TABLE I

Distribution of Complement Fixing Antibodies to Equine Khinopneumonitis Virus in army horses and mules

Class of animal	No. of sera tested	C.F. Titre 1/4	1/8	1/16	Total No. of positive sera	Total No. of negative sera
Horses	333	30 (9·01)	12 (3·60)	2 (0·60)	44 (13·21)	289 (86·79)
Mules	105	1 (0·95)	2 (1·90)		3 (2·86)	102 (97·14)
Total	438	31 (7·08)	14 (3·20)	2 (0·46)	47 (10·73)	391 (89·27)

Percentages shown in parentheses.

the CF reaction showing a minimal non-haemolysis of 60 to 80% sheep erythrocytes by the visual observation, was considered to reveal the presence of CF antibodies, which agrees with the findings of Petzoldt[11]. Shimizu *et al.*[3] reported 1 : 4 and above as significant titre on the basis of 50% CF reaction in their study on serological survey of horse population in Japan. We used in our study ERV antigen (EHVI, Kentucky D Strain), received from Dr. T. Shimizu and therefore a CF titre of 1 : 4 and above was considered as an indication for the presence of CF antibodies. Besides, it has also been reported, that the titre for EHVI (ERV) may be affected on account of antigenic cross relationship with other herpes viruses, *viz.*, EHV2 and EHV3[6].

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INDUCED MUTATIONS IN RELATION TO HETEROCHROMATIN IN *DROSOPHILA MELANOGASTER*

THE X-chromosome of *Drosophila melanogaster* carries a large block of heterochromatin in the proximal part which extends at least up to the region designated as section 20 in the polytene maps[1]. This region of the chromosome is more prone to radiation induced breaks and lethal mutations[1]. The visible mutation frequencies are also higher for the genes located near this heterochromatic region[2–4]. In addition to this major heterochromatic block, the X-chromosome is having intercalary heterochromatic regions[5]. The present investigation was aimed at analysing the role of heterochromatin in the induction of sex-linked recessive visible mutations by 4 different mutagens in *D. melanogaster*.

The mutagens used included gamma rays and three alkylating agents, *viz.*, N-methyl-N'-nitro-N

TABLE I

Distribution of Recessive Visible Mutations scored at 13 gene loci in the 4 segments of X chromosome
[*Relative rates (%) in arenthesis*).]

Mutagens	Segment I *y–cv*	Segment II *cv–v*	Segment III *v–f*	Segment IV *f-centromere*	Total mutations	Total chromosomes tested
Gamma rays (3 kR)	30 (10·4)	30 (10·4)	98 (34·1)	130 (45·1)	288	21,085
MNNG (0·03M)	28 (12·6)	39 (17·5)	54 (24·2)	102 (45·7)	223	20·679
NEU (0·004M)	37 (16·7)	28 (12·7)	69 (31·2)	87 (39·4)	221	17,835
DEB (0·01M)	79 (26·1)	0	95 (31·3)	129 (42·6)	303	27,871

nitrosoguanidine (MNNG), N-ethyl-N-nitrosourea (NEU) and 1, 2 : 3, 4-diepoxybutane (DEB). Third instar larvae of the Oregon-K strain were fed on a medium containing the chemical mutagen or were exposed to gamma rays in a Co^{60} gamma irradiation source. The doses used were comparable as all of them gave 70–80% fertile males from the treated larvae. Sex-lined recessive visible mutations were scored using the attached X method. The details of experimentation are given elsewhere[6].

Mutants were obtained for 13 gene loci with varying frequencies. All these were tested and found to be allelic to the already known mutations[7]. In order to find out whether the mutations observed in this study were randomly distributed on the X-chromosome, the chromosome was divided into four segments each demarcated with known marker genes. The frequencies of mutations induced in these segments are shown in Table I. Chi-square analyses revealed that their distribution was non-random for all the four mutagens. When their rates were calculated, it was found that the *f*-centromere segment carried the maximum number of mutations and the segments located distal to the centromere showed a steady decrease. The *f*-centromere segment is characterised by the proximal heterochromatic block. It is plausible that the higher number of visible mutations obtained in segment IV could be due to the large block of heterochromatin in this region. This can be substaintiated with the fact that the heterochromatic regions show higher rates of both radiation and chemical induced breaks and mutations[2,4,8–10]. Fahmy and Fahmy[3] have also reported that bobbed locus located near the centromeric heterochromatin in the X-chromosome of *Drosophila* shows a considerably higher response to chemical mutagens compared to euchromatic genes.

Further evidence to this suggestion comes from comparing the distribution of mutations obtained in this study with the intercalary heterochromatic blocks. For this comparison, the cytological map of X-chromosome was divided into 20 equal sections and the distribution of heterochromatin was taken with respect to them[5]. The band positions of the gene loci mutated are assigned to these 20 sections. The mutations produced by gamma rays and the alkylating agents (pooled) are presented in Fig. 1. Cytological positions of all the mutated gene loci, whose band positions are known, coincide with the heterochromatic regions. Moreover, the highest mutation frequency was recorded in section 19 of the cytological map in Fig. 1, which is adjacent to the centromeric heterochromatic block. Thus, the observation that 11 of the 13 gene loci which mutated in the present study lie in the vicinity of heterochromatic regions, either centromeric or intercalary, points to the fact that presumably heterochromatin increased the mutagen response of genes located near them. Hardly any mutations could be scored in our study for genes located in the true euchromatic regions.

It may be hypothesised that heterochromatin influences the behaviour of genes in its proximity because of its structural peculiarities. This influence is more pronounced in the case of the centromeric heterochromatin. One of the explanations can be that these genes are the ones which are located at the junctions of euchromatic and heterochromtic blocks,

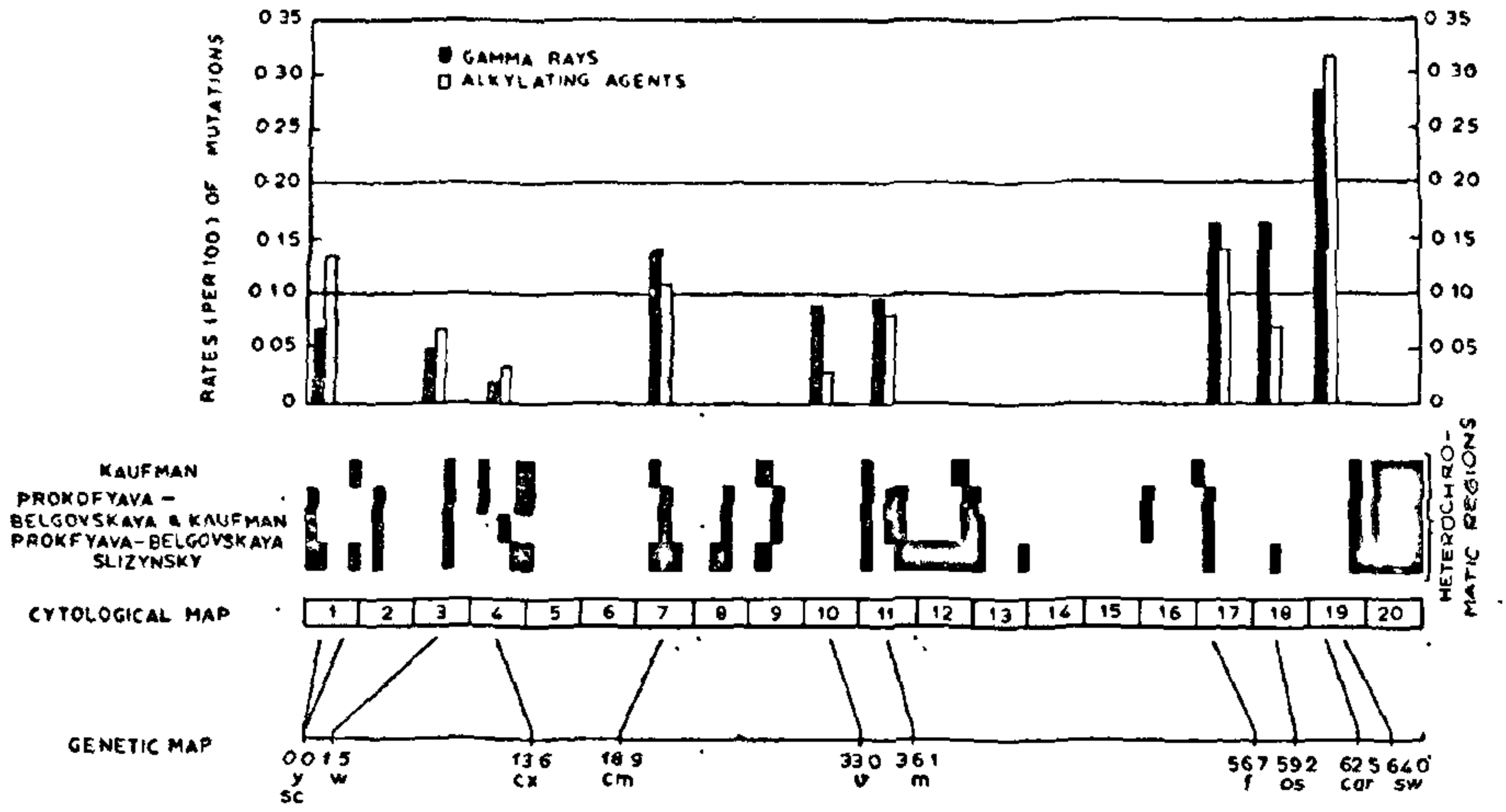

FIG. 1. Relationship between the distribution of heterochromatin and recessive visible mutation; on the X chromosome. (*y*, yellow; *sc*, scute; *cx*, curlex; *cm*, carmine; *v*, vermillion; *m*, ministures *f*, forked; *os*, outstretched; *car*, carnation; *sw*, short wing.)

Heterochromatic regions are known to be late replicating compared to the euchromatic portions[11]. This difference in the phase of replication may somehow make the genes at the junctions more prone to the action of mutagens. Further studies are required to explain the role of heterochromatin in induction of mutations.

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A NEW APPROACH TOWARDS DOUBLE GRAFTING IN MANGO

DOUBLE grafting or double working has been done successfully in temperate fruit-plants with a view to overcoming incompatibility between a desired variety and a good rootstock, transferring resistance to diseases and frost or inducing dwarf character and a strong framework without impairing the yield and quality of fruits[1].

Mango propagation although considered to be easy as compared to other tropical fruits, great difficulty was felt while preparing the plants for interstock trial using intermediate stem pieces. Some reports[2,3] have been published on double grafting/interstock trials on mango, but no attempts were made to