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Perspective

miRNAs as Key Modulators in Cell Biology

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DESCRIPTION

Over the past decade, miRNAs have been identified as key modulators of post-transcriptional gene silencing in mammals. Numerous studies have recently been published on the role of miRNAs in immune cell development and function, and several excellent reviews suggest that miRNAs represent a new level of control over cellular physiology. T cells that express the CD8 coreceptor and recognize peptide–MHC class I complexes have a key role in the control of numerous viral or intracellular bacterial infections. After acute infection, $CD8^+$ T cells rapidly mount a potent defense against pathogens and directly lyse infected cells. Most miRNAs expressed in a specific cell type have the ability to prevent gene expression that is inappropriate for that cell type, effectively deepening pre-existing differentiation programs. It is increasingly recognized that miRNAs directly regulate the levels of many regulatory proteins required for immune cell development and their peripheral responses in the thymus.

Specifically, in animals, miRNA genes are transcribed into pri-miRNA transcripts and processed in the nucleus by the RNase III enzyme complex Drosha and the microprocessor complex subunit DiGeorge syndrome critical region8 (DGCR8). The resulting pre-miRNA is exported to the cytoplasm by exportin-5. Pre-miRNAs are further processed by the RNase III enzyme Dicer and incorporated into miRNA-induced silencing complexes containing-argonautes that target mRNAs for regulation by different mechanisms, such as mRNA de-enylation and translational repression. This ultimately leads to mRNA decapping and degradation, thereby shutting down protein expression of the target gene. Interestingly, regulation of miRNA signaling pathways is not one-way, it is a two-way street pathway. Numerous base-pair interactions have been shown to limit miRNA recycling in human cells. This is caused by the addition of a non-templated uridine to the 3′ end of the miRNA. However, studies in *Caenorhabditis elegans* have revealed a model of target-dependent miRNA protection in which pairing with partially complementary target mRNAs stabilizes mature miRNAs. The explanation for this discrepancy is still unknown. Nonetheless, these data demonstrate a link between the degree of complementation and the effect of target on miRNA stability.

miRNAs provide specificity through complementary base-pairing with their target mRNAs. Recently, genetic, computational, and biochemical approaches have been applied to identify miRNA targets. Genetic approaches are based on the detection of gene deletions or conditional ablation that result in partial or complete rescue of mutant phenotypes caused by loss of specific miRNAs. Based on algorithms, computational approaches such as PicTar, miRanda, and TargetScan identify miRNA targets by requiring a conserved Watson-Crick pairing with the miRNA's 5' region. This criterion is intended to reduce false positive rates and improve sensitivity and overall accuracy. A drawback of these methods is that they may not identify the most biologically important miRNA targets. Biochemical methods such as cross-linking immunoprecipitation and high-throughput sequencing of RNA isolated by caged ribonucleoside-enhanced cross-linking and immunoprecipitation provide accurate sequences for targeting clinically relevant miRNA-mRNA interactions. Further work is needed to confirm whether the predicted target mRNAs are indeed regulated.

Robust data from *in vitro* and *in vivo* models are now accumulating, supporting critical roles for miRNAs as regulators of $CD8^+$ T cell development, proliferation, survival, migration, differentiation, and effector function. During these processes, miRNAs and signaling molecule 1 reciprocally regulate in feedback loops to fine-tune the amount of translatable mRNA in the face of different environmental cues.

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