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LOCALIZATION OF THE STRUCTURAL GENES FOR ARGININE KINASE AND GLYCERALDEHYDE-3-PHOSPHATE DEHYDROGENASE IN *DROSOPHILA MELANOGASTER*

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Liang-Ju Fu and Glen E. Collier (1982) Localization of the structural genes for arginine kinase and glyceraldehyde-3-phosphate Dehydrogenase in *Drosophila melanogaster*. *Bull. Inst. Zool., Academia Sinica* **22**(1): 25-35. Arginine kinase and glyceraldehyde-3-phosphate dehydrogenase are two important enzymes for energy metabolism in the flight muscle of most insects. The structural genes of these two enzymes in *Drosophila melanogaster* were cytologically localized based on gene dosage dependency. Region 66B/C-66F/67A of the left arm of the third chromosome affected arginine kinase. Region 50C-51A2 or region 52A12/B1-52E of the right arm of the second chromosome affected glyceraldehyde-3-phosphate dehydrogenase.

Gene mapping is usually done by recombination analysis of allelic variants. However, in some cases, no allelic variants are available, and genes can only be mapped by other methods. Segmental aneuploidy is such an alternative method for gene mapping. This method was first developed by Lindsley and sandler *et al.* in 1972. They produced Y-autosome translocation flies [T(Y:A) flies] by X-ray irradiation. When two T(Y:A) stocks with adjacent autosomal breakpoints are crossed, progenies can be produced that are either duplicated or deleted for the region between the breakpoints (Fig. 1). If the structural gene of a certain enzyme is dosage sensitive, the duplication aneuploid, containing three doses of this gene, will show 1.5± times as much enzyme activity as its diploid sibling (two doses). On the other hand, the deletion aneuploid (one dose) will have only half the enzyme activity of its diploid sibling.

Segmental aneuploidy has been extended to include X-chromosome by Stewart and Merriam in 1974. This method has been demonstrated to be useful and precise for gene localization by some known gene-enzyme systems (O'Brien and Gethmann, 1973), and has been sucessfully used to localize the structural genes for dopa decarboxylase (Hodgetts, 1975), trehalase (Oliver *et al*, 1978), sucrase (Oliver and Williamson, 1979) and some other enzymes (Hall and Kankel, 1976, Lubinsky and Bewley, 1979; O'Brien and Gethmann, 1973; Stewart and Merriam, 1974; Voelker *et al*, 1979).

This study was trying to use segmental aneuploidy to map the structural genes for arginine kinase and glyceraldehyde-3-phosphate dehydrogenase (GAPDH). The former enzyme is involved in the immediate supply of energy for the initiation of flight, and the latter one is involved in the constant supply of energy for the continuation of flight in insects.

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MATERIALS AND METHODS

Drosophila stocks

Stocks carrying Y-autosome translocations were from the *Drosophila* Stock Center, California Institute of Technology. Stocks carrying X:Y translocations were kindly supplied by Dr. John Merriam, UCLA, and Dr. Ross MacIntyre, Cornell University.

All the flies were grown on the standard cornmeal-agar-molasses medium at 24–26°C.

Each cross was made by putting 10–20 virgin females and 20–30 appropriate males into a vial or a bottle. The parents were transferred into a new vial or bottle every five to eight days. Progenies were collected every 12 hours, and aged for three to six days. The aneuploids and euploids of the same sex and the same age were compared for their eneyme activities and protein concentrations.

Production of aneuploids

Second and third chromosome: The production of duplication and deletion aneuploids has been described previously (Lindsley *et al.*, 1972; Stewart and Merriam, 1974) (Fig. 1).

In the initial screen, the T(Y:A) parents were chosen, so that each of the recovered duplications contains an average duplicated segment of chromosome of about 150 salivary gland chromosome bands long (we call these the duplications for the large regions). No viable deletions were recovered from these crosses because *Drosophila* is quite sensitive to segmental deficiency. Haploidy for 1 percent of the genome is frequently viable, but the upper limit that flies can tolerate is about 3 percent of the genome (Lindsley *et al.*, 1972). In the initial screen of our study, the deletions would be haploids for 9 percent of the genome in the average, which is much beyond what flies can tolerate.

After the large regions, which are dosage sensitive for each of the two enzymes, were found, another set of crosses was made for arginine kinase to further dissect the large region into smaller regions. It was not possible to further dissect the dosage sensitive region for GAPDH by segmental aneuploidy, because T(Y:A) stocks with breakpoints within this region were not available at the present.

X-Chromosome: X:Y translocations can be used similarly to produce aneuploids duplicated for parts of the X-chromosome. In this study, two different crosses were made for double check. In the first cross, virgins with attached X-chromosomes homozygous for ywf (y: yellow, w: white, f: forked) were crossed to X:Y translocated males with a free Y-chromosome. In the second cross, the same type of virgins were crossed to T(X:Y) males without a free Y-chromosome. The aneuploids recovered from these two crosses were duplicated for either the distal or the proximal half of the X-chromosome.

Fourth Chromosome: Virgins with compound fourth chromosome were crossed to males with normal fourth chromosomes. This cross produced triple-4 and haplo-4 progenies. The enzyme activities and protein concentrations of the triple-4 and haplo-4 males were compared to those of males of the same constitution as their paternals.

Sample preparations and enzyme assays

Arginine kinase: Tissue homogenates were made by homogenizing one fly per 0.1 ml of homogenization buffer. Flies were homogenized singly or in sets of five in 1 ml hand glass homogenizer. The crude homogenates were centrifuged for three minutes in a Beckman Microfuge. The clear supernatant was used for enzyme assay and protein determination.

The homogenization buffer was: 0.1 M glycine NaOH buffer, PH 8.6, 5 mM magnesium acetate, 50 mM potassium chloride and 5 mM dithiothreitol.

The assay for arginine kinase activity is as described by Wallimann and Eppenberger (Wallimann and Eppenberger, 1973). The method involves coupling the reaction of arginine kinase to pyruvate kinase (PK) and lactate dehydrogenase (LDH).

$$\text{Arginine} + \text{ATP} \xrightarrow{\text{Arginine Kinase}} \text{Arginine-P} + \text{ADP}$$

$$\text{ADP} + \text{Phosphoenolpyruvate} \xrightarrow{\text{PK}} \text{ATP} + \text{Pyruvate}$$

$$\text{Pyruvate} + \text{NADH} \xrightarrow{\text{LDH}} \text{Lactate} + \text{NAD}^+$$

Arginine kinase activity is detected by a decrease in absorbance at 340 nm as NADH is oxidized to NAD^+.

The assay mixture contains the following final concentrations of components: 5 mM magnesium acetate, 50 mM potasium chloride, 0.1 M glycine NaOH buffer, PH 8.6, 2.5 mM arginine, 0.7 I. U./ml PK and 1 I. U./ml LDH.

All the components in the assay mixture, except arginine, have to be incubated together for at least 15 minutes before they could indicate the presence of arginine kinase activity correctly. Activity was measured during the fourth minute of the assay, because the rate of decrease in absorbance was steady after the third minute of assay.

Glyceraldehyde-3-phosphate Dehydrogenase (GAPDH): Tissue homogenates were made in the same manner as for arginine kinase.

Homogenization buffer was: 0.02 M sodium phosphate buffer, PH 7.1, 5 mM dithiothreitol and 1 mM EDTA.

The assay for GAPDH activity is as described by Carlson and Brosemer (Carlson and Brosemer, 1971).

$$d\text{-GA-3-P} + \text{Pi} + \text{NAD}^+ \xrightarrow{\text{GAPDH}} \text{1, 3-diphospho-}d\text{-glycerate} + \text{NADH}$$

GAPDH activity is detected by an increase in absorbance at 340 nm as NAD^+ is reacted to NADH.

The assay mixture was: 0.02 M sodium phosphate buffer, PH 7.1, 13 mM sodium arsenate, 0.3 mM NAD^+, 5 mM dithiothreitol, 1 mM EDTA and 2 mM glyceraldehyde-3-phosphate.

All the enzyme activities were assayed in a Beckman Model 35 recording spectrophototometer at 25°C, the temperature was controlled by Haake circulating water bath.

Protein concentrations were estimated by the method described by Lowry *et al.* (Lowry *et al.*, 1951).

At least two replicates were made for each set of aneuploids and euploid siblings. The average of all replications is reported. The ratio of activities between the duplication-bearing aneuploids and the euploids is reported both on a per fly and specific activity bases.

RESULTS

Table 1 lists all the crosses made in the initial screen for the detection of dosage sensitive regions for arginine kinase and GAPDH. These crosses produced duplication-bearing aneuploids for all of the regions of the genome of *Drosophila melanogaster* except region 83DE, which contains an aneuploid-lethal locus (Lindsley, *et al*, 1972). In Table 1, we can see an increased arginine kinase activity on region 64E-67C, and an increased GAPDH activity on region 50C-52E.

Flies carrying duplications for region 64E-67C show significantly higher activity of arginine kinase than their euploid siblings both on per fly and specific activity bases. We have made reciprocal crosses for this region, *i.e.*, B141×G122 and G122×B141, to make sure that the elevation of activity occurred in both male and female duplication-bearing aneuploids. This turned out to be the case. These results indicated that the structural gene for arginine kinase is probably located within this region.

In order to localize the dosage sensitive locus for arginine kinase to a more precise location, region 64E-67C was further dissected into smaller regions. Fortunately, several T(Y:3) stocks with breakpoints within this region were available, and the results from this set of crosses were listed in Table 2 and diagrammed in Fig. 2. It is obvious that region

TABLE 1

Arginine kinase and glyceraldehyde-3-phosphate dehydrogenase activities of segmental aneuploids

Parents[a]	Duplicated region	Arginine kinase[h]		GAPDH[h]	
		act. dup./eup.[i]	sp. act. dup./eup.[j]	act. dup./eup.	sp. act. dup./eup.
+[b]×J96	21A-25A	1.00	0.98	0.98	0.94
J96×H52	25A-27E	0.90	1.10	0.95	0.73
H52×(L52×+[b])	27E-30F	1.22	1.06	0.97	1.04
L52×R15	30F-35BC	0.90	0.99	0.98	1.01
B110×(R15×+[b])	35BC-38C	0.95	1.08	0.96	0.96
B177×B110	38C-41	0.93	0.92	1.15	1.07
D20×R155	40-43C	0.90	0.91	0.94	1.08
R155×L23	43C-45F	0.74	0.96	0.96	1.06
B107×L23	45F-47E	0.89	0.87	0.85	0.95
B107×L110	47E-50C	0.88	0.91	1.03	0.65
R14×L110	50C-52E	1.20	1.06	1.39[k]	1.28[k]
L110×R14	50C-52E	—	—	1.52[k]	1.41[k]
H149×R14	52E-54F	1.04	1.09	0.92	0.89
L107×H149	54F-57B	1.04	1.04	1.14	1.13
P59×L107	57B-59B	1.12	1.14	0.98	1.01
+[b]×P59	59B-60F	0.81	0.80	0.96	0.98
B234×+[c]	61A-64E	0.88	0.91	0.92	0.92
B141×G122	64E-67C	1.26[k]	1.32[k]	1.07	1.08
G122×B141	64E-67C	1.36[k]	1.60[k]	—	—
G122×H156	67C-70C	0.95	1.03	0.98	1.09
H156×D228	70C-74A	0.77	0.80	1.16	1.03
J162×D228	74A-79D	0.81	0.81	0.81	0.82
A112×J162	76F-79D	0.86	1.05	0.91	0.87
L132×(J162×+[c])	79D-83CD	0.78	1.11	0.79	0.96
L136×R36	83EF-86B	0.91	0.66	0.93	1.10
G48×R36	86B-88C	1.10	1.12	0.74	0.98
A89×G48	88C-91B	0.99	0.99	1.05	1.10
B93×A89	91B-93F	0.87	1.12	0.88	0.95
G73×*B*93	93F-96A	1.13	1.19	0.97	0.95
R128××G73	96A-97F	0.93	1.00	0.94	1.09
+[d]×R128	97F-100F	0.98	0.98	1.00	0.98
C(4)[e]×ci, ey^{R}[f]	101A-102F	0.90	0.75	1.09	1.04
C(4)×ci, ey^{R}	101A-102F	1.00	0.87	1.14	0.94
ywf[g]×B26	1A-9C	0.76	0.98	0.79	1.29
ywf×B26	9C-20F	0.75	0.82	1.09	1.29
ywf×J8	1A-8C	0.99	0.73	1.03	0.98
ywf×J8	8C-20F	0.75	0.63	0.99	0.68

a. Unless otherwise indicated, all female parents were of the genotype C(1)RM, y/T(Y:2)/Cy or C(1)RM, y^{2}bb/T(Y:3)/In(3LR)TM6, Ubx^{67}e and the male parents were Y^{s}X.Y^{L}, In(1)EN, y/T(Y:2)/Cy or Y^{s}X.Y^{L}, y/T(Y:3)/In(3LR)TM6, Ubx^{67}e, where Cy is either In(2L+2R)Cy, Cy cn^{2} or In(2LR)SM1, al^{2} Cy cn^{2} sp^{2}.

b. Y^{s}X.Y^{L}, In(1)EN, y/Y^{s}X.Y^{L}, In(1)EN, y; In(2LR)SM1/Sco.

c. Y^{s}X.Y^{L}, In(1)EN, y; In(3LR)TM6, Ubx^{67b}e/Sb.

d. Y^{s}X.Y^{L}, In(1)EN, y/Y^{s}X.Y^{L}, In(1)EN, y; In(3LR)TM6, Ubx^{67b}e/Sb.

e. C(4)RM, ci ey^{R} gv1 sv^{a}.

f. ci ey^{R}/ci ey^{R}.

g. C(1)RM, ywf.

h. All activities and specific activities were the average of at least two multiple fly extracts or three single fly extracts.

i. Ratio of activity in duplication vs. activity in euploid sibling of the same sex and same age on per fly basis.

j. Ratio of specific activity in duplication vs. specific activity in euploid sibling of the same sex and same age on per mg soluble protein basis.

k. These data are the average of six single fly extracts.

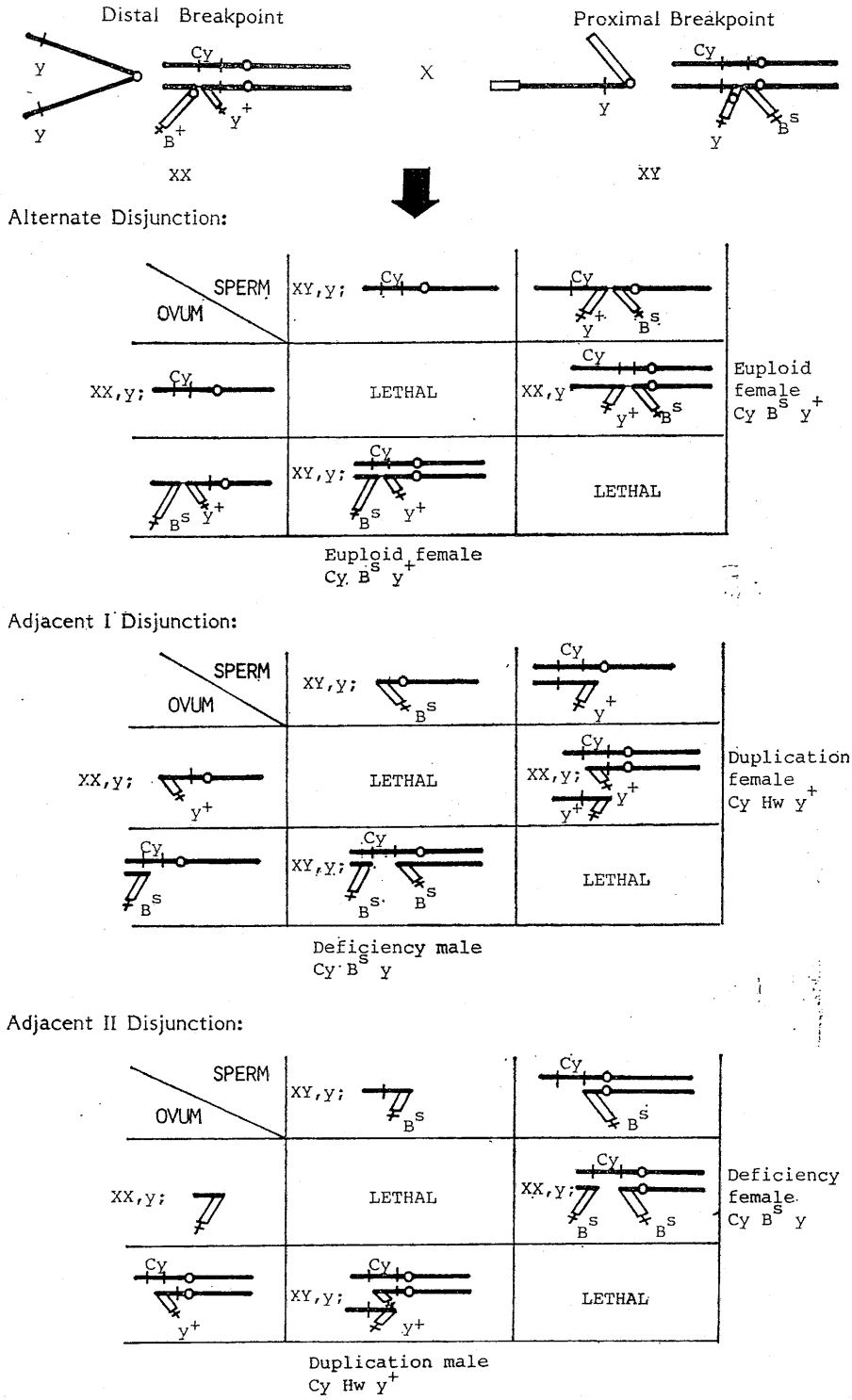

Fig. 1. Production of deficiency and duplication aneuploids by crossing two Y-autosome translocations, T(Y:A) stocks, with adjacent autosomal breakpoints. Only the zygotes from alternate, adjacent I and adjacent II disjunctions are shown. All other combinations are lethal.

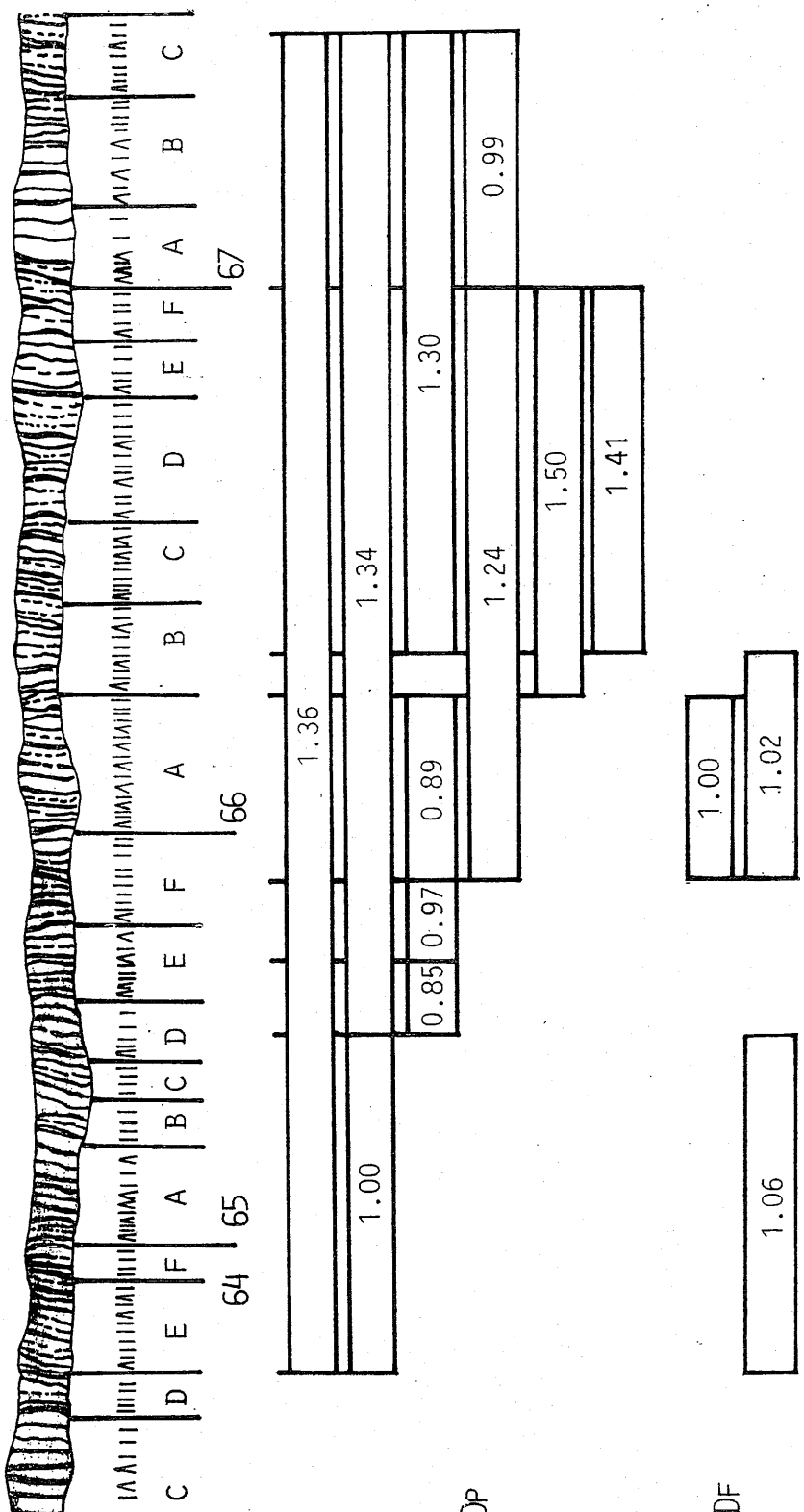

Fig. 2. Localization of the structural gene for arginine kinase within region 64E–67C. The drawing is a representation of the salivary gland chromosome map for region 64E to 67C of the left arm of chromosome III. The boxes below the chromosome represent the regions duplicated (D_P) or deleted (D_F) in the segmental aneuploids produced by the crosses listed in Table 2. The number in the boxes represents the ratio of activity in aneuploids vs. euploids.

66B/C-66F/67A is dosage sensitive, and therefore, it probably contains the structural gene for arginine kinase.

On the other hand, the dosage sensitive region for GAPDH appears to fall within region 50C-52E. Flies duplicated for this region have significantly higher activity than their euploid siblings both on per fly and specific activity bases (Table 1). Again, we made reciprocal crosses for this region, and did see the elevation of GAPDH activity in both male and female duplication aneuploids. In addition to region 50C-52E, the duplications for either the distal or the proximal half of the X-chromosome from the cross of *ywf*×B26 also show significantly higher specific activity than their euploid siblings (dup/eup=1.29), but have approximately equal levels of activity on per fly basis. This observation can be explained because the duplications have much less protein than the euploids. We have tested this assumption with another cross, ywf×J8, which also produced duplications for the distal and proximal half of the X-chromosome. The results from this cross (Table 1) indicated that the X-chromosome is not dosage sensitive for GAPDH.

Region 50C-52E cannot be further dissected by the segmental aneuploidy method because there are no T(Y:2) stocks with breakpoints within this region. Recently, Baker and Ridge (Baker and Ridge, 1980) induced a number of Lobe revertants by X-ray treatment. Two of these proved to be deficiencies with breakpoints within the 50C-52E region. Dr. MacIntyre of Cornell University has made one of these available to us, BL L^{+R4}. The region deleted in this stock is 51A2-52A12/B1. Euploids and flies bearing this deficiency have almost equal amount of GAPDH (Table 3). Therefore, the structural gene for GAPDH is not located in this region. It is either in region 50C-51A2 or

TABLE 2
Genetic dissection of region 64E through 67C for the screening of the structural gene for arginine kinase

Parents[a]	Aneuploid region	Arginine kinase activity[c]	
		dup./eup.	def./eup.
P50×B141	64E-65D	0.94	0.83
P50×G71	65D-67C	1.34	—
B186×P50	65D-65E	0.85	—
B186×J128	65E-65F	0.97	—
H138×J128	65F-66B	0.89	1.00
G130×J128	65F-66BC	0.90	—
G130×(G122×+[b])	66BC-67C	1.30	(d)
G71×G122	67C-67C	0.87	—
H167×G71	66F/67A-67C	0.92	—
H167×(G122×+[b])	66F/67A-67C	0.99	—
(+[b]×H167)×G130	66BC-66F/67A	1.61	(d)
G130×(+[b]×H167)	66BC-66F/67A	1.41	(d)
H138×(+[b]×H167)	66B-66F/67A	1.50	—
J128×(+[b]×H167)	65F-66F/67A	1.24	—

a. All female parents were C(1)RM, y^2bb/T(Y:3)/In(3LR)TM6, Ubx^{67b}e and all male parents were $Y^sX.Y^L$, y/T(Y:3)/In(3LR)TM6, Ubx^{67b}e.

b. $Y^sX.Y^L$, In(1)EN, y; In(3LR)TM6, Ubx^{67b}e/Sb.

c. All activities were the average of at least four single fly extracts, and were compared on per fly basis.

d. No deficiency has ever recovered out of approximately 250 offsprings of these crosses.

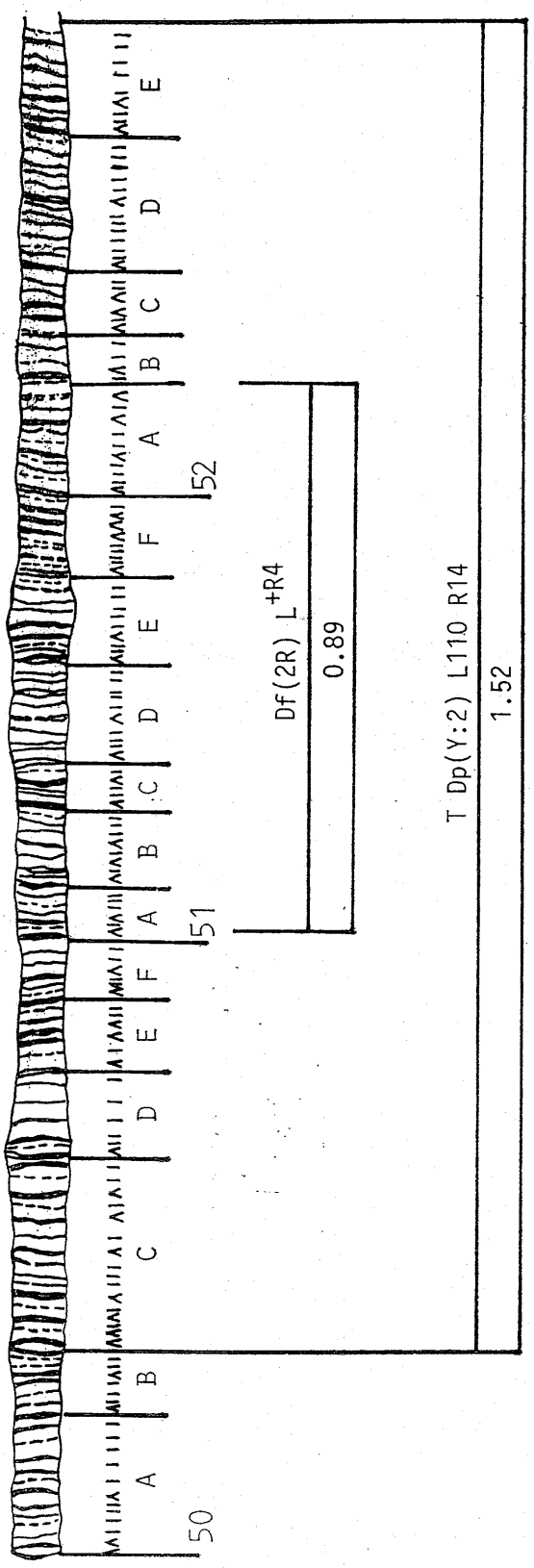

Fig. 3. Further dissection of the dosage sensitive region 50C–52E, for GAPDH. The drawing is a representation of the salivary gland chromosome map for region 50C to 52E of the right arm of chromosome II. Other than the segmental duplication produced by crossing T(Y:2) L110 and T(Y:2) R14, the only available chromosomal aberration for this region is an X-ray induced revertant of Lobe (Baker, 1980). It is a deficiency for 51A2 to 52A12/B1. The number in the boxes represents the ratio of activity in aneuploids vs. euploids.

TABLE 3
Comparison of the GAPDH activity between two new mutant stocks and wild Type flies

Stocks	Activity	Activity ratio	Specific activity	Specific activity ratio
$Df(2R)L^{+R4}$	0.78		0.26	
vs.		0.89		0.90
A/A wild type	0.88		0.29	
α-GPO^{hi}	1.38		0.58	
vs.		1.00		0.92
A/A wild type	1.38		0.63	

in region 52A12/B1-52E (Fig. 3). The localization can be tested as soon as other deficiencies for this region or appropriate T(Y:2) translocation stocks are available.

DISCUSSION

The results indicated that there is only one dosage sensitive region in the *Drosophila melanogaster* genome for arginine kinase, and it has been shown that there is only one molecular form of arginine kinase in *Drosophila melanogaster* (Wallimann and Eppenberger, 1973). These two results confirm each other.

The results also indicated that there is only one dosage sensitive region for GAPDH. This finding is also supported by the observation that after electrophoretic analysis, there is only one stained region for GAPDH on cellulose acetate membranes (Collier, unpublished data).

The probable location for the structural gene of arginine kinase is wihin region 66B/C-66F/67A, and that for GAPDH is either in region 50C-51A2 or in region 52A12/B1-52E. The cytological locations of these genes can be further narrowed by testing induced deficiencies within these regions. These deficiencies can be isolated by methods similar to those of Akam *et al.* (1978) or of Baker and Ridge (1980). Although it is very likely that a dosage sensitive region for an enzyme contains the structural gene for that enzyme, it is not conclusive evidence for the structural gene identity, because this region might contain a regulatory gene instead of the structural gene. Such a case was reported by Detweiler and MacIntyre (Detweiler and MacIntyre, 1978). They found two dosage sensitive regions for acid DNase, but only one has been proved to contain the structural gene for this enzyme. The other did not, it is very possible that this region contains a regulatory gene. Further evidence can be obtained by the analysis of electrophoretic variants, by analysis of null alleles that produce immunological crossreacting material (Bell and MacIntyre, 1973) and interallelic complementation (Bell *et al.*, 1972; O'Brien and MacIntyre, 1972).

Even though no electrophoretic variants are known for arginine kinase, if a deficiency for the dosage sensitive region was induced or constructed from stocks G130 and H167 (Pye *et al.*, 1980), it could be used in a cross scheme to detect recessive variants induced by a mutagen such as EMS (Lewis and Bacher, 1968).

Among these EMS-treated flies, if one fly is heterozygous for a wild type allele and a recessive null mutation, we will not be able to detect this mutant gene through the zymogram. The mutant band will be masked by the normal arginine kinase band. However, if we cross this fly to one heterozygous for a deficiency which includes the arginine kinase gene, the zymogram of their progeny will reveal the recessive mutant by the presence of a very faint band or by the absence of a band in the respective gel region. These results are due to F_1 flies heterozygous for the deficiency and the mutant allele for arginine kinase.

Similar schemes have been used successfully for α-glycerophosphate dehydrogenase (O'Brien

and MacIntyre, 1972), alcohol dehydrogenase (Grell *et al.*, 1965; Johnson and Denniston, 1964), acid phosphatase (MacIntyre, 1966; Morrison, 1973), and DNase (Detweiler and MacIntyre, 1978). If a convenient spot test (as for acid phosphatase or DNase) is developed for arginine kinase or GAPDH, then a similar cross scheme could be used to produce "null" mutants. In addition to confirming the location of the structural genes for these two enzymes, these mutants would be valuable in assessing the physiological role, genetic regulation, and molecular evolution of these enzymes.

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REFERENCES

AKAM, M. E. and D. B. ROBERTS (1978) Drosophila: the genetics of two major larval proteins. *Cell* **13**: 215-225.

BAKER, B. S. and K. A. RIDGE (1980) Sex and the single cell. I. On the action of major loci affecting sex determination in *Drosophila melanogaster*. *Genetics* **94**: 383-423.

BELL, J. B. and R. J. MACINTYRE (1973) Characterization of acid phosphatase-1 null activity mutants of *Drosophila melanogaster*. *Biochem. Genet.* **10**: 39-55.

BELL, J. B., R. J. MACINTYRE and A. P. OLIVIERI (1972) Induction of null activity mutants for the acid phosphatase-1 gene in *Drosophila melanogaster*. *Biochem. Genet.* **6**: 205-216.

CARLSON, C. W. and R. W. BROSEMER (1971) Comparative structural properties of insect triose phosphate dehydrogenase. *Biochemistry* **10**: 2113-2119.

DETWEILER, C. and R. J. MACINTYRE (1978) A genetic and developmental analysis of an acid deoxyribonuclease in *Drosophila melanogaster*. *Biochem. Genet.* **16**: 1113-1134.

GRELL, E. H., K. B. JACOBSON and J. B. MURPHY (1965) Alcohol dehydrogenase in *Drosophila melanogaster*. *Science* **149**: 80-82.

HALL, J. C. and D. R. KANKEL (1976) Genetics of acetylcholinesterase in *Drosophila melanogaster*. *Genetics* **83**: 517-535.

HODGETTS, R. B. (1975) The response of dopa decarboxylase activity to variations in gene dosage in *Drosophila*: A possible location of the structural gene. *Genetics* **79**: 45-54.

JOHNSON, F. M. and C. DENNISTON (1964) Genetic variation of alcohol dehydrogenase in *Drosophila melanogaster*. *Nature* **204**: 906-907.

LEWIS, E. B. and F. BACHER (1968) Method of feeding ethyl methane sulfonate (EMS) to *Drosophila* males. *Drosoph. Inf. Serv.* **43**: 193.

LINDSLEY, D. L., L. SANDLER, B. S. BAKER, A. T. C. CARPENTER, R. E. DENELL, J. C. HALL, P. A. JACOBS, G. L. GABOR MIKLOS, B. K. DAVIS, R. C. GETHMAN, R. W. HARDY, A. HESSLER, S. M. MILLER, H. NOZAWA, D. M. PARRY and M. GOULD-SOMERO (1972) Segmental aneuploidy and the genetic gross structure of the *Drosophila* genome. *Genetics* **71**: 157-184.

LOWRY, O. H., N. J., ROSEBROUGH, A. L. FARR and R. J. RANDALL (1951) Protein measurement with the folin phenol reagent. *J. Biol. Chem.* **193**: 265-275.

LUBINSKY, S. and G. C. BEWLEY (1979) Genetics of catalase in *Drosophila melanogaster*: Rates of synthesis and degradation of the enzyme in flies, aneuploid and euploid for the structural gene. *Genetics* **91**: 723-742.

MACINTYRE, R. J. (1966) The genetics of an acid phosphatase in *Drosophila melanogaster* and *Drosophila simulans*. *Genetics* **53**: 461-474.

MORRISON, W. J. (1973) Cytological localization of acid phosphatase-1 in *Drosophila melanogaster*. *Isozyme Bull.* **6**: 13.

O'BRIEN, S. J. and R. J. MACINTYRE (1972) The α-glycerophosphate cycle in *Drosophila melanogaster* II. Genetic aspects. *Genetics* **71**: 127-138.

O'BRIEN, S. J. and R. C. GETHMANN (1973) Segmental aneuploidy as a probe for structural genes in *Drosophila*: Mitochondrial membrane enzyme. *Genetics* **75**: 155-167.

OLIVER, M. J. and J. H. WILLIAMSON (1979) Genetic and biochemical aspects of sucrase from *Drosophila melanogaster*. *Biochem. Genet.* **17**: 897-907.

OLIVER, M. J., R. E. HUBER and J. H. WILIAMSON (1978) Genetic and biochemical aspects of trehalase from *Drosophila melanogaster*. *Biochem. Genet.* **16**: 927-940.

PYE, Q., KNIPPLE, D. and R. J. MACINTYRE (1980) Construction of segmental deficiency stocks from Y-autosome translocation stocks. *Drosoph. Inf. Serv.* **55**: 170.

STEWART, B.R. and J.R. MERRIAM (1974) Segmental aneuploidy and enzyme activity as a method for cytogenetic localization in *Drosophila melanogaster*. *Genetics* **76**: 301-310.

VOELKER, R.A., S. OHNISHI and C.H. LANGLEY (1979) Genetic and cytogenetic studies of the malate dehydrogenase of *Drosophila melanogaster*. *Biochem. Genet.* **17**: 947-956.

WALLIMANN, T. and H. M. EPPENBERGER, (1973) Properties of arginine kinase from *Drosophila melanogaster*. *Eur. J. Biochem.* **38**: 180-184.

果蠅體內兩種酵素的基因定位

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果蠅 *Drosophila melanogaster* 體內兩種與供應飛行所需能量有關的酵素 Arginine kinase 及 Glyceraldehyde-3-phosphate dehydrogenase 的基因已分別被初步定位。Arginine kinase 的基因可能位於第三條染色體左臂 66B/C-66F/67A 的部位。Glyceraldehyde-3-phosphate dehydrogenase 的基因可能位於第二條染色體右臂 50C-51A2 或 52A12/B1-52E 的部位。