Fas-activated serine/threonine phosphoprotein (FAST) is a regulator of alternative splicing. [1]

Enviado por Mariano Garcia-Blanco [2] el 9 octubre 2012 - 5:49pm

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Título	Fas-activated serine/threonine phosphoprotein (FAST) is a regulator of alternative splicing.
Publication Type	Journal Article
Year of Publication	2007
Autores	Simarro, M [3], Mauger, D [4], Rhee, K [5], Pujana, MA [6], Kedersha, NL [7], Yamasaki, S [8], Cusick, ME [9], Vidal, M [10], García-Blanco, MA [11], Anderson, P [12]
Journal	Proc Natl Acad Sci U S A
Volume	104
Issue	27
Pagination	11370-5
Date Published	2007 Jul 3
ISSN	0027-8424
Palabras clave	Alternative Splicing [13], Animals [14], Antigens, CD95 [15], Cell Line [16], Cell Nucleus [17], Gene Expression Regulation [18], Humans [19], Mice [20], Protein-Serine-Threonine Kinases [21], Receptor, Fibroblast Growth Factor, Type 2 [22], Yeasts [23]

Abstract	Fas-activated serine/threonine phosphoprotein (FAST) is a survival protein that is tethered to the outer mitochondrial membrane. In cells subjected to environmental stress, FAST moves to stress granules, where it interacts with TIA1 to modulate the process of stress-induced translational silencing. Both FAST and TIA1 are also found in the nucleus, where TIA1 promotes the inclusion of exons flanked by weak splice recognition sites such as exon IIIb of the fibroblast growth factor receptor 2 (FGFR2) mRNA. Two-hybrid interaction screens and biochemical analysis reveal that FAST binds to several alternative and constitutive splicing regulators, suggesting that FAST might participate in this process. The finding that FAST is concentrated at nuclear speckles also supports this contention. We show that FAST, like TIA1, promotes the inclusion of exon IIIb of the FGFR2 mRNA. Both FAST and TIA1 target a U-rich intronic sequence (IAS1) adjacent the 5' splice site of exon IIIb. However, unlike TIA1, FAST does not bind to the IAS1 sequence. Surprisingly, knockdown experiments reveal that FAST and TIA1 act independently of one another to promote the inclusion of exon IIIb. Mutational analysis reveals that FAST-mediated alternative splicing is separable from the survival effects of FAST. Our data reveal that nuclear FAST can regulate the splicing of FGFR2 transcripts.
DOI	10.1073/pnas.0704964104 [24]
Alternate Journal	Proc. Natl. Acad. Sci. U.S.A.
PubMed ID	17592127 [25]

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