The Human Transcription Enhancer Factor-1, TEF-1, Can Substitute for Drosophila scalloped during Wingblade Development*

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The human transcription enhancer factor-1 (TEF-1) belongs to a family of evolutionarily conserved proteins that have a DNA binding TEA domain. TEF-1 shares a 88% homology with Drosophila scalloped (sd) in the DNA binding domain and a 50% similarity in the activation domain. We have expressed human TEF-1 in Drosophila under the hsp-70 promoter and find that it can substitute for Sd function. The transformants rescue the wingblade defects as well as the lethality of loss-of-function alleles. Observation of reporter activity in the imaginal wing discs of the enhancer-trap alleles suggests that TEF-1 is capable of promoting sd gene regulation. The functional capability of the TEF-1 product was assessed by comparing the extent of rescue by heat shock (hs)-TEF-1 with that of hs-sd. The finding that TEF-1 can function in vivo during wingblade development offers a potent genetic system for the analysis of its function and in the identification of the molecular partners of TEF-1.

Transcription enhancer factor-1 (TEF-1) belongs to a family of transcription factors with the TEA/ATTS DNA binding domain (1) and was the first such gene identified in mammals. The protein was originally identified from HeLa cells by its ability to bind tandemly repeated GT-IIC or Sph motifs from the Simian virus 40 (SV40) (2, 3). It was subsequently shown to bind the P97 promoter of human papilloma virus-16 and act in the regulation of transcription of the E6 and E7 oncogenes (4). Results from transfection and biochemical fractionation experiments showed that the transcriptional activation of TEF-1 requires co-operation between several domains of the molecule and is modulated by transcriptional intermediary factors (TIFs) as well as at least two negatively acting factors (5, 6). The identity of these factors at present remains unknown.

TEF-1 homologues have been identified in Drosophila, mouse, chick, Aspergillus, and yeast, and the TEA domains of these genes show a remarkable degree of conservation (7–12).

Recent studies on the different mammalian TEFs suggest that these genes play partially overlapping roles in the development of the nervous system and musculature (13, 14). The Drosophila homologue, scalloped, has also been shown to play roles in the development of the nervous system and musculature, and viable alleles show aberrations in their response to taste stimuli (15). In TEF-1, the TEA domain has been shown to be sufficient for specific DNA binding of GT-IIC and Sph enhancer sequences (16). This domain in Drosophila Sd, which differs from that of TEF-1 by a single amino acid, is incapable of binding these sequences. Furthermore, the Sd protein fails to transactivate these sequences in cell lines where the TEF-1 protein acts at high efficiency (16). Interestingly, conversion of the serine residue in the Sd TEA to alanine (the corresponding residue in TEF-1) did not confer binding ability similar to that of TEF-1. The altered Sd molecule could bind GT-IIC weakly and still failed to bind Sph enhancer sequences. Similarly, a mutation of alanine to serine in the TEF-1 TEA domain did not significantly alter its DNA binding properties. These observations together suggest that DNA binding in both TEF-1 and Sd molecules is modulated by regions outside the TEA domain. This conclusion is further strengthened by experiments in which deletions of the C-terminal domain of TEF-1 were found to reduce the DNA binding efficiency, thus leading to the identification of putative activation domains of TEF-1. The mechanisms by which the activation domains of TEF-1 influence DNA binding efficiency have not been investigated.

The existence of a TEF-1 homologue in Drosophila raises the possibility of exploiting the rich repertoire of genetic information in the fly to study the regulation of the mammalian gene and the pathways in which it participates. In this paper, we use the wingblade phenotypes of sd mutants to show that TEF-1 can substitute for Sd in vivo. This observation allows the study of the role of TEF-1 in the context in which sd participates. sd along with several other genes, namely, engrailed (en), wingless (wg), hedgehog (hh), apterous (ap), Serrate (Ser), Notch (N), and vestigial (vg) plays an important role in the development of the wingblade (17). The Drosophila wingblade is an excellent system for the study of how complex genetic hierarchies operate during development. The exquisite design of the wing of the fly allows for the facile identification of mutations that cause even subtle changes in its structure (18). Vertebrate homologues of several of genes involved in wing development have now been identified, and it is becoming increasingly clear that despite several million years of evolutionary divergence between flies and mammals, molecular mechanisms fathomed from one system may not be too far removed from those occurring in another (19, 20). Our obser-

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1 The abbreviations used are: TEF-1, transcription enhancer/factor-1; TIF, transcriptional intermediary factor; kb, kilobase(s); X-gal, 5-bromo-4-chloro-3-indolyl β-D-galactopyranoside; hs, heat shock.

2 A. Chopra, unpublished data.

3 M. Inamdar, unpublished data.
vation that TEF-1 can function in Drosophila could provide a means by which the regulation of TEF-1 and its partners can be studied using a combination of genetics and biochemistry.

**EXPERIMENTAL PROCEDURES**

All standard molecular biology procedures were performed as described in Sambrook et al. (21).

**Strains**—The full-length TEF-1 cDNA was kindly provided by Pierre Chambon and Irwin Davidson, CNRS, Inserrm, Strasbourg, France. The pCasPer-Hs plasmid was obtained from Vince Pirotta, University of Geneva. The hs-sd strain carries four copies of Pu(w')-hs-ad) and was a kind gift from Sean Carroll, University of Wisconsin, Madison. The insertions in this strain were located on the second and third chromosomes, respectively. Flies carrying the transposase source P(6–2 3 ry'Sh/TM2 P(6–2 3 ry') Ubx were kindly provided by Hugh Robertson. Details about all marker and balancer strains can be obtained from Lindsley and Zimm (22).

**Generation of hs-TEF-1 Transgenic Flies**—The 1.2 kb TEF-1 cDNA was released from the PX40 plasmid by digestion with EcoR I and BglII and subcloned into the pCasPer-Hs plasmid in frame with the Drosophila hsp70 promoter. The recombinant vector (Pu(w'-hs')TEF-1) was micro-injected along with papw into w1118 embryos following protocols described by Rubin and Spradling (23). Germline transformants were selected by monitoring the w'-marker in subsequent generations. One transgenic line (hs-TEF-1(3.1)) was obtained in which the insertion mapped on the third chromosome and allowed homozygous viability.

**Mobilization of the Pu(w'-hs'-TEF-1A) Transposon**—The third chromosome insertion was mobilized by standard genetic crosses using the δ 2–3 transposase source to generate additional lines designated hs-TEF-1(2.1) and hs-TEF-1(2.6). Both these lines had insertions on the second chromosome, and the hs-TEF-1(2.1) insertion resulted in recessive lethality. All three lines were crossed again to a transposase source to generate lines in which the P element had been completely lost. The excision lines which were selected on the basis of their eye color and verified in Southern blots using 32P-labeled TEF-1 cDNA as a probe. Excision lines, in which the Pu(w'-hs'-TEF-1) had been precisely excised were served as controls for subsequent experiments.

**In Situ Hybridization to Polytene Chromosomes**—Polytene chromosomes were dissected from salivary glands of third instar larvae as described by Ashburner (24) and probed with digoxigenin-labeled TEF-1 cDNA.

**RNA Extraction and Northern Analysis**—Embryos from the hs-TEF-1(3.1) and w1118 strains were collected 12 h after egg laying and were heat shocked in a water bath at 37°C for 30 min. Total RNA was extracted as described in Ashburner (24), separated on formaldehyde/agarose gels, and transferred to nylon membranes as described in Sambrook et al. (21). Filters were probed using 32P-labeled TEF-1 cDNA and autoradiographed according to standard protocols.

**Cuticle Preparation**—All flies to be analyzed were stored in 70% ethanol for at least 24 h prior to preparation. Wings were mounted in a drop of Faure’s solution (34% v/v chloral hydrate, 13% v/v glycerol, 20 mg/ml gum arabic, and 0.3% cocaine chloralhydrate) and left at 70°C overnight to clear.

**Histochemical Detection of sd Reporter Gene Activity**—Larval imaginal wing discs were fixed in 4% paraformaldehyde in phosphate-buffered saline and stained using the chromogenic substrate X-gal according to the method described by Wilson et al. (25). Stained discs were washed in phosphate-buffered saline and mounted on slides with gelto.

**RESULTS**

The sd locus encodes at least three different transcripts which arise by alternative splicing of an approximately 12 kb transcription unit. The cDNA clone designated E21 corre-

![Image](http://www.jbc.org/)

**Fig. 1.** Comparison of the TEF family genes with sd. Sequences of the different TEF family genes are aligned with sd; the region of homology beginning at position 51 of sd. The TDA domain is completely conserved except for alanine to serine at position 106 of sd. There are two divergent domains, the N terminus and the region following the TDA domain.

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High levels of expression leads to lethality at the Embryonic and Pupal Stages—We showed that TEF-1 mRNA was transcribed in Drosophila under control of the hsp70 promoter. Embryos homozygous for hs-TEF-1(3.1) were heat shocked at 37°C for 1 h, and the mRNA was probed on Northern blots for the presence of TEF-1 transcripts. There was no endogenous TEF-1-like expression in control embryos while the hs-TEF-1 animals showed high levels of TEF-1 transcripts (data not shown).

High levels of TEF expression induced by heat shock resulted in variable levels of lethality throughout embryonic life. The extent of lethality observed was dependent upon the stage during embryogenesis when the temperature pulse was given. Previous studies using an enhancer trap allele of sd showed that the native expression of the sd gene was first detected at about stage 9 of embryonic development (10). Overexpression of TEF in the larval stages did not lead to significant lethality, but pupal pulses caused a high degree of lethality. Survivors that had experienced embryonic or pupal pulses did not, however, show any phenotypic defects typical of sd mutations. The production of lethality when TEF-1 was induced during embryonic and/or pupal life correlates well with the observation that all lethal alleles of sd die either during embryonic or the pupal stage.

**TEF-1 Can Substitute for Sd During Wingblade Development in Drosophila**—Most viable alleles at the sd locus show defects in wingblade development, resulting in a gapped or eroded appearance on the anterior and lateral margin (24). The extent of this phenotype is easy to quantitate and thus provides an attractive system by which we could monitor the functioning of TEF-1 in the animal. We chose the enhancer trap allele sd(w'ry+Ex1) (henceforth called as sdREx1) as our assay system because of its relatively high expressivity of the wingblade.
phenotype (Fig. 2B). sdETX4 carries an insertion of a P(lacZry1) element within the first intron of the transcription unit (10, 27). Genetic analysis suggests that sdETX4 is a partial loss-of-function allele since the phenotype in trans with a deficiency of the sd region is significantly more severe than that of the homozygous insertion strain (10, 15).

Males from the transgenic lines were crossed to females homozygous for sdETX4 and reared at 25 °C. The rescue of the wingblade defects in sdETX4 was scored in the male progeny. The wing defects observed were scored on an arbitrary scale of −1 to −5 where −5 denotes the strongest mutant phenotype and + indicates wild-type wings (Fig. 2A). In sdETX4 males, approximately 80% of the animals had wings corresponding to −3 (Fig. 2B). We compared the efficiency of different hs-TEF-1-bearing strains with that of hs-sd in rescuing the wingblade phenotypes in partial loss-of-function mutants. When sdETX4 was crossed to either the hs-sd or hs-TEF-1 transformants, the phenotype of the progeny was shifted significantly toward the wild-type (Fig. 2, C–E). 36% of sdETX4 carrying a single copy of the hs-TEF-1(2.1) transgene had completely normal wingblades; normal wings were never observed in the mutant strain in the absence of hs-TEF-1. While two copies of the hs-sd transformant significantly reduced the severity of the phenotype (Fig. 2C), four copies led to completely wild-type wings (data not shown). Of the three hs-TEF transformants that we used in this study, hs-TEF(2.1) was most effective at rescuing the wingblade phenotypes of sdETX4 (Fig. 2, D compared with E; data from hs-TEF(2.6) is comparable to hs-TEF(3.1)). This is in spite of the fact that the 3.1 line had two insertions of the hs-TEF-1 insertion on the chromosome. The hs-TEF(2.1) strain was also more efficient at rescuing the wingblade phenotype than the hs-sd transgenic. These differences could arise from positional influences on the regulation of genes inserted in different positions on the chromosome.

These data demonstrate that the expression of TEF-1 in flies leads to a striking rescue of the wingblade phenotype of sd
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TABLE I

Effect of hs-sd and hs-TEF transgenes on the viability of the pupal lethal allele sdETX81

| Genotype | sdETX81 males as percentage of total males |
|----------|-------------------------------------------|
| sdETX81, | 0                                         |
| sdETX81, | hasd/+ hassd/+                            |
| sdETX81, | has-TEF-1(3.1)                            |
| sdETX81, | has-TEF-1(3.1) exc.                      |
| sdETX81, | has-TEF-1(2.1)                            |
| sdETX81, | has-TEF-1(2.1) exc.                      |

FIG. 3. Third instar wing discs stained for β-galactosidase activity using the chromogenic substrate X-gal. (A) sdETX81 male. B, sdETX81/FM7 female. C, sdETX81; excision of hs-TEF-1(2.1) male. D, sdETX4; hs-sd/+ has-sd/+ male. E, sdETX81; hs-sd/+ has-sd/+ female. F, sdETX81; hs-TEF-1(2.1)/+ male. Arrowheads indicate the extent of β-galactosidase staining, which is increased in panels D–F. All discs were stained for the same amount of time.

mutants. A rescue of the sd mutant phenotype would also have been obtained if the insertion of the transposon resulted in mutations that could act as extragenic suppressors of sd mutation. If the rescue of the sd phenotype was indeed due to the expression of TEF-1, then, aberrations in the TEF-1 sequence within the transposon induced by imprecise excision would result in a loss of “rescue.” We generated lines in which the P(w+), hs-TEF-1) insertion was “excised” under the influence of transposase activity. Excision events were monitored by following the w+ marker on the transposon, and the chromosomes were analyzed on Southern blots using labeled TEF-1 cDNA as a probe. Lines in which no TEF-1 hybridization was detected were taken as complete excisions; and those in which hybridization was detected, but the restriction pattern had changed compared with the original line, were identified as events in which the P(w+, hs-TEF-1) was incompletely excised (data not shown). In the latter case, while some part of the transposon was still present, the TEF-1 gene would not be normally expressed. Several representative lines of both “complete” and “incomplete” excision events were crossed into sdETX81 backgrounds, and the phenotype of the wings of the progeny were observed. In both cases, the phenotype was not significantly altered from that of sdETX4, indicating that the expression of an intact TEF-1 sequence, and not merely the presence of an insertion into certain loci, was required for the rescue of the sd mutant wingblade defects (Fig. 2, F and G).

TEF-1 Function Can Rescue the Pupal Lethality of Strong sd Mutant Alleles—While weak (partial loss-of-function) alleles of sd show defects in the wingblade, strong mutants show embryonic or pupal lethality. The cause of lethality in embryonic lethal alleles is not clear although sd+ function has been implicated in axonal guidance as well as muscle development. In these roles, Sd presumably participates in a genetic pathway distinct from that involved in wingblade development. We tested whether TEF-1 could substitute for sd in this cellular context as well. sdPry-ETX81 (henceforth called sdETX81) dies in mid-pupation; genomic analysis revealed the presence of two insertions, one in the 5′ intron before the translational start site and a second within a 2-kb region located toward the 3′ end of the gene (26). Most of the lethal lesions in sd map to the latter interval (10). Preny of crosses of sdETX81 with the hs-TEF-1 and hs-sd transformants were reared at 30 °C, and the progeny were scored (Table I). Growth at 28 °C produced a smaller number of surviving males than at 30 °C. The viable mutant animals emerging from the crosses with the hs-sd and hs-TEF-1 transgenes still showed defects in wing morphology. This could mean that the levels and/or timings of sd/TEF-1 expression generated by the hsp70/TEF-1 promoter was insufficient to rescue the wingblade phenotypes even though this expression could rescue lethality. This explanation also applies to the roughened eye and maxillary palp phenotypes observed in sdETX81, hs-TEF-1/+ and sdETX81, and hs-sd/+ survivors. The pattern of β-galactosidase activity in the developing compound eye and maxillary palpal analage in the enhancer trap line sdETX81 suggests that sd+ is expressed in these regions (10).

Experiments with hs-TEF-1 Suggest That the sd Gene Is Autoregulated—Both sdETX81 and sdETX4 are alleles of sd generated by insertion of a Pazy′lacz2) transposon and express β-galactosidase in the wing disc (27). In sdETX4, the domain of β-galactosidase expression in the third instar wing discs parallels that of the mRNA expression seen in whole mount RNA in situ using sdE21 as a probe (10). We could neither detect Sd mRNA, nor any β-galactosidase reporter activity in the wing discs of sdETX81 hemizygous male larvae (Fig. 3A). The discs of sdETX81/+ heterozygous females, on the other hand, showed some staining in the third larval instar discs (Fig. 3B). The domain of expression was, however, significantly reduced over that seen in sdETX81/+ (10). In sdETX81/+ reporter enzyme expression was restricted to the dorsal/ventral boundary of the putative wing where sd+ expression is maximal. It is unlikely that this restricted pattern is due to an absence of precursors in the wing pouch region that would normally stain since sdETX81/+ adult wingblades are completely normal in morphology. These results together suggest that sd+ activity is required for driving reporter activity. Hence in sdETX84, a partial loss-of-function allele, the reporter activity more closely resembles the native gene expression than in the null allele (sdETX81) in trans with a wild-type chromosome. The lack of staining in sdETX81 males can thus be explained by an absence of sd+ activity.

We tested whether reporter enzyme activity could be enhanced by ectopic expression of Sd or TEF-1 using the hs-sd or hs-TEF-1 transgenes. β-Galactosidase activity was observed in wing discs of sdETX81 males carrying either hs-sd or any of the hs-TEF-1 insertions (arrowheads in Fig. 3, D and F). Interestingly, the effect observed with hs-TEF-1(2.1) was significantly greater than with hs-sd, and β-galactosidase staining was observed outside the expected wingpouch region (arrowheads in Fig. 3F). In sdETX81/+ the intensity as well as the domain of
expression were enhanced by sd (Fig. 3E), as well as TEF-1 expression (data not shown). The ability of the hs-TEF-1 and hs-sd transgenes to induce reporter activity suggests that the sd locus is autoregulated. Ectopic expression of sd/TEF-1 under a heat-shock promoter, interestingly, did not result in ubiquitous reporter enzyme activity, but this was restricted mainly to the wingblade region of the discs. This observation suggests that sd/TEF-1 may be necessary, but not sufficient, to drive sd activity, thus implicating the existence of other tissue specific factors.

**DISCUSSION**

We have shown that human TEF-1 can substitute for Sd in several different contexts during *Drosophila* development. Expression of this gene in *Drosophila* under control of the hsp70 promoter rescues the wingblade defects as well as the lethality seen in different sd mutations. Furthermore, TEF-1 functions as effectively as Sd in promoting expression of a sd reporter in the developing wing imaginal disc. Does TEF-1 protein interact with the same molecular targets as Sd when expressed in the fly? Data from biochemical experiments suggested that Sd and TEF-1 have different DNA binding capabilities and that the activation domains of the molecules resided outside the highly conserved TEA domains. The demonstration that TEF-1 can adopt most of the pleiotropic roles of Sd suggests that both molecules are capable of similar functions.

sd plays an important role in establishing the dorso-ventral patterning in the developing wingblade (28). Cells in the dorsal compartment of the disc are selected through the expression of *ap* which in turn activates *fringe* (*fr*) and Ser in these cells. Ser, which encodes a ligand for the N receptor induces the expression of *wg* in the ventral cells (17). sd is classified as a pro-wing gene and, like *vg*, has an expression pattern that straddles the D/V boundary; *vg* has recently been shown to be necessary and sufficient for the generation of wings in *Drosophila* (29). It is intriguing that TEF-1, a protein with only 68% homology, can participate in the complex interactions involved in wingblade development as efficiently as its *Drosophila* homologue. Recently other TEA domain-containing genes were identified in vertebrates that were similar to TEF-1 (14). Alignments of the amino acid sequences of various TEFs (TEF-1, TEF-3, and TEF-4) showed that the TEA domains were identical in all of them, and 200 amino acids in the C domain were well conserved. In contrast, the N-terminal regions of all TEFs and the region immediately following the TEA domain showed a considerable degree of divergence.

In *Drosophila*, the complexity of expression and the pleiotropic effects of sd mutations suggests a role for the different alternatively spliced variants from the locus. In addition, preliminary evidence does exist for the existence of additional genes with some resemblance to sd in the *Drosophila* genome. The biological function of TEF-1 is still unclear. The expression pattern is relatively widespread, and the results from mouse knockout experiments are difficult to interpret possibly due to redundancies of function (13). Studies of TEF-1 function in flies is a promising means by which to decipher its biological role. We are in the process of generating transgenic flies with other TEF-2 and TEF-4 genes along with chimeras of the activation domains and DNA binding domains and hope that this will contribute to a better understanding of the evolution and functions of the TEA domain family.

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