

RNAi-mediated PTB depletion leads to enhanced exon definition. ^[1]

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Abstract

Mutually exclusive use of exons IIIb or IIIc in FGF-R2 transcripts requires the silencing of exon IIIb. This repression is mediated by silencer elements upstream and downstream of the exon. Both silencers bind the polypyrimidine tract binding protein (PTB) and PTB binding sites within these elements are required for efficient silencing of exon IIIb. Recruitment of MS2-PTB fusion proteins upstream or downstream of exon IIIb causes repression of this exon. Depletion of endogenous PTB using RNAi increases exon IIIb inclusion in transcripts derived from minigenes and from the endogenous FGF-R2 gene. These data demonstrate that PTB is a negative regulator of exon definition in vivo.

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