Table 2. Three-way factorial ANOVA of the arcsine transformed values of the percent mortality data.

Source of variation	df	MS	Fs
A (female)	2	15.5148	14.52*
B (day)	4	0.9135	0.85
C (male)	2	0.3363	0.31
A x B (female x day)	8	2.2086	2.06
A x C (female x male)	4	0.2994	0.28
B x C (day x male)	8	0.4814	0.45
A x B x C	16	1.0682	

* P<0.05

& Rohlf 1969) was performed with the arcsine transformed values of the percent mortality. Table 2 shows the results of this test. As it can be seen, there is no significant effect of two main factors (male and day), the effect of the third factor (female) being slightly significant (fs=14.52; P<0.05). The significant effect of the female is due to the lower percent mortality in the progeny of 2 jz^3/st heterozygotes (1.26%) when compared with that of st/st and jz^3/jz^3 homozygotes (3.18% and 3.45%, respectively). This is just the opposite result of what we expected according to the hypothesis outlined above. If we look at the unfertilized egg data, the same pattern is observed (4.08% versus 23.25% and 4.55%). Therefore, a possible confusion between dead embryos and unfertilized eggs does not change the situation at all. There are two alternative explanations for the lack of embryonic lethality in the progeny of 2 jz^3/st heterozygous females. (1) In the meiosis of heterozygotes for complex inversions, such as 2 jz^3/st, crossing-over in the crucial region of the chromosomes is effectively decreased. (2) Abnormal chromosomes carrying duplications and deficiencies produced by crossing-over are not incorporated into functional egg cells. Whichever the solution to this question is, the low frequency of the 2 jz^3 arrangement in most natural populations and its correlation with that of the standard arrangement remain unexplained on cytological grounds.

References: Curtsinger, J.W. 1981, DIS 56:33-34; Fontdevila, A., A. Ruiz, G. Alonso & J. Ocana 1981, Evolution 35:148-157; Fontdevila, A., A. Ruiz, J. Ocana & G. Alonso 1982, Evolution 36:843-851; Riles, L. 1965, Genetics 52:1335-1343; Sokal, R.R. & F.J. Rohlf 1969, Biometry, W.H. Freeman & Co.; Sturtevant, A.H. 1938, Quant.Rev.Biol. 13:333-335; Wallace, B. 1953, Amer.Nat. 87:343-358; Wasserman, M. 1962, Texas Univ.Publ. 6205:85-117.

Schalet, A. University of Leiden, The Netherlands. Vital loci located at the junction of polytene X chromosome sections 2B and 2C in D. melanogaster.

Among approximately 150 independent spontaneous X-chromosome lethals, obtained from crossing wild-type males (M56i, Amherst) to In(1)sc^8 In(1)dl-49, y^{31d} sc^8 w^a v^{0f} f females, there were 5 lethals that mapped genetically between sc (1B4) and pn (2E1), but none were covered by $y^2Y^{67g19.1}$ or T(1→3)w^{vco}, or both of these duplications taken together. Nevertheless, all the lethals were covered by Dp(1;f)R (1A4-3A) and the 3 lethals tested, (5-114, 5-39, 6-62), were covered by Dp(1;f)Z9 (1A1-3E7). Lethal bearing chromosomes marked with y ac sc were obtained from linkage mapping experiments and used in combination with Df(1;f)R, which carries y^+ ac^+ sc^+, to perform allelism tests in the type cross: Dp(1;f)R/y ac sc 1^x/Y males X y ac sc 1^y/FM7 females. The 5 lethals fell into 3 complementation groups. The 3 groups and their estimated linkage map positions are: 1. 5-39, 6-62 (0.5); 2. 5-114, 14-28 (0.5); 3. 11-94 (0.7).

According to Lefevre (1981) the proximal limit of the distal region duplicated in $y^2Y^{67g19.1}$ is 2B17±. Lindsley & Grell (1968) gives the distal limit of the w^{vco} duplication as between 2B17 and 2C1, however, in a personal communication to P. Kramers, Lefevre indicates that the duplication does not include 2C1. Accordingly, our results suggest that there is indeed a "gap" between the regions covered by the two duplications, i.e., at the junction of polytene chromosome sections 2B and 2C, and that the 5 lethals described above are located in this interval. In the June 1982 Computerized Stock List 3 these lethals are designated under 1(1)S-2B-C.

References: Lefevre, G. 1981, Genetics 99:461-480; Lindsley, D.L. and E.H. Grell 1968, Carnegie Inst.Wash.Publ. 627.