Herreros, A.S. Universidad de Barcelona, Spain. Sensibility of the larvae of Drosophila to the electric field.

The larvas of several species of Drosophila are sensibles at the electric field. This may be easily shown by placing the larvae on the surface of agar-ethanol-acetic humidified on a watch glass 6 cm in diameter. In this medium two electrodes are inserted separated by 4 cm and a current of 9 V is passed through it.

After approximately 8 minutes, the number of larvae emigrating to each pole (or to within 1 cm of the pole) and the number of larvae that continue to move about the medium (neutral larvae) are counted.

The results I obtained are grouped from different time periods of the day and can be seen in the following table:

	number of larvae			
	at - pole	at + pole	neutral	total
D. subobscura	204	9	124	337
D. simulans	269	16	355	640
D. melanogaster	514	25	290	829

Testing for variation of sensibility throughout the day was omitted.

The behavior of the larvae is therefore significantly different from what would be expected in a random distribution. They are directed to the negative pole.

When the position of the electrodes are exchanged, many larvae also change their position quickly, thus indicating that the sensitivity is electrical and not chemical.

On the contrary, the larvae appear to not be oriented to the light or a magnetic field.

Holm, D.G. University of British Columbia, Vancouver, Canada. Analysis of nonrandom segregation of compound autosomes in males.

Several studies on the meiotic behavior of compound autosomes (Scriba 1967, 1969; Grell 1970; Evans 1971; Lutolf 1972; Fitz-Earle, Holm & Suzuki 1973; Holm & Chovnick 1975) have revealed that the recovery from females of gametes nonsegregational (disomic and nullosomic) for compound autosomes is not limited by the availability of complementary nonsegregational sperm. The regular and frequent production of sperm disomic and nullosomic for compound autosomes has led to the generally accepted concept that in males these aberrant chromosomes assort independently. Further support for this interpretation is offered by the frequency of egg hatch, which in most compound-autosomal strains is approximately 25%. While studies on a few compound-2 strains revealed egg-hatch frequencies somewhat greater than 25% (Clark & Sobels 1973; Holm 1976) recent findings disclose that in most strains of compound-2 males, C(2L) and C(2R) assort almost, if not totally, at random (Hilliker, Holm & Appels 1982). However, a major exception had been noted in earlier studies involving males carrying one particular compound-2R chromosome (Sandler et al. 1968; Evans 1971; Gethmann 1976). This compound, designated C(2R)cn, carries a duplication for a proximal segment of the 2L euchromatin and thus may carry a duplication for a 2L euchromatic pairing site.

In a recent study by Hilliker et al. (1982) a second major exception was uncovered, this one involving a pair of compound-2 chromosomes that had been generated in females heterozygous for the standard cn bw chromosome and the small pericentric inversion associated with the SD-72 chromosome. The breakpoints of the pericentric inversion in SD-72 lie in the proximal 2L euchromatin and proximal 2R euchromatin (Lewis 1962). Consequently, the C(2L)SD-72/+ is heterozygous for a proximal deficiency in 2L but carries a duplication for proximal 2R extending to 42A of the polytene chromosome map; the C(2R)SD-72/cn bw chromosome is heterozygous deficient for proximal 2R, but duplicated for proximal 2L extending to band position 39D3-4 (Ganetzky 1977; Hilliker et al. 1982).

From crosses involving males carrying these asymmetrical compound autosomes, which are designated C(2L)V12,SD72/+;C(2R)V43,SD72/cn bw, the recovery of nonsegregational progeny is reduced greatly in comparison to corresponding crosses involving males bearing compound-2 chromosomes in which the attached arms are homozygous for the proximal euchromatic segments. Such comparisons are revealed by the results entered in Table 1. Normally, when C(1)RM/Y; C(2L);C(2R) females are crossed to differentially marked compound-2 males, between 27 and 30%

Table 1. Recovery of compound-2 nonsegregational progeny from crosses involving C(1)RM/B^{S}Y;C(2L)P,b;C(2R)P,px females and +/+/B^{S}Y;C(2L)P,b;C(2R)P,px females mated with males bearing compound autosomes derived either from standard (Oregon-R) second chromosomes or from heterozygotes for the pericentric inversion in SD-72.

Cross	C(2L);C(2R) in male parent	Compound-2 chromosomes inherited from C(1)RM/B^{S}Y;C(2L)P,b;C(2R)P,px females: C(2L)	C(2R)	C(2L);C(2R)	0;0	Total	N**	y***
1	SH3,+;SH3,+	113	112	62	34	321	.299	
2	SH1,+;SH1,+*	433	571	177	231	1412	.289	
3	V12,SD72/+;V43,SD72/cn bw	157	122	6	9	294	.051	.112 .117
		Compound2 chromosomes inherited from +/+/B^{S}Y;C(2L)P,b;C(2R)P,px females						
4	SH3,+;SH3,+	483	573	463	232	1771	.404	
5	SH1,+;SH1,+*	309	335	198	244	1086	.407	
6	V12,SD72/+;V43,SD72/cn bw	115	101	4	13	233	.073	.104 .103

* C(2L)SH1,+ carries a duplication of 2R heterochromatin including rl^{+}.
** N = the frequency of nonsegregational progeny: progeny inheriting both C(2L) and C(2R) either from the female parent or from the male parent.
*** y = estimated frequency of nonsegregational (diplo-2 plus nullo-2) gametes produced by the C(2L)V12,SD72/+;C(2R)V43,SD72/cn bw males.

of the progeny arise from the fusion of diplo-2 and null-2 gametes, as exemplified by crosses 1 and 2 in Table 1. When +/+/Y;C(2L);C(2R) females are involved, the nonsegregational class represents approximately 40% of the total (e.g., crosses 4 and 5 in Table 1 and see Hilliker et al. 1982). The marked decrease in progeny arising from diplo-2 and nullo-2 sperm in crosses 3 and 6 (Table 1) indicate that the C(2L)V12,SD72/+ and C(2R)V43,SD72/cn bw chromosomes exhibit a high degree of meiotic segregation in males. This sixfold reduction from 29.9 and 28.9% to 5.1% and from 40.4 and 40.7% to 7.3% corresponds to a reduction in diplo-2 and nullo-2 sperm from 50% (random assortment of compound autosomes) to an estimated frequency of approximately 11% (see entries for y in the last column of Table 1). The relation between the frequency of nonsegregational sperm and the frequency of observed nonsegregational progeny, which is a function of the frequency of diplo-2 and nullo-2 gametes produced by the females, is explained in the following paragraphs.

♀ \ ♂	C(2L)′ (1-y)/2	C(2R)′ (1-y)/2	C(2L)′;C(2R)′ y/2	0;0 y/2	Frequency Viable Zygotes
C(2R) (1-x)/2	C(2L)′;C(2R)				$\frac{(1-x)(1-y)}{4}$
C(2L) (1-x)/2		C(2L);C(2R)′			$\frac{(1-x)(1-y)}{4}$
0;0 x/2			C(2L)′;C(2R)′		$\frac{xy}{4}$
C(2L);C(2R) x/2				C(2L);C(2R)	$\frac{xy}{4}$

Figure 1. The use of a Punnett Square to illustrate the distribution frequency of the four classes of viable zygotes arising from a cross between C(2L);C(2R) males and females.

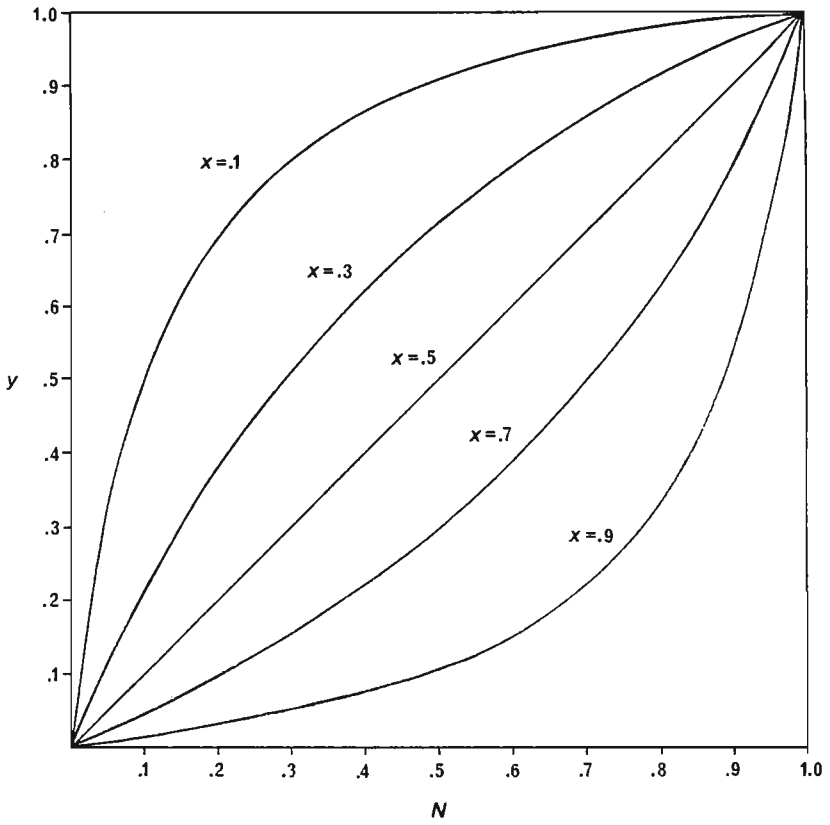

Figure 2. The frequency of y (the diplo-2 and nullo-2 products of spermatogenesis) as it relates to N (the frequency of nonsegregational progeny) for various values of x (the frequency of nonsegregational gametes produced by females).

Let x equal the frequency of nonsegregational meiotic events in females (i.e., frequency of diplo-2 plus nullo-2 gametes) and let y equal the nonsegregational frequency in males. The frequency of regular segregational products (i.e., gametes carrying only compound-2L or compound-2R chromosomes), therefore, will be 1-x and 1-y, respectively. Since, as shown in Figure 1, only C(2L) eggs fertilized by C(2R) sperm and C(2R) eggs fertilized by C(2L) sperm produce viable progeny in the segregational class (V_S), the frequency $V_S = 0.5(1-x)(1-y)$. Moreover, the frequency of viable progeny (V_N) arising from the fusion of diplo-2 and nullo-2 gametes (the nonsegregational gametes) equals 0.5xy. Although, in terms of the values of x and y, the frequencies of the two viable zygotic classes are not equal to the frequencies of nonsegregational (N) and segregational (S=1-N) progeny recovered, in the absence of differential viability, there is an equality of ratios such that:

$$N/1-N = xy/(1-x)(1-y) \qquad \text{(Equation 1)}$$

Equation 1 can be rearranged to give x as a function of y (Equation 2) or y as a function of x (Equation 3).

$$x = N(1-y)/\ N(1-y) + y(1-N) \qquad \text{(Equation 2)}$$
$$y = N(1-x)/\ N(1-x) + x(1-N) \qquad \text{(Equation 3)}$$

When the meiotic assortment of compound autosomes in males is random, that is y = 0.5, the frequency of nonsegregational gametes in females (x) is equal to the observed frequency of nonsegregational progeny (N). The same holds true when y is unknown, but the frequency x = 0.5. It will be noted that equations 2 and 3 are those used to correct for viability differences in such systems as SD (for example, Ganetzky 1977).

As demonstrated in Figure 2, for any value of x other than 0.5 the relation between y and N is nonlinear. However, if x is a known constant, then by measuring N, the value y can be estimated by Equation 3 given above. From the experiments reported in Table 1 we obtain from cross 4 involving +/+/B^SY;C(2L)P,b;C(2R)P,px females, a value of x = 0.404 and from cross 5 a value of x = 0.403. In the corresponding mating to compound-2, SD72 males (cross 6)

the frequency of nonsegregational products recovered is N = 0.073. Substituting these values for x and N in Equation 3 gives y = 0.104 and y = 0.103, respectively. For the two crosses (1 and 2) using C(1)RM/Y; compound-2 females we find x = 0.299 and x = 0.289. From cross 3, the frequency of nonsegregational products is N = 0.051. For x = 0.299 we obtain y = 0.112, and for x = 0.289, y = 0.117. These four values of y are in reasonably close agreement. Therefore, it would appear that between 10 and 12% of the compound-2 chromosomes in C(2L)V12;C(2R)V43 males fail to separate during meiosis. If this is a reflection of the proportion of meiotic events in which random assortment occurs, then approximately 80% of the meiotic products arise from segregation, that is C(2L)V12 and C(2R)V43 pair with approximately 80% fidelity. These findings suggest that C(2L)V12 and C(2R)V43 share homology for male meiotic pairing sites within the euchromatic segments defined by the limits of the SD-72 pericentric inversion.

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Irick, H.A. University of Washington, Seattle, Washington. Estimation of the number of genes in a region.

Several attempts have been made to determine the number of genes in a chromosomal region by mutational analysis. Two approaches to this are possible. Saturation of the region, such that each gene sustains multiple mutations, will detect all genes which can give rise to lethal, semi-lethal, or visible phenotypes (Hilliker et al. 1980; Lim & Snyder 1974; Judd et al. 1972). Other classes of genes, such as behavioral mutants, will frequently escape detection. Alternatively, the number of genes detected and the frequency of mutations per gene may be used to estimate the number of genes with zero mutations (Hochman 1973). The Poisson distribution allows estimation of the zero class, but assumes all genes have the same likelihood of mutation. Since the data generally do not approximate a Poisson (Hilliker, Chovnik & Clark 1980), only a subset of the data can be used for the estimate. It is therefore of interest to identify a distribution which allows a larger proportion of the observed data to be incorporated, and thus results in a more robust prediction of the zero class.

Hochman (1973) generated 182 mutations in 36 genes on chromosome 4 of D. melanogaster (Figure 1A). Deleting only a complex locus which received 35 mutations, the distribution of alleles detected per gene appears to resemble the discrete form of an exponential distribution:

$P(x) = \int_{x}^{x+1} Ae^{-Ax}\,dx$, where x is the number of alleles detected in a single gene and A is a constant defined only by the number of alleles recovered and the number of genes detected (Figure 1B and Figure legend). To determine whether the observed data is in fact a reasonable outcome of this theoretical distribution, a computer program was used to simulate the experiment. Random numbers were drawn from the candidate distribution, with the value of each number drawn determining the number of mutations observed in one gene. After 35 genes were mutated one or more times, the variance of the data from the theoretical distribution and the number of unmutated genes were saved. In 1000 trials, the variance of the simulated data was greater than the observed variance 473 times. Thus, the exponential distribution is an excellent model for determining mutability in this instance. In 95% of the trials, the number of unmutated genes fell within the range of 5 to 19, with a modal value of 11. The total number of genes on chromosome 4 is therefore predicted to be 47, with an allowed range between 41 and 55 (including the complex locus mentioned above). This corresponds very well to the salivary band number of 50 from Bridges (1942).