INNOVATIONS IN MOLECULAR BIOTECHNOLOGY
Jul-Dec 2024, VOL. 2, ISSUE 2, pp. 12 - 17
https://doi.org/10.61577/imb.2024.100008

RESEAPRO JOURNALS

MINI REVIEW

OPEN ACCESS

Emerging roles of RNA editing in cellular function and therapeutics

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ABSTRACT

RNA editing comprises of post-transcriptional modification that proposes site-specific nucleotide changes into RNA molecules, increasing transcriptomic and proteomic diversity well beyond the information encoded within the genome. The two principal biochemical mechanisms; adenosine-to-inosine (A-to-I) editing catalyzed by the adenosine deaminase acting on RNA (ADAR) family, and cytidine-to-uridine (C-to-U) editing mediated by the apolipoprotein B mRNA editing catalytic polypeptide-like (APOBEC) family together control a broad spectrum of physiological and pathological procedures. The objective of this review is to combine present evidence on the multifaceted roles of RNA editing across cellular contexts and to critically assess its translational potential within the therapeutic landscape. ADAR1 safeguards cellular homeostasis by avoiding irregular activation of the innate immune sensors MDA5 and PKR by endogenous double-stranded RNA (dsRNA), while de-regulation of ADAR-mediated editing has been implicated in tumorigenesis, viral pathogenesis, and neurological dysfunction. C-to-U editors of the AID/APOBEC family are majorly involved in innate and adaptive immunity, contributing to antibody diversification and antiviral restriction, and linked with cancer progression and neurological disease. On the therapeutic front, programmable A-to-I RNA editing platforms which include LEAPER, RESTORE, and antisense oligonucleotide-based approaches have emerged as powerful tools for rectifying disease-causing mutations at the transcript level, offering reversible and transient pharmacodynamic effects that avoid the permanence and heritability risks linked with genomic interventions. Some oligonucleotide-based RNA editing therapeutics are currently advancing through clinical trials, with Wave Life Sciences' WVE-006 representing an example of first-in-human ADAR-based programmable editing. Nevertheless, critical limitations persist off-target transcriptome-wide editing events, differential ADAR isoform expression across tissues, and the immunogenic risks linked with long-term interference with endogenous ADAR activity remain unresolved challenges that require both mechanistic understanding and clinical translation. Together, this review underscores RNA editing as an essential layer of epitranscriptomic regulation and positions programmable editing platforms as a compelling, yet still maturing, in precision medicine.

KEY WORDS

RNA editing; A-to-I editing; ADAR; C-to-U editing; APOBEC; Epitranscriptomics; programmable RNA therapeutics; site-directed RNA editing

ARTICLE HISTORY

Received 06 November 2024;
Revised 27 November 2024;
Accepted 09 December 2024

Introduction

The modulation of gene expression in eukaryotic cells does not terminate at the level of transcription; it is subject to an immense layer of post-transcriptional control that modulates the informational content of RNA molecules before and during translation. Between various mechanisms that operate at this post-transcriptional level, RNA editing shows a biochemically distinct and biologically significant process through which the nucleotide sequence of an RNA transcript is changed by site-specific enzymatic modification, independent of the underlying genomic template. Over 170 types of RNA modifications have been catalogued to date; though, only a subset lead to base pair substitutions that constitute canonical RNA editing events, the two most prevalent of which are A-to-I editing catalyzed by the adenosine deaminase acting on RNA (ADAR) family, and C-to-U editing mediated by the apolipoprotein B mRNA editing catalytic polypeptide-like (APOBEC) family [1,2]. These modifications extend proteomic and transcriptomic diversity far beyond the informational capacity encoded within the genome alone, showing a fundamental mechanism through which biological complexity is initiated from a finite genomic template.

The modulation of gene expression in eukaryotic cells does not terminate at the level of transcription; it is subject to an immense layer of post-transcriptional control that modulates the informational content of RNA molecules before and during translation. Between various mechanisms that operate at this post-transcriptional level, RNA editing shows a biochemically distinct and biologically significant process through which the nucleotide sequence of an RNA transcript is changed by site-specific enzymatic modification, independent of the underlying genomic template. Over 170 types of RNA modifications have been catalogued to date; though, only a subset lead to base pair substitutions that constitute canonical RNA editing events, the two most prevalent of which are A-to-I editing catalyzed by the adenosine deaminase acting on RNA (ADAR) family, and C-to-U editing mediated by the apolipoprotein B mRNA editing catalytic polypeptide-like (APOBEC) family [1,2]. These modifications extend proteomic and transcriptomic diversity far beyond the informational capacity encoded within the genome alone, showing a fundamental mechanism through which biological complexity is initiated from a finite genomic template.

In recent years, the application of high-throughput RNA sequencing technologies combined with advanced bioinformatics pipelines has enabled transcriptome-wide mapping of RNA editing sites at an unmatched resolution. Genome-wide profiling has revealed an extensive number of differentially edited loci across biological and pathological contexts, associating these post-transcriptional events in the regulation of immunity, viral infection, neurological function,

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metabolic homeostasis, and tumorigenesis. Especially, ADAR-mediated A-to-I RNA editing has been identified as a critical post-transcriptional event that suppresses cellular dsRNA-mediated innate immune interferon responses, and large-scale characterization of cis-RNA editing quantitative trait loci (edQTLs) across human tissues has revealed its mechanistic role in the genetic underpinning of common inflammatory diseases [5]. These discoveries have fundamentally repositioned RNA editing from biochemical curiosity to a central regulatory node within the epitranscriptomic landscape.

RNA editing is now acknowledged as a dominant type of RNA modification in mammals, with inosine mimicking guanosine in base pairing to alter dsRNA secondary structure, thereby prompting splicing, microRNA processing and targeting, and mRNA stability across virtually all RNA species, which includes pre-mRNA, mature mRNA, lncRNA, miRNA, and tRNA. C-to-U editors of the AID/APOBEC family are also implicated in innate and adaptive immunity, antibody diversification, and antiviral responses, with each family member exhibiting tissue-specific expression and different subcellular localization that govern their substrate accessibility and functional output [6]. Together, these two deaminase systems create a comprehensive, dynamically regulated epitranscriptomic code whose perturbation at any level enzyme expression, cofactor availability, or substrate architecture carries significant pathophysiological consequences.

The link between dysregulated RNA editing and human disease has been systematically documented across multiple pathological domains. Aberrant RNA modifications, which include A-to-I editing, have been demonstrated to influence the stability, translation, and localization of disease-related mRNAs, and their dysregulation is carefully linked with the onset and progression of cancer, neurological disorders, cardiovascular diseases, and immune conditions [7]. Within the neuro-oncological domain, ADAR1 upregulation has been reported to help glioblastoma stem cell self-renewal through JAK/STAT pathway activation, while changed ADAR2 activity in motor neurons causes the excitotoxic pathophysiology observed in amyotrophic lateral sclerosis through impaired GluA2 Q/R site editing. In the context of tumour immunology, ADAR1 overexpression and rise dsRNA editing have been known across multiple human cancers, and silencing ADAR1 in tumours refractory to immune checkpoint inhibitors has been offered as a capable combinatorial immunotherapeutic strategy [8].

On the therapeutic front, the reversibility and transcriptome-confined nature of RNA editing have positioned it as a convincing substitute for permanent genome-editing strategies. Programmable A-to-I RNA editing platforms, including LEAPER and RESTORE, leverage engineered antisense oligonucleotides or guide RNAs to recruit endogenous ADAR enzymes to target transcripts, enabling sequence-specific recoding without permanent genomic alteration, and have verified proof-of-concept correction of premature stop codons in disease-relevant transcripts. RNA base editing has since been applied in both biological research and therapeutic contexts, enabling site-directed editing within target transcripts with reversible and dose-dependent effects key advantages over permanent or heritable changes associated with DNA base editing [9].

Despite major advances, the existing body of literature is restrained by few substantive gaps. Most mechanistic studies on RNA editing have been shown in cell line models or rodent systems, which inadequately recapitulate the ADAR isoform expression landscape of human tissues, especially the central nervous system. Primary among the unresolved challenges is ADAR recruitment and editing sensitivity in tissues with low endogenous ADAR expression, and the risk of compounding biological effects when ADAR abundance or activity is improved either locally or systemically [10]. Additionally, the transcriptome-wide off-target editing profiles of current therapeutic platforms stay incompletely characterized, and the durability of editing-based therapeutic effects in long-lived, post-mitotic cell populations has not been rigorously well-known in vivo. A comparative limitation of endogenous ADAR-recruiting strategies relative to exogenous ADAR delivery is the relatively low editing efficiency achieved at target sites, which restricts their utility in biotechnological and therapeutic applications requiring high and sustained levels of recoding [11].

Against this background, the current mini review aims to comprehensively synthesize present evidence on the emerging roles of RNA editing in cellular function and to critically evaluate the state of its therapeutic exploitation, with the dual purpose of delineating the mechanistic intersections among the editome and human disease and of identifying the key biological and technical barriers that resolved to realize the full clinical potential of programmable RNA editing platforms.

Mechanisms of RNA Editing

RNA editing encompasses two main biochemical modalities in mammals that are governed by specific enzymatic machinery and substrate recognition principles: A-to-I editing mediated by the adenosine deaminase acting on RNA (ADAR) family, and C-to-U editing catalyzed by members of the apolipoprotein B mRNA editing catalytic polypeptide-like (APOBEC) family [12].

A-to-I editing is the most common form of RNA editing in the human transcriptome, regulating the activity of three members of the ADAR family ADAR1, ADAR2 and the catalytically inactive ADAR3. According to research, ADAR1 is expressed in all cell types, ADAR2 is abundant in most cell types in the brain, whereas ADAR3 expression is restricted to the central nervous system only. ADARs that are catalytically active have a similar composition at the structural level. They consist of an N-terminal double-stranded RNA-binding domain (dsRBD) that is conserved and a deaminase domain which is at the C-terminus [13]. Further, ADAR1 also possesses binding domains for Z-DNA/ZRNA. These are Zα and Zβ. This allows ADAR1 to recognize left-handed nucleic acid conformations. ADARs use a base-flipping mechanism at a catalytic level. The adenosine is extruded from the dsRNA helix into the active site. The active site possesses a zinc ion coordinated by two cysteine residues and one histidine. A water molecule acts as the fourth ligand. This water molecule attacks the C6 position of adenine. It releases the 6-amino group and then base imino group generates inosine. Cell Press ADARs display characteristic sequence preferences at the editing site, with both ADAR1 and ADAR2 preferring a 5′ uridine neighbour. Meanwhile, ADAR2 exhibits a unique preference of 5′-U ≈ A > C = G. Since inosine is structurally similar to guanosine, it is decoded as guanosine by the translational and splicing machinery, effectively producing an A-to-G substitution at the functional level. More than ninety-nine percent of ADAR-mediated deamination events occur at multiple clustered sites within complementary Alu repeat sequences, which destabilize the dsRNA duplex through the introduction of A-to-I mismatches [14].

APOBEC1 is a zinc-dependent cytidine deaminase and mediates C-to-U editing as part of a multi-protein editosome complex. In 1993, the enzyme that deaminated a C at position 6666 of ApoB mRNA was identified as APOBEC1. This deamination turned a CAA glutamine codon into a UAA stop codon, yielding the smaller APOB-48 isoform with intestinal lipid

https://doi.org/10.61577/imb.2024.100008

transport functions. The editosome requires the assembly process for APOBEC1-dependent substrate recognition. RNA-binding cofactor A1CF (APOBEC1 Complementation Factor) identifies an 11-nucleotide mooring sequence downstream of the edited cytidine. This process takes place in AU-rich context and places the catalytic subunit over its target [15]. More recently, a new RNA-binding protein, RBM47 was found to be a core editosome component capable of substituting for A1CF. Rbm47-deficient mice show a significant impairment C-to-U editing activity. APOBEC1 edit mRNA with C-to-U edits have been shown to be present in mRNA sequences beyond the canonical ApoB substrate. Recent sequencing using a broader transcriptome-wide target space has illustrated that ApoB mRNA 3′ UTRs show Editing in AU-rich sequences addition to the potential of APOBEC1 to edit other mRNAs. APOBEC3A has also been shown to catalyze site-specific C-to-U RNA editing in monocytes and macrophages during M1 polarization and in hypoxic conditions. Such activity alters the amino acid sequences of proteins that regulate antiviral pathogenesis. Thus, C-to-U editing activity is not restricted to intestinal tissue [16].

Together, these deaminase-mediated editing mechanisms constitute the biochemical foundation upon which the diverse cellular and pathophysiological consequences of RNA editing elaborated in subsequent sections are ultimately grounded.

Emerging Roles in Cellular Function

RNA editing has appeared as an indispensable post-transcriptional regulatory mechanism that operates across a broad spectrum of cellular processes, with its functional consequences extending well beyond the canonical concept of simple nucleotide recoding. The delineation of these roles has been substantially accelerated by transcriptome-wide sequencing technologies, genetic loss-of-function models, and quantitative editome profiling across human tissues [17].

Innate immunity and self/non-self-discrimination represent perhaps the most crucial emerging function of ADAR1-mediated editing. Genome-wide analysis of the in vivo substrates of ADAR1 has established that the enzyme catalyzes clustered hyperediting within long dsRNA stem loops in the 3′ untranslated regions of endogenous transcripts, and that abrogation of this editing activity in Adar1^E861A/E861A^ mice results in embryonic death attended by broad activation of interferon and dsRNA-sensing pathways a phenotype fully saved by simultaneous deletion of the cytosolic dsRNA sensor MDA5. Mechanistically, in cells lacking ADAR1, unedited endogenous RNAs activate multiple RNA sensors including MDA5, protein kinase R (PKR), oligoadenylate synthase (OAS), and Z-RNA binding protein 1 (ZBP1), collectively inducing a type I interferon response and inflammatory cell death. The cytoplasmic ADAR1 p150 isoform, which is interferon-inducible, plays the critical role in this context by undermining and reshaping cytosolic dsRNA structures to interfere with MDA5 filament formation, thereby calibrating the innate immune response and preventing autoimmunity [18].

Neuronal function and synaptic homeostasis symbolize a second domain of established biological importance. ADAR2-mediated A-to-I editing at the Q/R site of the GluA2 subunit of α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptors (AMPARs) recodes a genomically encoded glutamine codon to arginine, rendering the assembled receptor Ca^{2+}-impermeable under physiological conditions. Activity deprivation in cortical neurons results in a rapid and significant enlarge in unedited GluA2 expression mediated by a splicing factor-dependent suppression of nuclear ADAR2 abundance leading to increased incorporation of Ca^{2+}-permeable AMPARs at the synapse and donating to homeostatic synaptic upscaling. Beyond GluA2, editing of the serotonin 2C receptor (5-HT_2CR) pre-mRNA at up to five distinct sites by ADAR1 and ADAR2 generates combinatorial receptor isoforms with altered G-protein coupling efficiency and serotonin sensitivity, with altered editing profiles at this locus documented in association with psychiatric disorders [19]. Notably, GluA2 Q/R site editing occurs with approximately 99% efficiency in the healthy brain, and its impairment in Alzheimer's disease models has been causally linked to increased AMPA receptor calcium permeability, dendritic spine loss, synaptic degeneration, and cognitive impairment.

Cancer biology has appeared as a third domain of considerable relevance. ADAR1-mediated RNA editing dysregulation happening as a hyperediting phenomenon has been documented across multiple cancer types including liver, lung, breast, and esophageal cancers, in most cases promoting tumour progression through abnormal editing of substrates that regulate proliferative, survival, and immunomodulatory pathways. Concurrently, ADAR1 acts as a gatekeeper of the RNA-sensing pathway in tumour cells, and its overexpression enables malignant cells to silence endogenous immunogenic dsRNAs, thereby evading MDA5- and PKR-mediated immune surveillance a mechanism that confers resistance to immune checkpoint inhibitor therapies. Taken together, these examination position RNA editing not as a peripheral regulatory modifier, but as a core determinant of cellular identity, immune competence, neural circuit fidelity, and oncogenic potential [20].

RNA Editing in Disease

RNA editing is a post-transcriptional regulatory process that varies the transcriptome through nucleotide modifications without altering genomic DNA. The most common forms in mammals include A-to-I editing catalyzed by ADAR (adenosine deaminase acting on RNA) enzymes and C-to-U editing mediated by APOBEC family proteins. These alterations influence RNA stability, splicing, localization, and translational efficiency. Growing transcriptomic evidence demonstrates that dysregulation of RNA editing contributes to diverse pathological conditions including neurological disorders, autoimmune syndromes, cancer, metabolic diseases, and viral infections [21].

Neurological disorders

RNA editing plays a pivotal role in supporting neuronal homeostasis by regulating neurotransmission and synaptic plasticity. Reduced ADAR2 activity is strongly associated with amyotrophic lateral sclerosis (ALS), where deficient editing of the GluA2 subunit of AMPA receptors at the Q/R site results in increased Ca^{2+} permeability and excitotoxic neuronal degradation. Similarly, aberrant editing of ion channel transcripts has been noticed in epilepsy, influencing neuronal excitability and seizure susceptibility. In Alzheimer's disease, transcriptome-wide alterations in A-to-I editing patterns have been reported, suggesting involvement in neuroinflammation, synaptic dysfunction, and cognitive decline. These findings jointly indicate that precise RNA editing is crucial for neuronal signaling fidelity and neuroprotection [22].

Autoimmune disorders

ADAR1-mediated RNA editing is critical for preventing unsuitable activation of innate immune pathways. Loss-of-function mutations in ADAR1 are associated with Aicardi–Goutières syndrome (AGS), a severe autoimmune interferonopathy characterized by excessive type I interferon signaling. ADAR1 normally edits endogenous dsRNA, preventing recognition by cytosolic immune receptors such as MDA5.

https://doi.org/10.61577/imb.2024.100008

Failure of editing leads to accumulation of immunogenic dsRNA molecules, triggering chronic inflammatory responses and tissue damage. Thus, RNA editing functions as a molecular mechanism distinguishing self RNA from viral RNA, maintaining immune acceptance [23].

Cancer

RNA editing contributes to cancer growth by modulating gene expression, protein diversity, and microRNA targeting efficiency. Enlargement and overproduction of ADAR genes have been documented in several tumor types including breast cancer, hepatocellular carcinoma, glioblastoma, and lung cancer. Editing-induced recoding events may alter oncogenic signaling pathways, Moving cell proliferation, apoptosis, and metastasis. Transcriptome-wide studies demonstrate that RNA editing signatures can serve as diagnostic and prognostic biomarkers, reflecting tumor heterogeneity and therapeutic responsiveness. Consequently, RNA editing profiles are being explored as potential tools in precision oncology and targeted therapy development [24].

Metabolic diseases

C-to-U RNA editing regulates lipid metabolism through alteration of apolipoprotein B (ApoB) transcripts. APOBEC1-mediated editing introduces a premature stop codon in ApoB mRNA, producing two isoforms: ApoB100 synthesized in the liver and ApoB48 synthesized in the intestine. ApoB48 facilitates chylomicron formation and intestinal lipid transport, playing a central role in dietary lipid absorption. Dysregulation of ApoB editing has been associated with hyperlipidemia, obesity, and cardiovascular disorders, indicating that RNA editing contributes significantly to metabolic homeostasis [25].

Viral infections

RNA editing also purpose as an antiviral defense mechanism. ADAR enzymes catalyze extensive A-to-I substitutions in viral RNA genomes, generating hypermutation patterns that impair viral replication and protein synthesis. Hyperediting has been reported in multiple RNA viruses including measles virus, hepatitis delta virus, influenza virus, and SARS-CoV-2. However, certain viruses exploit host RNA editing machinery to increase genetic variability and evade immune surveillance. Therefore, RNA editing represents a complex host–pathogen interaction mechanism that may impact viral evolution and disease severity [26].

Therapeutic Applications of RNA Editing

RNA editing has developed as a transformative therapeutic strategy for correcting pathogenic mutations at the transcript level without permanent modification of genomic DNA. Programmable RNA editing platforms allow transient and reversible nucleotide alterations, presenting improved safety compared with DNA-editing technologies. Current progress in engineered ADAR systems, cytidine deaminases, and optimized delivery vehicles has accelerated translational development of RNA editing therapeutics [27].

Engineered ADAR-based Editing

Technologies such as RESTORE, LEAPER, and TRIBE utilize endogenous ADAR enzymes to catalyze site-specific A-to-I conversion. Guide RNAs are created to hybridize with target transcripts, creating dsRNA structures that recruit ADAR proteins. Since inosine is functionally interpreted as guanosine during translation, these ways enable correction of pathogenic G→A mutations with high transcript specificity and minimal genomic risk [28].

SNAP-ADAR and modified guide RNAs

SNAP-ADAR systems use engineered ADAR deaminase fused with SNAP-tag proteins that bind chemically modified guide RNAs. Integration of nucleotide modifications such as 2′-O-methyl and locked nucleic acids improves editing efficiency, enhances structural stability, and reduces off-target deamination, thereby increasing therapeutic precision [29].

C-to-U base editing

C-to-U editing platforms based on APOBEC1-derived cytidine deaminases enable correction of G·C→A·T pathogenic mutations at the RNA level. These C-to-U base editors increase therapeutic scope for nonsense mutations and truncated protein disorders, complementing A-to-I editing approaches [30].

RNA editing vs DNA base editing

RNA editing offers advantages including reversibility, absence of double-strand DNA breaks, and less long-term genotoxic risk. The transient nature of RNA modifications allows dose-dependent therapeutic control and minimizes irreversible off-target mutations, increasing clinical safety [31].

Delivery strategies

Efficient delivery systems include lipid nanoparticles (LNPs), adeno-associated viral vectors (AAV), and antisense oligonucleotide (ASO)-mediated guide RNA delivery. These platforms enable tissue-specific targeting, improved cellular uptake, and increased transcript editing efficiency [32].

Clinical pipeline

RNA editing therapeutics are proceeding through Phase I/II clinical trials, with companies such as Wave Life Sciences, ProQR Therapeutics, and Korro Bio developing treatments for neurological, ophthalmic, and metabolic disorders. Early results indicate favorable safety profiles and promising therapeutic efficacy, supporting RNA editing as a next-generation precision medicine platform.

Challenges and Future Perspective

Despite quick progress, some technical and translational challenges reduce the widespread clinical implementation of RNA editing technologies. One key concern involves off-target editing events within the transcriptome, where unintended adenosine or cytidine modifications can alter protein structure, RNA stability, or regulatory networks. Such off-target effects may lead to unpredictable phenotypic consequences, particularly when editing occurs in highly expressed or functionally essential transcripts. Tissue specificity represents another significant limitation, as capable and targeted in vivo delivery remains difficult. Recent delivery platforms, including lipid nanoparticles (LNPs), viral vectors, and antisense oligonucleotides, often show variable biodistribution, limiting editing efficiency in specific organs such as brain, cardiac tissue, and immune cells. Immunogenicity of editing components, involving engineered ADAR proteins and synthetic guide RNAs, may activate innate immune responses, reducing therapeutic efficacy and safety [33].

Another critical challenge links to the transient nature of RNA editing. While reversibility increases safety relative to permanent DNA modifications, sustained therapeutic benefit may require repeated dosing, increasing cost and potential toxicity. Furthermore, the complete landscape of the editome remains incompletely characterized, particularly in different physiological states, developmental stages, and disease conditions. Limited understanding of RNA structural causes

https://doi.org/10.61577/imb.2024.100008

governing editing efficiency further complicates rational design of editing strategies.

Future research is expected to significantly increase the editome through addition of single-cell transcriptomics and spatial RNA sequencing technologies, enabling precise mapping of cell-type-specific editing patterns. Artificial intelligence and machine learning approaches are gradually being applied to optimize guide RNA design, predict off-target interactions, and improve editing specificity. Combination of therapeutic strategies integrating RNA editing with immunotherapy or gene regulation technologies may improve treatment outcomes in cancer and genetic disorders. In vivo programmable RNA editing offers considerable promise for treatment of monogenic diseases, specifically those caused by point mutations. However, ethical considerations remain essential, particularly for germline-adjacent or heritable editing applications. Comprehensive regulatory frameworks and long-term safety studies will be essential for responsible clinical translation.

Conclusion

RNA editing has emerged as a fundamental post-transcriptional regulatory mechanism that significantly expands transcriptomic and proteomic diversity beyond the static information encoded in genomic DNA. Accumulating evidence demonstrates that site-specific nucleotide modifications, particularly A-to-I and C-to-U conversions, play key roles in regulating gene expression, RNA stability, immune recognition, neural signaling, and metabolic homeostasis. Dysregulation of RNA editing has been strongly linked with numerous pathological conditions, including neurological disorders, cancer, autoimmune syndromes, metabolic diseases, and viral infections, highlighting its broad biological significance and clinical relevance.

Importantly, improvements in programmable RNA editing technologies have positioned the field as a promising therapeutic platform capable of correcting pathogenic mutations in a reversible and controlled manner. Engineered ADAR-based systems, cytidine deaminase-mediated editing tools, and developed delivery strategies have demonstrated potential for precise transcript modification without introducing permanent genomic alterations. Such characteristics offer distinct advantages in terms of safety, specificity, and therapeutic flexibility compared to DNA editing approaches.

Overall, RNA editing research is rapidly evolving toward clinical translation, supported by high-throughput sequencing, artificial intelligence-guided design strategies, and advanced understanding of the editome. Continued interdisciplinary investigation is expected to unlock innovative therapeutic opportunities and establish RNA editing as a key component of next-generation precision medicine.

Disclosure Statement

No potential conflict of interest was reported by the author.

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