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.

Supported by research grant A5853 from NSERC of Canada.

References: Clark, A.M. & F.H. Sobels 1973, Mutation Res. 18:47-61; Evans, W.H. 1971, DIS 46:123-124; Fitz-Earle, M., D.G. Holm & D.T. Suzuki 1973, Genetics 74:461-475; Ganetzky, B. 1977, Genetics 86:321-355; Gethmann, R.C. 1976, Genetics 83:743-751; Grell, E.H. 1970, Genetics 65:65-74; Hilliker, A.J., D.G. Holm & R. Appels 1982, Genet. Res. 39:157-168; Holm, D.G. 1976, The Genetics and Biology of Drosophila Vol 1b:529-561; Holm, D.G. & A. Chovnik 1975, Genetics 81:293-311; Lewis, E.B. 1962, DIS 36:87; Lutolf, H.V. 1972, Genetica 43:431-442; Sandler, L., D.L. Lindsley, B. Nicoletti & G. Trippa 1968, Genetics 60:525-558; Scriba, M.E.L. 1967, Roux' Archiv. Entwment. 159:314-345; Scriba, M.E.L. 1969, Devel. Biol. 19:160-177.

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).

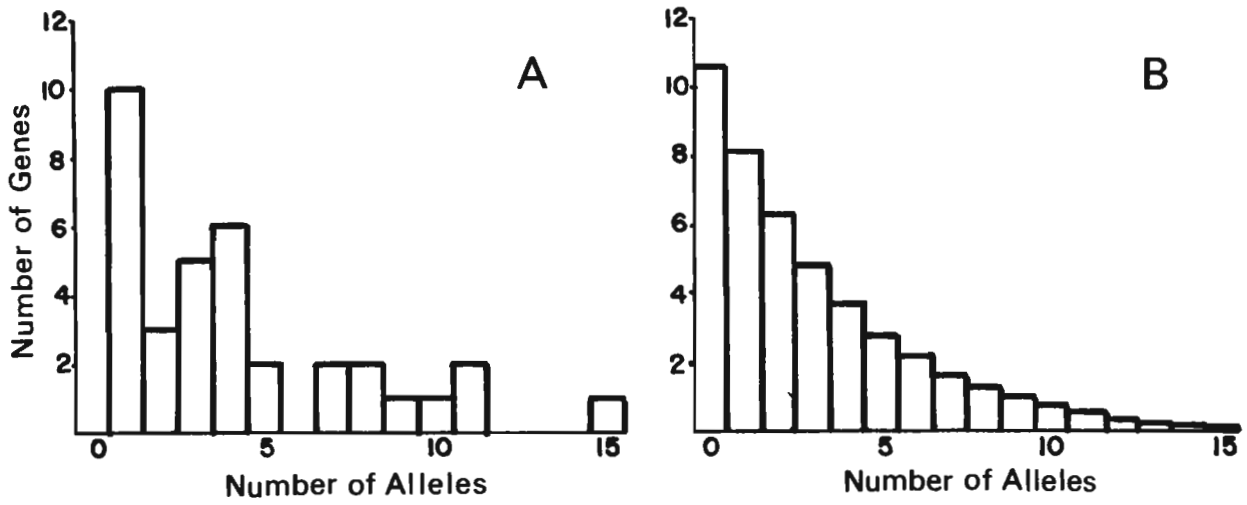

Figure 1. Comparison of actual mutation frequencies to the expected values derived from an exponential distribution.

A) Observed mutation frequency: 151 mutations were recovered in 35 genes on chromosome 4. (One gene with 35 mutants is omitted. Data of Hochman (1973)). Based on the number of genes in which 1 or 2 alleles were observed, 17 genes were predicted for the zero class by the Poisson distribution.

B) Expected frequency from an exponential distribution:

$P(x) = \int_{x}^{x+1} Ae^{-Ax}\,dx = (1-e^{-A})\,e^{-Ax}$ — Expected proportion of genes with x alleles. Since the number of alleles recovered must be an integer, P(x) is summed over the range of genes with at least x, but less than x+1, mutations.

A = Constant, defining the shape of the distribution.

$n(x) = N\,P(x) = N\,(1-e^{-A})\,e^{-Ax}$ — Expected number of genes with x alleles observed.

$N = 35 + n(0) = 35 / e^{-A}$ — Total number of genes.

$E(x) = \bar{x} = 151 / N$ — Mean number of alleles per gene.

$Mx(t) = (1-e^{-A}) / (1-e^{t-A})$ — Moment generating function of P(x).

$E(x) = [M\acute{x}(t)]_{t=0} = e^{-A} / (1-e^{-A})$

The two equations for E(x) allow solution of e^{-A}=0.7682, E(x)=3.314, A=0.2637, N=45.56, and n(0)=10.56.

It remains to develop a biological justification for this distribution. As mentioned earlier, the Poisson distribution is valid only if all genes have equal mutability. However, variations in mutation frequency between genes should be expected, due to differences in gene length, DNA sequence, protein conformation, and the presence of mobile elements. Furthermore, the likelihood of recovering mutations may be reduced due to duplicate genes, behavioral or nutritional mutants, or alternative enzyme pathways. It is useful to think of the actual distribution of mutability as un unknown function, where the mutability of most genes is within a single peak, such as a normal distribution. In a non-saturating screen, the less mutable genes are found in the classes of zero or one mutations, creating the observed exponential distribution. This analysis would not be meaningful for a saturation mutagenesis, as the exponential requires the zero class to be the most frequent.

References: Hilliker, A.J., S.H. Clark, A. Chovnick & W.M. Gelbart 1980, Genetics 95:95-110; Lim, J.K. & L.A. Snyder 1974, Genet. Res. 24:1-10; Judd, B.H., M.W. Shen & T.C. Kaufman 1972, Genetics 71:139-156; Hochman, B. 1973, CSHSQB 38:581-589; Hilliker, A.J., A. Chovnick & S.H.Clark 1980, DIS 56:64-65; Bridges, P.N. 1942, J.Heredity 33:403-408.

Kaytes, P. & D.L. Hartl. Washington University School of Medicine, St. Louis, Missouri. Note on electrophoretic mobility and tissue localization of β-flucuronidase.

In the hope of using β-glucuronidase as a model for studying genetic regulation of enzyme activity, we have attempted to identify putative structural gene(s) by surveying isofemale and chromosome substitution lines of Drosophila melanogaster for variation in electrophoretic mobility. Mass homogenates were prepared by sonicating 40 flies in sonication buffer (.1N sodium acetate, pH 5.0, 10% sucrose) for 3 15-second bursts from a Heat Systems sonicator at 35% duty cycle, output setting of 5 with intermittent ice cooling. After pelleting cellular debris by centrifugation in a Sorvall SS-34 rotor, 50 microliters of the supernatant was applied to a vertical gel run in the system of Clarke (1964) at 10 mA/gel for 2½ hours and stained by the method of Hayashi (1963) with the modification that gels were pre-incubated in the acetate buffer without sucrose for ½ hour to eliminate background staining. The stain is sufficiently sensitive that single-fly bands can be obtained by sonicating in 50 microliters of buffer and applying all of the supernatant to the gel. Enzyme activity was localized to a single insoluble red band. (A diffusely staining area not sharply banded was also observed but not studied in detail.) The sharp band was eliminated by the inclusion in the staining mixture of saccharolactone, a competitive inhibitor of β-glucuronidase. In all, 124 lines from 8 geographical locations were examined, but no mobility variation could be detected. In contrast, β-glucuronidase extracted from D. simulans exhibited a significantly slower (R_f=.91 relative to D. melanogaster) form of the enzyme. Isoelectric focusing by the method of Righetti and Drysdale (1971) showed the melanogaster enzyme to be slightly more acidic; mixing experiments failed to show interconversion of forms. We conclude that, under our electrophoretic conditions, the sharply banded form of β-glucuronidase is monomorphic in Drosophila melanogaster, but that interspecific variation does exist.

Tissue distribution studies were also carried out on the enzyme. Adult flies were mounted and thin frozen sections were taken as in Kankel and Hall (1976). The sections were stained for activity as for gels without pre-incubation; no fixation was necessary. The greatest level of activity could be seen in male reproductive structures, particularly the accessory glands, and also in the ejaculatory bulb and testes. The presence of β-glucuronidase in the male reproductive system was further confirmed by hand dissection and staining. Slight amounts of activity could be seen in the digestive tract, particularly the stomodeal valve and intermittently in the Malpighian tubules. All activity staining was abolished in the presence of saccharolactone.

References: Clarke, J.T. 1964, Ann.New York Acad.Sci. 121:428-436; Hayashi et al. 1963, J.Histochem.Cytochem. 12:293-297; Kankel & Hall 1976, Dev. Biol. 48:1-14; Righetti & Drysdale 1971, Biochim.Biophys.Acta 236:17-28.

Kekic, V., R. Hadziselimovic & Z. Smit. University of Belgrade and University of Sarajevo, Yugoslavia. Drosophila fauna of artificial microhabitats in Bosnia and Herzegovina, Yugoslavia.

During the fall of 1969, we collected Drosophila flies at 29 localities in Bosnia and Herzegovina, covering the heights from 90 to 1031 meters above sea level (see Figure).

Collecting was carried out at man-made microhabitats--in the immediate vicinity of barrels in which plums, prepared for home distillation of plum-brandy, were fermenting; vials with a small amount of fermenting plums were set out and after a certain time (every 3 hours) closed. Caught flies were taken out by means of aspirator, then fixed and kept in 70% ethanol until the time of identification.