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Drosophila Forked Locus

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A 40-kilobase-pair region of the *Drosophila* X chromosome from band 15F was cloned, and DNA insertions were indentified for the forked alleles f^1, f^3, f^{3n}, f^5, f^{36a}, f^s, and f^x. The positions of these insertions are consistent with the organization of the two pseudoallelic series present at the forked locus. Three RNAs of 0.8, 2.6, and 3.3 kilobases are transcribed from this chromosomal region. The 0.8-kilobase transcript(s), present at the larval and adult stages, and the 3.3-kilobase transcript, present at each developmental stage, are unaffected by the forked mutations examined. Only the 2.6-kilobase RNA, present exclusively at the pupal stage, was observed to be less abundant in each of the forked mutants analyzed, consistent with this transcript being the product of the forked gene.

The gene product of the *Drosophila* forked locus is required for correct bristle and hair formation. Mutations at this locus result in the shortening and bending of these structures on the adult cuticle compared with wild-type flies (11). Genetic and cytologic analysis had indicated that this locus is relatively simple, consisting of two pseudoallelic series located at band 15F (4, 5, 11, 15). In addition, the phenotypes of certain alleles of the forked locus are suppressed by second-site mutations at the *su(f)* locus (11). The availability of a temperature-sensitive allele of suppressor of forked [$l(1)su\ (f)^{ts67g}$] has permitted the determination of the temporal requirement for the product of the forked locus to a 24-h period of pupal development beginning just before the initiation of bristle information (2).

Since the forked locus could be cloned by transposon tagging by using the gypsy mobile genetic element (14), molecular analysis of this locus was performed with a view to examining the nature of forked mutations at the DNA and RNA level and to investigate the mechanism by which certain forked mutations are suppressed by mutation at the *su(f)* locus. From such analysis, DNA insertions were identified for several forked mutants. The insertions are clustered in two groups with the same organization as has been observed genetically and designated the right and left forked pseudoallelic series (4, 5, 11, 15). In addition, each of the forked mutants analyzed showed a reduced abundance of a 2.6-kilobase (kb) pupal RNA compared with the wild-type level, suggesting that this transcript encodes the forked gene product.

MATERIALS AND METHODS

The genotypes and sources of the fly strains used are given in Table 1. In the text irrelevant marker alleles are not included. Preparation and analysis of plasmid and phage DNA, total cellular *Drosophila* RNA, partial *Drosophila* DNA *Mbo*I libraries constructed with Charon 30 *Bam*HI phage arms, subclones in pUC13 DNA, restriction enzyme digestions, DNA ligation, nick-translation reactions, DNA and glyoxal RNA gel electrophoresis, and filter hybridizations with Biodyne membranes (ICN Corp.) were performed by standard procedures (12). The filter hybridization conditions used were 5× SSC (1× SSC is 0.15 M NaCl–15 mM sodium citrate), 10% dextran sulfate, 5× Denhart solution, 0.5% sodium dodecyl sulfate, 20 mM sodium phosphate (pH 6.8), 100 μg of denatured calf thymus DNA per ml, and 0.1 μg of ^{32}P-labelled denatured probe DNA. For DNA blots hybridization were performed for 16 h at 65°C, and for RNA blots hybridizations were performed for 16 h at 37°C under the same conditions, except the hybridization buffer contained 50% formamide. Filters were washed at 65°C for 30 min in 5× SSC–0.5% sodium dodecyl sulfate, for 60 min in 2× SSC–0.5% sodium dodecyl sulfate, and for 60 min in 0.2× SSC–0.5% sodium dodecyl sulfate. *Drosophila* DNA was isolated by the procedure of McGinnis et al. (13), and in situ hybridization was performed as described by Cohen and Meselson (1).

TABLE 1. *Drosophila* strains

Genotype	Source	Reference
Wild type (OreR)	Caltech *Drosophila* Stock Center	
$yvf^x car$	Mid-American *Drosophila* Stock Center	(11)
$vf^1\ su(f)$	Mid-American *Drosophila* Stock Center	(11)
$f^s l(1)\ su(f)^{ts67g}$	Caltech *Drosophila* Stock Center	(2)
$C(1)DX,yf^1/Dp(1;1)f^{+ih},f^1f^{+ih}\ B\ os$	Mid-American *Drosophila* Stock Center	(11)
$w^{bf}\ f^5$	Caltech *Drosophila* Stock Center	(11)
$f^5\ su(f)$	Mid-American *Drosophila* Stock Center	(11)
f^{36a}	Mid-American *Drosophila* Stock Center	(11)
$vf^{3n}\ car$	Mid-American *Drosophila* Stock Center	(11)
$y^2\ v\ f^{3n+}\ car$	M. M. Green	(6)
$w^if^3\ bb^n$	Mid-American *Drosophila* Stock Center	(11)
$C(1)DX,yf/f^{56e}; cn\ bw$	Mid-American *Drosophila* Stock Center	(11)

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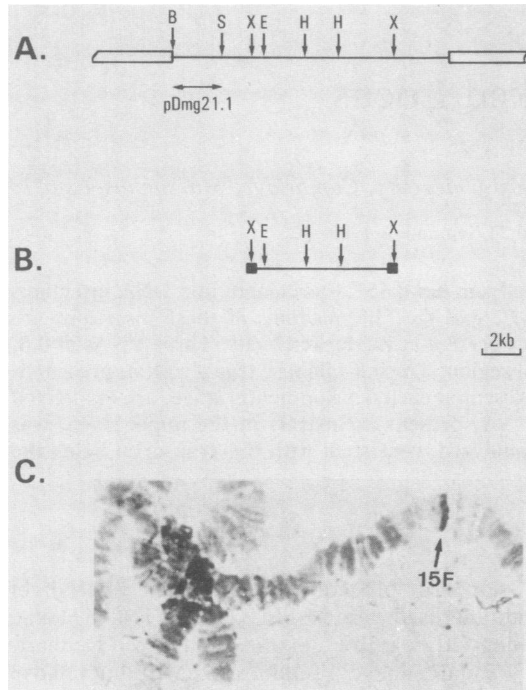
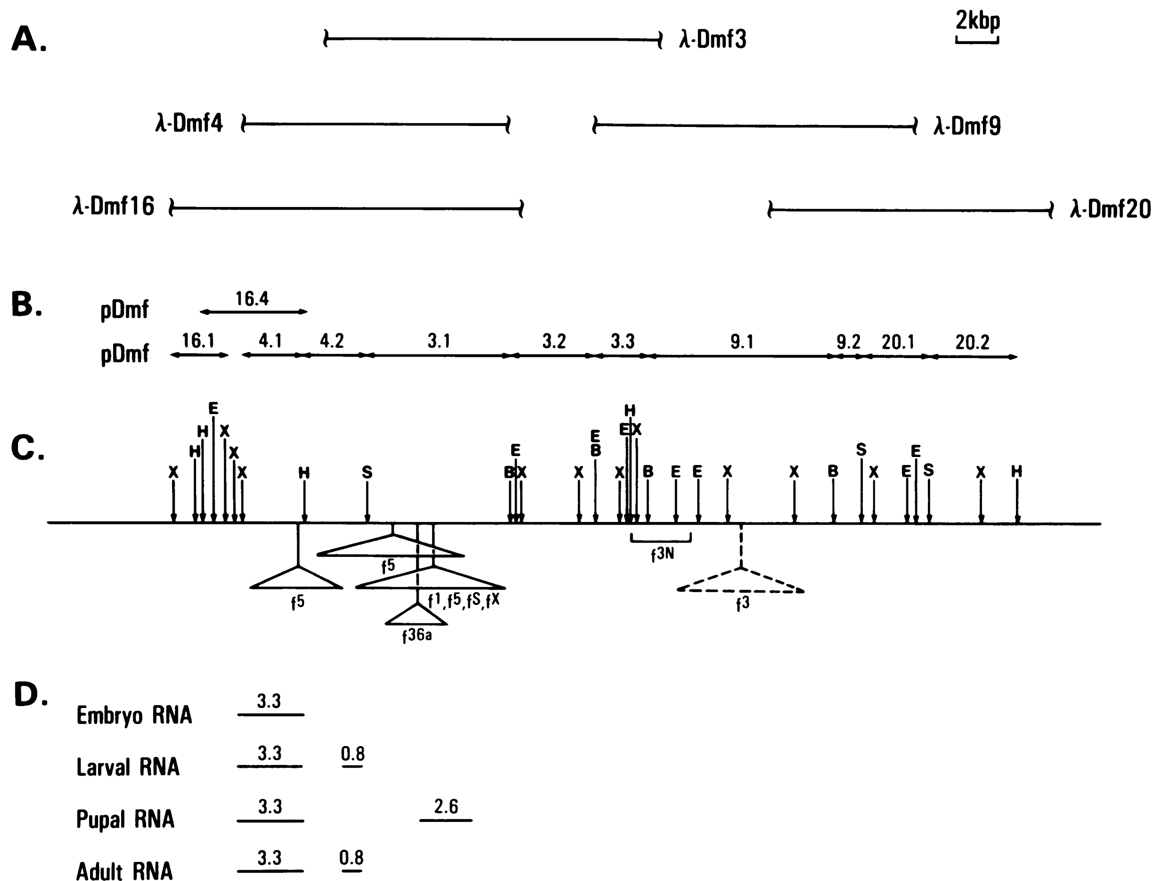


FIG. 1. A, Restriction map of λ-Dmg21 DNA insert showing *Bam*HI-*Sal*I fragment of pDmg21.1 subclone: (—) f^5 DNA;(▭) Charon 30 phage arms; B, *Bam*HI; E, *Eco*RI; H, *Hin*dIII; S, *Sal*I, X, *Xho*I. B, Restriction map of gypsy mobile element: (—) gypsy element DNA; (▬) terminal direct repeat sequences (14). C, In situ hybridization of OreR chromosome with λ-Dmg21.

RESULTS AND DISCUSSION

In *Drosophila*, the correlation between mutant alleles at a variety of different loci containing the gypsy mobile genetic element and the observation that these mutations can be suppressed by mutations at a second distant locus, the suppressor of hairy wing [*su*(*Hw*)], has permitted the cloning of a number of loci by transposon tagging (14). With this approach, it has been possible to clone the *Drosophila* forked locus. A genomic library was prepared with f^5 DNA and screened with the 6.8-kilobase-pair (kbp) *Xho*I gypsy DNA fragment from bx^{34e}-*6a* (14). Phage containing gypsy sequences were plaque purified and used for in situ hybridization to OreR salivary chromosomes. A single phage, λ-Dmg21 (Fig. 1A), was identified, containing a complete 7.3-kbp gypsy element (Fig. 1B), which hybridized in situ to the chromosomal band 15F (Fig. 1C). A 2.6-kbp *Bam*HI-*Sal*I DNA fragment, lacking gypsy DNA sequences, was subcloned (pDmg21.1) and used to initiate a chromosomal walk by utilizing a *Drosophila* OreR phage library. A 40-kbp region of chromosomal DNA was cloned and analyzed (Fig. 2).

Nine forked mutants were analyzed at the DNA level (Fig. 3 and 4). The 11 subclones (Fig. 2B) were used to probe genomic DNA digested with the same restriction enzymes as were used for the construction of the subclones. In the absence of any DNA insertions, deletions, and rearrangements, these probes hybridized to restriction fragments of the same length in OreR and forked mutant DNA. The probes pDmf4.2 and 3.2 (Fig. 3B and D) showed this pattern of hybridization, as did probes pDmf16.1, -9.2, -20.1, and



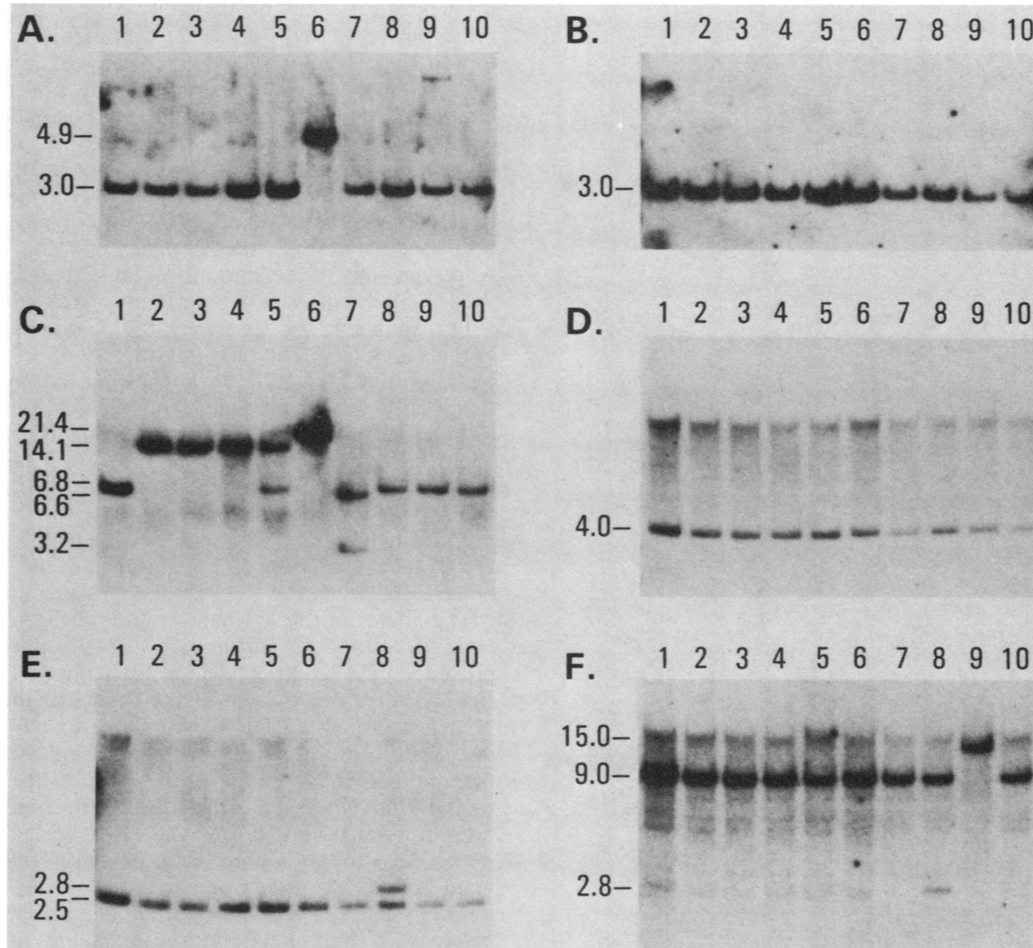


FIG. 3. DNA filter hybridization analysis of the forked locus. A, *Hin*dIII-*Xho*I-digested DNA probed with pDmf4.1; B, *Hin*dIII-*Sal*I-digested DNA probed with pDmf4.2; C, *Bam*HI-*Sal*I-digested DNA probed with pDmf3.1; D, *Bam*HI-digested DNA probe with pDmf3.2; E, *Bam*HI-digested DNA probed with pDmf3.3; F, *Bam*HI-digested DNA probed with pDmf9.1. Lanes 1 through 10 contained 1 μg of OreR, f^x, f^1, f^s, f^1f^{+ih} (male only), f^5, f^{36a}, f^{3n}, f^3, and f^{56e} (male only) DNA, respectively.

-20.2 (data not shown). With pDmf3.2 as a probe (Fig. 3D), it was also noted that this subclone contained a repetitive sequence. Filter hybridizations with pDmf16.4 (data not shown) and pDmf4.1 (Fig. 3A) as probes demonstrated the presence of an insertion in the f^5 chromosome in this region. Further analysis of this insertion (Fig. 4A; data not shown) demonstrated the insertion was 4.4 kbp in length and located less than 0.5 kbp to the left of an *Hin*dIII site (Fig. 2C).

Filter hybridization analysis of *Bam*HI-*Sal*I-digested genomic DNA with a pDmf3.1 DNA probe (Fig. 3C) revealed a 7.3-kbp insertion in f^1, f^s, and f^x DNA and 14.6 kb of additional sequences in f^5 DNA. Since all of these mutations are suppressible by the su(f) mutation (2, 11) and the f^1 and f^5 mutations are associated with gypsy DNA insertions (14), it seemed possible that these mutations were all related to insertion of gypsy mobile elements. To examine this, these DNAs were digested with *Xho*I and *Sal*I-*Xho*I and analyzed by filter hybridization with pDmf3.1 as a probe (Fig. 4B). Since *Xho*I digests the long terminal repeat sequence of gypsy DNA, these insertions would be released from DNA fragments containing such insertions. In addition, *Sal*I digestion can identify which fragment in a filter hybridization is derived from the left end of the region probed with pDmf3.1, and therefore the position of the gypsy insertions can be identified. Each of these mutations contains either one (f^1, f^s, and f^x) or two (f^5) insertions flanked by *Xho*I sites (Fig. 4B). In the case of the f^1, f^s, and f^x alleles the insertion is located 3.2 kbp to the right of the *Sal*I site (Fig. 2C). The f^5 DNA contains an insertion in this position plus another 7.3-kbp insertion located 1.2 kbp to the right of the *Sal*I site (Fig. 2C), as also observed in λ-Dmg21 (Fig. 1A). Additional filter hybridization analysis (data not shown) is consistent with the insertions in these mutants being complete gypsy

FIG. 2. A, Location of recombinant phage OreR insert DNA, relative to chromosomal walk, used to prepare subclones. B, Location of subclones relative to chromosomal walk. C, Restriction enzyme map of forked locus. Positions and sizes of various forked insertions are indicated. The f^3 insertion, indicated by dashed lines, is at least 6 kbp and has only been localized to the region homologous to pDmf9.1. Abbreviations are as in Fig. 1A. D, Transcription pattern of *Drosophila melanogaster* (OreR).

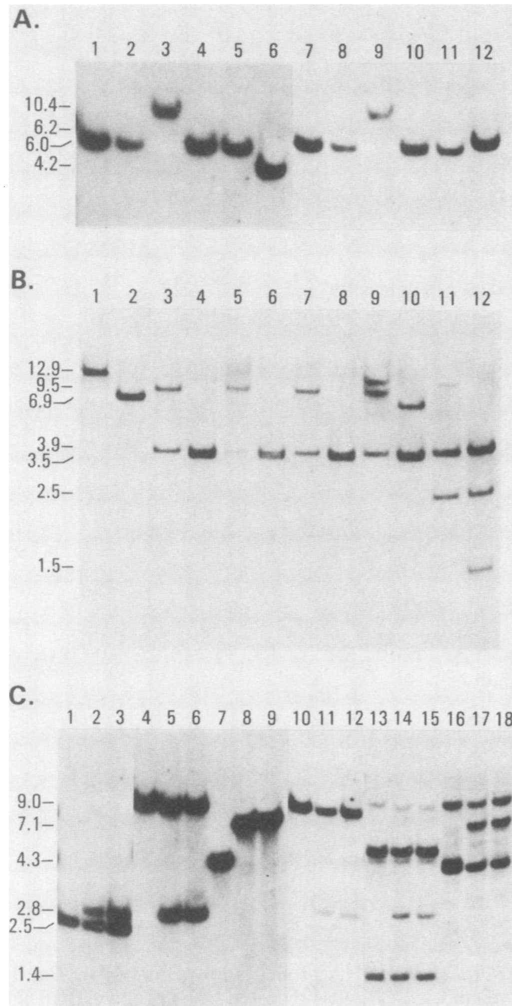


FIG. 4. DNA filter hybridization analysis of the forked locus. A, Lanes: 1 through 6, probed with pDmf4.1; 7 through 12, probed with pDmf4.2; 1 through 3 and 7 through 9, DNA digested with *Xho*I-*Sal*I; 4 through 6 and 10 through 12, DNA digested with *Bam*HI-*Xho*I-*Sal*I; 1 and 4, 1 μg of OreR DNA; 2 and 5, 1 μg of f^{1} DNA; 3 and 6, 1 μg of f^{5} DNA. Note that the data demonstrate this f^{5} insertion is 4.4 kbp [10.4 − 6.0 kbp and (6.2 + 4.2) − 6.0 kbp] and contains a *Bam*HI site. The difference in size between the bands of hybridization in Fig. 3A is 1.9 kbp (4.9 − 3.0 kbp) and results from the location of the insertion and the presence of a *Hin*dIII site within it. B, Probed with pDmf3.1. Lanes: 1, 3, 5, 7, 9, and 11, DNA digested with *Xho*I; 2, 4, 6, 8, 10, and 12, DNA digested with *Sal*I-*Xho*I; 1 and 2, 1 μg of OreR DNA; 3 and 4, 1 μg of f^{x} DNA; 5 and 6, 1 μg of f^{s} DNA; 7 and 8, 1 μg of f^{1} DNA; 9 and 10, 1 μg of $f^{1}f^{+ih}$ DNA; 11 and 12, 1 μg of f^{5} DNA. C, Lanes 1 through 9, probed with pDmf3.3; 10 through 18, probed with pDmf9.1; 1 through 3 and 10 through 12, DNA digested with *Bam*HI; 4 through 6 and 13 through 15, DNA digested with *Bgl*II; 7 through 9 and 16 through 18, DNA digested with *Sac*I; 1, 4, 7, 10, 13, and 16, 1 μg of OreR DNA; 2, 5, 8, 11, 14, and 17, 1 μg of f^{3n} DNA; 3, 6, 9, 12, 15, and 18, 1 μg of f^{3n+} DNA.

elements, each oriented in the same direction as in λ-Dmg21. Therefore, f^{1}, f^{5}, f^{s}, and f^{x} each contain a gypsy element in the same orientation inserted in a very similar location, possibly at precisely the same nucleotide, since gypsy element insertion is sequence specific (3). This suggests that either the same DNA insertion event has occurred independently several times or that this insertion occurred in a progenitor of each of these fly stocks. The f^{s} allele is presumed to be the f^{1} allele (2), and therefore their identical forked gene structure is expected. However since the f^{1}, f^{5}, and f^{x} alleles were isolated at different times (11), it seems likely that the same insertional event has occurred independently in these mutations. Interestingly, f^{1} and f^{5} are spontaneous mutations, whereas f^{x} was X ray induced. In addition, the analysis of $f^{1}f^{+ih}$ DNA (Fig. 3C and 4B) demonstrates the presence of both the gypsy element insertion associated with the f^{1} allele and the corresponding wild-type copy of this chromosomal segment represented by the f^{+ih} allele, present in heterochromatin, which is presumably responsible for the variegated position effect observed in this stock (15). The breakpoints of the f^{+ih} DNA segment were not detected (Fig. 3), suggesting that a region greater than 40 kbp was translocated from the wild-type forked locus to heterochromatin in the formation of this allele. The f^{36a} allele has a 3.0-kbp insertion (Fig. 3C, data not shown) located 2.5 kbp to the right of a *Sal*I site (Fig. 2C). This locates an insertion for all the alleles of the right-hand pseudoallelic series examined within a 0.7-kbp region of DNA and can account for the absence of observable recombination between the f^{1}, f^{36a}, and f^{x} alleles (5, 15).

The alleles of the left-hand pseudoallelic series examined were f^{3n} and f^{3}. Analysis of f^{3n} DNA demonstrated the presence of a 2.8-kbp tandem duplication (Fig. 3C and F; Fig. 4C; data not shown) of the sequence indicated in Fig. 2C. Digestion of f^{3n} DNA with *Bam*HI or *Bgl*II, which cut once within the duplication, released a 2.8-kbp DNA fragment homologous to both pDmf3.3 and pDmf9.1, in addition to the wild type-sized DNA fragments observed in these filter hybridizations (Fig. 4C). However, *Sac*I digestion, which does not cut within the duplication, results in a single band of hybridization which is 2.8 kbp larger in f^{3n} DNA than in wild-type DNA (Fig. 4C). The observation that the patterns of hybridization are the same for f^{3n} and the revertant f^{3n+} leaves unresolved the nature of the reversion event and raises the question as to whether the 2.8-kbp duplication is responsible for this forked mutation. The f^{3} allele has been shown to contain an insertion of at least 6 kbp in the region homologous to subclone pDmf9.1 (Fig. 3F). The proximity of the f^{3} and f^{3n} DNA insertions (Fig. 2c) suggests that they might be responsible for these mutations, since this would mean that the two pseudoallelic series at the forked locus are separated by approximately 10 kbp. This could account for the observable recombination frequency between alleles of the left and right pseudoallelic series (4, 5, 15). If these assumptions are correct, the left end of the chromosomal walk in Fig. 2C is proximal to the chromocenter, and the right end is distal to the chromocenter. At the DNA level no differences between the f^{56e} allele and wild-type DNA were observed.

The temporal transcription pattern of the forked locus and the effect of various forked and *su(f)* mutations were examined (Fig. 5). With the 11 subclones (Fig. 2B), transcripts were only detected with pDmf4.1, -4.2 and -3.1 as probes. A 3.3-kb transcript homologous to pDmf4.1 was observed at all developmental stages. This transcript was unaffected by the f^{3}, f^{3n}, f^{5}, and f^{x} mutations (Fig. 5A; data not shown). Similarly, the 0.8-kb transcript(s), present at larval and adult stages of development and homologous to pDmf4.2 and -3.1, was unaffected by the same mutations (Fig. 5B, data not shown). In contrast, a 2.6-kbp transcript present only at the pupal stage of development and homologous to pDmf3.1 was decreased in abundance in each of the mutants examined (Fig. 5C and D; data not shown). Quantitation of the 2.6-kb

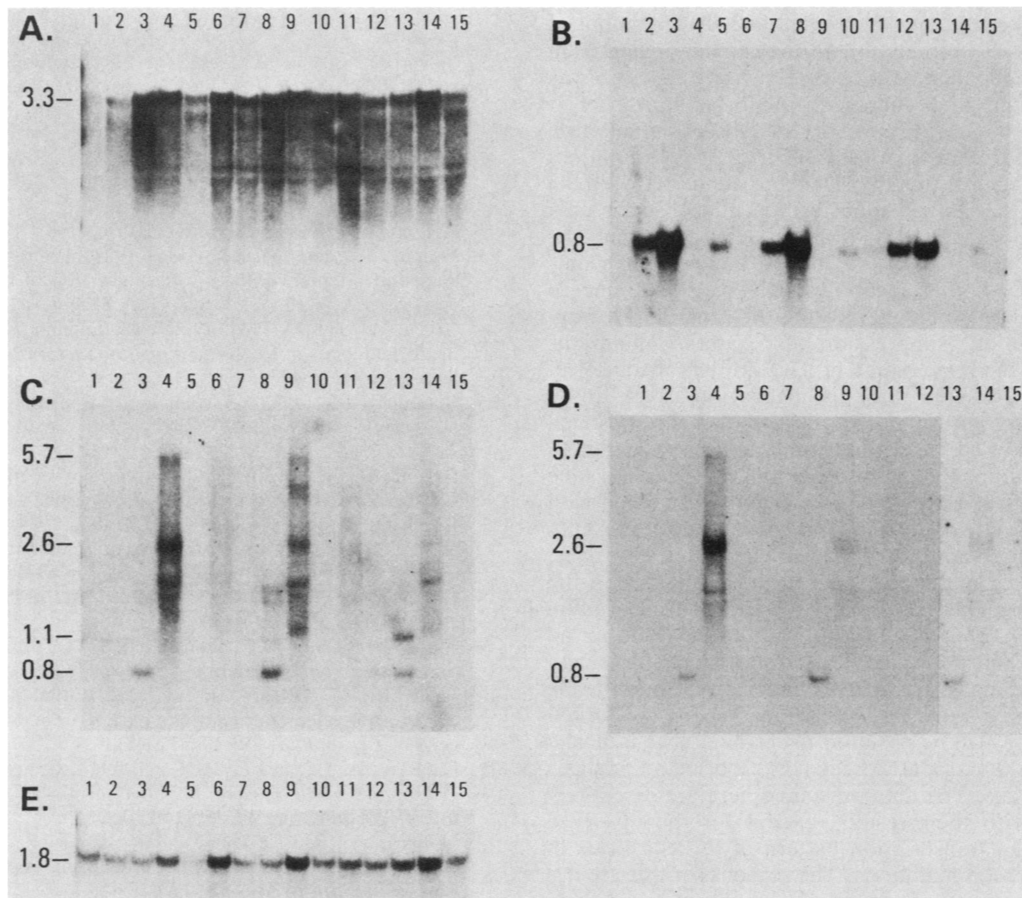


FIG. 5. RNA filter hybridization of the forked locus. A, Probed with pDmf4.1; B, probed with pDmf4.2; C and D, probed with pDmf3.1; E, probed with pDmTα1 (8). A, B, C, and E, Lanes: 1 through 5, 2.5 μg of OreR RNA; 6 through 10, 2.5 μg of f^s $su(f)^{ts67g}$ RNA prepared from *Drosophila* grown at 18°C; 11 through 15, 2.5 μg of f^s $su(f)^{ts67g}$ RNA prepared from *Drosophila* grown at 25°C. D, Lanes: 1 through 5, 2.5 μg of OreR RNA; 6 through 10, 2.5 μg of f^{3n} RNA; 11 through 15, 2.5 μg of f^3 RNA. A through E, Lanes: 1, 6, and 11, embryo RNA; 2, 7, and 12, first- and second-instar larval RNA; 3, 8, and 13, third-instar larval RNA; 4, 9, and 14, pupal RNA; 5, 10, and 15, adult RNA. The abundance of the 2.6-kb RNA reported in the text was estimated by densitometer scanning of autoradiograms (Fig. 5C and D; data not shown). The estimated abundances of the forked transcript were divided by the estimated abundances of the pupal tubulin transcripts in the same RNA sample (Fig. 5E, data not shown) (9) to correct for any variation in amount of RNA per lane and RNA blotting efficiencies. The estimated abundances of the pupal tubulin transcripts used to make these corrections were determined from filters reprobed with pDmTα 1.

RNA levels in these mutants demonstrated a 3- to 10-fold reduction of this transcript for the less extreme alleles examined [12, 13, and 35% of the wild-type level for f^s $su(f)^{ts67g}$ at 18°C, f^3, and f^{3n}, respectively] and its complete loss in the case of the extreme f^5 allele. Therefore, it seems likely that the 2.6-kb pupal RNA, present at the developmental stage when the product of the forked gene is required for bristle formation (2), represents the forked gene transcript. In addition to the 2.6-kb transcript, a low level of a 5.7-kb pupal transcript was observed (Fig. 5C and D). This transcript appears to be reduced in abundance in the various forked mutants examined. The bands of hybridization observed at 1.6 and 1.8 kb (Fig. 5C and D) may also represent low-level pupal transcripts which are less abundant in the forked mutants. However, since rRNA also migrates at this position, these bands may be artifactual, similar to the bands at the same position in Fig. 5A. For this reason, these bands are not considered further.

In the cases where a forked mutation is suppressed by the $su(f)$ mutation (Fig. 5C, data not shown), the level of the 2.6-kb transcript is not elevated to wild-type levels [0 and 2% of the wild-type level for f^5 $su(f)$ and f^s $su(f)^{ts67g}$ at 25°C, respectively]. This result contrasts with the transcription pattern observed at the *Drosophila* white locus, where suppression of the white-apricot mutation by the suppressor of white apricot results in the increased abundance of a wild type-sized transcript (10). However, in the case both of f^s $su(f)^{ts67g}$ maintained at 25°C (Fig. 5C) and f^5 $su(f)$ (data not shown), where the forked mutation is suppressed, a 1.1-kb transcript homologous to pDmf3.1 is observed at the third-instar larval stage. These observations suggest that this transcript may supply the forked gene product, resulting in suppression of the forked mutations. However, the inappropriate temporal expression and limited size of the transcript argue against this possibility. A detailed analysis of the transcription units coding for the 1.1- and 2.6-kb RNAs will be required to determine the relationship between these transcripts and to estimate the role the 1.1-kb RNA might have in the molecular mechanism of suppression. In addition, such analysis may indicate the location of transcription signals which regulate the temporal expression of these transcription units.

In conclusion, a 40-kbp chromosomal region containing the forked locus was cloned, and DNA insertions associated

with several forked mutants were identified. The positions of these insertions are consistent with the genetic organization of the two pseudoallelic series. A 2.6-kb transcript was identified which probably represents the forked transcript. It is reduced in abundance in a varity of forked mutants in a manner which correlates with the severity of the mutation. The second gypsy insertion in f^5 DNA, as compared with the f^s DNA, is presumably responsible for the absence of a detectable level of the 2.6-kb pupal transcript and the extreme phenotype in this mutant. In contrast, it seems likely that the 4.4-kbp insertion in f^5 DNA is silent, since it does not affect the abundance of the 0.8- and 3.3-kb transcripts. In addition, suppression of f^5 and f^s mutations correlates with the appearance of a 1.1-kb larval transcript homologous to the same chromosomal region as the 2.6-kb wild-type pupal transcript. This is also the chromosomal region where all of the insertions of the right-hand pseudoallelic series, including the gypsy elements of the suppressible f^5 and f^s alleles, are located. The insertions associated with the left-hand pseudoallelic series are located in a chromosomal region where no transcripts were detected. However, both the f^3 and f^{3n} mutations reduced the abundance of the 2.6-kb transcript. This may reflect the forked transcription unit extending to this region but contributing little or no sequence to the mature forked RNA. Alternatively, this may represent mutations acting at a distance as reported for the w^{DZL} mutation (7). Further detailed analysis of the forked transcription unit will be required to resolve these issues.

Recently, Parkhurst and Corces (16) reported an analysis of the forked locus. The data presented here are essentially in agreement with their results, except for the observed differences in the transcription pattern of forked alleles in response to the *su(f)* mutations. The reasons for this are not obvious, but may reflect differences in the materials and procedures utilized in these two studies such as the fly stocks used, the method of RNA preparation, and conditions of filter hybridization analysis.

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