

The Role of Genomics in the Development and Treatment of Multiple Myeloma: Understanding the Challenges and Opportunities

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Abstract: This review aims to provide a comprehensive overview of recent advances in the genomics of multiple myeloma and their clinical implications. Multiple myeloma is a hematologic malignancy originating from bone marrow plasma cells, characterized by the infiltration of pathological plasma cells, osteolytic bone lesions, and the presence of monoclonal immunoglobulins in serum and/or urine. Multiple myeloma exhibits significant genetic heterogeneity, which is the core reason for the considerable variability in patient prognosis, differential treatment responses, and the eventual development of drug resistance. In recent years, with the rapid development of high-throughput sequencing technologies (such as next-generation sequencing, single-cell sequencing) and bioinformatics, our understanding of genomic abnormalities in multiple myeloma has reached an unprecedented depth. Importantly, these genomic abnormalities have begun to directly inform clinical practice, holding significant promise particularly in risk stratification, treatment selection, minimal residual disease monitoring, and early warning of relapse. This review systematically outlines the latest research progress in the field of multiple myeloma genomics. It focuses on elucidating the function and clinical significance of key driver gene mutations (eg, NRAS/KRAS, TP53, MYC), delves into the roles of copy number variations (particularly 1q21 amplification), epigenetic dysregulation (including DNA methylation, histone modifications), and non-coding RNAs in multiple myeloma pathogenesis. Furthermore, this review looks forward to future developments, including targeted and immunotherapeutic strategies guided by genomics, as well as the use of liquid biopsy for minimal residual disease monitoring, offering new insights into the precision diagnosis and treatment of multiple myeloma.

Keywords: multiple myeloma, genomics, gene mutation, prognostic model, precision medicine

Introduction

Multiple myeloma (MM) is the second most common hematologic malignancy, accounting for approximately 10% of hematologic cancers.¹ The etiology of MM remains unclear, and its pathogenesis is a complex, multifactorial process involving genetic mutations, chromosomal aberrations, alterations in the bone marrow microenvironment, and immune dysfunction.² The classic “two-hit model” posits that the initial stage of the disease is driven by primary genetic events (such as immunoglobulin heavy chain translocations), followed by the acquisition of secondary genetic events (eg, RAS mutations, TP53 deletion) during disease progression.³ Xin et al elaborated on the core mechanisms underlying the progression from monoclonal gammopathy of undetermined significance (MGUS) to MM. They highlighted that the bone marrow microenvironment—characterized by immune dysregulation, aberrant cytokine networks, and disrupted bone remodeling—synergizes with intrinsic plasma cell defects, thereby forming a vicious cycle that drives clonal evolution, therapeutic resistance, and osteolytic lesions.⁴ In the precision diagnostic and therapeutic landscape of MM, FISH serves as the cornerstone for risk stratification, primarily enabling the rapid detection of IGH translocations and

core copy number abnormalities at initial diagnosis. However, FISH possesses inherent limitations in elucidating the full genomic landscape of MM. With the advancement of genomic technologies, targeted sequencing has emerged as an essential tool for identifying occult high-risk mutations and drug resistance-related genes, providing critical insights for guiding personalized treatment strategies. Furthermore, whole-genome sequencing (WGS) represents a promising technological frontier, particularly in deciphering mechanisms of resistance to novel immunotherapies such as CAR-T therapy. Collectively, from FISH-based initial stratification, through targeted sequencing-guided precision intervention, to WGS-driven tracing of drug resistance, these three modalities constitute a progressive precision medicine framework that has substantially advanced our understanding of the molecular classification and evolutionary mechanisms of MM. Research has found that nearly all MM patients harbor complex genomic abnormalities, including point mutations, insertions/deletions, copy number variations (CNVs), and structural variations (SVs).⁵ These genetic alterations collectively determine the clonal evolutionary trajectory of MM and directly influence patient treatment response and long-term survival. More importantly, genomic information is increasingly being utilized to guide clinical decision-making. For instance, according to the Chinese Guidelines for the Diagnosis and Treatment of Multiple Myeloma (2024 Revision), patients presenting with biallelic inactivation of TP53 or the presence of two or more high-risk cytogenetic abnormalities (eg, del(17p), t(4;14), 1q amplification) are classified as “ultra-high-risk”, and their prognosis is significantly worse than that of standard-risk patients.⁶ Furthermore, high-risk cytogenetic abnormalities (HRCA) serve as critical determinants in the selection of intensified treatment regimens. Multiple pivotal clinical trials focusing on newly diagnosed high-risk MM (eg, GMMG-CONCEPT, MASTER, etc.) have defined high-risk populations based on specific genomic aberrations such as del(17p) and t(4;14). These studies have explored the efficacy of combination therapies incorporating proteasome inhibitors, immunomodulatory agents, and monoclonal antibodies, as well as the early application of autologous stem cell transplantation. One of the primary therapeutic objectives in these approaches is to achieve deep remission, including minimal residual disease (MRD) negativity.⁷ However, MRD status has emerged as a robust surrogate endpoint for assessing treatment depth and predicting long-term survival. Studies have demonstrated that achieving and sustaining durable MRD negativity is strongly associated with significantly prolonged PFS and OS.⁸ Dynamic genomic MRD monitoring (eg, tracking clonal immunoglobulin gene rearrangements via NGS) can visually represent the trajectory of treatment response, offering real-time insights for adapting therapeutic strategies. Gan et al noted that specific molecular residual thresholds (eg, circulating tumor DNA levels) or patterns of clonal evolution may serve as early warnings for impending clinical relapse, thereby advancing the window for intervention.⁹ Performing repeat genomic analysis on relapse samples helps elucidate the evolutionary trajectories of drug-resistant clones and identify potential novel targets, thereby guiding subsequent therapeutic decisions.¹⁰ In recent years, the field of genomics in MM has undergone a paradigm shift, moving from a morphology- and clinically-driven approach toward a new era defined by multiomics and precision medicine (Figure 1).

Genomics has been deeply integrated into the full spectrum of MM diagnosis and treatment. By enabling precise risk stratification, it identifies high-risk patients requiring intensified therapy; through molecular classification, it provides a rationale for selecting therapeutic regimens; and via ultra-sensitive MRD monitoring, it dynamically assesses treatment response and provides early warning of relapse. As such, genomics has established itself as a cornerstone of contemporary MM research and clinical practice. Therefore, this article aims to systematically review key breakthroughs in MM

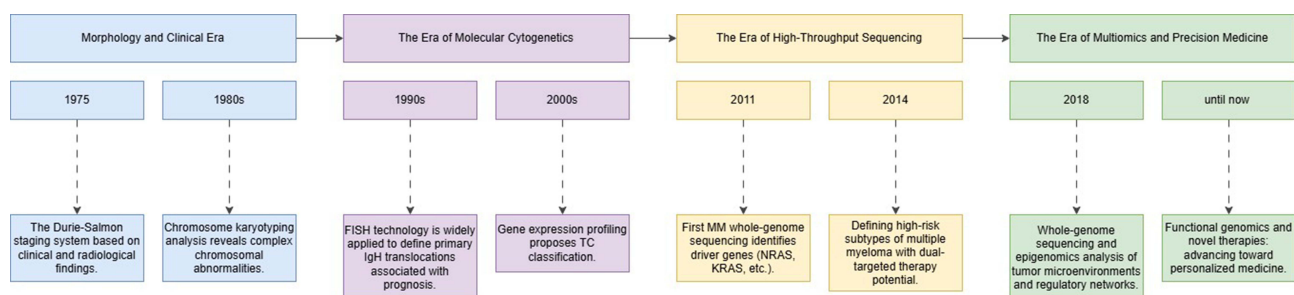


Figure 1 Genomics development in Multiple Myeloma.

genomics research in recent years, discuss their importance as prognostic and treatment decision tools in clinical practice, and finally, provide an outlook on future research directions.

Latest Research Advances in Classification and Function of Key Mutated Genes in MM

RAS/MAPK Signaling Pathway Genes

The RAS/MAPK pathway is one of the most frequently activated signaling pathways in MM. Mutations in NRAS, KRAS, and BRAF genes are most common within this pathway. In newly diagnosed MM (NDMM), the mutation frequency of NRAS and KRAS can reach 40%-50%.^{3,11} These mutations often occur at specific hotspot codons (eg, KRAS G12, G13; NRAS Q61), leading to loss of GTPase activity and constitutive activation of the protein, thereby promoting cell proliferation, survival, and inhibiting apoptosis. Beyond point mutations, studies using liquid biopsy of circulating tumor DNA (ctDNA) have also identified non-canonical site mutations in RAS pathway genes, which may be associated with unique clinical phenotypes and poor prognosis.¹² Clinical studies have confirmed that in patients with NDMM, the presence of NRAS, KRAS, or BRAF mutations is significantly correlated with a lower complete response rate following induction therapy with the VRd (bortezomib, lenalidomide, dexamethasone) regimen, suggesting that these mutations are independent adverse prognostic factors.¹³ Studies have shown that oncogenic RAS mutations can drive tumor growth by forming a complex with the amino acid transporter SLC3A2 and mTOR on endosomes to aberrantly activate mTORC1 signaling. This finding provides a rationale for the potential benefit of combining mTORC1 and MEK inhibitors in patients with RAS-mutant MM.¹⁴ A foundational study revealed that the GSK inhibitor TL4-12 induces cell cycle arrest and promotes apoptosis in RAS-mutant MM cells, and exhibits synergistic effects when combined with lenalidomide.¹⁵ However, the high heterogeneity of mutations within this pathway poses therapeutic challenges, as treatments targeting the dominant clone may fail to eradicate all mutated subclones, thereby contributing to persistent MRD and serving as a source of future relapse.

Tumor Suppressor Genes

Inactivation of tumor suppressor genes is a critical step in the malignant progression of MM, with TP53 and RB1 being the most important. TP53 abnormalities include deletion of the short arm of chromosome 17 [del(17p)], gene mutations (mostly missense), and biallelic inactivation.^{1,16} del(17p) is typically detected by FISH, while mutations require sequencing. TP53 is the central regulator of cellular stress response, capable of inducing cell cycle arrest, DNA repair, senescence, or apoptosis. Loss of TP53 function allows MM cells to evade apoptosis induced by genotoxic agents (eg, chemotherapy, proteasome inhibitors), leading to treatment resistance and disease progression.^{16,17} TP53 abnormality is one of the most well-defined high-risk factors in MM, closely associated with very short survival. Even in the absence of del(17p), TP53 mutation itself is an independent marker of poor prognosis.¹ Zhang et al pointed out that targeted therapeutic strategies for TP53 abnormalities (such as restoring p53 function or targeting its downstream pathways) are current research hotspots.¹⁶ In patients with biallelic inactivation of TP53, the depth and duration of response to conventional immunomodulatory drugs (IMiDs) and proteasome inhibitors (PIs) are often suboptimal, leading to early relapse. Moreover, the mutation frequency of TP53 abnormalities increases significantly at the time of disease relapse or progression, suggesting that therapeutic pressure may select for and expand these resistant clones.¹⁸ This has prompted clinical investigation into more cutting-edge therapeutic approaches. For instance, the early application of combination regimens containing monoclonal antibodies (such as CD38-targeted agents), bispecific antibodies, or CAR-T therapy for such patients has become a consensus.

The RB1 gene controls the G1 to S phase cell cycle transition, and its loss leads to uncontrolled cell cycle proliferation. Although its prognostic weight as an independent risk stratification factor may be lower than that of TP53, it is frequently found in conjunction with other high-risk abnormalities, synergistically driving disease progression. Lang et al's study provided an in-depth exploration of the association between RB1 gene deletion and renal insufficiency in MM patients. They found a positive correlation between the number of RB1 deletion-positive cells and serum creatinine levels, and patients with RB1 deletion combined with renal insufficiency had worse PFS.¹⁹ In clones harboring

RB1 deletion, the resulting dysregulation of the cyclin-dependent kinase (CDK) 4/6 pathway provides a rationale for exploring combination therapies involving CDK4/6 inhibitors (eg, palbociclib) with existing agents, although their clinical application in MM requires further validation.²⁰

Oncogenes and Cell Cycle Regulatory Genes

MYC is a potent transcriptional regulator acting as an “accelerator” in MM disease progression. Although mutations in MYC itself are uncommon, its dysregulated expression (eg, overexpression) is extremely common in MM, often caused by upstream pathway activation or genomic rearrangements (eg, translocations).²¹ Aberrant activation of MYC drives metabolic reprogramming, ribosome biogenesis, and cell proliferation, closely associated with disease transformation from MGUS to active MM.²¹ Concurrent MYC abnormalities and other high-risk events, such as t(4;14) and del(17p), define cases as “double-hit” or “triple-hit” myeloma, which are associated with poor response to current therapies and significantly shorter survival.²² Studies have also shown that upregulation of MYC is associated with multidrug resistance.²³ However, directly targeting the MYC protein remains challenging, and current strategies primarily focus on its upstream regulatory pathways or downstream synergistic factors. For instance, BET bromodomain inhibitors are designed to interfere with MYC transcription; however, their efficacy as monotherapies remains limited. Recent clinical trials are exploring their combination with PIs or IMiDs.²⁴ In addition, emerging therapies such as PROTAC degraders targeting MYC protein stability are under investigation in preclinical studies.²⁴

The CCND family (CCND1, CCND2, CCND3) are key regulators of G1 phase progression. In MM, CCND gene dysregulation primarily occurs through translocations (eg, t(11;14) leading to CCND1 overexpression, t(6;14) leading to CCND3 overexpression) or copy number amplification (eg, 1q21 amplification often accompanies CCND2 overexpression),³ collectively driving uncontrolled cell cycle progression through aberrantly high cyclin D expression.²⁵ The abnormality of the CCND1 gene is most commonly observed in the t(11;14)(q13;q32) chromosomal translocation, which places the CCND1 gene under the control of the immunoglobulin heavy chain (IGH) enhancer, thereby driving its overexpression. t(11;14) is often associated with lymphoplasmacytic morphology, CD20 expression, and survival dependence on BCL2 protein, making it remarkably sensitive to the BCL2 inhibitor venetoclax.²⁶ Preclinical and early-phase clinical data suggest that patients with CCND1 overexpression resulting from the t(11;14) translocation may exhibit increased sensitivity to treatment with CDK4/6 inhibitors.²⁷ Notably, t(11;14)-positive patients exhibit internal heterogeneity and can be further subdivided into prognostically distinct CD-1 and CD-2 subtypes based on gene expression profiling.²⁸ Although t(11;14) has traditionally been classified as standard risk, recent studies suggest its prognosis is closer to intermediate risk, and it exhibits specific clinical behaviors when associated with primary plasma cell leukemia.²⁹ Upregulation of CCND2 is primarily associated with the t(4;14)(p16;q32) translocation, which results in the co-expression of FGFR3 and CCND2. The t(4;14) translocation has long been classified as a HRCA and is correlated with a more aggressive disease course and shorter survival.³⁰ To address the poor prognosis in this high-risk patient subgroup, contemporary therapeutic strategies emphasize the use of multi-drug combinations, including agents such as daratumumab and carfilzomib, aiming to overcome adverse outcomes and achieve deeper MRD-negative status. Aberrant expression of CCND3 is frequently observed in the context of the t(6;14)(p21;q32) translocation. The t(6;14) translocation, though rare, shares biological similarities with t(11;14) and carries a standard prognostic risk for both progressive and active myeloma.³¹ A groundbreaking study has, for the first time, identified the deubiquitinase USP10 as a critical stabilizer of CCND3. By inhibiting the ubiquitination-mediated degradation of CCND3, USP10 activates the CCND3/CDK4/6-Rb signaling axis, thereby driving the proliferation of myeloma cells. This finding suggests that targeting the USP10/CCND3 axis represents a novel therapeutic strategy, which, when combined with CDK4/6 inhibitors, exhibited synergistic anti-tumor effects.³²

DNA Damage Repair and Ubiquitin-Proteasome Pathway Related Genes

The ubiquitin-proteasome pathway (UPP) is the primary pathway for intracellular protein degradation and the target of proteasome inhibitors like bortezomib. Specific alterations in UPP-related genes have emerged as important complements for identifying high-risk disease. For instance, deletions or inactivating mutations in E3 ubiquitin ligase genes such as HUWE1, FBXO9, and FBXW7 are significantly associated with enhanced genomic instability, DNA repair

deficiencies, and poorer survival outcomes.³³ Among these, FBXW7 functions as a tumor suppressor, and its inactivation leads to the aberrant accumulation of oncoproteins such as c-MYC and Cyclin E, thereby driving aggressive clonal proliferation. These alterations can be identified through NGS or WGS, and their detection enables further stratification of patients into an “ultra-high-risk” subgroup, guiding more intensive therapeutic interventions.³⁴ Mutations or reduced expression of the CRBN gene have been identified as one of the key mechanisms underlying primary or acquired resistance to IMiDs (eg, lenalidomide, pomalidomide), as they disrupt the drug-induced degradation of substrate proteins such as IKZF1/3.³³ Evidence suggests that the COP9 signalosome is critical for maintaining the stability of the CUL4-DDB1-CRBN E3 ubiquitin ligase complex, in which CRBN resides, and its loss could lead to impaired function of this complex, thereby contributing to drug resistance.³⁵ Therefore, assessing CRBN status prior to treatment may help avoid ineffective therapy. Sun et al, through bioinformatics analysis, found that the proteasome pathway-related gene DCAF8 is highly expressed in MM and associated with poor patient prognosis.³⁶ DCAF8 may interact with XPO1 protein, participating in nucleocytoplasmic transport of proteins; its high expression might represent a novel oncogenic dependency pathway in MM.³⁷ Achieving sustained MRD negativity is currently a key therapeutic goal in MM, and clonal evolution of UPP-related genes represents a major driver of MRD resurgence and clinical relapse. Dynamic monitoring of bone marrow or circulating tumor DNA via highly sensitive NGS allows for the tracking of acquired mutations in genes such as CRBN. These mutations, emerging under therapeutic pressure, can be detected months prior to clinical relapse and serve as early biomarkers for relapse warning.³⁴

In recent years, the role of DNA damage repair (DDR) genes in the pathogenesis, risk stratification, and treatment of MM has become increasingly clear, establishing them as key targets in precision medicine. In the context of risk stratification, large-scale genomic studies have revealed that pathogenic rare variants in DDR genes—such as TP53, ATM, CHEK2, KDM1A, and ARID1A—are significantly associated with genetic susceptibility to MM. These variants are particularly enriched in patients with early-onset disease or a family history of MM. Patients carrying mutations in TP53 or ATM exhibit worse OS, indicating that these genes may serve as independent molecular markers for identifying high-risk populations.³⁸ In terms of treatment selection, the functional status of DDR directly influences cellular sensitivity to genotoxic agents, as evidenced by studies confirming that melphalan resistance in MM cells is closely associated with DDR-related parameters including endogenous DNA damage levels, glutathione metabolism, and nucleotide excision repair capacity.³⁹ Meanwhile, the upregulation of key base excision repair pathway genes, such as PARP1, correlates with poor prognosis in patients undergoing autologous stem cell transplantation, providing a rationale for DDR-targeted therapeutic strategies, including PARP inhibitors.⁴⁰

CNVs and SVs

1q21 Amplification

1q21 amplification is currently one of the most common high-risk genetic abnormalities in MM, occurring in approximately 30%-40% of NDMM patients and up to 70% in relapsed/refractory multiple myeloma (RRMM) patients.⁴¹ The 1q21 region contains numerous genes, making the identification of true driver genes a research challenge. Garcia et al, through integrative analysis, identified PSMB4 and PSMD4 as novel target genes associated with 1q21 amplification that may contribute to proteasome inhibitor resistance.⁴¹ PSMB4 is a proteasome subunit; its overexpression may directly alter proteasome composition and function, thereby affecting bortezomib efficacy. Additionally, CKS1B, BCL-9, PDZK1, among others, are also considered potential driver genes in this region.⁴² Xu et al, through bioinformatics analysis, further screened NVL, IL6R, and DUSP23 as hub genes in 1q21 amplified MM associated with poor prognosis, providing new clues for targeted intervention.⁴³ Research indicates that 1q21 amplification (typically defined as ≥ 4 copies) is associated with shorter PFS and OS compared to simple gain (3 copies).⁴⁴ Therefore, the precise detection of 1q21 copy number alterations and accompanying co-aberrations using FISH has emerged as a cornerstone of modern initial risk stratification in MM. In clinical practice, for treatment-naïve patients harboring 1q21 gain/amplification—particularly those with high copy numbers or concurrent HRCA—both domestic and international guidelines recommend the use of intensified combination regimens incorporating novel agents (PIs, IMiDs, and anti-CD38 monoclonal antibodies) as the first-line choice to overcome their inherently poor prognosis. Notably, some studies have revealed

that tumor cells with 1q21 amplification may exhibit unique sensitivity to MCL-1 inhibitors, providing a theoretical rationale for the future development of targeted therapies for this specific subgroup.⁴⁵

Other High-Risk CNVs

del(17p), as mentioned, is the primary form of TP53 deletion. Biallelic inactivation of TP53 is recognized as one of the most significant high-risk factors. Its frequency increases dynamically with disease progression, reaching up to 20% in RRMM, which serves as a hallmark of robust clonal evolution and aggressive biological behavior.⁴⁶ Monitoring the dynamic changes in TP53 status, particularly biallelic inactivation, before and after treatment serves as a powerful tool for detecting the emergence of drug-resistant clones and predicting early relapse. Patients who achieve MRD negativity but retain persistent TP53-aberrant clones face a significantly increased risk of recurrence.⁴⁶ In contrast, the independent prognostic value of del(13q) remains controversial, and it is now generally considered to act more as a surrogate marker or a contributory factor that synergizes with other high-risk abnormalities, such as t(4;14) and del(17p), to exacerbate poor outcomes, rather than an independent strong prognostic factor.⁴⁷ Additionally, some studies have suggested that IMiDs (eg, thalidomide) may help improve the prognosis of patients with isolated del(13q).⁴⁸ The t(4;14) translocation leads to overexpression of FGFR3 and MMSET genes. Shen et al's research indicated that MM patients with FGFR3 mutations had lower albumin levels, higher β 2-microglobulin levels, more advanced R-ISS stage, and shorter PFS and OS.⁴⁹ FGFR3 mutations and/or t(4;14) positivity are independent risk factors affecting prognosis.

In summary, a deeper understanding of HRCAs such as TP53 deletion, del(13q), and t(4;14) has shifted their role from mere prognostic markers to cornerstone decision-making tools guiding the whole-process management of MM, including risk assessment, treatment selection, and dynamic monitoring. Therefore, integrating cytogenetic information into the MRD monitoring system in the future—shifting the focus from “disease burden monitoring” to “high-risk clone kinetics surveillance”—would significantly enhance the precision of relapse warning.

Epigenetic Dysregulation

DNA Methylation

DNA methylation typically occurs at CpG islands in gene promoter regions, with hypermethylation leading to gene silencing. Ying et al found that the RASSF2A gene promoter was hypermethylated in MM, with correspondingly decreased mRNA expression levels, suggesting RASSF2A acts as a potential tumor suppressor gene inactivated by methylation in MM.⁵⁰ The study by Roy Choudhury demonstrated that the DNA methylation profiles of telomere-related genes (TRGs) are significantly altered in patients with newly diagnosed multiple myeloma (NDMM), and identified an epigenetically regulated panel comprising five TRGs that shows potential as early diagnostic biomarkers or therapeutic targets.⁵¹ Furthermore, the RRAD gene was downregulated in MM due to promoter hypermethylation; the demethylating agent decitabine could re-activate its expression and inhibit MM cell proliferation while inducing apoptosis.⁵² A preclinical study has demonstrated that DNA methyltransferase inhibitors can enhance the efficacy of immunotherapies, such as monoclonal antibodies (eg, daratumumab) or CAR-T cells, by reversing the epigenetic silencing of immune-related molecules, thereby offering new insights into combination therapeutic strategies.⁵³

Histone Modifications

Histone modifications influence gene transcription by altering chromatin structure. Dupere-Richer et al found that the histone demethylase KDM6A plays a tumor suppressor role in MM.⁵⁴ KDM6A, by demethylating H3K27me3, activates immune response genes including NLRC5 and CIITA, the latter being key regulators of major histocompatibility complex (MHC) genes. KDM6A deficiency leads to downregulated MHC expression, enabling tumor cells to evade immune surveillance. The study also found that using histone deacetylase inhibitors (HDACis) could partially restore MHC expression, providing a new combination immunotherapy strategy for tumors with KDM6A deficiency.⁵⁴ Studies have shown that HDACis, such as panobinostat, demonstrate considerable efficacy in RRMM, particularly in patient subgroups resistant to lenalidomide or within specific age demographics.⁵⁵ However, due to experimental limitations, most studies have focused on a single epigenetic mark. Future research should be directed toward more comprehensive

and systematic investigations. Establishing specific histone coding patterns, chromatin accessibility profiles, and DNA spatial organization maps, along with delineating their association maps throughout cancer progression, would be of great value.

Non-Coding RNAs

miRNAs, as key post-transcriptional regulators, are aberrantly expressed in MM. Cevik et al found that hsa-miR-29b-3p was upregulated in the bone marrow of NDMM patients and showed diagnostic potential for MM.⁵⁶ Its target genes include DNMT3A and TET2, linking miRNA regulation with DNA methylation/demethylation processes. Kaya et al confirmed that miR-145-5p inhibits cell proliferation and functions as a tumor suppressor in MM cells by directly targeting IGF1R and NRAS genes.¹⁴ Studies have demonstrated that the long non-coding RNA (lncRNA) FEZF1-AS1 promotes MM progression through the IGF2BP1/BZW2 signaling axis, and its expression levels may correlate with patient response to specific treatment regimens. Furthermore, targeting oncogenic lncRNAs—for example, via antisense oligonucleotide technology against MALAT1—has shown significant anti-myeloma activity in preclinical models, providing proof-of-concept for therapeutic strategies that target ncRNAs.⁵⁷ Due to their stable presence in body fluids, particularly peripheral blood, ncRNAs have emerged as highly promising biomarkers for non-invasive, dynamic monitoring. Research has indicated that the evolution of the serum exosomal miRNA profile in patients before and after treatment is highly consistent with MRD status, and its sensitivity may even surpass that of traditional M-protein detection.⁵⁷

Novel Cell Death Mode-Related Genes and Prognostic Models

Ferroptosis-Related Genes

Ferroptosis is an iron-dependent novel form of programmed cell death. Su et al, through integrative analysis, found that ferroptosis suppressor genes were progressively enriched during MM progression; their high expression was significantly associated with high ISS stage, R-ISS stage, and poor PFS and OS.⁵⁸ Kuang constructed a risk score model comprising three ferroptosis-related genes—EPAS1, RRM2, and FH—and validated its predictive power in an independent cohort. This model stratified patients into high-risk and low-risk groups, with the high-risk group having significantly shorter OS, and the risk score was an independent prognostic factor.⁵⁹ Wang et al studied the mechanism by which the key lipid metabolism pathway gene UCP2 regulates ferroptosis in MM cells, revealing its clinical value in MM.⁶⁰

Autophagy-Related Genes

Zhang et al screened 13 genes (eg, NKX2-3, BIRC5, PARP1) from autophagy-related genes (ARGs) to construct an autophagy-related prognostic model for MM.⁶¹ This model effectively distinguished high-risk from low-risk patients, with significantly lower survival rates in the high-risk group. Research by Ayna Duran et al also indicated that the impact of ARGs on MM patient survival depends on the chromosomal abnormality status, emphasizing the necessity of studying biological processes within specific genetic contexts.⁶²

Other Types of Prognostic Models

Beyond the aforementioned models, researchers have constructed various prognostic models based on mitophagy,⁶³ cuproptosis,^{64,65} angiogenesis-related genes,⁶⁶ and ubiquitination-related genes.⁶⁷ These models reveal the heterogeneity of MM from different biological perspectives and provide complementary prognostic information. For example, the mitophagy risk model constructed by Min et al not only predicted prognosis but also suggested that high-risk group patients might be more sensitive to drugs targeting TOP2A, such as etoposide and doxorubicin.⁶³

Clonal Evolution and Tumor Heterogeneity

MM is a disease with significant spatiotemporal heterogeneity, and its clonal architecture continuously evolves during disease progression. In her doctoral research, Chen et al compared bone marrow plasma cells (BM-PCs) and circulating plasma cells (CPCs) from the same patient, finding that they shared some mutations but also harbored unique ones,

indicating that CPCs might evolve from BM-PC clones and could carry new genetic alterations.⁵ Soloveva et al visually demonstrated the clonal diversity of MM at different sites by comparing mutation profiles from bone marrow, plasmacytomas, and ctDNA.¹² Clonal evolution is the fundamental cause of drug resistance in MM. Chen et al's research tracked clonal dynamics from diagnosis to relapse, finding that mutations in genes like *OBSCN*, *CACNA1H*, and *TP53* were selectively enriched under therapeutic pressure, becoming dominant clones that drove disease relapse.⁵ The study by Gooding et al also showed that 2q37 loss of *COP9* signalosome genes became enriched with increasing lines of therapy in patients exposed to IMiDs.³⁵ Therefore, static genomic profiling based on a single biopsy is insufficient to guide later-line therapy. In summary, our understanding of clonal evolution and tumor heterogeneity in MM has advanced from descriptive biology to a new phase of clinically integrated application. Genomics, as a core tool, is systematically mapping the dynamic landscape of the disease across its full cycle—from pathogenesis and treatment to relapse. Future challenges lie in translating real-time, dynamic clonal monitoring into clinically actionable decision-making systems, as well as developing novel therapies capable of effectively intervening in the process of clonal evolution.

Discussion and Future Perspectives

Research in MM genomics has advanced our understanding of this disease from a purely morphological level to a complex molecular classification. Current progress is highlighted in the following aspects: 1) Driver Events: From common *RAS*, *TP53* mutations to emerging epigenetic regulators, driver events are continuously being expanded and refined. 2) High-Risk Genetic Abnormalities: For traditional high-risk abnormalities like +1q, del(17p), researchers are striving to elucidate their specific driver genes and pathogenic mechanisms. 3) Prognostic Tools: Multi-gene models incorporating novel biological processes like ferroptosis and autophagy provide precise risk stratification tools beyond traditional staging systems. 4) Clonal Evolution: MM is a dynamically evolving ecosystem, providing a theoretical basis for understanding drug resistance and designing curative strategies.

However, challenges remain. Currently, the gold standard for efficacy monitoring in MM—bone marrow-based MRD assessment—is invasive and logistically cumbersome, hindering its repeated use. Moreover, it may produce false-negative results due to multifocal or extramedullary lesions, as well as suboptimal bone marrow aspiration or biopsy quality. Therefore, there is an urgent clinical need to develop easily interpretable, minimally invasive novel liquid biopsy biomarkers (such as ctDNA, circulating tumor cells, and miRNAs) to enable precise prognostic prediction and dynamic monitoring. However, the sensitivity of current blood-based assays appears to fall short of that achieved with bone marrow samples, and further research is warranted to enhance their sensitivity. This notion has been corroborated in the studies by Li⁶⁸ and Gozzetti.⁶⁹ Second, translating vast genomic information into effective treatment strategies is a core future task. For example, HDAC inhibitor combination therapy for *KDM6A* deficiency,⁵⁴ CELMoDs drugs targeting the *CRBN* complex,³⁵ and targeted therapy for specific mutations (eg, *BRAF* V600E) are all promising directions. Third, Despite the acknowledged potential of multi-omics integration to advance precision medicine, significant barriers—such as small sample sizes, technical batch effects, and lack of robust external validation—continue to impede its clinical applicability. Combining data from genomics, transcriptomics, proteomics, and the immune microenvironment is essential for delineating a holistic landscape of disease pathogenesis and facilitating the discovery of clinically actionable insights.

Genomics research in MM will continue to deepen clinical translation, promoting molecular classification based on NGS and WGS analysis to become routine diagnostic standards, and developing precise targeted treatment pathways on this foundation. Concurrently, immunogenomics will become a key focus area, aiming to identify biomarkers predictive of responses to immunotherapies like CAR-T cell therapy and bispecific antibodies by deeply investigating the interaction between the tumor genome and anti-tumor immunity. As artificial intelligence and machine learning become increasingly integral to biomedical research, their potential to construct reliable prognostic models and treatment recommendation systems hinges on the effective synthesis of multi-omics and clinical data. A key priority lies in ensuring algorithmic robustness and interpretability, while simultaneously mitigating challenges related to validation deficits to facilitate clinical translation. Notably, genomic exploration of MGUS and smoldering multiple myeloma will also intensify, aiming to identify early high-risk markers for transformation to active MM, ultimately enabling early intervention and disease prevention.

Conclusion

In summary, genomics research in MM has achieved revolutionary progress, profoundly revealing its genetic complexity, clonal dynamic evolution patterns, and molecular-level heterogeneity. These discoveries have not only refined the risk stratification system for MM but, more importantly, laid a solid scientific foundation for developing novel targeted therapies and realizing individualized precision medicine. With the continuous advancement of sequencing technologies, decreasing detection costs, and improved capabilities for multi-omics integrative analysis, we have reason to believe that in the future, every MM patient will possess their own genomic “identity card”, receive the most effective treatment accordingly, ultimately improving prognosis and moving towards a cure.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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