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EDITORIAL

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Components of specific immunity in host defense

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Invading microbial pathogens are immediately trapped in the nearest specialized organ, known as lymph nodes, where necessary immune responses will develop to clear the microbes. The pathogen is transported to the lymph nodes through a network of channels and, collectively, called as lymphatic system. In these organs, microbial pathogens are ingested by the phagocytic or similar types of cells known as antigen presenting cells (APCs) and its component is processed and presented by these cells to the T cells to develop appropriate immune responses. Another cell type known as B cells produce wide range of molecules that bind to the surfaces of the microbes known as antibody and subsequently inactivate the pathogen. Based on the pathogen, T cells can differentiate into various functional subtypes to provide necessary pathogen-specific immune responses. In addition to microbial pathogens, these cells play an important role in immune homeostasis. The dysregulated B and T cells can also result to the immune catastrophe that may result to the development of cancer or autoimmune diseases. This issue of *International Reviews of Immunology* discusses some aspects of the tertiary lymphoid structure, APCs, B and T cells in normal immune function and in immunopathogenesis (Figure 1).

Inflammation is an essential protective response to infection; however, excessive inflammation could be detrimental and can cause collateral damage to the host. It is also associated with noninfectious diseases such as cancer, autoimmune diseases, and transplant rejection. Heightened inflammation can result in the formation of new lymphoid tissue aggregates known as Ectopic lymphoid-like structures (ELS) or tertiary lymphoid organ that consist of various professional immune cells like B and T cells. The first review article in this issue by Marinkovic *et al*. discusses the organization and biology of ELS in different disease set-ups and its role in disease development. The article also discusses ELS as a therapeutic target for various complex diseases [1]. The audience for this article will be researchers working in different fields of immune-biology, particularly scientists working in development of diagnostics and therapeutics (Figure 1).

The dynamicity of immune cells and soluble mediators are key for host defense and immune homeostasis. It is mediated through Immune cell trafficking via complex signaling pathways. The movement of immune cells are primarily supported by cell surface molecule like leukocyte adhesion molecules, integrins, lectins, tetraspanines, and/or induction of chemokines and chemokine receptors and so on. A member of the semaphorin family protein known as semaphorin 4D (Sema4D) is reported to play an important role in axon guidance and an important role in immunity, particularly, cell migration. The second review in this issue by Kuklin *et al.* discusses the role of Sema4D in immune regulation in cell migration. The article also discusses the underline molecular mechanism of Sema4D-mediated cell migration [2]. This article will be interesting to immunologists, neurologists, and scientists working in neuroimmunology (Figure 1).

Post-translational modification of proteins is most essential for maintenance of protein structure and its biological function. Various proteins such as receptors, ligand, immunoglobulins and so on are few examples of glycosylated protein. The immunoglobulin is one example which is highly glycosylate and this glycosylation is needed for the biological function. The third review article of this issue by Gomez-Henao *et al.* describes the importance of glycosylation of proteins involved in T cells and APC interaction and its functional impact on various immunological processes such as cytokine production and T cell proliferation. The article discusses dysregulated post-translational machinery or lack of glycosylation that results in the development of immunopathology such as autoimmune diseases. The article also suggests that fundamental understanding of glycosylation that

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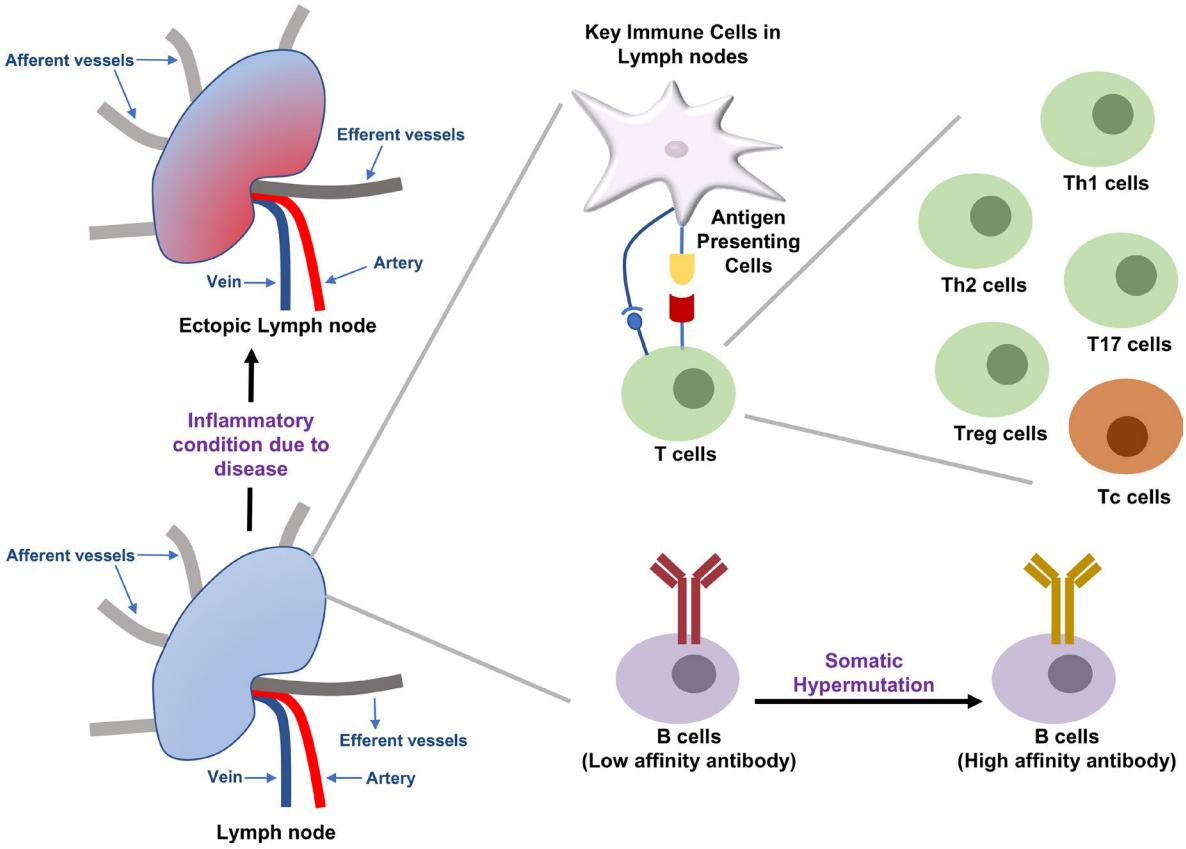

Figure 1. Adaptive immune components in host defense.

can also help in the development of immunotherapies [3]. This article will be interesting to wide range of researchers working in fundamental and translational immunology (Figure 1).

The mammalian host can produce numerous kinds of antibodies against numerous biotic and abiotic entities. The generation of diverse antibodies by limited genome was a challenging quest solved by constant efforts of several immunologist, particularly by Susumu Tonegawa whose work received the prestigious Nobel prize for providing molecular basis of antibody diversity in 1987. Initially, B cells produce low affinity antibodies against the antigen and have a limited role in pathogen clearance. To produce high affinity antibodies, B cells undergo the phenomena known as somatic hypermutation (SHM) and class switching recombination (CSR) to efficiently clear the invading pathogen. The fourth review article of this issue by Jaiswal *et al.* focuses on SHM and CSR in antibody diversity, particularly a crucial enzyme known as Activation-induced cytidine deaminase (AID) and molecular events taking place during antibody diversity [4]. This article will be interesting to the fundamental immunologist working or investigating B cells.

CD4+ T cells or T helper (Th) cells are master regulators of adaptive or pathogen-specific immunity. Th cells play a crucial role in shaping adaptive immunity via differentiating into various functional subtypes such as Th1, Th2, Th17, Th9 and Treg cell. These cell types not only activate immunity but also dampen the adaptive immunity and dysregulation in these cells, which may result in oncogenesis or autoimmune disease. The last article in this issue by Zohouri *et al.* discusses the role of one subset of Th cells known as CD4+CD25-FoxP3+ T cells in cancer and autoimmune diseases [5]. This article will be interesting to broad readers of fundamental immunology (Figure 1).

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