acre was 11.6 with a range of 2.4 to 21.7. The agria cacti averaged 46.9 plants per acre with a range of 4.3 to 167.1. The latter figure represents a survey of about 4 acres in southern Baja California. These occasional thickets are produced vegetatively and are probably nurtured by the decaying stems themselves as they bend down and take root. We can tentatively state that agria cactus is generally more abundant than organ pipe even though it is not as large. The mean frequency of rotting plants among all plants scored was 13.5% of 4,100 agria cacti compared to 6.1% of 286 organ pipes. The biological difference is even greater, however, since many organ pipe rots do not contain D. mojavensis larvae while the majority of agria rots do.

The second method for measuring resource predictability was attempted by correlating the variation in concentration of yeasts, low molecular weight volatiles, and abiotic factors such as pH and temperature, with each other, and with the presence or absence of adult D. mojavensis (information kindly supplied by Don C. Vacek). Four significant correlations were found in agria rots while only one was found in organ pipe rots. Therefore, a D. mojavensis female can better assess an agria rot for both feeding and egg laying. For instance, there is a positive correlation between the concentration of ethanol, 2-propanol and other volatiles with the presence of adult D. mojavensis and a negative correlation between the concentration of these volatiles and the concentration of yeasts in the substrate. As the necrotic tissue advances, the yeasts increase in density, at least in part, at the expense of the volatiles and subsequently for the benefit of the maturing larvae. In the case of organ pipe necrotic tissue, the only significant correlation found was a negative one between pH and volatile concentration. No correlations were detected for temperature in either host plant.

Yeast species comparisons among host plants showed agria rots to be more predictable because they are less variable (data kindly offered by William T. Starmer). There were 4 effective species out of 12 species recovered in this host compared to 5.4 effective species out of 9 species recovered in organ pipe. On a per-plant basis, agria averages 1.8 species of yeast while the organ pipe average is 2.2. Furthermore, on a per-isolate basis, the yeasts from agria utilize an average of 8.5 compounds for growth (N=183) while those from organ pipe utilize an average of 10.1 compounds (N=83). Since the variance of these means is twice as high in organ pipe (31.0 vs 16.5) it means this plant is a more variable environment. The most notable evidence we have for this lies in the presence or absence of Pichia cactophila, probably the most important yeast for D. mojavensis since it is a good indicator of the presence or absence of larvae. This yeast was recovered in 73% of 105 agria rots sampled compared to 59% of 41 organ pipe rots. The difference is marginally significant.

In summary, central populations live in a more predictable environment than marginal populations because of the greater abundance of rots which have higher resolving power for the flies and which are more suitable (chemically stable?) for the growth of the nutritionally favored yeast. The first and last points may be a reflection of the asexual reproduction periodically exhibited in agria cactus.

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Heterochromatic duplications and the meiotic segregation of compound second autosomes during spermatogenesis of D. melanogaster.

It is well documented that in D. melanogaster females bearing compound second or third autosomes (and no additional structural heterologues or supernumerary chromosomes) that the complementary compound autosomes regularly disjoin from each other at meiosis (Holm 1969; Grell 1970; Holm and Chovnick 1975). That this segregation was not dependent on heterochromatic homology between the two compound autosomes was clear as it could be easily perturbed by the introduction of a Y chromosome, by the substitution of an attached X chromosome for the two free X chromosomes or by structural heterozygosity for the other autosome.

The present of meiotic pairing sites active during spermatogenesis and responsible for the meiotic segregation of the X and Y chromosomes (reviewed in Peacock and Miklos 1973) led to the speculation that analogous sites may exist in the autosomal heterochromatin. Nevertheless, Holm (1969; Holm and Chovnick 1975) in the analysis of compound third autosomes

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found that they behaved as nonhomologous chromosomes during spermatogenesis. As recent work has demonstrated that compound autosomes arise by a translocation mechanism (reviewed in Holm 1976; Hilliker 1978), any pair of compound autosomes share heterochromatic homology and, hence, one would expect some pairs of compound third autosomes to exhibit non-random segregation during male meiosis given the existence of pairing sites in the autosomal heterochromatin.

Holm (1969) in examining the meiotic segregation during spermatogenesis of compound second autosomes in several Drosophila strains found that none of the compound second autosomes examined segregated randomly, that is, there was a smaller proportion of sperm recovered nonsegregational for the compound autosomes than would be predicted from a model of random segregation. However, such departures from random segregation are minor and may be the consequence of elimination of one compound autosome from a proportion of diplo-2 spermatid nuclei (see Hardy 1975).

Sandler et al. (1968) found a C(2R) chromosome which apparently segregated in males with relatively high efficiency from any C(2L) chromosome with which it was tested in combination. The segregational properties of this chromosome, C(2R)cn, were further analyzed by Evans (1971) and Gethmann (1976). C(2R)cn carries a 2L duplication extending from the centomere to 38 or 39 (E.B. Lewis, unpubl., cited in Gethmann 1976) and is, therefore, duplicated for the 2L heterochromatin as well as 2L proximal euchromatin. Gethamnn (1976) suggests that the relatively efficient segregation of C(2R)cn from C(2L) is due to the 2L duplication and specifically to the duplication of pairing sites in the heterochromatin. Equally likely is that C(2R)cn is duplicated for a pairing site in the 2L proximal euchromatin. Yamamoto's (1979) cytological analysis of the meiotic segregation of C(2R)cn from a complement C(2L) during spermatogenesis led him to conclude that these chromosomes segregated randomly; however the data in support of this conclusion are not presented in sufficient detail to allow statistical evaluation.

In our laboratory a number of compound autosomes carrying heterochromatic duplications of a portion of the complementary arm have been constructed. For example, C(2L)SH1+ carries a duplication of 2R extending from the centromere to a point distal to the rl^+ locus within the 2R heterochromatin. Our previous analysis of the second chromosome proximal heterochromatin (Hilliker and Holm 1975; Hilliker 1976) has enabled us to set limits on the extent of the duplications associated with compound second autosomes. C(2L)SH1+ and C(2L)VH1 1t, which bear rl^+ duplications of 2R, are duplicated for at least half of the 2R heterochromatin and C(2R)VK2bw is duplicated for most of the 2L heterochromatin.

In order to examine the role, if any, of heterochromatin homology in meiotic pairing in males, the segregation of compound autosomes bearing duplications for heterochromatic material of the complementary compound autosomes was assayed. Segregation was assayed by crossing males of the selected compound-second autosome bearing strains to differentially marked compound-second autosome bearing females possessing a Y-chromosome. These B^SY; C(2L)P,b; C(2R)P,px females give, as first demonstrated by E.H. Grell (1970) for B^SY; C(2L); C(2R) bearing females in general, a high frequency of compound-second autosome nonsegregation. Female gametes nonsegregational for the compound second autosomes will result in a viable zygote only if fertilized by a sperm nonsegregational for the paternal compound-second autosomes. Thus a strain in which compound-second autosomes partially segregate in males when crossed to B^SY; C(2L)P,b; C(2R)P,px females will give a lower frequency of progeny completely matroclinous or patroclinous for the two compound-second autosomes than will a strain in which C(2L) and C(2R) segregate at random in the male.

Therefore, males of the C(2L)SH3+;C(2R)SH3+ strain, one in which nearly equal frequencies of C(2L); C(2R); diplo-2; and nullo-2 sperm are produced (Holm 1969), and of several other strains in which one or both compound autosomes bore heterochromatic duplications of the other arm were crossed singly in shell vials to B^SY;C(2L)P,b;C(2R)P,px virgin females. The results are presented in Table 1 and in summary form in Table 2.

Since there is no significant reduction in the frequency of nonsegregation in those crosses involving males carrying C(2L) and C(2R) chromosomes with extensive heterochromatic homology (Table 2), it is probable that heterochromatic homology per se is not a major factor in the meiotic segregation of autosomes in male D. melanogaster. Further, in conjunction with the compound third chromosome studies, as well as studies involving autosomes (and autosomal derivatives) heterozygous for heterochromatic deletions (Yamamoto 1979; Hilliker 1980), these data provide evidence for the absence of male meiotic pairing sites in the bulk of the autosomal heterochromatin.

The foregoing data have been abstracted from Hilliker (1975).

Table 1. Progeny of B^{Sy}; C(2L)P,b; C(2R)P,px females and various compound second autosome bearing males.

	Chromosome from mother								
	B^{Sy}		B^{Sy}		B^{Sy}		B^{Sy}		
Male genotype	C(2L)P,b; C(2R)P,px	C(2L)P,b; C(2R)P,px	C(2R)P,px	C(2R)P,px	C(2L)P,b	C(2L)P,b		0	Total
C(2L)SH3,+ C(2R)SH3,+	1	462	300	273	204	279	247	5	1771
C(2L)SH1,Dp(2R)r1^{+} C(2R)SH1,+	1	567	395	365	286	383	447	4	2448
C(2L)SH1,Dp(2R)r1^{+} C(2R)SH3,+	0	57	39	46	31	47	44	0	264
C(2L)SH3,+ C(2R)VK2,Dp(2L)1t^{+},bw	0	127	153	146	122	174	215	3	940
C(2L)VH1,Dp(2R)r1^{+},1t C(2R)VK2,Dp(2L)1t^{+},bw	1	122	127	102	80	146	163	1	742

Table 2. Frequency of progeny nonsegregational for compound-second autosomes from B^{Sy}; C(2L)P,b; C(2R)P,px virgin females crossed to various strains of compound-second autosome bearing males.

C(2L)	C(2R)	Percent nonsegregational progeny	N
SH3+	SH3+	40.4	1771
SH1+*	SH1+	41.6	2448
SH1*	SH3+	38.3	264
SH3+	VK2bw**	36.7	940
VH11t*	VK2bw**	38.7	742

* Bears a r1^{+} duplication of 2R.

** Bears a 1t^{+} duplication of 2L.

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Hilliker, A.J.*, A. Chovnick and S.H. Clark. Univ. of Connecticut, Storrs, Connecticut. The relative mutabilities of vital genes in D. melanogaster.

In recent years, several chromosomal regions of D. melanogaster have been subjected to intensive analysis. Most of these studies have focused primarily on the identification of genes capable of mutating to a lethal or semi-lethal state within a short, defined chromosome segment. However, the screens employed were generally competent to detect genes whose mutant alleles exhibit a recessive alteration in visible phenotype.

If all vital genes in a given region were equally mutable, then it would be possible to employ the Poisson distribution to determine the number of unmutated genes remaining after completion of a mutagenesis study. Cohen (1960) has discussed such methods for truncated Poisson distributions. It is, however, generally appreciated that vital genes within a given chromosomal interval are not of equal mutability, a point we herein substantiate and document for several regions of the Drosophila genome that have been extensively analyzed.

Since the Poisson distribution has only one parameter, the mean (m), which is equal to the variance, it is possible to determine if a given distribution of counts differs from a Poisson distribution by use of the variance ratio, s^2/m, where the sample mean square, s^2, estimates the variance of the distribution, and where m has infinite degrees of freedom (see discussion in Gilbert 1973). The variance ratio test of significance for deviation from the Poisson distribution is preferable to the chi square test in that it is more readily applicable to smaller data samples. Moreover, even in situations where one can apply the chi square test, the variance ratio test is associated with a greater number of degrees of freedom. Where the variance ratio is not significantly different from one, indicating possible agreement with a Poisson distribution, it is possible that the count distribution does differ from a Poisson distribution. Such cases would be better revealed by the chi square test which examines the entire distribution. However, this issue is irrelevant with respect to the present analysis since (1) the sample counts are too small to employ the chi square test, and (2) none of the 11 data sets examined are in good agreement with a Poisson distribution by the variance ratio test.

Table 1 summarizes a series of mutagenesis experiments that are competent to determine if vital genes within a given region are equally mutable by a given mutagen. Each entry represents a mutagenesis screen in which lethal mutations for all genes within a region are detected following treatment of sperm with the indicated mutagen. The data for the regions defined by $Df(3R)ry^{614}$ (Df(3R)87D2-4; 87D11-14) and $Df(3R)ry^{619}$ (Df(3R)87D7-9; 87E12-F1) were obtained in this laboratory as part of a larger analysis of the chromosome interval adjacent to the rosy locus (Hilliker et al. 1980).

The six regions included in the analysis of Table 1 involve both euchromatic and heterochromatic segments of the Drosophila genome. In no instance are the vital genes within a segment of equal mutability. Of the 11 experiments examined in Table 1, only the analysis of fourth chromosome spontaneous mutations failed to show a significant deviation from the Poisson expectation. The P value for this count distribution is greater than 0.05 but less than 0.10.

On the basis of these data, we are led to conclude that Drosophila vital genes within a defined chromosome segment are not of equal mutability. Hence, one cannot use the Poisson distribution to estimate the number of remaining unmutated vital genes within such an extensively analyzed segment.

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