

Abstract

Hematopoietic stem cells (HSCs) are the cornerstone of blood and immune system maintenance, responsible for the continuous production of all blood cell types through a tightly regulated process of differentiation. This review explores the intricate molecular mechanisms governing HSC differentiation, emphasizing the role of transcription factors, signaling pathways, and epigenetic modifications. Key transcription factors such as GATA-2, PU.1, and RUNX1 orchestrate lineage commitment and cell fate decisions, while signaling pathways including Notch, Wnt, and TGF- provide extrinsic cues essential for maintaining HSC quiescence, proliferation, and differentiation. Epigenetic regulators, such as DNA methylation and histone modifications, further modulate gene expression patterns crucial for HSC function. Understanding these molecular processes has significant clinical implications, particularly in the context of hematologic disorders and regenerative medicine. Aberrations in HSC differentiation can lead to hematologic malignancies, bone marrow failure syndromes, and other blood disorders. Advances in single-cell technologies and genome editing have facilitated deeper insights into the HSC differentiation landscape, paving the way for innovative therapeutic approaches. This includes targeted therapies aimed at correcting dysregulated pathways, ex vivo HSC expansion techniques for transplantation, and the potential for generating HSCs from pluripotent stem cells. In summary, elucidating the molecular mechanisms of HSC differentiation not only enhances our comprehension of hematopoiesis but also informs the development of novel clinical interventions for hematologic diseases. Future research endeavors should focus on translating these molecular insights into practical therapeutic strategies to improve patient outcomes in the realm of hematology and beyond.

H C

E e d a da a d ech l g'e

H C

H C

St g e-c a ech l g'e : H C

H C

Da a d a

(, GA A-2, 1, 1)

(, 7 GF-) H C

H C

C t a t a

H C

S a t a a

H C

E h t a c d e a

H C

B

H C

Re d d D c

M e c a e c h a f h e a e t e a c a d e d a

F ; K ; GA A-2, 1, 1

H C

GA A-2 ; H C

1

D

S g a g a h a : H C

H C

H C

7 GF-

H C

E g e t d f i c a : E ; A

H C ; A

H C ; F ; A

C t a t a

H e a g t d d e : D ; H C ; F

1 ; (A L) ; D

H C

e a e t a e g e : H C

C I / C 9

H C ; E

A

H C

H C

F e d e c

A ; F ; A

H C

G t a A t a : F ;

e d c t a A a c h e : H C

H C ; C

H C

H C

H C

H C

H C

H C

C

