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The Therapeutic Potential of METTL3 Inhibition in Modulating N6-Methyladenosine for Chronic Kidney Disease

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Abstract: Chronic kidney disease (CKD) is a progressive disease with limited therapeutic options, necessitating novel approaches for disease management. N6-methyladenosine (m6A) modification has emerged as a key regulator of gene expression in CKD, influencing pathways involved in inflammation, fibrosis, and cellular stress. METTL3 plays a crucial role in modifying RNA, with its overexpression linked to the activation of pro-fibrotic and inflammatory signaling pathways, such as TGF-β1 and NF-κB. Consequently, METTL3 inhibition has been explored as a potential therapeutic strategy for CKD. Preclinical studies suggest that METTL3 inhibitors can attenuate renal fibrosis, reduce inflammation, and restore oxidative stress and apoptosis by modulating m6A-dependent gene expression. However, challenges remain in developing selective and bioavailable METTL3 inhibitors, understanding the context-dependent effects of m6A modifications, and ensuring precise targeting in renal cells. Advances in small-molecule drug discovery, RNA-based therapies, and nanoparticle-mediated delivery may improve the clinical applicability of METTL3 inhibitors. Future research should focus on optimizing these therapeutic strategies to enhance efficacy while minimizing off-target effects. By targeting aberrant m6A methylation, METTL3 inhibition represents a promising avenue for CKD treatment, offering new possibilities for precision medical science.

Keywords: chronic kidney disease, m6A modification, METTL3 inhibitors, TGF-β signaling

1. Introduction

Chronic kidney disease (CKD) is a common global health challenge, affecting millions of people worldwide. It is defined as structural or functional abnormalities in the kidney that persist for more than three months and are associated with significant health implications. CKD often results in end-stage renal disease (ESRD), requiring dialysis or kidney transplantation. It is closely linked to cardiovascular disease, which increases the risk of morbidity and mortality. Despite advancements in nephrology and the development of therapeutic strategies aimed at controlling blood pressure, glucose levels, and proteinuria, effective treatments that specifically target the molecular mechanisms driving CKD progression remain limited [1]. This gap in treatment choices reveals the urgent need for modern approaches that address the underlying epigenetic and molecular pathways contributing to CKD pathogenesis. In recent years, N6-methyladenosine (m6A) methylation has emerged as a critical epigenetic modification regulating gene expression in various biological processes. M6A is also one of the most prevalent internal mRNA modifications in eukaryotic cells.

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It has a crucial role in influencing RNA metabolism, such as mRNA stability, splicing, transport and protein synthesis. Dysregulation of m6A has been increasingly associated with the development and progression of CKD. Studies have shown that it can cause kidney injury by modulating key cellular pathways involved in renal fibrosis and epithelial-to-mesenchymal transition (EMT). Given the fundamental role of these processes in CKD pathophysiology, targeting m6A methylation represents a promising therapeutic strategy for disease progression.

Among the key regulators of m6A methylation, methyltransferase-like 3 (METTL3) plays an essential role in catalyzing the addition of m6A marks onto RNA. METTL3 is part of the m6A methyltransferase complex and is responsible for m6A deposition on mRNA, thereby influencing post-transcriptional gene expression. The overexpression of METTL3 has been found greatly associating with abnormal m6A patterns and promote pathogenic cellular responses. It has also been linked to the activation of pro-fibrotic and inflammatory pathways, particularly transforming growth factor-beta 1 (TGF-β1) signaling, EMT, and nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) activation. These signaling pathways are important contributors to CKD pathogenesis, as they drive fibrosis, inflammation, and cellular dysfunction—hallmarks of progressive kidney disease. Due to the negative side and importance of METTL3 in CKD progression, METTL3 inhibition has emerged as a potential therapeutic strategy to counteract pathologically with m6A modifications. By suppressing METTL3 activity, small-molecule inhibitors have shown promise in pre-clinical studies for lightening fibrosis, reducing inflammation, and restoring cellular homeostasis in kidney tissues. Experimental evidence has demonstrated that METTL3 inhibition can possibly reverse previous negative pathways, including depression of TGF-β1 pathway, prevention of EMT, and NF-κB-mediated inflammatory responses suppression. Additionally, small-molecule inhibitors such as STM2457 ($C_{25}H_{28}N_6O_2$) and other METTL3-targeting compounds have shown encouraging results in experimental models of kidney injury, further highlighting their potential as targeted therapies for CKD [2].

Given the increasing prevalence of CKD and the urgent need for innovative treatment options, exploring METTL3 inhibition as a therapeutic approach presents a promising avenue for improving patient outcomes. Targeting METTL3-mediated m6A methylation could provide a advancing strategy for limiting CKD progression and addressing its underlying molecular benefits. Additionally, METTL3 inhibitors may be incorporated into combination therapies, enhancing the effectiveness of existing CKD treatments such as renin-angiotensin-aldosterone system (RAAS) inhibitors, sodium-glucose cotransporter-2 (SGLT2) inhibitors, and other anti-fibrotic agents [3,4]. By investigating the therapeutic potential of METTL3 inhibitors, this study aims to contribute valuable insights into the development of precision therapies for CKD. Understanding how METTL3-driven m6A methylation contributes to CKD progression may create an easier way for modern treatment strategies that not only slow disease progression but also improve long-term renal outcomes and patient survival.

2. The role of m6A methylation in chronic kidney disease

m6A is the commonest gene expression regulator that modified mRNA post-transcriptionally, especially in eukaryotic mRNA. It is a dynamic and reversible process that controls multiple steps in RNA life cycle, such as mRNA splicing, transcription and translation regulation and mRNA stability [5]. The m6A methylation involves three main steps, each governed by different classes of enzymes: writers, readers, and erasers. These enzymes work together to install, recognize, and remove the m6A modification, ensuring proper gene regulation and cellular function.

The first step is m6A installation/methylation, commonly referred to as the “writing” process. It is catalyzed by the m6A methyltransferase complex (MTC), which is composed of METTL3, methyltransferase-like 14 (METTL14), Wilms’ tumor 1-associated protein (WTAP), and additional

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cofactors such as vir like m6A methyltransferase associated protein (VIRMA) and RNA binding motif protein 15 (RBM15). METTL3 acts as the major catalytic enzyme, while METTL14 functions as a structural assistant that enhances METTL3 activity. WTAP is not involved in catalytic activities itself. Instead, it plays an essential role in stabilizing the METTL3-METTL14 complex and ensuring their proper position inside nucleus during m6A methylation. MTC specifically targets DRACH motifs (where D = A/G/U nuclear bases, R = A/G, and H = A/C/U) in mRNA sequences, ensuring precise methylation at the N6 position of adenosine residues. This methylation reaction utilizes S-adenosylmethionine (SAM) as a methyl donor, transferring a methyl group to the target site [5].

The second step, m6A recognition, determines the fate of m6A-modified mRNA. Reader proteins will bind to m6A residues and regulate mRNA stability and translation, or allow degradation. There are two major categories of m6A readers: YTH domain family proteins (YTHDF1, YTHDF2, YTHDF3) and insulin-like growth factor 2 mRNA-binding proteins (IGF2BP1, IGF2BP2, IGF2BP3). YTHDF1 enhances translation efficiency by recruiting ribosomes, whereas YTHDF2 promotes rapid mRNA degradation by directing transcripts to process mRNA breakdown. Interestingly, YTHDF3 acts as a co-regulator for both YTHDF1 and YTHDF2. It can choose to assist either one of them depending on the cellular context. Meanwhile, all 3 IGF2BP proteins function as stabilizers. They bind to m6A-modified mRNAs and protect them from degradation, which can also prolong their life-time and increase protein production. This complicated balance between stabilization and degradation is crucial for fine-tuning gene expression and cellular homeostasis.

The final step involves m6A demethylation, known as the "erasing" process. This step is carried out by two demethylases: fat mass and obesity-associated protein (FTO) and AlkB homolog 5 (ALKBH5). These enzymes actively remove the methyl group from m6A residues, restoring the mRNA to its unmethylated state. This demethylation step allows for a new cycle of m6A modifications, ensuring dynamic control over RNA metabolism and cellular signaling pathways [2,6].

Although the mechanism and progress of m6A towards CKD seemed complete in detail, there are still challenges for m6A-targeted therapies. For example, the context-dependent effects of m6A are complicated by the exhibition of both protective and harmful modifications at the same time, due to different CKD cell types and stages involved. It’s hard to target specifically the negative modifications without affecting positive ones. This can also explain another crucial issue, drug delivery, while directing small renal cells as targets remains a challenge. Advancing regulators include FTO, and METTL3. They are still in development but compared to the others, MELLT3 shows more promising effects. Traditional small-molecule inhibitors often suffer from poor bioavailability and off-target effects, necessitating the exploration of advanced delivery methods such as nanoparticles and RNA-based therapeutics. Furthermore, the long-term effects of m6A methylation are not well understood, raising concerns about potential side effects on normal kidney function and systemic homeostasis. Given these challenges, future research may need to focus on refining original medicine strategies, identifying m6A-specific targets, and improving therapeutic delivery systems to ensure both efficiency and safety in CKD treatment.

3. The benefits and mechanisms of METTL3 inhibition

MRTTL3 inhibition is defined by the process of blocking the catalytic activity of METTL3, reducing the m6A methylation on specific mRNA. The major effect of METTL3 inhibition is to reduce mRNA stability, translation efficiency and RNA-protein interactions. Compared to eraser or reader proteins during m6A methylation, METTL3 stands out because it acts upstream in the signaling pathway[7]. METTL3 inhibition can broadly suppress aberrant m6A methylation at the

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earliest stage before showing downstream effects on gene expression and RNA stability, which makes it a more effective m6A-targeted treatment. In contrast, eraser proteins such as FTO and ALKBH5, act in later stages with highly dynamic and context-dependent functions. This means eraser inhibitions may lead to unexpected cell-type-specific effects or disrupt normal gene regulation processes. The least potential choice is m6A reader proteins, who function by interpreting m6A signals then influence RNA processing. Inhibition of them requires super specific targeting of individual proteins based on their transcript information. This is most complex and challenging as it asks for deeper cell-type-specific interventions and its specific mechanisms and development remain incomplete.

CKD has 4 major characteristics: renal fibrosis, chronic inflammation, oxidative stress and apoptosis. This is also the main reason for choosing METTL3 inhibitors as a promising treatment since it is significantly involved in all of them [8]. Renal fibrosis is defined by the excessive production of extracellular matrix (ECM), leading to normal kidney functions destruction. METTL3 inhibition can suppress key fibrotic signaling pathways by destabilizing pro-fibrotic transcripts, including TGF-β, Smad family member 3 (SMAD3), and collagen type I alpha 1 (COL1A1). By reducing gene expression on these mRNAs, METTL3 inhibitors are able to limit fibroblast activation and ECM accumulation [9]. For chronic inflammation, METTL3 inhibition reduces m6A modifications on inflammatory cytokine transcripts, including interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and NF-κB. This leads to decreased cytokine stability and translation, thereby dampening immune activation and inflammatory signaling [2]. Both oxidative stress and apoptosis contribute to CKD progression by damaging renal cells and depleting functional nephrons [10]. To fight against oxidative stress, METTL3 inhibitors function differently. Instead of focusing on gene deactivations, METTL3 inhibition leads to stabilizing antioxidant defense genes, such as nuclear factor erythroid 2-related factor 2 (Nrf2) and heme oxygenase-1 (HO-1), therefore, enhancing cellular resistance to oxidative damage. Simultaneously, it reduces m6A modifications again on pro-apoptotic factors such as Bcl-2-like protein 4 (BAX) and the B-cell lymphoma 2 (Bcl-2) family, leading to a decrease in apoptosis-related kidney damage [11-13]. By targeting METTL3, this inhibition approach offers a highly specific method to mitigate multiple CKD-related pathways, making it an effective therapeutic strategy.

Furthermore, there are 3 more recent findings for METTL3 inhibition. The first is relating to regulation of ATP-binding cassette subfamily G member 2 (ABCG2) signaling [14]. It is highly expressed in kidney and has a major role in promoting excretion of harmful metabolites and xenobiotics. Inhibiting METTL3 is proven to increase ABCG2 expression, which aid greatly in enhance kidney's detoxification mechanism, resulting in reduction of oxidative stress, inflammation and slow down CKD fibrosis progression. The second is RAAS inhibitors. It is a broad category for multiple classes of drugs which acting on different positions and targeting different proteins in RAAS pathways. The angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor blockers (ARBs) in RAAS are the main examples for kidney diseases, usually functioning to reduce blood pressure and kidney damage. These drugs help alleviate intraglomerular hypertension and limit kidney damage by inhibiting angiotensin II-mediated activation of pro-fibrotic pathways, particularly relating to TGF-β1 signaling. Since renal fibrosis is a major driver of CKD progression, RAAS inhibition plays a crucial role in slowing disease advancement [3]. The last one involved is called SGLT2 inhibitors. Differ than RAAS inhibitors, it is a more specific class of drugs which target SGLT2 protein only in kidneys. Example inhibitors contain dapagliflozin and empagliflozin. They have emerged as key treatment for CKD, primarily by lowering glucose reabsorption, reducing glomerular hyperfiltration, and improving metabolic and hemodynamic stability. Since it can decrease kidney stress and reduce inflammation and oxidative stress, it cooperates well with RAAS inhibitors, offering additional nephroprotection [4]. The combination of RAAS and SGLT2

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inhibitors provides a dual approach by targeting both hemodynamic and metabolic factors, reducing kidney injury more effectively than monotherapy.

This newly discovered link between METTL3 inhibition and ABCG2 upregulation further reinforces the potential of METTL3 inhibitors as a multifaceted therapeutic strategy for CKD. These valuable discoveries together offer great confidence for nowadays studies to keep moving forward. Future studies could focus on optimizing METTL3 inhibitors for greater specificity, improved drug delivery, and minimizing off-target effects. As research advances, METTL3 inhibition in combination with existing CKD therapies may open the gate for more effective, personalized treatments that enhance renal function and improve patient outcomes.

4. The therapeutic potential and future directions of METTL3 inhibitors in chronic kidney disease

Modern scientific advancements have significantly improved our understanding of RNA regulation in kidney diseases, with the inhibition of METTL3 emerging as a promising therapeutic strategy for CKD. The development of RNA sequencing technologies and molecular biology techniques has provided strong evidence that METTL3 inhibition can reduce fibrosis, suppress inflammatory signaling pathways, and slow disease progression [9]. Additionally, recent breakthroughs in small-molecule screening technologies have accelerated the identification and development of selective METTL3 inhibitors, increasing the feasibility of translating this approach into real-world clinical applications.

Despite these advantages, several critical challenges remain. One major concern is the specificity of m6A modification. Its widespread presence across different tissues in the human body makes it hard to target a specific area. While METTL3 inhibition has shown potential benefits in CKD, the long-term effects of sustained m6A suppression remain unclear. It is possible that long-termed METTL3 inhibition treatment could cause damage to normal renal functions, immune responses and kidney regeneration, raising concerns about potential unintended consequences. Furthermore, CKD is a heterogeneous disease influenced by multiple factors, including environmental exposures, genetic predisposition, and metabolic conditions. Due to this complexity, there will be difficulties towards personalized therapeutic strategies, which should be used to maximize the benefits of METTL3-targeted interventions. Identifying relevant biomarkers that predict individual patient responses to METTL3 inhibition will be a key step toward developing precision medicine approaches for CKD treatment. Another limitation is the lack of highly selective and clinically validated METTL3 inhibitors. While early small-molecule inhibitors have demonstrated efficacy in preclinical models, they require further optimization regarding potency, stability, and bioavailability. Additionally, there need to be more researches focusing on potential off-target effects since METTL3 is involved in various physiological processes beyond kidney disease. To ensure both safety and efficacy, further research is needed to refine inhibitor design, optimize drug delivery methods, and conduct comprehensive clinical trials.

Looking ahead, the future of METTL3-targeted therapy may involve combination treatments with existing CKD therapies to enhance therapeutic efficacy. For example, METTL3 inhibition could be combined with renin-angiotensin system (RAS) blockers, sodium-glucose cotransporter 2 (SGLT2) inhibitors, and other anti-fibrotic agents. They create synergistic treatment strategies which can improve kidney function and reduce fibrosis and inflammation symptoms. Moreover, gene-editing technologies such as CRISPR-Cas13 present another exciting avenue for targeting RNA modifications. Unlike traditional small-molecule inhibitors, CRISPR-Cas13 can precisely target and modulate m6A levels in specific cell types, potentially minimizing side effects while maximizing therapeutic benefits. Future research may also explore further for the modulation of m6A erasers (such as FTO and ALKBH5) and reader proteins. They might not produce well

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consequences as before, but once in combination with METTL3 inhibitors, there may be surprising positive outcomes. This innovation may provide additional therapeutic choices for patients too. Furthermore, advances in single-cell RNA sequencing will allow researchers to analyze disease progression at a cellular level, offering deeper insights into m6A-related mechanisms. At the same time, AI-driven drug discovery is expected to accelerate the identification of novel targets and the development of highly selective METTL3 inhibitors, enabling the creation of precision therapies tailored to individual patients [15].

5. Conclusion

This review has provided a comprehensive summary of the crucial role of m6A methylation in chronic kidney disease (CKD) and explored the therapeutic potential of METTL3 inhibition. By modulating aberrant m6A methylation and its downstream signaling pathways, METTL3 inhibitors offer a promising aspect for therapeutic intervention. Preclinical studies have demonstrated that targeting METTL3 can promote key pathological processes in CKD, including fibrosis, inflammation, oxidative stress, and apoptosis. These mechanisms contribute significantly to CKD progression, as METTL3 inhibition represents a novel strategy to slow and control disease advancement. These findings provide a strong foundation for future research and clinical applications.

However, despite the encouraging potential of METTL3-targeted therapies, several challenges must be addressed before they can be widely implemented in clinical settings. One of the major concerns is METTL3 inhibition specification. M6A modifications are critical to a wide range of physiological functions, including normal kidney function, immune system regulation, and cellular homeostasis. While METTL3 overexpression has been implicated in CKD pathogenesis, over-controlled METTL3 inhibition could disrupt essential biological processes and lead to unintended side effects. This highlights the necessity for developing highly selective inhibitors that can precisely target pathogenic m6A modifications while preserving physiological methylation processes. Another significant challenge is the heterogeneity of CKD. The disease manifests differently among patients due to variations in genetic, environmental, and lifestyle factors, making it hard to offer personalized therapeutic treatments. A deeper understanding of patient-specific molecular signatures is essential to overcome this obstacle, and future research should focus on identifying biomarkers that can predict an individual's response to METTL3 inhibition, thereby enabling more targeted and effective treatments. Advancements in drug delivery technologies may also play a crucial role in overcoming the limitations of METTL3-targeted therapies. Nanoparticle-based drug delivery systems offer a potential solution by enhancing the precision and efficiency of METTL3 inhibitors, reducing off-target effects, and improving drug bioavailability. Additionally, gene-editing techniques such as CRISPR-Cas13 could also provide an innovative approach to regulating METTL3 activity with greater specificity. Technologies like this offer chances for more refined and safer therapeutic interventions in CKD. Furthermore, integrating METTL3 inhibitors with existing CKD treatments may enhance their overall efficacy. Current CKD management primarily focuses on controlling symptoms and slowing disease progression through RAAS inhibitors, anti-inflammatory agents, and antifibrotic drugs. Combining these established therapies with METTL3 inhibitors may yield synergistic effects, providing a more comprehensive approach to disease treatments. Future studies should investigate potential combination therapies to determine their effectiveness and safety profiles.

As the field of RNA epigenetics continues to advance, the inhibition of METTL3 stands as a promising pioneer in CKD treatment. While challenges remain, ongoing research efforts to refine specificity, enhance delivery mechanisms, and integrate novel technologies, which will be basics in delivering preclinical success into clinical applications. By overcoming these barriers, METTL3-

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targeted therapies could contribute greatly to CKD treatment, offering new hope for patients and moving the field of nephrology toward a more precise and personalized approach.

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