MAZ elements alter transcription elongation and silencing of the fibroblast growth factor receptor 2 exon IIIb. [1]

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Abstract	The fibroblast growth factor receptor 2 (FGFR2) gene exons IIIb and IIIc are alternatively spliced in a mutually exclusive and cell type-specific manner. FGFR2 exon choice depends on both activation and silencing. Exon IIIb silencing requires cis-acting elements upstream and downstream of the exon. To examine the influence of transcription on exon IIIb silencing, the putative RNA polymerase II (RNAPII)-pausing MAZ4 element was inserted at different positions within the FGFR2 minigene construct. MAZ4 insertions 5' to the upstream silencing elements or between exon IIIb and downstream silencing elements result in decreased silencing. An insertion 3' of the downstream silencing elements, however, has no effect on splicing. An RT-PCR elongation assay shows that the MAZ4 site in these constructs is likely to be a RNAPII pause site. Insertion of another RNAPII pause site into the minigene has a similar effect on exon IIIb silencing. Transfection of in vitro transcribed RNA demonstrates that the cell type specificity of FGFR2 alternative splicing requires co-transcriptional splicing. Additionally, changing the promoter alters both FGFR2 minigene splicing and the MAZ4 effect. We propose that RNAPII pauses at the MAZ4 elements resulting in a change in the transcription elongation complex that influences alternative splicing decisions downstream.
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