Special Issue

Aberrant Pre-mRNA Splicing in Disease

Message from the Guest Editor

After the discovery of pre-mRNA splicing in the late 1970s, it became apparent that exons can be spliced together in different ways: in other words, pre-mRNA is alternatively spliced. The extent of alternative splicing in different species is remarkable; indeed, in humans, it is now thought that over 94% of our genes are alternatively spliced. Genes can even express dozens, if not hundreds of splice isoforms; alternative splicing is a major contributor to proteomic complexity. Alternative splicing affects all parts of mRNAs; not only the open reading frame altering the amino-acid sequence, but also the 5' and 3' UTRs influencing mRNA translation, localization and stability. Splice isoforms often encode functionally distinct proteins. Mutations that disrupt normal pre-mRNA splicing—as many as one in six mutations in humans—are associated with a wide range of diseases. The purpose of this Special Issue is to illustrate the growing prominence of alternative splicing in biomedical research.

Guest Editor

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