Stocker, A.J. University of Texas (Southwestern) Medical School at Dallas. An apparent developmental anomaly in Drosophila pseudoobscura induced by injection.

In an experiment to determine the effects of LSD and related compounds on Drosophila, third instar female larvae were injected with either a phosphate-buffered-saline LSD-25 solution or with phosphate-buffered-saline alone. A considerable number of the F-1 progeny from both the experimental and control animals exhibited abdominal segmentation anomalies in which the sternites and tergites were disrupted. These progeny were usually produced from eggs laid during the first few days after initiation of egg laying. An example of a fly bearing such a segmentation anomaly is shown in figure 1.

Figure 1. An example of a fly exhibiting the described segmentation anomaly.

These "segmentation defective" flies were usually fertile and produced normal progeny. The cause of this defect is not known. It is clear, however, that there is no increased incidence of such anomalies among the offspring of the LSD-25 (and related compounds) treated animals; neither were such anomalies observed in uninjected flies. The effect could not be attributed to the injection itself since the adults produced from the injected larvae completed their development without exhibiting any such abnormalities. It appears, therefore, that some agent transmitted during the injection affects the developing ovary in some unknown manner to produce a specific effect, which seems to be of a developmental rather than a genetic nature.

Work supported in part by Grant no. MH 15743-01 from the National Institutes of Mental Health, and a grant from the Moody Foundation.

Lefevre, G. and M.M. Green. San Fernando Valley State College, Northridge, California; University of California, Davis, California. Interactions of deficiencies in the 3C region.

Unexpected phenotypes appear among females heterozygous for different deficiencies involving the 3C region. Specifically, a short, male-viable deficiency for band 3C2-3 expresses only a white-eyed phenotype, but is delayed in emergence for 2 to 3 days (in uncrowded cultures) as compared with normal brothers. This deficiency, $w^{67c23(3)}$, when combined with N^{64i16} or N^{64j15}, both deficient from 3C3 through 3C7 and beyond, produces viable females that express the vertical phenotype, but are not rough-eyed. $Df\ w^{67k30}$, deficient for bands 3C2-6 inclusive, is male lethal, but when combined with N^{5419}, deficient from 3C6 to 3C11, again produces viable females that express vt, but not rst. Thus, homozygous deficiency for either 3C3 or 3C6 accompanied by heterozygous deficiency for the other, gives rise to the vt phenotype. This suggests the persistence of a genetic duplication associated with the 3C2-3 and 3C5-6 doublets. Furthermore, such a duplication would explain the anomalous lethality of the $w^{m4L}rst^{3R}$ chromosome, in which band 3C2-3 is deficient (which should not be lethal by itself), but band 3C5-6 is inactivated by position effect. When either one or the other of these two bands remains, the condition is not lethal and a vt phenotype is expressed, as in the rst^2 deficiency. When 3C5 is affected, the rst phenotype is observed, but its homozygous deficiency is not lethal. The only male-lethal mutants between w and spl are deficiencies that remove, at least, both 3C3 and 3C6, as in $Df\ w^{67k30}$. The effect of 3C5 on the viability of the 3C3, 3C6 double deficiency is being determined.