

The B Lymphocyte

Clyde Schultz*

Department of Biology, University of Calgary, Calgary, Alberta, Canada

Abstract

B lymphocytes, (B cells) are one of the most important cell types in humans and higher animals. They are the key cell involved with antibody production in the form of programming plasma cells which produce specific antibody that targets antigens or pathogens. B lymphocytes have evolved in concert with other cells such as macrophages, T Lymphocytes and the cytokine system as the primary defense against disease. Vaccines largely function by eliciting an antibody response from B lymphocytes.

Introduction

B and T cells (lymphocytes) are known more broadly as white blood cells T lymphocytes originate in the thymus while B lymphocytes originate in the bone marrow and lymphatic system. B cells are the backbone of humoral immunity that in they are ultimately program plasma cells which are actual antibody producing cell. B cells make up about 20% of the total number of lymphocytes in the body at any given time. In most mammals B cells mature in the bone marrow, however in birds B cells mature in the Bursa of Fabricius [1]. This discovery in birds led to the designation of “B” for bursa not bone marrow.

Immune interactions

Development of B cells

B lymphocytes develop from hematopoietic stem cells (HSC) that occur in the bone marrow [2,3]. There are several stages of development or differentiation, beginning with the development of HSC into multipotent progenitor cells (MPP) or early or pro-B cells. There development into immature B cells is enhanced by different gene expression patterns. The development and additions of B cell receptors on the surface of the cells occurs during development in the bone marrow. Immature B cells must receive antigen independent signaling which involves B cell receptors or development of a particular B cell will not continue. If however, the B cell receptor binds to self-antigen then B cells may either; undergo clonal deletion, editing of receptors, anergy (tolerance) or ignoring of certain negative process signals and continual development [4]. At the end of this process, immature but fully formed non-programmed B cells will matriculate to the spleen passing through the transitional stages T1 (travel to and entry into the spleen) and T2 which occurs in the spleen and further development into either marginal zone or follicular B cells [5]. Once this occurs, they are naïve, but mature B cells and may be programmed to respond to specific antigen. The B cell at this stage acts as an antigen presenting cell and may endocytose pathogens. Sub parts of the antigen or pathogen are uploaded to MHCII molecules and presented to a subset of T cells referred to as T Helper cells [5,6]. This interaction of the T helper cell with the MHCII region on the B lymphocyte activates the B lymphocyte.

B cell activation and plasma cell development

Activation of B cells occurs in the spleen or lymph nodes where the cells are exposed to antigens. At one of these lymphoid organs,

activation of B cells occurs by interaction via the B cell receptor(s) with an antigen [6]. Up to this stage a B cell can be programmed to respond to any antigen (pathogen). However, once this interaction occurs (with the interaction of T helper cells), the pattern for ultimate antibody response is programmed for a specific target. Current thought in immunology is that B cells are activated by receptor diffusion through membranes and there is interaction with Lck and CD 45 with somewhat equal frequency, thus creating an equilibrium between phosphorylation and non-phosphorylation. When a mature but unprogrammed B cell comes into contact with an antigen that CD 45 is displaced which provides a mechanism for phosphorylation of B cell receptors and thus initiation of signal transduction. Looking at this in relation to the B cells suggests, follicular B cells will tend toward T cell-independent activation. Marginal zone cells will tend to undergo T-Cell dependent activation [7]. Once activated the B cells will differentiate to a mature plasma cell. It is the plasma cell that produces specific antibody. Plasma cells are terminal cells. They are large lymphocytes that have a large basophilic cytoplasm. They will eventually die and not sub-divide. Plasma cells will produce antibody up to thousands of antibody molecules per second per cell [8].

T cell dependent activation of B cells

Most antigens activate B cells with the assistance of T cells. T cells must be present for this type of immunologic reaction to occur, which leads to the formation of antibody to antigen. This type of activation begins with a B cell receptor binding to a T cell dependent antigen. Endocytosis of the antigen to the interior of the B cell occurs, there is degradation and reconfiguration of the antigen followed by “presentation” of the antigen to T cells as degraded peptide pieces [9]. These “pieces” interact with the MHCII molecules on the cell membrane. The sub set of T cells referred to as helper or (H) cells bind the MHCII-antigen peptide segments through the T cell receptor. At this point IL-4

*Correspondence to: Clyde Schultz, Department of Biology, University of Calgary 2500 University Drive, Calgary, Alberta, Canada T2N 1N4, Tel: 403-220-5278; E-mail: schultzc@ucalgary.ca

Key words: bursa, immune, antibody, plasma, cell

Received: August 23, 2021; **Accepted:** September 03, 2021; **Published:** September 06, 2021

and IL-21 cytokines are expressed which serve to enhance activation as well as the surface receptor CD 40. All these effector molecules and receptors act to enhance and promote B cell proliferation and sustains T cell growth and differentiation. At this juncture activation is completed [10].

B cells then undergo a multistep process that yields some acute protection but also plasma cells which are the cell type that produce the antibody molecules against the programmed antigen, and B memory cells which become active should that antigen reappear. Some examples of antigens that are considered T cell dependent are polio, measles, and small pox.

T cell independent activation of B cells

Activation of B cells can occur without the assistance of T cells. Activation signals are received from binding of microbial “particulates” in the form of toll-like receptors. These cell-independent antigens include polysaccharides and some viruses, including potentially the SARS-Cov-2 virus. Because T cells are not as a rule involved in activation of B cells, there is often a non-existent or limited immune memory of the antigen interaction (event). For the practical purposes of vaccine administration, booster shot administration may be required.

Diseases as a result of B cell deficiency

B lymphocytes and the associated plasma cells are essential for healthy immune development and thus disease resistance [11]. Abnormal B cells and related pathology include autoimmune diseases such as multiple sclerosis, type 1 diabetes and rheumatoid arthritis.

While there are treatments for these diseases there is no cure as the underlying B cell deficiency is not treated.

References

1. Cooper C (2015) The early history of B cells. *Nat Rev Immunol* 15: 191-197. [[Crossref](#)]
2. Fischer U, Yang JJ, Ikawa T, Hein D, Vicente-Dueñas C, et al. (2020) Cell Fate Decisions: The role of transcription factors in early B cell development and Leukemia. *Blood Cancer Discov.* 1: 224-233. [[Crossref](#)]
3. Kondo M (2010) Lymphoid and myeloid lineage commitment in multipotent hematopoietic progenitors. *Immunol Rev* 238: 37-46. [[Crossref](#)]
4. LeBien T, Tedder TF (2008) B lymphocytes: How they develop and function. *Blood* 112: 1570-1580. [[Crossref](#)]
5. Loder F, Mutschler B, Ray RJ, Paige CJ, Sideras P (1999) B cell development in the spleen takes place in discrete steps and is determined by the quality of the B cell receptor-derived signals. *J Exp Med* 190: 75-90. [[Crossref](#)]
6. Yuseff MI, Pierobon P, Reversat A, Lennon-Duménil AM (2013) How B cells capture, process and present antigens: a crucial role for cell polarity. *Nature Reviews Immunology* 13: 475-486. [[Crossref](#)]
7. Nuff S, Hodgkin PD, Tarlinton DM, Corcoran LM (2015) The generation of antibody secreting plasma cells. *Nature Reviews Immunology.* 15: 160-171. [[Crossref](#)]
8. Kuby J (2007) *Kuby Immunology*. Freeman WH (Eds) San Francisco, p. 13.
9. Blum J, Wearsch PA, Cresswell P (2013) Pathways of antigen processing. *Annu Rev Immunol* 31: 443-473. [[Crossref](#)]
10. Cotty S (2015) A brief history of T cell help to B cells. *Nat Rev Immunol* 15: 185-189. [[Crossref](#)]
11. Yanaba K (2008) B lymphocyte contributions to human autoimmune disease. *Immunol Rev* 223: 284-299. [[Crossref](#)]