生物化学セミナー

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『Gene regulatory networks orchestrating the B-lymphopoiesis』

We have demonstrated that hematopoietic multipotent progenitor cells (MPPs) are promoted to adopt B cell fate choice by Ebf1, which is induced by the combined activities of E2A and PU.1, and IL7 receptor-mediated signaling. Furthermore, we have shown that substantial epigenetic modifications and extensive rewiring of chromatin folding facilitate interaction between gene promoters and their regulatory elements concurrently with B cell fate commitment. Ebf1 is required for the development of pre-B cells, whereas IRF4 is required for the developmental progression from the pre-B to the mature B cell stage. In the absence of IRF-4 and IRF-8, B cell development is arrested at the large pre-B cell stage, and mutant cells are unable to undergo light-chain recombination. Complementing IRF4 expression or lowering IL7R signaling initiates Igκ rearrangement, and cells transit from the pre-B to the B cell stage. But it's not clear how these divergent regulatory inputs resolve to initiate Igκ recombination at the pre-B cell stage. When IL-7R signaling is downregulated, several genes that are needed for B cell development are activated. These genes include the TET family of enzymes, which helps reverse DNA methylation at specific loci. Together, these studies affirm the notion that Ebf1 and IRF4 define genetic and epigenetic networks that interplay and coordinate in regulating gene expression and promoting the orderly development of B cells.

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