

Ohnishi, S. and R.A. Voelker, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina. Genetic mapping of hexokinase-C in *D. simulans*.

In order to map hexokinase-C locus (Hex-C) genetically, a wild type stock with a variant allele (Hex-C⁶) was crossed to a multiple marker stock for the second chromosome [net (net, 0) b (black, 43) py (polychaete, 74) sd (spread, 80) pm (plum, 103)], which carries the common allele (Hex-C⁴).

F₁ females obtained from the cross were backcrossed to males of the marker stock. These offspring were electrophoretically analyzed after scoring for visible markers. The data are summarized in Table 1.

Table 1. The summary for genetic mapping of Hex-C in *D. simulans*.

Visible Phenotype	Hex-C ⁴ /Hex-C ⁶	Hex-C ⁴ /Hex-C ⁴
sd pm	3*	31
+ +	40	0
sd +	3	14
+ pm	26	13

*These are double crossovers between sd and pm.

Hex-C was located between sd(80) and pm(103) at 86.6 (= 80 + 23 x 16/56) with the 95% binomial confidence interval extending from 83.7 to 89.2 on the second chromosome. The location corresponds to that in *D. melanogaster* (Jelnes 1971, 2-73.5; Mukai and Voelker 1977, 2-74.5).

References: Jelnes, J.E. 1971, *Hereditas* 67:291-293; Mukai, T. and R. A. Voelker 1977, *Genetics* 86:175-185.

O'Tousa, J. and P. Szauter, University of Washington, Seattle, Washington. The initial characterization of non-claret disjunctional (ncd): evidence that cand is the double mutant, ca ncd.

Females homozygous for cand show two distinct phenotypes: an abnormal eye color and aberrant meiotic chromosome behavior. Several lines of evidence have suggested that this compound phenotype is actually the result of cand being defective at two closely linked loci, one governing eye pigment metabolism and the other meiotic

chromosome behavior (see Baker and Hall 1976 for a review on the literature of cand). We report here on the recovery of a new meiotic mutant, non-claret disjunctional (ncd) that provides strong evidence that cand is indeed a double mutant.

ncd was isolated in a search for meiotic mutants among EMS-treated 3rd chromosomes in the laboratory of D.L. Lindsley at U.C. San Diego and analyzed by us in Seattle. Females homozygous for this mutant exhibit meiotic abnormalities similar to those of cand / cand females, and the mutant fails to complement cand (Table 1). Furthermore, the ncd mutation has been mapped to the distal region of 3R where cand is located. These results confirm that cand and ncd are allelic; however, both ncd/ncd homozygotes and ncd/cand compound heterozygotes have wild-type pigment. Therefore, ncd must be defective only at the cand locus governing chromosome behavior. The recovery of ncd, then, is evidence that cand is a double mutant with the implied constitution: ca ncd.

Table 1. The results of the crosses of the indicated females by \widehat{XY} , vFB; $\widehat{44}$, ci ey males.

maternal genotypes:	progeny classes									exceptions				
	y $\sigma\sigma$ & B/+ $\sigma\sigma$				vFB $\sigma\sigma$				y $\sigma\sigma$		per 10 ³ ova			
	+	ci	ey	pol	+	ci	ey	pol	+	ci	ey	pol	X	4
$\frac{y, ncd, pol}{y, ncd, pol}$	45	39	1		1	25	0		0	1	2		405	650
$\frac{y, ca^{nd}, pol}{y, ncd, pol}$	13	35	0		7	20	0		3	1	0		564	700
$\frac{y, ncd, pol}{y, +, pol}$	137	0	0		0	0	0		0	0	0		0	0

References: Baker, B.S. and J.C. Hall 1976, in: *Genetics and Biology of Drosophila*, vol. I (ed. by E. Novitski and M. Ashburner), Academic Press, NY.