

Research Progress and Regulatory Role of Long Non-coding RNAs Associated with N6-methyladenosine Modification in Important Economic Traits of Poultry

Luyu Yang^{1,2}, Zhiyuan Zhang^{1,2}, Fumin He^{1,2}, Ruilong Xu^{1,2}, Li Zhou^{1,2}, Yadong Tian^{1,2}, Ruili Han^{1,2}, Ruirui Jiang^{1,2}, Wenting Li^{1,2}, Guirong Sun^{1,2}, Zhuanjian Li^{1,2}, Xiangtao Kang^{1,2}, Donghua Li^{1,2,*} 

¹ College of Animal Science and Technology, Henan Agricultural University, Zhengzhou 450046, China.

² Key Laboratory of Livestock and Poultry Resources (Poultry) Evaluation and Utilization of Ministry of Agriculture and Rural Affairs, Henan Agricultural University, Zhengzhou 450046, China.

*Corresponding Author: Donghua Li; E-mail: lidonghua6656@126.com

Abstract

N6-methyladenosine (m6A) is the most common and abundant internal chemical modification in eukaryotic mRNA and long non-coding RNA (lncRNA). This dynamic and reversible epitranscriptional modification is mediated by a collaborative action of methyltransferases (writers), demethylases (erasers), and recognition proteins (readers) and represents an important epigenetic regulatory mechanism at the post-transcriptional level. lncRNAs are a class of RNA molecules that are longer than 200 nucleotides and lack the potential to encode proteins. They can participate in the fine regulation of gene expression at the transcriptional, post-transcriptional and epigenetic levels through various mechanisms such as cis-regulation and trans-regulation. In recent years, the interactive regulation of m6A modification and lncRNA has become a research hotspot. The precise regulatory network jointly constructed by these two elements plays a core role in the formation and plasticity regulation of complex traits in animals. This article systematically elaborates on the regulatory roles and research progress of m6A modification-related lncRNAs in important economic traits such as muscle development, fat deposition, and immune response in poultry. In response to the current issues of insufficient functional validation and insufficient mechanism elucidation, it proposes that future research should integrate multi-omics combined analysis, temporal and spatial dynamic analysis, cross-species comparative studies, and gene editing methods to systematically clarify their intrinsic regulatory mechanisms. This will open up a new path for molecular design breeding of poultry for disease resistance, high yield, high quality, and high efficiency, and is of great significance for promoting genetic improvement of poultry and the sustainable development of the industry.

Keywords

M6A modification; Long non-coding RNA; Poultry; Economic traits; Muscle development; Fat deposition; Immune response

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Introduction

M6A, the most abundant internal chemical modification on mRNA and lncRNA in eukaryotes, constitutes a core regulatory layer of epitranscriptomics^[1]. Its regulatory network is built on dynamically reversible enzymatic modifications: it is catalyzed by methyltransferase complexes (termed 'writers', the core complex comprising methyltransferase-like 3 (METTL3), methyltransferase-like 14 (METTL14), and wilms' tumor 1-associating protein (WTAP)), and can be specifically removed by demethylases (termed 'erasers', such as fat mass and obesity-associated protein (FTO) and alkB homolog 5 (ALKBH5)), enabling precise temporal and spatial regulation. The biological functions of this chemical modification are recognized and mediated by a suite of specific binding proteins termed 'readers', such as the YTH domain family proteins, the nuclear reader YTH domain-containing protein 1 (YTHDC1), and the Insulin-like growth factor 2 mRNA-binding protein (IGF2BP) family^[2]. Reader proteins, by binding to m6A sites, extensively and precisely regulate various stages of RNA metabolism, including the alternative splicing of precursor mRNAs, nuclear-cytoplasmic transport of

mature transcripts, regulation of translation efficiency, and attenuation of RNA stability, among other key processes^[3]. Thus, they play a central role in crucial biological functions such as embryonic development, cell differentiation, stress response, biological rhythms, muscle development, fat deposition, reproduction, and so on^[4]. The M6A modification mainly occurs on mRNA, and is also widely present in various non-coding RNAs, such as lncRNA, circRNA and miRNA^[5]. These modifications dynamically regulate the metabolic process of RNA, thereby influencing gene expression. In recent years, the abnormality of its regulatory mechanism has been clearly associated with various pathological processes such as tumor occurrence and development, metabolic syndrome, and neurological diseases^[6], thus making it a highly promising frontier field for the discovery of disease biomarkers and the development of targeted treatment strategies.

lncRNA represent a category of non-coding transcripts characterized by a length of more than 200 nucleotides and no protein-coding potential. It is widely present in the genome and plays a crucial regulatory role in various processes such as the growth and development of organisms, cell differentiation, and the occurrence of



diseases^[7]. Based on their positions relative to adjacent protein-coding genes in the genome and their transcriptional directions, lncRNAs can mainly be classified into several types such as intergenic lncRNAs (lincRNAs), antisense RNAs, intronic lncRNAs, enhancer-related lncRNAs, and bidirectional lncRNAs^[8]. As the research progressed, it was discovered that lncRNAs exert extensive regulation of gene expression at the epigenetic, transcriptional and post-transcriptional levels through complex molecular mechanisms^[9]. Its functional modes mainly include: as a signaling molecule, the change in its expression level itself is an important manifestation of cell differentiation or environmental stress response^[10]; as a guide, it specifically recruits chromatin modification complexes (such as polycomb repressive complex 2 (PRC2)) to the target gene sites^[11]; as a molecular scaffold, it assembles multi-protein complexes to integrate regulatory signals^[12]; it can also act as a competitive endogenous RNA (ceRNA), like a molecular sponge, adsorbing microRNAs to relieve their inhibitory effect on downstream target genes^[13]; in addition, some lncRNAs can also encode functional micropeptides, directly participating in processes such as cell proliferation and metabolism^[14].

Poultry, as a crucial source of animal protein worldwide, has an efficient production system that holds irreplaceable economic value for ensuring food security and meeting the growing demand for food^[15]. To address the multiple challenges in production, such as disease prevention and control, resource constraints, and sustainable development of the industry, by leveraging modern biotechnologies

like genomics and molecular breeding, continuous improvement of key economic traits including meat production performance, feed utilization efficiency, and disease resistance and stress tolerance has become an inevitable trend for ensuring the high-quality development of the livestock industry^[16]. With the continuous advancement of epigenetic research, it has been confirmed that the interaction between m6A modification and lncRNA constitutes a dynamic and intricate gene expression regulatory network, and plays a collaborative role in key biological processes such as muscle development and lipid metabolism in livestock^[17]. M6A modification serves as a dynamic and reversible post-transcriptional regulatory mechanism, which directly governs the differentiation of muscle satellite cells and fat production by dynamically regulating the RNA metabolism of key genes^[18]. Meanwhile, lncRNAs mainly exert indirect regulation through mechanisms such as constructing ceRNA networks^[13]. The two interact deeply with each other. M6A modification can directly affect the stability and function of lncRNAs^[19], and some lncRNAs can also interact back with the m6A modification process (Figure 1)^[20]. Therefore, this paper, from the perspective of epigenetic regulation by m6A-lncRNA, systematically analyzes the research progress of this phenomenon in important economic traits such as muscle growth and fat deposition in poultry. It provides a new theoretical framework and application prospects for developing new molecular markers and achieving precise breeding to cultivate modern poultry breeds with high yield, high quality, and strong adaptability.

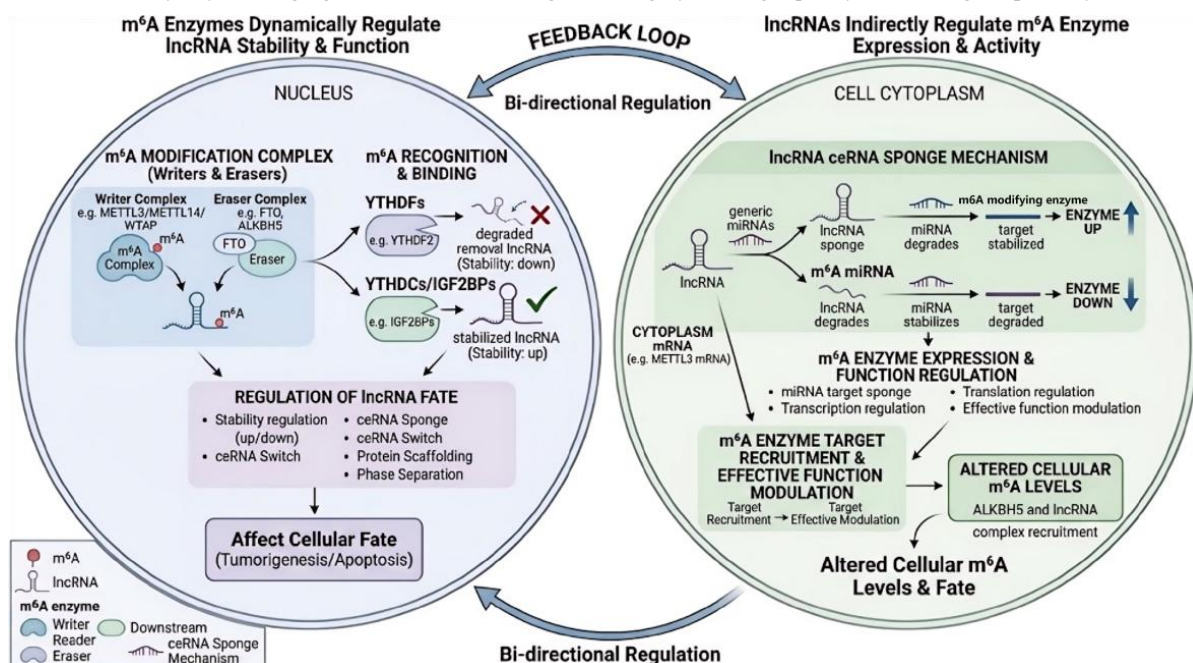


Figure. 1 The bidirectional regulatory mechanism of m6A-lncRNA. METTL3: methyltransferase-like 3; METTL14: methyltransferase-like 14; WTAP: wilms' tumor 1-associating protein; FTO: fat mass and obesity-associated protein; YTHDFs: YTH domain-containing family protein; YTHDCs: YTH domain-containing proteins; IGF2BPs: Insulin-like growth factor 2 mRNA-binding proteins; ceRNA: competitive endogenous RNA. Figures were edited using Adobe Illustrator 2026 (Version 30.0).

1. M6A Modification and lncRNA Detection Technology

In the research on the interaction between m6A and lncRNA in poultry, the commonly used technical systems mainly include three categories: comprehensive transcriptional profile mapping, targeted verification, and functional analysis.

The detection of m6A modifications in the entire transcriptome

mainly relies on methylated RNA immunoprecipitation sequencing (MeRIP-seq). This technique uses specific anti-m6A antibodies to enrich methylated RNA fragments, combined with high-throughput sequencing, to systematically map the entire transcriptome m6A methylation profile of specific tissues or cells under specific physiological conditions, locate m6A modification peaks and analyze their distribution characteristics^[21].

The systematic identification and expression analysis of lncRNAs

mainly rely on RNA sequencing (RNA-seq), especially strand-specific library sequencing, which can accurately distinguish the sense and antisense transcripts and is crucial for discovering new lncRNAs and analyzing their expression patterns^[22]. By comparing the sequencing data to the reference genome, the transcript structure of lncRNAs can be predicted, and candidate lncRNAs related to specific traits can be screened through differential expression analysis^[23]. After obtaining the candidate molecules, functional validation is required. Quantitative PCR combined with immunoprecipitation (such as RIP-qPCR or MeRIP-qPCR) is used to verify whether specific RNAs (such as lncRNAs or mRNAs) are bound to the target protein (such as the m6A reader YTH domain-containing family protein (YTHDF)) or whether they contain m6A modifications^[24].

The Chromatin Immunoprecipitation Sequencing (CLIP-seq) technique can precisely identify the direct target RNAs of RNA-binding proteins such as m6A recognition proteins at the whole transcriptome level, and analyze the characteristics of the recognition and binding motifs^[25]. The final functional gain and loss experiments are the core of mechanism elucidation. They are usually conducted in in vitro cell models (such as chicken myoblasts, preadipocytes, or macrophage lines) by siRNA/shRNA-mediated gene knockdown or plasmid transfection-mediated gene overexpression, to observe the effects on cell phenotypes (such as proliferation, differentiation, and lipid droplet accumulation) and the expression of downstream target genes, thereby confirming the functions of specific m6A modification enzymes or lncRNAs^[26]. The combined application of these technologies constitutes a complete research path from discovery, verification to mechanism analysis, serving as the foundation for deeply revealing the m6A and lncRNA interaction regulatory network in poultry.

2. Research on the M6A-lncRNA Regulatory Network in Economic Traits

2.1 The Role of the M6A-lncRNA Regulatory Network in Muscle Development and Meat Quality Formation

At present, the research on lncRNAs related to m6A modification or regulation in poultry muscle tissue is still in its infancy, but preliminary findings have revealed their potential core role in regulating muscle development and the formation of meat quality. Recent studies have shown that lncRNA, as a substrate for m6A modification, its stability and function are directly regulated by the dynamic changes of m6A. This is one of the important mechanisms by which lncRNA affects muscle development^[47]. Mao *et al.* found that up to 59.67% of lncRNA transcripts in the development of bovine skeletal muscle were modified by m6A, and the proportion of this modification showed dynamic changes during the development process^[27]. It indicates that m6A modification may be widely involved in regulating the stability and turnover of lncRNAs related to muscle development. The research in chickens has provided a more specific explanation of the mechanism by which m6A modification affects the transition of muscle fiber types through regulating key lncRNAs. Studies have confirmed that the transcription of the skeletal muscle-specific lncRNA MSTRG.15576.9 (MYH1G-AS) is jointly regulated by the transcription factors SMAD family member 3 (SMAD3) and SP2 transcription factor (SP2). Moreover, SP2 can also transcriptionally inhibit the expression of the m6A demethylase ALKBH5. The reduction in ALKBH5 expression leads to a weakened m6A demethylation effect on MYH1G-AS RNA, thereby increasing the m6A modification level of MYH1G-AS RNA and decreasing its RNA stability. This regulatory

axis ultimately accelerates myoblast proliferation, inhibits their differentiation, and promotes the transformation of slow muscle fibers to fast muscle fibers, thereby affecting the growth and development of muscles and the composition of muscle fibers^[28]. Similar patterns were also observed in pigs, where the level of m6A modification was positively correlated with the expression of most lncRNAs, suggesting that m6A modification might positively regulate their stability. For instance, when the m6A modification of lncRNA MSTRG.14200.1 was inhibited, it delayed the differentiation of pig skeletal muscle satellite cells^[29].

lncRNA can also indirectly regulate the downstream gene network related to muscle development, metabolism and m6A modification by adsorbing specific miRNA through the classic ceRNA network mechanism. This network itself may also be regulated by m6A modification, forming a multi-dimensional regulatory intersection^[30]. In the development of poultry muscle, lncIRS1 is a well-verified ceRNA, which can act as a molecular sponge to adsorb the miR-15 family, relieve its inhibition on downstream target genes, thereby activating the insulin-like growth factor 1-phosphatidylinositol 3-kinase/AKT (IGF1-PI3K/AKT) signaling pathway and promoting the proliferation and differentiation of chicken myoblasts^[31]. Another study has shown that lncRNA-Six1, on the one hand, can act as a ceRNA to adsorb miR-1611 and up-regulate the expression of Six1 protein; on the other hand, its transcript can also encode a functional micropeptide to cis-activate the Six1 gene, jointly promoting the proliferation of chicken myoblasts and muscle growth^[32,33]. These core pathways regulated by ceRNA mechanisms have been confirmed to be key pathways affecting muscle fiber hypertrophy, type transformation, and muscle energy metabolism.

Furthermore, lncRNAs can indirectly affect the m6A modification levels of global or specific genes by trans-regulating the activities of methyltransferases or demethylases during the m6A modification process, thereby influencing the muscle development process. At present, in poultry, there are limited reports on lncRNAs that directly target and regulate the expression of m6A enzymes, but several studies have revealed that the m6A enzymes themselves act as key regulatory nodes influencing muscle development. Studies have shown that lncRNA MEG3 can act as a molecular scaffold to bind to the transcription factor SPI1, recruit and activate the METTL3 methyltransferase, and increase the m6A modification level of target genes^[34]. At the same time, METTL3 can catalyze the m6A methylation level of lncRNA MEG3 and promote its degradation^[35]. The reduction of MEG3 can promote muscle atrophy, regulate muscle fiber types and the stability of muscle metabolism processes^[36]. Furthermore, METTL3 knockdown will affect the m6A modification level of the Notch signaling pathway in the body, reduce the mRNA translation efficiency of pathway components, inhibit the proliferation of muscle stem cells, and cause damage to muscle regeneration^[37]. In myoblast cells, the m6A modification mediated by METTL3 can promote the translation of the myocyte enhancer factor 2C (MEF2C) protein through the YTHDF1 reading protein. The MEF2C protein, in turn, can bind to the promoter of METTL3 to promote its expression, forming a self-reinforcing positive feedback loop, which jointly drives myoblast differentiation^[38]. At the same time, studies have shown that up to 59.67% of lncRNAs in bovine skeletal muscle are modified by m6A, and this proportion changes dynamically with the developmental process, further supporting the close interaction between m6A and lncRNAs in muscle development^[27].

2.2 The Regulatory Role of the M6A-lncRNA Regulatory Network in Fat Deposition and Metabolism

In addition to muscle development, the m6A-lncRNA axis also

plays an important role in fat deposition and lipid metabolism. In the regulation of poultry fat metabolism, the interaction between m6A modification and lncRNA forms a precise regulatory network in key metabolic tissues such as abdominal fat and liver, jointly participating in the regulation of core biological processes such as the proliferation and differentiation of pre-fat cells, lipid synthesis and decomposition metabolism, and thereby determining the efficiency of abdominal fat deposition. The steady-state imbalance of this interaction network not only leads to excessive accumulation of abdominal fat in poultry, reducing feed conversion rate and carcass quality, but also increases the risk of metabolic diseases such as fatty liver, seriously restricting the healthy development of the poultry industry.

In the abdominal adipose tissue, m6A modification deeply participates in the proliferation and differentiation process of preadipocytes by dynamically regulating the stability of key lncRNAs and the ceRNA network they mediate. Studies have shown that in chicken abdominal adipose tissue, the overall level of m6A modification is negatively correlated with fat production^[39]. Chao *et al.* conducted a study comparing abdominal fat in high-fat and low-fat line broilers. Through MeRIP-seq, they discovered that the differentially methylated m6A-modified genes were enriched in the lipid metabolism pathway. Among them, the m6A modification level of the lecithin cholesterol acyltransferase (LCAT) gene was higher in the low-fat line, and its mRNA stability was precisely regulated by the demethylase ALKBH5^[40]. ALKBH5 was highly expressed in the abdominal fat of the low-fat line, and it promoted the degradation of genes such as LCAT by reducing their m6A modification levels, thereby inhibiting fat production^[26]. This process may be mediated by specific lncRNAs. For instance, in the study of the ceRNA regulatory network for fat deposition in the duck's abdomen, multiple lncRNAs related to lipid metabolism were identified through whole transcriptome sequencing. These lncRNAs can act as "sponges" for miRNAs, competitively binding to miRNAs and relieving their inhibitory effect on target genes^[41]. Although the abdominal fat-specific lncRNAs directly regulated by m6A modification have not been well reported in poultry, a study by Luo *et al.* in porcine adipocytes suggested a possible pattern. M6A modification can promote the degradation of lncRNAs involved in adipogenesis through the protein-mediated recognition by YTHDF2, thereby inhibiting fat deposition^[42]. Additionally, the m6A methyltransferase METTL3 has been confirmed to reduce the expression of PHKG1 through an m6A-YTHDF2-dependent manner, thereby promoting adipogenic differentiation of porcine precursor adipocytes^[43]. This suggests that METTL3 may regulate the stability of key lncRNAs in poultry abdominal fat through a similar mechanism.

As the central organ for lipid metabolism, in liver tissue, the m6A-lncRNA interaction network has a global regulatory effect on lipid synthesis, transport and output. The m6A modification apparatus (methyltransferase, demethylase and recognition protein) plays a key role in liver lipid metabolism. In the egg-laying chicken fatty liver syndrome (FLS) model, liver tissue shows upregulation of FTO and downregulation of YTHDF2, resulting in specific demethylation and stable expression of mRNA of fatogenic genes such as fatty acid synthase (FASN) and sterol regulatory element binding protein 1 (SREBP1), thereby driving abnormal accumulation of lipids in the liver^[44]. Furthermore, the liver-specific deletion of METTL14 can inhibit the translation of β -adrenergic signaling-related transcripts by maintaining their high m6A modification levels, thereby enhancing fat breakdown and resisting obesity and fatty liver induced by a high-fat diet^[45]. These m6A enzymes may reshape the liver lipid metabolism

network by influencing the entire transcriptome, including lncRNAs. In the liver of mice, the lncRNA lncLSTR can regulate the expression of CYP8B1 by interacting with TAR DNA-binding protein 43 (TDP-43), thereby influencing bile acid synthesis and lipid homeostasis^[46]. The m6A modification has been shown to regulate the stability of lncRNAs through mechanisms such as the METTL3/YTHDF2 axis^[26], suggesting that a similar mechanism may also exist in the liver of poultry, that is, the m6A modification can regulate the stability of lncLSTR, thereby reshaping the lipid metabolism network of the liver.

2.3 The Role of the M6A-lncRNA Regulatory Network in Immune Response and Disease Resistance

In addition to muscle development and fat metabolism, the m6A-lncRNA regulatory network also exerts indispensable effects on the immune defense and disease resistance of poultry. During the process of pathogen infection, lncRNAs with changes in m6A modification exist in the immune organs or cells of poultry. These molecules play a significant role in disease resistance by regulating the inflammatory response and innate antiviral immunity. Sun *et al.* conducted a systematic analysis of the full transcriptome m6A modification profile of lncRNAs in chicken embryo fibroblast cells (CEF) infected by Marek's disease virus (MDV) for the first time^[47]. This study, through MeRIP-seq, discovered that compared with the uninfected control group, MDV infection significantly increased the expression of lncRNA m6A modification sites, indicating that m6A modification is highly conserved on lncRNAs, and MDV infection can reshape the m6A modification profile of lncRNAs. Further Gene Ontology and KEGG pathway analysis showed that these lncRNAs with m6A modification changes were significantly enriched in signaling pathways closely related to MDV infection, suggesting that m6A modification may participate in regulating the viral replication process by affecting the stability or function of lncRNAs. Zhao *et al.* systematically depicted the dynamic changes of m6A modification during viral infection through combined analysis of MeRIP-seq and RNA-seq of chicken tumor liver induced by J subgenus avian leukemia virus (ALV-J), and identified 1757 differentially expressed mRNAs and 126 differentially expressed lncRNAs. Gene enrichment analysis indicated that they may be involved in biological processes and pathways such as immune-related and apoptosis, and through co-expression network analysis, it was found that these lncRNAs were associated with 18 potential m6A-related methyltransferases, suggesting that m6A modification may participate in the tumor formation process induced by ALV-J through affecting the expression of lncRNAs^[48].

The above studies collectively reveal that during the pathological process of immune organs or cells of poultry responding to viral infections, m6A modification not only reshapes the expression profile of host lncRNAs, but also directly alters the methylation status of specific lncRNAs, and through potential interactions with methyltransferases, constitutes a multi-level epigenetic transcriptional regulatory network, laying a solid foundation for understanding the function of m6A modification and lncRNA interaction in the avian antiviral immune response.

3. Challenges and Prospects

Although the interaction between m6A modification and lncRNA has shown potential in regulating important economic traits in poultry, this field is still in its infancy and exploratory stage. It faces numerous challenges and also holds vast research prospects.

3.1 Limitations of the Current Study

At present, most studies are still at the stage of using high-throughput sequencing techniques (such as MeRIP-seq and RNA-seq) to describe correlations and screen candidate molecules. For the identified differentially methylated m6A-modified lncRNAs, there is a lack of in-depth and systematic *in vitro* and *in vivo* functional validation of their specific biological functions, precise molecular mechanisms (such as which miRNAs they specifically adsorb as ceRNA, which proteins they interact with, and how they affect chromatin status), and the dynamic changes of their m6A modifications. For instance, in the development of chicken muscle, ALKBH5 has been identified as a key regulatory node^[21], but the downstream key lncRNA targets directly regulated by it and the complete signaling pathway have not yet been fully elucidated.

In terms of the important economic traits of poultry, current research mainly focuses on muscle and fat tissues. The exploration of the m6A-lncRNA regulatory network in the liver, immune organs (spleen, bursa of Fabricius), and reproductive system (ovary, oviduct, testis) is relatively scarce. For example, in the reproductive traits, it is only known that m6A modification is involved in the process of egg follicle selection in chickens^[49], but which lncRNAs are regulated by this modification and affect the egg-laying number remains unknown. In addition, current studies are predominantly concentrated in chickens, with only a limited number of reports in ducks and other poultry species. The overall research coverage of different poultry types remains insufficient, which needs to be expanded in future investigations. This limitation restricts the global understanding of the formation mechanism of poultry economic traits.

At the technical level, although the MeRIP-seq technology that relies on antibody enrichment can achieve the mapping of the entire transcriptome m6A modification profile, it has limitations such as low resolution and difficulty in precisely locating individual m6A sites. Moreover, it is prone to generating false positive signals due to non-specific binding of antibodies. Additionally, the annotation information of lncRNAs in poultry is still incomplete. Coupled with their complex transcript structures and low expression levels, it also poses significant challenges for their precise identification and quantitative analysis. In terms of mechanism research, there are significant differences in the homology and functional conservation of m6A modification-related enzymes between poultry (such as chickens and ducks) and mammals (such as humans and mice)^[50]. Currently, most of the proposed regulatory mechanisms are still based on the research models of mammals. The unique m6A modification patterns, lncRNA functional patterns, and their evolutionary characteristics under the selection pressure of domestication and breeding in poultry still need to be further systematically clarified.

3.2 Future Research Directions

Future research should further delve into the mechanism exploration and establish a precise regulatory network. It is necessary to go beyond simple correlation analysis and comprehensively utilize techniques such as RIP-qPCR, CLIP-seq, dual luciferase reporter gene, RNA pulldown-MS, etc., to verify the direct interactions between key m6A-modified lncRNAs and reading proteins (such as the YTHDF family), miRNAs or transcription factors; by using CRISPR/Cas9 gene editing, conditional gene knockout and overexpression models, the causal regulatory relationships of specific m6A-lncRNA axes on muscle growth, fat deposition, disease resistance and reproductive performance should be clarified *in vivo*. At the same time, efforts should be made to promote the integration of multi-omics data and temporal-spatial dynamic analysis. The data of epigenetic transcriptome (m6A seq),

transcriptome (RNA seq), translationome (Ribo seq), proteome, and epigenome (ATAC seq, ChIP seq) should be jointly analyzed. Combined with single-cell nuclear sequencing (snRNA seq) and spatial transcriptomics techniques, the spatiotemporal specific expression and functional heterogeneity of m6A-modified lncRNAs in different tissues, cell subpopulations, and developmental stages should be revealed.

In addition, it is necessary to enhance species comparison and evolutionary analysis. By comparing genomes and epigenetic transcriptomes across species (chickens, ducks, geese, and mammals), identify conserved or specific m6A lncRNA regulatory modules in poultry, and analyze their evolutionary characteristics and association mechanisms with economic traits during domestication and breeding selection. Ultimately, efforts should be focused on achieving the transformation from basic research to breeding applications. Identify the key quantitative trait loci (m6A QTL) with stable phenotypic effects and associated lncRNAs, and develop functional markers that can be used for molecular marker-assisted selection (MAS) and genomic selection (GS). Explore precise intervention strategies for the key m6A-lncRNA axis using technologies such as nutritional regulation (such as methyl donor additives) and RNA-targeted editing, providing feasible approaches for precise nutrition and molecular breeding. At the same time, evaluate the impact of related interventions on animal health and welfare to ensure the scientificity and sustainability of breeding practices.

In conclusion, the research in the field of poultry m6A lncRNA regulation is still in a stage of rapid development. By breaking through the limitations of existing technical and mechanism understanding, conducting more in-depth functional analysis, multi-omics combined analysis, cross-species evolutionary comparison, and transformational application research for breeding, it will significantly deepen the understanding of the epigenetic regulatory mechanism of important economic traits in poultry, providing new epigenetic theoretical basis and breeding new ideas for cultivating modern poultry varieties with high yield, high quality, and strong adaptability.

4. Conclusions

In summary, the m6A-lncRNA regulatory network acts as a key epigenetic mechanism governing poultry muscle development, fat deposition, and immune response. M6A modification dynamically controls lncRNA stability and function, while lncRNAs feed back-regulate m6A machinery to shape economic traits.

This interdisciplinary field has become an important frontier in the study of epigenetics in agricultural animals. Systematically conducting the mapping of m6A-lncRNA profiles in multiple tissues, deeply analyzing their molecular regulatory mechanisms, and extending to waterfowl species such as ducks and geese will deepen our understanding of the formation laws of economic traits in poultry. Eventually, these basic research results will be transformed into molecular breeding practices, identifying key markers and targets that can be used for genetic improvement, which has significant theoretical value and practical significance for promoting independent innovation in poultry breeding and enhancing the core competitiveness of the industry.

Author Contributions

Luyu Yang conceived the framework, collected and analyzed key literature, and drafted and revised the full manuscript. Zhiyuan Zhang, Fumin He, Ruilong Xu, Li Zhou helped revise the article. Yadong Tian, Ruili Han, Ruirui Jiang, Wenting Li, Guirong Sun, Zhuanjian

Li, Xiangtao Kang, Donghua Li assisted in literature collection, data sorting and manuscript proofreading. Donghua Li provided guidance and finalized the manuscript. All authors read and approved the final version.

Artificial Intelligence (AI) Statements

During the preparation of this manuscript, artificial intelligence tools were only used for language polishing, writing refinement, and optimization of English expression. All research content, including data acquisition, result analysis, and literature citation, was independently completed and rigorously verified by the authors. Figures were created by the authors using Adobe Illustrator 2026 (Version30.0). All final versions of the text were comprehensively reviewed and revised by the authors, who take full responsibility for the accuracy and originality of the manuscript.

Conflicts of Interest

The authors declare no competing financial interests.

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