Coding and non-coding RNA polymerases.

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Description

The discovery of small peptides encoded by long non-coding RNAs (lncRNAs) as well as regulatory functions mediated by mRNA non-coding regions has complicated the understanding of RNA's multifaceted functions. Long non-coding RNAs have the ability to regulate gene expression through a variety of mechanisms, including epigenetic, transcriptional, post-transcriptional, translational, and protein location effects. The modes of action of lncRNAs are as diverse as their functional diversity. lncRNAs can recruit epigenetic factors to modify chromatin state, assemble transcriptional machinery to initiate transcription, or act as a structural organizer to help form subcellular organelles. The size of the long class of ncRNAs ranges from about 200 nt to more than 100 kb. While they appear to be mostly transcribed by RNA polymerase II (although an example of a Pol III transcript is mentioned later), many long ncRNAs avoid, or at least do not undergo, the subsequent standard mRNA processing steps, a feature intimately linked to their regulatory functions, which frequently entail nuclear retention close to the sites of transcription.

These include lncRNAs transcribed primarily by RNA polymerase II (Pol II), but also by other RNA polymerases, as well as lncRNAs from intergenic regions (lincRNAs) and sense or antisense transcripts that overlap with other genes. The resulting lncRNAs are frequently capped at their 5′ ends by 7-methylguanosine (m7G), polyadenylated at their 3′ ends, and spliced similarly to mRNAs. It's worth noting that enhancer and promoter regions are both transcribed into enhancer RNAs (eRNAs) and promoter upstream transcripts. Growing evidence links ncRNA misregulation to human diseases, diagnostic and therapeutic applications. In the small ncRNA category, miRNAs have demonstrated clinical utility for diagnostic testing with the development of an mRNA signature blood test (miR-Test) for lung cancer screening. MiRNAs are also valuable markers for classifying cancer tumors and tissues of origin, which is critical for selecting the best treatment. Researchers used miRNA-based assays to confidently identify 42 tumour types. MiRNA expression levels have also been linked to treatment response in various cancers, implying that miRNAs can be used to detect resistance or other suboptimal treatment outcomes.

Over the last decade, studies have demonstrated the utility of miRNAs and lncRNAs in cancer diagnosis, such as using NGS sequencing to measure the expression of 10 miRNAs involved in thyroid cancer. To detect cancer in thyroid nodules, this information is combined with sequencing data from a thyroid oncogene panel. Although lncRNAs are not as well understood as miRNAs, reports of diagnostic applications are available. Overexpression of the prostate-specific lncRNA PCA3 has been detected in prostate cancer cells, and PCA3 urine assays are widely used in the clinic. Such lncRNAs appear to be weakly co-transcriptionally spliced, and transcription termination at these genes appears to be independent of polyadenylation signals, resulting in the temporal accumulation of lncRNAs on chromatin and rapid degradation by the RNA exosome. Because lncRNAs are frequently nuclear, it appears that functional lncRNAs must avoid nuclear surveillance in order to accumulate at high levels in specific cell types. However, the nuclear surveillance process may not always target chromatin-tethered lncRNAs. Some chromatin-localized lncRNAs have a high number of U1 small nuclear RNA binding sites, which recruit the U1 small nuclear ribonucleoprotein (U1 snRNP) to transcriptionally active Pol II, resulting in the tethering of many non-coding RNAs to chromatin. When the function of the Pol II-associated elongation factor SPT6 is disrupted, certain lncRNAs can accumulate on chromatin. SPT6 deficiency causes histone H3 trimethylated at *Lys36* (*H3K36me3*, a marker of active transcription) to be redistributed from protein-coding genes to lncRNA genes, increasing their transcription. Concurrently, SPT6 deficiency impairs transcription termination and Integrator complex recruitment to chromatin, resulting in the accumulation of long non-coding transcripts on chromatin in the form of DNA damage-associated R-loops.

Conclusion

In general, lncRNAs splice less efficiently than mRNAs. They have weaker internal splicing signals and longer distances between the 3′ splice site and the branch point, both of which are associated with augmented nuclear retention. Other factors, such as differential splicing regulator expression, also contribute to the accumulation of lncRNAs in the nucleus. For example, the highly expressed splicing inhibitor peptidyl prolyl isomerase E suppresses the splicing of a subset of lncRNAs in mouse embryonic stem cells (mESCs), resulting in significant nuclear accumulation of many lncRNAs in mESCs.

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