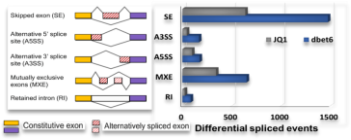
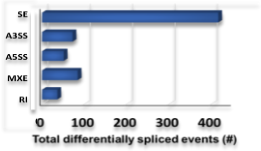
BRD4 a transcription factor and druggable cancer target has been characterized for its role in alternate splicing (protein diversity) in normal and cancer cells.

The nuclear genes encoding proteins are interrupted by intervening sequences called introns in the eukaryotes. Therefore, the nascent RNAs transcribed from these coding units contain both exons and introns and undergo through various processing steps before they are used for protein synthesis. On the other hand, the number of proteins present in eukaryotic systems is much more than the number of these encoding units and that is suggested through alternate splicing at mRNA level. Unlike normal cells, the cancer cells make a large number of variants of certain proteins that have roles in cancer development. We identified a protein BRD4 a transcription factor and epigenetic regulator and showed that BRD4 regulates alternate splicing in cancer cells different from normal cells.

❑ **BRD4 regulates alternative splicing in mice thymocytes and AML cells**

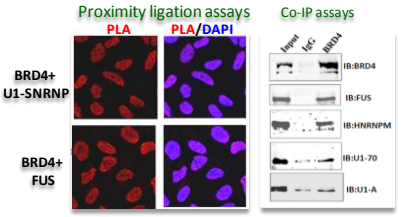


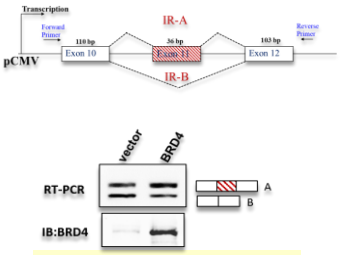
Bromodomain degrader small molecule (dBET6)/ Bromodomain inhibitor (JQ1) treatment causes differential splicing in Acute Myeloid Leukemia (AML) cancer cells



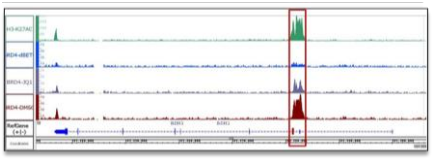
BRD4 deficiency leads to differential splicing in Mice thymocytes

❑ **BRD4 interacts with spliceosome machinery**

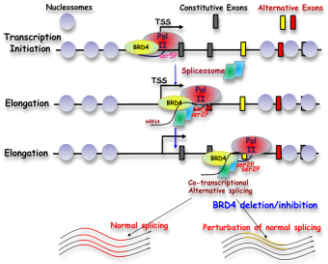




BRD4 regulates alternative splicing of a mini gene in Hela cells



BRD4-CHIP shows BRD4 peaks at the alternative exons



A model showing BRD4 role in alternative splicing during transcription elongation