and Kwangju-Chunnam), faster in the three strains (Taiku, Taijun, and Jeungpyung), and intermediate between them in the remaining strains from ten localities.
3. The activity of the ADH isozyme is extremely strong in the Taiku strain.
4. Such a difference of the ADH isozyme patterns, mobility, and activity among strains of Drosophila melanogaster from various localities of Korea implies that the genetic constitutions of the ADH isozymes is different among the strains from various localities of Korea.
5. The genetic analysis on this difference of the ADH isozyme patterns is going on now.

Baker, B.S. University of Washington, Seattle, Washington. Tests for chromosome breakage in the meiotic mutant paternal loss.

The recessive male meiotic mutant paternal loss (2-35.7) (pal = mei-W5 of Sandler, 1971) when homozygous in males causes frequent loss of paternal chromosomes (Baker, 1972). Loss may be either complete (nullisomic exceptions) or somatic during the early cleavage divisions of progeny of pal males. To examine whether chromosome loss might be the result of chromosome breakage the following tests were performed. (1) Muller-5 tests: 9 lethals were found among 1312 chromosomes recovered from pal/pal fathers in a Muller-5 test. From pal^+/pal^+ control males 4 lethals were recovered among 1299 chromosomes. An F_2 Muller-5 test gave 4 lethals/1154 chromosomes from pal males and 4 lethals/997 chromosomes from pal^+ males. (2) Translocation tests: $y/\overline{y^+YB^S}$;pal/pal males were crossed to $\overline{XY}$, EN(1)y/y, dl-49, Hw m^4 g^2;bw;st females and 495 $\overline{XY}/\overline{y^+YB^S}$ F_1 males were crossed to females of their mothers' genotype and one T(Y;3) was found. $\overline{XY}/y^+YB^S$;+/bw;+/st sons of the F_1 males were similarly tested for the presence of translocations and none found (454 males tested). (3) Male recombination. No recombinants between Pr(3-90.0) and Ly(3-40.5) were found among 5548 progeny of pal/pal;Pr Ly/++ males. (4) Isochromosomes. No new compound third chromosomes were found in a cross of 210 pal/pal;+/+ males to +/+;C(3L)RM, se h^2 rs^2;C(3R)RM, sbd gl e^S females. (5) Dominant lethality. Egg hatch experiments revealed that there was 12-17% more dominant lethality in crosses involving pal/pal males than in crosses using females of the same stocks to SM1/pal or +/+ males (Table 1).

Table 1. Effect of pal on egghatch. Flies premated 3 days and only those matings that showed larvae in the premating vial were used to obtain eggs.

	male	female	total eggs	% unhatched
1.	pal/pal	Canton-S	814	30.1
	pal/SM1	"	985	12.4
2.	pal/pal	Canton-S	1254	40.6
	pal/SM1	"	1066	28.6
	+/+	"	807	24.9
3.	pal/pal	$y/y;spa^{pol}/spa^{pol}$	561	21.0
	pal/SM1	"	665	3.9
	+/+	"	714	3.5

That this dominant lethality is probably due to sperm exceptional for the major autosomes and not chromosome breakage is suggested by the following considerations. Between 0.3 and 0.75 sperm nondisjunctional for one major autosome are recovered per male in cross of pal/pal; +/+ males to $X/X/B^SY$ females bearing either compound second or third chromosomes. Only about 1/6 of the ova produced by such females are complimentary to a given type of exceptional sperm (Grell 1970). Thus considering both major autosomes there are 3.6 to 9 autosomal exceptional sperm produced per pal male. As 120 progeny are produced per male in crosses to free autosome females using the same mating regime 3 to 7.5% of the recoverable sperm produced by a pal male are exceptional for one major autosome. Hence 18% (3/17) to 63% (7.5/12) of the dominent lethality results from aneuploidy for a major autosome. As these calculations do not take into account somatic loss of the major autosomes or the concomitant loss of both major autosomes it seems likely that all of the dominant lethality caused by pal is the result of aneuploidy for the major autosomes. In summary, all tests for chromosome breakage in pal males have been negative.

References: Baker, B.S. 1972 (in preparation); Grell, E.H. 1970, Genetics 65:65-74; Sandler, L. 1971, DIS 47:68.