxed populations. Therefore, natural populations may regularly pass from a period of high selection for dominance modifiers to one of relaxed selection for the same modifiers: the overt manifestations of such strong and relaxed selection are the changes in the degree of dominance (see Table 6).

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