In *Drosophila melanogaster* translocations of all possible kinds were observed in 17.92% of the F_1 cultures (N = 1000) after irradiation of males with 4452 R of X-rays[21]. The majority of these translocations (12.36%) were such ones between the chromosomes 2 and 3. This last figure compares well with more recent data for 2–3 translocations. With a dosage of 4000 R of X-rays 14.39% were recovered[22] and with 3360 R X-rays 14.3%[23].

These values for total translocation production in *Drosophila* are only half or less of the values we have observed with *Culex*. It might be mentioned here that also in 2 other mosquito species, i.e. *Culex tritaeniorhynchus* and *Aedes albopictus*, the figures were of the same order of magnitude.

It is difficult to give an explanation for the exceptionally high values for translocation production in mosquitos. One possibility could be that the chromosomes in the sperm of mosquitos are in such a peculiar spatial position to each other that translocations can occur much more easily than in *Drosophila*. The very long and slender sperm head in mosquitos seems to indicate a position of the chromosomes parallel to each other. But this idea has to be discarded because the actual exchange of chromosomal segments occurs probably during karyogamy when the chromosomes have the possibility of free movement.

Another explanations seems to be more likely and is offered here as a working hypothesis. The mitotic chromosomes of *Drosophila* have a total length of 7.6 µm (I = 1.8 µm; II = 2.6 µm; III = 3.2 µm; IV, the dotlike chromosome, not taken into account), but the total length for *Culex* is 21.1 µm (I = 5.6 µm; II = 7.4 µm; III = 8.1 µm). Accordingly the total chromosome length of *Culex* is almost 3 times (2.8) that of *Drosophila*. The observed values of translocations show almost the same numerical correlation. Therefore the number of translocations which can be produced in an animal with a certain dosage of irradiation possibly depends on the total length of the chromosomes. This can only be taken as a very crude and approximate statement of a hypothesis, as it does not take into consideration other factors like volume and density of the chromosomes. Confirmation or rejection of the hypothesis needs comparative data from other objects besides *Drosophila* and *Culex*. It can be expected that such data become available in due time with the extension of the semisterility principle for control of other harmful insects[24].

Zusammenfassung. In Anbetracht der theoretischen und in einem Freilandexperiment bereits erwiesenen Möglichkeit der Bekämpfung von Schadinsekten durch Freilassung semisteriler Tiere wurde die Produktion von Translokationen und damit verbundener Semisterilität bei der Stechmücke *Culex pipiens* untersucht. Die Totalrate der erzeugten Translokationen liegt bei Stechmücken im Vergleich zu *Drosophila* auffallend hoch. Es besteht offenbar eine Korrelation zwischen der Gesamtlänge der Chromosomen und der Translokationsrate.

H. Laven and E. Jost

Institut für Genetik, Johannes-Gutenberg-Universität, D-65 Mainz (Germany), 6 January 1971.

[21] J. T. Patterson, W. S. Stone, S. Bedichek and M. Suche, Am. Nat. *68*, 359 (1934).
[22] I. I. Oster, in *Repair from Genetic Radiation Damage* (Ed. F. H. Sobels; Pergamon Press, Oxford 1963), p. 51.
[23] I. H. Herskowitz, Genetics *42*, 375 (1957).
[24] We want to thank Prof. H. Traut (Münster) for very helpful discussions of radiobiological aspects of translocation production.

The Specific Localization of Diethyl Sulphate-Induced Second Chromosome Recessive Lethal Mutations in *Drosophila melanogaster*

The current interest in the specificity for gene mutation shown by chemical and physical mutagens on eukaryotic microbial systems[1], has encouraged us to look for specificity in the distribution of recessive lethal mutations along the second chromosome of *Drosophila* after treatment with diethyl sulphate.

Material and methods. Newly-hatched wild type (Oregon-K) *Drosophila* males were treated with 0.5% diethyl sulphate for 48 h by an adult feeding method[2]; they were then, individually mated to 2 CyL[4]/Pm females, for 3 days, as the first mating for the detection of second chromosome recessive lethal mutations[3]. The sampling procedure used allows the recovery of germ cells which were present as spermatozoa over the period of the treatment, and consequently lethals arising in the brood sample from individual males, do so independently of one another, i.e. they do not occur by clonal origin. However, in order to be sure of the independent origin of the lethals, only 1 lethal was taken from anyone male. Moreover, to eliminate the presence of spontaneous lethals which might have been present before treatment, males which gave approximately equal or greater numbers of lethal to non-lethal cultures in the F_3, were not used.

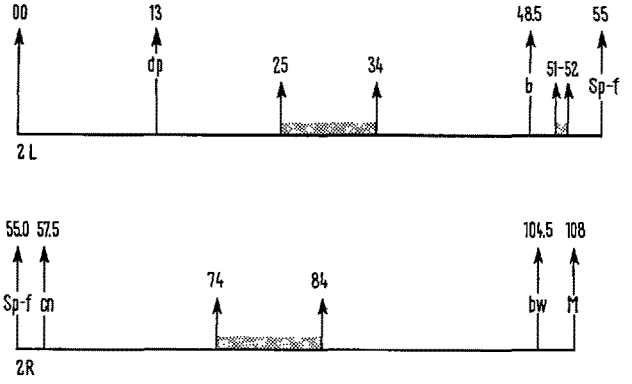
Results and discussion. The treatment induced 225 lethals in 884 chromosomes tested from 91 males, i.e. 25.4% second chromosome recessive lethal mutations. 47 of the 91 males yielded 1 or 2 lethals, but it was possible to examine only 38 out of the 47 lethals chosen owing to loss during maintenance. Semi-lethal mutations were excluded.

Tests of the 38 lethals for allelism in all possible pairwise combinations (703 separate crosses) showed that 18 of the lethals were non-identical (non-allelic); all these crosses showed the usual 2:1 phenotypic ratio expected from non-allelic crosses[3]. The majority of the remainder of the lethals behaved, when crossed with certain others, in a manner expected from a series of overlapping deficiencies, i.e. a X b and b X c show allelism, whilst a X c show non-allelism. 4 of the lethals showed identity by the allelism tests. These latter 20 lethal stocks were again tested for allelism 3 more times with the same results, but now including also reciprocal crosses to exclude the type of synthetic lethal reported by Batten and Thoday[4]. Thus, the lethals

[1] T. Alderson and M. J. Hartley, Mutation Res. *8*, 255 (1969).
[2] M. Pelecanos and T. Alderson, Mutation Res. *1*, 173 (1964).
[3] C. Auerbach, *Mutation*, Part I: Methods (Oliver and Boyd, Edinburgh 1961).
[4] J. L. Batten and J. M. Thody, Heredity *24*, 445 (1969).

may be divided into 3 groups; 18 non-identical, 4 identical and 16 'partially' identical. The lethals from all 3 groups were next mapped on the chromosome by separately crossing individuals (CyL^4/lethal) from each lethal stock in mass culture to a stock homozygous for the recessive visible second chromosome markers, dumpy wing (dp), black body (b), cinnabar eye (cn) and brown eye (bw); these markers span both arms of the chromosome (Figure). Phenotypically wild type F_1 females (lethal/dp, b, cn, bw) from each cross were mass mated to CyL^4/lethal males from their own lethal stock to produce F_2 wild type progeny which have either the non-crossover lethal/dp, b, cn, bw genotype or the lethal/dp, b, cn, bw, where one or more of the visible markers has been lost by crossingover with the lethal chromosome in the F_1 female (crossover types). The occurrence of a non-crossover or a crossover type in these phenotypically wild type individuals is determined by separately test crossing males, in which crossing-over does not occur to dp, b, cn, bw females. The position of a lethal on the chromosome is obtained from the occurrence of both reciprocal recombinants in the region in which the lethal lies, and the absence of one or other of the reciprocal recombinants in other marked regions. The mapping of the lethal between the markers flanking its position is determined from the relative frequency of each reciprocal recombinant in the progeny. The standard error of a location was calculated by the formula of Reeve (NAFEI and AUERBACH[5]), namely, S.E. = $(a - 1)\, x\, (1 - b)/n$, where a and b are the loci of the outside markers limiting the segment in which the lethal is found, $(a - 1)$ and $(1 - b)$ are the distances of the lethal to these loci, and n is the number of males scored in the relevant segment. The locations reported in the present paper have standard errors of between 0.6–0.8 cM[6].

The striking result from the mapping of the diethyl sulphate-induced lethals is the localization of all lethals at one or more of 3 distinct regions along the chromosome. Each of the 4 identical lethals map at each of 3 regions, 25–34 cM, 51–52 cM, and 74–84 cM, that is, each behaves from its mapping as a triple lethal. The 18 non-identical lethals all map at either or both of 2 regions; 7 map between 25–34 cM, 8 map between 74–84 cM, and 3 map as double mutants at both the 25–34 cM and the 74–84 cM regions. The partially identical lethals map in a similar way to the non-identical lethals, 5 between 25–34 cM, 7 between 74–84 cM and 4 as double mutants at both the 25–34 and 74–84 cM regions. The behaviour of the partially identical lethals by test of allelism is compatible with their localization, that is, they map in the positions necessary for their grouping as particular sets of partially identical lethals.



Distribution of lethals along the second chromosome. The areas between 2 arrows indicate the regions in which the lethals were localized. Scale: 1 centimorgan = 2 mm. cM = centimorgans, and are units of crossing-over.

It is to be noted that the 25–34 cM region lies in the middle of the left arm of the second chromosome (2L), and that of the 74–84 cM region in the middle of the right arm (2R), whilst the 51–52 cM region is close to the heterochromatic region (Sp-f, spindle fibre attachement of the Figure). It is also perhaps of interest that all three regions in which the lethals map include positions of rather intense puffing activity in the salivary gland chromosomes of the Oregon-K stock[6].

It has not been possible to study the association of the lethals with chromosome aberrations since the lethals were lost during our move from Thessaloniki to Patras. However, no disturbance of the normal linkage relationships of the markers used has been observed in the mappings of the lethals. It is hoped to repeat and extend the work in view of the striking results obtained.

Résumé. La localisation des mutations récessives létales produites par le diéthyl-sulphate sur le second chromosome de la Drosophile, a montré que ce mutagène possède une action remarquablement spécifique. Ainsi toutes les mutations se localisent dans trois régions. D'autre part, l'analyse par «le test d'allélisme» de 38 mutations létales provenant indépendamment l'une de l'autre a permis de distinguer 3 groupes de létaux: létaux identiques, non-identiques et «partiellement» identiques.

M. PELECANOS[7]

Department of Genetics, University of Patras (Greece), 25 September 1970.

[5] H. NAFEI and C. AUERBACH, Z. VererbLehre *95*, 351 (1964).

[6] M. ASHBURNER, Chromosoma *21*, 398 (1967).

[7] Acknowledgments. Part of this work was carried out at the Department of Biology, University of Thessaloniki, Greece. I thank Prof. A. KANELLIS for providing the laboratory facilities. Thanks are due also to O.E.C.D. (Organisation for Economic Cooperation and Development) for financial support as well as to Prof. J. M. THODAY, F.R.S., Department of Genetics, University of Cambridge, England, for allowing me to use the Department's library.

Laich-Räubern und -Kannibalismus bei sympatrischen Anuren-Kaulquappen[1]

Material und Methode. Von der Feststellung ausgehend, dass Kaulquappen von *Rana temporaria* in einem Kunstweiher Laich von verschiedenen sympatrischen Anurenarten verzehren[2], wurden vom April bis im Juli 1970 unter vereinheitlichten Bedingungen 147 Versuche mit Kaulquappen von *Rana temporaria, R. ridibunda, Bufo calamita, Hyla arborea* und *Bombina variegata* versus Laich von *Rana esculenta, Bufo calamita, Hyla arborea* und *Bombina variegata* durchgeführt.

Die Kaulquappen verschiedener Altersstadien wurden artweise in Plastikgefässen mit 10 l Wasser im Freien gehalten. Der 0–3tägige, befruchtete Laich wurde in