

Human homologs of a *Drosophila* Enhancer of split gene product define a novel family of nuclear proteins

S. Stifani, C. M. Blaumueller, N. J. Redhead, R. E. Hill & S. Artavanis-Tsakonas
Nature Genetics 2, 119–127 (1992)

The following parts of this paper appear now in their correct format.

Table 2 Comparison of the CcN motif of E(spl) m9/10, TLE proteins and proteins with demonstrated nuclear localization and susceptibility to phosphorylation by casein kinase II and/or cdc2

Protein	CK-II site ^a	cdc2 site ^a
E(spl) m9/10	231SDQD	247SPRP
TLE 1	239SDGD	259SPRA ²⁶³ SPA ²⁶⁷ SFR
TLE 2	228SDED	249SPAT ²⁵³ TPCGK
TLE 3	239SDGD	258TPRV ²⁶² SPA ²⁶⁶ SPP
SV 40 T antigen	111S ¹¹² SDDE	S ¹²⁴ TPPK
human c-myc	S ³⁸⁴ SDTE	T ³⁴⁴ SFRS
human p53	284TEEE	S ³¹⁵ SPQP
human A-myb dorsal ^b	467SLND 312SDGV ³¹⁶ TSEA	479TRLK 290TPRY

^aPhosphorylatable Ser/Thr residues are numbered.

^bThe *Drosophila* protein dorsal was included as one example of several other proteins bearing a putative CcN motif for which only translocation to the nucleus has been demonstrated.

Linkage disequilibrium mapping in isolated founder populations: diastrophic dysplasia in Finland

J. Hästbacka, A. de la Chapelle, I. Kaitila, P. Sistonen, A. Weaver & E. Lander
Nature Genetics 2, 204–211 (1992)

In the Methodology section of this paper the section entitled 'Estimation of recombination and mutation' appeared without important mathematical symbols in the explanation of equations. The correct version is as follows:

(Equations (1) and (2) correspond essentially to equations (8) and (12) of Luria and Delbrück, with the substitutions $N = N, C$ and $\sigma' = (\text{var } \bar{y}) / (C)$. With regard to the second substitution, our standard deviation σ' is smaller by a factor (C) than Luria-Delbrück's standard deviation $(\text{var } \bar{y})$ because we are interested in the standard deviation of the average of the proportions of mutants in the descendants of the C founders, whereas Luria and Delbrück were interested in the standard deviation among the proportions.)

A frameshift mutation in the γ E-crystallin gene of the Elo mouse

Mireille Cartier, Martin L. Breitman & Lap-Chee Tsui
Nature Genetics 2, 42–45 (1992)

The correct alignment for Table 1 in this paper is as follows:

Table 1 Complete association of Elo with the G403 deletion

Phenotype	Number of mice	G403 deletion	Polymorphism at position 444
Wild-type	143	no	T
Elo	131	yes	A
(Total)	274		

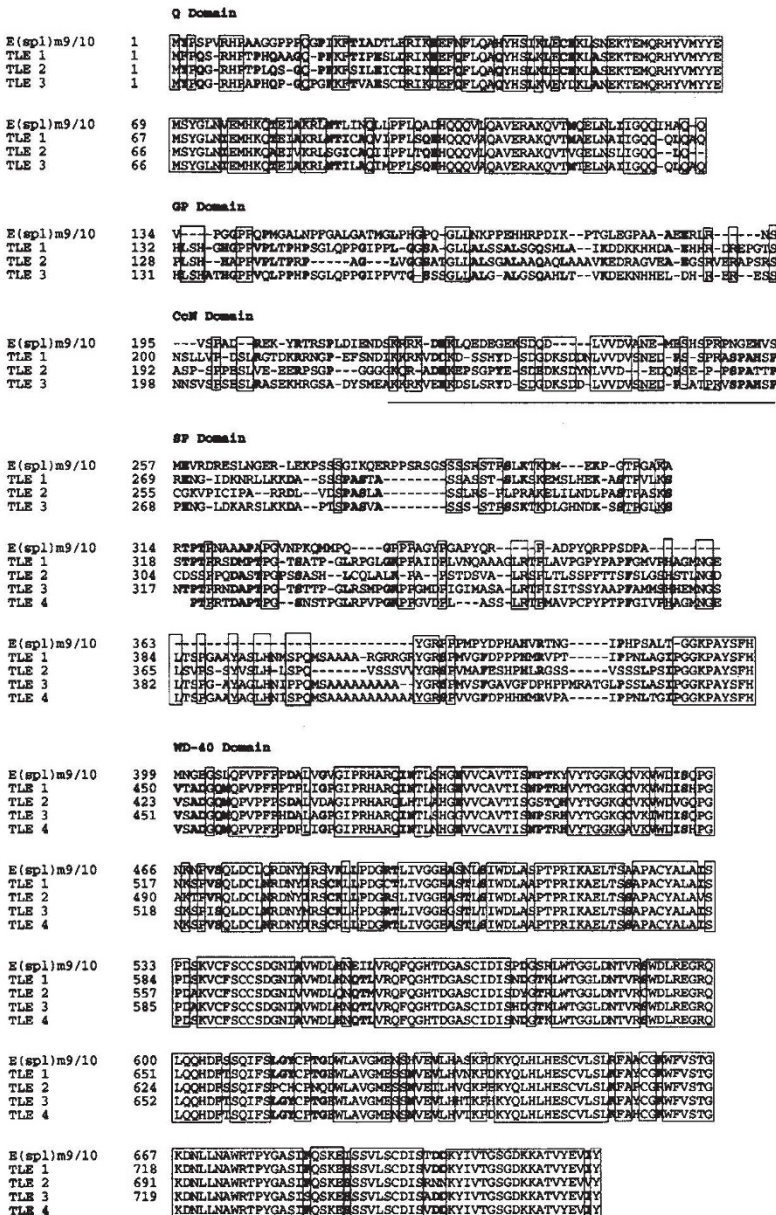


Fig. 1 Comparison of the amino acid sequence of *Drosophila* E(spl) m9/10 and human TLE proteins. Amino acids are numbered on the left side. Identical residues in all compared sequences are boxed, while residues identical in either three out of four or four out of five sequences are indicated in boldface type. Alignments maximize continuity between all sequences. Underlined amino acid residues correspond to the CcN motif (see text). The GenBank accession numbers for the corresponding nucleotide sequences are: TLE 1, M99435; TLE 2, M99436; TLE 3, M99438; TLE 4, M99439.