because of rapid autoagglutination of the cells. None of the vertebrate erythrocytes and the trypsin-treated mammalian cultured cells were agglutinated by the SF.

Our results indicate that the SF attaches to the surfaces of cell plasma membranes of insect cultured cells. The SF-attaching sites (cellular receptors) are restricted to certain surface areas of the plasma membranes. The agglutinating capacity of the SF suggests the existence on the SF molecule of polyvalent active sites which are responsible for the binding of the SF to cellular receptors on the plasma membrane. Since the SF greatly enhances NPV infection in vitro, our present findings strongly indicate that the SF acts in vitro as an enhancer in the attachment of cell-membrane-budded nucleocapsids of NPV to cultured insect cells as in the case of NPV enhancement in vivo[8-10]. This phenomenon may be analogous to the antibody-mediated enhancement of animal virus infections, such as the enhancement of the virus-specific antibody at a subneutralizing concentration in the attachment of the virus to Fc-receptor-bearing cells[15].

The NPVs are known to cause the hemagglutination of vertebrate erythrocytes[16-20]. Recently, Anderson et al.[21] found that the enveloped nucleocapsids of a GV of *Plodia interpunctella* agglutinated several species of vertebrate erythrocytes, in particular, rabbit erythrocytes. In our present study, the SF did not agglutinate the erythrocytes of several vertebrate species including that of the rabbit. Thus, the hemagglutinin present in enveloped nucleocapsids of *P. interpunctella* GV appeared not to be related to the SF.

The SF agglutinates not only lepidopterous insect cells but also those of mosquitoes and a cockroach. Since the enveloped arboviruses, i.e. *Alphavirus* group, multiply in the vector mosquito, the SF through its effective attachment may enhance the infection of these viruses in mosquito cells in vitro. Such a study may explain, in part, the variations in the efficiency of mosquitoes as vectors.

The basis for the attachment of the enveloped baculoviruses to the host plasma membrane to initiate fusion has not been clarified. Our previous study in vivo suggested that the phospholipids and ionic charges of the SF and viral envelopes were involved in attachment[22-24]. The SF appeared to enhance the attachment and/or fusion of the enveloped virion to the plasma membrane.

The question of virus receptor sites has not been resolved[25]. The adsorption of the SF to certain regions of the plasma membrane suggests the presence of receptor sites on the cell membrane. The attachment of the SF to the host cell membrane and its capacity to enhance the baculovirus infection provide us with a unique opportunity to further investigate this problem.

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The effect of X-rays and EMS on the behavior of the transposing element, TE98, in *Drosophila melanogaster*

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Summary. The sensitivity of TE98 (carrying w^{+R} and rst^+) to X-rays does not differ significantly from the mutability of *curled* and *karmoisin* loci. In addition no spontaneous mutants of TE98 were recovered, indicating its extreme stability. On the effect of EMS no *white* mutants were found supporting the view that the w^+ gene of TE98 is duplicated.

Several classes of transposable genetic elements are known in *Drosophila melanogaster*. Similarly to procaryotic transposons, they play an important role in spontaneous and induced mutational events[2]. The mutagen sensitivity of a transposable DNA sequence inserted adjacent to the *white* locus has been demonstrated[3]. Few data are available on the response to mutagenic agents of other *Drosophila* transposons. We report on the behavior of a large transposing element TE (in a position named TE98 in 3R) under conditions of induced mutagenesis.

In the course of a cytogenetic analysis of the 87A–C region of the 3rd chromosome, we isolated a large number of deficiencies induced by X-rays. In these experiments we used the transposing element *TE98* described by Ising and Block[4] as a genetic marker. This transposing element is cytologically visible in polytene chromosome preparations and it carries a piece of the X chromosome with the *white* (w^{+R}) and *roughest* (rst^+) genes. *TE98* is a derivative of *TE1* from which more than 100 new positions have been identified. The transposing elements

Table 1. Frequency of flies with mutant phenotypes in controls and after X-ray treatment

Chromosomal region examined	Number of flies examined	Number of flies with mutant phenotype	Mutation frequency
*cu**	62919	45	7.1×10^{-4}
*kar**	62919	48	7.6×10^{-4}
TE98	205975	176	8.5×10^{-4}
Control *cu** and *kar**	3919	–	–
Control *TE98*	82284	–	–

*Data are taken from Gausz et al.[9].

Table 2. Frequencies of flies with mutant phenotypes in controls and after EMS treatment

Chromosomal region examined	Number of flies examined	Number of flies with mutant phenotype	Mutation frequency
*cu**	8303	7	8.4×10^{-4}
*kar**	14029	13	9.2×10^{-4}
TE98	34495	2	5.7×10^{-5}
Control *TE98*	82284	–	–

*Data are taken from Gausz et al.[8].

TE28 and *TE98* are flanked by foldback elements of the FB family[5,6].

The cytological position of *TE98* is distal to the 87A4–5 band in the right arm of the 3rd chromosome (Gyurkovics, unpublished observation). In the presence of *w* on the X chromosome, *TE98*-carrying flies exhibit the wild type (red) eye color. However, when the transposing element is eliminated by a deletion, the loss of the w^{+R} allele results in white-eyed flies. If *TEs* are placed near to heterochromatin by inversion or translocation, this results in flies with white-variegated eyes. To obtain more data on the repsonse of *TE98* to mutagens, in addition to the X-rays, we also used ethyl methane sulphonate (EMS).

In X-ray experiments males carrying *w* on their X-chromosome as well as *TE98* on their 3rd chromosome *(w/Y; TE98* w^{+R} rst^{+}*/TM3)* were irradiated with a 4000 rad (1000 rad/min) dose. The treated males were crossed to *w spl/w spl; Sb/Ser* virgin females (for an explanation of genetic symbols see Lindsley and Grell[7]). In the F_1 progeny all the flies carrying the transposon exhibited a wild-type eye color, but in flies where the w^{+R} gene was mutated, excised or translocated to heterochromatin, the eyes were white or white-variegated, respectively.

EMS was used at a concentration of 0.025 M according to the method of Lewis and Bacher[8]. The crosses were identical with those described for the X-ray experiment.

For a comparison, we use our previous data obtained for the mutability of the *curled (cu)* and *karmoisin (kar)* loci[9,10].

The results of the X-ray experiments are summarized in table 1. The X-ray treatment induced changes affecting the w^{+R} marker with a frequency of 8.5×10^{-4}, whereas flies with *cu* and *kar* phenotype appeared with a frequency of 7.1×10^{-4} and 7.6×10^{-4}, respectively. These frequencies do not differ significantly from each other indicating that the sensitivity of *TE98* to X-rays is not higher than the value for induced visible recessive mutations. Of the 176 X-ray induced mutations 95 showed white and 81, variegated eyes. 32 of the white-eyed ones were identified as deletions in crosses with lethal mutations from the surroudning region. The remaining 63 mutants were lost due to sterility or reduced viability. 14 out of the 32 deletions were examined cytologically and 7 of them had one breakpoint in the 87A4–5 band, which is the insertion site of *TE98* (Gyurkovics, unpublished results).

After EMS treatment (table 2) mutations at the *cu* and *kar* loci were recovered with almost identical frequencies, 8.4×10^{-4} and 9.3×10^{-4}, respectively. Changes involving *TE98* appeared at a significantly lower frequency. Only 2 flies with white-variegated eyes were found among 34,495 progeny, which is a frequency of 5.8×10^{-5}, significantly lower than that for *cu* and *kar* loci. The variegated eye color indicates that these 2 mutations are probably the result of chromosome rearrangements. No *w* point mutations or deletions were isolated. Our inability to induce point mutations by EMS treatment supports the hypothesis of Ising and Block[4] that *TE1,* from which *TE98* is originated, contains a duplication for w^{+R}. Recently[5] it has been shown by whole genome Southern analysis of *TE98* that there are 3 bands of hybridization to single copy *white* sequences: 1 from the original location on the X chromosome, and 2 from *TE98*. Therefore it can be concluded that *TE98* carries a duplication of the *white* locus region.

The frequencies of spontaneous and induced loss of 2 other transposing elements *(TE6* and *TE30)* have been reported by Ising and Block[4] and by Ising and Ramel[11]. The red form of *TE30* is lost spontaneously with a frequency of 2×10^{-4}. The values for *TE6* vary between 3×10^{-4} and 8.3×10^{-3} depending on the genetic background. *TE98* is very stable; we did not find white-eyed flies among 82,284 tested F_1 progeny (frequency is less than 1.2×10^{-5}). In spite of the fact that *TE6* and *TE30* are less stable than *TE98* Ising and Ramel[11] were not able to influence the frequency of loss by X-rays, temperature shock or interchromosomal effects of inversions on recombination. The sensitivity of the transposing elements to these external influences is different compared to the results of Rasmussen et al.[3]. However, the fact that 7 out of the 14 cytologically analyzed deletions induced on *TE98* carrying chromosomes had one breakpoint adjacent to the transposing element, indicates that in spite of its relative stability *TE98* is a 'hot spot' for induced chromosome breaks. The other breakpoints are more randomly distributed. It is not excluded that some of these breakpoints may be connected with insertion sites of dispersed repeated elements and in this case the *FB-NOF* sequences in *TE98* may serve as portable regions of homology between the 2 breakpoints[12]. However, to test this possibility further studies are needed.

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