

ORIGINAL ARTICLE

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Clustering Based on Innate Immunity Reveals Differential Dysregulation Based on Disease Severity in Myelodysplastic Neoplasms

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ABSTRACT

Myelodysplastic neoplasms (MDS) are clonal hematologic disorders characterized by ineffective hematopoiesis and a variable risk of progression to acute myeloid leukemia (AML). Despite growing recognition of the role of innate immunity in MDS pathogenesis, the precise mechanisms remain unclear. In this study, we analyzed bone marrow CD34+ expression data from 183 MDS patients to investigate the impact of the Toll-like receptor (TLR) pathway on disease progression. Six key innate immunity genes (*IRAK1*, *IRAK2*, *IRAK4*, *MYD88*, *TRAF6*, and *NF-κB1*) were used to define two distinct immune clusters: a hyperactive immune cluster (HIC) and a moderate immune cluster (MIC). The HIC was enriched in 155 immune-related pathways and showed higher infiltration of activated natural killer cells and M1 macrophages, while the MIC exhibited increased infiltration of naïve B cells and mast cells. Differential expression analysis identified 35 genes that distinguished the clusters. Validation in an independent cohort of 82 patients revealed that reduced expression of these genes correlated with markers of advanced disease, including lower hemoglobin levels, lower neutrophil counts, altered cytogenetics, and higher bone marrow blast percentages. These findings underscore the critical role of immune dysregulation in MDS progression and highlight novel therapeutic opportunities within the innate immunity pathway for tailored interventions.

Abbreviations: AML, Acute Myeloid Leukemia; AML-EM, Acute Myeloid Leukemia Related to Myelodysplasia; BMDPs, Bone Marrow Pooled Cells; Cq, Quantification Cycle; DEGs, Differentially Expressed Genes; GSEA, Gene Set Enrichment Analysis; HIC, Hyperactive Immune Cluster; IDPC, Hematopoietic Stem and Progenitor Cells; IL, Interleukin; IRAK, Interleukin-1 Receptor Associated Kinase; KEGG, Kyoto Encyclopedia of Genes and Genomes; MDS, Myelodysplastic Neoplasms; MIC, Moderate Immune Cluster; NF-κB, Nuclear Factor kappa B; TLR, Toll-Like Receptor.

The first two authors contributed equally to this article.

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