DROSOPHILA GENMAPS DATABASE REPORT FORM

Return to John Merriam, UCLA Biology, Los Angeles CA 90026-1606. FAX [redacted]

660

Doa FBrf0075186

location: 78F1-2 keys for this paper:

Gene/mutant name: DARKENER OF APRICOT

abbreviation: Doa

biochemical name/product: ?

(1) CLONES:

kb_genomic: ~30 vector/library name: λEMBL3

kb_cDNA: vector/library name:

METHOD:

prior clone: walk:

expression: differential screen:

microclone: oligonucleotide: PCR?

homology: transposon tag:

(2) REARRANGEMENTS: $Df(d^{RX1})$ IS A Doa ALLELE!

↳ HAZELRIGG & KAUFMAN 1983 GENETICS

(3) TRANSFORM INSERTS:

(5) SEQUENCE: NONE YET

kb_genomic: accession number:

kb_cDNA: accession number:

ORF-size:

ORF_function/homology group:

ORF_domains:

(6) TRANSCRIPT DESCRIPTION:

transcript_size:

distribution:

(7) PROTEIN ANTIBODY:

distribution:

(8) LAC Z FUSION STAIN:

distribution:

MISC_INFO: INFO: CONTACT: LENNY RABINOW

[redacted]

EMAIL: RABINO[redacted]

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DROSOPHILA GENMAPS DATABASE RECORD PRINTOUT 6/21/92

Return corrections to John Merriam, UCLA Biology, Los Angeles, CA 90026-1606; FAX [redacted]

source_number 660

full_location 98F1,2

table_numbers 1 cross_locations

gene_name Darkener of apricot

gene_abbrev Doa

product dosage sensitive suppresso

1 kb_cloned_genom 30 0 vector_genom λ DASH2

kb_cloned_cDNA 5 0 vector_cDNA λgt10/Bluescript

METHOD: expression walk_jump_org xxx YACs 0

microcloning oligonucleotide

homology transposon_tag

clonetechtext walk from Dfd breakpoint directly into Doa locus

2 rearrangement

3 T_strain

markers

5 kb_seq_genomic 0 0 acc_number_gen kb_seq_old 0

kb_seq_cDNA 2.7Kb 0 acc_number_cDNA

comments sequencing still on going, not yet submitted

ORF_size

ORF_function

ORF_domains

6 kb_trans_size ~2.7, ~4.4

transc_dist spatial unknown//embryonic, 3rd instar, PN PNE adults all express

7 protein_dist

8 fusion_dist

misc_info

full citation Rabinow L; Birchler JA; A dosage-sensitive modifier of retrotransposon-induced alleles of the Drosophila white locus. EMBO J 1989:[illegible]879.

[redacted]

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keywords EMAIL [redacted]/RABINOW [redacted]

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UCLA 660

SANTA BARBARA • SANTA CRUZ

December 20, 1990

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John Merriam
UCLA Drosophila Genmaps Database

DEPARTMENT OF BIOLOGY
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FAX [redacted]

Dear Colleagues:

I would like to use information from the clone list to construct a "physical map" of the Drosophila genome. You have (a) clone(s) listed, as described below, so I would like to enlist your help. What I need for this project are the names and locations of chromosome rearrangement breakpoints that lie within the region of your clone/walk. Mapping breakpoints is the most reliable way of connecting the molecular with the cytological scales.

As you know, the clone list has been useful to Drosophila workers in finding starting points for their walks. The database is organized by references, so that the original cloning paper from a lab is cited as key #1, with subsequent papers cited by subject (key #5 is sequence information, for example). The clone list is therefore also a gene-by-gene literature index. The clone list is now mounted on the Indiana University BioArchive computer bulletin board in the FLY subdirectory. Use Internet address 129.79.1.101 and your local File Transfer Program.

Please take a few minutes to complete this form and return to me by FAX or mail. It will be most useful if you can update your clone sizes and make more exact your clone locations. Make any other changes you want also. List the rearrangements and which breakpoints you have identified. Any cytologically visible rearrangement (deletion, inversion, translocation, transposition, duplication) is useful. Please indicate if the same citation is the best reference, or indicate a better one (or a personal communication).

I thank you for your help as always. With your prompt and generous support I [redacted] to show you the results in March at the Chicago fly meeting.

List Rearrangements :	Breakpoint found:	Other breakpoints:

CHANGE or CONFIRM indexing for original clone cit'n: Locations __ Clone sizes __ Names/Products __

Rabinow, L; (1989) EMBO Journal 8:879

Location	Key	kb gen/cDNA	Gene name (abbreviation) product rearrangements...
98F1,2	1	10 0	dosage sensitive suppressor (doa)

RECORD NO 1933 REF NO. 660 ash

24/87

e

Location 98 F1, 2

6. Table Num 0 (1) 2 3 4 5 6

7. Clone Name

8. Genes Info doa & dosage sensitive suppressor

9. Stock Orgn

10. KB Size

11. Cross Ref

12. Misc Info

13. Reference

14. Breakpoint

15. BPCont1

16. BPCont2

17. T Strain

18. Markers

19. DIS Vol

20. Comments

21. KB Sequenced

22. Coordinates

please return to:

John Merriam

Biology Department
Univ California
Los Angeles, CA 90024

FAX # [redacted]

Robinow and Bridges

Biological Labs
Harvard
Cambridge MA 02138

key to Table num for this record: 0 = new data and ref on one previously entered, 1 = clone, 2 = rearrangement breakpoint, 3 = insert, 4 = walk, 5 = sequenced, 6 = transcription
multiple entries ok (ie 1,2,4,5,6)

Fax ref

(66)

...ow, Birchler
...logical Laboratories
...rvard Unviersity
Cambridge, MA 02138

Rabinow and Birchley (1989) 11th Euro

RABINOW, L. + BIRCHLER, J.A. 1989 EMBO J., 8, 879-889

98F1,2 — CLONING DATA BELOW IS VERY PRELIMINARY

_____ location changes

_____ kb per location?

GENOMIC genomic or cDNA?

key details of cloning method

JUMP FROM Dfd, BREAKPOINT DIRECTLY INTO Dfd LOCUS walk/jump

NO INFORMATION YET ON STRUCTURE/EXPRESSION etc. homology

_____ transposon tag

_____ expression, etc

_____ oligonucleotide

_____ other (specify)

Is the reference correct? (changes?) SEE ABOVE

Your name LEONARD RABINOW

telephone number [redacted]

FAX number [redacted]

Please return to John Merriam, Biology Department,
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[redacted]

Journal vol.8 no.3 pp.879 - 889, 1989

may relate to 660

Dosage-sensitive modifier of retrotransposon-induced alleles of the *Drosophila white* locus

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Communicated by M.Ashburner

The *apricot* allele of the *white* locus results from the insertion of the retrotransposon *copia*. Mutations in a newly discovered locus, the *Darkener-of-apricot* (*Doa*), suppress w^a and some of its revertants. Of 44 other *white* alleles tested, only w^{sp55} is affected by *Doa*, although, in contrast, it is enhanced by *Doa* mutations. The *Doa* locus modulates w^a and w^{sp55} expression as a function of its own dosage. Mutations in *Doa* are dominant suppressors or enhancers and are recessive lethals. Rare *Doa* mutant homozygotes escaping lethality demonstrate extreme phenotypic suppression of w^a and enhancement of w^{sp55}. RNA from w^a is substantially wild-type in structure in escapers, although reduced in quantity.

Key words: copia/Drosophila/enhancement/retrotransposon/suppression

Introduction

Insertion of transposable elements is a major cause of spontaneous mutation in eukaryotic organisms (Bender *et al.*, 1983; Modolell *et al.*, 1983; Levis *et al.*, 1984; Silverman and Fink, 1984; Campuzano *et al.*, 1986; Nusse, 1986; Morse *et al.*, 1988). Similarities between certain transposable elements and mammalian retroviruses have been extensively noted, and include sequence homologies, overall organization of encoded genes (Scherer *et al.*, 1982; Shiba and Saigo, 1983; Kugiyama *et al.*, 1983; Mount and Rubin, 1985; Marlur *et al.*, 1986), and the replication and transposition through RNA intermediates of the yeast Ty element (Boeke *et al.*, 1985), and apparently the *Drosophila copia* transposon (Flavell, 1984; Kikuchi *et al.*, 1986). These similarities have led to the term retrotransposon to describe transposable elements that replicate and transpose through an RNA intermediate (Boeke *et al.*, 1985). As opposed to retroviruses, however, retrotransposons have not been reported to have intercellular mobility.

Second-site modifiers that enhance (increase), or suppress (decrease) the severity of the phenotype associated with transposable element insertion have been described in yeast and *Drosophila* (Modolell *et al.*, 1983; Winston *et al.*, 1984a, 1987; Kubli, 1986; Fassler and Winston, 1988). Mammalian mutations due to the insertion of retroviruses an also be affected by mutations in second-site modifiers (Levy *et al.*, 1982, 1985; Traina-Dorge *et al.*, 1985). Second-site modifier genes may define functions encoded by host genes, utilized by the transposon. For example, many *Drosophila* retrotransposons are differentially expressed during development, implying responsiveness to developmentally regulated cues (Schwartz *et al.*, 1982; Parkhurst and Corces, 1987). Several mutations affecting Ty and solo-LTR insertions in yeast have both specific and/or pleiotropic phenotypes, implicating them as modifiers of gene expression (Winston *et al.*, 1984a,b; Clark-Adams and Winston, 1987; Neigeborn *et al.*, 1987; Clark-Adams *et al.*, 1988).

Suppressor and enhancer mutations almost certainly define a number of different processes, since many of them are additive in their effects (Mount *et al.*, 1988; Rutledge *et al.*, 1988; Rabinow and Birchler, in preparation), or have differing effects on the transposons acted upon. For example, mutations in *SPT3*, *SPT7* and *SPT8* genes suppress the effects of Ty and delta (Ty-LTR) insertions, and also change the pattern of transcription from delta elements (Winston *et al.*, 1984b, 1987). Mutations in the *SPT6* gene have no effect on the transcription of Ty elements, although they suppress at least one of the same mutations suppressed by *SPT3* mutations (Clark-Adams and Winston, 1987).

In *Drosophila*, several recessive mutations in second-site modifiers have been described which affect specific retrotransposon insertions at various loci (Green, 1959; Modolell *et al.*, 1983; Parkhurst and Corces, 1985; Chang *et al.*, 1986; Searles and Voelker, 1986). The mode of action of these modifiers has been examined in a few cases, with effects seen on the RNA of the transposable element, and/or on the product of the locus into which the element has inserted. One of the characterized retrotransposons, *gypsy*, is responsible for the mutations y^2 and f^1, among others (Modellel *et al.*, 1983). These alleles are suppressible by mutations in *su(f)* and *su(Hw)*, both of which partially return transcript levels of the host gene to normal (Parkhurst and Corces, 1985, 1986a,b).

The role of these modifier loci in normal *Drosophila* gene expression and development remains unclear, although at least two of these genes are vital to the fly. The *su(f)* locus can mutate to a recessive or hemizygous lethal form (Dudick *et al.*, 1974; Russell, 1974), while some alleles of *su(Hw)* are sterile in homozygous females (Lindsley and Grell, 1969). A third modifier locus, the *su(w^a)*, has been cloned and autoregulates its expression at the level of splicing (Chou *et al.*, 1987; Zachar *et al.*, 1987). Its gene product is thought to play a role in the production of *copia* RNA, although no pleiotropic or specific effects on host gene expression have yet been reported.

Here we describe the identification and characterization of the *Doa* locus, a second-site modifier of w^a with previously undescribed properties. *Doa* mutants are dominant suppressors of the *copia* insertion allele, w^a, while extra wild-type copies of the *Doa* locus enhance w^a. All mutant *Doa* alleles are associated with recessive lethality. Finally, the RNA originating from w^a is drastically altered in both quantity and quality in rare homozygous *Doa* mutants escaping lethality.