THE GENOME OF *Drosophila melanogaster*

Dan L. Lindsley Georgianna G. Zimm

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We dedicate this book to the memory of George Lefevre in recognition of his exhaustive cytogenetic analysis of the X chromosome and in gratitude for his many helpful comments on the manuscript version of this revision prior to his untimely death in January, 1990.

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PREFACE

The last twenty years have witnessed a remarkable expansion in the definition of the Drosophila genome. The emergence of Drosophila as an organism of choice for molecular-genetic investigations of eukaryotic biology has attracted a large number of talented workers to the field, and the rapid advances in molecular technology have provided new and sophisticated tools and generated novel kinds of information.

This work is a revision of "The Genetic Variations of *Drosophila melanogaster*" by D. L. Lindsley and E. H. Grell, which appeared in 1968 and was essentially a complete catalogue of mutations and chromosome rearrangements of *Drosophila melanogaster* as of the end of 1966. The present volume purports to be such a catalogue current until the end of 1989. The illustrations are primarily the work of Edith M. Wallace, the artist employed by T. H. Morgan; they were mostly drawn between 1920 and 1940. The same illustrations were used in "The Genetic Variations of *Drosophila melanogaster*" and its predecessor "The Mutants of *Drosophila melanogaster*" by Calvin B. Bridges and Katherine S. Brehme. At the time of the 1968 publication, genes were identified exclusively through the existence of mutant alleles; the only wild-type alleles considered were electrophoretic variants of a few enzymes, and the only gene for which there was any molecular information was *bb*. Amino-acid and nucleotide sequencing and the polymerase chain reaction were concepts for the future; cloning of DNA sequences had not been imagined; transposable elements, hybrid dysgenesis, and transformation were unsuspected. These technological advances have shifted the emphasis to normal gene structure and function rather than exclusive consideration of mutant alleles. This shift in emphasis is reflected in the title of the present volume, "The Genome of *Drosophila melanogaster*." The ability to identify a gene from either its protein product or the homologous product from another species, rather than the converse, has led to the discovery of many new genes for which no variant had been previously recognized. In addition, new genes with interesting expression patterns are being discovered in enhancer-detection lines.

Interim versions of the majority of the material contained herein have appeared in the form of volumes 62, 64, 65, and 68 of Drosophila Information Service. This volume contains information on upwards of 4000 genes and 9000 chromosome rearrangements. There are categories of effects, little if at all represented in the 1968 edition, that have assumed major proportions in the present version. These include developmental mutations, behavioral mutations, female-sterile mutations, meiotic and mitotic mutations, *Y*-autosome translocations, and transposable elements; in addition, many regions of the genome have been subjected to saturation mutagenesis so that large numbers of lethally mutable loci have been identified and deficiency mapped. A major consequence of the mapping efforts, utilizing both chromosome rearrangements and *in situ* hybridization, is that the polytene map has displaced the recombination map as the more useful standard. The 1968 volume was subdivided into seven sections: Mutations, Chromosome Aberrations,

Special Chromosomes, Cytological Markers, Departures from Diploidy, Nonchromosomal Inheritance, and Wild-Type Stocks. In the present version, the section on Wild-Type Stocks has been eliminated, new sections on Transposable Elements and DNA Sequences have been added, and a molecular biology category has been added to the descriptions of genes and rearrangements if the information is available.

We are grateful to our colleagues throughout the world for their contributions and corrections to draft copies. Those who have submitted entries or sections of entries are acknowledged on the first line of the entry. Special thanks are due to a number of colleagues whose efforts on behalf of this volume have been more than substantial. In particular, Jeff Hall has provided almost all of the material on behavioral and neuronal genes; George Lefevre and especially his colleague, Catherine Coyle-Thompson, provided massive amounts of information and corrections to the sections on *X*-linked lethals; Trudi Schüpbach provided descriptions of female-sterile and maternal-effect-lethal mutations. Jim Boyd and Scott Hawley provided the entries on mutagen-sensitive and meiotic mutants, respectively. Michael Ashburner has been especially helpful in keeping us supplied with his encyclopedic lists of mutations, chromosome rearrangements, and references; in addition, he has gone over the draft copies of the work and provided detailed additions and corrections. Finally, Loring Craymer and Abraham Schalet were most helpful in reviewing material and pointing out errors and omissions.

We apologize for the omissions, inconsistencies, and errors in this compilation. Every time we reread it we find new ones, but, mercifully, revision has to stop sometime.

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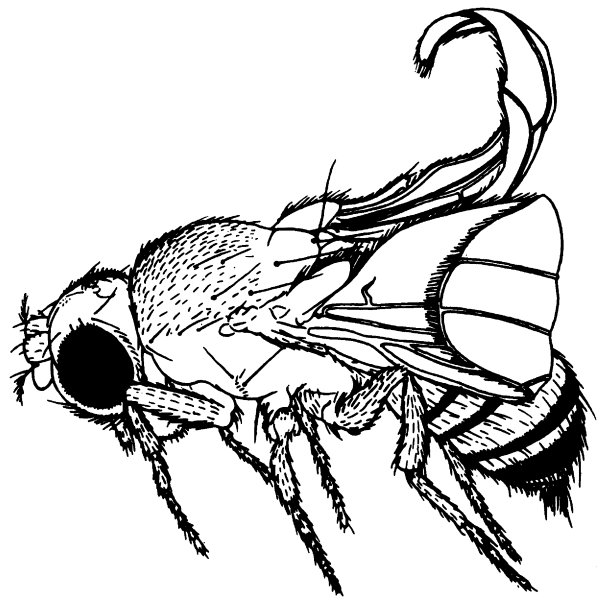


GENES

LOCI. When the last edition of this book was prepared, it was necessary to have two alleles of a gene in order to identify and map a locus genetically. The subsequent development of methods for identifying and mapping loci by gene-dosage manipulation and by *in situ* hybridization with cloned probes has led to the identification and cytological localization of many genes for which no allelic variability has yet been detected. Thus, genes are now recognized by virtue of a phenotypic response to the dosage of a specific circumscribed chromosome segment, by the *in situ* hybridization of a specific transcribed sequence to a polytene-chromosome band, or by the existence of allelic variation. Each locus so recognized is given a name which is descriptive of its mutant phenotype or its wild-type function. The name is concise and is preferably a simple noun or adjective; for example, cabbage, canoe, and kidney or Curly, outstretched, pink and rough. Loci recognized by virtue of the protein that they encode are named as the protein; e.g., Alcohol dehydrogenase, Actin, Calmodulin, etc.

The phenotype of flies obtained from natural populations is considered normal or wild type, and the alleles carried by such individuals, the normal or wild-type alleles. When the main characteristic of the nominate mutant allele is recognized when it is heterozygous with a wild-type allele, the mutant is considered dominant and its name begins with an upper-case letter; when the nominate allele is recessive, an initial lower-case letter is used. The names of genes specifying proteins, for many of which only the wild-type allele is known, begin with upper-case letters. Cases arise in which the same locus has received two or more names; other things being equal, the earlier-applied name is adopted.

For convenience, a symbol is assigned to each locus. This symbol is an abbreviation of the name that uniquely designates the locus in question; it combines brevity with information. It usually begins with the same letter as the name, is always italicized, and does not contain subscripts, or spaces; e.g., *r* for rudimentary, *R* for Roughened, *ro* for rough, *rs* for rose, and *ry* for rosy. In designations of genotypes with several mutant genes, symbols of genes on the same chromosome are separated by spaces (e.g., *y w f B*); symbols of genes on homologous chromosomes are separated by a slash bar (e.g., *y w f/B*); symbols of genes on nonhomologous chromosomes are separated by semicolons and spaces (e.g., *bw; e; ey*). Names are not italicized in text.

ALLELES. The alternatives or alleles at a particular genetic locus are designated by the same name and symbol and are differentiated by distinguishing superscripts. The superscript notation designating alleles may take a number of different forms. A common device is an abbreviation that further characterizes the particular allele or that was used as the locus symbol before allelism was established. This practice is avoided because it has the disadvantage of preempting useful symbols and names from use as locus designations. Another unacceptable device is the use, as superscripts, of elements of the genotype in which the allele arose, since such a designation implies something more than a trivial connection between allele and element. Finally, lengthy acquisition numbers are avoided as allelic designations, since the information that they contain is of no use to the general user, and greatly exceeds what is necessary to differentiate one allele from another; in the present version, many such allelic designations have been abbreviated. In a large number of cases we have replaced complex, and to most users uninformative, superscripts with simple numerial designations; we have not, however, been consistent in this practice. Other acceptable superscripts for allelic designations are arbitrary numbers, capitalized initials of the finder or laboratory, or the date of discovery.

The numeral 1 is the implied superscript of nonsuperscripted symbols. Whereas genes in the same allelic series are designated by the same symbol but with different superscripts, mutants with similar phenotypes at different loci are not given the same symbol.

For a recessive allele of a preponderantly dominant series or a dominant allele of a predominantly recessive series, the superscripts *r* and *D*, respectively, may be used; e.g., Hn^{r}, Hn^{r2}, *and* bw^{D}. Finally, for the normal allele in a series, a superscript plus sign may be used; e.g., b^{+} or B^{+}. The plus symbol alone implies the normal (wild-type) allele or alleles in any context, such as *y*/+ or *y m f*/+. Absence of a particular locus may be noted by use of a superscript minus sign with the symbol; e.g., bb^{-}. Revertants or partial revertants of mutant alleles are designated by the superscript *rv* followed by a distinguishing number; revertants of dominant mutations that are deficiencies are treated not as alleles but as deficiencies and are accordingly not superscripted but listed with the distinguishing number.

Loci encoding specific polypeptides or transcripts require special conventions. In many instances, such loci lack recognized allelic variants, but a single wild-type allele is known; in others, polymorphisms exist in such attributes as electrophoretic mobility, abundance, or stability, or mutants affecting activity, developmental-stage specificity, or tissue specificity may occur; these are designated as alleles in the standard manner; e.g., Adh^{F} and Adh^{S}. Alleles specifying the absence of a particular enzyme or other protein are designated by the superscript *n* (null) followed by a distinguishing number or letter, e.g., Adh^{n1} or where lack of function is inviable by *l* (lethal), e.g., Nrg^{ll}. All such loci identified by virtue of their wild-type gene product are treated as dominants and are thus named and symbolized with initial upper-case letters. Since all the genes described in this compilation are Drosophila genes, we have not used an initial "*D*" to designate the Drosophila homologues of genes originally characterized in other species. Abbreviations for the protein and the gene are frequently identical, and both are used in most discussions. The gene symbol may be differentiated from the protein symbol by having only its initial letter capitalized and by being italicized, whereas the protein symbol is in roman capitals; e.g., ADH.

In several instances where two members of the same allelic series were formerly given different locus names, both are here included under one name; e.g., $Pm = bw^{V1}$. In other cases, we assume allelism of mutants with similar phenotypes and genetic positions even though they have not been tested for phenotypic interaction. In such instances, the basis for the assumption is usually noted. Since the practice has not been consistent, some alleles may be described as different genes. Except in special cases, investigation of allelic interaction of sex-linked recessive lethals is not feasible; consequently, they are often given distinctive symbols where allelism may actually exist.

TRANSFORMANTS. Loci transposed to new chromosome locations by transposable elements are enclosed in brackets to indicate that they are not in their normal position, followed by a parenthetical indication of their new position; e.g., $[w^{+}](35BC)$ and $[ry^{+}](sd)$. As such constructs become more complex, a complete description cannot be incorporated into the symbol. Accordingly, our policy is to sacrifice information in order to keep the symbol as simple as possible; thus, transformants of genes of interest selected by cotransformation with a selectable marker are designated according to the gene of interest rather than the selectable marker; e.g., *[Cp16](52D)* designates an insertion of Chorion protein 16 into 52D, which was selected by cotransformation of ry^{+}.

MIMICS. Mutants at different loci sometimes have similar phenotypic effects. Such loci may be handled in several ways. The simplest is to give each a distinctive name (e.g., vermilion, cinnabar, scarlet, karmoisin, cardinal); this method has the effect of scattering such mimics throughout the alphabetical listing. Or a common symbol followed by a distinguishing symbol may be used (e.g., *tu-1a, tu-1b, tu-2* for genes controlling production of melanotic pseudotumors). Loci encoding proteins of similar function are differentiated by arbitrary numbers (e.g., *Sgs3, Sgs7, Sgs8*), by polytene chromosome position (e.g., *Act5C, Act42A, Act57A,* etc.), or by molecular weight (e.g., *Hsp68, Hsp70, Hsp83,* etc.). Distinctive suffixes are also useful (e.g., rough, roughoid, roughish, roughex; plexus, Plexate; dachs, dachsous; maroon, maroonlike). The latter schemes frequently have the virtue of placing like phenotypes or gene functions in sequence in an alphabetical listing. Some phenotypes result from mutation at many loci in all chromosomes; these are given a common symbol followed by a parenthetical designation of the chromosome and then by a distinguishing designation. Examples of this type of mutant are the female steriles, the lethals, the Minutes, and the male steriles [e.g., *fs(2)B, l(1)1Ac, M(1)18C, ms(2)73d,* respectively]. We endeavor in this work to replace arbitrarily chosen distinguishing designations with polytene locations where possible. This has become feasible as the result of remarkable strides in cytogenetic mapping made possible by the selection characterization and maintenance of many deficiencies and by *in situ* hybridization.

MODIFIERS. The primary effect of some mutants is to cause another mutant to exhibit a more-extreme departure from normal (enhancer) or a more nearly normal phenotype (suppressor). Such mutants are symbolized *e* or *E* and *su* or *Su*, followed in parentheses by the gene modified. Designation of the particular allele modified appears as a superscript within the parentheses and alleles of the modifier gene as superscripts outside the parentheses; e.g., $su(lz^{34})$ and $su(Hw)^{2}$. Terms such as dilutor, exaggerator, inhibitor, intensifier, and modifier were also formerly used, but we have usually attempted to classify such genes as enhancers or suppressors.

FORMAT. Mutants with their descriptions are listed alphabetically according to symbol and cross-indexed according to name. Current terminology is listed in bold face. All cases of synonymy are also listed in italics with cross-references to current usage. Mutants known to be lost are preceded by an asterisk; however, mutants not preceded by an asterisk are not therefore known to be extant. Each mutant is described according to the following format:

symbol: name (Author of entry)

location: The location is indicated by the chromosome number, separated by a hyphen from the genetic position

on the chromosome. Three levels of accuracy of the genetic location are indicated, those carried to tenths of a unit being the more accurately determined; e.g., 3.0 represents a more accurate location than 3. In regions saturated for mutants, map positions may be given in hundreths, either as the result of detailed recombinational mapping or by interpolation using deficiency mapping data. Map positions enclosed in braces are inferred from cytological map position. Accuracy of a map position determination is of course dependent on the accuracy of the positions assigned to the reference markers; i.e., on the accuracy of the map. We treat the map as a rough guide to the relative positions of loci but, considered on a refined level, it may be inaccurate with respect to both position and order of genes. Intense activity in determining cytological positions in recent years is resulting in the rapid replacement of the genetic map by the cytological map as the more useful indicator of gene position.

origin: For induced mutants, the agent is given; mutants recovered from untreated parents or a wild population are listed as spontaneous. Isoallelic variants found as major components of stocks or populations are listed as naturally occurring alleles. Mutagenic agents are frequently abbreviated, especially in tables of alleles; abbreviations used are indicated in the accompanying table:

abbreviation [α]	compound
CB 1246	triethylmelamine
CB 1414	nitrogen mustard
CB 1506	2-chloroethyl methanesulfonate
CB 1522	2-fluoroethyl methanesulfonate
CB 1528	ethyl methanesulfonate
CB 1540	methyl methanesulfonate
CB 1592	S-2-chloroethylcysteine
CB 1735	S-mustard
CB 2041	1:4-dimethanesulfonoxybutane
CB 2058	1:4-dimethanesulfonoxybut-2-yne
CB 2348	1:4-dimethanesulfonoxy-1:4-dimethylbutane
CB 2511	D-1:6-dimethanesulfonyl mannitol
CB 2628	L-1:6-dimethanesulfonyl mannitol
CB 3007	DL-p-N,N-di-(2-chloroethyl)aminophenylalanine
CB 3025	L-p-N,N-di-(2-chloroethyl)aminophenylalanine
CB 3026	D-p-N,N-di-(2-chloroethyl)aminophenylalanine
CB 3034	p-N-N,di-(2-chloroethyl)aminophenylethylamine
CB 3086	styrylquinoline
DCE	dicholorethane
DEB	diepoxy butane
EI	ethylenimine
EMS	ethyl methanesulfonate
ENU	ethyl nitrosourea
HCOH	formaldehyde
HD	hybrid dysgenesis
HMS	hycanthon methanesulfonate
ICR170	2-methoxy-6-dichloro-9-(3-ethyl-2-chloroethyl amino-propylamino)acridine dihydrochloride
MMS	methyl methanesulfonate
MR	male recombination factor = P element
NMS	nitrogen mustard
NMU	nitrosomethyl urea
NNG	N-methyl-N-nitro-N-nitrosoguanidine
P	P-element hybrid dysgenesis
SMS	sulfur mustard
spont	spontaneous
TEM	triethylmelamine

[α] CB Chester Beatty; ICR Institute for Cancer Research.

The chromosome of origin of mutations is of interest when DNA sequence is being studied; such information is not generally available, however, and is usually not included.

discoverer: Name, date of discovery.

synonym: Alternative symbol or name or both, mostly obsolete terminology.

references: Sources of the major descriptive material are listed, but bibliographic material may also appear in some of the other categories. The second reference to a paper in an entry is generally abbreviated to just the author's name or to name and year. Such abbreviated references not preceded by a fuller reference in the same entry are generally to unpublished information. References to CP552 refer to Carnegie Publication 552, which is "The Mutants of *Drosophila melanogaster*" by C.B. Bridges and K.S. Brehme; CP627 refers to "Genetic Variations of *Drosophila melanogaster*" by D.L. Lindsley and E.H. Grell.

phenotype: The most important departures from normal, which are usually those suggested by the name, are described first. Other information about the phenotype follows, and finally there may be data on viability and fertility. This revision contains considerable information on the normal functions of the genes described, including observations on stage and tissue specificity of expression; the techniques of *in situ* hybridization and immunostaining of whole embryos and sectional material have added a new dimension to phenotypic description. The last item in the phenotypic description is the rank, abbreviated RK. In this revision, we have not attempted to assign rank to mutants; however, we have retained those assignments appearing in early editions. Mutants were classified by Bridges into three different ranks according to their utility in experiments in which counts are made: RK1 mutants are easily scored; RK2 mutants are usable but less convenient; RK3 mutants have limited usefulness. An RK3 mutant may be one with good expression and viability but simply not convenient to use in counting experiments; e.g., enzyme polymorphisms. The letter A follows the rank of mutants associated with chromosome aberrations.

alleles: Rather than describing alleles in separate entries, as was done in previous versions, we have attempted, wherever possible, to tabulate them. Different grouping of the types of information itemized above appear as columns in the tables of alleles. When a type of information, such as phenotype, is too extensive for tabulation, subentries follow the nominate entry. The types of information included in each table are decided on a case-by-case basis, but the order of columns approximates the order in which information is included in full entries. Deficiencies may be listed in tables of alleles for the purpose of cross-referencing.

cytology: This category is primarily to provide the cytological location of the gene, as determined by rearranged breakpoint-associated alleles, by deficiency mapping, or by *in situ* hybridization to polytene chromosomes. It may also indicate that the mutant was induced in a rearranged chromosome or occurred in association with a *de novo* rearrangement; in tables of alleles, pre-existing rearrangements are listed by name; *de novo* rearrangements likely to have caused the mutant receive the same designation as the mutant, and are listed by breakpoints in tables and by name in the section on chromosome rearrangements.

molecular biology: This is a new category of information that is expanding at an unprecedented rate. It includes references to the cloning, restriction mapping, sequenc-

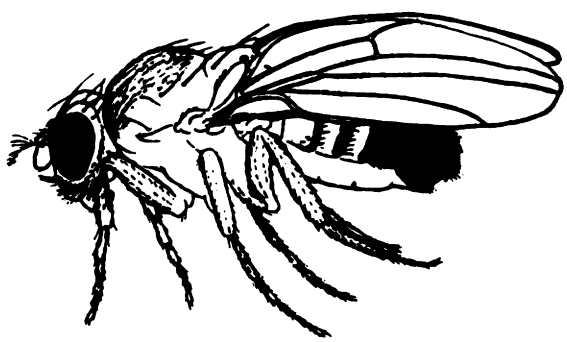
ing, and conceptual amino-acid sequences with indicated homologies to other proteins and motifs. In allele tables, it includes sequence alterations associated with different alleles. Molecular mapping results are currently presented without regard to an established convention. Zero coordinates have frequently been chosen at sites not easily identified in a normal chromosome complement, such as a rearrangement breakpoint, the site of insertion of a transposable element, or the end of a random-shear fragment. We propose that the midpoint of an endonuclease restriction site, preferably one shown to be present in choromosomes of several independent origins, be chosen as the origin of a restriction map and that 0 marks not a nucleotide pair, but the plane of symmetry of the restriction site; ambiguity can result only from restriction site polymorphism. We also propose that when the chromosomal orientation of the map is determined, positive values extend to the right and negative values to the left so that all restriction maps have the same orientation; this will be especially useful when adjacent restriction maps fuse. When two studies of the same region have used different sets of coordinates, we have perforce chosen the one that conforms more closely with the above conventions. No convention has been established for defining the origin of nucleotide maps of transcription units, and of course either orientation with respect to the restriction map may obtain, depending on which strand is transcribed.

other information: This category contains miscellaneous information that does not fit into one of the other categories.

Loci that share phenotypic and nomenclatural features (i.e., mimics) are frequently presented in a single entry in which the common information is presented once, and the information that distinguishes among loci is tabulated; the order of the columns of information roughly corresponds to the order in which the same categories of information appear in full entries.

a: arc

location: 2-99.2.
discoverer: Bridges, 12e24.
references: Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 202 (fig.).
Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 212 (fig.).
Bridges, 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.
phenotype: Wings broader; bent downward in slight, even arc; edges drawn down to diamond shape. Sometimes in stock, wings are bent upward instead of downward. Crossveins closer together. RK2.
alleles: a^{1}, a^{ba} (see below), $*a^{ba1}$, $*a^{ba2}$, $*a^{ba3}$, $*a^{ba4}$, $*a^{bad6}$, $*a^{badp}$, $*a^{bar}$, $*a^{Ba}$, $*a^{BaC}$, $*a^{Bap1}$, $*a^{Bap2}$, $*a^{BaX}$, $*a^{Bay}$ (Goldschmidt, 1945, Univ. Calif. Berkeley Publ. Zool. 49: 351-56, 388-89, 519; CP627), and $*a^{M60}$ (Meyer, 1963, DIS 37: 50).
cytology: Placed between 57F11 and 58E4 on the basis of its inclusion within *Df(2R)M-1* = *Df(2R)57F11-58A1;58F8-59A1* but not *Dp(2;3)P* = *Dp(2;3)58E3-4;60D14-E2;96B5-C1* (Bridges, 1937). Likely in band 58D6 or 7 based on *Df(2R)a-ba2* = *Df(2R)58D5-6;58D7-8*.



a: arc
From Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 148.

a^{ba}: arc-broad angular

origin: Spontaneous.
discoverer: Goldschmidt, 1934.
synonym: Referred to as *bran: broad angular* by Goldschmidt, but shown by him to be an allele of *arc*.
references: 1945, Univ. Calif. Berkeley Publ. Zool. 49: 351-56, 388-89, 519.
phenotype: Wings broader and shorter than wild type, blunt at the tip. Frequently shows upturned posterior scutellar bristles. In combination with svr^{poi}, produces soft blistered wing. Other interactions described by Goldschmidt, 1945, table 74. Wing grows in pupal stage to full length and then retracts, possibly with histolysis [Goldschmidt, 1934, Z. Indukt. Abstamm. Vererbungsl. 69: 38-131 (fig.)]. RK2.
cytology: Salivary chromosomes normal (Kodani).
other information: Claimed to recur repeatedly in certain lines (Goldschmidt, 1945).

α^{1}: see *tyr1*

**A: Abnormal abdomen*

location: 1-4.5.
discoverer: Morgan, 11g.
synonym: *Abnormal.*
references: 1915, Am. Naturalist 49: 384-429 (fig.).
Morgan and Bridges, 1916, Carnegie Inst. Washington Publ. No. 237: 27 (fig.).
phenotype: Tergites and sternites raggedly incomplete, exposing a thin crinkled cuticle; bristles and hairs on abdomen correspondingly eliminated. Highly variable, wild phenotype in old dry cultures. *A/+* less extreme than *A/A* and *A* male; homozygous female fully viable and fertile. RK2 in well-fed cultures.
alleles: $*A^{1}$ (Morgan and Bridges, 1916), A^{53g} (see below), A^{70} [allelism conjectural (Gooskov, 1971, DIS 46: 41)].
other information: Lost by reversion to wild type.

A: see bw^{A}

A53g

location: 1- (between *y* and *w;* may not be allelic to *A*).
origin: Spontaneous.
discoverer: Hillman, 53g.
references: 1953, DIS 27: 56.
1973, Genet. Res. 22: 37-53.
1977, Amer. Zool. 17: 521-33.
Hillman and Barbour, 1963, Proc. Intern. Congr. Genet., 11th, Vol. 1: 170.
phenotype: Epidermal foldings of abdomen abnormal. Tergite formation incomplete, ranging from loss of tergites 2-8 in extreme cases to loss of lateral part of tergite in one or more segments. Expression in *A53g/A53g* females > *A53g/Y* males > *A53g/+* females. Expression maternally influenced (Shafer and Hillman, 1974, J. Insect Physiol. 20: 223-230). Highly variable; sensitive to modifiers on *X, 2,* and *3,* including *E(A53g)* on *2L*. Sensitive to culture conditions; expression reduced in old cultures and under conditions of crowding, low temperature (TSP in late second and early third instar), and low humidity. Also reduced by agents that inhibit RNA or protein synthesis or oxidative phosphorylation (Hillman, Shafer, and Sang, 1973, Genet. Res. 21: 229-38). Supernatents from homogenates of *A53g*-bearing adults stimulate amino acid incorporation and aminoacylation of tRNA more than those from wild type (Rose and Hillman, 1969, Biochem. Biophys. Res. Commun. 35: 197-204). Mutant late pupae and adults show increased concentrations of soluble protein. Expression of biochemical phenotype correlated with that of visible phenotype (Rose and Hillman, 1973, Genet. Res. 21: 239-245). RK2 in young cultures.
cytology: Deficiency analysis places *A53g* in 3A5 (Hillman), which is at variance with the genetic position of *A*.

a-3: see *a(3)26*

**A-p: Abnormal abdomen-polygenic*

location: Polygenic.
discoverer: Sobels, 49i.
references: 1950, DIS 24: 62.
1951, DIS 25: 75-76.
1952, Genetica 26: 117-279 (fig.).
1952, Trans. Intern. Congr. Entomol., 9th, Vol. 1: 225-30.
synonym: *AA; Asy: Asymmetric.*

phenotype: Incomplete mediodorsal fusion and onesided reduction of tergites. When more than one tergite is abnormal, spiral segmentation types are most frequent. Expression strongly dependent on environment. Penetrance and expressivity correlated (Bezem and Sobels, 1953, Koninkl. Ned. Akad. Wetenschap., Proc. Ser. C. 56: 48-61). In strains selected for penetrance of *A-p*, mediodorsal fusion or asymmetrical reduction of head and thorax also occur. RK3.

*a(1)48: abnormal abdomen in chromosome 1

location: 1- (not located).
origin: Spontaneous.
discoverer: Zimmerman, 1948.
references: 1952, DIS 26: 69.
1954, Z. Indukt. Abstamm. Vererbungsl. 86: 327-72 (fig.).
phenotype: Used to describe three *X* chromosomes with little or no effect of their own but which increase the incidence of abdominal malformations in crosses with *a(2)* and *a(3)*. Viability and fertility good. RK3.
alleles: The three chromosomes designated **a(1)48*, **a(1)50*, and **a(1)51* (CP627). Genetic relations not worked out.

a(1)HM26

location: 1-(*y-cv*).
origin: Induced by ethyl methanesulfonate.
synonym: *l(1)HM26*.
references: Mayoh and Suzuki, 1973, Can. J. Genet. Cytol. 15: 237-54.
phenotype: Missing or reduced sternites; missing or angled tergites; black specks on ventral surface of abdomen in about one-third of males at 22° and more than half of males at 17°. Viability reduced at 17° relative to that at 22°.

a(1)HM27

location: 1-(near *y*).
origin: Induced by ethyl methanesulfonate.
synonym: *l(1)HM26*.
references: Mayoh and Suzuki, 1973, Can. J. Genet. Cytol. 15: 237-54.
phenotype: Same as *a(1)HM26;* more severe at 17° than at 22°. Viability slightly reduced at 17° relative to that at 22°.

*a(2)48

location: 2- (not located).
origin: Spontaneous.
discoverer: Zimmerman, 1948.
references: 1952, DIS 26: 69.
1954, Z. Indukt. Abstamm. Vererbungsl. 86: 327-72 (fig.).
phenotype: Abdominal irregularities most frequently involve anterior segments. Penetrance 7%. Also shows maternal effect. Viability and fertility good. RK3.
alleles: Second chromosomes with some or all of these effects are **a(2)50*, **a(2)51*, and **A(2)51*. Genetic relations not worked out.

a(3)26: see *abd*

*a(3)48

location: 3- (not located).
origin: Spontaneous.
discoverer: Zimmerman, 1948.
references: 1952, DIS 26: 69.
1954, Z. Indukt. Abstamm. Vererbungsl. 86: 327-72 (fig.).
phenotype: Only a maternal effect affecting 2.5% of progeny. Irregularities most frequently involve posterior segments of abdomen. Viability and fertility good. RK3.

A34: see bw^{V6}

aa: anarista

location: 3-0.
discoverer: Bridges, 23d10.
synonym: *al-b: aristaless-b.*
references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 218.
phenotype: Aristae bare or tufted. Wings somewhat broader than wild type. Expression variable, overlaps wild type often in female and sometimes in male. RK3.
cytology: Placed between 61E2 and 62A6 on basis of its inclusion in *Df(3L)D = Df(3L)61E2-F1;62A4-6* from *T(Y;2;3)D*.

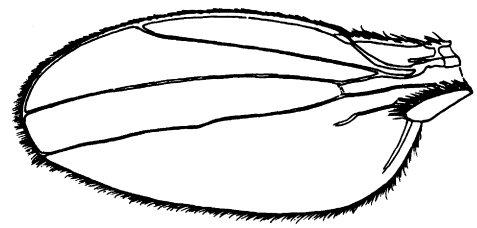
Aa: Altered abdomen

location: 1- (not located).
origin: X ray induced in the *In(1)dl-49, y w f* component of *C(1)DX*.
discoverer: Cicak, 56f.
references: Cicak and Oster, 1957, DIS 31: 80.
phenotype: Heavy deposition of melanin in tergites of females and males. *Aa* detachment-bearing males sterile. RK2A.
cytology: Possibly associated with a rearrangement in addition to *In(1)dl-49*.

AA: see *A-p*

ab: abrupt

location: 2-44.0.
origin: Spontaneous.
discoverer: Bridges, 16j16.
references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 218 (fig.).
phenotype: Vein L5 usually stops after posterior crossvein. Scutellar bristles usually fewer. Wing effect probably acts during contraction period (Waddington). Overlaps wild type. Expression more severe in females than in males and when pupal stage takes place at 20° than at 25°. TSP during the first 10% of pupal stage. (Thompson, Bruni, Carbonaro, and Russo, 1988, DIS 67: 86). RK2.
alleles: ab^{1}, ab^{2} (see below), ab^{51g}, like ab^{2} in *In(2L+2R)Cy;* $*ab^{l\text{-}60h}$*: abrupt lethal* (CP627).



ab: abrupt
Edith M. Wallace, unpublished.

ab^{2}

origin: Spontaneous.
discoverer: Bridges, 23g6.
synonym: *pt: parted.*
references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 232.
phenotype: Vein L5 does not reach margin. Scutellar bristles always fewer than wild type. Hairs parted down midline of thorax and abdomen. Supra-alar bristles sometimes absent. Coxae tend to be thickened. Males sterile and have rotated genitalia. *ab/ab*^{2} resembles *ab/ab* but has a stronger bristle effect. RK2.

abb: abbreviated

location: 2-105.5.
discoverer: Bridges, 28d6.
references: 1937, Cytologia (Tokyo), Fujii Jub., Vol. 2: 745-55.
phenotype: Bristles smaller, especially posterior scutellars. Developmental time slightly longer than normal. Viability only slightly reduced. Classification difficult, especially in early eclosions; improves with age of culture. Enhanced by *shr* (2-2.3), making classification easy. RK3; RK2 with *shr*.
cytology: Placed in region between 59E2 and 60B10 by Bridges (1937) on basis of its being to the right of *In(2R)bw*^{VDe1} = *In(2R)41B2-C1;59E2-4* and to the left of *Df(2R)Px* = *Df(2R)60B8-10;60D1-2*.

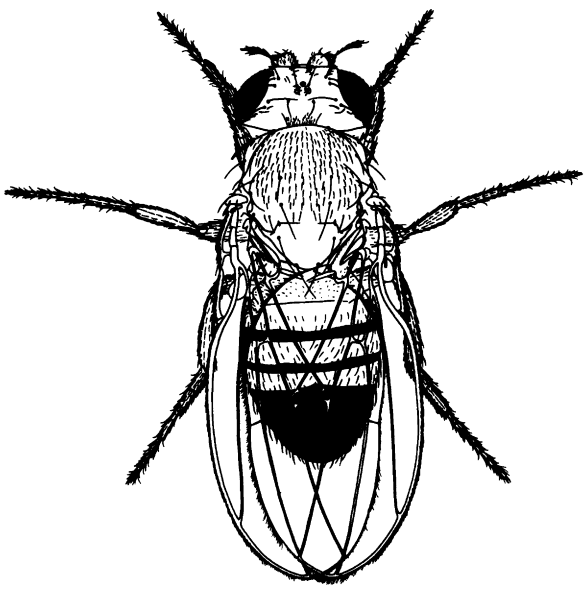


abb: abbreviated
From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 11.

abd: abdominal

location: 3-27 (to the right of *se*).
origin: Spontaneous.
discoverer: H. A. and N. W. Timoféeff-Ressovsky.
synonym: *a-3, a(3)26.*
references: 1927, Wilhelm Roux's Arch. Entwicklungsmech. Organ. 109: 70-109.
Schäffer, 1935, Z. Indukt. Abstamm. Vererbungsl. 68: 336-60 (fig.).
phenotype: Irregular reduction of abdominal tergites, sternites, pigmentation, and bristles; more marked in females and increased by crowding and dry food (Braun, 1938, Am. Naturalist 72: 189-92). Schäffer's data (1935) suggest irregular dominance in heterozygote, overlapping of wild type in homozygote, and genetic modifiers. RK3.
alleles: *abd*^{2}, spontaneous; recovered by Gottschewski, 1939. Partially complements *abd*^{1}. Allelism inferred from similarity in genetic location and phenotype and incomplete complementation.
cytology: Placed in 66D9-E1 based on its inclusion in *Df(3L)h-i22* = *Df(3L)66D9-E1* (Ingham, Pinchin, Howard, and Ish-Horowicz, 1985, Genetics 111: 463-86).

abd-A: see ***BXC***

Abd-B: see ***BXC***

abdomen rotatum: see ***ar***

abdominal: see ***abd***

abe: see *mit15*

abero: see ***abr***

Abl: Cellular abl oncogene sequence

location: 3-{44}.
origin: Isolated from genome library using *v-abl* probe.
synonym: *Dash.*
references: Shilo and Weinberg, 1981, Proc. Nat. Acad. Sci. USA 78: 6789-92.
Simon, Kornberg, and Bishop, 1983, Nature (London) 302: 837-39.
phenotype: Considered to be the Drosophila sequence homologous to mammalian *c-abl* based both on its origin and amino acid sequence as inferred from its nucleotide sequence.

ABL protein detected at the time of germ-band shortening in the axons of the central nervous system in a bilateral symmetrical series of points that correspond to the positions of neuromeres; later, protein appears in the axons growing across the midline, but not in the cell bodies of the CNS nor in the PNS. As development proceeds, staining of the longitudinal fascicles and to a lesser extent the commissural fascicles becomes intense; staining also seen in association with axonal outgrowth of neural cells in the eye imaginal disk (Bennett and Hoffmann).

Recessive alleles in combination with *Df(3L)st-j7* = *Df(3L)73A1-2;73B1-2* either die as late pupae or pharate adults with complete cuticle and roughened eyes, *Abl*^{l1}, or as short lived (5-6 days), rough-eyed adults, *Abl*^{l2} and *Abl*^{l3}. Surviving females lay few eggs, some of which develop into adults; surviving males have motile sperm, but do not mate and produce no progeny. The rough eye is a reflection of some loss of photoreceptor cells plus ommatidial fusion. In combination with a deficiency extending further to the left, e.g., *Df(3L)std11* = *Df(3L)73A11-B1;73D1-2* to include the locus of *Dab*, *Abl*^{l}/*Abl*$^{-}$ genotypes die as late embryos or early first-instar larvae with disrupted axonal organization in the ventral nerve cord (Henkemeyer, Gertler, Goodman, and Hoffmann, Cell 51: 821-28). CNS of doubly deficient embryos, i.e., *Abl*$^{-}$ *Dab*$^{-}$, fails to form commissures and is defective in axonal outgrowth, although the PNS develops normally.

alleles: Three ethyl-methanesulfonate-induced recessive lethal or semilethal alleles recovered in combination with *Df(3L)st4* or *Df(3L)st-e5* by Belote, McKeown, and Hoffmann are designated Abl^{l1}, Abl^{l2}, and Abl^{l3}. Phenotypic descriptions given above.

cytology: Localized to 73B by *in situ* hybridization with genomic clone (Simon *et al.*).

molecular biology: Sequence isolated using *v-abl* probe from murine leukemia virus (Hoffman-Falk, Einat, Shilo, and Hoffmann, 1983, Cell 35: 393-401). cDNA and genomic sequencing (Telford, Burkhardt, Butler, and Pirrotta, 1985, EMBO J. 4: 2609-15; Henkemeyer, Bennett, Gertler, and Hoffmann, 1988, Mol. Cell Biol. 8: 843-53) reveal a gene of ten exons distributed over 26 kb of genomic DNA; the exons encode a protein of 1520 amino acids, whose sequence is more similar to mammalian *c-abl* sequence than to that of any other gene; between residues 187 and 656, which contains the tyrosine kinase essential domain, the Drosophila sequence is 75-85% similar to that of the human *abl* gene. 33 bp region beginning at tyrosine-416 84% homologous to mammalian nucleotide sequence and 62% homologous to *C-src* DNA from Drosophila (Hoffmann, Fresco, Hoffman-Falk and Shilo, 1983, Cell 35: 393-401). The polypeptide product as yet unidentified but presumed to be a protein kinase; Drosophila extracts do contain a tyrosine kinase activity (Simon *et al.*). The carboxy half of the ABL protein is not conserved between flies and mammals. Expression of the kinase essential domain in bacteria leads to excessive phosphorylation of proteins at tyrosine residues. Developmental Northerns probed with 800 base-pair sequence from region of highest homology with *v-abl* reveal a 6.2 kb polyadenylated transcript in early but not late embryos, larvae or adults; most abundant in 0-4 hr embryos; absent after 8 hr (Lev, Liebovitz, Segev, and Shilo, 1984, Mol. Cell. Biol. 4: 982-84); returns in a burst of activity in early pupae.

Abnormal: see ***A***

abnormal abdomen: see ***a( )***

Abnormal abdomen: see ***A***

abnormal eye: see *mit15*

abnormal oocytes: see ***abo***

abnormal tergites: see ***abt***

abnormal wings: see ***abw***

abo: abnormal oocyte

location: 2-44.0 (mapped with respect to *J*, 2-41).

origin: Naturally occurring allele recovered near Rome, Italy.

references: Sandler, Lindsley, Nicoletti, and Trippa, 1969, Genetics 64: 481-93.
Mange and Sandler, 1973, Genetics 73: 73-86.
Sandler, 1970, Genetics 64: 481-93.
1975, Israel J. Mol. Sci. 11: 1124-34.
1977, Genetics 86: 567-82.

phenotype: Probability of survival of embryos produced by *abo/abo* mothers reduced; male embryos more severely affected than female embryos. Both preblastoderm and postblastoderm embryonic death observed; partial rescue of postblastoderm mortality effected by paternally inherited abo^{+} allele; partial rescue of preblastoderm mortality by heterochromatic *ABO* elements located in *Xh* between 3/4 and 7/8 of the distance from the centromere, in *YL* region h10-11, in *YS* region h19, in *2R* proximal, and perhaps in other heterochromatic regions (Pimpinelli, Sullivan, Prout, and Sandler, 1985, Genetics 109: 701-24). Gradual loss of phenotype in homozygous *abo* stocks accompanied by increase in quantity of ribosomal DNA (Krider and Levine, 1975, Genetics 81: 501-13). New restriction fragments appear in Hind III/Hae III double digests of such homozygous lines probed with nontranscribed spacer sequences of ribosomal genes (Graziani, Vicari, Boncinelli, Malva, Manzi, and Mariani, 1981, Proc. Nat. Acad. Sci. USA 78: 7662-64). *abo* phenotype returns with subsequent maintenance in heterozygous condition. Homozygous *abo* females exhibit moderate decrease in recombination with concommitant increase in exceptional progeny (Carpenter and Sandler, 1974, Genetics 76: 453-75).

cytology: Located in 31F-32E based on its inclusion in *Df(2L)J39 = Df(2L)31A-B;32E* but not *Df(2L)J27 = Df(2L)31B-D;31F* or *Df(2L)Mdh = Df(2L)30D-F;31F*. *abo*-bearing chromosomes differ from others in having a *blood* insertion sequence in 32E (Lavorgna, Malva, Manzi, Gigliotti, and Graziani, 1989, Genetics 123: 485-94).

ABO

A series of heterochromatic elements capable of reducing the level of maternally influenced preblastoderm, but not postblastoderm, mortality among the progeny of *abo/abo* mothers; embryos that carry ABO elements survive better than those that do not (Pimpinelli, Sullivan, Prout, and Sandler, 1985, Genetics 109: 701-24). Elements identified to date and their heterochromatic locations, where known, are listed in the accompanying table. The rescuing capability of *ABO-X* approximates that of *ABO-YL* + *ABO-YS*. *ABO-X* apparently defective in $In(1)sc^{4}$ (Malva, Labella, Manzi, Salzano, Lavorgna, De Ponti, & Graziani, 1985, Genetics 111: 487-94). Effectiveness of *ABO-X* and *ABO-2R* appears to be enhanced by maintenance in a homozygous *abo* stock (Sullivan and Pimpinelli, 1986, Genetics 114: 885-95).

element	cytology
ABO-2R	
ABO-X	h26-28 [α]
ABO-YL	h10-11
ABO-YS	h19

[α] In region that includes proximal half of h26 and distal half of h28 (Pimpinelli, Sullivan, Prout, and Sander, 1985, Genetics 109: 701-24).

abr: abero

location: 2-83.

origin: Spontaneous.

discoverer: Bridges, 33b10.

phenotype: Abdominal banding etched and irregular. Wing margins irregular. Eyes rough. Bristles and hairs sparse and disarranged. *abr/+* sometimes lacks anterior dorsocentrals. Viability usually poor. RK3.

other information: Not allelic to *fr* or *nw*.

abrupt: see ***ab***

Abruptex: see ***Ax,*** listed under ***N: Notch***

*abt: abnormal tergites

location: 1-45.6.
origin: Induced by 2-chloroethyl methanesulfonate.
discoverer: Fahmy, 1955.
references: 1959, DIS 33: 83.
phenotype: Abdomen affected to various degrees, from extreme deformation of tergites to slight abnormalities in distribution of pigment and hairs. Eyes also deformed to various degrees from gross alterations in shape to slight derangement of ommatidia. Wings vary from alterations in size, outline, and venation to small incisions of the inner margin. Most-extreme effects not always positively correlated, and all flies show several atypical characters. Males viable; fertility severely reduced. RK3.

*abw: abnormal wings

location: 1-60.
origin: X ray induced.
discoverer: Halfer, 1963.
phenotype: Wing size reduced; wings upturned; L5 and crossveins absent. Plexus of veins between L3 and L4. RK1.

abx: see BXC

ac: see ASC

Ac: see Cu^A

Ac-SD: see *Rsp*

acc: acclinal wing

location: 1-54.5.
origin: Induced by triethylenemelamine.
discoverer: Fahmy, 1952.
references: 1958, DIS 32: 67.
phenotype: Wings upheld but slope backward at 45° angle from abdomen. Unable to fly or jump; muscles normal in gross and ultrastructural morphology. Mosaic experiment suggests possible thoracic neural etiology (Deak, 1976, J. Insect Physiol. 22: 1159-65). Viability and fertility good in both sexes. RK1.
alleles: One allele each induced by D-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine and by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine.

Ace: Acetyl cholinesterase (J.C. Hall)

location: 3-52.2.
synonym: *l(3)26.*
references: Hall and Kankel, 1976, Genetics 83: 517-35.
Greenspan, Finn, and Hall, 1980, J. Comp. Neurol. 189: 741-74.
Hall, Alahiotis, Strumpf, and White, 1980, Genetics 96: 939-65.
phenotype: The structural gene for acetylcholinesterase [AChE, acetylcholine acetyl hydrolase (EC 3.1.1.7)], the enzyme that terminates synaptic transmission by rapidly hydrolyzing the neurotransmitter acetylcholine. Biochemical analysis (*e.g.*, Zingde, Rodrigues, Joshi, and Krishnan, 1983, J. Neurochem. 41: 1243-52; Gnagey, Forte, and Rosenberry, 1987, J. Biol. Chem. 262: 13290-98; Fournier, Bride, Karch, and Bergé, 1988, FEBS Lett. 238: 333-37; Haas, Marshall, and Rosenberry, 1988, Biochemistry 27: 6453-57; Toutant, Arpagaus, and Fournier, 1988, J. Neurochem. 50: 209-18; Fournier, Bergé, Almeida, and Bordier, 1988, J. Neurochem. 50: 1158-63), indicates that the mature enzyme contains noncovalently associated subunits of 16 and 55 kd, which are processed from a primary translation product of *ca* 70 kd such that the 16-kd moiety is from the N terminus and the 55-kd moiety is from the C terminus; two such associations are linked via disulfide bonds connecting the 55-kd polypeptides anchored to membrane via a glycoinositol phospholipid anchor covalently linked to the C termini of the 55-kd subunits. Extracts contain amphiphilic dimers and monomers as well as hydrophilic dimers and monomers, which lack the glycoinositol phospholipid anchor. Developmental profile studied by Dewhurst, McCaman, and Kaplan (1970, Biochem. Genet. 4: 499-508; see also Arpagaus, Fournier, and Toutant, 1988, Insect Biochem. 18: 539-49); total AChE activity shows a transient peak during first larval instar and rises again to a maximum in the adult. In the developing eye disc, AChE first appears in retinula cells three to four days before they are functional and when it cannot have a synaptic function; levels are reduced in retinula cells midway through pupal development, and the enzyme accumulates rapidly in the neuropils of the optic lobes of the brain and the midbrain (Wolfgang and Forte, 1989, Dev. Biol. 131: 321-30). Putative nulls are lethal at end of embryonic stage; then ultrastructural observations of CNS in such mutants suggest neural-degenerative defects (Chase and Kankel, 1988, Dev. Biol. 125: 361-80). ACE-minus tissues survive in mosaics unless enzyme absent from posterior midbrain; surviving mosaics have defective visual physiology, optomotor behavior or courtship, depending on location of mutant clone. Such clones associated with defective morphology or neuropile of various ganglia in central nervous system (Greenspan *et al.*, 1980). In heat-sensitive combinations of *Ace* mutations (Greenspan *et al.*, 1980), both membrane-bound and soluble enzyme has reduced activity (Zador, 1989, Mol. Gen. Genet. 218: 487-90).
alleles: Unless noted otherwise in comments column alleles are null as is Ace^1.

allele	origin	discoverer	synonym	ref [α]	comments
Ace ^{1} β	X ray	Schalet	*l(3)26*	*4, 8*	
Ace ^{2}	EMS	Deland	*l(3)m15*	*4*	
Ace ^{3}	EMS	Hilliker, Clark	*l(3)B2-5*	*4*	
Ace ^{4}	EMS	Hilliker, Clark	*l(3)B4-2*	*4*	
Ace ^{5}	EMS	Hilliker, Clark	*l(3)B8-2*	*4*	
Ace ^{6}	EMS	Hilliker, Clark	*l(3)B15-2*	*4*	hypomorphic
Ace ^{7}	EMS	Hilliker, Clark	*l(3)B22-1*	*4*	
Ace ^{8}	EMS	Hilliker, Clark	*l(3)B22-2*	*4*	hypomorphic
Ace ^{9}	EMS	Hilliker, Clark	*l(3)B27-1*	*4*	
Ace ^{10}	EMS	Hilliker, Clark	*l(3)B29-1*	*4*	
Ace ^{11}	EMS	Hilliker, Clark	*l(3)B29-2*	*4*	
Ace ^{12}	EMS	Hilliker, Clark	*l(3)H36*	*4*	
Ace ^{13}	EMS	Hilliker, Clark	*l(3)H41*	*4*	
Ace ^{14}	EMS	Hilliker, Clark	*l(3)H89*	*4*	
Ace ^{15}	EMS	Hilliker, Clark	*l(3)B21-5*	*4*	
Ace ^{16}	EMS	Hilliker, Clark	*l(3)H15*	*4*	
Ace ^{HD1} β	HD			*6*	
Ace ^{j19}	EMS			*1*	
Ace ^{j21}	EMS			*1*	
Ace ^{j27}	EMS			*1*	
Ace ^{j29} β	EMS			*1, 2*	cold sensitive
Ace ^{j31}	EMS			*1*	
Ace ^{j32}	EMS			*1*	
Ace ^{j33}	EMS			*1*	
Ace ^{j39}	EMS			*1*	
Ace ^{j40} β	EMS			*1, 2, 7*	few survivors; suppressor of variegation
Ace ^{j41}	EMS			*1*	

allele	origin	discoverer	synonym	ref α	comments
Ace^{j43}	EMS			*1*	
Ace^{j44} β	EMS			*1*	
Ace^{j45}	EMS			*1*	
Ace^{j50}	EMS			*1*	
Ace^{j51}	EMS			*1*	
Ace^{j53}	EMS			*1*	
Ace^{j55}	EMS			*1*	
Ace^{lm35} β	EMS			*3*	hypomorphic
Ace^{lm38}	EMS			*3*	
Ace^{lm115}	EMS			*3*	
Ace^{G11}	EMS	Gelbart			
Ace^{mr} β	spont			*5*	

α *1* = Greenspan, Finn, and Hall, 1980, J. Comp. Neurol. 189: 741-74; *2* = Hall, Alahiotis, Strumpf, and White, 1980, Genetics 96: 939-65; *3* = Hall and Kankel, 1976, Genetics 83: 517-35; *4* = Hilliker, Clark, Gelbart, and Chovnick, 1981, DIS 56: 65-72; *5* = Mortan and Singh, 1982, Biochem. Genet. 20: 179-98; *6* = Nagoshi and Gelbart, 1987, Genetics 117: 487-502; *7* = Reuter, Gausz, Gyurkovics, Friede, Bang, Spierer, Hall, and Spierer, 1987, Mol. Gen. Genet. 210: 429-36; *8* = Schalet, Kernaghan, and Chovnick, 1964, Genetics 50: 1261-68.

β More detailed description below.

cytology: Located in 87E3 based on its location between *Df(3R)ry1301* = *Df(3R)87D2-4;87E1-2* and *Df(3R)GE41* = *Df(3R)87E4* (Hall and Spierer 1986, EMBO J. 5: 2949-54).

molecular biology: Locus comprises ten exons and nine introns distributed within a 34-kb transcription unit (Fournier, Karch, Bride, Hall, Bergé, and Spierer, 1989, J. Mol. Biol. 210: 15-22) extending from approximately coordinates 18 to 52 kb on the molecular map of Bender, Spierer, and Hogness (1983, J. Mol. Biol. 168: 17-33) whose origin is 6.5 kb to the left of the left breakpoint of *In(3R)Cbx^{rv1}* with positive values extending to the right. Transcription takes place from right to left. The 5′ untranslated region is transcribed from exons I and II; the signal sequence is encoded in exon II, the 16-kd polypeptide by exons II, III, and IV; the 55-kd polypeptide by exons IV, V, VI, VII, VIII, and IX; the hydrophobic peptide exchanged in mature protein with a glycolipid anchor as well as the 3′ untranslated region by exon X. The mature transcripts are estimated at 4.2 and 4.5 kb (Nagoshi and Gelbart, 1987, Genetics 117: 487-502). cDNA sequencing (Hall and Spierer, 1986; Fournier *et al.*, 1989) indicates mature transcript of 4291 nucleotides encoding a 650-amino-acid protein product, which displays a high degree of homology with AChE from *Torpedo californica*; however, the Drosophila protein carries a 38-amino-acid signal sequence lacking in Torpedo, and a 41-amino-acid hydrophilic sequence extending from residues 140 to 180 that is not contained in the Torpedo sequence; the latter is encoded by exons III and IV, and contains the site of proteolytic cleavage of the Drosophila AChE primary translation product.

Ace^{1}

phenotype: Temperature insensitive lethal; lethal in homozygotes or in combination with deficiency for *Ace^{+}*. Lethality at end of embryonic stage. Greatly reduced levels of acetylcholinesterase. AChE-minus tissues survive in mosaics unless enzyme absent from posterior midbrain; surviving mosaics have defective visual physiology, optomotor behavior, or courtship depending on location of mutant clone. Such clones associated with defective morphology of neuropile of various ganglia in central nervous system.

molecular biology: DNA insert in fifth intron (Fournier *et al.*, 1989) observed at approximately coordinate +30; separable by recombination from the *Ace^{1}* mutation (Nagoshi and Gelbart, 1987, Genetics 117: 487-502).

Ace^{HD1}

phenotype: Retains some ACE activity (Nagoshi and Gelbart, 1987, Genetics 117: 487-502), but only as soluble enzyme outside CNS (Zudor *et al.*, 1986).

molecular biology: Deleted of promoter region and first (non-coding) exon (Fournier *et al.*, 1989).

Ace^{j29}

phenotype: The original allele of this complementation group. Cold sensitive lethal. Maximum survival of *Ace^{j29}/Df(3R)l26d* at 27°, no survival at 18°. Exposure to 18° does not reduce AChE activity. *Ace^{j29}* alters Km of enzyme, further implying structural gene locus.

Ace^{j40}

phenotype: Nearly completely lethal. Two percent survival in combination with *Df(3R)l26d* at 18°, none at 29°. Partial complementation of *Ace^{j19}* and *Ace^{j50}*; heat sensitive; extracts of *Ace^{j40}* lack the 110 kilodalton molecular species, whereas *Ace^{j19}* and *Ace^{j50}* lack the 64 and 75 kilodalton species (Zingde, Rodrigues, Joshi, and Krishnan, 1983, J. Neurochem. 41: 1243-52). Enzyme produced by heteroallelic combinations raised under permissive conditions is thermolabile. Exposure of *Ace^{j40}/Ace^{j19}* or *Ace^{j40}/Ace^{j50}* flies to restrictive temperature during late embryonic-early larvae stage lethal; little effect on mid and late larval stages; pupal exposure causes defects in adult phototaxis and motor activity. Heat treatment of adults causes no decline in ACE activity but decrements in phototaxis (29°), and cessation of movement (31°) observed. *Ace^{j40}* produces enzyme with altered Km.

molecular biology: Appears to map proximal to a DNA insert located between coordinates +43 and +48 (Nagoshi and Gelbart, 1987).

Ace^{j44}

molecular biology: Associated with a molecularly defined structural variation; probably loss of a *Bam* H1 site around coordinate +33 (Gausz, Hall, Spierer, and Spierer, 1986, Genetics 112: 65-78). Structural variant and mutation appear to be inseparable by recombination (Nagoshi and Gelbart, 1987).

Ace^{lm35}

phenotype: Hypomorphic allele. Exhibits reduced survival (< 30%) in combination with *Df(3R)l26d*. Enzyme activity in *Ace^{lm35}/+* heterozygotes lower than in heterozygotes for more severe alleles.

Ace^{mr}: Acetylcholinesterase-malathion resistant

origin: Recovered from line selected for malathion resistance.

phenotype: Acetylcholinesterase from homozygotes has lower K_m, lower activity, and slightly increased electrophoretic mobility compared to wild type. Relation to malathion resistance unclear.

ACE1: Amplification Control Element on chromosome 1

A sequence required for amplification in ovarian follicle cells of the cluster of chorion-protein genes located at 7F1-2 *(Cp36* and *Cp38);* provisionally located between 654 and 266 base pairs upstream from *Cp38* (Wakimoto).

ACE3

A sequence required for amplification in ovarian follicle cells of the cluster of chorion-protein genes located at 66D11-15 *(Cp15, Cp16, Cp18* and *Cp19);* located between 615 and 187 base pairs upstream from *Cp18* (Kalfayan, Levine, Orr-Weaver, Parks, Wakimoto, deCicco, and Spradling, 1985, Cold Spring Harbor Symp. Quant. Biol. 50: 527-35).

Acetyl choline receptor: see *Acr*

achaete: see *ac* under *ASC*

Ach: see *emc*^{D}

Acp: Accessory gland protein

Genes inferred from bands on SDS polyacrylamide gels. Six polypeptides are highly polymorphic, exhibiting several electrophoretic variants; these all map to chromosome *2* and are tabulated below. Codominant expression indicates that variants are in structural genes and not attributable to differences in post-translational modification (Whalen and Wilson, 1986, Genetics 114: 77-92).

locus	genetic location	cytological location	molecular mass (kd)
AcpA	2-		165-70
AcpB	2-42.8	36D1-E4	130-140
AcpC$^{\alpha}$	2-53.0		125-128
Acp-g1$^{\alpha}$	2-13.5		145-163
AcpJ	2-		45
AcpK$^{\alpha}$	2-54.1		43

$^{\alpha}$ variants include a null allele.

Acp70A: Accessory gland peptide

location: 3-{40}.

references: Chen, Stumm-Zollinger, Aigaki, Balmer, Bienz, and Böhlen, 1986, Cell 54: 291-98).

phenotype: Encodes a 36-amino-acid peptide that is synthesized in the accessory gland and is transferred to the female where it represses female sexual receptivity and stimulates oviposition. The peptide contains a high concentration of basic amino acids, tryptophan and hydroxyproline as well as an unique residue of unknown nature that is encoded by a leucine codon.

cytology: Placed in 70A by *in situ* hybridization.

molecular biology: Gene cloned and sequenced; conceptual sequence indicates a hydrophobic amino-terminal signal sequence of 19 residues. mRNA for prepeptide accumulates exclusively in the male accessory gland.

Acph-1: Acid phosphatase 1

location: 3-101.1 (between *ca* and *bv*).

discoverer: MacIntyre, 1964.

references: 1966, DIS 41: 61.
1966, Genetics 53: 461-74.

phenotype: Structural gene for acid phosphatase 1 [ACPH-1 (EC3.1.3.2)], the major phosphatase in adults; responsible for approximately 90% of the low-pH nucleotidase activity throughout development. Glycoprotein homodimer with subunit molecular weight of 50,000 daltons. Purification and biochemical characterization by Feigen, Mitrick, Johns, Postlethwait, and Sederoff (1980, J. Biol. Chem. 255: 10338-43). Serves as a reliable cytochemical marker in many tissues (Hall, 1979, Genetics 92: 437-57). Enzyme appears to be produced in nurse cells and follicular cells of ovary and transferred to oocyte through the ring canals and by pinocytosis, respectively (Sawicki and MacIntyre, 1977, Dev. Biol. 60: 1-13); maternally produced enzyme persists to third instar; paternal gene function detectable in gels after 9-10 hr of embryonic development (Yasbin, Sawicki, and MacIntyre, 1978, Dev. Biol. 63: 35-46); and after 5 hr histochemically (Sawicki and MacIntyre, 1978, Dev. Biol. 63: 47-58). Enzyme found in larvae, pupae, and adults; levels increase during adult life (Postlethwait and Gray, 1975, Dev. Biol. 47: 196-205).

alleles: In addition to the information tabulated below, pairwise combinations of *Acph-1*^{n2}, *Acph-1*^{n3}, *Acph-1*^{n6}, and *Acph-1*^{n9} exhibit 20-40% normal levels of cross reacting material (CRM).

allele	origin	derivative of	discoverer	ref$^{\alpha}$	comments
Acph-1^{A}	spont		MacIntyre	*4*	slow
Acph-1^{B}	spont		MacIntyre	*4*	intermediate
Acph-1^{C}	spont		MacIntyre	*4*	fast
Acph-1^{n1}	EMS	*Acph-1*^{B}	MacIntyre	*1*	A-like mobility in heterodimer
Acph-1^{n2}	EMS	*Acph-1*^{A}	MacIntyre	*1*	
Acph-1^{n3}	EMS	*Acph-1*^{B}	MacIntyre	*1*	
Acph-1^{n4}	EMS	*Acph-1*^{A}	MacIntyre	*1*	0-5% normal CRM
Acph-1^{n5}	EMS	*Acph-1*^{A}	MacIntyre	*1*	0-5% normal CRM
Acph-1^{n6}	EMS	*Acph-1*^{B}	MacIntyre	*1*	A-like mobility in heterodimer
Acph-1^{n7}	EMS	*Acph-1*^{B}	MacIntyre	*1*	
Acph-1^{n8}	EMS	*Acph-1*^{A}	MacIntyre	*1*	0-5% normal CRM
Acph-1^{n9}	EMS	*Acph-1*^{B}	MacIntyre	*1*	
Acph-1^{n10}	EMS	*Acph-1*^{A}	MacIntyre	*1*	
Acph-1^{n11}	EMS	*Acph-1*^{B}	MacIntyre	*1*	0-5% normal CRM
Acph-1^{n12}	EMS	*Acph-1*^{B}	MacIntyre	*1*	
Acph-1^{n13}	EMS	*Acph-1*^{B}	MacIntyre	*1*	0-5% normal CRM
Acph-1^{n14}	EMS	*Acph-1*^{A}	MacIntyre	*1*	B-like mobility in heterodimer
Acph-1^{n15}	EMS	*Acph-1*^{A}	MacIntyre	*1*	0-5% normal CRM
Acph-1^{nGB1}	spont	*Acph-1*^{B}		*2, 3*	B-like mobility in heterodimer
Acph-1^{nGB2}	spont	*Acph-1*^{B}		*2, 3*	
Acph-1^{nNC1}	spont	*Acph-1*^{B}		*2, 3*	

$^{\alpha}$ *1* = Bell, MacIntyre, and Olivieri, 1972, Biochem. Genet. 6: 205-16; *2* = Burkhart, Montgomery, Langley, and Voelker, 1984, Genetics 107: 295-306; *3* = Langley, Voelker, Leigh Brown, Ohnishi, Dickson, and Montgomery, 1981, Genetics 99: 151-56; *4* = MacIntyre, 1968, DIS 3: 60.

cytology: Located between 99C5 and 7 based on its deletion by *Df(3R)ca-R14 = Df(3R)99A8-9;99D1-2* but not by *Df(3R)ca-165P = Df(3R)99B2-4;99C5-6* (Frisardi and MacIntyre, 1984, Mol. Gen. Genet. 197: 403-13).

Acr60C: Acetyl choline receptor in 60C

location: 2-{107}.

references: Shapiro, Wakimoto, Subers, and Nathanson, 1989, Proc. Nat. Acad. Sci. USA 86: 9039-43.
Onai, FitzGerald, Arakawa, Gocayne, Urquhart, Hall, Fraser, McCombie, and Venter, 1989, FEBS Lett. 255: 219-25.

phenotype: The structural gene encoding a Drosophila homologue of vertebrate muscarinic acetylcholine recep-

tor (mAChR). When expressed in Y1 adrenal cells it is physiologically active as measured by agonist dependent stimulation of phosphatidylinositol metabolism.

cytology: Placed in 60C7-8 by *in situ* hybridization.

molecular biology: Genomic clone isolated from library using a probes from vertebrate muscarinic acetylcholine receptor genes. Nucleotide sequences of cDNA clones reveal a long open reading frame that encodes a 788-amino-acid protein with calculated molecular weight of 84,807 (Onai *et al.*). The amino-acid sequence shows a number of features characteristic of the muscarinic/adrenergic receptor gene superfamily in vertebrates: three potential N-linked glycosylation sites (Asn 65, 84, and 87), seven putative membrane-spanning domains. It displays a high degree of amino-acid identity with vertebrate muscarinic acetylcholine receptors, ~60% overall and up to 88% in transmembrane regions; the segment between transmembrane domains 5 and 6 is considerably longer than that of vertebrate sequences; also the gene has three introns in the region.

Acr64B

location: 3-{8}.

synonym: *ard.*

references: Hermans-Borgmeyer, Zopf, Ryseck, Hovemann, Betz, and Gundelfinger, 1986, EMBO J. 5: 1503-08.

Wadsworth, Rosenthal, Kammermeyer, Potter, and Nelson, 1988, Mol. Cell Biol. 8: 778-85.

Sawruk, Hermans-Borgmeyer, Betz, and Gundelfinger, 1988, FEBS Lett. 235: 40-46.

phenotype: Structural gene encoding a Drosophila homologue of a subunit of vertebrate nicotinic acetylcholine receptors (nAChR). Antibody raised against *Acr64B* fusion proteins immunoprecipitate one of two high-affinity α-bungarotoxin-binding sites from detergent extracts of Drosophila head membranes (Schloss, Hermans-Borgmeyer, Betz, and Gundelfinger, 1988, EMBO J. 7: 2889-94). *In situ* hybridization localizes *Acr64B* expression to nervous tissue, especially in late embryos, pupae, and newly eclosed adults (Hermans-Borgmeyer, Hoffmeister, Sawruk, Betz, Schmitt, and Gundelfinger, 1989, Neuron. 2: 1147-56).

cytology: Placed in 64B by means of *in situ* hybridization.

molecular biology: Genomic clones identified using a *Torpedo californica* nAChR probe; these hybridize to a 3.2-kb mRNA present at high levels on developmental Northern blots in late embryos and during metamorphosis, periods of neuronal differentiation. Genomic probes used to isolate overlapping cDNA clones. The gene comprises six exons distributed over approximately seven kb of genomic sequence. The predicted mature protein after cleavage of a 24 amino-acid signal sequence, consists of 497 amino acids, has a calculated molecular weight of 57,340 and shows extensive homology to all known nAChR genes of other species along its entire amino acid sequence, conforming most closely to neuronal α subunits, although it lacks the cysteine doublet at residues 201 and 202 characteristic of all other α subunits. It contains four putative transmembrane domains, a potential amphipathic α helix, and a canonical N-glycosylation site Asn48; however, the N-linked glycosylation site found at residue 141 in all vertebrate nAChR's is absent in Drosophila.

Acr96A

location: 3-{83}.

synonym: *ALS: Alpha-Like Subunit.*

references: Bossy, Ballivet, and Spierer, 1988, EMBO J. 7: 611-18.

phenotype: Structural gene encoding a Drosophila homologue of a subunit of vertebrate nicotinic acetylcholine receptors (nAChR); inferred to be homologous to neuronal α subunits based on the cystein doublet at amino-acid residues 201 and 202.

cytology: Placed in 96A by *in situ* hybridization.

molecular biology: Genomic clones identified using as a probe a fragment of the chick neuronal nAChRα2 gene; these hybridize to a 10.5 kb mRNA present at high levels on developmental Northern blots from late embryo to pupation, decreasing in late pupae and adults; genomic probes used to isolate overlapping cDNA clones. The gene comprises ten exons distributed over 54 kb of genomic sequence; combined nucleotide sequence from the cDNA clones defines a single long open reading frame of 1701 nucleotides bracketed by 1282 5′ and 514 3′ nucleotides. The ORF encodes 567 amino acids, which show 40-44% sequence conservation with mammalian neuronal nAChR α subunits and with Drosophila *Acr64B* product. Structural domains homologous to those of vertebrate nAChR subunits include a cystein doublet at residues 201 and 202 that characterizes all α subunits, four transmembrane domains, two potential glycosylation sites (Asn 24 and 212) characteristic of vertebrate neuronal α subunits, and an amphipathic α-helical region in the C-terminal quarter of the polypeptide. In addition, the positions of four Drosophila introns correspond exactly to those of four of seven vertebrate AChR introns.

Activator of SD: see *Rsp*

act: actidione sensitive

location: 3-90 (21 units to the right of *H*).

origin: Naturally occurring allele.

references: Marzluf, 1969, Biochem. Genet. 3: 229-38.

phenotype: *act/act* killed by 0.1 the concentration of actidione (cycloheximide) that *act* $^{+}$-bearing strains survive.

alleles: Recessive allele fixed in Oregon-R and Canton-S strains. Urbana-S and Swedish-b carry *act* $^{+}$.

Act5C: Actin in region 5C

location: 1-{14}.

references: Tobin, Zulauf, Sánchez, Craig, and McCarthy, 1980, Cell 19: 121-31.

Fyrberg, Kindle, Davidson, and Sodja, 1980, Cell 19: 365-78.

Fyrberg, Bond, Hershey, Mixter, and Davidson, 1981, Cell 24: 107-16.

phenotype: Codes for cytoplasmic actin; transcribed throughout development; one of two actin genes transcribed in Kc cells and several other cell lines (Fyrberg, Mahaffy, Bond, and Davidson, 1983, Cell 33: 115-23). One of three actin genes expressed in the wing disc during wing development, each with its characteristic profile. Peak expression at 44h of pupal age, little or no expression at 60h rising again at 80h. 44h peak corresponds to time of maximum change in cell shape (Peterson, Bond, Mitchell, and Davidson, 1985, Dev.

Genet. 5: 219-25). Transcripts present in the preblastoderm embryo suggesting maternal transcription; during blastoderm formation, *Act5C* transcripts accumulate at the periphery of the embryo; local concentrations of transcript observed in anterior and posterior midgut rudiments in stage 13 embryos; hybridization also observed in the developing ventral nervous system. Later in embryogenesis, transcript found in all tissues but with dramatic concentrations of transcripts in the anterior and posterior midgut and the proventriculus. Exon specific probes demonstrate that transcripts containing exon 1 tend to be concentrated in anterior portions of early embryos, including the anterior midgut primordium and the proventriculus, as well as in the posterior midgut primordium; during germ-band extension, small local concentrations of exon 2 transcripts are seen in posterior and ventral regions of the embryo (Burn, Vigoreaux, and Tobin, 1989, Dev. Biol. 131: 345-55).

cytology: Localized to 5C2-5 based on failure of *Act5* specific probe to hybridize to either *Df(1)N73 = Df(1)5C2;5C5-6* or *Df(1)C149 = Df(1)5A8-9;5C5-6* (Sodja, Rizki, Rizki and Zafar, 1982, Chromosoma 86: 293-98).

molecular biology: Genomic clone restriction mapped (Fyrberg *et al.*, 1980) and partially sequenced (Fyrberg *et al.*, 1981). Comparison with cDNA clones indicates the presence of three exons, two of which are included in any cDNA (Bond and Davidson, 1986, Mol. Cell Biol. 6: 2080-88). Partial sequence (Bond and Davidson; Vigoreaux and Tobin, 1987, Genes Dev. 1: 1161-71) indicate that all protein encoding sequences reside in exon 3, that either exon 1 or exon 2 is spliced to exon 3, and that there are three transcription start sites, one in exon 1 and two in exon 2, giving rise to different 5′ untranslated regions; also there are indications of at least three polyadenylation sites generating messages with 3′ untranslated regions of 375, 655, and 945 nucleotides. Using discriminating 5′ and 3′ probes, Burn, Vigoreaux, and Tobin (1989) have shown that transcripts differing in 5′ untranslated regions display different tissue specificities; no 3′ specificities are seen. Exons 1 and 2 are each preceded by a functional promoter (Bond-Matthews and Davidson, 1988, Gene 62: 289-300).

Act42A

location: 2-55.4 (inferred from polytene position).

references: Tobin, Zulauf, Sánchez, Craig, and McCarthy, 1980, Cell 19: 121-31.

Fyrberg, Bond, Hershey, Mixter, and Davidson, 1981, Cell 24: 107-16.

phenotype: Codes for cytoplasmic actin; transcribed throughout development; one of two actin genes transcribed in Kc cells and several other cell lines (Fyrberg, Mahaffy, Bond, and Davidson, 1983, Cell 33: 115-23). One of three actin genes expressed in the wing disc during wing development, each with its characteristic profile. Peak expression at 44h of pupal age, little or no expression at 60h rising again at 80h. 44h peak corresponds to time of maximum change in cell shape (Peterson, Bond, Mitchell, and Davidson, 1985, Dev. Genet. 5: 219-25).

cytology: Located in 42A by *in situ* hybridization.

molecular biology: Genomic clone = λDmA3. Partial nucleotide sequence in Fyrberg *et al.* (1981).

Act57A

location: 2-{93}.

references: Tobin, Zulauf, Sánchez, Craig, and McCarthy, 1980, Cell 19: 121-31.

Fyrberg, Kindle, Davidson, and Sodja, 1980, Cell 19: 365-78.

Fyrberg, Bond, Hershey, Mixter, and Davidson, 1981, Cell 24: 107-16.

phenotype: According to Fyrberg and Davidson, *Act57A* encodes the actin I protein isoform, which is the major actin species of larval intersegmental muscle; also encodes adult cephalic and abdominal muscle (Fyrberg, Mahaffy, Bond and Davidson, 1983, Cell 33: 115-33).

cytology: Localized to 57A by *in situ* hybridization.

molecular biology: Genomic clone = λDmA4; coding region restriction mapped and partially sequenced (Fyrberg *et al.*, 1981). Intervening sequence of approximately 630 base pairs inserted in the glycine codon at amino acid position 13 (Fyrberg *et al.*, 1981).

Act79B

location: 3-{47.5}.

references: Tobin, Zulauf, Sánchez, Craig, and McCarthy, 1980, Cell 19: 121-31.

Fyrberg, Kindle, Davidson, and Sodja, 1980, Cell 19: 365-78.

Fyrberg, Bond, Hershey, Mixter, and Davidson, 1981, Cell 24: 107-16.

Zulauf, Sánchez, Tobin, Rdest, and McCarthy, 1981, Nature 292: 556-58.

Sánchez, Tobin, Rdest, Zulauf, and McCarthy, 1983, J. Mol. Biol. 163: 533-51.

phenotype: According to Zulauf *et al.* (1981), *Act79B* appears to code for actin I, a larval muscle-specific actin. Initial translation product, which migrates as actin II, apparently acetylated to become actin I. Using probe from 3′ transcribed-but-not-translated sequences, Sánchez *et al.* demonstrated two minor peaks of transcription during embryogenesis and major peaks during first and second instar and to a lesser degree in the pupa. Fyrberg, Mahaffy, Bond, and Davidson, (1983, Cell 33: 115-23) on the other hand, find *Act79B* to be expressed in adult legs and tubular muscles of thorax, including direct flight muscles, pleurosternal muscles, and muscles of various leg segments. In addition, *Act79B* is expressed in muscles that support the head and abdomen, in the scutellar pulsatile organ, and in two pairs of abdominal muscles that are present only in male flies (Courchesne-Smith, and Tobin, 1989, Dev. Biol. 133: 313-21). One of three actin genes expressed in wing development each with its characteristic developmental profile; peak activity at 80h of pupal age (Petersen, Bond, Mitchell, and Davidson, 1985, Dev. Genet. 5: 219-25.

cytology: Localized to 79B by *in situ* hybridization.

molecular biology: Genomic clone by Zulauf *et al.* (1981) and as λDmA6 by Fyrberg *et al.* (1981). Coding region restriction mapped and partially sequenced by Fyrberg *et al.* (1981). Intervening sequence of 357 nucleotides within a glycine codon at position 307 (Fyrberg *et al.*, 1981). Coding sequences, intervening sequences and flanking sequences completely sequenced (Sánchez *et al.*). Encodes a 374-amino-acid 43-kd polypeptide which is 95% homologous with the product of *Act88F*

and 91% homologous with rabbit actin.

Act87E

location: 3-{52.3}.

references: Tobin, Zulauf, Sánchez, Craig, and McCarthy, 1980, Cell 19: 121-31.

Fyrberg, Kindle, Davidson, and Sodja, 1980, Cell 19: 365-78.

Fyrberg, Bond, Hershey, Mixter, and Davidson, 1981, Cell 24: 107-16.

phenotype: Encodes actin found in larval muscle and adult cephalic and abdominal muscle (Fyrberg, Mahaffey, Bond, and Davidson, 1983, Cell 33: 115-23).

alleles: No lethal alleles of *Act87E* recovered in a lethal-saturation study (Manseau, Ganetzky, and Craig, 1988, Genetics 119: 407-20).

cytology: Placed in 87E9-12 by *in situ* hybridization; included in *Df(3R)ry619 = Df(3R)87D7-9;87E12-F1,* but not in *Df(3R)ry1168 = Df(3R)87B15-C1;87E9-12* (Manseau *et al.*, 1988).

molecular biology: Genome clone restriction mapped and partially sequenced by Fyrberg *et al.* (1981). Comparison of genomic and cDNA sequence indicates a 556 nucleotide intron in the 5′ untranslated region. Conceptual amino-acid sequence shows ~95% identity with other Drosophila actins (Manseau *et al.*, 1988).

Act88F

location: 3-57.1 (based on 41 *cu-sr* and 84 *red-e* recombinants).

references: Tobin, Zulauf, Sánchez, Craig, and McCarthy, 1980, Cell 19: 121-31.

Fyrberg, Kindle, Davidson, and Sodja, 1980, Cell 19: 365-78.

Fyrberg, Bond, Hershey, Mixter, and Davidson, 1981, Cell 24: 107-16.

Sánchez, Tobin, Rdest, Zulauf, and McCarthy, 1983, J. Mol. Biol. 163: 533-51.

phenotype: Structural gene encoding actin III; expressed only in the developing thorax, specifically in the indirect flight muscles (Fyrberg, Mahaffey, Bond, and Davidson, 1983, Cell 33: 115-23; Hiromi and Hotta, 1985, EMBO J. 4: 1681-87). The only actin expressed in indirect flight muscle (Fyrberg). Heterozygotes for null mutations or *Act88F* deficiencies are flightless; flightlessness is apparently caused by imbalance between myosin heavy chains and actin III; whereas hemizygosity for either *Mhc* or *Act88F* leads to complex myofibrillar defects and flightlessness, double hemizygotes have nearly normal fibrillar structure and are able to fly [Beall, Sepanski, and Fyrberg, 1989, Genes Dev. 3: 131-40 (fig.)]. Major peaks of transcription during pupal stage (Sánchez *et al.*, 1983). Heterozygotes and to a greater degree, homozygotes and heteroallelic heterozygotes for antimorphic alleles *Act88F*^{4} and *Act88F*^{5} show constitutive synthesis of heat-shock proteins, with HSP26 and HSP27 less actively synthesized than HSP22, HSP70, and HSP84; response to heat shock normal (Hiromi and Hotta).

alleles:

allele	origin	synonym	ref[α]	comments
***Act88F*^{1}**	EMS	*Ifm(3)1, Ifm(3)2*	*3, 4, 7*	dominant antimorphic allele weak inducer of HSP arg 28 → cys
***Act88F*^{2}**	EMS	*Ifm(3)4*	*3, 4, 7*	dominant antimorphic allele weak inducer of HSP ile 76 → phe
***Act88F*^{3}**	EMS	*Ifm(3)6*	*1, 7*	
***Act88F*^{4}**	EMS	*Ifm(3)7, Act88F*^{KM75}	*3, 7, 8*	dominant antimorphic allele strong inducer of HSP trp 356 → opal
***Act88F*^{5}**		*Act88F*^{HH5}	*2, 8*	dominant antimorphic allele strong inducer of HSP gly 366 → ser
***Act88F*^{6}**		*Act88F*^{KM88}	*2, 8*	dominant hypomorphic allele trp 75 → ambera
***Act88F*^{7}**		*Act88F*^{KM129}	*2, 8*	
***Act88F*^{8}**	spont	*Act88F*^{rsd}	*5, 6*	

α *1* = Ball, Karlik, Beall, Saville, Sparrow, Bullard, and Fyrberg, 1987, Cell 51: 221-28; *2* = Hiromi and Hotta, 1985, EMBO J. 4: 1681-87; *3* = Karlik, Coutu, and Fyrberg, 1984, Cell 38: 711-19 (fig.); *4* = Karlik, Saville, and Fyrberg, 1987, Mol. Cell Biol. 7: 3084-91; *5* = Lang, Wyss, and Eppenberger, 1981, Nature 291: 506-08; *6* = Mahaffey, Coutu, Fyrberg, and Inwood, 1985, Cell 40: 101-10; *7* = Mogami and Hotta, 1981, Mol. Gen. Genet 183: 409-17; *8* = Okamoto, Hiromi, Ishikawa, Yamada, Isoda, Mackasa, and Hotta, 1986, EMBO J. 5: 589-96.

cytology: Placed in 88F by *in situ* hybridization.

molecular biology: Genomic clones isolated by Tobin *et al.* (1980) and by Fyrberg *et al.* (1981). Sequence analysis reveals a translated sequence accounting encoding a 374-amino-acid polypeptide of molecular weight 43 kd, which shows 95% homology with the *Act79B* gene product and 92% homology with rabbit actin. The genomic sequence shows an intron of 60 nucleotides within codon 307 (Sánchez *et al.*, 1983). Deletion analysis of upstream *cis*-acting regulatory sequences carried out by Geyer and Fyrberg (1986, Mol. Cell Biol. 6: 3388-96). Arthrin, a 55-kd protein found in indirect flight muscle shown to be an uniquinated form of actin III (Ball, Karlik, Beall, Saville, Sparrow, Bullard, and Fyrberg, 1987, Cell 51: 221-28).

Act88F^{4}

phenotype: Dominant flightless allele; actin III replaced by a truncated polypeptide of 42 kd that is stable and capable of incorporation into myofibrils; actin II reduced in homozygotes (Hiromi and Hotta, 1985). Myofibrils in indirect flight muscles of homozygotes severely deranged; sarcomere structure obliterated; indirect-flight-muscle nuclei enlarged. Skeins of morphologically normal but highly disorgainzed thick filaments present, but Z discs absent. Thin filaments scarce. Electron dense material of unknown origin seen in sections. Wild-type flies transformed with cloned *Act*^{4} sequence produces both the 42-kd and the heat-shock proteins (Hiromi, Okamoto, Gehring, and Hotta, 1986, Cell 44: 293-301).

Act88F^{5}

phenotype: Produces half normal amount of actin isoform III; shows increased synthesis of actin I, normally present in only trace amounts in indirect flight muscle. Indirect-flight-muscle nuclei enlarged and myofibrils disrupted. Heterozygotes flightless.

Act88F^{6}

phenotype: Actin III entirely absent from indirect flight muscle in homozygotes; levels of actin II also reduced.

Act88F^{7}

phenotype: Actin III replaced by a truncated polypeptide of 38 kd; its low concentrations on gels suggests high instability (Hiromi and Hotta, 1985).

Act88F^{8}

phenotype: Studied only in combination with *rsd* at 95.4 on chromosome *3*; not examined in *rsd*$^{+}$ background. Wings of homozygotes held straight up, nearly meeting over thorax; heterozygotes have wings held normally, but are nearly flightless. Electron microscropy of homozygotes reveals grossly abnormal indirect-flight-muscle structure; lack thin filaments and Z discs (Deak, Bellamy, Bienz, Dubuis, Fenner, Gollin, Rahmi, Ramp, Reinhardt, and Cotton, 1982, J. Embryol. Exp. Morphol. 69: 61-81). Abnormal protein accumulation observed in thoraces. Actin III and its ubiquinated derivative, arthrin, absent in *Act88F*^{8} homozygotes (Lang *et al.*); six other polypeptides, including an indirect-flight-muscle tropomyosin isoform and two indirect-flight-muscle tropomyosin-related isoforms, markedly reduced. Homozygotes transformed with *Act88F*$^{+}$ show restoration to approximately normal levels of the six reduced polypeptides. Accumulation of actin III and arthrin still negative, however; the latter attributed to the failure of post-translational modification in the presence of homozygous *rsd*. Viability and fertility normal.

molecular biology: a null mutant; *Act88F* mRNA reduced 10-15 fold; alteration of normal sequence apparently outside the coding region; mRNA level, and to some degree, the phenotype rescuable by germ-line transformation using *Act88F* normal genomic sequence. Sequence of coding region of DNA including 60-nucleotide intron reveals differences from that of Canton-S that account for five amino-acids substitution (Mahaffey *et al.*).

other information: Conceivable that *Act88F*^{9} is a wild-type isoallele with normal phenotype in the absence of *rsd*.

actidione sensitive: see ***act***

Actin: see ***Act***

Actn: α ***Actinin***

location: 1-1.0.

synonym: *l(1)2Cb*

phenotype: The structural gene for α Actinin (Fyrberg). Both lethal and viable alleles recovered; allelism determined by Homyk and Emerson. Viable alleles unconditionally flightless; wing position normal, but unable to fly or beat wings; jump abnormally short distances. Gynandromorph studies of *Actn*^{1} indicate a bilateral pair of submissive foci located mid ventrally close to the embryonic midline (Homyk and Emerson). ERG normal (Homyk and Pye, 1989, J. Neurogenet. 5: 37-48). *Actn*^{4} is a heat sensitive lethal, and when raised at low temperature, causes aberrant wing display of courting males; *Actn*^{1}/*Actn*^{4} jumps and flies abnormally when raised at 22° but normally when raised at 29°; temperature sensitive period for this effect in first half of pupal stage (Homyk *et al.*,1980). Trans heterozygotes (i.e., *Actn*^{3}, *Actn*^{4}, *Actn*^{8}, and *Actn*^{14}) with *hdp-a*^{2} also flightless. Lethal alleles die in late larval or early pupal stages; homozygous maternal germline clones produce normal ova. Polyphasic lethality of *Actn*^{8} attributed to position effect of the inversion on arm (Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41).

alleles:

allele	origin	discoverer	synonym	ref α	comments
Actn^{1}	EMS	Homyk	*fliA*^{1}	*1, 2, 3*	
Actn^{2}	EMS	Homyk	*fliA*^{2}	*1, 2, 3*	
Actn^{3}	EMS	Homyk	*fliA*^{3}	*1, 2, 3*	
Actn^{4}	EMS	Homyk	*fliA*^{4}	*1, 2, 4*	heat-sensitive pupal lethal
Actn^{5}	X ray	Lefevre	*l(1)A115*	*6, 8*	larval lethal
Actn^{6}	X ray	Lefevre	*l(1)C212*	*6*	*T(1;3)1A7;2C3;80*
Actn^{7}	X ray	Lefevre	*l(1)GA17*	*6, 8*	embryonic lethal (double mutant)
Actn^{8}	X ray	Lefevre	*l(1)HC207*	*1, 6, 8*	*In(1)2C3;7B1;* polyphasic lethal
Actn^{9}	X ray	Lefevre	*l(1)HC288*	*6, 8*	larval-pupal lethal
Actn^{10}	X ray	Lefevre	*l(1)HF356*	*6*	
Actn^{11}	X ray	Lefevre	*l(1)JC111*	*6*	*In(1)2C3;9A2-3*
Actn^{12}	EMS	Lefevre	*l(1)EA43*	*7, 8*	larval lethal
Actn^{13}	EMS	Lefevre	*l(1)EA45*	*7*	
Actn^{14}	EMS	Lefevre	*l(1)EA82*	*1, 7, 8*	larval lethal
Actn^{15}	EMS	Lefevre	*l(1)EA111*	*7*	
Actn^{16}	EMS	Lefevre	*l(1)VE692*	*7, 8*	larval lethal
Actn^{17}	spont	Schalet	*l(1)4-3*		
Actn^{18}	spont	Schalet	*l(1)17-44-1*		
Actn^{19}	HMS		*l(1)HM29*	*5*	

α *1* = Homyk and Emerson, 1988, Genetics 119: 105-21; *2* = Homyk and Grigliatti, 1983, Dev. Genet. 4: 77-97; *3* = Homyk and Sheppard, 1977, Genetics 87: 95-104; *4* = Homyk, Szidonya, and Suzuki, 1980, Mol. Gen. Genet. 177: 553-65; *5* = Kramers, Schalet, Paradi, and Huiser-Hoogteyling, 1983, Mutat. Res. 107: 187-201; *6* = Lefevre, 1981, Genetics 99: 461-80; *7* = Lefevre and Watkins, 1986, Genetics 113: 869-95; *8* = Perrimon, Engstrom, and Mahowald, 1985, Genetics 111: 23-41.

cytology: Placed in 2C3 based on breakpoint common to three rearrangement associated lethal alleles (Lefevre). Covered by *Dp(1;3)w*^{vco} = *Dp(1;3)2B17-C1;3C4-5;77D3-5;81* but not by *Dp(1;Y)w*$^{+303}$ = *Dp(1;Y)2D1-2;3D3-4* (Perrimon, *et al.*).

ad: arcoid

location: 2-60.7.

origin: Spontaneous.

discoverer: Curry, 38a2.

references: 1939, DIS 12: 45.

phenotype: Wings arched, broad, and somewhat shortened; crossveins close; scutellar groove shallow. Legs may be slightly shorter than wild type. RK3.

add-B: see *dmd*^{2}

Additional sex combs: see ***Asx***

ade1: adenosine1

location: 1-57 (right of *f*).

origin: Induced by ethyl methanesulfonate.

synonym: *ade1-1*^{sd}.

references: Falk and Nash, 1974, Genetics 76: 755-766.

phenotype: Eclosion delayed 2 or 3 days; delay abolished by supplementation of minimal medium with adenosine or guanosine.

ade2 *(S. Henikoff and D. Nash)*

location: 2-17.7 [based on 73 *cl-spd* recombinants (Keizer, Nash, and Tiong, 1989, Biochem. Genet. 27: 349-53)].

references: Johnstone, Nash, and Naguib, 1985, Biochem. Genet. 23: 539-55.

Henikoff, Nash, Hards, Bleskan, Woolford, Naguib, and Patterson, 1986, Proc. Nat. Acad. Sci. USA 83: 3919-23.

Tiong, Keizer, Nash, Bleskan, and Patterson, 1989, Biochem. Genet. 27: 333-48.

phenotype: Purine nucleoside auxotroph supplementable with adenine, adenosine, and inosine. Eye color reddish-brown similar to rosy. Lacks detectable levels of the fourth purine *de novo* synthetic-pathway enzyme, formylglycineamide ribotide amidotransferase (FGARAT; EC 6.33.5.3). Homozygotes and heteroallelic combinations of many alleles have defective wings; the defects include reduced wing size, deranged posterior wing margins, and extra wing veins. Macrochaetae are somewhat thinner than normal and reduced in length. *ade2*^{5}, *ade2*^{6}, and *ade2*^{7} are sterile, perhaps owing to general debility, when homozygous or in heteroallelic combination with one another. Only *ade2*^{7} was not tested owing to a linked lethal. Most homozygotes and heteroallelic heterozygotes display reduced viability, with the appearance of pharate adults unable to eclose.

alleles:

allele	origin	discoverer	synonym	ref $^{\alpha}$	comments
***ade2*^{1}**	EMS	Naguib		*2, 3*	weak allele
***ade2*^{2}**	γ ray			*5*	
***ade2*^{3}**	γ ray			*5*	
***ade2*^{4}**	γ ray			*5*	
***ade2*^{5}**	γ ray			*5*	
***ade2*^{6}**	γ ray			*5*	
***ade2*^{7}**	γ ray			*5*	
***ade2*^{8}**	γ ray			*5*	*In(2LR)26B;* 40-41;57B-C
***ade2*^{9}**	γ ray			*5*	*T(2;3)26B1-2;97D*
***ade2*^{10}**	spont	Bryson, 1939	*pym*^{1}	*1, 5*	
****ade2*^{11}**	spont	Neel, 1941	*pym*^{2}	*4*	

$^{\alpha}$ *1* = Bryson, 1940, DIS 13: 49; *2* = Henikoff, Nash, Hards, Bleskan, Woolford, Naguib, and Patterson, 1986, Proc. Nat. Acad. Sci. USA 83: 3919-23; *3* = Johnstone, Nash, and Naguib, 1985, Biochem. Genet. 23: 539-55; *4* = Neel, 1942, Am. Nat. 76: 630-34; *5* = Tiong, Keizer, Nash, Bleskan, and Patterson, 1989, Biochem. Genet. 27: 333-48.

cytology: Placed in 26B, probably 26B1-2, based on breakpoints common to *In(2LR)ade*^{8} = *In(2LR)26B;40-41;57B-C* and *T(2;3)ade*^{9} = *T(2;3)26B1-2;97D*. Also included in *Df(2L)ade2-1* = *Df(2L)25F;26B5-6*, *Df(2L)ade2-2* = *Df(2L)25F2-3;26D-E*, and *Df(2L)ade2-3* = *Df(2L)26A;26B5-6*.

ade3 (S. Henikoff and D. Nash)

location: 2-20.

origin: Induced by ethyl methanesulfonate.

discoverer: Nash.

synonym: *Gart*.

references: Johnstone, Nash, and Naguib, 1985, Biochem. Genet. 23: 539-55.

Henikoff, Nash, Hards, Bleskan, Woolford, Naguib, and Patterson, 1986a, Proc. Nat. Acad. Sci. USA 83: 3919-23.

Henikoff, Keene, Sloan, Bleskan, Hards, and Patterson, 1986b, Proc. Nat. Acad. Sci. USA 83: 720-24.

phenotype: Purine nucleoside auxotroph supplementable with adenine, adenosine, and inosine. Recovery of *ade3* progeny from crosses between *ade3* and *ade3/SM5* is about 1% when raised on minimal medium. Less than 3% of the normal activity purine *de novo* synthetic pathway enzyme, glycineamide ribotide transformylase [EC 2.1.2.2 (GART)]. Eye color normal.

cytology: 27C by means of *in situ* hybridization of cloned sequence.

molecular biology: Corresponds to the cloned sequence selected by Henikoff, Keene, Tatchell, Hall, and Nasmyth [1981, Nature (London) 289: 37] by its ability to complement *ade8* in yeast, which codes for GART. Seven exons specify a 4.7 kb mRNA encoding the second, third, and fifth *de novo* purine biosynthetic-pathway enzyme activities, glycineamide ribotide synthetase [EC 6.3.4.13 (GARS)], aminoimidazole ribotide synthetase [EC 6.3.31 (AIRS)], and GART, on a 1353 amino-acid polypeptide. The first four exons also specify a 1.7 kb mRNA encoding Gars alone on a 434 amino acid polypeptide (Henikoff *et al.*). This smaller polypeptide is identical to the NH_2-terminal portion of the larger, except for the last amino acid, as a consequence of alternative processing of the primary transcript. The *ade3* mutation is a single base transition changing a conserved glycine (found at that position in yeast *ade8*) to a serine at amino acid 1164 of the large polypeptide.

A functional pupal cuticle protein gene is found within the first intron (interrupting the GARS domain), is encoded on the other DNA strand, and is itself interrupted by a single intron between codons 4 and 5 of a 184 amino-acid open reading frame. This intronic gene *(Pcp)* is expressed primarily in abdominal epidermal cells that secrete the pupal cuticle (Henikoff, Keene, Fechtel, and Fristrom, 1986, Cell 44: 33-42)

Adenine phosphoribosyl transferase: see *Aprt*

Adenylate kinase C: see *Adk-C*

Adh: Alcohol dehydrogenase (M. Ashburner)

location: 2-50.1.

references: Johnson and Denniston, 1964, Nature (London) 204: 906-07.

Grell, Jacobson, and Murphy, 1965, Science 149: 80-82.

Ursprung and Leone, 1965, J. Exp. Zool. 160: 147-54.

phenotype: Structural gene for alcohol dehydrogenase [ADH (EC 1.1.1.1)]. Natural populations are polymorphic for three electrophoretic alleles (*Adh*^{F}, *Adh*^{S}, *Adh*$^{F\text{-}ChD}$) and for three rarer electrophoretic alleles (*Adh*^{US}, *Adh*$^{F'}$, *Adh*^{UF}). The frequency of the *Adh*^{F} allele increases, at the expense of *Adh*^{S}, with increasing latitude in both northern and southern hemispheres [Johnson and Schaffer, 1973, Biochem. Genet. 10: 149-63; Vigue and Johnson, 1973, Biochem. Genet. 9: 213-27; Wilks, Gibson, Oakeshott and Chambers, 1980, Aust. J. Biol. Sci. 33: 575-85; Anderson, 1981, Genetic Studies of Drosophila Populations (Gibson and Oakes, eds.). Australian National University Press, pp. 237-50; Anderson and Chambers, 1982, Evolution 36: 86-96].

Confers resistance to ethanol; flies lacking ADH rapidly become intoxicated and eventually die on exposure to ethanol (Grell, Jacobson and Murphy, 1968, Ann. N.Y. Acad. Sci 151: 441-45; Vigue and Sofer, 1976, Biochem. Genet. 14: 127-135; David, Bocquet, Arens and Fouillet, 1976, Biochem. Genet. 14: 989-97). However, ethanol sensitivity is complex since even *Adh* nulls are more resistant to ethanol when young than when old (Vigue and Sofer, 1976; Tsubota). *Adh*$^{+}$ flies are killed by low concentrations of unsaturated secondary alcohols (*e.g.* 1-penten-3-ol; 1-pentyn-3-ol) but not by unsa-

turated primary alcohols (*e.g.* 1-penten-1-ol) (Sofer and Hatkoff, 1972, Genetics 72: 545-49), presumably due to the formation of toxic ketones. This allows the chemical selection of *Adh* nulls (Sofer and Hatkoff, 1972; O'Donnell, Gerace, Leister and Sofer, 1975, Genetics 79: 73-83). ADH may play a metabolic role independent of alcohol detoxication, *i.e.* in the metabolism of higher alcohols (see Winberg, Thatcher and McKinley-McKee, 1982, Biochem. Biophys. Acta 704: 7-16). ADH also catalyses the oxidation of acetaldehyde to acetate (Heinstra, Eisses, Schoonen, Aben, de Winter, van de Horst, van Marrewijk, Beenakkers, Scharloo and Thörig, 1983, Genetica 60: 129-37; Moxon, Holmes, Parsons, Irving, and Doddrell, 1985, Comp. Biochem. Physiol. 80B: 525-35).

Specific activity of ADH changes with development, with peaks at the end of the third larval instar and about four days after eclosion (Ursprung, Sofer and Burroughs, 1970, Wilhelm Roux's Arch. Entwicklungsmech. Org. 164: 201-08; Dunn, Wilson and Jacobson, 1969, J. Exp. Zool. 171: 185-90; Leibenguth, Rammo and Dubiczky, 1979, Wilhelm Roux's Arch. Dev. Biol. 187: 81-88; Maroni and Stamey, 1983, DIS 59: 77-79; Anderson and McDonald, 1981, Canad. J. Genet. Cytol. 23: 305-13). Most of the activity is in the larval fat body and gut and the adult fat body (Ursprung, Sofer and Burroughs). Maternal inheritance of ADH by embryos and larvae (O'Donnell *et al.;* Leibenguth *et al.*). Half life of ADH-F *in vivo* estimated as 55.3 hours (Anderson and McDonald, 1981, Biochem. Genet. 19: 411-19). Not expressed in SL2 tissue culture cells, but transfected cloned gene is (Benyajati and Dray, 1984, Proc. Nat. Acad. Sci. 1701-05).

Ethanol tolerance usually correlated with ADH activity and polymorphic experimental populations exposed to ethanol usually show an increase in the frequency Adh^F (McDonald and Avise, 1976, Biochem. Genet. 14: 347-55; Cavener and Clegg, 1978, Genetics 90: 629-44; van Delden, Kamping and van Dijk, 1975, Experientia 31: 418-19; Oakeshott, Gibson, Anderson and Champ, 1980, Aust. J. Biol. Sci. 33: 105-14; McDonald, Chambers, David and Ayala, 1977, Proc. Nat. Acad. Sci. USA 74: 4562-66). Flies carrying Adh^F tend to be more resistant than those carrying only Adh^S to ethanol [Kamping and van Delden, 1978, Biochem. Genet. 16: 541-55; Ainsley and Kitto, 1975, Isozymes (C. Markert, ed.). Academic Press, Vol. II, pp. 733-43; Briscoe, Robertson and Malpica, 1975, Nature (London) 253: 148-49].

Electrophoresis of homozygous genotypes usually reveals three interconvertable isozymes [Ursprung and Leone; Johnson and Denniston; Grell *et al.*, 1965; Ursprung and Carlin, 1968, Ann. N.Y. Acad. Sci. 151: 456-75; Jacobson, Murphy and Hartmann, 1970, J. Biol. Chem. 245: 1075-83; Jacobson and Pfuderer, 1970, J. Biol. Chem. 245: 3938-44; Jacobson, Murphy and Ortiz, 1972, Arch. Biochem. Biophys. 149: 22-35; Knopp and Jacobson, 1972, Arch. Biochem. Biophys. 149: 36-41; Schwartz, Gerace, O'Donnell and Sofer, 1975, Isoenzymes (C. Markert, ed.). Academic Press, Vol. I, pp. 725-51]. These vary in activity and stability, the most cathodal being more active, but less stable, than the more anodal forms. They probably result from the binding of 0, 1 or 2 moles per mole of a NAD^+ addition complex with a carbonyl compound [Schwartz and Sofer, 1976, Nature (London) 263: 129-31; Schwartz, O'Donnell and Sofer, 1979, Arch. Biochem. Biophys. 194: 365-78; Winberg, Thatcher and McKinley-McKee, 1983, Biochem. Genet. 21: 63-80]. Feeding flies acetone, propan-2-ol, or 3-hydroxy-butanone, for example, converts isozymes to most anodal form and results in loss of enzyme activity *in vitro* and *in vivo* (Schwartz and Sofer, 1976; Papel, Henderson, van Herrewege, David and Sofer, 1979, Biochem. Genet. 17: 533-63). ADH has been purified (Sofer and Ursprung, 1968, J. Biol. Chem. 243: 3118-25; Schwartz *et al.*, 1975; Thatcher, 1977, Biochem. J. 163: 317-23; Leigh Brown and Lee, 1979, Biochem. J. 179: 479-82; Juan and Gonzalez-Duarte, 1980, Biochem. J. 189: 105-10; Elliot and Knopp, 1975, Methods Enzymol. 41: 374-79; Chambers, 1984, Biochem. Genet. 22: 529-50). It is a homodimer with monomeric subunit molecular weight of 27500 daltons (Thatcher, 1980, Biochem. J. 187: 875-83); molecular extinction coefficient 4.8×10^4 liter/mol/cm (Juan and Gonzalez-Duarte, for ADH-S). Complete amino acid sequence determined by Thatcher (1980; see also Schwartz and Jornvall, 1976, Europ. J. Biochem. 68: 159-68; Auffret, Williams and Thatcher, 1978, FEBS Lett. 90: 324-26; Benyajati, Place, Powers, and Sofer, 1981, Proc. Nat. Acad. Sci. USA 78: 2317-21; Chambers, Laver, Campbell and Gibson, 1981, Proc. Nat. Acad. Sci. USA 78: 3103-07) with secondary structure predictions (Thatcher and Sawyer, 1980, Biochem J. 187: 884-86; Benyajati *et al.*, 1981). Limited homology in supposed catalytic region with ribitol dehydrogenase of Klebsiella (Jornvall, Persson and Jeffry, 1981, Proc. Nat. Acad. Sci. USA 78: 4226-30).

ADH shows a broad substrate specificity but is more active (by at least a factor of 5) with secondary than primary alcohols and shows highest activity to 3-6 carbon alcohols (Sofer and Ursprung; Thatcher and Camfield, 1977, Winberg *et al.*, 1982, Chambers *et al.*). Differences in substrate specificity, kinetic constants and stability of different electrophoretic variants often reported (Anderson and McDonald, 1983, Proc. Nat. Acad. Sci. USA 80: 4798-802). Considerable heterogeneity in the specific activity of ADH within and between different Adh^F and Adh^S strains, though Adh^S strains tend to be lower than Adh^F [Day, Hillier and Clarke, 1974, Biochem. Genet. 11: 141-53, 155-65; Day and Needham, 1974, Biochem. Genet. 11: 167-75; Gibson, 1970, Nature (London) 227: 959-61; Gibson, Chambers, Wilkes and Oakeshott, 1980, Aust. J. Biol. Sci. 33: 479-89; Gibson and Miklovitch, 1971, Experientia 27: 99-100; Kreitman, 1980, Genetics 95: 467-75; Oakeshott, 1976, Aust. J. Biol. Sci. 29: 365-73; Sampsell, 1977, Biochem. Genet. 15: 971-88; Sampsell and Sims, 1982, Nature (London) 296: 853-55; Thörig, Schoone and Scharloo, 1975; Biochem. Genet. 13: 721-31; Vigue and Johnson; Hewitt, Pipkin, Williams and Chakrabartty, 1974, J. Hered. 65: 141-44; Ward, 1974, Biochem. Genet. 12: 449-58; Ward, 1975, Genet. Res. 26: 81-93; Maroni, Laurie-Ahlberg, Adams and Wilton, 1982, Genetics 101: 431-66; Rasmuson, Nilson and Zeppezauer, 1966, Hereditas 56: 313-16; Clarke, Camfield, Garvin and Pitts, 1979, Nature (London) 180: 517-18; Laurie-Ahlberg, Maroni, Bewley, Lucchesi and Weil, 1980, Proc. Nat. Acad. Sci. USA 77: 1073-77; Barnes

and Birley, 1978, Heredity 40: 51-57; Barnes and Birley, 1978, Biochem. Genet. 16: 155-65; McDonald and Ayala, 1978, Genetics 89: 371-88; McDonald *et al.*, 1980; Lewis and Gibson, 1978, Biochem. Genet. 16: 159-70]. With the exception of the studies by Thatcher and Sheik (1981, Biochem. J. 197: 111-17), Winberg *et al.* (1982), McDonald, Anderson and Santos (1980, Genetics 95: 1013-22); Eisses, Schoonen, Aben, Scharloo, and Thörig (1985, Mol. Gen. Genet. 199: 76-81) and Moxon *et al.* (1985), these were all done with crude extracts and not purified enzyme. Thatcher and Sheikh find the relative thermostabilities to be ADH-S > ADH-F > ADH-n5 > ADH-D. ADH-S shows slower dissociation of NADH from NADN-enzyme complex than ADH-F (Winberg, Hovik, and McKinley-McKee, 1985, Biochem. Genet. 23: 205-16).

ADH is not a metalloenzyme (Place, Powers and Sofer, 1980, Fed. Proc. 39: 1640); but, paradoxically, is inhibited by certain metal ion chelators, *e.g.* pyrazole (Place, Powers and Sofer; Winberg *et al.*, 1982; Moxon *et al.*, 1985).

Utilization of ethanol as an energy source (van Herrewege and David, 1974, C. Rend. Acad. Sci. Paris 279D: 335-38; van Herrewege, David and Grantham, 1980, Experientia 36: 846-47; Libion-Mannaert, Delcour, Deltombe-Lietaert, Lenelle-Montfort and Elens, 1976, Experentia 32: 22-23) depends on ADH activity (David, Bocquet, van Herrewege, Fouillet and Arens, 1978, Biochem. Genet. 16: 203-11). Adh^F homozygotes usually show a better ability to survive on ethanol as a sole energy source than Adh^S homozygotes (Daly and Clarke, 1981, Heredity 46: 219-26; Anderson, McDonald and Santos, 1981, Experientia 37: 463-64). Adh^F and Adh^S homozygotes also show behavioural differences in their response to ethanol (Parsons, 1977 Oecologia 30: 141-46; Cavener, 1979, Behav. Genet. 9: 359-65; Gelan and McDonald, 1980, Behav. Genet. 10: 237-49; Hougonto, Lietaert, Libion-Mannaert, Feytmans and Elens, 1982, Genetica 58: 121-28; Parsons, 1980, Behav. Genet. 10: 183-90; Parsons, 1980, Experientia 36: 1070-71).

D. simulans enzyme monomers form heterodimers with those of *D. melanogaster* (E.H. Grell); *D. simulans* enzyme purified (Juan and Gonzalez-Duarte, 1981, Biochem. J. 195: 61-69). Sequence of *D. simulans* ADH (from DNA) similar to that of Adh^S with following changes: ser1 → ala1; gln82 → lys82; ile184 → val184 (Bodmer and Ashburner, 1984, Nature 309: 425-30). *D. simulans* and *D. melanogaster* enzymes differentially regulated in hybrids (Dickenson, Rowan, and Brennan, 1984, Heredity 52: 215-25). The *Adh* genes from *D. orena* and *D. mauritiana* have also been sequenced (Bodmer and Ashburner), and those of *D. erecta, D. teissieri* and *D. yakuba* mapped with restriction enzymes (Langley, Montgomery and Quattlebaum, 1982, Proc. Nat. Acad. Sci. USA 79: 5631-35).

alleles: Large numbers of alleles have been selected and characterized. This information is summarized in the following tables: The first table describes the origins and phenotypes of the electrophoretic variants, the majority of which were isolated from natural populations; the second describes the origins and phenotypes of the null alleles; In addition 16 isolations of null alleles from four Australian locations have been described (Freeth and Gibson, 1985, Heredity 55: 369-74); not clear how many mutational events represented.

allele	origin	source	discoverer	ref α	migration β rate (pI)	thermo stability
Adh ^{71k} γ	spont		Thörig	*5, 6, 13, 22*	(6.4)	> Adh ^{F}
Adh ^{A1} δ	recomb.	*Adh* ^{n1} /*Adh* ^{n5}	Maroni	*13, 15*	(7)	< Adh ^{S}
Adh ^{B7} δ	recomb.	*Adh* ^{n1} /*Adh* ^{n5}	Maroni	*13, 15*	(7)	< Adh ^{S}
Adh ^{D}	EMS	*Adh* ^{F}	Grell	*9, 19*	(6)	
Adh ^{F}	spont		Johnson and Denniston	*1, 7, 12, 16, 20*	6.4	
Adh $^{F'}$	spont	(Congo)	David	*4, 17, 20*	6.5	
Adh $^{F(o)}$ γ ε	spont		Eisses	*6*	(6.4)	> Adh ^{F}
Adh $^{F\text{-}ChD}$ ε	spont		Lewis	*2, 3, 8, 14, 24*	(6.4)	> Adh ^{F} ; > Adh ^{S}
Adh ^{Fm} ζ	spont		Sampsell	*18*	(6.4)	
Adh ^{Fr} ε	spont		Sampsell	*13, 18*	(6.4)	> Adh ^{F}
Adh ^{Fs}	spont		Sampsell	*13, 18*	(6.4)	< Adh ^{F}
Adh ^{I} ζ	spont		Ursprung and Leone	*11, 23*	(6.4)	
Adh ^{II} η	spont		Ursprung and Leone	*11, 23*	(7)	
Adh ^{S}	spont		Johnson and Denniston		7	
Adh ^{Sm} η	spont		Sampsell	*18*	(7)	
Adh ^{Ss}	spont		Sampsell	*13, 18*	(7)	< Adh ^{F}
Adh ^{UF}	spont			*20, 21*	6.0	
Adh ^{US}	spont	(Congo)	David	*4, 10, 20*	7.8	

α *1* = Benyajati, Place, Powers, and Sofer, 1981, Proc. Nat. Acad. Sci. USA 78: 2717-21; *2* = Chambers, Laver, Campbell, and Gibson, 1981, Proc. Nat. Acad. Sci. USA 78: 3103-07; *3* = Chambers, Wilks, and Gibson, 1981, Aust. J. Biol. Sci. 34: 625-37; *4* = David, 1978, Recherche 9: 482-83; *5* = Eisses, Schoonen, Scharloo, Thörig, 1985, Comp. Biochem. Physiol. 82: 863-68. *6* = Eisses, Thörig, and Scharloo, 1981, Genetics 97: s33; *7* = Fletcher, Ayala, Thatcher, and Chambers, 1978, Proc. Nat. Acad. Sci. USA 75: 5609-12; *8* = Gibson, Chambers, Wilkes, and Oakeshott, 1980, Aust. J. Biol. Sci. 33: 479-89; *9* = Grell, Jacobson, and Murphy, 1968, Ann. NY Acad. Sci. 151: 441-55; *10* = Grossman, Koreneva, and Ulitscaya, 1970, Genetika (Moscow) 6(2): 91-96; *11* = Hewitt, Pipkin, Williams, and Chakrabartty, 1974, J. Hered. 65: 141-48; *12* = Johnson and Denniston, 1964; Nature (London) 204: 906-07; *13* = Kreitman, 1980, Genetics 95: 467-75; *14* = Lewis and Gibson, 1978, Biochem. Genet. 16: 159-70; *15* = Maroni, 1978, Biochem. Genet. 16: 509-23; *16* = Retzios and Thatcher, 1979, Biochemie 61: 701-04; *17* = Retzios and Thatcher, 1980, Biochem. Soc. Trans. 9: 298-99; *18* = Sampsell, 1977, Biochem. Genet. 15: 971-88; *19* = Schwartz and Jornvall, 1976, Europ. J. Biochem. 68: 159-68; *20* = Thatcher, 1980, Biochem. J. 187: 875-83; *21* = Thatcher and Camfield, 1977, Biochem. Soc. Trans. 5: 271-72; *22* = Thörig, Schoone, and Scharloo, 1977, Biochem. Genet. 13: 721-731; *23* = Ursprung and Leone, 1965, J. Exp. Zool. 160: 147-54; *24* = Wilks, Gibson, Oakeshott, and Chambers, 1980, Aust. J. Sci. 33: 375-85.

β Numbers in parentheses inferred from phenotypic description; others represent actual measurements (Thatcher, 1980, Biochem J. 187: 875-83).

γ Unlike *Adh* ^{F}, will oxidize dihydroorotic acid to orotic acid and sarcosine to glycine.

δ Probably identical.

ε Probably the same as *Adh* ^{71k}.

ζ = *Adh* ^{F}.

η = *Adh* ^{S}.

allele	origin α	derivative of	discoverer	ref β	activity	CRM	forms active hybrid enzyme	notes
Adh ^{fn4}	formaldehyde	*Adh* ^{D}	Sofer	*2-4, 9*	–	–		
Adh ^{fn6}	formaldehyde	*Adh* ^{D}	Sofer	*2-4, 9*	–	–		
Adh ^{fn23}	formaldehyde	*Adh* ^{D}	Sofer	*2-4, 9*	–	+		γ
Adh ^{fn24}	formaldehyde	*Adh* ^{D}	Sofer	*2-4, 9*	–	–		
Adh ^{n1}	EMS	*Adh* ^{S}	E.H. Grell	*6, 7, 10, 14*	20%	+	+	δ
Adh ^{n2}	EMS	*Adh* ^{S}	E.H. Grell	*6, 7, 10, 14*	–	5%	–	
Adh ^{n3}	EMS	*Adh* ^{S}	E.H. Grell	*6, 7, ,10, 14*	–	15%	–	
Adh ^{n4}	EMS	*Adh* ^{D}	E.H. Grell	*6, 7, 10, 14*	–	–		
Adh ^{n5}	EMS	*Adh* ^{D}	E.H. Grell	*6, 12-14, 17, 18*	leaky ts		+	ε
Adh ^{n6}	EMS	*Adh* ^{F}	Gerace	*5, 9, 14*	–	44%	–	
Adh ^{n7}	EMS	*Adh* ^{F}	Gerace	*5, 9, 14*	–	54%	–	
Adh ^{n8}	EMS	*Adh* ^{F}	Gerace	*5, 9, 14*	–	61%	–	
Adh ^{n9}	EMS	*Adh* ^{F}	Gerace	*5, 9, 14*	–	71%	–	
Adh ^{n10}	EMS	*Adh* ^{F}	Gerace	*5, 9, 14*	–	–		
Adh ^{n11}	EMS	*Adh* ^{F}	Sofer	*9-12, 13, 14-17*	0.02%	27%	+	ζ
Adh ^{n12}	EMS	*Adh* ^{F}	Sofer	*9, 10, 14*	–	73%	–	
Adh ^{n13}	EMS	*Adh* ^{F}	Sofer	*9, 10, 14*	–	5%	–	
Adh ^{n14}	EMS	*Adh* ^{F}	Sofer	*9, 10, 14*	–	–		
Adh ^{n967}	spont			*18*				η
Adh ^{nA}	EMS	*CyO, Adh* ^{F}	Sofer	*5, 14*	–	–		
Adh ^{nB}	EMS	*CyO, Adh* ^{F}	Sofer	*5, 14*	–	+		θ
Adh ^{nC1}	EMS	*Adh* ^{UF}	Ashburner					
Adh ^{nC2}	EMS	*Adh* ^{UF}	Ashburner		leaky ts			
Adh ^{nLA2}	X ray	*Adh* ^{F}	Aaron	*1, 8*	–	+		ι
Ash ^{nLA73}	X ray	*Adh* ^{F}	Aaron	*1, 8*	–	–		
Adh ^{nLA74}	X ray	*Adh* ^{F}	Aaron	*1, 8*	–	+		
Adh ^{nLA80}	X ray	*Adh* ^{F}	Aaron	*1, 8*	–	+		κ
Adh ^{nLA248}	X ray	*Adh* ^{F}	Aaron	*1, 8*	–	–		

allele	origin α	derivative of	discoverer	ref β	activity	CRM	forms active hybrid enzyme	notes
Adh ^{nLA249}	X ray	*Adh* ^{F}	Aaron	*1, 8*	–	+		
Adh ^{nLA252}	X ray	*Adh* ^{F}	Aaron	*1, 8*	–	+		
Adh ^{nLA319}	spont	*Adh* ^{D}	Aaron	*1, 8*	–	+		
Adh ^{nLA378}	X ray	*Adh* ^{F}	Aaron	*1, 8*	–			
Adh ^{nLA405}	X ray	*Adh* ^{F}	Aaron	*1, 8*	–	+		ι

α *Adh*^{fn4} – *Adh*^{fn24} and *Adh* ^{n11} – *Adh* ^{n14} selected as larvae on pentenol; *Adh* ^{n6} – *Adh* ^{n10}, *Adh* ^{nA}, *Adh* ^{nB}, and the *Adh* ^{nLA} series of alleles selected as adults on pentenol.

β *1* = Aaron, 1979, Mutat. Res. 63: 127-37; *2* = Benyajati, Place, Powers, and Sofer, 1981, Proc. Nat. Acad. Sci. USA 78: 2717-21; *3* = Benyajati, Place, and Sofer, 1983, Mutat. Res. 111: 1-7; *4* = Benyajati, Place, Wang, Pentz, and Sofer, 1982, Nucleic Acids Res. 10: 7261-72; *5* = Gerace and Sofer, 1972, DIS 49: 39; *6* = Grell, Jacobson, and Murphy, 1968, Ann. N.Y. Acad. Sci. 151: 441-55; *7* = Kamping and van Delden, 1980, DIS 55: 89; *8* = Kelley, Mims, Farnet, Dicharry, and Lee, 1985, Genetics 109: 365-77; *9* = O'Donnell, Gerace, Leister, and Sofer, 1975, Genetics 79: 73-83; *10* = Pelliccia and Sofer, 1982, Biochem. Genet. 20: 297-313; *11* = Reddy, Pelliccia, and Sofer, 1980, Biochem. Genet. 18: 339-51; *12* = Sampsell, 1977, Biochem. Genet. 15: 971-88; *13* = Schwartz and Jornvall, 1976, Europ. J. Biochem. 68: 159-68; *14* = Schwartz and Sofer, 1976, Genetics 83: 126-36; *15* = Thatcher, 1980, Biochem. J. 187: 875-83; *16* = Thatcher and Retzios, 1980, Protides of Biol. Fluids 28: 157-60; *17* = Thatcher and Sheikh, 1981, Biochem. J. 197: 111-17; *18* = Vigue and Sofer, 1974, Biochem. Genet. 11: 387-96.

γ Polypeptide smaller than wild type.

δ Polypeptide slightly larger than wild type on SDS gels and electrophoretic mobility altered on non-denaturing gels.

ε Purified enzyme thermolabile; *Adh* ^{n5} flies grown at 18° show loss of both ADH activity and CRM following a shift to 30° (Tsubota); recovery on return to 18° takes several days (Vigue and Sofer; Tsubota).

ζ Mutation in adenine ribose pocket of coenzyme binding domain; is not bound to 5′-AMP sepharose and cannot recognize NAD^+ (Thatcher and Retzios), *Adh* ^{F} */Adh* ^{n11} forms an active dimer that migrates as *Adh* ^{F} */Adh* ^{UF} (Schwartz and Jornvall; Schwartz and Sofer, Pelliccia and Sofer). Shows weak intracistronic complementation with *Adh* ^{n6} *Adh* ^{n7} *Adh* ^{n12} and *Adh* ^{nA} (Thatcher; Reddy *et al.*). *Adh* ^{n6} */Adh* ^{n11} heterozygotes display partial resistance to alcohol; hybrid enzyme activity heat labile; displays altered substrate binding properties (Pelliccia and Couper, 1984, DIS 60: 160-62).

η Isolated from natural population.

θ Polypeptide shorter than normal (24 kilodaltons).

ι *In vitro* translation product of mRNA smaller than that of wild type (Pelham and Bodmer).

κ Protein unstable by two-dimensional gel electrophoretic analysis.

cytology: Placed in 35B3 by *in situ* hybridization.

molecular biology: Structural gene cloned (Goldberg, 1980, Proc. Nat. Acad. Sci. USA 77: 5794-98) and sequenced [Goldberg; Benyajati *et al.*, 1981; Haymerle, 1983, Thesis, University of Cambridge; Kreitman, 1983, Nature (London) 304: 412-17; Benyajati, Place, Wang, Pentz, and Sofer, 1982, Nucleic Acids Res. 10: 7261-72]. Partial sequence (3′ end) of cDNA clone by Benyajati, Wang, Reddy, Weinberg and Sofer (1980, Nucleic Acids Res. 8: 5649- 67). Variation in restriction enzyme sites within and around *Adh* (Langley *et al.*). *Adh* ^{F} alleles are polymorphic for insertion substitution changes within the 5′ non-coding region intron (Kreitman, 1983). Sequence comparisons between 5′ flanking regions and exons in *D. melanogaster* and *D. simulans* indicate excess polymorphism in the *D. melanogaster* 5′ flanking region (Kreitman and Aguade, 1986, Genetics 114: 93-100; Aquadro, Desse, Blond, Langley, and Laurie-Ahlberg, 1986, Genetics 114: 1165-90).

Standard amino acid sequence taken to be that of ADH-S (Thatcher, 1980 with two corrections: glu25 (not gln) and an extra tryptophan at 251 (Benyajati *et al.*, 1981). Standard DNA sequence is that of *Adh* ^{S} allele from clone pSAC1 of Goldberg (Benyajati *et al.*, 1981, 1982, 1983; Haymerle); numbered from -1/+1, +1 being the 'A' of the ATG initiating codon. All changes with respect to coding strand.

Two primary transcripts: major larval transcript initiated from -69, 24 bp from a TATA box (-100 to -94); major adult transcript initiated from -766, 24 bp from a TATA box (-808 to -800). The major adult transcript is processed by the removal of an intervening sequence between -689 and -36. There are two introns within the coding sequence, from +100 to +164 and from +571 to +639. The polyA addition site is from +1028 to +1034 and the 3′ end of the mRNA at +1079. (Benyajati *et al.*, 1981, 1983, Henikoff, 1983, Nucleic Acids Res. 11: 4735-52).

An 11.8 kb *Sac*I restriction fragment of the *Adh* ^{F} allele shown by P-element-mediated germline transformation to contain all *cis*-acting DNA sequences necessary for correct expression (quantitative levels of mRNA and enzyme; tissue specificity; developmental switch in promoter usage)(Goldberg, Posakony, and Maniatis, 1983, Cell 34: 59-73. *In vitro* recombinants rule out the 5′ flanking sequences as responsible for the two- to threefold higher enzyme activity and increased amount of ADH protein in *Adh* ^{F} compared to *Adh* ^{S}; the only consistent differences are at three nucleotide positions, one at 1490 responsible for the electrophoretic difference and two silent third-codon substitutions at nucleotides 1443 and 1527 (Laurie-Ahlberg and Stamm, 1987, Genetics 115: 129-40); increase probably not attributable to increased levels of mRNA (Laurie and Stamm, 1988, Proc. Nat. Acad. Sci. USA 85: 5161-65). Transcript of Drosophila gene transfected into yeast spliced normally (Watts, Castle, and Beggs, 1983, EMBO J. 2: 2085-91). A fusion of *Adh* to the *Hsp70* promoter has been inserted into a *P* element and used in transformation experiments in *Adh* deficient flies; in such transformants *Adh* $^{+}$ function is under heat-shock control (Bonner, Parks, Parker-Thornberg, Mortin and Pelham, 1984, Cell 37: 979-91). Molecular information on alleles in following table.

allele	molecular biology	ref α
Adh ^{71k}	lys 192 → thr 192; pro 214 → ser 214	*7*
Adh ^{D}	lys 192 → thr 192; gly 232 → glu 232	*10, 12*
Adh ^{F}	lys 192 → thr 192; A*713* → C*713*	*1, 10, 11, 14*
Adh $^{F'}$	ala 51 → glu 51	*10*
Adh $^{F\text{-}ChD}$	lys 192 → thr 192; pro 214 → ser 214	*4*
Adh ^{fn4}	No mature mRNA; 16 bp deletion in first intron (*146-162*); AG(*163-164*) splice acceptor changed to GG splicing defective.	*2, 3*
Adh ^{fn6}	No mature mRNA; 6 bp deletion in	*2, 3*

allele	molecular biology	ref α
	first intron (*106-111*); 101-105 substituted by CGATC; splicing defective.	
Adh ^{fn23}	34 bp deletion in 3′ coding region (*724-758*); read through of normal termination triplet.	*2, 3*
Adh ^{fn24}	50% wild-type mRNA level; 11 bp deletion in second exon (*256-266*); Premature chain termination	*2, 3*
Adh ^{n4}	C*312* → T*312*; gln 83 → ter 83	*2, 3*
Adh ^{n11}	Destroys Pvu II site (Chia)	
Adh ^{nB}	gly 14 → asn 14	*10, 12, 13*
	UGG → UGA in trp 234 codon; suppressible in vitro with yeast ochre suppressor tRNA	*8, 9*
Adh ^{nLA248}	250 bp insertion formed by unequal crossover between exon 3 (at *+708*) and exon 2 (at *+465*) with 7bp (GTGCAAC) inserted at the junction.	*5, 6*
Adh ^{S}	standard nucleotide sequence	*1, 2, 3, 10, 13*
Adh ^{UF}	asn ^{8} → ala ^{8}; ala ^{45} → asp ^{45}; lys 192 → thr 192	*10, 13, 14*
Adh ^{US}	lys 192 unchanged	*11*

α *1* = Benyajati, Place, Powers, and Sofer, 1981, Proc. Nat. Acad. Sci. USA 78: 2717-21; *2* = Benyajati, Place, and Sofer, 1983, Mutat. Res. 111: 1-7; *3* = Benyajati, Place, Wang, Pentz, and Sofer, 1982, Nucleic Acids Res. 10: 7261-72; *4* = Chambers, Laver, Campbell, and Gibson, 1981; Proc. Nat. Acad. Sci. USA 78: 3103-07; *5* = Chia, Karp, McGill, and Ashburner, 1985, J. Mol. Biol. 186: 689-706; *6* = Chia, Savakis, Karp, Pelham, and Ashburner, 1985, J. Mol. Biol. 186: 679-88; *7* = de Boer, Andriesse, and Weisbeek; *8* = Kubli, Schmidt, Martin, and Sofer, 1982, Nucleic Acids Res. 10: 7145-52; *9* = Martin, Place, Pentz, and Sofer, 1985, J. Mol. Biol. 184: 221-29; *10* = Retzios and Thatcher, 1979, Biochimie 61: 701-04; *11* = Retzios and Thatcher, 1980, Biochem. Soc. Trans. 9: 298-99; *12* = Schwartz and Jornvall, 1976, European J. Biochem. 68:: 159-68; *13* = Thatcher, 1980, Biochem. J. 187: 875-83; *14* = Thatcher and Camfield, 1977, Biochem. Soc. Trans. 5: 271-72.

adl: see *l(1)adl*

adp^{60}: adipose

location: 2-83.4.
origin: Spontaneous.
discoverer: Doane, 1960.
references: 1961, DIS 35: 78.
1963, DIS 38: 32.
1963, Proc. 23rd Ann. Biol. Coll., Oregon State Univ. Press, Corvallis, pp. 65-88.
1969, J. Exp. Zool. 171: 321-42.
phenotype: Adult fat body hypertrophies as cells become distorted by enormous oil globules. Lipid accumulated at expense of glycogen in fat body; yolk deposition retarded (Doane, 1963, DIS 37: 73-74; Doane, 1980, Evolution 34: 868-74). Lipid content and fatty acid profiles compared for various developmental stages in *adp* ^{60} and wild type (Teague, Clark, and Doane, 1986, J. Exp. Zool. 240: 95-104). Abnormal fat bodies visible through body wall of 6-day-old and older adults when submerged in 95% alcohol and then water. Corpus allatum of mated females hypertrophies. Females fertile but egg hatchability reduced to 45-90%, depending on residual genome; dorsal appendages of chorion convoluted or fused (King and Koch, 1963, Quant. J. Microscop. Sci. 104: 297-320); adult emergence lowered to 33-85%. (Doane, 1963, DIS. 37: 73-74). Males viable and fertile. Heterozygotes show desiccation tolerance superior to that of wild type or *adp* ^{60} homozygotes (Clark and Doane, 1983, Hereditas 99: 165-75). RK3.
cytology: Placed in 55A-C1 based on its inclusion in *Df(2R)PC4* = *Df(2R)55A;55F* but not *Df(2R)P29* = *Df(2R)55C1-2;56B1-2* (Doane and Dumapias, 1987, DIS 66: 49).

adp^{fs}: adipose-female sterile

origin: Spontaneous.
discoverer: Counce, 1956.
synonym: *fs(2)adp: female sterile(2) adipose.*
references: Doane, 1959, Genetics 44: 506.
1960a, J. Exp. Zool. 145: 1-22 (fig.).
1960b, J. Exp. Zool. 145: 23-42.
1961, J. Exp. Zool. 146: 275-98.
phenotype: Adult fat body phenotype like *adp* ^{60}; lipid accumulated at expense of glycogen in fat body; yolk deposition in ovaries retarded; carbohydrate levels low in 8-day-old adults (Cummings and Ganetzky, 1972, DIS 30: 48. Corpus allatum hypertrophies in mated females to same degree as in *adp* ^{60}. Females completely sterile; sterility autonomous. Eggs laid by homozygotes show meiotic or mitotic abnormalities, or both, never develop beyond early cleavage stages; chorion, chorionic filaments and vitelline membrane defective in some. Males 78% fertile. Heterozygotes fertile, but females become sterile with age. Viability generally good but longevity reduced; homozygotes with selective advantage under starvation; heterozygotes superior under desiccation. Average water content of well-fed adults reduced; percentage of lipids, as a function of dry body weight, almost double that of wild type. Iodine numbers show greater degree of saturation of mutant lipid extracts than of wild type. RK3.

**ae: aeroplane*

location: 2-55.8.
origin: Spontaneous.
discoverer: Mohr, 26k24.
references: Quelprud, 1931, Hereditas 15: 97-119 (fig.).
phenotype: Wings spread, balancers drooping. Overlaps wild type. RK3.

**Ae: Aechna*

location: 3- (rearrangement).
origin: X ray induced.
discoverer: Belgovsky, 45a14.
references: 1946, DIS 20: 63.
phenotype: Wings spread at right angles to body axis. Homozygous lethal. RK1A.
other information: Reduced crossing over in the *th-e* region suggests presence of pericentric inversion.

aea: see *dv* ^{2}

aeroplane: see *ae*

ag: agametic

location: 1-20.7.
origin: Spontaneous.
references: Engstrom, Caulton, Underwood, and Mahowald, 1982, Dev. Biol. 91: 163-70 (fig.).
phenotype: Maternal effect mutant; approximately 40% of gonads of progeny of homozygous females agametic. Although some eggs of homozygous females exhibit

abnormal polar granules, normal numbers of pole cells form; some pole cells abnormal with degenerating polar granules and nuclear bodies, but pole cells reach gonads at 14 hr of development and then in 40% of the gonads become necrotic and disappear; responses of right and left gonads correlated. Phenotype most pronounced at 25°, decreasing at higher and lower temperatures. Mutant not completely recessive; expression in progeny of heterozygous females half that in those of homozygotes.

cytology: Placed between 7B4 and 7C1 based on its position to the right of *ct* and its inclusion in *Df(1)ct268-42 = Df(1)7A5-6;7B8-C1*.

*agl: angle winglike

location: 1- (not located).

origin: Recovered among descendants of flies treated with natural gas.

discoverer: Mickey, 49c7.

synonym: Originally called *angle wing* but this name preoccupied by *ang*.

references: 1950, DIS 24: 60.

phenotype: Wing bent upward in middle. Overlaps wild type. RK3.

agn: agnostic

location: 1-38.9.

references: Savvateeva, Korochkina, Peresleny, and Kamyshev, 1985, DIS 61: 144.

Savvateeva, Peresleny, Ivanushina, and Korochkin, 1985, Dev. Genet 5: 157-72.

phenotype: Identified as three temperature sensitive lethal mutations. Adenylate cyclase activity somewhat higher than normal at 22° and readily activated at 29°. Phosphodiesterase activity assayed in heat-pretreated homogenates higher than normal. Locomotor activity decreased and learning activity increased at 22°, like *dnc* at 29°.

alleles:

allele	origin	synonym
agn ^{1}	EMS	*l(1)ts398*
agn ^{2}	EMS	*l(1)ts622*
agn ^{3}	EMS	*l(1)ts980*

agq: atrophie gonadique

location: 2-3 polygenic.

origin: Recovered from natural population on French Mediterranean coast.

references: Periquet, 1970, DIS 45: 33.

1979, Biol. Cell. 33: 33-38.

phenotype: Gonads atrophic either unilaterally or bilaterally owing to pole cell degeneration. Degree of effect correlated with both temperature during the first hours of development and with the number of *agq*-derived autosomes. Pole cells, but not oocytes, thermosensitive. Responses of right and left gonads correlated. Penetrance higher in females than males.

al: aristaless

location: 2-0.4 [Golubovsky, Kulakov, and Korochkina, 1978, Genetika (Moscow) 14: 294-305].

references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 213 (fig.).

Stern and Bridges, 1926, Genetics 11: 510 (fig.).

phenotype: Aristae strongly reduced. Postscutellars widely separated and erect but strongly divergent. Scutellum shortened; sternopleurals irregular in size, position, and number; wings slightly bowed downward, narrowed, and pointed; first longitudinal vein raised and thickened. Tarsal claws transformed into bristle-like structures or absent; effect enhanced by presence of *th* or *ss* ^{a40a} or both [Mglinetz and Ivanova, 1975, Genetika (Moscow) 11, 4: 88-96]. Enhances transformations of *Antp* ^{NS} [Mikuta and Mglinetz, 1978, Genetika (Moscow) 14: 1578-85] and *ss* ^{a40a}; *al ss* ^{a40a} flies exhibit partial transformation of third antennal segment to basitarsus; anomalous outgrowths of distal basitarsus and foreshortening of second and third tarsal joints of thoracic legs; spineless phenotype also enhanced. *al* females exhibit reduced mating success (Burnet, Connolly, and Dennis, 1971, Anim. Behav. 19: 409-15). RK1.

alleles:

allele	origin	discoverer	ref $^{\alpha}$	comments
al ^{1}	spont	Bridges, 17k7	*2, 6, 7*	viable
al ^{2}	spont	Stern, 26a	*7*	<*al* ^{1}; poorly viable
****al*** ^{3}	spont	Bridges, 33g2		>*al* ^{1}; semilethal; female sterile
al ^{4}	spont	Bridges, 33l27	*1, 2*	<*al* ^{1}; viable; *In(2LR)bw* ^{V1}
al ^{8}	X ray		*4*	>*al* ^{1}; lethal; *In(2LR)21C1-2;41C*
al ^{36}	X ray	Glass, 36c	*2, 3*	=*al* ^{1}; viable
****al M60***	X ray	Meyer, 60f	*2, 5*	lethal; *In(2LR)*; variegated?
al ^{V}	X ray	E.B. Lewis, 1940	*1*	lethal; *In(2LR)21B-C1;41*; variegated

$^{\alpha}$ *1* = Bridges, 1935, DIS 3: 5; *2* = CP627; *3* = Glass, 1939, DIS 12: 47; *4* = Korochkina and Golubovsky, 1978, DIS 53: 197-200; *5* = Meyer, 1963, DIS 37: 50; *6* = Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 213 (fig.); *7* = Stern and Bridges, 1926, Genetics 11: 510-511 (fig.).

$^{\beta}$ Phenotype defined with respect to homozygous viablility and strength of expression in comparison with *al* ^{1}.

cytology: Placed in 21C1-2 doublet on the basis of its inclusion in *Df(2L)al = Df(2L)21B8-C1;21C8-D1* but not in *Df(2L)S5 = Df(2L)21C2-3;22A3-4* (Lewis, 1945, Genetics 30: 137-166).

al ^{8}

phenotype: Homozygous lethal; 1% survival in combination with *Df(2L)al = Df(2L)21B8-C1;21C8-D1;* survivors have reduced aristae, broad thorax, arched wings with incomplete veins, enlarged eyes.

cytology: Associated with *In(2LR)al* ^{8} = *In(2LR)21C1-2;41C*.

al-b: see *aa*

ala: see *dy* ^{ala}

ala parvae: see *dy* ^{ala}

β *alanyl dopamine hydrolase*: see *t*

β *alanyl dopamine synthetase*: see *e*

alarless: see ***alr***

alb: alberich

location: 2-(unmapped).

origin: Induced by ethyl methanesulfonate.

references: Tearle and Nüsslein-Volhard, 1987, DIS

66: 209-26.

phenotype: Maternal-effect lethal; occasional embryo lacks abdominal segments (Lehmann).

Alcohol dehydrogenase: see *Adh*

ald: altered disjunction (A.T.C. Carpenter)

location: 3-61.

origin: Induced by ethyl methanesulfonate.

references: O'Tousa, 1982, Genetics 102: 503-24.

phenotype: Homozygous females display elevated levels of nondisjunction of *X* and fourth chromosomes (9.5 and 6.0% respectively); double exceptions are predominantly *XX;0* and *0;44*, products expected from nonhomologous disjunction; behavior of large autosomes nearly normal. Exchange frequencies normal, and sex-chromosome exchange tetrads contribute to exceptional products.

Ald: Aldolase

location: 3-91.5.

origin: Naturally occurring polymorphism.

references: Voelker, Ohnishi, and Langley, 1979, Biochem. Genet. 17: 769-83.

phenotype: Structural gene for fructose-biphosphate aldolase (EC 4.1.2.13). Enzyme multimeric based on the formation of heteromultimeric bands on gels from heterozygotes for electrophoretic variants. Amino-acid sequence determined; protein is a 158-kd multimer comprising four 360-amino-acid polypeptides. Monomers show 71% identity with rabbit muscle aldolase (Malek, Suter, Frank, and Brenner-Holzach, 1985, Biochem. Biophys. Res. Comm. 126: 199-205). Mixed multimers of rabbit and Drosophila subunits able to function (Brenner-Holzach and Leuthard, 1972, Eur. J. Biochem. 31: 423-26). Sequence analysis suggests alternating domains of alpha helix and beta sheet; domain boundaries correspond to boundaries between exons as seen in rat-liver aldolase (Sawyer, Fothergill-Gilmore, and Freemont, 1988, Biochem. J. 249: 789-93).

cytology: Placed in 97A-B on the basis of its being between the autosomal breakpoints of *T(Y;3)R87* = *T(Y;3)97A* and *T(Y;3)B158* = *T(Y;3)97B*.

alleles: Two electrophoretic variants described; Ald^4 product migrates toward the anode more rapidly than that of Ald^2.

Aldehyde oxidase: see *Aldox*

Aldolase: see *Ald*

Aldox-1: Aldehyde oxidase-1

location: 3-57.2 (between *red* and *sbd*).

origin: Naturally occurring polymorphism.

synonym: *Ao.*

references: Dickinson, 1970, Genetics 66: 487-96.

Warner, Watts, and Finnerty, 1980, Mol. Gen. Genet. 180: 449-53.

Warner and Finnerty, 1981, Mol. Gen. Genet. 184: 92-96.

Bogart and Bernini, 1981, Biochem. Genet. 19: 929-46.

phenotype: Structural gene for aldehyde oxidase [AO-1 (EC 1.2.3.1)]. A molybdenum-containing homodimer with subunits of 140,000-dalton molecular weight. As with other molybdenum hydroxylases, activity is inhibited by tungsten and depends on the presence of a low-molecular-weight cofactor (Warner and Finnerty). Enzyme activity increases nonuniformly during development with step increases at pupation and midway through pupal stage. First increase appears to be controlled by a closely linked *cis*-acting element (Dickinson, 1975, Dev. Biol. 42: 131-40) and the latter by $Aldox\text{-}2^+$ (Bentley and Williamson, 1979, Z. Naturforsch. 34: 304-05). Control independent of that of *lpo* located 0.08 unit to the left (Dickinson and Weisbrod, 1976, Biochem. Genet. 14: 709-21). Enzyme activity absent in *cin* (Browder and Williamson, 1976, Develop. Biol. 53: 241-49) and *mal* and reduced in *lxd* (Courtright, 1967, Genetics 57: 25-39); cross-reacting material observed in all three genotypes (Browder, Wilkes, and Tucker, 1982, Biochem. Genet. 20: 111-24). These mutants presumably affect the availability of molybdenum cofactor (Warner and Finnerty). Autonomous in transplants. Enzyme composition in egg and early larva reflects maternal genotype, giving way to that of zygotic genotype during larval life. Tissue specificity varies with stage and strain (Dickinson, 1972, Genetics 71: s14; Cypher, Tedesco, Courtright and Kumaran, 1982, Biochem. Genet. 20: 315-32). Heptaldehyde serves as specific substrate (Cypher *et al.*). Differential staining for enzyme noted in different compartments of the wing imaginal disc (Kuhn and Cunningham, 1976, Genetics 83: s42).

alleles: Three electrophoretic variants superscripted *1*, *2*, and *3* in order of increasing mobility described by Dickinson (1970) presumably correspond to $Aldox\text{-}1^4$, $Aldox\text{-}1^6$, and $Aldox\text{-}1^8$ (1978, DIS 53: 117); 6 electrophoretic variants numbered in order of increasing mobility from *1* through *6* described by Langley, Tobari, and Kojima (1974, Genetics 78: 921-36). Correspondence of two sets of alleles unknown. Two null alleles superscripted *n1* and *n2* are homozygous viable but produce no recognizible product either in the form of enzyme activity or by the formation of heterodimers with functional gene products; however, cross-reacting material found in larval hemolymph but not in extracts of adults (Browder *et al.*, 1982). Thirteen other null alleles isolated from natural populations in Great Britain ($Aldox^{nGB1}$ to $Aldox^{nGB4}$) and North Carolina ($Aldox^{nNC1}$ *to* $Aldox^{nNC9}$); all thirteen exhibit residual enzyme activity and except for $Aldox^{nGB1}$ are $Aldox^4$ derivatives and fail to participate in heterodimer formation with normal gene products. $Aldox^{nGB1}$ is an $Aldox^8$ derivative and forms heterodimers (Burkhart, Montgomery, Langley, and Voelker, 1984, Genetics 107: 295-306). *lao:* low aldehyde oxidase (Collins, Duck, and Glassman, 1971, Biochem. Genet. 5: 1-13) considered allelic based on its phenotype and genetic position (3-56); aldehyde oxidase levels of $Aldox^n/Aldox^{lao}$ higher than expected if $Aldox^n$ amorphic.

cytology: Placed in region between 88F9 and 89B4 on the basis of its inclusion in *Df(3)sbd105* = *Df(3R)88F9-88A1;89B4-5* but not *Df(3R)sbd45* = *Df(3R)89B1-4;89B10-13* (Spillmann and Nöthiger, 1978, DIS 53: 124). Narrowed to 89A1-2 by Langhout and van Breugel (1985, DIS 61: 181) on basis of reduced staining of that doublet in $Aldox^{n1}$.

Aldox-1^{Pa1}

origin: Polymorphic in laboratory and natural populations.

references: Dickinson, 1975, Dev. Biol. 42: 131-40.

phenotype: Exhibits substantial increase in enzyme

activity between late larval and early pupal stages followed by a second increase in late pupal stage. Results in relatively high pupal:adult activity ratio.

Aldox-1 Pa2

origin: Polymorphic in laboratory and natural populations.
references: Dickinson, 1975, Dev. Biol. 42: 131-40.
phenotype: Exhibits little change in enzyme activity between late larval and early pupal stages, but there is a substantial increase in late pupal and adult stages. Results in relatively low pupal:adult activity ratio. *Aldox-1 Pa1/Aldox-1 Pa2* has intermediate phenotype.
other information: 1.5% recombination with electrophoretic *Aldox-1* alleles recorded. The difference between *Pa1* and *Pa2* postulated to reside in a cis-acting site that exerts temporal control on gene activity.

Aldox-1 Pb1

origin: Polymorphic in natural population from Lima, Peru.
references: Dickinson, 1978, J. Exp. Zool. 206: 333-42.
phenotype: Cytochemically, enzyme activity in paragonia uniformly distributed and high at eclosion. By end of first week of adult life enzyme accumulated into intracellular bodies giving accessory gland a spotted appearance.
alleles: *Aldox-1 Pb2* characterized by lower activity that remains uniformly distributed in paragonia as detected histochemically.
other information: Not separated genetically from electrophoretic alleles of *Aldox-1*. *Pb1* and *Pb2* postulated to differ in a cis-acting control site with tissue-specific effects on gene activity.

aldox2: aldehyde oxidase 2

location: 2-82.9 (based on 112 *c-px* recombinants).
origin: Naturally occurring polymorphism.
references: Bentley and Williamson, 1979, Z. Naturforsch. 34: 304-05.
1980, Genetics 94: s8.
Meidinger and Bentley, 1986, Biochem. Genet. 24: 683-99.
Bentley, Meidinger, and Braaten, 1989, Biochem. Genet. 27: 99-118.
phenotype: Not a structural gene for aldehyde oxidase. Homozygotes for *aldox2* fail to show increased levels of molybdoenzymes, aldehyde oxidase, pyridoxal oxidase, sulfite oxidase, and xanthine oxidase, that normally occur late in the pupal period; adult levels lower than normal. Enzyme activity less sensitive to tungsten and more responsive to molybdenum than in wild type; aldehyde oxidase activity, at least, more heat labile and pH optimum slightly more acidic than normal (Meidinger and Bentley, 1984, Genetics 107: s73). Duplication of *aldox2+* without effect on enzyme levels.
cytology: Placed in region 54 based on the failure of *Y P2 D* from *T(Y;2)H149 = T(Y;2)h21;54F* to cover it and its genetic map position to the right of *Amy*, which has been placed in 54A1-B1 by *in situ* hybridization.

ale: almond eye

location: 3-47.5 (located with respect to *D* and *Sb*).
origin: Spontaneous in natural population.
references: Golubovsky and Zakharov, 1972, DIS 49: 112.
Golubovsky, Ivanov, and Zakharov, 1973, Genetika (Moscow) 9(8): 168-71.
phenotype: In homozygotes, eye almond shaped but with normal facet development. Phenotype normal in heterozygotes with normal third but mutant when heterozygous to *D. Dfd/ale* show normal eye size, and 20% of flies show tufted vibrissae characteristic of *Dfd/+*. *ale Mc* homozygotes completely eyeless.
other information: Both *Dfd* and *ale* map to 47.5. No wild-type recombinants recovered among 569 tested progeny of *Dfd/ale* females. *ale* acts as a transdominant suppressor of *Dfd*.

Ali n: Aliesterase-negative

location: 3- (not located).
origin: Spontaneous.
synonym: *ali: aliesteraseless.*
references: Ogita, 1961, Botyu-Kagaku 26: 93-97.
1962, DIS 36: 103.
phenotype: Probably the structural gene for aliesterase [ALI (EC 3.1.1.1)]. Homozygotes for *Ali n* practically unable to hydrolyze methyl butyrate, whereas wild type shows high activity; *Ali n/+* exhibits intermediate activity. Homozygotes shown by Beckman and Johnson to lack a normally present esterase that migrates slowly on starch gel (their band F). RK3.
alleles: *Ali n* is a null allele; no other variants reported.

Alkaline phosphatase: see ***Aph and Aph-2***

aliesteraseless: see *Ali n*

almond: see *Dfd r*

almondex: see ***amx***

almondex-55: see *lz K*

almond eye: see ***ale***

**alo: alopecia*

location: 1-38.3.
origin: Induced by 2-chloroethyl methanesulfonate.
discoverer: Fahmy, 1956.
references: 1958, DIS 32: 67.
phenotype: Abdominal hairs much reduced in number; pigmentation frequently lighter and patchy. Effect very pronounced in females reared at 25° but overlaps wild type in both sexes when reared at a low temperature. Viability and fertility good in males but reduced in females. RK3.

Alp: Abnormal leg pattern

location: 2-10.
references: Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.
phenotype: Defined by two dominant gain-of-function alleles. Heterozygotes viable with fusion of metatarsal and second tarsal segments. *Alp 1/Alp 2* is pupal lethal with more extreme tarsal fusions.
alleles: Two X-ray induced alleles. *Alp 1* is associated with *T(2;3)XT1* and *Alp 2* is associated with *T(2;4)X2*.
cytology: Placed in 23F6-24A1 based on breakpoint common to translocations.

alpha: see *tyr-1*

alpha methyldopa hypersensitive: see ***amd***

*alr: alarless

location: 3- (not located).
origin: Spontaneous.
discoverer: Steinberg, 40b.
references: 1940, DIS 13: 51.
phenotype: Outer postalar bristle always missing; posterior supra-alar missing in about 80% of the flies. Anterior scutellars, humerals, and notopleurals frequently duplicated. Never overlaps. Viability and fertility excellent. RK3.

ALS: see *Acr96A*

Altered abdomen: see Aa

altered disjunction: see ald

Alu: Alula

location: 2-54.9 (Muller places *Alu* to the left of *pr*).
origin: Spontaneous.
discoverer: Bridges, 38a12.
references: Curry, 1939, DIS 12: 45.
phenotype: Heterozygote has alula fused to main wing; wings often bent, broader. May overlap wild type but intensified by cold and by heterozygous *ds* with buckling effect increased. Homozygote at 19° shows extreme buckling owing to rotation of wing and alula. Homozygote viable and resembles heterozygote. RK2.
alleles: $*Alu^{56c}$ (CP627).

*alw: arclike wing

location: 2- (near *b*).
discoverer: Sturtevant, 1948.
references: 1948, DIS 22: 55.
phenotype: Wings evenly bent downward at tips. Overlaps wild type. RK2.

am: see Dfd^r

Ama: see ANTC

Ama: see $RpII^{C4}$

Ama-1: α-amanatin resistant 1

location: 3-19.
origin: Recovered from natural populations from India, Malaysia, and Taiwan.
references: Phillips, Willms, and Pitt, 1982, Can. J. Genet. Cytol. 24: 151-62.
phenotype: Flies homozygous or heterozygous for *Ama-1* and *Ama-2* have LD_{50} to α-amanatin 10-30 times that of wild type. Sensitivity of RNA polymerase II activity to α-amanatin same as wild type. *Ama-1* alone sufficient to confer resistance, but in three independent isolations both *Ama-1* and *Ama-2* present.

Ama-2

location: 3-100.
origin: Recovered from natural populations in India, Malaysia, and Taiwan.
references: Phillips, Willms, and Pitt, 1982, Can. J. Genet. Cytol. 24: 151-62.
phenotype: Same as for *Ama-1*.

Amalgam: see Ama under ANTC

Amanatin resistant: see also Amr and RpII

amb: amber

location: 1-6.8.
origin: Induced by triethylenemelamine.
discoverer: Fahmy, 1950.
references: 1958, DIS 32: 67.
phenotype: *amb* has pale yellow body color; bristles very thin and short; hairs less affected. Eyes slightly brighter red. Males sterile. Viability 10-50% wild type. RK2. amb^2 less extreme, males viable and fertile, females sterile.
alleles: $*amb^1$, amb^2 (Fahmy, 1958 and CP627). Also one allele each induced by triethylenemelamine, DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine, 2-chloroethyl methanesulfonate, and nitrogen mustard and two alleles induced by *p*-N,N-di-(2-chloroethyl)amino-phenylethylamine.
cytology: Placed in 4C7-8 on the basis of its inclusion in *Df(1)bi-D3* = *Df(1)4C5-6;4C7-8* but not *Df(1)rb41* = *Df(1)4B6-C1;4C7-8* (Banga, Bloomquist, Brodberg, Pye, Larrivee, Mason, Boyd, and Pak, 1985, Chromosoma 93: 341-46).

amd: alpha methyldopa hypersensitive

location: 2-53.9 [.002 units (2.1 kb) to the left of *Ddc*].
synonym: *amd*; $l(2)amd^H$, *l(2)37Bk*.
references: Sparrow and Wright, 1974, Mol. Gen. Genet. 130: 127-41.
Wright, 1977, Am. Zool. 17: 707-21.
Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.
Gilbert, Hirsh, and Wright, 1984, Genetics 106: 679-94.
Marsh and Wright, 1986, Genetics 112: 249-65.
Black, Pentz, and Wright, 1987, Mol. Gen. Genet. 209: 306-12.
Wright, 1987, Adv. Genet. 24: 127-222.
phenotype: *amd*/+ flies die when reared on levels of alpha methyl dopa that are not lethal to wild type; resistance proportional to the number of amd^+ loci present. Adult *amd*/+ females fed alpha methyldopa become sterile and lay eggs that cannot complete embryogenesis. Dopa decarboxylase levels normal. *amd* homozygotes lethal; lethal phase at times of larval hatching, larval molts, and pupariation; larval anal organ extruded and necrotic; pupal cuticle thin and friable. Appears to play role in cuticle formation. amd^1/amd^6 complementing adults deficient for one or more unidentified catecholamines involved in the colorless sclerotization of cuticle.
alleles: The first seven alleles selected as alpha methyl dopa hypersensitive in the heterozygous condition; the remainder recovered as recessive lethal mutations. Interallelic complementation observed, suggesting dimeric product.

allele	origin	discoverer	synonym	ref α	comments
amd^1	EMS	Sparrow	$l(2)amd^{H1}$	1, 2	
amd^2	EMS	Sparrow	$l(2)amd^{H7}$	2	
amd^3	EMS	Sparrow	$l(2)amd^{H14}$	2	
amd^4	EMS	Sparrow	$l(2)amd^{H45}$	2	
amd^5	EMS	Sparrow	$l(2)amd^{H82}$	2	
amd^6	EMS	Sparrow	$l(2)amd^{H89}$	1, 2	
amd^7	EMS	Sparrow	$l(2)amd^{H121}$	1, 2	
amd^8	EMS	Wright	$l(2)amd^{H8}$	3, 4	
amd^9	EMS	Wright	$l(2)amd^{H60}$	3, 4	
amd^{10}	EMS	Wright	$l(2)amd^{H122}$	3, 4	
amd^{11}	EMS	Wright	$l(2)amd^{H149}$	3, 4	
amd^{12}	X ray	Hodgetts	$l(2)amd^{HX1}$	3, 4	

allele	origin	discoverer	synonym	ref [α]	comments
***amd*^{21}**	EMS	Wright	*l(2)203*	*4*	
***amd*^{22}**	EMS	Wright	*l(2)245*	*4*	
***amd*^{23}**	EMS	Wright	*l(2)258*	*4*	
***amd*^{24}**	EMS	Wright	*l(2)283*	*4*	
***amd*^{25}**	EMS + HCOH	Wright	*l(2)305*	*4*	
***amd*^{26}**	EMS + HCOH	Wright	*l(2)329*	*4*	
***amd*^{27}**	EMS + HCOH	Wright	*l(2)337*	*4*	
***amd*^{28}**	EMS + HCOH	Wright	*l(2)341*	*4*	
***amd*^{29}**	EMS + HCOH	Wright	*l(2)346*	*4*	
***amd*^{30}**	EMS	Wright	*l(2)602*	*4*	
***amd*^{31}**	EMS	Wright	*l(2)616*	*4*	
***amd*^{32}**	EMS	Wright	*l(2)638*	*4*	
***amd*^{33}**	EMS	Wright	*l(2)640*	*4*	
***amd*^{34}**	EMS	Wright	*l(2)674*	*4*	
***amd*^{35}**	EMS	Steward	*l(2)RS1*	*4*	
***amd*^{36}**	EMS + γ ray	Wright	*l(2)7301*	*4*	
***amd*^{37}**	EMS + γ ray	Wright	*l(2)7401*	*1, 4*	750-bp insert, destroys *Eco*RI site at -5.6-kb
***amd*^{38}**	EMS + γ ray	Wright	*l(2)7413*	*4*	
***amd*^{39}**	EMS + γ ray	Wright	*l(2)7433*	*4*	
***amd*^{40}**	EMS + γ ray	Wright	*l(2)7439*	*1, 4*	*Bgl*II site at -4.8-kb altered; at intron splice acceptor
***amd*^{41}**	EMS + γ ray	Wright	*l(2)7445*	*4*	
***amd*^{42}**	EMS	Schüpbach	*l(2)WK26*		
***amd*^{43}**	EMS + γ ray	Cecil	*l(2)C7*		
***amd*^{44}**	EMS + γ ray	Cecil	*l(2)AA3*		
***amd*^{45}**	EMS + γ ray	Cecil	*l(2)BB2*		
***amd*^{46}**	EMS + γ ray	Cecil	*l(2)BB3*		
***amd*^{47}**	EMS + γ ray	Cecil	*l(2)B1*		

α *1* = Black, Pentz, and Wright, 1987, Mol. Gen. Genet. 209: 306-12; *2* = Sparrow and Wright, 1974, Mol. Gen. Genet. 130: 127-41; *3* = Wright, Bewley, and Sherald, 1976, Genetics 84: 287-310; *4* = Wright, Black, Bishop, Marsh, Pentz, Steward, and Wright, 1982, Mol. Gen. Genet. 188: 18-26.

cytology: Placed in 37B9-C1 based on its inclusion in *Df(2L)NST* but not *Df(2L)VA17*.

molecular biology: Located between coordinates -4.7 and -2.42, where 0 is the axis of symmetry of the *Hpa*I site near the terminus of the *Ddc* coding sequence and positive values extend to the right. Genomic sequence contains a 483-bp intron near the 5′ end; the usual upstream regulatory sequences identified as well (Marsh, Erfle, and Leeds, 1986, Genetics 114: 453-67). A 2.0-kb *amd* transcript first detectable early in embryogenesis; reaching maximum level at 12-16 hours; low levels observed in adults; concentrated in the nurse cells of stage 8-9 oocytes; smaller transcripts with sequence homology to the 2.0-kb transcript observed in third-instar larvae. *amd* and *Ddc* transcribed from opposite strands; two regions of extensive homology between *amd* and *Ddc* detected; intron sequences and positions not conserved, although homology across intron junctions is high (Eveleth and Marsh, 1986, Genetics 114: 469-83). Sequence predicts a 50,481 dalton polypeptide with a slight negative charge; 38% amino acid homology with dopa decarboxylase.

Amdr: Alpha methyl dopa resistant

location: 3- (between *h* and *tn*).

origin: Induced by ethyl methanesulfonate.

references: Bishop and Sherald, 1981, DIS 56: 21.

phenotype: Based on two of 16 chromosomes selected for conferring resistance to α methyl dopa when heterozygous. LD_{50} to L-α-methyl dopa for the two chromosomes is 0.325 mM for *Amdr* ^{1}/+ and 0.35 mM for *Amdr* ^{2}/+, compared to 0.10 mM for wild type. Both chromosomes are homozygous lethal, and *Amdr* ^{1}/*Amdr* ^{2} is nearly lethal, allowing the inference of a single locus.

amethyst: see *amy*

amiel

location: Autosomal.

origin: Spontaneous.

synonym: *Amiel.*

references: Rushton and Metcalfe, 1971, DIS 46: 61.

phenotype: Homozygous males court abnormally; wing vibrations and copulation attempts more vigorous than in wild type, but mutant males take longer to achieve copulation and have higher incidence of unsuccessful courtships. Homozygous females behave normally.

Amiel: see *amiel*

Aminoimidazole ribotide synthetase: see *ade2*

amn: amnesiac (J.C. Hall)

location: 1-63.

discoverer: Sziber.

origin: Induced by ethyl methanesulfonate.

references: Quinn, Sziber, and Booker, 1979, Nature (London) 277: 212-14.

phenotype: Homozygous or hemizygous mutant flies can be conditioned to avoid odors associated with electric shocks, but effects of conditioning decay with a half life of 15 min compared to 60 min for normal. Memory decay biphasic; rapid for first hour and slow thereafter (Tully). Substitution of reward (1.0 M sucrose) for punishment (electric shock) lengthens memory span from one hour to six hours (Tempel, Bonini, Dawson, and Quinn, 1983, Proc. Nat. Acad. Sci. USA 80: 1482-86). Groups of *amn* flies exhibit apparently abnormal acquisition of learning in tests using visual cues (Folkers, 1982, J. Insect. Physiol. 28: 535-39); it appears that short-term memory is defective in the mutant (in shock-odor tests), with long-term memory being normal (Tully and Quinn, 1985, J. Comp. Physiol. 157: 263-77); in experiments involving "operant" conditioning, with heat as the aversive unconditioned stimulus, *amn* exhibits a small decrement in learning *per se* and subsequently has no detectable memory (Mariath, 1985, J. Insect Physiol. 31: 779-81). In tests of "simple learning," amnesiac individuals habituate to or are sensitized by sugar stimuli subnormally; the sensitization defect maps to the same proximal locus as that affecting associative conditioning (Duerr and Quinn, 1982, Proc. Nat. Acad. Sci. USA 79: 3646-50). The effects on courtship behavior or pre-exposure to fertilized females decay more rapidly in amnesiac than in normal males (Siegel and Hall, 1979, Proc. Nat. Acad. Sci. USA 76: 3430-34; Ackerman and Siegel, 1986, J. Neurogenet. 3: 111-23), but amnesiac males are defective in expressing after-effects of exposure to immature wild-type males when tested immediately after such exposure (Gailey, Jackson, and Siegel, 1982, Genetics 102: 771-82). Females defective in ability to be primed by courtship song (Kyriacou and Hall, 1984, Nature (London) 308: 62-65).

cytology: Placed in 19A1 based on its inclusion in *Df(1)mal12* = *Df(1)19A1;20F* but not *Df(1)mal11* = *Df(1)19A2-3;19E1* or *Df(1)mal3* = *Df(1)19A2-3;20E-F* (Tully and Gergen, 1986, J. Neurogenet. 3: 33-47).

Amplification Control Element: see *ACE*

Amr: Amanatin resistant

location: 3- (not mapped).
origin: Induced by ethyl methanesulfonate.
references: Nishiura, 1981, Biochem. Genet. 19: 31-46.
phenotype: Heterozygotes survive 5 µg/ml α amanatin. RNA polymerase II activity in *Amr*-bearing flies resistant to α amanatin.
alleles: Three lines possibly containing different alleles designated Amr^{010}, Amr^{018}, and Amr^{106}.
other information: Genetic analysis lacking. If it is demonstrated that this locus codes for an RNA polymerase II subunit, it will be renamed *RpII* plus a subunit designation.

amx: almondex

location: 1-27.7 [to the left of *lz* (Green and Green, 1956, Z. Indukt. Abstamm. Vererbungsl. 87: 708-21)].
origin: X ray induced.
discoverer: Ball, 32k20.
phenotype: Eyes slightly reduced, narrower below. Trident pattern stronger than in *lz*. Maternal effect lethal. Studies by Shannon [1972, Genetica (The Hague) 43: 244-56] show that *amx* progeny and many *amx*/+ progeny of *amx* mothers are embryonic lethals. Ovaries and egg production of *amx* females normal. General disorganization of early embryo with *amx*/+ progeny of *amx* mothers less extreme than *amx* progeny (Shannon, 1973, J. Exp. Zool. 183: 383-400); *amx*/+ daughters show 0.2% survival; *amx*/*Dp(1;1)lz-2* show considerably higher survival (Campos-Ortega); Lethal embryos exhibit hypertrophy of central nervous system at the expense of epidermal tissue (Lehmann, Dietrich, Jiménez, and Campos-Ortega, 1981, Wilhelm Roux's Arch. Dev. Biol. 190: 226-29; Lehmann, Jiménez, Dietrich, and Campos-Ortega, 1983, Wilhelm Roux's Arch. Dev. Biol. 192: 62-74). Similarly peripheral nervous elements, the sensilla, exhibit increased numbers and abnormal morphology; cells diverted from epidermal to neurological pathway (Hartenstein and Campos-Ortega, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 210-21). Embryonic phenotype locally rescuable by injections of ooplasm from wild-type or *pcx* ova during preblastoderm stages (Campos-Ortega, La Bonne and Mahowald, 1985, Dev. Biol. 110: 264-67). *lz*/*amx* is wild type. Mosaics in amx/+ daughters of ±/± or *amx*/+ females show that ventral tissues are sensitive to reduced amx^{+} activity; no clones of *amx* tissue found in cuticle of *amx*/+ daughters of *amx* mothers (Germeraad and Disano, 1984, Genetics 107: s36). RK2.
cytology: Located in 8D (region 8D4 through 8E2) by Green and Green (1956).

amx^{55}: see lz^{K}

**amy: amethyst*

location: 2- (not located).
discoverer: Bridges.
references: Morgan, Bridges, and Sturtevant, 1925, Bibliog. Genet. 2: 218.
phenotype: Transparent, light-purplish eye color. RK3.

Amy: Amylase

location: 2-77.9 (based on 5039 *c-wt* recombinants).
origin: Polymorphic locus.
discoverer: Kikkawa, 1957.
references: Kikkawa and Abe, 1960, Annotationes Zool. Jpn. 33: 14-23.
Kikkawa, 1960, Jpn. J. Genet. 35: 382-87.
Kikkawa and Ogita, 1962, Jpn. J. Genet. 37: 394-95.
Kikkawa, 1963, DIS 37: 94.
Bahn, 1967, Hereditas 58: 1-12.
1964, Jpn. J. Genet. 39: 401-11.
Doane, 1969, J. Exp. Zool. 171: 321-42.
1969, Problems in Biology: RNA in development (Hanly, ed.). U. of Utah Press, Salt Lake City, pp. 73-109 (fig.).
Hickey and Benkel, 1986, CRC Crit. Rev. Biotech. (fig.).
phenotype: The structural gene for α-amylase [AMY (EC 3.2.1.1)]. A monomeric protein based on failure to form hybrid enzyme molecules of intermediate mobility in heterozygotes for alleles coding for electrophoretic variants. Activity mainly in midgut and hemolymph with smaller amounts in other tissues; activity found in anterior or posterior, or both, but not middle, region of midgut; three spatial patterns of adult posterior midgut activity encountered on standard medium; controlled by the trans-regulatory effect of *map* (2-80) (Abraham and Doane, 1978, Proc. Nat. Acad. Sci. USA 75: 4446-50); adult anterior midgut activity under regulation of another separable regulatory locus (Doane, 1980, DIS 55: 36-39). Larval midgut activity affected by closely linked *cis*-acting regulatory elements (Klarenberg, Kisser, Willemse, and Scharloo, 1986, Genetics 114: 1131-45). Amylase activity is glucose repressible (Hickey and Benkel, 1982, Biochem. Genet. 20: 1117-29); the degree of repression can be greater than one hundred fold in larvae and occurs at a pretranslational, probably transcriptional, level of regulation (Benkel and Hickey, 1985, Genetics 110: S25; 1986, Genetics 114: 137-44, 943-54; 1987, Proc. Nat. Acad. Sci. USA 84: 1337-39).
alleles: Eight electrophoretic variants of a-amylase have been recorded; they are numbered, in order of decreasing rates of migration toward the anode, from -1 through +7 (Doane, Treat-Clemons, Gemmill. Levy, Hawley, Buchenberg, and Paigen, 1983, Isozymes: Curr. Top. Biol. Med. Res. 9: 63-70). Enzymes with mobilities 2 and 3 exist in forms with different heat sensitivities: Amy^{1} the most frequent allele, may be expressed at three different activity levels in different strains, 1a, 1b, and 1c in which 1a has twice the activity of 1b and 1b has twice the activity of 1c; purified α-amylases from 1a and 1c strains have identical specific activities (Treat-Clemmons and Doane, 1982, Isozyme Bull. 15: 90-91); enzyme levels here are apparently under the control of closely linked transacting regulatory elements (Hickey, 1981, Biochem. Genet. 19: 783-96). A chromosome may express none, one, or two of these forms. Bahn recovered one $Amy^{1,3}$ and two Amy^{2} recombinants from $Amy^{1}/Amy^{2,3}$ heterozygotes and one $Amy^{4,3}$ and two $Amy^{2,6}$ recombinants from $Amy^{4,6}/Amy^{2,3}$ heterozygotes. From these observations it was concluded that the *Amy* locus is duplicated and the two copies are separated by 0.008 cm; furthermore, flanking marker segregations indicated that determinants of forms 1,2 (thermostable), and 4 are to the left of those for 3 (thermostable) and 6. Conservation of res-

triction endocuclease sites in DNA from Bahn's *Amy* ^{2,3} in comparison with *Amy* ^{1,3} (from Canton-S) and *Amy* ^{1,6} (from Suyama, Japan) indicates that the determinant of form 1 is to the left of those of forms 3 and 6 in the latter two chromosomes (Gemmill, Schwartz, and Doane, 1986, Nucl. Acids Res. 14: 5337-52). *Amy* ^{1} monomorphic allele in Oregon-R has been shown to be *Amy* ^{1,1} (Hawley, 1989, Ph.D thesis, Arizona State University).

cytology: Placed in 54A based on *in situ* hybridization (Gemmill, Levy, and Doane, 1985, Genetics 110: 299-312).

Amy-d: Amylase distal

The distal member of the *Amy* repeat. Electrophoretic alleles include *Amy-d* ^{3} (thermostable). *Amy-d* ^{6} and likely *Amy-d* ^{2} (thermolabile); some chromosomes apparently lack *Amy-d* activity.

Amy-p: Amylase proximal

The proximal member of the *Amy* repeat. Electrophoretic alleles include *Amy-p* ^{1}, *Amy-p* ^{2} (thermostable), *Amy-p* ^{4}, probably *Amy-p* ^{3} (thermolabile), and *Amy-p* ^{5}; a null allele also exists. Allelic compositions of various strains are tabulated in the accompanying table.

strain	source	Amy-p	Amy-d	ref $^{\alpha}$
Amy ^{1a}		*Amy-p* ^{1a}	inactive?	
Amy ^{1b}		*Amy-p* ^{1b}	inactive?	
Amy ^{1c}		*Amy-p* ^{1c}	inactive?	
Amy $^{1.2}$ β	*adp* ^{fs}, Kaduna	*Amy-p* ^{1}	*Amy-d* ^{2} thermolabile	*2, 3, 4, 5*
Amy $^{1.3}$	Canton-S	*Amy-p* ^{1}	*Amy-d* ^{3}	*3, 4, 5, 6*
Amy $^{1.4}$		*Amy-p* ^{1}	*Amy-d* ^{4}	
Amy $^{1.6}$	Suyama	*Amy-p* ^{1}	*Amy-d* ^{6} thermolabile	*3, 4, 5*
Amy ^{2}		*Amy-p* ^{2}	inactive?	
Amy $^{2.3}$	Copenhagen	*Amy-p* ^{2}	*Amy-d* ^{3}	*1, 3, 5*
Amy $^{2.6}$		*Amy-p* ^{2}	*Amy-d* ^{6}	
Amy $^{3.6}$ β	Kyoto	*Amy-p* ^{3} thermolabile	*Amy-d* ^{6} thermolabile	*3, 4*
Amy $^{4.5}$		*Amy-p* ^{4}	*Amy-d* ^{5}	
Amy $^{4.6}$	*adp* ^{60}, Kaduna	*Amy-p* ^{4} thermolabile	*Amy-d* ^{6} thermolabile	*1, 3, 4*
Amy ^{5}	Africa	*Adp-p* ^{5}	inactive ?	*8*
Amy $^{5.6}$	Africa	*Adp-p* ^{5}	*Adp-d* ^{6}	*8*
Amy ^{n}	Texas	inactive	inactive	*7*

$^{\alpha}$ *1* = Bahn, 1967, Hereditas 58: 1-12; *2* = Doane, 1967, J. Exp. Zool. 164: 363-78; *3* = Doane, 1969, Problems in Biology. RNA in Development (W. E. Hanley, ed.). Univ. Utah Press, Salt Lake City, pp. 73-109; *4* = Doane, 1969, J. Exp. Zool. 171: 321-42; *5* = Gemmill, Schwartz, and Doane, 1986, Nucl. Acids Res. 14: 5337-52; *6* = Levy, Gemmill, and Doane, 1985, Genetics 110: 313-24; *7* = Haj-Ahmed and Hickey, 1982, Nature 299: 350-52; *8* = Puijk and DeJong, 1972, DIS 49: 61.

$^{\beta}$ Tentative assignments.

molecular biology: Clones homologous to mouse α-amylase gene isolated from Maniatis library (Doane, Treat-Clemons, Gemmill, Levy, Hawley, Buchberg, and Paigen, 1983, Curr. Top. Biol. Med. Res. 9: 63-90). λDm32 hybridizes to polytene region 53CD; no homologous mRNA detected; postulated to be pseudogene. λDm65 hybridizes to 54A1-B1 and is homologous to a 1450-1500 nucleotide transcript (Gemmill, Levy, and Doane, 1985, Genetics 110: 299-312). 15kb insert in λDm65 contains reverse repeat by restriction mapping; subclones, each containing one of the repeated sequences, injected into Xenopus oocytes; one subclone capable of producing isozyme 1 of α-amylase; the other capable of producing isozyme 3; confirms duplicated nature of locus (Levy, Gemmill, and Doane, 1984, Isozyme Bull. 17; 1985, Genetics 110: 313-24). Directions of transcription of the two genes divergent. (Levy, 1985, Genetics 110: 137); separated by 4 kb. Seven strains exhibiting no, one, or two electrophoretic forms of α-amylase all carry the duplication as ascertained from restriction analysis. *Amy* ^{1c} contains an approximately 10 kilobase insert some 10 kb proximal to *Amy-p*. 12% of chromosomes isolated from diverse natural populations contain large inserts in the vicinity of the *Amy* loci (Langley, Shrimpton, Yamazaki, Miyashita, Matsuo, and Aguadro, 1988, Genetics 119: 619-29). Two molecular inversions that could have arisen through interlocus exchange recorded; one had normal levels of amylase activity (Langley *et al.*) and the other was a null allele that produced reduced levels of mRNA and was insensitive to glucose repression (Hickey, Benkel, Abukashawa, and Haus, 1988, Biochem. Genet. 26: 757-68; Schwartz and Doane, 1989, Biochem. Genet. 27: 31-46). An amylase cDNA has been cloned and sequenced (Boer, and Hickey, 1986, Nucleic Acid Res. 14: 8399-8411). This sequence shows 57% identity to mouse amylase; the predicted amino-acid sequence indicates a 54.5-kd polypeptide of 493 residues, the 18 N-terminal ones of which are signal sequence; there is 55.4% amino-acid identity with mouse amylase. Upstream sequence contains a repeated motif also found in a negatively regulated mammalian gene (Hickey, Genest, and Benkel, 1987, Nucleic Acid Res. 15: 7184). Northern blots probed with this cDNA show that the glucose repression effect is at the level of amylase mRNA abundance.

Amy ^{4}: see *Amy* ^{1,4}

Amy $^{+}$: see *Amy* ^{1}

Amy ^{ad}: see *Amy* ^{4,6}

Amy ^{s}: see *Amy* ^{2,6}

Amy ^{wh}: see *Amy* ^{1,4}

Amyloid protein precursor-like: see ***Appl***

an: ancon

location: 2-44 (34-54).

discoverer: Bridges, 30e3.

phenotype: Wings and legs somewhat short in *an* ^{1}; *an* ^{2} (CP627) more extreme with gnarled legs, scraggly abdominal bristles, etched sclerites; eyes small and roughish. *an* ^{1}/*an* ^{2} like *an* ^{2}.

alleles: *an* ^{1} and *an* ^{2}.

anarista: see ***aa***

ancon: see ***an***

And: Andante (J.C. Hall)

location: 1-36.2

origin: Induced by ethyl methanesulfonate.

discoverer: Konopka, R. Smith and Orr, 1976.

references: Jackson, Gailey, and Siegel, 1983, J. Comp.

Physiol. 151: 545-52.

phenotype: The normal free-running 24 hr periods of the circadian rhythms of eclosion and adult locomotor activity (in constant conditions) are lengthened by 1.5-2 hr/cycle; *And*/+ heterozygotes have a period phenotype intermediate between wild-type and mutant homozygotes (Konopka, R. Smith and Orr). The phase-response curves (PRCs) for eclosion and activity rhythms, indicating light-induced phase shifts, show a similar degree of lengthening as seen in free-running periodicities. *And* rhythms are highly temperature-compensated, as are those of wild-type (Konopka *et al.*). *And* males are defective in after effects on courtship behavior that are usually induced by prior exposure to mated females or very young males (Jackson *et al.*, 1983).

cytology: Placed in 10E1-2;10F1. The *And* homozygote-like activity rhythm phenotype is uncovered by *Df(1)KA6 = Df(1)10E1;11A7-8* and *Df(1)KA7 = Df(1)10A9;10F6-7,* but heterozygotes involving *And* and *Df(1)N105 = Df(1)10F7;11D1, Df(1)RA47 = Df(1)10F1;19F9-10,* or $Df(1)m^{259\text{-}4}$ = *Df(1)10C2-3;10E1-2* are like *And*/+ (Konopka *et al.*). The two *And*-uncovering *Df*'s just noted over wild type give normal periods. An anomaly then, is that *Df(1)HA85 = Df(1)10C1-2;11A1-2,* which uncovers *And* as it should (see above), leads to significantly longer-than-normal periods when over wild type (Konopka *et al.*).

other information: *And* lengthens in an additive manner, the periodicities associated with certain other rhythm mutants, *i.e.*, those which by themselves cause shorter- or longer-than-normal locomotor activity periods (*viz*, per^{S}, per^{L1}, per^{L2}, *Clk*). *And*, on its isolation, was associated with a *dy* wing phenotype, and the rhythm abnormality maps to the *dy-m* locus (see "cytology"); but *dy* and m^{D} have normal activity rhythm periods, and *And* over either of these two visibles gives the same periods as seen in *And*/+ (Konopka *et al.*); however, of four gamma-ray induced *dy*'s - dy^{n1}, dy^{n2}, dy^{n3}, and dy^{n4}, all but dy^{n2} are *And*-like in their locomotor activity rhythms (Jackson, Newby, and DiBartolomeis, 1989, Neurosci. Abstr. 15: 461).

ang: angle wing

location: 2-10.5.

origin: Spontaneous.

discoverer: Mittler and Goldberg, 48i16.

references: Mittler, 1950, DIS 24: 61.

phenotype: Wings held up from dorsal surfaces and extended outward 15-90° from the mid-dorsal line. Longitudinal dorsal median muscles 5 and 6 fused (Goldberg, 1954, Ph.D. Thesis, Ill. Inst. Technol.). No increase in expressivity with temperature. Does not overlap wild type. RK2.

ang: see *ano*

angle wing: see ***ang***

angle wing: see *agl*

angle winglike: see ***agl***

**ano: anomogenitals*

location: 1-35.7.

origin: Induced by triethylenemelamine.

discoverer: Fahmy, 1952.

synonym: Originally symbolized *ang*, but this symbol preoccupied.

references: 1958, DIS 32: 67.

phenotype: Many bristles on head and thorax either reduced in size or absent. Thoracic and abdominal hairs appreciably fewer. External male genitalia invariably abnormal, sometimes completely absent. Melanized exudate frequently present in furrow between mesonotum and scutellum near anterior scutellar bristles. Males sterile; viability less than 10% wild type. RK3.

ant: antennaless

location: 2- (not located).

origin: Spontaneous.

discoverer: Gordon, 1936.

references: 1941, DIS 14: 39.

1941, Proc. Intern. Congr. Genet., 7th. p. 131.

Gordon and Sang, 1941, Proc. Roy. Soc. (London), Ser. B 130: 151-84 (fig.).

Vogt, 1947, Biol. Zentr. 66: 388-95 (fig.).

phenotype: Antennae missing on one or both sides. Expression affected by residual genotype, nutritional environment, and temperature. Time of action about 70 hours after hatching [Begg and Sang, 1945, J. Exp. Biol. 21: 1-4 (fig.)]. Used in experiments to locate chemoreceptors [Begg and Hogben, 1946, Proc. Roy. Soc. (London), Ser. B 133: 1-19] and in studies of mating behavior (Begg and Packman, 1951, Nature 168: 953). RK3.

ANTC: The Antennapedia Complex
(T.C. Kaufman)

location: 3-47.5.

references: Kaufman, Lewis, and Wakimoto, 1980, Genetics 94: 115-33.

Lewis, Wakimoto, Denell, and Kaufman, 1980, Genetics 95: 393-97.

Denell, Hummels, Wakimoto, and Kaufman, 1981, Dev. Biol. 81: 43-50.

Kaufman, 1983, Time, Space, and Pattern in Embryonic Development, Alan R. Liss, New York, pp. 365-83.

Kaufman and Abbott, 1984, Molecular Aspects of Early Development, Plenum, New York, pp. 189-218.

Wakimoto, Turner, and Kaufman, 1984, Dev. Biol. 102: 147-72.

Regulski, Harding, Kostriken, Karch, Levin, and McGinnis, 1985, Cell 43: 71-80.

Gehring and Hiromi, 1986, Ann. Rev. Genet. 20: 147-73.

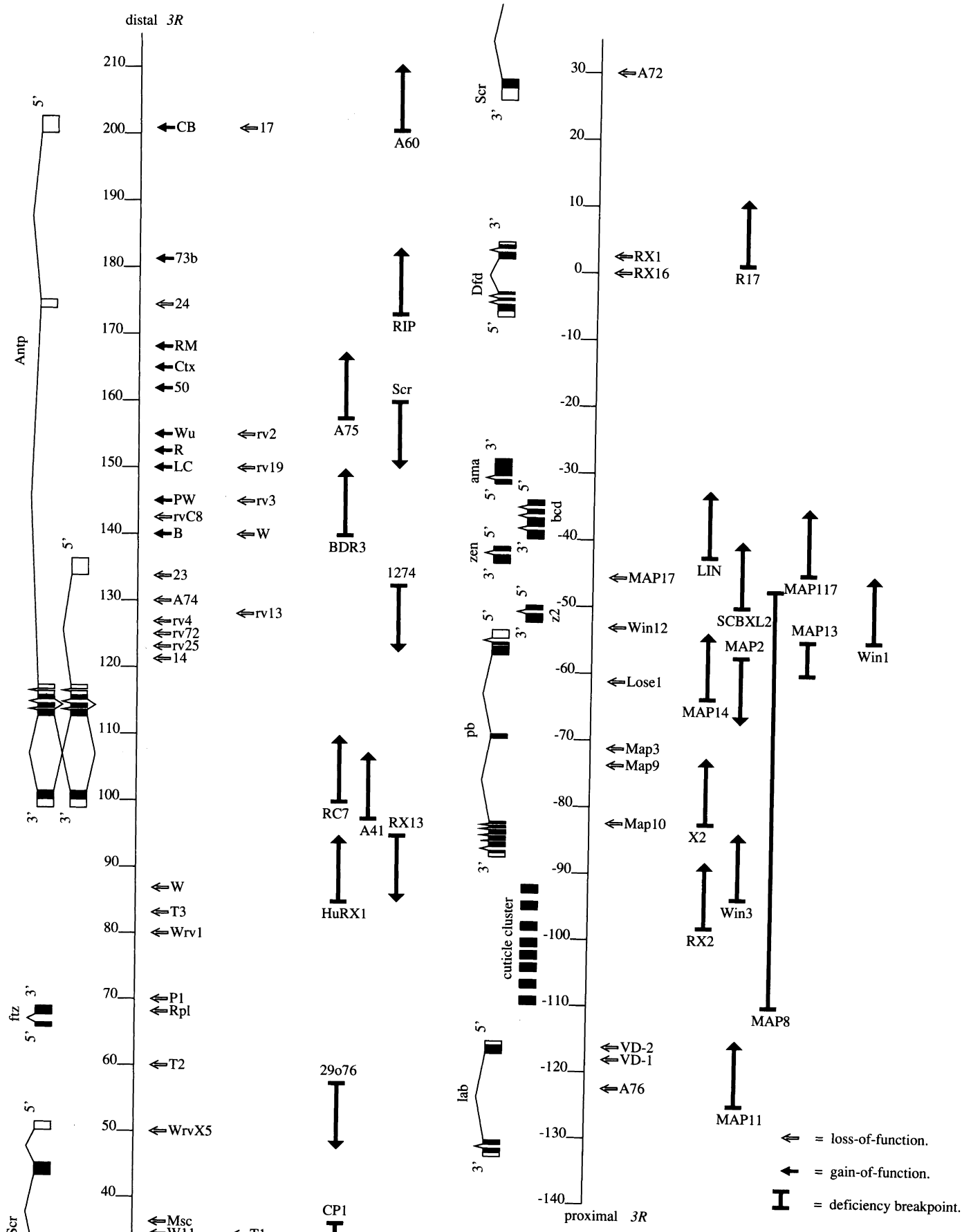
Akam, 1987, Development 101: 1-22.

Fechtel, Natzle, Brown, and Fristrom, 1988, Genes 120: 465-74.

Mahaffey and Kaufman, 1988, Developmental Genetics of Higher Organisms: A Primer in Developmental Biology, Macmillan, New York, pp. 329-59.

Kaufman, Seeger, and Olsen, 1990, Genetic Regulatory Hierarchies in Development, Academic Press, New York, pp. 309-62.

phenotype: The existence of the homeotic *ANTC* was originally proposed based on the tight linkage of the proboscipedia (*pb*). Sex combs reduced (*Scr*) and Antennapedia (*Antp*) loci. All were found to reside in a set of three doublet bands at the proximal end of section 84 (84A1,2-3,4 and 84B1,2) in the right arm of polytene chromosome 3. Subsequent genetic analyses have shown that two other homeotic loci labial (*lab*) and Deformed



ANTC: The Antennapedia Complex Map
From data supplied by T.C. Kaufman. Program by D. Conner.

(*Dfd*) are also members of the complex. The homeotic loci of the *ANTC* are involved in the specification of segmental identity in the posterior head (gnathocephalic) and anterior thoracic regions of the embryo and adult. Moreover the linear order of the homeotic loci in the complex *lab, pb, Dfd, Scr*, and *Antp* corresponds to the anterior posterior order of altered segments (intercalary, mandibular, maxillary, labial, and thoracic) found in animals bearing mutations in each of the resident loci. Specifically *Antp* transforms posterior T1, all of T2, and the anterior of T3, *Scr* transforms T1 and labial, *Dfd* affects the maxillary and mandibular lobes, *pb* affects the derivatives of the maxillary and labial segments and finally *lab* functions in the intercalary segment. Taken together the results of mutational analyses indicate that members of the complex are necessary to repress head development in the thorax (*Antp*) and elicit normal segmental identity in the anterior thorax (*Scr*) and posterior head (*Scr, Dfd, pb*, and *lab*). The *ANTC* is distinguished from the bithorax complex not only by virtue of the domain of action of its homeotic loci (anterior vs. posterior) but also by the residence of loci which are not homeotic in nature. Two of these fushi tarazu (*ftz*) and zerknüllt (*zen*) have been shown to affect segment enumeration (*ftz*) and the formation of dorsal structures (*zen*) in the early embryo. A third nonhomeotic gene is bicoid (*bcd*). Mutations in this locus result in female sterility and maternal effect lethality. Eggs laid by *bcd* females fail to develop normal anterior ends and instead produce mirror image duplications of structures normally produced at the posterior terminus of the embryo. In addition to these genetically defined loci there are several other "genes" which have been found in the *ANTC* by molecular mapping. The first of these is a cluster of cuticle-protein-related genes which map between the *lab* and *pb* loci. Eight small (*ca.* 1 kb) transcription units make up the cluster and all have sequence similarities to known cuticle protein genes. These "genes" are also apparently regulated by ecdysone in imaginal discs. Deletion of the entire cluster has no apparent effect on the development or cuticle morphology of embryos, larvae, or adults. The second molecularly identified "gene" is the Amalgam (*Ama*) transcription unit. The encoded protein places the gene in the immunoglobulin superfamily and like the cuticle cluster the locus can be deleted from the genome with no discernible effect on the organism.

cytology: Placed in the 84A1-B2 interval by the inclusion of the complex in *Df(3R)Scr* and the location of breakpoint associated inactivations of the *lab, pb, Dfd, Scr, ftz*, and *Antp* loci.

molecular biology: The entire complex has been cloned and has been shown to cover 335 kb of genomic DNA. The most distal transcription unit is *Antp* which covers the distal-most 100 kb of the complex and is made up of eight exons. Proximally the next 75 kb contain the *Scr* and *ftz* loci. The distal 50 kb of this interval house sequences necessary for *Scr* expression as well as the two exons of the *ftz* locus and its associated regulatory elements. The proximal 25-kb contain the three identified exons of the *Scr* transcription unit. The five exons of the *Dfd* gene are found in the central portion of the next-most-proximal 55-kb interval. The *Dfd* transcription unit covers only 11 kb of this region and it is likely that one or both of the 20-kb intervals flanking the gene are the location of *cis*-acting regulatory elements for the locus. The next 25-kb interval contains four of the nonhomeotic transcription units which help distinguish the *ANTC* and *BXC*. The distal most is *Ama*, next *bcd*, and finally *zen* and *z2*. The *z2*, *zen*, and *Ama* transcription units are all relatively small (1-2 kb) and comprise two exons each. The *bcd* gene is somewhat larger (3.6 kb) and is made up of four exons. Immediately proximal to the *z2* transcription unit (*ca.* 1 kb from its 3′ end) is the 5′ end of *pb*; the latter gene extends over the next 35 kb of genomic DNA and contains nine exons. The next 25 kb of the complex contain the cuticle cluster and its eight identified transcription units. The final 25 kb are the residence of the *lab* gene which is made up of three exons.

Despite the nonhomeotic nature of three of the smaller transcription units (*zen, bcd,* and *ftz*) resident in the complex, these loci are tied to the larger homeotic genes of the region by the nature of their protein products. All five of the large homeotics (*Antp, Scr, Dfd, pb*, and *lab*) and the three small genes have a homeobox motif and their protein products are found in the nuclei of the cells in which they are expressed. Thus eight of the genes in the *ANTC* encode regulatory proteins which act as transcription factors. The *z2* gene also contains a homeobox; however, the biological significance of the gene is not known as deletions of this transcription unit have no discernible effect. The cuticle-like genes and *Ama* do not contain a homeobox.

The reasons for the clustering of these developmentally significant loci of similar function is not known. The existence of common or overlapping regulatory elements, the need to insulate regulatory sequences from position effect and the possibility of higher order chromatin structures for proper expression have all been proposed. Whatever the reason, the homeotic complex structure has a long evolutionary standing. Similar clusters are found in vertebrates, an observation consistent with a very early origin of these genes, likely predating the separation of protostomes and deuterostomes.

Ama: Amalgam

location: 1-{47.5}.

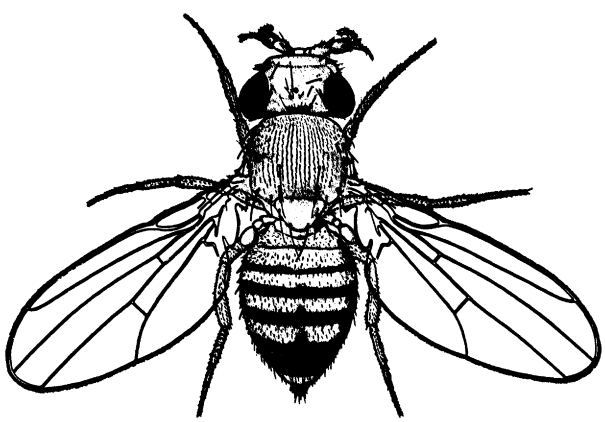
origin: Isolated as an unidentified third transcription unit in a 50 kb region known to harbor *bcd* and *zen*.

references: Seeger, Haffley, and Kaufman, 1988, Cell 55: 589-600.

phenotype: Antibody staining first detects Amalgam in the mesoderm during gastrulation; as neuroblasts delaminate from the ectoderm staining appears in a row of mesectodermal cells along the ventral midline of the extended germ band. Amalgam appears in the first neurons generated from the ganglion-mother cells, but not in the neuroblast precursors of these cells. Neuronal accumulation of *Ama* gene product increases during CNS development, but appears to be confined to the CNS and initially does not extend to axons exiting the CNS in segmental, intersegmental, or peripheral nerves; with time three rows of PNS-associated cells accumulate *Ama* protein; staining heavy around spiracle sensory organ and several cephalic sensory structures. Simultaneously there is a complicated temporal and spatial sequence of staining of mesodermal derivatives. Embryonic phenotype of deletion of *Ama* attributable to simultaneous deletion of *zen*; no effect of Ama^{-} detectable.

cytology: Placed in 84A1 based on its juxtaposition with *bcd*.

molecular biology: Sequence of a putatively full-length cDNA clone compared with that of the corresponding genomic region reveals a gene with a 316 bp intron in the 5′ untranslated region. Transcription from left to right. Conceptual amino-acid sequence indicates a protein product of 333 amino acids, the first 23 of which have the characteristics of a signal sequence. The sequence contains three internal repeats of approximately 100 amino acids each that exhibit homology to the immunoglobulin or Ig domain of vertebrates; each contains two widely spaced cysteine residues and the show 22-36% identity to one another with greatest identity found around the cysteines. There are two potential N-linked glycosylation sites in the first domain and one in the third. In addition there is a potential C-terminal membrane-attachment domain of amino acids. Comparison with sequences in the data base indicate that the Amalgam sequence is closest to members of the Ig class of proteins that act as cell-adhesion molecules.



Antp^{LC}: Antennapedia of Le Calvez

From Le Calvez, 1948, Bull. Biol. France Belg. 82: 97-113.

Antp: Antennapedia

location: 3-47.5.

references: Denell, 1973, Genetics 75: 279-97.
Struhl, 1981, Nature 292: 635-38.
Garber, Kuroiwa, and Gehring, 1983, EMBO J. 2: 2027-34.
Hafen, Levine, Garber, and Gehring, 1983, EMBO J. 2: 617-23.
Hazelrigg and Kaufman, 1983, Genetics 105: 581-600.
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Scott, Weiner, Polisky, Hazelrigg, Pirrotta, Scalenghe, and Kaufman, 1983, Cell 35: 763-76.
Hafen, Levine, and Gehring, 1984, Nature 307: 287-89.
Abbott and Kaufman, 1986, Genetics 114: 919-42.
Carroll, Laymon, McCutcheon, Riley, and Scott, 1986, Cell 47: 113-22.
Frischer, Hagen, and Garber, 1986, Cell 47: 1017-23.
Laughnon, Boulet, Bermingham, Laymon, and Scott, 1986, Mol. Cell Biol. 6: 4676-89.
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Schneuwly, Kuroiwa, Baumgartern, and Gehring, 1986, EMBO J. 5: 733-39.
Wirz, Fessler, and Gehring, 1986, EMBO J. 5: 3327-34.
Jorgensen and Garber, 1987, Genes Dev. 1: 544-55.
Schneuwly, Klemenz, and Gehring, 1987, Nature 325: 816-18.
Schneuwly, Klemenz, and Gehring, 1987, EMBO J. 6: 201-06.
Bermingham and Scott, 1988, EMBO J. 7: 3211-22.
Boulet and Scott, 1988, Genes Dev. 2: 1600-14.
Gibson and Gehring, 1988, Development 102: 657-75.
Muller, Affolter, Leupin, Otting, Wuthrich, and Gehring, 1988, EMBO J. 7: 4299-304.
Otting, Gottfried, Qian, Muller, Affolter, Gehring, and Wuthrich, 1988, EMBO J. 7: 4305-09.
Perkins, Daly, and Tjian, 1988, Genes Dev. 2: 1615-26.
Stroeher, Gaiser, and Garber, 1988, Mol. Cell Biol. 8: 4667-75.
Bermingham, Martinez-Arias, Petitt, and Scott, 1990, Development 109: 553-66.

phenotype: Null loss-of-function alleles result in embryonic lethality. Animals succumb at the end of embryogenesis and show homeotic transformations in the larval cuticle of the first, second, and third thoracic segments. Specifically the cuticle derived from parasegments 4 and 5 are transformed to a more anterior identity such that the posterior of the first thorax produces fragments of mouth hook material on its dorsal surface presumably owing to a new posterior labial identity, whereas the anterior of the second thorax resembles the first thorax. The anterior of the third thoracic segment is weakly transformed toward a T1-like identity. The posterior of T2 is presumably T1 like as there are no gnathal structures seen in this compartment. There are also partial loss-of-function mutations which allow survival into the larval, pupal, and adult stages. Those that allow adult survival produce animals in which the anterior of the dorsal mesothorax shows a transformation to prothorax. There are no other apparent defects associated with these lesions. Those "leaky" mutants which die in the pupal and larval stages show similar parasegmental transformations as the null alleles, except that only the parasegment 4 to 3 homeosis is generally apparent. Animals which survive to the pupal stage fail to evert their anterior spiracles resulting in a blunt appearance of the anterior pupa. This same phenotype is seen in genotypes which survive to the adult stage. These partial mutants in many cases are associated with chromosome rearrangements notably deletions which approach the locus from its distal end. Moreover these mutations have been shown to complement fully other seemingly null mutations. Subsequent molecular analyses have shown that these results are accounted for by the presence of two promotors, one, P1, distal to the other, P2. The partial mutants affect the ability of the P1 promotor to initiate transcription, while the complementing lesions inactivate P2. Null mutants affect the transcription unit and protein encoding portion of the gene which is common to both promotors (see below).

X-ray induced somatic clones of *Antp*$^{-}$ cells demonstrate that the locus is required in the adult for the proper development of the dorsal pro and mesothorax, and legs. The former is reduced in size presumably reflecting an anteriorward transformation while the latter are

transformed to antennae. Thus *Antp*$^{+}$ function is required in the embryo and adult in parasegments 4 and 5 to prevent more anterior segmental identities, specifically those normally found in the anterior thorax and head.

The *Antp* locus was initially recognized by virtue of several striking dominant gain-of-function alleles. Thirteen of these transform the antenna of the adult into a mesothoracic leg (*Antp*^{49}, *Antp*^{B}, *Antp*^{Yu}, *Antp*^{Pw}, *Antp*^{LC}, *Antp*^{R}, *Antp*^{Wu}, *Antp*^{50}, *Antp*^{RM}, *Antp*^{73b}, *Antp*^{CB}, *Antp*^{72j}, and *Antp*^{Ns}). Three of these also have effects on the orbit of the eye and the vibrissal region of the ventral head (*Antp*^{RM}, *Antp*^{72j}, and *Antp*^{Ns}). There are also two dominant alleles (*Antp*^{Ctx} *and Antp*^{W}) which transform portions of the head capsule (dorsal and posterior) and the eye to a dorsal mesothoracic identity. In some cases this includes the production of wing tissue in the eye. Finally, a unique dominant *Antp*^{Hu} produces bristles on the normally bald propleurae just ventral to the mesothoracic spiricle. This latter phenotype has been interpreted as the production of sternopleural bristles on the propleurae, and thus a T1 to T2 transformation. With the exception of *Antp*^{Ns} and *Antp*^{72j} all these dominant lesions are associated with recessive lethality and gross chromosome rearrangements. All the breakpoints fall in the interval between the distal and proximal promotors. The dominant gain-of-function phenotype results from the misregulation of the P2 promotor by position affect or by the production of novel transcripts initiated in the newly juxtaposed sequences and spliced to the downstream *Antp* coding sequences. Both events result in the ectopic accumulation of the *Antp* protein product in the eye-antennal disc where the normal head repressive function of the gene causes the observed alteration. The recessive lethality associated with these lesions falls into the partially deficient category mentioned above. That is, these lesions show complementation with the P2 specific (*Antp*^{1} and *Antp*^{23}) mutations and in general show only strong parasegment 4 → parasegment 3 transformations. However, there is a gradient of this affect among the breakpoints. Those closest to P1 and furthest from P2 are the weakest, whereas those close to P2 show the strongest phenotype and earlier lethal phase. This same result is obtained with breakpoint mutations in the P2-to-P1 interval which are not associated with a dominant phenotype. Therefore this interval likely contains sequences necessary for the proper regulation of the P2 promoter.

Three of the dominant gain-of-function lesions (*Antp*^{Hu}, *Antp*^{73b}, and *Antp*^{Ns}) have been reverted. The revertants are either complete nulls, thus obviating the potential for ectopic expression, or are partial mutants; the latter mutants likely remove the potential for ectopic expression by altering the juxtaposed sequences required for abnormal P2 activity.

Both *in situ* hybridization and immunostaining have been used to determine the spatio-temporal pattern of *Antp* expression. Both the protein and RNA are strongly accumulated in the ventral nerve cord and more weakly in the epidermis and mesoderm of the embryo. Protein and RNA are first detected during cellular blastoderm in a band of cells in the parasegment 4-6 anlagen. This initial spatial pattern is further elaborated at full germ-band extension. In the ectoderm *Antp* products are found starting in the region of the first thoracic segment (parasegments 3 and 4) and extending posteriorly to the level of the seventh abdominal segment. In the mesoderm, they are found in parasegments 4-6. During germ band shortening the gene products are accumulated in the CNS from parasegment 4 (posterior T1) through to the posterior end of the ventral nerve cord. In the integument transcripts and protein are mainly restricted to the parasegments 4-5 interval although some weak expression can be seen in parasegments 3. As embryogenesis proceeds, the posterior CNS expression diminishes but is still detectable at the end of embryogenesis. The major accumulation in the CNS at this time is in the neuromeres of parasegments 4 and 5. The mesodermal expression is found in the anterior midgut; quenching of *Antp* expression is found in the posterior portion of the anterior midgut and has been shown to be dependent on the expression of *Ubx*. In later stages *Antp* protein can be detected in the leg, dorsal prothoracic, and wing discs.

alleles:

allele	origin	discoverer	synonym	type	cytology
***Antp*^{1}**	X ray	Abbott	*Antp*^{a58}	hypomorphic	normal
***Antp*^{2}**	X ray	Abbott	*Antp*^{a60}	hypomorphic	*Df(3R)84B2;84D3*
***Antp*^{3}**	X ray	Abbott	*Antp*^{a62}	null	normal
***Antp*^{4}**	X ray	Abbott	*Antp*^{a71}	null	normal
***Antp*^{5}**	X ray	Abbott	*Antp*^{a74}	null	*In(3R)84B2;87C*
***Antp*^{6}**	X ray	Abbott	*Antp*^{a75}	hypomorphic	*Df(3R)84B2-C6*
***Antp*^{7}**	EMS	Denell	*Antp*^{d7}	null	normal
***Antp*^{8}**	DEB	Stephenson	*Antp*^{e8}	null	normal
***Antp*^{9}**	EMS	Fornili	*Antp*^{f9}	hypomorphic	normal
***Antp*^{10}**	EMS	Fornili	*Antp*^{f22}	null	normal
***Antp*^{11}**	EMS	Fornili	*Antp*^{f36}	null	normal
***Antp*^{12}**	EMS	Fornili	*Antp*^{f40}	null	normal
***Antp*^{13}**	EMS	Fornili	*Antp*^{f69}	null	normal
***Antp*^{14}**	X ray	Kaufman	*Antp*^{k4}	null	*T(2;3)36C-D; 84B1-2 + In(3LR)62B; 98F*
***Antp*^{15}**	EMS	Kaufman	*Antp*^{k5}	null	normal
***Antp*^{16}**	EMS	Matthews	*Antp*^{kml}	null	?
***Antp*^{17}**	X ray	Lopez	*Antp*^{l1}	hypomorphic	*T(2;3)25F;84B1-2*
***Antp*^{18}**	X ray	Lopez	*Antp*^{l2}	null	normal
***Antp*^{19}**	X ray	Pultz	*Antp*^{p4}	null	normal
***Antp*^{20}**	EMS	R. Lewis	*Antp*^{r4}	hypomorphic	normal
***Antp*^{21}**	EMS	R. Lewis	*Antp*^{r10}	null	normal
***Antp*^{22}**	EMS	R. Lewis	*Antp*^{r17}	null	normal
***Antp*^{23}**	X ray	Scott	*Antp*^{s1}	hypomorphic	normal
***Antp*^{24}**	X ray	Scott	*Antp*^{s2}	hypomorphic	*In(3R)80;84B1-2*
***Antp*^{25}**	EMS	Wakimoto	*Antp*^{w10}	null	normal
***Antp*^{26}**	EMS	Wakimoto	*Antp*^{w24}	null	normal
***Antp*^{49}**	X ray	Piternick	*Antp*^{4703}	weak	lesion in 84B1-2
***Antp*^{50}**	X ray	Piternick	*Antp*^{4715}	strong	extra band distal to 84B1-2
***Antp*^{59}**	X ray	Piternick		weak	= *Antp*^{49} ?
***Antp*^{60}**	X ray	Piternick		weak	= *Antp*^{50} ?
***Antp*^{72j}**	spont	Baker		viable	normal
***Antp*^{73b}**	spont	Green		strong	*In(3R)84B1-2*
***Antp*$^{73b\text{-}rv1}$**	spont	Green		*Antp*^{73b} revertant	
***Antp*$^{73b\text{-}rv2}$**	spont	Green		*Antp*^{73b} revertant	
***Antp*$^{73b\text{-}rv5}$**	X ray	Hazelrigg		*Antp*^{73b} revertant	*T(2;3)57B6-8; 84B1-2;97B3*
***Antp*$^{73b\text{-}rv7}$**	X ray	Hazelrigg		*Antp*^{73b} revertant	*T(2;3)40;84B1-2*
***Antp*$^{73b\text{-}rv8}$**	X ray	Hazelrigg		*Antp*^{73b} revertant	*Dp(3;3)84D5-8; 85F5-8*
***Antp*$^{73b\text{-}rv9}$**	X ray	Hazelrigg		*Antp*^{73b} revertant	*In(3R)84B1-2; 84C5-6*
***Antp*^{B}**	X ray	Bacon		moderate dominant	*In(3R)84B1-2;85E*
***Antp*^{CB}**	X ray	Black		moderate dominant	*In(3R)84B1-2; 99F-100A*
***Antp*^{Ctx}**	X ray	Lewis	*Ctx*	strong dominant	*T(2;3)35B;84B1-2*
***Antp*^{Hu}**	X ray	Ruch	*Hu*	moderate dominant	*In(3R)84B1-2; 84F4;86C7-8*
***Antp*$^{Hu\text{-}rv1}$**	X ray	Hazelrigg		*Antp*^{Hu} revertant	*Df(3R)84B1-2; 84D6-F4*

allele	origin	discoverer	synonym	type	cytology
Antp ^{JK}	spont	Kennison		recessive	
Antp ^{LC}	Neutron	LeCalvez	*Ar;ss* ^{Ar}	moderate dominant	*In(3R)84A5-6;92A5-6*
Antp ^{Ns}	spont	Gehring	*Ns*	viable dominant	normal
Antp $^{Ns\text{-}rv1}$	X ray	Denell		*Antp* ^{Ns} revertant	*In(3R)81;84B1-2*
Antp $^{Ns\text{-}rv2}$	γ ray	Duncan		*Antp* ^{Ns} revertant	*In(3R)81F;90BC*
Antp $^{Ns\text{-}rv3}$	γ ray	Duncan		*Antp* ^{Ns} revertant	*T(Y;3)Y;84A4-B2*
Antp $^{Ns\text{-}rv6}$	γ ray	Duncan		*Antp* ^{Ns} revertant	*In(3LR)79D1-2; 84A4-B2*
Antp $^{Ns\text{-}rv8}$	γ ray	Duncan		*Antp* ^{Ns} revertant	normal
Antp $^{Ns\text{-}rv11}$	X ray	Denell		*Antp* ^{Ns} revertant	normal
Antp $^{Ns\text{-}rv13}$	γ ray	Duncan		*Antp* ^{Ns} revertant	*T(2;3)84A4-B2; 40-41*
Antp $^{Ns\text{-}rv16}$	γ ray	Duncan		*Antp* ^{Ns} revertant	Complex
Antp $^{Ns\text{-}rv18}$	γ ray	Duncan		*Antp* ^{Ns} revertant	*T(Y;3)Y;84A4-B2*
Antp $^{Ns\text{-}rv19}$	γ ray	Duncan		*Antp* ^{Ns} revertant	*T(Y;3)Y;84B1-3*
Antp $^{Ns\text{-}rv25}$	X ray	Denell		*Antp* ^{Ns} revertant	*In(3R)81;* 84B1-2;85A
Antp $^{Ns\text{-}rv70}$	X ray	Denell		*Antp* ^{Ns} revertant	normal
Antp $^{Ns\text{-}rv72}$	X ray	Denell		*Antp* ^{Ns} revertant	*Df(3R)84B3;84D*
Antp $^{Ns\text{-}rv85}$	X ray	Denell		*Antp* ^{Ns} revertant	*In(3R)81;84B1-2*
Antp $^{Ns\text{-}rv96}$	X ray	Denell		*Antp* ^{Ns} revertant	*T(Y;3)Y;84B1-2;94C*
Antp $^{Ns\text{-}rvC1}$	EMS	Struhl		*Antp* ^{Ns} revertant	normal
Antp $^{Ns\text{-}rvC2}$	EMS	Struhl		*Antp* ^{Ns} revertant	normal
Antp $^{Ns\text{-}rvC3}$	EMS	Struhl		*Antp* ^{Ns} revertant	normal
Antp $^{Ns\text{-}rvC4}$	EMS	Struhl		*Antp* ^{Ns} revertant	*In(3LR)75B;84B1-2*
Antp $^{Ns\text{-}rvC5}$	EMS	Struhl		*Antp* ^{Ns} revertant	normal
Antp $^{Ns\text{-}rvC6}$	EMS	Struhl		*Antp* ^{Ns} revertant	normal
Antp $^{Ns\text{-}rvC8}$	EMS	Struhl		*Antp* ^{Ns} revertant	*T(2;3)41;84B1-2*
Antp $^{Ns\text{-}rvC9}$	EMS	Struhl		*Antp* ^{Ns} revertant	normal
Antp $^{Ns\text{-}rvC10}$	EMS	Struhl		*Antp* ^{Ns} revertant	*T(Y;3)Y;84B1-2*
Antp $^{Ns\text{-}rvC11}$	EMS	Struhl		*Antp* ^{Ns} revertant	normal
Antp $^{Ns\text{-}rvC12}$	EMS	Struhl		*Antp* ^{Ns} revertant	normal
Antp ^{PW}	MDN $^{\alpha}$	Pinchin		strong dominant	*In(3LR)71F;84B1-2*
Antp ^{R}	X ray	Rappaport	*ss* ^{a}	moderate dominant	*In(3R)84B1-2;86C*
Antp ^{RM}	X ray	R. Meyer		moderate dominant	*In(3R)82E1;84B1-2*
Antp ^{Scx}	spont	Hannah	*Scx*	weak dominant	normal
Antp ^{W}	X ray	Wohlwill		moderate dominant	*T(3;4)84B1-2;102F + T(2;3)33E;66C*
Antp ^{Wu}	γ ray	Wu		strong dominant	*In(3LR)75C;84B1-2*
Antp ^{Yu}	X ray	Yu		strong dominant	*T(2;3)22B;83E-F + T(2;3)38E;98A*

$^{\alpha}$ MDN = methoxy diethylnitrosamine.

cytology: Placed in 84B1-2 based on *Antp's* inclusion in the overlap region between *Df(3R)Scr* and *Df(3R)A41* as well as the commonly held breakpoint of four forward, eleven gain-of-function and eighteen revertant of gain-of-function mutations (see table of alleles).

molecular biology: The *Antp* transcription unit lies at the distal end of the *ANTC* and is transcribed in a distal to proximal (*i.e.*, left to right) direction with respect to the right arm of the third chromosome. The locus has been identified in the DNA through the localization of breakpoints associated with both loss- and gain-of-function mutations. Additionally regulatory portions of the gene have been used to drive the expression of β galactosidase reporter constructs *in vivo* and these constructs produce spatial patterns of expression similar to those seen for the normal gene. The identified transcription unit is 100 kb long and is made up of eight exons. Exons 1 and 2 are the most distal and are found at the 5′ end of RNAs initiated from the P1 promotor mentioned previously. Exon 3 is approximately 60 kb downstream of the P1 5′ end and represents the leader sequences unique to transcripts initiated at the P2 promotor. The remaining five exons (E4-E8) are common to transcripts initiated at both P1 and P2. Exon 4 also encodes a leader sequence and the identified open reading frame begins in exon 5, 36 nucleotides downstream of the splice acceptor. The open reading frame continues through exons 6, 7, and 8 ending 240 nucleotides downstream of the splice acceptor of E8. Two polyadenylation sites are used at the downstream end of E8. The first (A1) is *ca.* 875 nucleotides downstream of the 5′ end of the exon; the other (A2) is *ca.* 2300 nucleotides more proximal. The two promotors coupled with the two adenylation sites result in the production of four size classes of transcript (P1/A1 = 3.2 kb, P1/A2 = 4.6 kb, P2/A1 = 3.4 kb, P2/A2 = 4.8 kb). All of these have been seen on Northern blots. There is no apparent preferential association of promotor with respect to 3′ end formation. However, the two promotors do have different spatial patterns of expression. Notably the P1 promotor is seen to be strongly expressed in the anlagen of the dorsal prothoracic disc, a tissue dramatically affected by its deletion. The P2 promotor is more evenly expressed in *Antp's* spatial domain (see below), consistent with the defects associated with its inactivation. The 3′ end of the transcription unit is *ca.* 30 kb distal to the 3′ end of *ftz* and 50 kb distal to the identified 5′ end of *Scr*. The distance to the next most distal transcription unit from the P1 5′ end is nearly 50 kb. The site of the *Antp* ^{Hu} breakpoint is in this 50 kb interval.

In addition to the transcript heterogeneity mentioned above, *Antp* also undergoes alternate splicing among the ORF-containing introns. Specifically exon 6 which encodes thirteen amino acids is found predominantly in embryonic transcripts and less frequently in imaginal disc derived RNAs. Additionally there is an alternate splice at the 3′ end of exon 7, resulting in the deletion of four amino acids just upstream of the homeobox motif if the short splice is made. It appears that the long form splice is used preferentially but that all four potential protein forms are made in imaginal discs. The exon-6-less transcripts are rare in embryonic RNA. There is no apparent preferential association of alternate splicing patterns with either of the two promotors. The longest potential protein (E6 + 7L) is 378 amino acids in length, and has a predicted molecular weight of 43 kd. The homeobox motif is encoded in E8 and the opa like repeats in E5.

bcd: bicoid

location: 3-{47.5} (between *zen* and *Ama*).

synonym: *mum: multimorph.*

references: Frohnhöfer and Nüsslein-Vollhard, 1986, Nature (London) 324: 120-25 (fig.).

Fronhöfer and Nüsslein-Volhard, 1987, Genes Dev. 1: 603-14.

phenotype: Maternal-effect lethal mutations showing defective head and thorax development. Females homozygous for strong alleles produce embryos in which head and thorax are replaced by duplicated telson, including anal plates, tuft, spiracles, and filzkörper; however, no pole cells formed at the anterior end. Deletions and fusions of anterior abdominal segments and occasionally anterior abdominal segments in reversed polarity are also observed. Strong alleles amorphic based on phenotypic similarities of embryos produced by homozygous and hemizygous females. Weak alleles result in pattern defects in heads of embryos; lack only labral derivatives (median tooth, dorsal bridge); intermediate weak genotypes produce reduced head but retain normal thoracic development; intermediate strong produce further reduction of head, deletion of second and third and reduction

of first thoracic dentical belts; thoracic segments fused. Partial rescue of embryonic phenotype effected by injection of cytoplasm (5% of volume) from the anterior ends of unfertilized wild-type eggs into the anterior pole of newly fertilized eggs of *bcd* mothers; injection into ectopic sites stimulates differentiation of anterior structures at site of injection; efficiency proportional to number of bcd^+ alleles carried by cytoplasm donor. Phenocopies result from leakage of 5% of egg volume from anterior perforation of normal embryos. The distance of the head fold at gastrulation is proportional to the number of bcd^+ alleles in the maternal genotype. *bcd* mRNA appears as a flattened disc plastered to the anterior extremity of early embryos; by the time of pole cell migration it has become localized to the clear cytoplasm at the periphery, forming a cap over the anterior end of the egg and is distributed in a steeply decreasing gradient such that 90% of the RNA is in the anterior 18% of egg length; by nuclear cycle 14 the RNA begins to disappear and becomes undetectable by midway through cellularization. *bcd* protein on the other hand forms a shallower gradient in which 57% of protein is in the anterior 18% of egg length, and the gradient doesn't reach baseline until the posterior 30% of egg length; the gradient forms from two to four hours after oviposition in both fertilized and unfertilized eggs, and except during mitosis is concentrated in nuclei; diffusion postulated to account for the establishment of the protein gradient following translation from anteriorly anchored RNA. Protein levels decrease during cellularization, although some nuclear staining persists until the end of germ-band elongation. *bcd* transcript first detectable in the ovaries of *bcd* females; forms a ring around the anterior margin of the developing oocyte in stages 5 and 6; in stages 9 and 10 nurse-cell accumulation observed to be localized toward the periphery of the cyst; by stage 12 the nurse cells have emptied their contents into the oocyte and the *bcd* transcript appears as an anterior cap (St. Johnston, Driever, Berleth, Richstein, and Nüsslein-Volhard, 1989, Development Supplement: 13-19). No evidence of translation of *bcd* protein during oogenesis. Formation of the *bcd* gradient is regulated by three maternally active genes *exu, sww,* and *stau; exu* appears necessary for nurse cell accumulation; *sww* is required for anterior localization of *bcd* mRNA in the oocyte; and *stau* appears to be involved in RNA localization in the embryo. A defect in any of these functions results in little or no gradient of *bcd* activity. *bcd* in turn appears to control the activity of anterior gene activity; specifically the anterior pattern of *hb* expression is not observed and is replaced by a mirror-image posterior *hb* stripe in bcd^- embryos (Tautz, 1988, Nature 332: 281-84; Schröder, Tautz, Seifertz, and Jäckle, 1988, EMBO J. 7: 2881-87).

alleles:

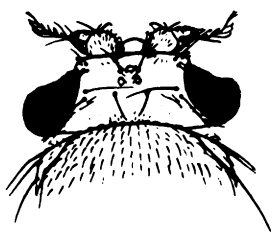
allele	origin	synonym	ref [α]	comments
***bcd*^{1}**	EMS	bcd^{085}	2, 5	intermediate allele; *2564* C → T; 184 gln → amber
***bcd*^{2}**	EMS	$bcd^{2\text{-}13}$	2, 5	weak allele; *3885* T → A; 453 leu → his
***bcd*^{3}**	EMS	$bcd^{23\text{-}16}$	2	strong allele
***bcd*^{4}**	EMS	$bcd^{33\text{-}5}$	2	strong allele
***bcd*^{5}**	EMS	bcd^{111}	2, 5	weak allele; *2798* C → T; 262 gln → amber
***bcd*^{6}**	EMS	bcd^{E1}	1, 2, 5	strong allele; *2482-2650* deleted + TA inserted; frameshift → 55 out-of-frame amino acids replacing amino acids 156-494, including homeodomain
***bcd*^{7}**	EMS	bcd^{E2}	1, 2	Strong allele; 260 base-pair deletion overlapping homeodomain
***bcd*^{8}**	EMS	bcd^{E3}	2, 5	intermediate allele; strongly temperature sensitive; *2406* C → T; 131 ser → leu
***bcd*^{9}**	EMS	bcd^{E4}	2, 5	intermediate allele; *2393* C → T; 127 leu → phe
***bcd*^{10}**	EMS	bcd^{E5}	2, 5	weak allele; *2804* C → T; 264 gln → amber
***bcd*^{11}**	EMS	bcd^{E6}	5	*2388-2420* deleted; amino acids 125-135 deleted
***bcd*^{12}**	EMS	bcd^{GB}	2, 5	strong allele; *2486* C → T; 158 gln → amber
***bcd*^{13}**			3	hypomorphic allele
***bcd*^{14}**			3	hypomorphic allele
***bcd*^{15}**			4	strong hypomorphic allele
***bcd*^{16}**			4	strong hypomorphic allele

α *1* = Berleth, Burri, Thoma, Bopp, Richstein, Frigerio, Noll, and Nüsslein-Volhard, 1988, EMBO J. 7: 1749-56; *2* = Fronhöfer and Nüsslein-Volhard, 1986, Nature 324: 120-25; *3* = Lambert, 1985, PhD Thesis, Indiana University; *4* = Seeger, 1989, PhD Thesis, Indiana University; *5* = Struhl, Struhl, and MacDonald, 1989, Cell 12: 59-73.

cytology: Placed in region 84A1 on the basis of failure to be complemented by *Df(3R)9A99 = Df(3R)83F2-84A1;84B1-2; Df(3R)LIN,* and *Df(3R)Scr = Df(3R)84A1-2;84B1-2,* and complementation by *Df(3R)4SCB = Df(3R)84A6-B1;84B2-3,* and *Df(3R)Antp17 = Df(3R)84A6;84D13-14.*

molecular biology: Gene identified in an 8.7-kb genomic fragment from coordinates –42 to –33 kb of the chromosome walk of Scott, Weiner, Hazelrigg, Polisky, Pirrotta, Scalenghe, and Kaufman (1983, Cell 35: 763-76) by germ-line transformants that completely rescue the mutant phenotype (Berleth, Burri, Thoma, Bopp, Richstein, Frigerio, Noll, and Nüsslein-Volhard, 1988, EMBO J. 7: 1749-56; see also Frigerio, Burri, Bopp, Baumgardner, and Noll, 1986, Cell 47: 735-46; Kilchherr, Baumgardner, Bopp, Frei, and Noll, 1986, Nature 321: 493-97). The transcription unit comprises four exons and produces a major mRNA of 2.6 kb, which contains all four exons, and a minor 1.6-kb mRNA from which exons 2 and 3 are spliced. Splice-acceptor-site variation in the third exon leads to translation products of 489 and 494 amino acids (53.9 kd). The first exon contains a PRD repeat, consisting essentially of alternating histidines and prolines, found within a number of genes, including *prd*, expressed early in development; the 5′ end of exon 3 encodes a novel homeodomain with no more than 40% amino-acid homology with other homeobox sequences; the 3′ end contains a series of repeated glutamines, opa repeats. Also contains a RNA-recognition motif, mostly in exon 4 (Rebagliatti, 1989, Cell 58: 231-32). A highly acidic C-terminal domain is thought to provide transcriptional activation; the latter can be replaced with heterologous activating sequences and still display bcd^+ activity (Driever, Ma, Nüsslein-Volhard, and Ptashne, 1989, Nature 342: 149-54). The sequence responsible for the anterior localization of *bcd* RNA at the anterior embryonic pole localized to 625 nucleotides in the 3′ untranslated region, which include regions capable of forming extensive secondary structure

(Macdonald and Struhl, 1988, Nature 336: 595-600). The ten residues from 138 to 147 comprise the DNA recognition helix of the *bcd* homeodomain; replacing the lysine in the ninth position of this ten-amino-acid sequence with either alanine or glutamine is sufficient to destroy recognition of *hb* sequences; in addition, the latter substitution confers a new specificity for *Antp* and *Ubx* upstream target sequences (Hanes and Brent, 1989, Cell 57: 1275-83). Bicoid protein binds to five high-affinity binding sites (consensus sequence TCTAATCCC) upstream from the *hb* transcription start site (Driever and Nüsslein-Volhard, 1989, Nature 337: 138-43). The posterior boundary of the anterior *hb* domain responds to changes in the number or affinity of these sites as well as to the dose of bcd^{+} such that increases cause a more posterior and decreases a more anterior boundary (Driever, Thoma, and Nüsslein-Volhard, 1989, Nature 340: 363-67; Struhl, Struhl, and Macdonald, 1989, Cell 57: 1259-73).



Dfd: Deformed

From Bridges and Morgan, 1923, Carnegie Inst. Washington Publ. No. 327: 94.

Dfd: Deformed

location: 3-47.5.

references: Chadwick and McGinnis, 1987, EMBO J. 3: 779-89.

Hazelrigg and Kaufman, 1983, Genetics 105: 581-600.

Jack, Regulski, and McGinnis, 1988, Genes Dev. 2: 635-51.

Kuziora and McGinnis, 1988, Cell 55: 477-85.

Martinez-Arias, Ingham, Scott, and Akam, 1987, Dev. 100: 673-83.

Merrill, Turner, and Kaufman, 1987, Dev. Biol. 122: 379-95.

Regulski, McGinnis, Chadwick, and McGinnis, 1987, EMBO J. 3: 767-77.

Chadwick, Jones, Jack, and McGinnis, 1990, Dev. Biol. 141: 130-40.

phenotype: Null mutations act as recessive lethals. Homozygous or hemizygous animals die at the end of embryogenesis and show a spectrum of defects in the head. There are no discernible defects in the trunk. The head defects are associated with missing structures normally derived from the mandibular and maxillary segments, the dorsal lateral papillae of the maxillary sense organ, the mouth hooks, and the maxillary cirri. The remaining gnathal structures are present albeit disarranged likely due to abnormalities in the movements associated with head involution. A weak homeotic transformation (30-50% penetrance) has also been noted in animals hemizygous for a breakpoint-associated revertant of the single dominant gain-of-function allele (Dfd^{rv1}). The phenotype is an apparent transformation of the H piece and lateral-graten which appear to be replaced by cephalopharyngeal plates. This phenotype has not been observed in any other mutant genotype and the reason for its low-penetrance production by this particular allele is not known. X-ray-induced somatic clones of Dfd^{-} cells have shown that the locus is also required for adult head development. These cells develop normally in the thorax and abdomen but do not form structures in the ventral anterior aspect of the head; specifically the vibrissae and maxillary palps. Clones in the dorsal posterior part of the head form ectopic bristles which have been interpreted as a head to thoracic transformation. A temperature-conditional allele has been used to define two temperature-critical periods for Dfd^{+} activity. The first is during embryogenesis during segmentation and head involution, while the second occurs in the late third instar larval through mid pupal stages. These times correlate nicely with the observed cuticular defects in mutant animals and the times of peak gene product accumulation. There is a single dominant gain-of-function allele which causes defects in the ventral aspects of the adult head similar to those seen in the Dfd^{-} head clones mentioned above. There are no defects seen in the posterior of the head nor does this allele cause any embryonic or larval defects as a heterozygote, homozygote, or hemizygote. This allele is associated with a group of *B104* (*roo*) insertion elements (*ca.* 50 kb of inserted DNA) as well as a duplication of the 3′ exons of the *Dfd* transcription unit (see below). The mutant causes an extended spatial domain of expression of the locus into the eye portion of the eye-antennal disc as compared to the pattern seen in normal animals. The precise cause-effect relationship between the observed molecular defect and the mutant phenotype is not known except that partial deletion of the *B104* elements but not the 3′ end duplication causes a reversion of the dominant phenotype and has no apparent effect on the wild type function of the resident *Dfd* gene. This dominant allele has been reverted and these revertants act as a simple recessive loss-of-function alleles with the one exception noted above. The *Dfd* transcript is initially detected at the blastoderm stage in a band of cells at the position of the future cephalic furrow. This RNA shows maximal accumulation from 6-12 hours of embryogenesis when it is found in the mandibular and maxillary lobes, as well as in the subesophageal reigon of the CNS. The amount of *Dfd* RNA diminishes through the first and second larval instars and peaks again during the third instar. At this point, it is found in the peripodial membrane cells of the eye-antennal discs. The cells which accumulate the RNA are those which have been fate mapped to give rise to the adult-head-capsule structures which are defective in Dfd^{-} clones. Antibodies raised to *Dfd* protein have shown a similar pattern of accumulation to that seen for the RNA. The protein is first detected in cellular blastoderm stage in a stripe of six cells which circumscribes the embryo. As germ-band elongation proceeds and segmentation becomes evident *Dfd* protein is detected in the mandibular and maxillary lobes and a portion of the dorsal ridge. During germ-band shortening protein is no longer detect-

able in the mandibular lobe or in the anterior lateral aspect of the maxillary lobe. The process of head involution carries the *Dfd*-expressing cells interiorly where they are found in portions of the pharynx at the end of embryogenesis. *Dfd*-positive cells are also found in the subesophageal region of the CNS in the maxillary ganglion. This expression pattern has been shown to be dependent on the prior expression of the gap and pair-rule segmentation genes for its inception and on an autogenous regulatory element upstream of the *Dfd* transcription initiation site for the maintenance of *Dfd* expression into the later stages of embryogenesis. Immunostaining of imaginal discs shows *Dfd*-positive cells in the peripodial membrane of the eye-antennal discs with no detectable accumulation in the disc proper. There are also a few cells in the stalk of the labial discs which appear to accumulate *Dfd* protein. The *Dfd* cDNA driven by a heat shock promotor has been returned to flies and used to ectopically express *Dfd* protein. Animals carrying this construct subjected to heat shock produce ectopic mouth hooks and maxillary cirri in the ventral aspect of their thoracic segments, two structures missing in Dfd^{-} animals. There is no phenotypic affect on abdominal pattern; however, head development is severely disrupted in heat-pulsed animals.

alleles:

allele	origin	discoverer	synonym	type	cytology
Dfd^{1}	spont	Cattell, 13g		dominant allele	normal
Dfd^{2}	EMS	Cain	Dfd^{rC9}	hypomorphic allele	normal
Dfd^{3}	EMS	Cain	Dfd^{rC11}	temperature sensitive	normal
Dfd^{4}	EMS	Fornili	Dfd^{rf1}	hypomorphic allele	normal
Dfd^{5}	EMS	Fornili	Dfd^{rf78}	hypomorphic allele	normal
Dfd^{6}	X ray	Kaufman	Dfd^{rK2}	null allele	?
Dfd^{7}	X ray	Kaufman	Dfd^{rK26}	hypomorphic allele	?
Dfd^{8}	EMS	Matthews	Dfd^{rKM2}	hypomorphic allele	normal
Dfd^{9}	EMS	Matthews	Dfd^{rKM24}	hypomorphic allele	normal
Dfd^{10}	EMS	R. Lewis	Dfd^{rR1}	hypomorphic allele	normal
Dfd^{11}	EMS	R. Lewis	Dfd^{rR3}	null allele	normal
Dfd^{12}	EMS	R. Lewis	Dfd^{rR11}	hypomorphic allele	normal
Dfd^{13}	EMS	Merrill	Dfd^{rV8}	hypomorphic allele	normal
Dfd^{14}	EMS	Merrill	Dfd^{rV13}	hypomorphic allele	normal
Dfd^{15}	EMS	Wakimoto	Dfd^{rW6}	hypomorphic allele	normal
Dfd^{16}	EMS	Wakimoto	Dfd^{rW21}	null allele	normal
Dfd^{rv1}	X ray	Hazelrigg	Dfd^{+RX1}	Dfd^{1} revertant	*Tp(3;3)83D4-5; 84A4-5;98F1-2*
Dfd^{rv2}	X ray	Hazelrigg	Dfd^{+RX13}	Dfd^{1} revertant	*Df(3R)83E3; 84A4-5*
Dfd^{rv3}	X ray	Hazelrigg	Dfd^{+RX16}	Dfd^{1} revertant	*Tp(3;3)86F11; 87D14;84A4-5*
Dfd^{rv4}	X ray	Hazelrigg	Dfd^{+RX17}	Dfd^{1} revertant	normal

cytology: Placed in 84A4-5 by its inclusion in *Df(3R)Scr, Df(3R)Antp17,* and *Df(3R)Dfd13* and the location of two revertant-associated breakpoints Dfd^{rv1} and Dfd^{rv2}.

molecular biology: The *Dfd* transcription unit has been identified in the *ANTC* by its association with two *Dfd* revertant breakpoints which interrupt it and result in the recessive lethal mutant phenotype. The identified transcription unit covers 11 kb of genomic DNA and is made up of five exons. The 5′-most three exons are separated by two relatively small introns and these are separated from the 3′-most two exons by a large 7-kb intron. Transcription proceeds from proximal to distal (with respect to the chromosome centromere to telomere). This orientation is opposite to that of all the other homeotic loci in the *ANTC*. The next most proximal gene in the complex is *Ama*, the 3′ end of which is just over 20 kb from the 5′ end of *Dfd*. Distally the 3′ end of *Dfd* is 20 kb from the 3′ end of Scr. The five exons sum to 2.75 kb, a figure in good agreement with the 2.8 kb transcript size seen in Northern blots. Sequence analysis of a full length cDNA shows a long open reading frame of 1758 nucleotides encoding a protein of 586 amino acids, yielding a molecular weight of 63.5 kd. The homeobox is encoded by exon four and the opa repeats are downstream in exon five.

ftz: fushi tarazu

location: 3-47.5.

references: Hafen, Kuroiwa, and Gehring, 1984, Cell 37: 833-41.
Jürgens, Wieschaus, Nüsslein-Volhard, and Kluding, 1984, Wilheim Roux's Arch. Dev. Biol. 193: 283-95.
Kuroiwa, Hafen, and Gehring, 1984, Cell 37: 825-31.
Laughon and Scott, 1984, Nature 310: 23-31.
Wakimoto, Turner, and Kaufman, 1984, Dev. Biol. 102: 147-72.
Weiner, Scott, and Kaufman, 1984, Cell 37: 843-51.
Carroll and Scott, 1985, Cell 43: 47-57.
Hiromi, Kuroiwa, and Gehring, 1985, Cell 43: 603-13.
Duncan, 1986, Cell 47: 297-309.
Hiromi and Gehring, 1987, Cell 50: 963-74.
Doe, Hiromi, Gehring, and Goodman, 1988, Science 239: 170-75.

phenotype: Null loss-of-function mutations result in embryonic lethality. Animals survive to the end of embryogenesis and exhibit a pair-rule mutant phenotype in the cuticle. This same phenotype is observable in animals at the beginning of segmentation of the germ band. Prior to deposition of cuticle, ftz^{-} animals have two rather than three mouth (gnathocephalic) segments and five as compared to ten trunk metameres. The material deleted is derived from the even-numbered parasegments, ps2 through ps12. Similar metameric deletions/fusions are seen in the neuromeres of the ventral nerve cord of the CNS. The name of the locus derives from the phenotype and is Japanese for "segment" (fushi) "deficient" (tarazu) (*N.B.* — there is only one letter t in tarazu; it is at the start of the word *i.e.*, there is **no** second t preceding the z). Temperature-sensitive alleles of the gene have shown that the temperature-critical period for viability and phenotype falls between 1 and 4 hours of embryogenesis with the mid point of 2.5 hours at the blastoderm stage. The recovery of clones of ftz^{-} cells created by X-ray-induced somatic exchange after cellular blastoderm have demonstrated that ftz^{+} activity is not necessary for normal cuticular morphogenesis subsequent to this point in development. In addition to these recessive null and hypomorphic alleles there are two classes of dominant gain-of-function lesions at the *ftz* locus. The first, *ftz-Regulator of postbithorax-like*, causes a variable transformation of the posterior haltere into posterior wing. The second, *ftz-Ultra-abdominal-like*, is associated with a patchy transformation of the adult first abdominal segment toward third abdominal identity. The former (ftz^{Rpl}) lesion also shows a recessive loss-of-function phenotype while the latter class (ftz^{Ual}) has no discernable embryonic phenotype and is homozygous viable. The fact that these dominant alleles produce mutant phenotypes that mimic lesions in the *BXC* has been inter-

preted as demonstrating a regulatory link between the segment enumeration genes and the homeotics.

alleles:

allele	origin	discoverer	synonym	type	cytology
ftz ^{1}	DEB	M. Bender	*ftz* ^{b54ts}	hypomorphic allele	normal
ftz ^{2}	DEB	M. Bender	*ftz* ^{b5ts}	hypomorphic allele	normal
ftz ^{3}	EMS	Cain	*ftz* ^{c15}	null allele	normal
ftz ^{4}	EMS	Cain	*ftz* ^{clts}	hypomorphic allele	normal
ftz ^{5}	EMS	Fornili	*ftz* ^{f47ts}	hypomorphic allele	normal
ftz ^{6}	EMS	Kaufman	*ftz* ^{k5}	null allele	normal
ftz ^{7}	EMS	Matthews	*ftz* ^{km13}	null allele	normal
ftz ^{8}	EMS	K. Matthews	*ftz* ^{kmQ}	null allele	normal
ftz ^{9}	EMS	R. Lewis	*ftz* ^{R13}	null allele	normal
ftz ^{10}	EMS	R. Lewis	*ftz* ^{R14}	null allele	normal
ftz ^{11}	EMS	Wakimoto	*ftz* ^{w20}	null allele	normal
ftz ^{12} α	EMS	Jürgens	*ftz* ^{7B}		
ftz ^{13}	EMS	Jürgens	*ftz* ^{9H34}		
ftz ^{14}	EMS	Jürgens	*ftz* ^{9093}		
ftz ^{15}	EMS	Jürgens	*ftz* ^{E66}		
ftz ^{16}	EMS	Jürgens	*ftz* ^{E193}		
ftz Rpl	X ray	Duncan	*ftz* ^{Rpl}	dominant allele	*T(2;3)84A6-B1;41*
ftz Ual1	EMS	E.B. Lewis	*ftz* ^{Ual1}	dominant allele	normal
ftz Ual2	ENU	Chiang	*ftz* ^{Ual2}	dominant allele	normal
ftz Ual3	EMS	Duncan	*ftz* ^{Ual3}	dominant allele	normal
ftz Ual2rv1	EMS	Duncan	*ftz*$^{-}$	revertant of *ftz* ^{Ual2}	normal
ftz Ual2rv2	X ray	Duncan	*ftz*$^{-}$	revertant of *ftz* ^{Ual2}	normal
ftz Ual2rv3 β	spont	Duncan	*ftz*$^{-}$	revertant of *ftz* ^{Ual2}	?

α Associated with a 5-kb insertion element in the transcribed region of *ftz*.

β Behaves genetically as a deletion of *ftz*, *Scr*, and *Antp*.

cytology: Placed in 84B1-2 based on its inclusion in *Df(3R)Scr* and the *3R* breakpoint of *T(2;3)ftz* ^{Rpl}, which is known to interrupt the coding region of the *ftz* transcription unit.

molecular biology: The localization and identification of the *ftz* transcription unit within the *ANTC* has been accomplished through the mapping of *ftz*-associated aberrations in the DNA [*ftz* ^{11} and *T(2;3)ftz* ^{Rpl}] and the rescue of *ftz*$^{-}$ genotypes using *P*-element mediated transformation. The transcription unit is just over 2 kb in length and is made up of two exons of 800 and 980 base pairs and a single 150-base-pair intron. The open reading frame is 1,239 nucleotides long and initiates in the (800 bp) 5′ exon. Conceptual translation of the open reading frame predicts a protein of 398 amino acids with a molecular weight of 43 kd. The most prominent motifs in the protein are the homeodomain (encoded in the second exon) and a PEST domain which may be important in the dynamic pattern of *ftz* expression. Northern blots have shown that the *ftz* transcript is accumulated in early embryos starting at about 2 hours (syncytial blastoderm), peaking shortly afterwards and declining at about 4 hours. These times are coincident with the temperature-sensitive-period data noted above. The spatial pattern of RNA accumulation is first seen as a broad band at syncytial blastoderm extending from the position of the future cephalic furrow posteriorly to about 15% egg length. At cellular blastoderm this broad single band resolves into seven transverse stripes which circumscribe the embryo. These stripes disappear as gastrulation proceeds and are gone by mid gastrulation. Protein accumulation lags behind the RNA and is first detected at cellular blastoderm in the seven-stripe pattern. The position and width of the stripes indicates that *ftz* expression occurs within the even-numbered parasegmental anlagen, which are missing in *ftz*$^{-}$ animals. Subsequent to the ectodermal expression in the germ band, the *ftz* protein product is again detected in the later stages of germ-band shortening, in a subset of cells in each of the segmental ganglia of the ventral nerve cord. This expression continues to the end of embryogenesis and has been shown to be important in the proper morphogenesis of a specific set of neurons repeated in each ganglion. Transformation studies have resulted in the identification of at least three *cis*-acting upstream regulatory elements necessary for normal *ftz* expression. An 1-kb fragment just upstream of the start of transcription is necessary for the establishment of the striped pattern at cellular blastoderm. Another fragment just distal to this element is needed for expression in the CNS, while about 6 kb upstream is an element necessary for the maintenance of stripes. It has also been shown that this *cis*-acting maintenance element requires the presence of *ftz* protein and therefore that *ftz* is apparently autogenously regulated in the later stages of its expression.

lab: labial

location: 3-47.5.

references: Diederich, Merrill, Pultz, and Kaufman, 1989, Genes Dev. 3: 399-414.

Merrill, Diederich, Turner, and Kaufman, 1989, Dev. Biol. 135: 376-91.

Mlodzik, Fjose, and Gehring, 1988, EMBO J. 7: 2569-78.

phenotype: Null mutations act as recessive embryonic lethals. Animals survive to the end of embryogenesis and have normal thoracic, abdominal, and caudal segments. However, the head is abnormal, and shows defects in derivatives of all of the gnathocephalic segments. There is no obvious homeotic transformation in these animals. Analysis of earlier stages shows abnormalities in the process of head involution. X-ray-induced clones of *lab*$^{-}$ cells demonstrate that *lab* function is unnecessary for the development of the adult thorax and abdomen. However, clones in the head fail to develop normally and show deletions in the maxilla and eye. Dorsally the posterior head capsule is transformed toward an apparent thoracic identity. A temperature conditional allele has been used to show a temperature critical period between 6 and 14 hours of embryogenesis. This period coincides with an interval in which head involution, a process disrupted by *lab*$^{-}$, takes place. Antisera raised to *lab* protein have shown it to initially accumulated just anterior to the gnathocephalic region of the germ band at the early stages of segmentation. This protein also is found in a row of cells extending above the gnathal region in the procephalic lobe and more dorsally into the dorsal ridge. As segmentation, germ-band shortening and head involution proceed, the cells expressing the protein are involved in the process complexities of head involution. Finally at the end of morphogenesis, *lab* positive cells are found in the lateral aspects of the pharynx, the tritocerebral ganglia of the CNS, and the frontal sac. In addition to this expression in the head, *lab* protein is also found in endodermal cells at the posterior of the anterior midgut and the anterior cells of the posterior midgut. The position and movements of the cephalic cells accumulating *lab* is consistent with the interpretation that this locus is expressed in the intercalary or most anterior of the gnathal segments.

alleles:

allele	origin	discoverer	synonym	comments
lab ^{1}	EMS	R. Lewis	*lab* ^{r9}	hypomorphic allele
lab ^{2}	X ray	Kaufman	*lab* ^{k3}	temperature-sensitive allele
lab ^{3}	EMS	Fornili	*lab* ^{f7}	hypomorphic allele
lab ^{4}	EMS	Fornili	*lab* ^{f8}	null allele
lab ^{5}	EMS	Fornili	*lab* ^{f10}	hypomorphic allele
lab ^{6}	EMS	Fornili	*lab* ^{f33}	hypomorphic allele
lab ^{7}	EMS	Fornili	*lab* ^{f40}	null allele
lab ^{8}	EMS	Fornili	*lab* ^{f56}	hypomorphic allele
lab ^{9}	X ray	Abbott	*lab* ^{a76}	null allele; *In(3R)84A1-2;84E*
lab ^{10}	EMS	Merrill	*lab* ^{v14}	hypomorphic allele
lab ^{11}	DEB	Seeger	*lab* ^{l5}	null allele
lab ^{12}	DEB	Seeger	*lab* ^{l10}	hypomorphic allele
lab ^{13}	DEB	Seeger	*lab* ^{lB1}	hypomorphic allele
lab ^{14}	X ray	Diederich	*lab* ^{vd1}	null allele; (insertion)
lab ^{15}	X ray	Diederich	*lab* ^{vd2}	hypomorphic allele; (deletion)
lab ^{16}	X ray	Merrill	*lab* ^{vd21}	null allele; *T(3;4)84A1-2;101*
lab ^{17}	X ray	Merrill	*lab* ^{vd22}	hypomorphic allele
lab ^{18}	X ray	Merrill	*lab* ^{vd35}	hypomorphic allele

cytology: Placed in 84A1-2 based on its inclusion in *Df(3R)Scr* and the location of the proximal *3R* breakpoints of two rearranged alleles *In(3R)lab* ^{9} and *T(3;4)lab* ^{16}. These latter two breakpoints have been located in the DNA and are known to interrupt the *lab* transcription unit.

molecular biology: The *lab* transcription unit is the most proximal in the *ANTC* and has been localized and identified by mapping the position of four *lab*$^{-}$ associated rearrangements in the DNA (*lab* ^{9}, *lab* ^{14}, *lab* ^{15}, and *lab* ^{16}) and the rescue of *lab*$^{-}$ animals by a minigene constructed from the transcription unit implicated by the breakpoints. The *lab* transcription unit is 17 kb in length, is made up of three exons and is transcribed from distal to proximal on the chromosome. Exons two and three are separated by a 245-bp intron and these from the 5′ exon by a 13.8-kb intron. The open reading frame begins at nucleotide +239 in the first exon and extends through the third. Conceptual translation of the open reading frame predicts a protein of 629 amino acids and a molecular weight of 67.5 kd. Northern blot analysis detects a single Poly(A)$^{+}$ RNA of 3.0 kb, a size in good agreement with the identified exons of 1455, 416, and 935 bp. This RNA is first detected at 2-4 hours of embryogenesis and remains present through the larval and pupal stages. There is no detectable accumulation in adults. The encoded protein contains opa sequences as well as a homeodomain. The latter is encoded by sequences in exons two and three, has its closest similarity to the *pb* homeodomain, and shares with that homeobox the position of its intronic interruption.

pb: proboscipedia

location: 3-47.5.

references: Bridges and Dobzhansky, 1933, Wilhelm Roux's Arch. Dev. Biol. 127: 575-90.

Kaufman, 1978, Genetics 90: 579-96.

Lewis, Wakimoto, Denell, and Kaufman, 1980, Genetics 95: 383-97.

Pultz, Diederich, Cribbs, and Kaufman, 1988, Genes Dev. 2: 901-20.

phenotype: Null alleles transform the labial palps of the adult into portions of the prothoracic leg. The distal tarsal segments are present, including claws and pulvilli. The distal portion of the first tarsal segment including the sex comb in males is fused directly to the proximal portion of the femur. Thus proximal first tarsus, tibia, and distal femur are absent. Leg segments proximal to femur are not present. Hypomorphic alleles produce a labial-palp-to-antenna transformation. Generally only more distal (arista) antennal structures are seen. Extremely weak hypomorphic alleles exist which produce no ostensible phenotype as homozygotes but do reveal a weak antennal transformation in combination with a deletion or null allele. Both null and hypomorphic alleles also show an alteration in maxillary palp morphology which has been interpreted as a transformation toward an antennal identity.

alleles:

allele	origin	discoverer	synonym	phenotype	cytology
pb ^{1}	spont	Bridges and Dobzhansky		18° → antenna + 29° → leg	+
pb ^{2}	γ ray	Duncan and Kaufman		leg	+
pb ^{3}	γ ray	Duncan and Kaufman		leg	+
pb ^{4}	γ ray	Duncan and Kaufman		antenna	+
pb ^{5}	γ ray	Duncan and Kaufman		leg	+
pb ^{6}	spont	Baker, W.K.	*pb* ^{72j}	weak antenna	+
pb ^{7}	X ray	Abbott	*pb* ^{a19}	leg	+
pb ^{8}	X ray	Abbott	*pb* ^{a21}	leg	+
pb ^{9}	X ray	Abbott	*pb* ^{a70}	leg	+
pb ^{10}	X ray	Diederich	*pb* ^{bd4}	leg	+
pb ^{11}	EMS	Cain	*pb* ^{c13}	antenna	+
pb ^{12}	X ray	Kaufman	*pb* ^{draw1}	leg	+
pb ^{13}	EMS	Fornili	*pb* ^{f4}	weak antenna	+
pb ^{14}	EMS	Fornili	*pb* ^{f73}	18° → antenna + 29° → leg	+
pb ^{15}	EMS	Hazelrigg	*pb* ^{h62}	antenna	+
pb ^{16}	X ray	Kaufman	*pb* ^{lose1}	leg	*In(3LR)66B;84A4-5*
pb ^{17}	EMS	Matthews	*pb* ^{m1}	leg	+
pb ^{18}	EMS	Matthews	*pb* ^{m2}	leg	+
pb ^{19}	EMS	Matthews	*pb* ^{m3}	leg	+
pb ^{20}	X ray	Pultz	*pb* ^{map2}	leg	*Df(3R)84A4-5*
pb ^{21}	X ray	Pultz	*pb* ^{map3}	leg	*T(2;3)84A4-5;26D-F*
pb ^{22}	X ray	Pultz	*pb* ^{map6}	leg	+
pb ^{23}	X ray	Pultz	*pb* ^{map8}	leg	+ (deletion)
pb ^{24}	X ray	Pultz	*pb* ^{map9}	leg	*In(3R)84A4-5;84D*
pb ^{25}	X ray	Pultz	*pb* ^{map10}	leg	*In(3R)84A4-5;het*
pb ^{26}	X ray	Pultz	*pb* ^{map12}	leg	*In(3R)84A4-5;85D*
pb ^{27}	X ray	Pultz	*pb* ^{map13}	leg	+ (deletion)
pb ^{28}	X ray	Pultz	*pb* ^{map17}	variegating leg	*In(3R)84A4-5;het*
pb ^{29}	EMS	Merrill	*pb* ^{v10}	leg	+
pb ^{30}	EMS	Merrill	*pb* ^{v12}	leg	+
pb ^{31}	EMS	Wakimoto	*pb* ^{w4}	18° → leg 29° → antenna	+
pb ^{32}	EMS	Wakimoto	*pb* ^{w19}	antenna	+
pb ^{33}	X ray	Kaufman	*pb* ^{win1}	leg	*Df(3R)84A4-5;84B1-2*
pb ^{34}	X ray	Kaufman	*pb* ^{win3}	leg	*Df(3R)84A4-5;84C1-2*
pb ^{35}	X ray	Kaufman	*pb* ^{win5}	leg	+
pb ^{36}	X ray	Kaufman	*pb* ^{win12}	leg	*In(3R)84A4-5;87A5*
pb ^{37}	X ray	Kaufman	*pb* ^{x1}	leg	+
pb ^{38}	X ray	Kaufman	*pb* ^{x2}	leg	*Df(3R)84A4-5;84B1-2*
pb ^{39}	X ray	Kaufman	*pb* ^{x3}	leg	*T(2;3)44F;84D*
pb ^{40}	X ray	Matthews	*pb* ^{x4}	leg	+

cytology: Placed in 84A4-5 based on its inclusion in *Df(3R)Scr* and the location of eleven *pb*-associated breakpoints in this doublet (see table of alleles).

molecular biology: The *pb* transcription unit extends over

35 kb of DNA in the proximal portion of the *ANTC*. It is bounded distally by the z2 transcription unit and proximally by a cluster of at least eight cuticle-like genes. Neither the proximal nor distal transcripts have any demonstrable function in the fly. The transcription unit produces a single 4.3-kb mRNA which is derived from nine exons distributed over the interval. The open reading frame begins in exon two and ends in exon nine. The homeobox motif is encoded in exons four and five and is split by intron four in the same position as the homeobox is split in the labial gene. The opa sequences are in exon eight and are therefore downstream of the homeobox. Exon three is roughly equidistant between its two flanking exons and the two largest introns of the gene. This exon is 15 nucleotides long and is alternately spliced. The RNA and protein products of the gene are accumulated in the maxillary and mandibular lobes of the embryo and the labial discs of the larvae.

Scr: Sex combs reduced

location: 3-47.5.

references: Kuroiwa, Kloter, Baumgartner, and Gehring, 1985, EMBO J. 4: 3757-64.
Sato, Hayes, and Denell, 1985, Dev. Biol. 111: 171-92.
Mahaffey and Kaufman, 1987, Genetics 117: 51-60.
Martinez-Arias, Ingham, Scott, and Akam, 1987, Development 100: 673-83.
Riley, Carroll, and Scott, 1987, Genes Dev. 1: 716-30.
Carroll, DiNardo, O'Farrell, White and Scott, 1988, Genes Dev 2: 350-60.
Glicksman and Brower, 1988, Dev. Biol. 127: 113-18.
LeMotte, Kuroiwa, Fessler, and Gehring, 1989, EMBO J. 8: 219-27.
Mahaffey, Diederich, and Kaufman, 1989, Development 105: 167-74.

phenotype: Null mutations at the locus result in embryonic lethality. Animals die at the end of embryogenesis and show evidence of homeotic transformation in the cuticle derived from the labial and first thoracic segments. The first thorax is transformed to a second thoracic identity and the labial segment toward maxillary. This latter phenotype is seen as a duplication of the maxillary sense organs and the cirri. Deletions of the locus as well as null alleles also produce a dominant phenotype most clearly seen in males as a reduction in the number of sex-comb teeth. This reduction is indicative of a partial transformation of first leg to second, a conclusion borne out by the recovery of hypomorphic alleles of the locus which as hemizygotes allow survival to the adult stage and have no obvious effect in the embryo. These survivors show a complete transformation of ventral prothorax to mesothorax including the presence of stenopleural bristles on the propleurae; they also show an apparent transformation of the dorsal prothorax toward a mesothoracic identity. In addition to these thoracic transformations, the labial palps are transformed toward a maxillary palp morphology. All of these adult transformations can also been seen in X-ray-induced somatic clones of Scr^- cells. Thus *Scr* activity is needed for proper segmental identity in both the embryo and adult in the anterior-most segment of the thorax and the posterior-most metamere of the head. In the absence of *Scr* product these two segments are transformed divergently to the identity of the next most posterior and anterior metamere respectively. The only other homeotic mutation to produce such a divergent homeosis is *pb*, which appears to act similarly in the adjacent maxillary and labial segments of the adult head. In addition to these loss-of-function mutations there are several gain-of-function dominant alleles. All result in a similar phenotype in adults, most clearly seen in males as the production of sex combs on the second and third thoracic legs. Additionally, strong alleles of this type (Scr^{ScxW}, Scr^{ScxP}, and Scr^{ScxS}) show the loss of sternopleural bristles indicative of a more complete transformation of mesothorax to prothorax. All of these dominants are associated with genomic rearrangements and with the exception of Scr^{ScxS} act as recessive lethals (Scr^{Msc}, Scr^{ScxT1}, Scr^{ScxT2}, and Scr^{ScxP}) or semilethals (Scr^{ScxW} and Scr^{ScxT3}) at the locus. Examination of animals carrying these lesions at the end of embryogenesis as heterozygotes with a normal chromosome or hemizygotes reveals no evidence of the gain-of-function transformation of T2 and T3 → T1, only the loss-of-function phenotypes described above. These phenotypic observations have been extended by showing that *Scr* protein is accumulated ectopically in the second and third leg imaginal discs in dominant gain-of-function genotypes but not in the second and third thoracic segments at any point in embryogenesis. Thus it appears that the spatial pattern of *Scr* expression is differentially regulated at these two times. Genetic analyses have shown that at least one difference lies in *Scr* imaginal expression being subject to a transvection-like effect. The gain-of-function lesions cause or allow the ectopic expression of the structural gene on the *trans*- rather than the *cis*-coupled transcription unit. This is most clearly seen in the case of Scr^{ScxT1}, which is broken within the transcribed portion of *Scr* and is therefore incapable of making a functional gene product. *Scr* mRNA is first detected in embryos in early gastrulae in a band of cells just posterior to the cephalic furrow. Protein is not detected at this time but later during germ-band elongation; it is found in the region of the labial lobe. Subsequently, during germ-band retraction, RNA and protein are detected in the first thoracic segment with the highest concentration at the anterior border of this segment. RNA and protein are also detected in the subesophageal region of the CNS in the labial ganglion and in mesodermal cells associated with the anterior midgut. As head involution proceeds, the *Scr*-expressing cells of the labial segment are carried inside where they are found associated with the pharynx and the mouthparts at the end of embryogenesis. In the third larval instar, protein is found in the prothoracic leg discs, the dorsal prothoracic discs, the labial discs, and a small group of cells in the stalk of the antennal portion of the eye-antennal disc where it attaches to the mouthparts. In addition to this disc expression, *Scr* protein is accumulated in the subesophageal region of the CNS. This spatial pattern of expression in the epidermis is consistant with the spectrum of defects seen in Scr^- animals and clones.

alleles:

allele	origin	discoverer	synonym	type	cytology
***Scr*^{1}**	EMS	Denell	Scr^{d8}	null allele	normal
***Scr*^{2}**	X ray	Kaufman	Scr^{k6}	null allele	normal
***Scr*^{3}**	EMS	R. Lewis	Scr^{r18}	hypomorphic allele	normal

allele	origin	discoverer	synonym	type	cytology
Scr^{4}	EMS	Wakimoto	*Scr*^{w17}	null allele	normal
Scr^{5}	EMS	Wakimoto	*Scr*^{w22}	hypomorphic allele	normal
Scr^{6}	EMS	Fornili	*Scr*^{f2cs}	cold-sensitive hypomorphic allele	normal
Scr^{7}	EMS	Fornili	*Scr*^{f71}	hypomorphic allele	normal
Scr^{8}	EMS	Fornili	*Scr*^{f76cs}	cold-sensitive hypomorphic allele	normal
Scr^{9}	X ray	Abbott	*Scr*^{a68}	null allele	*In(3LR)77D; 84B1-2*
Scr^{10}	X ray	Abbott	*Scr*^{a72}	null allele	*In(3LR)75B; 84B1-2*
Scr^{11}	EMS	Lambert	*Scr*^{c12}	null allele	normal
Scr^{12}	EMS	Stephenson	*Scr*^{e40}	null allele	normal
Scr^{13}	EMS	Matthews	*Scr*^{km0}	null allele	normal
Scr^{14}	EMS	Matthews	*Scr*^{km7}	hypomorphic allele	normal
Scr^{15}	EMS	Matthews	*Scr*^{km12}	hypomorphic allele	normal
Scr^{16}	EMS	Matthews	*Scr*^{km15}	null allele	normal
Scr^{17}	X ray	Pultz	*Scr*^{p18}	null allele	normal
Scr^{18}	X ray	Merrill	*Scr*^{VD30}	null allele	*In(3R)84B1-2; 95F*
Scr^{19}	X ray	Jürgens	*Scr*^{XF5}	null allele	*T(2;3)?*
Scr^{20}	X ray	Jürgens	*Scr*^{XT145}	null allele	*T(2;3)?*
Scr^{Msc}	spont	Tokunaga	*Msc*	Dominant allele	*In(3R)84B1-2; 84F1-2*
Scr^{W}	EMS	Wakimoto	*Scr*^{w15}	Dominant allele	50 kb inversion in 84B1-2
Scr^{Wrv1}	X ray	Hazelrigg		Scr^{W} revertant	*T(2;3)58F1-2; 84B1-2*
Scr^{Wrv3}	X ray	Hazelrigg		Scr^{W} revertant	normal
Scr^{Wrv5}	X ray	Hazelrigg		Scr^{W} revertant	*In(3R)81;84B1-2*
Scr^{Wrv6}	X ray	Hazelrigg		Scr^{W} revertant	*T(2;3)22D; 63A1-2+ T(2;3)54A1; 80-81*
Scr^{T1}	X ray	Tiong	*Scr*^{T1}	Dominant allele	*Tp(3;3)80-81; 84B1-2;84D5-6*
Scr^{T2}	X ray	Tiong	*Scr*^{T2}	Dominant allele	*T(2;3)40-41; 84B1-2*
Scr^{T3}	X ray	Tiong	*Scr*^{T3}	Dominant allele	*T(2;3)25D;40; 84B1-2+ T(2:3)29B; 91E*
Scr^{P}	X ray	Pultz	*Scr*^{P1}	Dominant allele	*T(3;4)80-81; 84B1-2;102F*
Scr^{S}	DEB	Seeger	*Scr*^{msl}	Dominant allele	

cytology: Placed in 84B1-2 based on its inclusion in *Df(3R)Scr,* and the common 84B1-2 breakpoints of eleven *Scr* mutations.

molecular biology: The *Scr* transcription unit has been identified by the localization of twelve *Scr*-associated breakpoints and the overlap junction of six deletions. Three of these approach *Scr* from its distal limit [*Df(3R)Antp7, Df(3R)A41,* and *Df(3R)Hu*]; the remainder delete the proximal end of the gene [*Df(3R)Dfd13*]. The identified transcription unit spans 25 kb of genomic DNA and is made up of three exons. Proceeding from 5′ to 3′ they are 0.5, 1.0, and 2.5 kb in length. The two introns are 6.0 and 15 kb respectively. The 3′ end of *Scr* is 20 kb distal to the 3′ end of *Dfd,* and the 5′ end of *Scr* is 18 kb proximal to the 5′ end of *ftz* and 50 kb proximal to the 3′ end of *Antp.* Breakpoint associated mutations in this latter 50-kb interval all affect *Scr* function indicating that this region is important for the normal expression of the transcription unit. The sum of the three identified exons is in close agreement with the 3.9-kb mRNA detected on Northern blots. There is a single large open reading frame, which initiates in exon 2 just downstream of the splice acceptor, and terminates in exon 3 about 300 nucleotides downstream of the splice acceptor. Thus the 3′ tail is just over 2 kb in length. The total open reading frame is 1,245 nucleotides in length and encodes a protein of 413 amino acids with a predicted molecular weight of 45 kd. The homeobox motif is encoded in exon 3 and opa-like repeats are found in exon 2.

z2: zen-2

location: 3-47.5 (inferred from close proximity to *zen*).

synonym: *zpr: zen pattern related.*

references: Rushlow, Doyle, Hoey, and Levine, 1987, Genes Dev. 1: 1268-79.

Pultz, Diederich, Cribbs, and Kaufman, 1988, Genes Dev. 2: 901-20.

phenotype: Simultaneous deletion of both *z2* and *pb* produces only a *pb* mutant phenotype. Thus absence of *z2* function has no discernible effect on development or morphology.

cytology: Placed in 84A4-5 based on its inclusion in *Df(3R)Scr* and exclusion from *Df(3R)LIN.*

molecular biology: The *z2* transcription unit maps 10 kb proximal to *zen* and about 1 kb distal to the transcription initiation site of *pb.* An open reading frame starts 67 bp downstream of the transcription start site in exon 1 and extends to 114 bp upstream of a consensus poly(A) addition site. The transcript produced is 1.0 kb in length and shows the same spatio-temporal expression pattern as the neighboring *zen* gene. Like *zen* conceptual translation of the *z2* open reading frame reveals the presence of a homeobox domain (encoded in exon 2). This sequence shows good similarity to the *zen* homeobox (75%), but there is little other sequence similarity found in the remainder of the proteins. The function of this locus is not known, but in light of the fact that its deletion causes no detectable effect, and *zen* mutants can be rescued by a genomic fragment which does not contain *z2*, it is likely the locus represents a pseudogene. Consistent with this conclusion is the finding that a *z2* homologue is not found in the *ANTC* of *D. pseudoobscura.*

zen: zerknüllt

Location: 3-47.5 (between *pb* and *bcd* in the *ANTC*).

references: Wakimoto, Turner, and Kaufman, 1984, Dev. Biol. 102: 147-72.

Rushlow, Doyle, Hoey, and Levine, 1987, Genes and Dev. 1: 1268-79.

Rushlow, Frasch, Doyle, and Levine, 1987, Nature 330: 583-86.

Doyle, Harding, Hoey, and Levine, 1986, Nature 323: 76-9.

phenotype: Null mutations result in embryonic lethality and the loss of several dorsally derived embryonic structures, including the amnioserosa, optic lobe, and dorsal ridge. These animals also fail to fully extend their germ bands and go through the process of head involution. The name for the locus derives from the characteristic "wrinkled" appearance of the germ band seen in the SEM at the time of normal germ-band retraction. Hypomorphic mutations result in the absence of dorsal structures but do undergo normal gastrulation movements. A temperature-sensitive allele has been used to define the time of zen^{+} action between 2 and 4 hours of embryogenesis, just prior to and overlapping the earliest observable morphogenic defects. X-ray induced somatic clones have further shown that zen^{+} function is unnecessary for postembryonic development. The RNA product of *zen* is first detected at about 2 hours of development during the

eleventh to twelfth cell cycle of the syncytial blastoderm. At this early stage the RNA is found on the dorsal surface of the embryo extending around the anterior and posterior poles. As cellularization proceeds and the early events of gastrulation begin, the RNA becomes restricted to a mid-dorsal stripe of cells. These cells have been fate mapped and give rise to the amnioserosa and the lobes in the dorsal posterior of the embryonic head, *i.e.*, the structures absent in *zen*$^{-}$ animals. The time of appearance of *zen* RNA also correlates nicely with the temperature-sensitive-period data obtained using the conditional allele. Antisera to the *zen* protein product has been used to follow its accumulation pattern, and this analysis agrees with and expands the *in situ* results. The protein is located in the nuclei of cells expressing the gene and at cellular blastoderm is found in a mid-dorsal stripe seven cells wide and seventy cells in length. During gastrulation these cells eventually give rise to the amnioserosa, the optic lobe, and dorsal ridge; these structures continue to show *zen* protein accumulation until the end of germband extension at about 4 to 6 hours of development. This end point also correlates well with the end of the temperature-sensitive period of the conditional allele. The spatial pattern of *zen* expression has been shown to be dependent on the products of several of the maternally expressed genes which specify the anterior-posterior and dorsal-ventral polarity of the embryo, and *zen* would appear to lie near the end of the axis-determining pathway.

alleles: Seven ethyl-methanesulfonate-induced alleles, all of which have normal cytology.

allele	discoverer	synonym	comments
***zen*^{1}**	Fornili	*zen*^{f16}	temperature-sensitive hypomorphic allele
***zen*^{2}**	Fornili	*zen*^{f27}	null allele
***zen*^{3}**	Fornili	*zen*^{f55}	null allele
***zen*^{4}**	Fornili	*zen*^{f62}	null allele
***zen*^{5}**	Fornili	*zen*^{f75}	hypomorphic allele
***zen*^{6}**	Merrill	*zen*^{v1}	null allele
***zen*^{7}**	Wakimoto	*zen*^{w36}	null allele

cytology: Placed in 84B1-2 based on its inclusion in *Df(3R)Scr* and *Df(3R)SCB-XL2*.

molecular biology: One of a pair of regions between *pb* and *bcd* in the *ANTC* has been shown to be *zen* by *P*-element mediated transformation and rescue of a *zen*$^{-}$ genotype. The rescuing fragment is 4.5 kb in length and carries a single 1.3 kb transcription unit. It is composed of two exons separated by a 64-base pair intron. The start of translation is 52 base pairs downstream of the transcription start site in the first exon. The open reading frame ends 169 base pairs upstream of a poly(A) addition site in exon 2. The predicted size of mature message from genomic and cDNA sequence analysis is 1.3 kb which is in agreement with the transcript size observed on Northern blots. Conceptual translation of the open reading frame shows the presence of a homeodomain and PEST sequences, which are enriched for the amino acids serine, threonine, proline, and glutamic acid. The presence of both of these motifs correlates well with the DNA binding activity of *zen* protein and the dynamic pattern of protein accumulation seen for *zen* protein *in vivo*.

antenna: see *ems*

Antennapedex: see ***Apx***

Antennapedia: see ***Antp*** under ***ANTC***

anterior open: see ***aop***

anterobithorax: see ***abx*** under ***BXC***

Antp: see ***ANTC***

ants: antennas

location: 3-49.4.
origin: Spontaneous.
references: Ribó, 1968, DIS 43: 59.
phenotype: Antennae modified—lengthened or reduced—especially in males. Viability good.

aop: anterior open

location: 2-12 (approximate).
references: Nüsslein-Volhard, Wieschaus, and Kluding, Wilhelm Roux's Arch. Dev. Biol. 193: 267-82. (fig.).
Tearle and Nüsslein-Volhard, 1987, DIS 66: 209-26.
phenotype: Embryonic lethal. Homozygous embryos have anterior dorsal hole in epidermis. Brain and sometimes gut extrude through hole. Head involution normal. Visible during dorsal closure.
alleles: Six ethyl-methanesulfonate induced alleles; *aop*^{1} and *aop*^{2} (recovered as *IP* and *IIS*) retained.

aor: abdominmal one reduced

location: 3-{85}.
references: González, Molina, Casal, and Ripoll, 1989, Genetics 123: 371-77.
phenotype: Hemizygotes lack the first abdominal segment. Histoblasts of third-instar larvae normally present in A1.
cytology: Placed in 96A1-7 based on its association with *In(3R)Ubx*^{7L} = *In(3R)89E;96A1-7* and its inclusion in *Df(3R)L16* = *Df(3R)96A1-10;96E*.

**ap: apterous* (T.G. Wilson)

location: 2-55.2.
references: Metz, 1914, Am. Naturalist 48: 675-92.
Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. No. 278: 236 (fig.).
Stevens and Bryant, 1985, Genetics 110: 281-97.
1986, Genetics 112: 217-28.
phenotype: Wings and halteres reduced to traces. Bristles eliminated from area around wing base (including posterior notopleurals, anterior and posterior supra-alars, and anterior postalars); posterior scutellars erect when present but missing in first counts; dorsocentrals smaller and fewer; hairs on thorax sparse and irregular. Sutural furrow reduced; thorax disproportionately small. Flies small, pale, weak, and very short lived. Viability about 70% that of wild type but erratic. Both sexes sterile. RK2.
alleles: Apterous alleles generally fall into three groups based on phenotypic differences. Most of the characterized apterous alleles belong to the first group and have basically the *ap* or *ap*^{4} phenotype. Some alleles (*ap*^{blt2} and *ap*^{T60}) have a less severe wing phenotype, being straplike. Alleles also vary in their expressivity of the precocious adult death and nonvitellogenic ovary phenotypic characters; some alleles result in a low number of escapers, similar to *ap*^{4}, while others have an escaper percentage of as much as 50%. There is little correlation between expressivity of the wing deficiency phenotype

and either precocious adult death or nonvitellogenic ovary development, but a good correlation exists between expressivity of the latter phenotypic characters (Wilson, 1980). Generally, heterozygous combinations of these alleles do not show complementation for any phenotypic characters. Another group, represented by *ap* ^{blt}, exhibits a less severe, somewhat different phenotype; attributable to localized lysosomal cell death in the presumptive wing blade. (Sedlock, Mango, and Stevend, 1984, Dev. Biol. 104: 489-96). A third group includes two dominant alleles. The apterous locus appears to be a complex locus, containing several partially complementing groups for the wing deficiency and adult-death/female-sterility phenotypic characteristics. However, by studying the effects of a number of different temperature regimens on phenotypic expression of three different temperature-sensitive alleles, Stevens and Bryant (1986) conclude that all phenotypes are explicable in terms of changes in quantity rather than quality of gene product.

allele	origin	discoverer	synonym	ref [α]	phenotype [β]
*ap ^{1}	spont	E.M. Wallace, 13h		4, 8, 14	
*ap ^{2}	spont	Bridges, 16j20	*ap-c*	3, 4, 8	*ap* ^{4}
ap ^{2a}	EMS	Wilson, 1978		20	
*ap ^{3}	spont	Morgan, 23a	*ap-c*, no wings	8, 17	*ap* ^{4}
ap ^{3a}	EMS	Wilson, 1978		20	*ap* ^{56f}
ap ^{4}		Medvedev, 32a15	*ap-d*	8, 13, 20	γ
ap ^{4a}	EMS	Wilson, 1978		20	
ap ^{5}	U.V.	Byers, 49f		8, 16	*ap* ^{4}
ap ^{6}	spont	Faulhaber		6, 8, 10	*ap* ^{4}
ap ^{13}	EMS	Wilson		20	
ap ^{18}	EMS	Wilson, 1978		20	
ap ^{25}	EMS	Wilson, 1978		20	
ap ^{32}	EMS	Wilson, 1978		20	
ap ^{40}	EMS	Wilson, 1978		20	*ap* ^{56f}
ap ^{46}	EMS	Wilson, 1978		20	
ap ^{49}	EMS	Wilson, 1978		20, 21	
ap ^{49j}	spont	Ritterhoff, 49j		8, 11, 20	*ap* ^{4}
ap ^{54}	EMS	Wilson, 1978		8, 20, 21	*ap* ^{78j}
ap ^{56f}	spont	Thompson, 56f		5, 8, 20	γ
ap ^{57}	EMS	Wilson, 1978		20	
ap ^{58}	EMS	Wilson, 1978		20	
ap ^{67e}	spont	Lee		12	
ap ^{69c1}	EMS	Au		7	
ap ^{69c2}	EMS	Gottschalk		7	
ap ^{69c3}	EMS	Nadler		7	
ap ^{73n}	spont	Altorfer, 73n		1	*ap* ^{4}
ap ^{77f}	EMS	Wilson, 77f		20, 23	γ
ap ^{78e}	EMS	Wilson, 78e			
ap ^{78j}	EMS	Wilson, 78j		20, 21, 24	γ
ap ^{blt}	spont	Groscurth, 31b1	*blt*	8	γ
ap ^{blt*}				20	
ap ^{blt2}	spont	Whittinghill, 44h		8, 22	< *ap* ^{4}
ap ^{blt3}	spont	Semenza, 49k	*blt* ^{S49k}	2, 8	*ap* ^{4}
ap ^{c}				20	
ap ^{e}	spont			18	γ
ap ^{ID}			*ap* ^{id}	20	*ap* ^{Xa} ?
ap ^{T60}	X ray	Thomas, 60g		8, 15	< *ap* ^{4}
ap ^{trw}	spont		*trw*	9	γ
ap ^{Xa}	X ray	Serebrovsky, 28a	*Xa*	8, 19, 20	γ

α *1* = Altorfer, 1977, DIS 52: 2; *2* = Barigozzi, 1950, DIS 24: 54; *3* = Bridges, 1919, J. Exp. Zool. 28: 370. *4* = Bridges and Morgan, 1919, Carnegie Inst. Washington Publ. 278: 236 (fig.); *5* = Burdick, 1956, DIS 30: 69; *6* = Butterworth and King, 1965, Genetics 52: 1153-74; *7* = Butterworth, Nolph, Au, Gottschalk, Nadler, and Tuma, 1970, DIS 45: 36; *8* = CP627; *9* = Crist, Fontaine, and Merrell, 1980, DIS 55: 204; *10* = Faulhaber, 1963, DIS 37: 48; *11* = Glass, 1951, DIS 25: 76-77; *12* = Lee, 1972, DIS 48: 18; *13* = Medvedev and Bridges, 1935, Tr. Inst. Genet. Akad. Nauk. SSR 10: 119-209; *14* = Metz, 1914, Am. Nat. 48: 675-92 (fig.); *15* = Meyer, 1963, DIS 37: 50; *16* = Meyer, Edmondson, Byers, and Erickson, 1950, DIS 24: 59; *17* = Morgan, 1929, Carnegie Inst. Washington Publ. 399: 183; *18* = Roberts and Bownes, 1982, DIS 58: 209; *19* = Serebrovsky and Dubinin, 1930, J. Hered. 21: 259-65; *20* = Stevens and Bryant, 1985, Genetics 110: 281-97; *21* = Stevens and Bryant, 1986, Genetics 112: 217-28; *22* = Whittinghill, 1947, DIS 21: 71; *23* = Wilson, 1980, Dev. Genet. 1: 195-204; *24* = Wilson, 1981, Dev. Biol. 85: 425-33.

β Designation of allele with similar phenotype.

γ Phenotypes described below in separate entries.

cytology: Placed in salivary region 41B-C (Schultz).

ap ^{4}

phenotype: Wings less than 10% normal length, lacking all wing blade structures. Halteres reduced to structureless remnants less than 25% normal size. Scutellar and dorsocentral bristles sometimes missing (Butterworth and King, 1965, Genetics 52: 1153-74). Wing phenotype disc autonomous in *ap* ^{4}/*ap* $^{+}$ mosaic flies, although small patches of *ap* ^{4} wing structures are found in *ap* ^{4}/*ap* $^{+}$ mosaic wings. Haltere phenotype disc autonomous (Wilson, 1981, Dev. Biol. 85: 434-45). Adults become paralyzed about 30 hr following eclosion and die soon thereafter. Around 1% of adults are long-lived "escapers" of this phenotype (Wilson, 1980, Dev. Genet. 1: 195-204). Precocious adult-death phenotype fate-maps to proximity of Malpighian tubules, and tubule malfunctioning postulated to result in this phenotype (Wilson, 1981). Foregut of females swollen owing to accumulation of peritrophic membrane (King and Sang, 1958, DIS 32: 133). Female sterile with underdeveloped ovaries; nurse cell nuclei become pycnotic after stage 7, and stage-8 oocytes are the most advanced (King and Burnett, 1957, Growth 21: 263-80; Wilson, 1980). *ap* ^{4} ovaries develop nonautonomously when transplanted to a wild-type host (King and Bodenstein, 1965, Z. Naturforsch. 20B: 292-97). Application of juvenile hormone mimic, ZR-515, to newly eclosed *ap* ^{4} females results in vitellogenic oocytes [Postlethwait and Weiser, 1973, Nature (London) New Biol. 244: 284-85]. Membranes of vitellogenic oocytes lack microvilli and pinocytoxic vesicles normally present; development of these structures stimulated by administration of ZR-515 (Tedesco, Courtwright, and Kumaran, 1981, J. Insect. Physiol. 27: 895-902). Corpora allata from adult *ap* ^{4} are juvenile-hormone deficient when bioassayed [Postlethwait, Handler, and Gray, 1975, The Juvenile Hormones (L.I. Gilbert, ed.). pp. 449-69]. Nonvitellogenic oocyte phenotype fate-maps to same or similar location as precocious adult death phenotype (Wilson, 1981). Escaper females develop stage-14 oocytes (King and Sang, 1958) and are fertile (Wilson, 1980). Males show immature sexual behavior and are sterile, but testes appear normal with motile sperm (King and Sang, 1958). Larval fat body histolysis delayed; this phenotype is nonautonomous as determined by transplantation experiments (Butterworth, 1972, Dev. Biol. 28: 311-25). Application of ZR-515 accelerates larval fat body histolysis in *ap* ^{4} adults (Postlethwait and Jones, 1978, J. Expt.

Zool. 203: 207-14). Ovarian acid phosphatase level low in *ap*^{4} females and is restored after application of ZR-515 (Postlethwait *et al.*, 1975). *ap*^{4} ovaries cultured *in vitro* are capable of yolk protein synthesis (Redfern and Bownes, 1982, Mol. Gen. Genet. 195: 181-83). *ap*^{4}/*Df(2L)M41A-54* hemizygote has nearly normal complement of bristles but otherwise resembles *ap*^{4} homozygote (Butterworth and King, 1965).

ap^{56f}

phenotype: Wing and haltere phenotype like *ap*^{4}. Scutellar and dorsocentral bristles missing (Butterworth and King, 1965, Genetics 52: 1153-74). Rear and middle legs occasionally twisted, more frequently in female than in male. Both sexes fertile and long lived when homozygous and in combination with other *ap* alleles. *ap*^{56f}/*M(2)S2*^{4} have normal complement of dorsocentral and scutellar bristles (Butterworth and King, 1965).

ap^{77f}

phenotype: Weakest non-temperature-sensitive allele known. Wing has reasonably good wing blade development, with missing triple-row elements and posterior wing margin. Haltere less well developed but more so than *ap*^{4}. Adults long lived and fertile. Less dominant in heteroallelic combination with *ap*^{4}-like alleles than is *ap*^{56f}. *ap*^{77f}/*Df(2R)M41A4* has more severe phenotype than *ap*^{77f} homozygotes.

ap^{78j}

phenotype: A temperature-sensitive allele of apterous. When raised at 22°, wing and haltere phenotype approaches wild type except for missing patches of triple-row bristles and posterior wing margin. When raised at higher temperatures, phenotype becomes more severe and resembles *ap*^{4} at 29°. Two nonoverlapping temperature-sensitive periods in development, one in late-second to middle-third instar for wing and haltere deficiency phenotype and the other during the first day of pupal development for precocious adult death and nonvitellogenesis phenotype. Wing discs of heat-pulsed larvae failed to exhibit cell death by trypan blue exclusion.

ap^{blt}: *apterous-blot*

phenotype: Wings blistered, sometimes inflated and dark due to trapped hemolymph. Mirror-image duplication of posterior wing blade structures occurs [Waddington, 1939, Proc. Nat. Acad. Sci. USA 25: 299-307; Whittle, 1979, J. Embryol. Exp. Morphol. 53: 292-303 (fig.)]. Wing venation may be disrupted. Portions of posterior wing compartment may be transformed into anterior compartment structures, an effect like that of engrailed (*en;* 2-62.0). Despite relatively mild adult phenotype, extensive cell death observed, localized to wing pouch of imaginal discs; associated with acid phosphatase and lysosomal activity (Sedlak, Manzo, and Stevens, 1984, Dev. Biol. 104: 489-96). Clonal analysis revealed nonautonomous expression of phenotype. Heterozygotes with *ap*^{4} or *ap*^{56f} and hemizygotes show blistering phenotype only (Whittle). *ap*^{blt}/*ap*^{73n} shows transformation phenotype, and aldehyde oxidase histochemical staining of these wing discs is consistent with transformation (Whittle and Sprey, 1982, Wilhelm Roux's Arch. Dev. Biol. 191: 285-88). Much overlapping with wild type, and expressivity variable. Adults long lived and fertile.

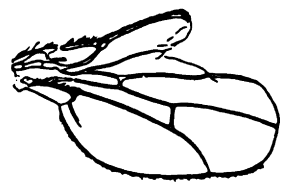
ap^{e}

phenotype: Homozygotes display extreme wing reduction, particularly of the posterior wing compartment. Approximately 50% of the flies have duplications of the anterior wing margin, distal costa, and triple row bristles. In wings with large amounts of wing blade, very little venation is present; however, these may often have triplications or even four copies of the anterior wing margin, some located in the posterior part of the wing. Dried hemolymph sometimes trapped between the dorsal and ventral wing surfaces giving the wing a puffy blackened appearance. This mutant therefore has duplications and deficiencies characteristic of cell death followed by regulation in the wing, but also has transformations of the posterior wing compartment to the anterior wing compartment. 8% of the flies have defective third legs, more frequently in females than in males. Halteres and scutellar bristles appear to be normal. Homozygotes viable and fertile.

ap^{trw}: *apterous-torn wing*

phenotype: Distal part of wing in homozygotes shows sawtooth pattern as if tip torn away. Expression uniform in males and females. Viability and fertility good.

other information: Genetic location and phenotype suggests allelism with apterous, but not tested with viable *ap* alleles.



ap^{Xa}**: apterous-Xasta**

From Bridges and Brehme, 1944, Carnegie Inst. Washington Publ. No. 552: 228.

ap^{Xa}: *apterous-Xasta*

phenotype: Wings reduced in length to about 70% normal; irregular in outline with a V-shaped incision with apex at L2, uniformly present giving wing a mitten-like shape with the thumb between marginal vein and L2. Excellent dominant with no overlap. Fertile and fully viable in heterozygote. Usually lethal in homozygous conditions, but occasionally ecloses very late as pale dwarf with wings and balancers like *vg*. Deep notch visible in tip of wing fold in prepupa (Waddington, 1939, Proc. Nat. Acad. Sci. USA 25: 299-307). In homozygotes and in combination with *ap*^{4}, *ap*^{6}, or *Df(2R)M41A4*, wings are straplike and 30-70% normal length, and haltere length is 25-50% normal; longevity and fertility like *ap*^{4}/*ap*^{4} except for an occasional long-lived *ap*^{Xa}/*Df(2R)M41A4* female that may be fertile [Butterworth and King, 1965, Genetics 52: 1153-74 (fig.)]. In heterozygous combination with *ap*^{ID}, duplications of the notum occur frequently. Wing disc cell death found in both *ap*^{Xa}/+ (Fristrom, 1969, Mol. Gen. Genet. 103: 363-79) and *ap*^{Xa}/*ap*^{ID} [Postlethwait, 1978, Genetics and Biology of Drosophila (Ashburner and Wright, eds.). Academic Press, London, New York, San Franciso, Vol. 2C,

pp. 418-19 (fig.)].

cytology: Shown by Sturtevant (1934, DIS 2: 19) to be associated with *T(2;3)ap* ^{Xa} = *T(2;3)41F;89E8-F1* which is superimposed on *In(2R)Cy* and *In(3R)P* (Morgan, Bridges, and Schultz, 1936, Year Book - Carnegie Inst. Washington 35: 294; Lewis, 1951, DIS 25: 109).

ap-c: see *ap* ^{2}

ap-c: see *ap* ^{3}

ap-d: see *ap* ^{4}

apang: see ***apg***

Apart: see ***Apt***

**apb: apterblister*

location: 2-44.7.

origin: Ultraviolet induced.

discoverer: Edmondson, 49k.

references: Meyer, Edmondson, Byers, and Erickson, 1950, DIS 24: 59-60.

phenotype: Wings always notched, nearly always spread, and usually blistered but expression somewhat variable. Homozygous imagos live less than 24 hr, owing to intestinal obstructions. Abdomens characteristically turn dark grey before death because of accumulation of digested food products. Although not at same locus as *ap, apb* +/+ *ap* ^{4}, flies show slight notching of wings and many die within a day; those that survive are fertile. *ap* ^{5} gives a similar heterozygous effect. RK2.

Ape: Apurinic endonuclease

location: 3-{47}.

synonym: *AP3*.

references: Kelly, Venugopal, Harless, and Deutsch, 1989, Mol. Cell Biol. 9: 965-73.

phenotype: Encodes an apurinic-apyrimidinic DNA endonuclease, AP3. Biochemical studies by Spiering and Deutsch (1986, J. Biol. Chem. 261: 3222-28).

cytology: Placed in 79C-D by *in situ* hybridization.

molecular biology: Isolated from a cDNA expression library using antiserum directed against human enzyme known to cross react with the Drosophila enzyme (Spiering and Deutsch). The conceptual translation product predicts a 317-amino-acid polypeptide of molecular weight 34.2 kd. Region between nucleotides 30 and 173 shows 66% homology with *recA* of *E. coli* and 42% amino-acid identity. Two helix-turn-helix domains detected in the carboxy-terminal end of the polypeptide. Northern blots identify a 1.3-kb transcript at all stages of development, but somewhat reduced in pupae and adult males; there is also transiently present, a 3.5-kb transcript in four-to-eight-hour embryos that disappears after second larval instar.

apexless: see ***apx***

aperA: abnormal proboscis extension reflex A (J.C. Hall)

location: 1-22.1.

origin: Induced by ethyl methanesulfonate.

discoverer: Kimura.

references: Kimura, Shimozawa and Tanimura, 1986, J. Exp. Zool. 239: 393-99.

phenotype: Variable phenotypic defects in the sugar-induced proboscis extension reflex (PER): some *aperA* flies cannot extend their probosces at all, whereas they are able to open the labellar lobes; some individuals extend their probosces only to the right or the left side of the body; each mutant individual seems to have a fixed phenotype, *e.g.*, a fly which shows one-sided PER always extends its proboscis to the same side; the array of aberrant phenotypes is different under the influence of the two mutant alleles: for *aperA* ^{1}, 47.0% were unable to extend their probosces, 16% only to the left side, and the remainder extended their probosces normally; for *aperA* ^{2}: 32% could not extend their probosces, 23% could extend them only to the right side, and 22% only to the left side (the remainder behaved normally).

alleles: Two alleles; *aperA* ^{1} *(=TT1), aperA* ^{2} *(=TT360)*, with the overall penetrance for the former ca. 79%; 77% for the latter.

aperB (J.C. Hall)

location: 1-0.6.

origin: Induced by ethyl methanesulfonate.

discoverer: Kimura.

references: Kimura, Shimozawa and Tanimura, 1986, J. Exp. Zool. 239: 393-99.

phenotype: Given sugar stimuli *aperB* flies extended their probosces, not straight forward (as does wild-type), but backward; when these mutants show a partial extension of their probosces, the direction of the extensions is normal; the expression of the *aperB* gene is sensitive to culture temperature: when the *aperB* ^{1} mutants were reared at low temperature (18° or 20°C), over 90% of the flies were normal, whereas the high culture temperature (over 25°C) caused an abnormal PER; the temperature at which the proboscis extension reflex was tested did not affect the phenotype.

cytology: Maps to 2D3-F3; based on its inclusion in *Df(1)Pgd* = *Df(1)2D3;2F5* but not *Df(1)JC19* = *Df(1)2F3;3C5; w* $^{+}$*Y* = *Dp(1;Y)2D2;3D2-3* covers *aperB* ^{1}.

alleles: Two alleles: *aperB* ^{1} *(=TT665)* and *aperB* ^{2} *(=TF48)*, which lead to indistinguishable phenotypic defects.

other information: *aperB* mutations are completely recessive, and complement the closely linked *aperC* mutation.

aperC (J.C. Hall)

location: 1-0.4.

origin: Induced by ethyl methanesulfonate.

discoverer: Kimura.

references: Kimura, Shimozawa and Tanimura, 1986, J. Exp. Zool. 239: 393-99.
1986, Devel. Biol. 117: 194-203.
1987, J. Neurogenet. 4: 21-28.

phenotype: Sugar-induced proboscis extension nearly absent (*i.e.* no extension at all of rostrum and haustellum), but not until adults are three to six days old; this defect, which is completely recessive, wanes such that at least half of the adults behave normally again by approximately day 10-11; correlated with these behavioral changes is time-dependent degeneration and regeneration of a pair of muscles, the rostral protractors; behavioral and histological phenotypes are temperature-sensitive: 18° causes defects later in adult life, and yet there is no recovery; 29° causes lower than usual (*i.e.* 25°) proportion of adults developing the defects, and high temperature is compatible with recovery; temperature-sensitive

period is from two to four days post-eclosion.

cytology: Maps to 1F5-2A, based on its inclusion in *Df(1)A94* = *Df(1)1F5;2B15* and *Df(1)S39* = *Df(1)1E4;2B11-20* plus the fact that the *X*-chromosome duplication from the distal tip to *2A*, from *T(1;Y)G20*, covers *aperC*.

other information: *aperC* completely recessive and complements the closely linked *aperB* mutations.

apg: apang

location: 2-7.7.

origin: Induced by ethyl methanesulfonate.

references: Shakaron and Sharma, 1983, DIS 59: 110 (fig.).

phenotype: Homozygotes when raised at 19° show occasional absence of one or both claws; veins L4 and L5 interrupted; fertile at 19° but become sterile when shifted to 28°; produce embryos with range of germ band abnormalities. Homozygous pupal lethal when raised at 28°; pharate adults show defective tarsal development of all six legs; condensed, poorly developed and curved metatarsus and tarsi; duplications in tibial and tarsal segments; claws absent. Temperature sensitive period first instar to early pupa.

Aph-1: Alkaline phosphatase-1

location: 3-47.3 (between *W* and *p*) (Wallis and Fox).

references: Beckman and Johnson, 1964, Nature 201: 321 (fig.).
1964, Genetics 49: 829-35 (fig.).
Wallis and Fox, 1969, Biochem. Genet. 2: 141-58.

phenotype: Locus responsible for one of several different alkaline phosphatase species [APH1 (EC 3.1.3.1)] formed during the life cycle. Specifies the enzyme that becomes active in the larval cuticle and muscle during the third instar. Electrophoretic mobility of a pupal form of the enzyme, which differs from that found in the larva, also appears to be controlled by this locus (Wallis and Fox). Dimeric nature of enzyme inferred from the presence of enzymes of hybrid mobility in larvae heterozygous for electrophoretic variants. Biochemical characterization of larval enzyme by Harper and Armstrong (1972, Biochem. Genet. 6: 75-82; 1973, Biochem. Genet. 10: 29-38; 1974, Biochem. Genet. 11: 177-80).

alleles: Naturally occurring alleles superscripted *F* and *S* reported by Beckman. Wallis and Fox describe Aph^{A} which specifies larval enzyme migrating faster than Aph^{F}, but a pupal enzyme with same characteristics as that produced by Aph^{F}. Aph^{0} reported by Johnson (1966, Science 152: 361-62) produces no detectable enzyme activity but causes the appearance in extracts of Aph^{S}/Aph^{0} larvae of a band migrating slightly faster than the hybrid band produced by Aph^{F}/Aph^{S} larvae. Naturally occurring alleles superscripted *2, 4, 6,* and *10* characterized by Harper and Armstrong (1972, 1973, 1974); *4* is synonymous with *F* as is *6* with *S; 2* migrates more slowly than *S* and *10* more rapidly than *F;* not clear that *10* and *A* are different. That the larval and pupal enzymes are differently modified products of the same locus is indicated by genetic inseparability and by concordance in the orders of mobilities of electrophoretic alleles (Wallis and Fox).

Aph-2

location: 2- not mapped.

references: Schneiderman, Young, and Childs, 1966, Science 151: 461-63.

phenotype: The alkaline phosphatase found in adult hindgut.

alleles: Two different alleles recorded superscripted *A* and *B*. Enzyme produced by $Aph\text{-}2^{A}$ homozygotes migrates more rapidly than that produced by $Aph\text{-}2^{B}$ homozygotes; enzyme produced by $Aph\text{-}2^{A}/Aph\text{-}2^{B}$ has same mobility as that produced by $Aph\text{-}2^{A}$ homozygotes.

apo: altered pattern orientation (J.C. Hall)

location: 1- (not localized).

origin: Induced by ethyl methanesulfonate.

synonym: apo^{S129}.

references: Heisenberg, 1979, Handbook of Sensory Physiology (H. Autrum, ed.). Springer-Verlag, Berlin, Vol. VII/6A, pp. 665-79.
Bülthoff, 1982, DIS 58: 31.
1982, Biol. Cybernet. 45: 63-70.

phenotype: Poor orientation to objects, including spots in Y-maze test; electroretinogram normal.

app: approximated

location: 3-37.5.

discoverer: Curry, 34a25.

references: 1935, DIS 3: 6.

phenotype: Crossveins close together; veins diverge at greater angle than wild type; effect visible in prepupal wing [Waddington, 1940, J. Genet. 41: 75-139 (fig.)]. Legs short with four-jointed tarsi; the penultimate joint characteristically swollen [Waddington, 1939, Growth Suppl. 37-44 (fig.)]. Joint between second and third tarsal segments often incomplete; invaginations or internalization of cuticle seen in tarsi 1, 3, and 4 (Held, Duarte, and Derakhshanian, 1986, Wilhelm Roux's Arch. Dev. Biol. 195: 145-57). Thickset body. Posterior scutellars farther apart than normal. Eyes smaller and flatter than normal, also bumpy. Spread wings; thickened veins. RK1.

alleles: app^{61e} (CP627).

cytology: Placed in 69A2-4 on the basis of its inclusion in *Df(3L)vin6* = *Df(3L)68C8-11;69A4-5* but not *Df(3L)vin5* = *Df(3L)68A3;69A1-2* (Akam, Roberts, Richards, and Ashburner, 1978, Cell 13: 215-26).

Appl: β-Amyloid protein precursor like (K. White; J.C. Hall)

Location: 1-{0}.

origin: Isolated as cDNA clones derived from cloned genomic DNA in the 1B division.

references: Rosen, Martin-Morris, Luo, and White, 1989, Proc. Nat. Acad. Sci. USA 86: 2478-82.
Martin-Morris, and White, 1990, Dev. (In press).

molecular biology: A 6.5 kb transcript corresponding to the cDNA clones encodes a polypeptide that is conceptually an 886 amino acid transmembrane protein; this predicted amino-acid sequence shows strong homology in certain of its regions to the β-amyloid protein precursor protein of humans (Rosen *et al.*, 1989). Two forms of the actual protein, which is N-glycosylated, are detectable (in studies involving extracts, primary cultures, and transfected cells); an 145 kd membrane-associated precursor and a 130 kd secreted form lacking the

cytoplasmic domain inferred from sequencing (Luo, Martin-Morris, and White, 1990, J. Neurosci., in press). The source of the *Appl* transcript spans *ca.* 38 kb of genomic DNA; this RNA localizes to post-mitotic neurons (and apparently not to non-neuronal tissues) in all developmental stages and in adults (Martin-Morris and White, 1990). Consistent with these *in situ* hybridization data are those showing APPL protein immunoreactivity in developing neurons, concomitant with axonogenesis; this staining remains associated with differentiated neuronal cell bodies and axonal tracts (including neuropil regions) in embryos, APPL immunoreactivity is observed exclusively in post-mitotic CNS and PNS neurons (Luo *et al.*, 1990).

other information: The APPL-encoding gene initially suggested (Rosen *et al.*, 1989) to correspond to *vnd* (which was defined originally by embryonic neural-lethal mutations). This has been disproved, in that a terminal deletion *Df(1)78* which retains vnd^{+} function removes most of the *Appl* coding sequences (Martin-Morris and White).

apr: see w^{a}

Aprt: Adenine phosphoribosyltransferase

location: 3-1.49 (0.13 cM to the right of *R;* estimated by Johnson and Friedman to be 3.03 units from the tip of *3L*).

synonym: *aprt.*

references: Johnson and Friedman, 1981, Science 212: 1035-36.
1983, Proc. Nat. Acad. Sci. USA 80: 2990-94.

phenotype: Is the structural gene for adenine phosphoribosyltransferase [APRT, AMP: pyrophosphate phosphoribosyltransferase (EC 2.4.2.7)], a homodimer with 23,000 dalton subunits. It is a purine salvage enzyme which catalyzes the synthesis of AMP from 5-phosphoribosyl-1-pyrophosphate. Flies homozygous for a null allele $Aprt^{1}$ survive on 15 times the concentration of purine that wild type tolerates and show about 2% wild-type enzyme activity; $Aprt^{1}/+$ exhibit about half wild-type activity. $Aprt^{2}$ has 9% normal enzyme activity. The dosage response suggests that the mutant affects the structural gene for APRT.

alleles: Electrophoretic variants $Aprt^{A}$ (more acidic) and $Aprt^{B}$ (more basic) in wild-type stocks Oregon R and Canton S, respectively. $Aprt^{1}$ (Duck), $Aprt^{2}$ and $Aprt^{3}$ (Gelbart and Chovnick) induced by ethyl methanesulfonate; $Aprt^{4}$ and $Aprt^{5}$ selected on purine food by Johnson and Friedman (1983).

cytology: Placed in 62B8-12 based on its inclusion in the region of overlap of *Df(3L)R-G7* = *Df(3L)62B8-9;62F2-5* and *Df(3L)R-G2* = *Df(3L)62B2-4;62B11-12* (Sliter, Henrich, Tucker, and Gilbert, 1989, Genetics 123: 327-36). *in situ* hybridization identifies 62B9 as the site of *Aprt* (Johnson *et al.*).

molecular biology: Genomic clone isolated by chromosomally walking from sequences isolated and cloned by microdissection of region 62B from polytene chromosomes. Gene recognized by hybrid selection of an 1-kb mRNA that translated an APRT product. cDNA's have a common 5′ initiation site but two different 3′ polyadenylation sites. The primary transcript contains two introns, the first of which has alternative 5′ sites, which are spliced to the same 3′ site; one product encodes the functional enzyme and the other a prematurely terminated and presumably nonfunctional polypeptide (Johnson and Henikoff, 1989, Mol. Cell Biol. 9: 2220-23). Conceptual amino-acid sequence predicts a polypeptide of 194 amino acids and about 20 kd in molecular weight. Drosophila APRT amino-acid sequence displays approximately 40% identity and nearly 80% homology with all known APRT proteins (Johnson, Edström, Burnett, and Friedman, 1987, Gene 59: 77-86).

**Apt: Apart*

location: 3- (between *h* and *p*).

origin: X ray induced.

discoverer: Belgovsky, 34e23.

references: 1935, DIS 3: 27.

phenotype: Wings spread widely. Viability, fertility, and separability good. Homozygous lethal. RK2A.

cytology: Associated with *In(3L)Apt* — no salivary analysis.

other information: *Apt/D* survive; therefore not an allele of *D*.

apterblister: see *apb*

apterous: see *ap*

Apurinic endonuclease: see *Ape*

**apx: apexless*

location: 1-11.3.

origin: Induced by DL-*p*-N,N-di-(2-chloroethyl)amino-phenylalanine.

discoverer: Fahmy, 1954.

references: 1959, DIS 33: 83.

phenotype: Slightly larger fly with large eyes containing various numbers of deranged ommatidia. Wings broad and blunt; in many flies, margin removed to various degrees, from a small incision of inner margin to removal of entire inner margin, costal vein, and parts of the membrane as far as L3. Region from L3 to costal cell unaffected. Rarely L4 and 5 are interrupted. Males viable and fertile; female fertility reduced. RK3.

Apx: Antennapedex (R.E. Denell)

location: 1-70 (said to map 12 units to the right of *B*).

origin: Neutron induced.

references: Ginter, 1969, DIS 44: 50.

phenotype: Males and heterozygous females show variable expression from small additional segment on the third antennal segment to a nearly complete leg including femur, tibia, and tarsus. Arista usually present. Homozygous females lethal but *X0* males survive. Crosses involving either *Apx* males or females produce many inviable embryos.

cytology: Polytene *X* appears normal, but genetic results suggest a *T(1;3)* with breakpoints in the proximal part of *Xh* and at *Antp*.

Apx-2: see *Antp*

ar: abdomen rotatum

location: 4- (proximal to *bt;* Fung and Stern, 1951, Proc. Nat. Acad. Sci. USA 37: 403-4.

origin: Spontaneous.

discoverer: Beliajeff, 1926.

references: 1931, Biol. Zentralbl. 51: 701-8 (fig.).
Bridges, 1935, Biol. Zh. 4: 401-20.