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Sommario/riassunto	The process of generating differentiated cell types performing specific effector functions from their respective undifferentiated precursors is dictated by extracellular signals and the recipient cell's ability to transmit those signals to effect changes in cellular functions. One major mechanism for bringing about such changes is at the level of transcription. Thus, inducing transcription of previously silent genes and suppressing active genes in response to the extracellular signal can result in acquiring new functions by the cells. The transcriptional machinery, comprising of RNA Polymerase II and associated general transcription factors, assemble at the core promoter of eukaryotic protein coding genes. The rate and/or stability of formation of this machinery dictate the transcriptional regulation of the corresponding gene, which can be at the level of chromatin regulation as well as enhancer-promoter communication. Such coordinated temporal and spatial regulation of gene expression in response to specific signals determines lineage differentiation, cellular proliferation and development. Every event in the life cycle of a lymphocyte is modulated

by the signals they receive. For instance, expression of the B cell antigen receptor (BCR) on the surface of B cells is a hallmark of various stages of B cell development--signaling via the BCR is important both during early/antigen independent (tonic) and late/antigen dependent phases of development. Despite the established requirement for BCR signaling during various phases of B cell maturation, how BCR signaling connects to chromatin changes and downstream transcriptional pathways in each step of development remains poorly understood. Similar questions also remain in other cells of the immune system. Moreover, how the enhancers communicate to the promoters in a stage specific fashion and in the context of chromatin also remain unclear. Chromatin modifiers are generally present and active in most cell types. How could then there be differences in chromatin architecture dependent on a particular stage of development? The B (and T) lymphocytes also perform a unique developmental program because they have an unparalleled genetic makeup—the genetic loci that encode their cell surface receptors are in an ‘unrearranged’ or “germline” configuration during the early stages of development. Thus, they not only express stage specific genes and transcription factors during each developmental stage, they need to undergo rearrangement of their cognate receptor loci in a strictly ordered fashion to generate a pool of receptor proteins, each capable of recognizing a specific antigen, which they encounter at a much later step. Hence, there must be a strict negotiation between the recombination machinery and the transcriptional machinery at every developmental step of the way. Importantly, along the way, the B cells expressing receptors capable of recognizing self-antigens must be eliminated to avoid autoimmune responses and only those cells capable of recognizing foreign-antigens are preserved to reach peripheral organs where they eventually meet pathogens. How are these processes coordinately regulated in a stage specific fashion and what role does chromatin play? Are the rules of engagement different in innate versus adaptive immune responses? Here we seek to address some of these questions and provide our current understanding of signal-induced chromatin and transcriptional regulation of the immune system.
