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Narda, R.D. and R.K. Gupta. Punjab Agricultural University, Ludhiana, India.
Mutation studies in D. melanogaster.

Role of protein synthesis in the induction of chromosomal aberrations was studied in the larvae of Oregon-K stock of D. melanogaster. Protein synthesis was inhibited by chloramphenicol (CPL) or streptomycin (ST), chromosomal aberrations were induced by ethylmethane sulphonate (EMS), methylmethane sulphonate (MMS), and hydrazine sulphate (HZ). For treatment with mutagen and/or the inhibitor, the larval period was divided into two halves. Chromosomes were examined for aberrations in fully grown third instar larvae.

EMS induced higher frequency of inversions when applied in the second larval half, whereas, MMS and HZ did so when applied in the first larval half. A few deletions were also induced by EMS and MMS and one translocation was induced by HZ (Table 1).

Regarding the effect of inhibition of protein synthesis on inversion frequency, excluding one case there was an overall increase in all treatments. This indicates that protein synthesis is involved in induction of chromosomal aberrations.

Further EMS, MMS and HZ induced maximum frequency of inversions (46.81%, 30.77% and 41.82% respectively) in 3L chromosome. In X-chromosome HZ induced higher frequency of inversions (13.62%) as compared to EMS (6.37%) and MMS (5.12%) and HZ induced not even a single

Table 1. Effect of protein inhibitors on the frequency of induced by EMS, MMS and HZ

Treatment	No. of larvae studied	No. of inversions scored	Frequency of inversions
NIL	95	0	0.0%
O + EMS	86	10	11.6%
CPL + EMS	107	13	12.1%
ST + EMS	90	5	4.3%
*EMS + O	85	7	8.2%
EMS + CPL	71	7	9.8%
EMS + ST	76	7	9.2%
O + MMS	70	8	11.4%
CPL + MMS	81	13	16.0%
ST + MMS	75	18	24.0%
**MMS + O	104	14	14.2%
MMS + ST	96	38	39.5%
MMS + ST	90	29	32.2%
***O + HZ	90	12	13.3%
CPL + HZ	70	19	27.1%
ST + HZ	70	11	15.7%
HZ + O	79	16	13.3%
HZ + CPL	75	30	40.0%
HZ + ST	75	25	33.3%

* 3 deletions were also induced
** 5 deletions were also induced
*** 1 translocation was also induced

inversion in the 2L chromosome in any of the treatments. The observed number of inversions induced by different mutagens in different chromosomal arms is non-random. Possibility that the number of aberrations induced on a chromosome depends upon its length is thus ruled out.

It was also revealed by the study of breakage-union points that EMS acts specifically at proximal end of 3L chromosome, MMS does so at the distal end of 2R and proximal end of 3R and HZ in the central one third 3L chromosome.

It is concluded that the type of spectrum of mutations induced by various mutagens is different and mode of action of each mutagen is specific in itself.