The preferred direction of a typical local motion detector in Drosophila seems to be invariably determined by the relative position of two contiguous elements in the hexagonal array of visual units. Control of the preferred direction is more likely to occur at higher levels of motion processing, e.g., at the level of spatial integration of the detector signals. However that may be, wind-induced shift is not compatible with a conservative wiring scheme of the optomotor control system.

References: Heisenberg & Wolf 1979, J.Comp.Physiol. 130:113-130; Wolf & Heisenberg 1980, J.Comp.Physiol. 140:69-80; Collett 1980a, J.Comp.Physiol. 138:271-282; Collett 1980b, J.Comp.Physiol. 140:145-158; Buchner, Goetz, & Straub 1978, Biol.Cybernetics 31:235-242; Goetz & Buchner 1978, Biol.Cybernetics 31:243-248.

Golubovsky, M.D. Institute of Cytology and Genetics, Novosibirsk, USSR. The increase of X-linked lethal and non-disjunction rates in genotypes with unstable singed alleles in D. melanogaster.

As it was suggested in 1977 the instability of different sn alleles isolated from wild populations was due to insertion of movable or transposable elements (TE) and action of MR natural factors (Golubovsky et al. 1977; Green 1977). It is well known that one of the main features of TE is the induction of the site specific mutations and chromosome breakage. The appearance of lethal mutations is the most typical consequence of TE integration in a vital locus. It is possible to expect that the breakage of chromosomes may be connected with the disturbance of their disjunction. So it is interesting to estimate the same components of the mutation process in lines having insertional unstable mutations. For this purpose we made an attempt to estimate: (1) the frequency of sex-linked lethals in X-chromosome containing unstable sn; (2) to what extent the allelic transition in the unstable sn is directly connected with the appearance of lethal mutation in other loci of the X-chromosome; (3) the rate of non-disjunction of X-chromosomes with unstable sn. All these parameters have been obtained by using the slightly modified Muller-5 technique:

F_0 $1\sigma \xrightarrow{sn^*}$ x $\frac{w^a\ B\ (Basc)}{w^a\ B\ (Basc)}$ ♀♀

(sn* - the unstable)

F_1 $1♀ \frac{sn^*}{w^a\ B}$ x $\xrightarrow{w\ sn^3}$ ♂♂

F_2 regular phenotypes:

♀♀ w^aB; sn. ♂♂ w^aB;

sn* (are absent if the lethal occurred in a gamete of the F_0 male)

Table 1. The increased lethal mutation frequency in the X-chromosomes containing unstable singed alleles. The absence of direct association between the sn mutations and the appearance of lethals in other loci of the same X-chromosome.

Allele in X-chrom.of F_0 males	number chrom. tested	Mutational events in X chromosome of $\xrightarrow{sn^*}$ ♂♂		
		I only sn mutations	II sn mutat. and lethals in same chrom.	III only lethals and strong semilethals
sn^{11}	406[a]	4(27)	1(3)	2
	106[b]	8	1	4
sn^{+10}	222[b]	2	0	2
sn^{B8}	168	5(12)	0	2
sn^{B14}	117	4(11)	0	2
Total	1019	23(50)	2	11

Experiment: 1019 chrom. - 13 lethals or 1.28%±0.35%

Control[c] : 4605 chrom. - 17 lethals or 0.37±0.09%

a = numbers in parenthesis represent the mutations appearing in clusters;

b = in these cases there were mass crosses in F_0 but individual in F_1 (see above the scheme);

c = X-chromosomes from wild populations tested in laboratory by Yu.N. Ivanov.

nonregular phenotypes:

non-disjunction of X-chromosomes	mutations of sn^* allele in F_0
in F_1 females:	(for example $sn^* \rightarrow sn^+$):
♀♀ $w^aB/sn^*/Y$ or "B non w^a".	♀♀ w sn^3/sn^+ or "+" ;
♂♂ w $sn^3/0$ (sterile)	♂♂ sn^+ or "+" .

The main data of experiments performed in 1976 are presented in Tables 1-3 (see also the report (Golubovsky & Erokhina 1977) in Russian). Four different alleles were tested. Two alleles sn^{11} and sn^{+10} represent the unstable derivatives of the original unstable mutant sn^{77}. They mutate according to the rule "all or none": from strong mutant expression to absolutely normal phenotypes and vice versa; so called the "A type"of instability. The other two alleles sn^{B8} and sn^{B14} are highly mutable derivatives of the original mutant sn^{63} having "B type" of instability: they have moderate mutant expression (hooked bristles) and mutate to the subliminal or pseudonormal one (slightly waved bristles)(Golubovsky 1978a & 1978b). The X-chromosomes containing four tested unstable alleles have lethal frequency about 1% (4 independent tests) that is considerably higher than the usual level of mutability 0.2-0.4% for X-chromosomes originated from wild fly samples (Table 1). The similar increased level of mutability was found for X-chromosomes which bear other unstable sn allele (Engels & Preston 1981; Raymond & Simmons 1981) or unstable allele of cut locus (Gerasimova 1981). As shown in Table 1, out of 13 chromosomes in which lethal and strong semilethal mutations occurred only 2 have carried the sn mutations. On the other hand about 90% of chromosomes with changed sn^* had neither lethals nor semilethals in other loci. Two simultaneous events at the same X-chromosome--sn mutation and lethal occurrence--were found no more than in 10% cases (2 out of 23). The lethals tested on Df(1)sn were localised outside the sn region, the similar picture was found in (Engels & Preston 1981; Raymond & Simmons 1981; Gerasimova 1981). The comparison of the numbers of apparent sn mutants in the male and female progeny of individual crosses ♀sn^*/Basc x w sn^3 ♂♂ also gave indirect evidence that lethals did not occure predominantly in those X-chromosomes where the unstable sn allele mutated. Such results confirmed by other authors (Engels & Preston 1981; Raymond & Simmons 1981; Gerasimova 1981) may be explained by remarkable feature of TE: replicative transposition. Namely they may stay in the original integration site but their replicated copy may be transposed to the other sites. By that process it is possible to explain the increase of total level of mutability in the absence of strict correlation with allelic

Table 2. Similarity of numbers of singed mutants in the male and female progeny of crosses ♀ sn^*/Basc x w sn^3 ♂. The indirect evidence that lethals did not occur predominantly in those X-chromosomes where the unstable sn allele mutated.

	male progeny		female progeny	
Allele	n	% sn mutat.	n	% sn mutat.
sn^{11}	1757	4.38	2119	4.25
sn^{+10}	5865	1.24	2625[a]	1.30
sn^{B8}	3739	0.51	4300[b]	0.79
sn^{B14}	1830	1.64	2100[b]	1.95

a= exact count of females given
b= estimated from sampling individual cultures.

Table 3. The increased rate of X-chromosome non-disjunction in the females heterozygous for unstable sn^*.
Crosses: ♀ sn^*/Basc /x w sn^3 ♂♂ .

Genotype	number of progeny	nonregular phenotypes ♀"B non w^a"	nonregular phenotypes ♂ w sn (sterile)	non-disjunction frequency x10^{+3}
Basc/sn^{11}	7400[a]	16	20	4.9
Basc/sn^{+10}	23500	30	78	4.6
Basc/sn^{B8}	15000	11	17	1.9
Basc/sn^{B14}	7300	8	22	4.1
Total	53200	65	137	3.8
Control				
Basc/+	3000	1	4	1.7

a= total was estimated by sampling individual cultures.

transition at the unstable sn. There are preferential sites of integrations for each mobile genetic element (Engels & Preston 1981). As for the elements like "copia" or "mobile dispersed genes" the average number of integration sites in D. melanogaster genome is about 50 or approximately 10-15 for the X-chromosome. Taking it into account we may suppose that the increase of X-chromosome mutability by 0.5% for one generation needs the rising of mutation rate for each of these 10-15 sites by hundreds of times. The X-chromosome non-disjunction frequency in females sn^*/Basc is also higher than in the control (Table 3). The hybrid dysgenesis systems induce the transposition of TE inherent to the genome and as a result the level of genic and chromosomal mutations is also increasing. From this point of view it is interesting that high levels of X non-disjunction had been shown for the I-R system of hybrid dysgenesis in the germ line of dysgenic F_1 females (Picard et al. 1978). Two possible explanations may be suggested: (1) the increased level of chromosome breakage, and (2) the sn locus may take part in the genetic control of chromosomal disjunction as it was established for w-z region (Robins 1981).

References: Golubovsky, M.D., Yu.N. Ivanov & M.M. Green 1977, PNAS 74:2973-2975; Green, M.M. 1977, PNAS 74:3490-3493; Golubovsky, M. & I.D. Erokhina 1977,Genetika (Russ.) 13:1210-1219; Golubovsky, M.D. 1978a, DIS 53:171; Golubovsky, M.D. 1978b, DIS 53:196; Engels, W. & Ch.R. Preston 1981, Cell 26:421-428; Raymond, J.D. & M.J. Simmons 1981, Genetics 98:291-302; Gerasimova, T.I. 1981, Mol.Gen.Genet. 184:544-547; Picard, G., J.C. Bregliano, A. Bucheton, J.M. Lavige & A. Pelisson 1978, Genet.Res. 32:275-287; Robins, L.G. 1981, Mol.Gen.Genet. 183:264-269.

Golubovsky, M.D. Institute of Cytology and Genetics, Novosibirsk, USSR.

Recessive sex-linked female-specific lethals at deltex locus discovered in natural populations of D. melanogaster.

Sex-specific lethals provide a remarkable means for analysis of sex determination and genetically controlled developmental processes (Baker & Ridge 1980). The recent extensive studies have shown (Belote & Lucchesi 1980): (1) such lethals are associated with a small number of loci, and (2) at the moment the majority of these mutants are male-specific; many of them are clustered at the centromere region of chromosome 2.

It is to be mentioned that such mutations are the component of sex-limited genetic load in wild populations (Dresher 1964). For example, the recessive mutation killing the males (mak-male killer, 2-54±) was first discovered in a natural population of Crimea, USSR (Golubovsky & Ivanov 1972), then the other allele (named mle) was found in the wild population from Japan (Fukunaga et al. 1975) and only the third allele was recently isolated during special extensive screening by chemical mutagenesis (Belote & Lucchesi 1980). The first male-killing lethal in chromosome 3 was also originated from a natural population (Uchida et al. 1981).

Taking into account the unequal deficit of female limited lethals, I report here the description of the mutation. It was isolated from nature in 1972 and studied briefly in 1973. In autumn 1972 together with Dr. R.L. Berg we investigated the phenotype of wild flies in several remote populations of USSR. We also searched the visible sex-linked mutations crossing all aberrant males with X-attached females. Among about 1300 males captured in Kashira population (about 100 km to the south from Moscow), we found two sex-linked mutations, yellow and "deltex vein," the latter first discovered by Dr.R.L. Berg as male having slight terminal deltas on veins. Unfortunately (but it turned out that it was lucky) at that time the common deltex mutation (dx) was absent in our laboratory. So I carried out the typical crosses for localisation of the founded vein mutation (let's designate it as dx^*). I combined dx^* with v and f markers and analyzed the progeny of crosses:

♀♀ dx^* v f /+ + + on ♂♂ dx^* v f. Results are shown at the left.

phenotypes	male progeny	female progeny
1. dx v f	57	0
2. + + +	55	51
3. dx + +	12	0
4. + v f	15	12
5. dx v +	20	0
6. + + f	23	13
7. dx + f	4	0
8. + + +	7	12
	193	88

The two facts are evident from these recombinational data: the lethality of the homozygous dx^*/dx^* females and localisation of dx^* in the region of 17 m.u. where deltex locus is placed. Then I received the "ec dx" line from Oregon stock