Editorial

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Clonal Hematopoiesis and Inflammation: Unraveling Links to Aging and Disease

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Introduction

Clonal hematopoiesis (CH) is the age-associated expansion of blood cell clones carrying somatic mutations in hematopoietic stem cells. While often asymptomatic, CH is increasingly recognized as a driver of systemic inflammation and a risk factor for cardiovascular disease, hematologic malignancies, and other chronic conditions. Understanding the relationship between clonal hematopoiesis and inflammatory processes is critical for deciphering age-related disease mechanisms and developing targeted interventions.

Mechanisms Linking Clonal Hematopoiesis to Inflammation

CH commonly involves mutations in genes such as DNMT3A, TET2, and ASXL1, which regulate epigenetic modifications and hematopoietic differentiation. Mutated clones often produce myeloid cells with heightened pro-inflammatory activity, secreting cytokines like IL-1β, IL-6, and TNF-α. These inflammatory signals contribute to tissue damage, accelerate atherosclerosis, and increase susceptibility to systemic inflammatory conditions.

Additionally, CH-driven inflammation can create a feedback loop that promotes further clonal expansion and immune dysregulation. Emerging evidence suggests that specific CH mutations may confer varying inflammatory potentials, emphasizing the heterogeneity of this phenomenon.

Clinical Implications

The intersection of CH and inflammation has significant clinical relevance. Individuals with CH are at higher risk of cardiovascular events, independent of traditional risk factors, and may develop low-grade chronic inflammation. Identifying CH through next-generation sequencing can help stratify patients for cardiovascular and oncologic risk monitoring.

Therapeutic strategies targeting CH-related inflammation, including anti-cytokine therapies or interventions modulating clonal expansion, are being explored. Understanding how different CH mutations influence inflammatory pathways will be crucial for developing precision therapies aimed at reducing disease burden while preserving normal hematopoiesis.

Conclusion

Clonal hematopoiesis links somatic mutations in hematopoietic cells to systemic inflammation and age-related disease. By elucidating the molecular mechanisms connecting clonal expansion to pro-inflammatory states, researchers are uncovering new opportunities for risk stratification and targeted intervention. Continued study of CH and inflammation promises to advance our understanding of aging, cardiovascular disease, and hematologic malignancies, ultimately improving patient outcomes through personalized approaches.