

Barker 1 sample became adapted to the different conditions in my laboratory (a different food and the change from vials to bottles are probably the most dramatic differences) before experiment 1 was conducted. This adaptation increased the "competitive ability" of Barker 1 so that it quickly eliminated simulans 1. Simulans 1 also adapted in some manner so that it could outcompete the fresh Chicago 1 sample in experiment 2. The fact that experiment 3 results were as initially predicted was because all samples were obtained simultaneously and the experiment was run immediately after they were obtained. Further support of this hypothesis comes from Barker (pers. comm.) who has found in a second one-generation test that his stock in a recent retest produced approximately 40% *D. melanogaster* from 50% *D. melanogaster* parents, a value very close to his earlier results and quite unlike mine in experiments 1 and 2.

Grossfield, J. Purdue University, Lafayette, Indiana. A non-heuristic attribute of the ERG.

In conversation with several colleagues the question of electroretinograms (ERG's) being used to trace evolutionary patterns in the genus has arisen. I wish to point out that all species of *Drosophila* tested to date have the same waveform and time course when ERG's are recorded under comparable conditions. This has held true over the past few years when ERG's have been cursorily checked in this laboratory for members of the *melanogaster*, *obscura*, *virilis*, *quinaria*, *robusta*, and *annulimana* species groups. Indeed, on the basis of available information, the same is true for all Diptera, with flies such as *Calliphora* and *Musca* showing larger amplitude responses. The presence of screening or accessory pigments in the eyes of various species may change the sensitivity of the response somewhat but that would be the maximal effect expected.

Hall, J.C. University of Washington, Seattle, Washington. The failure of two alleles of *c(3)G* to increase frequencies of X-linked lethals.

Green (Mut. Research 10:353, 1970) has discovered a putative allele of the recombination-deficient mutant, *c(3)G*, picked up as a mutator gene. It is a third chromosome semidominant whose locus is absent from *Df(3R) sbd*¹⁰⁵--as is true

for *c(3)G*. The frequencies of mutations at certain X chromosome loci are increased in the presence of the mutator gene in females. In addition, recombination is somewhat reduced by this mutant (M. Green, personal communication). Green's preliminary allelism tests show that females bearing the mutator and *c(3)G* in heterozygous condition do not generate mutations in the relatively high frequency found for the mutator in homozygous condition. This means that a) *c(3)G* and the mutator are alleles, but *c(3)G* is not a mutator; or b) *c(3)G* may or may not be a mutator, but it is not an allele of Green's mutant.

Both *c(3)G* and *c(3)G*⁶⁸ -- a newly arisen allele of this meiotic mutant (*mei-W22* of Sandler, DIS 47, 1971, in press) -- have been directly assayed for possible mutator properties. Parry (Ph.D. Dissertation, University of Washington, 1970) found that a meiotic mutant which lowers recombination and increases nondisjunction generates increased frequencies of sex-linked lethals. Such sex-linked lethal tests were carried out for the two alleles of *c(3)G*, in which the treatment of X chromosomes consisted of passage of these X chromosomes through females homozygous for either meiotic mutant. For *c(3)G* only one of 473 treated X chromosomes carried a lethal. And for *c(3)G*⁶⁸ none of 553 X's had a lethal induced. In a control, seven of 931 X's recovered from *c(3)G*⁺/*c(3)G*⁺ females bore a lethal. Four of these lethals arose from one female, and three from another, so the seven lethals probably represent only two mutations, each of which occurred at an oogonial stage and was proliferated. In any event, *c(3)G* does not appear to be a mutator gene.

Samples of the X chromosomes passed through these three kinds of females were examined for the presence of half chromatid (mosaic) lethals (produced in high frequency by chemical mutagens -- e.g. Carlson and Southin, Genetics 48:663, 1963). If *c(3)G* were generating increased frequencies of such half chromatid lethals in meiosis, after chromosome replication, they would go undetected among the *F*₁ males in a sex-linked lethal test (defining the P generation mothers as those bearing an X chromosome balancer and an X from a *c(3)G* female). However, of 87 X's from *c(3)G*, 70 from *c(3)G*⁶⁸, and 292 from *c(3)G*⁺, none was found (in the *F*₂) to have originally carried a half chromatid lethal.