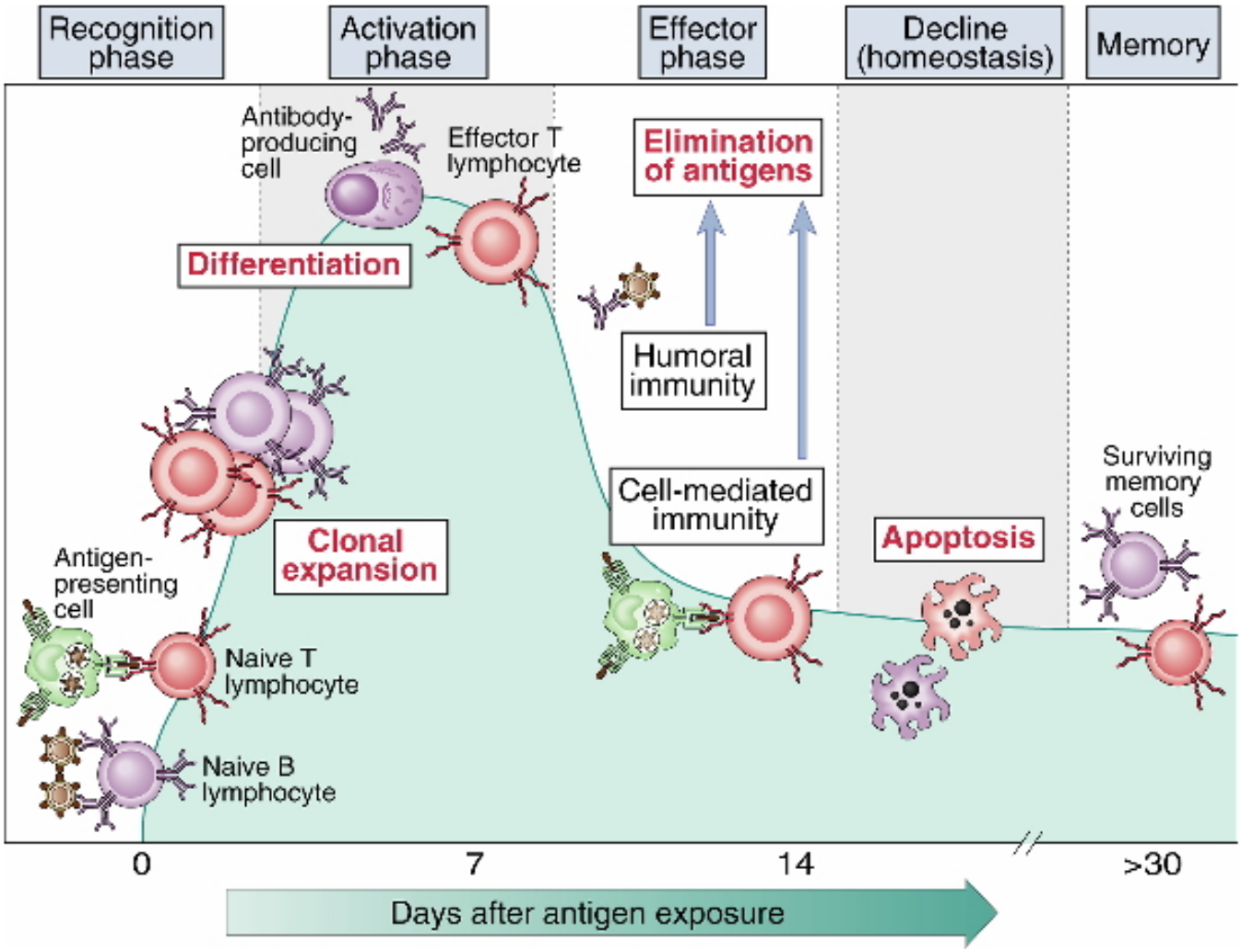
Lecture 1.
Learning Objectives and Summary



1. Introduction to the immune system

Learning objectives:

1. Understand the overall organization of the immune system
2. Conceptualize how the collection of individual clones of lymphocytes (termed the "immune repertoire") arises from rearrangement within two genetic loci: the Ig gene in B cells and the antigen receptor in T cells.
3. Learn how "clonal selection" allows for the expansion of a limited number of antigen-recognizing lymphocytes in response to an specific antigenic stimulus
4. Appreciate the structure and function of MHC molecules.
5. Understand how multiple signals converge on T cells to effect clonal expansion.
6. Begin to appreciate the significance of maintaining a state of immune tolerance sufficient to prevent the emergence of autoimmunity.

SUMMARY

1. The immune system is complex. Try to understand it in terms of specific functional modules.

2. Diversity in antigen recognition is accomplished, in part, by rearrangements in the Ig and TCR loci. This occurs in the bone marrow and thymus, respectively.

3. The T and B cell repertoire determines the spectrum of antigens that can be recognized in an individual's lifetime. The nature of this repertoire is determined by the Major Histocompatibility Complex (MHC), which binds peptide antigen.

4. In the primary immune response, antigen presenting cells (APCs) present antigen bound to MHC molecules to T cells in the lymph nodes and spleen. T cells "help" B cells to develop further and clonally expand in germinal centers of these organs.

5. Lymphocytes exit these organs to become effector or memory cells. Effector cells secrete Ab (plasma cells) or cytokines (CD4+ T cells) and kill virally-infected cells (CD8+ T cells). Memory cells re-circulate until they encounter Ag again.

6. The immune system is tightly regulated. It exists in a delicate balance of immunity and tolerance. A lack of tolerance to self antigen coupled to excessive immune activation (or inadequate immune suppression) can lead to autoimmunity.