

homogeneity was highly significant ($\chi^2 = 427.7, 32 \text{ df}, p < .001$), in good agreement with the earlier observations in *D.melanogaster* mutants made by Waddington, Woolf, and Perry.

Reference: Waddington, C.H., B. Woolf, and M.M. Perry 1954, *Evolution* 8:89-96.

Thompson, V. Roosevelt University, Chicago, Illinois. Failure of the Hn^{r3} ry^6 combination to behave as a recessive synthetic lethal.

Taira (1960) and Goldberg et al. (1962) report that the *D.melanogaster* third chromosome mutant eye color alleles Hn^{r3} and ry^6 combine to form a recessive synthetic lethal and Lucchesi (1968) lists the Hn^{r3} ry^6 combination among the established synthetic lethal systems involving

laboratory mutants. I have synthesized Hn^{r3} ry^6 chromosomes and find the homozygotes to be viable.

The ry^6 allele was obtained from A. Chovnick in the form of a balanced stock of genotype $\text{M}(3)\text{S34 Dfd kar ry}^6/\text{In}(3\text{R})\text{Ubx}^{\text{A}}, \text{cu kar Ubx}^{\text{A}}$. Males of this stock were crossed to females homozygous for Hn^{r3} and sr (Fig. 1) following the general scheme of Goldberg et al. Heterozygous $\text{M}(3)\text{S34 Dfd kar ry}^6/\text{Hn}^{\text{r3}} \text{sr}$ daughters were backcrossed in turn to males homozygous for Hn^{r3} and sr . F2 males with Henna eye color and no Deformed eye phenotype were individually test crossed to a stock carrying the ry^2 allele linked to Sb (sr could not be readily scored and was ignored). F3 Stubble rosy phenotype males from test crosses that produced rosy off-balancer chromosome and resulting F4 Ubx^{130} heterozygotes (without Sb) were crossed inter se to produce the F5 generation. All crosses were performed at $25 \pm 1^\circ\text{C}$ on cornmeal-malt-yeast medium.

One hundred twenty-four Henna phenotype F2 males were successfully test crossed. Twenty-six proved to carry the ry^6 allele on the maternally derived Hn^{r3} bearing chromosome. Sixteen of the twenty-six also bore the Dfd allele and were discarded. The F3 and F4 crosses were carried out independently and in parallel for the ten remaining lines each of which carried an independently arising $\text{Hn}^{\text{r3}} \text{ry}^6$ chromosome.

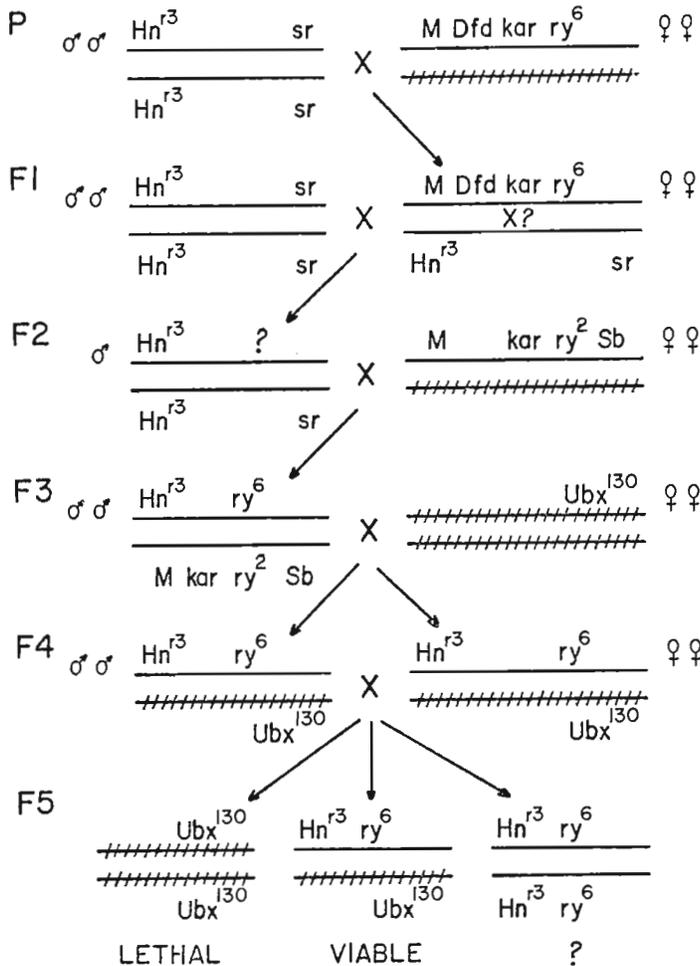


Fig. 1. Crosses used to synthesize and test the homozygous viability of $\text{Hn}^{\text{r3}} \text{ry}^6$ third chromosomes. Only mutant alleles are shown and no attempt is made to indicate proportional map distances. Cross hatched chromosomes carry recombination suppressing rearrangements. Genotypes of balancer chromosomes are omitted when not directly relevant.

In six of these ten lines every F5 individual exhibited the Ultrabithorax phenotype, indicating that the synthesized $Hnr^3 ry^6$ chromosome was lethal in homozygous condition. In the other four lines 10-20% of the F5 individuals exhibited orange eye color and failed to exhibit the Ultrabithorax phenotype. The orange eye color was distinct from the eye color of kar , ry and $kar ry$ homozygotes. These observations indicated that four of the independently arisen $Hnr^3 ry^6$ chromosomes were viable in homozygous condition, a suggestion confirmed by test crossing putative homozygotes to appropriate tester stocks.

Subsequently, Ubx heterozygotes from the six lines which produced no viable homozygotes were also test crossed. In each case the presence of the Hnr^3 allele was confirmed, but heterozygotes for the synthesized chromosomes and the original ry^6 bearing chromosome proved to be lethal. Apparently, the recessive lethality of these six chromosomes was due to homozygosity for factors present on the original ry^6 bearing chromosome, not to homozygosity for the $Hnr^3 ry^6$ combination per se.

The reported $Hn ry$ synthetic lethal system was peculiar in its limitation to only one of five ry alleles tested in combination with Hnr^3 (Goldberg et al. 1962). It is possible that in the previous work on this system the ry^6 allele served as a marker for a linked hidden lethal interaction factor that has since been lost. Alternatively, the viability of $Hnr^3 ry^6$ homozygotes may depend critically on the genetic background at one or more other loci (three of my four homozygously viable $Hnr^3 ry^6$ chromosomes carried the kar allele, which was probably present in the $Hnr^3 ry^6$ chromosomes of Goldberg et al. as well). Whatever the source of the discrepancy in results, the extant ry^6 allele does not combine with Hnr^3 to form an unconditional synthetic lethal.

References: Goldberg, A., A. Schalet & A. Chovnick 1962, DIS 36:67-68; Lucchesi, J.C. 1968, Genetics 59:37-44; Taira, T. 1960 DIS 34:107.

Thompson, V. Roosevelt University, Chicago, Illinois. Second chromosome crossing over in *D.melanogaster* females heterozygous for first, second and third chromosome balancers.

Multiple heterozygosity for balancer chromosomes often leads to a breakdown in the effectiveness of individual balancers (MacIntyre & Wright 1966). Here I report the effect on second chromosome recombination of simultaneous heterozygosity for the first chromosome balancer $winscy [In(1)sc^S Lsc^{8R}+dl-49, y sc^{S1} sc^8 w]$ and

Wallace's "A1" second-third chromosome translocation. The A1 rearrangement is the result of a reciprocal translocation between two balancer chromosomes, $In(2L) Cy In(2R) Cy, Cy L$ and $In(3LR) Ubx^{130}, Ubx^{130eS}(=TM2)$ (Wallace 1966; _____ et al. 1966). In the absence of first chromosome structural heterozygosity the A1 rearrangement suppresses most second chromosome recombination, with the notable exception of about 4% crossing over in the vicinity of the centromere (Thompson 1977).

Males hemizygous for the $winscy$ chromosome and heterozygous for the A1 rearrangement were crossed to females from stocks homozygous for net, vg and $dp b bw$. Daughters carrying Cy, L and Ubx were backcrossed to males from the appropriate mutant stock and the progeny scored for phenotype. The results, based on 1004 vg cross offspring, 582 net cross offspring and 383 $dp b bw$ cross offspring, appear in Table 1. Left arm recombination is not affected by the introduction of $winscy$ heterozygosity and remains at very low levels, perhaps because the left arm includes the second chromosome break point of the translocation. Right arm recombination is markedly increased (about 10-20 fold over A1 heterozygote levels). Not unexpectedly, most or all of the increase in recombination appears to take the form of double crossing over.

This is reflected in strong negative crossover interference in the $Cy-vg-L$ and $Cy-L-bw$ intervals, which exhibit interference values of -2.3 and -1.8 respectively.

Table 1. Second chromosome crossing over in females heterozygous for the $winscy$ and A1 balancer chromosomes.

| Map position | 0.0 | 6.1 | 13.0 | 48.5 | 67.0 | 72.0 | 104.5 |
|-----------------|-------|------|------|-------|------|------|-------|
| Marker | net | Cy | dp | b | vg | L | bw |
| % crossing over | 0.0 | 1.0 | 0.5 | 16.1* | 4.5 | 9.4 | |

*Estimated from $Cy-vg$ and $Cy-L$ crossover values in conjunction with the other values given.