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Nucleotide sequences and their uses.

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Within a gene, introns and exons are nucleotide sequences. Exons are chemically bound to one another in order to form mature mRNA, whereas introns are deleted by RNA splicing as RNA matures, meaning they are not expressed in the final messenger RNA (mRNA) product. Exons are expressed sequences, whereas introns are intervening sequences. Each human gene has an average of 8.8 exons and 7.8 introns.

Exons

Exons are conserved nucleotide sequences in DNA and RNA that are used to make mature RNA. The process of using DNA as a template to make mRNA is known as transcription.

In a process known as translation, mRNA collaborates with ribosomes and transfer RNA (tRNA), both of which are found in the cytoplasm, to produce proteins.

In addition to any protein coding sequences, exons normally include both the 5' and 3' untranslated regions of mRNA, which contain start and stop codons.

Introns

Introns are nucleotide regions in DNA and RNA that do not code for proteins and are deleted by RNA splicing during the precursor messenger RNA (pre-mRNA) stage of mRNA maturation.

Introns are found in a wide variety of genes that create RNA in most living species, including viruses, and can range in size from tens to thousands of base pairs.

Introns can be classified into four different types:

1. Spliceosomes delete introns from protein-coding genes.

2. Proteins that delete introns from tRNA genes

3. Self-splicing introns, which use guanosine-5'-triphosphate (GTP) or another nucleotide cofactor to remove themselves from mRNA, tRNA, and rRNA precursors (Group 1)

4. Self-splicing introns, which can remove themselves without the use of GTP (Group 2)

It's critical to delete the introns accurately, as any leftover intron nucleotides or exon nucleotide deletion could result in a defective protein being created. This is because codons, which are made up of three nucleotides, are used to link the amino acids that make up proteins. As a result, an erroneous intron removal could cause a frameshift, causing the genetic code to be read wrongly.

RNA Splicing

RNA splicing is the process of converting pre-mRNA into mature mRNA by removing introns and connecting exons together. Splicing can be done in a variety of ways, depending on the organism, the type of RNA or intron structure, and whether or not catalysts are present.

The donor site is a highly conserved GU sequence at the 5' end of an intron, whereas the acceptor site is a highly conserved AG sequence at the 3' end. The spliceosome, a large RNA-protein complex made up of five small nuclear ribonucleoproteins (snRNPs), uses these sites to recognise the start and end points of the intron and catalyse the removal of the intron. The intron is formed into a loop that can be easily cleaved by the spliceosome, and the leftover RNA on each side of the intron is linked. Minor spliceosomes are other forms of spliceosomes that recognise uncommon or altered intron sequences.

Splicing of tRNA is much less common, but it does happen in all three major domains of life: bacteria, archaea, and eukaryote. snRNPs are replaced by many enzymes in a step-by-step process that varies greatly between organisms.

Self-splicing introns are most commonly seen in RNA molecules called ribozymes, which are designed to catalyse biological reactions. A nucleotide cofactor, which may be free in the biological milieu or a part of the intron itself, attacks Group 1 introns at the 5' splice site, causing the neighbouring exon's 3'OH to become nucleophilic and so link to the 5' end of another exon, causing the intron to loop. Splicing of Group 2 introns is identical to that of Group 1 introns, but with the addition of a particular adenosine that assaults the 5' splice site.

Alternative Splicing

Alternative splicing describes how distinct exon combinations can be linked together to produce a single gene that codes for several proteins. Walter Gilbert was the first to suggest this theory, claiming that different exon permutations could result in different protein isoforms. These would then have a variety of chemical and biological actions.

Between 30 and 60 per cent of human genes are known to undergo alternative splicing. Furthermore, splice faults, rather than coding sequence defects, account for more than 60% of disease-causing mutations in humans.

Fibronectin, a glycoprotein that extends from the cell into the extracellular matrix, is an example of a human gene that undergoes alternative splicing. Fibronectin has been identified in approximately 20 distinct isoforms. All of these were made using different combinations of fibronectin gene exons.

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