III- chromosome influence on II-chromosome nondisjunction, that gives the additional complexity to the male chromosome behavior in meiosis.

The elements of yeast site-specific recombination system FLP-FRT contain FLP-recombinase construct inserted in X- chromosome under a heat shock promoter and the vector P[ry^+; hs-neo; FRT] 80B inserted in 80B region of the 3L chromosome were described in Xu and Rubin (1993). The results of crossing of individuals containing different combinations of those elements to *y; C(2)EN; ru ca* females is shown in Table 1. It could be seen that most low level of II- chromosome nondisjunction takes place in the absence of FRT-sites. FRT homo- and heterozygous individuals give a higher level of II- chromosome nondisjunction in comparison to wild type (0.92×10^{-3} in accord to Ashburner's Table 27.8 [1989]) despite the presence of FLP-chromosome. This gives the evidence that one III- chromosome FRT-site is enough to ensure increased level of the III-chromosome nondisjunction, *i.e.* FRT is dominantly acting. The heat shock induction (40 min. at 37°C) of FLP-recombinase has no effect on the nondisjunction. This means that the phenomena could not be due to the site-specific exchanges in the FRT area.

Table 1.

Sex of flies	Genotype of tested individuals	Heat shock treatment	Number of embryos	Number of alive progeny	Frequency of nondisjunction
Male	FRT /*ry e*	—	2224	5	2.2×10^{-3}
Male	FRT /FRT	—	1149	8	7.0×10^{-3}
Male	*w* FLP; *ry e* / TM3	—	2427	1	0.4×10^{-3}
Male	*w* FLP; FRT / FRT	+	3752	5	1.3×10^{-3}
Male		—	1252	51	40.7×10^{-3}
	w FLP; FRT / TM3	—	2629	57	21.7×10^{-3}
Female		—	2903	0	0
	FRT / FRT	—	2237	0	0
Female	*w* FLP; FRT / FRT	—	2574	0	0
Female	*w* FLP; FRT / FRT	+	2563	1	0.4×10^{-3}

Dramatic increase of II- chromosome nondisjunction could be seen when FRT-site presents together with III-chromosome balancer TM3.

The phenomenon described here clearly shows that the behavior of different pairs of homologous chromosomes in male meiosis are not independent. Some cellular structures such as a spatial organization of chromatin or a spindle structure may be mediators of the observed interaction.

Similar experiments with female meiosis (Table 1) show no FRT induced nondisjunction.

Acknowledgments: The work done was supported by CDA-National Institute Health grant, USA.

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Chubykin, V., S. Fedorova, and L. Omelyanchuk.
Institute of Cytology and Genetics, Novosibirsk 630090 Russia. Chromosome abnormality in new mutant allelic to *aar* gene.

Earlier we have described some P[1ArB] insertion mutants, which demonstrated an increasing of lethality and mitotic abnormalities in the third instar larvae (Omelyanchuk and Volkova, 1996). One of those insertions, named as *v158*, was mapped in the 85F region of 3R chromosome by *in situ* hybridization. In result of cytogenetic analysis we have found an abnormal mitotic chromosome condensation and appearance of chromosome-like bodies, the quantity of which did not coincide with chromosome karyotype; nondisjunction of the X and the IV-chromosome also took place. It was shown (C. Sunkel, personal communication) that the *v158* mutant is an allele of the *aar* gene (Gomes, 1993). Further detailed analysis demonstrated characteristic mitotic defects of the *v158* mutant, that distinguish it from already known aar^1 and aar^2 alleles.

Mitotic chromosome preparations of neuroblasts and its C-band staining were made by standard techniques (Ashburner, 1989).

In Figure 1 A, B, C a positive C-staining of heterochromatic blocks is shown at different and sequential stages of mitosis in wild type strain (normal). In *Drosophila*, pairing of homologous chromosomes and nonhomologous association of asynaptic paracentromeric region in the chromocenter are both in meiotic and mitotic cells. At the late stages the pairing of heterochromatic regions and transition from nonhomologous bounds to homologous ones that orient

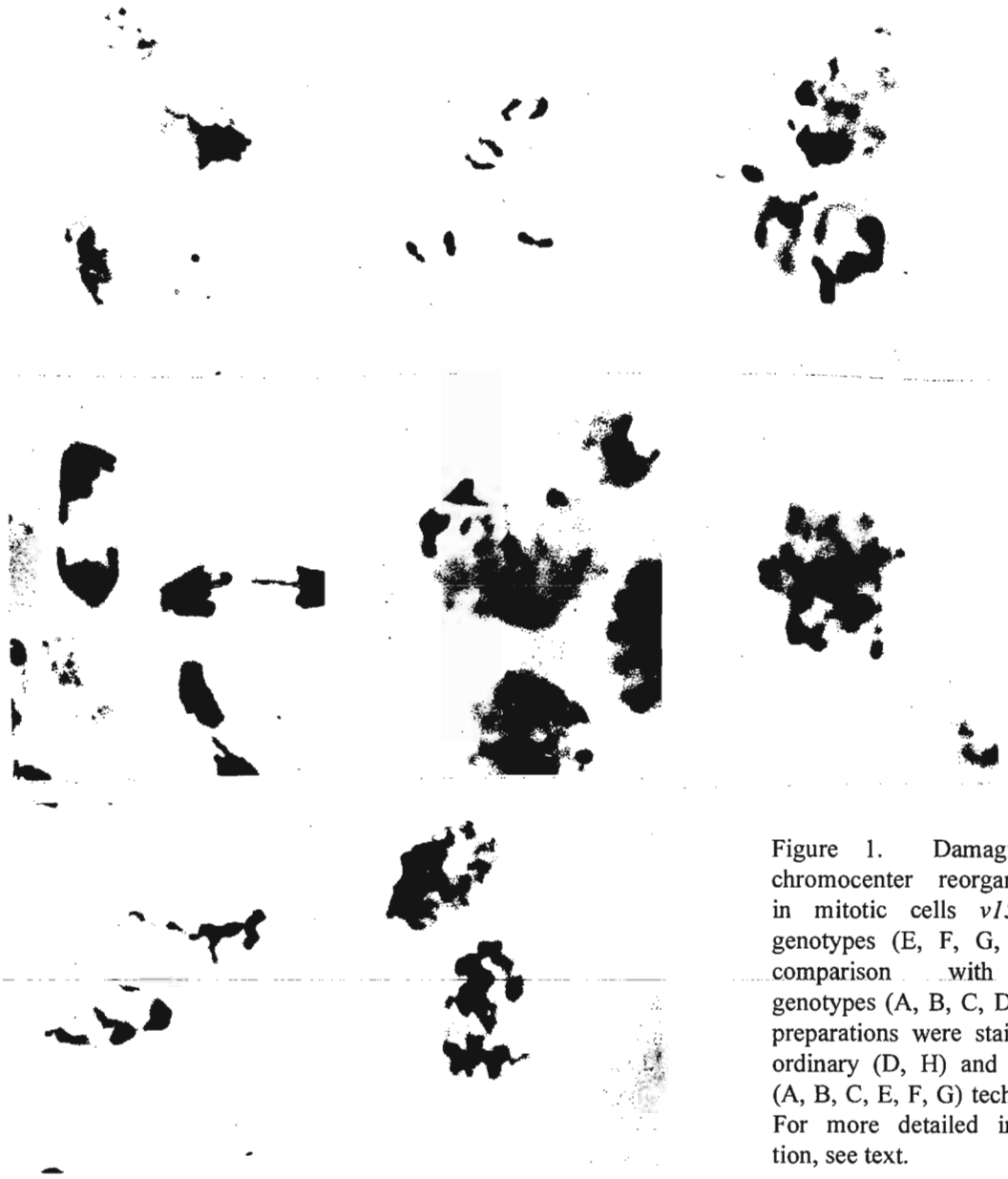

Figure 1. Damaging of chromocenter reorganization in mitotic cells *v158/v158* genotypes (E, F, G, H.) in comparison with +/+ genotypes (A, B, C, D). The preparations were stained by ordinary (D, H) and C-band (A, B, C, E, F, G) techniques. For more detailed information, see text.

the chromosomes in meiotic metaphase occur (Chubykin, 1996). In mitotic cells the reorganization of the chromocenter is terminated before microtubule attachment to kinetochores in prometaphase. The bounds between homologous heterochromatic regions in normal mitotic prometaphase are absent (Figure 1 B, C). Figure 1 E, F, G demonstrate heterochromatin in *v158/v158* individuals at similar mitotic stages which were determined by degree of compacting of chromosome euchromatic arms. In comparison with normal strain (Figure 1 A) the process of chromocenter restructuring in the *v158* homozygotes at the end of interphase is absent (Figure 1 E). Then, in prophase and prometaphase of the *v158* there are huge nonhomologous asynaptic heterochromatic blocks or associations which probably are formed by conservation of chromocentral bonds (Figure 1 F, G).

Table 1.

Genetic Constitution and Sex	Metaphase M	Anaphase A	Interphase I	M/A •10^{-2}	A/I • 10^{-3}
v158/v158					
females	41	19	4276	0.96	4.4
males	82	61	11334	0.72	0.54
v158/+					
females	62	56	2874	2.2	9.5
males	92	50	3664	2.5	13.6

Figure 2. Mitotic chromosomes containing isolated and more condensed chromatids without their division.

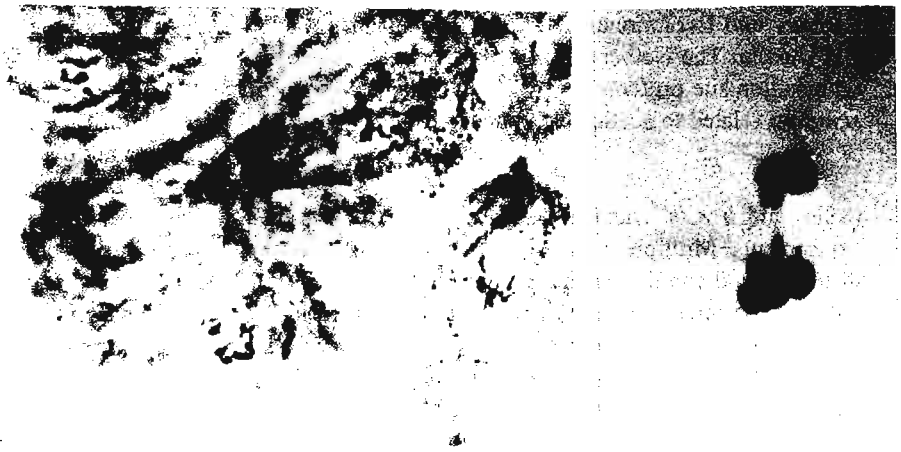

Figure 3. Asynchronous divisions (A, left) and nondisjunction of chromosomes (B, right) in meiosis of *v158/v158* male.

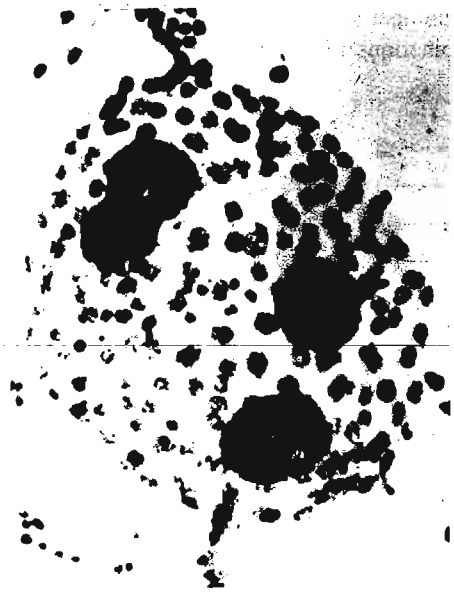

Figure 4. Damaging of ovarian development in *v158/v158* female.

Such abnormal associations are preserved during metaphase-anaphase and results in formation of fibrils, which connect the chromosomes (Figure 1 D, H).

Figure 2 illustrates C-banding of chromosome-like bodies in *v158/v158* individuals which are never observed in normal. Each such formation has a deeply stained heterochromatic region. Therefore we believe that a separate body can represent one or two paired chromatids: variations in its number can be explained to account for the difference in pairing ability.

Data of metaphase and anaphase indexes are presented in Table 1. Unlike the data of *aar* (Gomes, 1993), in *v158* these indexes are smaller in *v158/v158* than in *v158/+*. This corresponds to our data that the effect of *v158* allele is determined before metaphase in cell cycle.

The *v158* insertion is semilethal. Figure 3 illustrates asynchronous nuclear divisions (Fig. 3A) and nondisjunction of chromosomes (Fig. 3B) in meiosis of a homozygous male. Ovaries development are also defective. Figure 4 shows ovarian chamber with four nurse cells in the *v158*, while in normal there are 15.

The data obtained in this study demonstrate some unique mitotic abnormalities in the *v158* mutant, conditioned by heterochromatin behavior in prometaphase.

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