# Research and Reviews: Journal of Medical and Health Sciences

## **Types Of Immunity**

## Manu Tomar

<sup>1</sup>Department of Biotechnology, Abdul Kalam Technical University, Lucknow, India

## **Review Article**

Abstract

Received: 25/07/2016 Accepted: 19/08/2016 Published: 28/08/2016

#### \*For Correspondence

Manu Tomar, Department of Biotechnology, Abdul Kalam Technical University, Lucknow, India

#### E-Mail:

manutomar234@gmail.com

Keywords: immunity, natural killer cell, eosinophils, mast cells, basophil, neutrophil, macrophage, dendritic cell, adaptive immunity, immunization, innate immunity As immunity is the power of our body to kill the germs or pathogenic substances entered in our body so that we can easily maintain our health. This article reviews different types of immunity and their functions. Also reviews about different types of cell which are involved in that particular immunity.

## **INTRODUCTION**

Immunity is the capability of the body to fight against pathogenic microbes in order to prevent us from infectious diseases. Immunity involves 2 types of components i.e., specific and nonspecific components <sup>[1-5]</sup>. Specific components acclimate themselves to each and every types of new disease encountered and hence able to produce pathogen-specific immunity whereas nonspecific components behave as an eliminators or chemical mediators of pathogens regardless of antigenic specificity <sup>[6-10]</sup>.

## **TYPES OF IMMUNITY**

There are basically two types of immunity:

- 1) Innate immunity
- 2) Adaptive immunity

#### 1) Innate immune system

This is basically a natural immunity of an individual that's why it is also called as non-specific immune system or we can say in-born immunity. It includes all the mechanisms that protect the host from pathogenic microbes. It provide prompt defense against infection but does not create any memory against a particular pathogen for future encountered <sup>[11-17]</sup>. Hence its response is independent for an antigen <sup>[18]</sup>. Functions of innate immune system are:

- Behaves as a physical and chemical boundary to pathogenic agents <sup>[19]</sup>.
- Recognition and eradication of foreign substances present in our body with the help of leukocytes <sup>[20,21]</sup>.
- Enrolling immune cells to the section of germs, through the secretion of chemical substances called cytokines <sup>[22]</sup>.
- Stimulation of the adaptive immune system through a process known as antigen presentation.

• Recognition of bacteria by the stimulation of the complement cascade

#### Cells involved in innate immunity:

White blood cells are main source to induce innate immunity. These cells are also known as leukocytes. WBC differs from other cells of the body in that they are not associated with a specific organ and hence their function is autonomous <sup>[23]</sup> These cells are the products of hematopoietic stem cells <sup>[24,25]</sup>. They move freely inside the plasma and capture infectious particles and invading microorganisms.

These leukocytes cells include: Natural killer cells, eosinophils, mast cells, basophils; and the phagocytic cells comprises neutrophils, macrophages, and dendritic cells, and works along with the immune system by eliminating pathogenic agents which causes infection.

**NK cells** are the basic part of innate immunity that does not directly attack the foreign substances. The role of NK cells is like that of cytotoxic T cells . These cells are called natural killer because they do not require any activation. They kill the microbes as soon as they produced inside the body <sup>[26-29]</sup>.

**Eosinophils** not only digest the pathogenic organism which enters into our body but also target those foreign cells which are enlarging enough to digest. Their efficiency is low as compare to neutrophils and basophils.

**Basophils** play an important role in immunity. Due to activation of these cells histamine-releasing basophils are produced. They also take part in allergic reactions. For e.g.: Asthma. Toxin proteins are also released due to activation of eosinophil which are effective in killing parasites but may also damage other tissue during an allergic reaction <sup>[30]</sup>.

**Mast cells** are associated with lesion curing and defense against pathogens. These cells are linked with allergic reactions <sup>[31-34]</sup>. After activation, mast cells rapidly produce granules which are rich in histamine along with chemokine <sup>[35]</sup>. Histamine recruits neutrophils and macrophages <sup>[36-39]</sup>.

**Phagocyte** are immune cells that engulf pathogens .During engulfing, a phagocyte enlarge its plasma membrane, folding the membrane around the particle until it is encompassed. After that it kills and digests the pathogen.

**Neutrophils** are also called as granulocytes due to the existence of granules in their cytoplasm. These granules consists a mixture of toxic substances that hinders the growth of microbes <sup>[40-42]</sup>. Like that of macrophages, neutrophils also kill the pathogens through respiratory burst process <sup>[43-46]</sup>.

**Macrophage** means large eaters. These are the most efficient phagocytes and can phagocytize large number of bacteria or microbes. Bacterial molecule binds to receptor on the surface of a macrophage and kills the bacteria through the process of respiratory burst. Bacteria also stimulate the macrophage to produce chemokine, which gathers other cell at the site of infection <sup>[47]</sup>.

**Cytokines** operate only with that type of cells which having specific receptors on their surface. Each cytokines are pleiotropic i.e., having different effects on different cells in nature. Sometimes cytokines also considered as chemokine because both can reenergize each other.

**Dendritic cells** are present in tissues that comes in acquaintance with the external environment, mainly with the skin (i.e. Langerhans cells), and the inner mucosal lining of the stomach, nose and lungs <sup>[48]</sup>. Dendritic cells play an important role in the mechanism of antigen presentation, and provide a network between the innate and adaptive immune system <sup>[49]</sup>.

### 2) Adaptive immune system

Adaptive immune system also called acquired immune system or rarely known as specific immune system. It is a subsystem of overall immune system because it composed of systemic cells and processes that inhibits pathogen growth. As this system is destructive in nature so it is essential that they are made in such a manner so that they only target to foreign particles not to it. To recognize self and non-self-components inside the body makes adaptive immunity distinctive one from other type of immunity <sup>[50-52]</sup>. Acquired immune system produces an immunological memory after early response to a specific pathogen, and leads to an enhanced response when encounters with the same pathogen next time. This process is the basis of vaccination in adaptive immune system. Adaptive immune system comprises both the components i.e., humoral and cell-mediated immunity. Functions of the acquired immune system are:

- Pathogens are remembered by the development of immunological memory through memory B cells and memory T cells.
- Recognition of self and non- self-antigens during the process of antigen presentation.
- Activation of responses to eliminate specific pathogen-infected cells.

#### Cells involved in adaptive immunity

T and B lymphocytes are the major cells of the acquired immune system. These cells are derived from the same hematopoietic stem cells. B cells involved in the humoral immune response while T cells play an important role in cell-mediated immune responses <sup>[53-56]</sup>.

## T lymphocytes

Cytotoxic T cells also called cytotoxic T-lymphocyte or TC cells .T cells induce the death of that cells which are infected with viruses <sup>[57,58]</sup>. When T-cell receptor (TCR) strongly binds with a peptide-bound MHC class-I molecule, naive cytotoxic T cells are activated. T cells are of two types: Cytotoxic T cells and Helper T cells.

## Cytotoxic T cells

Cytotoxic T cells are also called CD8+ lymphocytes <sup>[59]</sup>. These cells mainly target to those cells which are infected with virus. For e.g. HIV infected cells <sup>[60-64]</sup>.

## Helper T-cells

Helper T cells are also called CD4+ lymphocytes <sup>[65].</sup> These cells cannot kill infected cells, but they can regulate the immune response of other cells to perform their functions properly <sup>[66,67]</sup>.

## **B** lymphocytes

B Cells are the primary cells involved in the formation of antibodies and hence called as humoral immunity. 5 different types of antibody produced by B-lymphocytes are: IgA, IgD, IgE, IgG, and IgM <sup>[68-70]</sup>. These Abs are evolved to handle different types of antigens. Antibodies are produced during the stimulation of B cells, which recognizing a uncommon antigen and hence neutralizing particular pathogens <sup>[71-73]</sup>.

Adaptive immunity is further categorized into two type of immunity:

- i. Passive immunity
- ii. Active immunity

## i) Passive immunity

Passive immunity is the transmission of active immunity, in the form of prefabricated antibodies, from one person to another person. Passive immunity appears as naturally or artificially <sup>[74-79]</sup>. When maternal antibodies through the placenta are transferred to the fetus, here passive immunity is developed naturally but when high levels of human antibodies specific for a particular pathogen are transferred to non-immune individuals, this is called artificial acquire passive immunity <sup>[80-84]</sup>. In passive immunity, the patient is at risk of epidemic by the same microbe later because body can't develop memory cells. Passive immunization is used in order to lower the symptoms of immunosuppressive diseases <sup>[85]</sup>.

#### Naturally acquired passive immunity

Maternal passive immunity is an illustration of naturally acquired passive immunity and indicates antibodymediated immunity which is transfer to a fetus from its mother at the time of pregnancy. These maternal antibodies are transferred to the fetus only through placental cells. This occurs during the third month of gestation. IgG is the antibody that can transfer through the placenta <sup>[86-89]</sup>.

#### Artificially acquired passive immunity

Artificially acquired passive immunity is a type of short-term immunization which can be activated by the transfer of antibodies. These antibodies are administered into the body in several forms such as animal blood plasma in the form of monoclonal antibodies. Artificially acquired passive immunity is activated in the case of immunodeficiency diseases <sup>[90-92]</sup>. For e.g: hypogammaglobulinemia.Abnormal heartbeat, which causes fainting in rare cases

#### ii) Active immunity

During the entry of pathogen inside the body both B cells and T cells are stimulated and develop memory B-cells and T- cells <sup>[93-96]</sup>. These memory cells produce secondary response when encountered with the duplicate pathogen again during the life. Active immunity may also be natural or artificial.

## Naturally acquired active immunity

This type of immunity develops when an individual is exposed to a live pathogen, and produces a primary immune response, and then it leads to immunological memory. This immunity is natal because it is not convinced by deliberate exposure <sup>[97,98]</sup>. Immunodeficiency and immunosuppression are the disorders of immune system operation that can affect the development of active immunity.

#### Artificially acquired active immunity

We can stimulate artificially acquired active immunity through vaccine. This is because of presence of antigens in vaccine. They also induce a primary response against the antigen without causing any symptoms of the disease [99, 100].

## CONCLUSION

Innate immunity is the first non-specific immunity for fighting against microbes. Here, the developed immune response is very fast and is negotiated by various cells like T cells, mast cells, B cells, basophils and eosinophil. Adaptive immunity relies on the tightly organized coaction between B and T cells. This immunity also developed immunologic memory so that it can give rapid response when exposure to the same pathogen again. The remaining of this article will focus on different types of cell involved in particular immunity.

## REFERENCES

- 1. Mann ER, et al. Review: Skin and the Immune System. J Clin Exp Dermatol Res. 2012;S2:003.
- 2. Tandoi F, et al. Immunity and nutrition: from research to clinical practice. Italian Journal of Pediatrics. 2014;40:31.
- 3. Deepak T and Goyal K. The Role of Helminths in Immunity. J Allergy Ther. 2014;7:231.
- 4. Marchant A. Mother and fetal immunity to cytomegalovirus. Retrovirology. 2009;6:3.
- 5. Sallusto F. Human T helper cells in immunity and autoimmunity: Th17 and beyond. Journal of Translational Medicine. 2010;8:19.
- 6. Poletaev AB and Churilov LP. Immunophysiology, natural autoimmunity and human health. Anosia. 2010;6:11-18.
- 7. Giannattasio, et al. Nutrition and immunity in newborns. Italian Journal of Pediatrics. 2015;41:33.
- 8. Kanwar JR, et al. Toll Like Receptors Play a Role in General Immunity, Eye Infection and Inflammation: TIrs for Nanodelivery. J Clin Cell Immunol. 2011;2:114.
- 9. Mathias R and von der Weid PY. Immunity and Gastrointestinal Disease: ARole for Lymphatic Vessels. J Clin Cell Immunol. 2014;5:262.
- 10. Abd El-Gawad EA, et al. Enhancement of Antioxidant Activity, Non-specific Immunity and Growth Performance of Nile Tilapia, Oreochromis Niloticus by Dietary Fructooligosaccharide. J Aquac Res Development. 2016;7:427.
- 11. Farage MA, et al. Innate Immunity in the Lower Female Mucosal Tract. J Steroids Hormon Sci. 2011;2:106.
- 12. Saikh KU. Innate Immunity and Sepsis: MyD88 as a Target for Therapeutics. J Clin Cell Immunol. 2011;2:e102.
- 13. Sparo and Sánchez Bruni SF. Approach to Optimise the Treatment of Bacterial Infections: Combined Antimicrobial Therapy with Enhancers of Innate Immunity. Clin Exp Pharmacol. 2012;2:e106.
- 14. Srinivasan PC. Neutrophils-the Sentinels of Periodontal Innate Immunity. J Clin Cell Immunol. 2013;S13:002.
- 15. Bomfim GF, et al. The Toll way to hypertension: role of the innate immune response. Endocrinol Metab Synd. 2011;2: 117.
- 16. Huszar JM and Payne CJ. miR-146a Influences Energy Metabolism, Cell Differentiation and Innate Immunity. Metabolomics. 2013;3:119.
- 17. Reygaert WC. Innate Immune Response to Urinary Tract Infections Involving Escherichia coli. J Clin Cell Immunol. 2014;5:280.
- 18. Jack C.S, et al. TLR signaling tailors innate immune responses in human microglia and astrocytes. J.Immunol. 2005;175:4320–4330.
- 19. Chung WO. Receptors in Oral Epithelial Innate Immunity. J Bioanal Biomed. 2015;7:e134.
- 20. Bomfim GF, et al. The Toll way to hypertension: role of the innate immune response. Endocrinol Metab Synd. 2011;2: 117.
- 21. Reygaert WC. Innate Immune Response to Urinary Tract Infections Involving Escherichia coli. J Clin Cell Immunol. 2014;5:280.
- 22. Chung WO. Receptors in Oral Epithelial Innate Immunity. J Bioanal Biomed. 2015;7:e134.

- 23. D. B. Learn, et al. Taurine and hypotaurine content of human leukocytes. Journal of Leukocyte Biology. 1990;48:174–182.
- 24. Abad C and Var Tan Y. VIP and PACAP as Regulators of Immunity: New Perspectives from A Receptor Point of View. J Clin Exp Neuroimmunol. 2016;1:104.
- 25. Frøsig TM. Re-Thinking the Interplay between Tumorigenesis and Immunity. J Cell Signal. 2016;1:112
- 26. Wang D, et al. Natural Killer cells in Innate Defense against Infective Pathogens. J Clin Cell Immunol. 2013;S13:006.
- 27. Umemoto S, et al. Mucosal Immune Responses Associated with NKT Cell Activation and Dendritic Cell Expansion by Nasal administration of a-galactosylceramide in the Nasopharynx. Otolaryngology. 2015;5:216.
- 28. MJ Robertson, et al. Response of human natural killer (NK) cells to NK cell stimulatory factor (NKSF): cytolytic activity and proliferation of NK cells are differentially regulated by NKSF. J Exp Med. 1992;175:779-788.
- 29. Sabrina B, et al. Dissecting the Roles of Natural Killer T cells in Autoimmune Disorders and Malignancy. J Clin Cell Immunol. 2012;S10:005.
- 30. Crivellato E and Ribatti D. Cooperation of Mast Cells and Basophils in Allergy. J Allergy Ther. 2013;4:135.
- 31. Erol AYG. The Role of Mast Cells and Neuroglia in Neuroinfectious Diseases. J Neuroinfect Dis. 2015;6:190.
- 32. Yazdani Shaik BD and Conti P. Relationship between Vitamin C, Mast Cells and Inflammation. J Nutr Sci. 2016;6:456.
- 33. Law B, et al. A Role for Mast Cells in Alcohol-Induced Tissue Damage and Remodeling. J Clin Exp Pathol. 2015;5:218.
- 34. Crivellato E and Ribatti D. Cooperation of Mast Cells and Basophils in Allergy. J Allergy Ther. 2013;4:135.
- 35. Vaillant AAJ, et al. Autoimmunity in Neurological and Psychiatric Disorders: Participation of Antibodies and Cytokines in the Immunopathogenesis of these Diseases. Immunome Res. 2015;11:089.
- 36. Erol AYG. The Role of Mast Cells and Neuroglia in Neuroinfectious Diseases. J Neuroinfect Dis. 2015;6:190.
- 37. Spínola A, et al. Are Basophils and T helper 2 Cells Implicated in Mastocytosis? J Allergy Ther. 2014;5:192.
- 38. Crivellato E and Ribatti D. Cooperation of Mast Cells and Basophils in Allergy. J Allergy Ther. 2013;4:135.
- 39. Yazdani Shaik BD and Conti P. Relationship between Vitamin C, Mast Cells and Inflammation. J Nutr Sci. 2016;6:456.
- 40. Xuehua Xu. Molecular Mechanisms Underlying Chemotaxis in the Model System Dictyostelium discoideum and Mammalian Neutrophils and Breast Cancer Cells. Cell Dev Biol. 2015;4:e135.
- 41. Der E, et al. Pro- and Anti-inflammatory Neutrophils in Lupus. J Clin Cell Immunol. 2014;5:239.
- 42. F. C. Weber, et al. Neutrophils are required for both the sensitization and elicitation phase of contact hypersensitivity. Journal of Experimental Medicine. 2015;212:15–22.
- 43. Ritis K and Kambas K. Neutrophil Extracellular Traps The Invisible Inflammatory Mediator in Fibrosis. J Clin Exp Pathol. 2015;5:239.
- 44. C.-W. Yang, et al. Neutrophils influences the level of antigen presentation during the immune response to protein antigens in adjuvants. Journal of Immunology. 2010;185: 2927–2934.
- 45. Berezin A. The Neutrophil Extracellular Traps: The Missed Link between Microvascular Inflammation and Diabetes? Metabolomics. 2016;6:163.
- 46. T. N. Mayadas, et al. The multifaceted functions of neutrophils. Annual Review of Pathology: Mechanisms of Disease. 2014;9:181–218.
- 47. V. L. Shepherd and J. R. Hoidal. Clearance of neutrophil-derived myeloperoxidase by the macrophage mannose receptor. American Journal of Respiratory Cell and Molecular Biology. 1990;2:335–340.
- 48. Pugholm LH, et al. Antibody-Mediated Delivery of Antigen to Dendritic Cells . Immunother Open Acc. 2016;2:119.
- 49. Bikash Sahay and Mahesh Kathania. Directional Activation of Intestinal Dendritic Cells by Oral Targeted Multivalent Vaccine. J Vaccines Vaccin. 2012;3:1.
- 50. Y.S. Jeelall, et al. Human lymphoma mutations reveal CARD11 as the switch between self-antigen-induced B cell death or proliferation and autoantibody production. J Exp Med. 2012;209: 1907–1917.
- 51. J.C. Weill, et al. Human marginal zone B cells. Annu Rev Immunol. 2009;27:267–285.
- 52. E. Meffre. The establishment of early B cell tolerance in humans: lessons from primary immunodeficiency diseases. Ann N Y Acad Sci. 2011;1246: 1–10.
- 53. Vista ES and Aragones M. The Role of B Cells in Autoimmunity: Insights on B Lymphocyte Stimulation (Blys) as a Target for Biologics in Systemic Lupus Erythematosus. J Clin Cell Immunol. 2015;6:360.

- 54. Sheng W, et al. T Cell-Derived GM-CSF, Regulation of Expression and Function. Immunome Res. 2015;11:098.
- 55. Chhabra A. Engineering Anti-Tumor T Cell Immunity. Adv Genet Eng. 2013;2:104.
- 56. Francelin C and Verinaud L. Apoptosis and the Developing T Cells. J Clin Cell Immunol. 2011;S3:001.
- 57. Tsai YG and Lin CY. Mechanism of Regulatory T Cells During Allergen-Specific Immunotherapy: from the Past to Future. J Aller Ther. 2011;S6:001.
- 58. Lowder TW and Kunz HE. Regulatory T Cells in Asthma and Airway Hyperresponsiveness. J Aller Ther. 2011;S1:002.
- 59. LetschEmail A. Peptide vaccination induces specific effector and memory T cells but fails to enhance preexisting T cell immunity. Cancer Cell International. 2004;5: 48.
- 60. Kleinschek MA, et al. Circulating and gut-resident human Th17 cells express CD161 and promote intestinal inflammation. J Exp Med. 2009;206: 525-534.
- 61. Takahashi T, et al. Expression of CD161 (NKR-P1A) defines subsets of human CD4 and CD8 T cells with different functional activities. J Immunol. 2006;176: 211-216.
- 62. Jewett A, et al. Dedifferentiation of Epithelial Tumors Enhances Cytotoxicity, Survival and Expansion of Allogeneic CD8+ T Cells and Natural Killer Cells. J Carcinogen Mutagen. 2012;S1:007
- 63. Palmer D, et al. Cish attenuates proximal TCR-signaling and CD8+ T cell immunity. Journal for ImmunoTherapy of Cancer. 2014;2: 32.
- 64. Trivedi S, et al. Interleukin-3 and Granulocyte-Macrophage Colony-Stimulating Factor Expression, a Biomarker of Memory CD8+ T Cell Immunity and Vaccine Efficacy. J Vaccines Vaccin. 2012;3:166.
- 65. Liu MF and Wang CR. Increased Th17 cells in flow cytometer-sorted CD45RO-positive memory CD4 T cells from patients with systemic lupus erythematosus. Lupus Sci Med. 2014;1: e000062.
- 66. Janik DK and Lee WT. Staphylococcal Enterotoxin B (SEB) Induces Memory CD4 T Cell Anergy in vivo and Impairs Recall Immunity to Unrelated Antigens. J Clin Cell Immunol. 2015;6:346.
- 67. Liu H, et al. The Role of CD4+ T cells in the Development of an Efficacious HIV Vaccine. J Clin Cell Immunol. 2015;6:350.
- 68. Lorton D and Bellinger DL. Sympathetic Nervous System Regulation of Th Cells in Autoimmunity: Beyond Th1 and Th2 Cell Balance. J Clin Cell Immunol. 2015;6:356.
- 69. Nair J.R, et al. CD28 expressed on malignant plasma cells induces a prosurvival and immunosuppressive microenvironment. J Immunol. 2011;187:1243–1253.
- 70. Dwyer J. M. Manipulating the Immune System with Immune Globulin. New Engl. J. Med. 1992;326: 107-116.
- 71. Bouras C. Immunity and neuron: new evidence relating immunoglobulins to cytosceletal damage. Annals of General Hospital Psychiatry. 2003;2: 19.
- 72. Heaps A, et al. Patients with Chronic Sarcoidosis have Reduced CD27+IgM+IgD+ Unswitched Memory B cells and an Expanded Population of Terminal Effector CD8+CD27-CD28- T cells. J Clin Cell Immunol. 2012;3:132.
- 73. Grönwall C, et al. Protective Roles of Natural IgM Antibodies. Immunol. 2012;3: 66.
- 74. Mckarns SC and Moisson FE. TNF Blockade, CNS Autoimmunity, Sex, and the Microbiome. J Mult Scler (Foster City). 2016;3:172.
- 75. R Sauerwein. Methods, markers and mechanisms for protective immunity in the controlled human malaria infection model. Malaria Journal. 2014;13:32.
- 76. Mackaness and G. B. Cellular resistance to infection. J. Exp. Med. 1962;116:381-406.
- 77. Lochmanova A and Lochman I. Immunity to Cytomegalovirus. J Antivir Antiretrovir. 2011;3:040-044.
- 78. Abad C and Var Tan Y. VIP and PACAP as Regulators of Immunity: New Perspectives from A Receptor Point of View. J Clin Exp Neuroimmunol Y. 2016;1:104.
- 79. Piatkiewicz P, et al. Autoimmunity Markers in Patients with Type 2 Diabetes. J Clin Diabetes Pract. 2016;1:107.
- 80. Tello N, et al. Levamisole Induced Autoimmunity. J Arthritis. 2016;5:187.
- 81. Caldeira EJ. The Diabetes and Allergies. J Allergy Ther. 2015;6:e111.
- 82. Gupta B and Raghav SK. Single Cell Transcriptomics for Autoimmune Disorders. J Mol Genet Med. 2015;9:191.
- 83. Malemud CJ. Endosomes and Toll-Like Receptors-Role in Autoimmunity. Rheumatology (Sunnyvale). 2015;5:158.
- 84. Molnár I, et al. The Prevalence and Characteristics of Allergy in Autoimmune Thyroid Diseases. J Clin Cell Immunol. 2015;6:306.
- 85. Boesman Finkelstein M. Passive oral immunization of children. Lancet. 1989;49: 1336.

- 86. U. Korthauer, D. Graf, et al. Defective expression of T-cell CD40 ligand causes X-linked immunodeficiency with hyper-lgM. Nature. 1993;361: 539–541.
- 87. Yang DH, et al. Expression of Complement Receptor Type 1 on Erythrocytes in Autoimmune Diseases. J Mol Biomark Diagn. 2014;5:163.
- 88. Racine J and Zeng D. Induction of Mixed Chimerism for Reversal of Autoimmunity in Type 1 Diabetes. J Clin Cell Immunol. 2013;S2:006.
- 89. Udristioiu A, et al. A New Approach of Abnormal Apoptosis Implicated in Malignancy and Autoimmunity. J Bioanal Biomed. 2012;4:034-038.
- 90. Guilherme L, et al. Rheumatic Heart Disease: Genes, Inflammation and Autoimmunity. Rheumatol Curr Res. 2012;S4:001.
- 91. Zhao Y. Autoimmunity and Therapeutic Challenges of Type 1 Diabetes. Translational Medic. 2011;1:104e.
- 92. J. Hutcheson, et al. Modulating proximal cell signaling by targeting Btk ameliorates humoral autoimmunity and end-organ disease in murine lupus. Arthritis Res Ther. 2012;14:R243.
- 93. Waugh KA, et al. Tolerance of Tumor-Specific T cells in Melanoma Metastases. J Clin Cell Immunol. 2016;7:409.
- 94. Cheriyan VT, et al. T cells in Immunobiology of Tumors and Immunotherapy. J Clin Cell Immunol. 2016;7:392.
- 95. Mitsuhashi M, et al. Peptide-induced mRNAs in whole blood ex vivo as a unique assay for peptide-specific T cell immunity. Journal for ImmunoTherapy of Cancer. 2013;1: 108.
- 96. Bodor J, et al. Hematopoietic Stem Cell-Based Therapy for HIV Disease: A Role for Regulatory T Cells. J Clin Cell Immunol. 2015;6:300.
- 97. Caprioli F, et al. Targeting T cells in Chronic Inflammatory Bowel Diseases. J Clin Cell Immunol. 2013;4:155.
- 98. Pastrana JL, et al. Regulatory T Cells and Atherosclerosis. J Clin Exp Cardiolog 2012;S12:00
- 99. Shahana A Choudhury and Fazle Matin. Seroprevalence of Antibodies to Measles, Mumps and Rubella (MMR) Vaccines in Previously Vaccinated Human Immunodeficiency Virus-Infected Children and their Control Counterparts. J Vaccines Vaccin. 2014;5:1.
- 100. Mortensen R and Dietrich J. New Generation Combination Vaccines against Streptococcus pyogenes. J Vaccines Vaccin. 2016;7:323.