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Janeway's Immunobiology Role of T Cells and T Cell Subpopulations in Protective Immunity Against Infection with Treponema Pallidum Subsp. Pertenue in Hamsters Specific T Cell Repertoires Mediate Protective Immunity to Histoplasma Capsulatum Molecular Biology of the Cell **Generation of CD8+ T Cell Mediated Protective Immunity Upon Vaccination with Soluble Antigen** *Programming CD8 T Cell Memory Development and Protective Immunity Transcriptome Response Associated with Protective Immunity in T and B Cell Deficient Zebrafish* **Antibody Fc: The Role of Reactive Oxygen Species in Protective Immunity** Persistent Viral Infections **The New Paradigm of Immunity to Tuberculosis** *Immunomodulation During the Development of Protective Immunity to Chlamydia* **Correlates of Protective Immunity Against Hepatitis C Virus Dendritic Cells Mediate Protective Immunity Against Salmonella Typhimurium by Regulating Antigen Presentation, Inflammation and Cell Death** *Assessment of Protective Immunity Against Plasmodium Berghei Sporozoites* **The Role of Bluetongue Virus Protein-specific Immune Response in Protective Immunity of Sheep Protective Immunity to Cryptosporidium Parvum Role of IL-17 in HIV-specific CD8+T Cell Immunity** T Cell Immunity in a Small Animal Surrogate of Hepatitis C Virus Infection **Immune Memory and Vaccines: Great Debates T-cell Activation in Health and Disease Microbial DNA and Host Immunity Protective Immune Response to Dengue Virus Infection and Vaccines: perspectives from the field to the bench Diverse functions of mucosal resident memory T cells** *B Cells in Immunity and Tolerance* **T-Cell Paradigms in Parasitic and Bacterial Infections Antibody Fc Janeway's Immunobiology** *CD8 T Cell Immunity to Viral Infection* **The Role of CD4+ T Cell Help and the Co-stimulatory Molecule CD40 in Protective Immunity Against Liver Stage Plasmodium Infection** Mucosal Vaccines **Protective Immunity Against Transmissible Gastroenteritis Virus (TGEW) Avian Immunology** *The Th2 Type Immune Response in Health and Disease* **Insights Into CD4 T Cell-Mediated Immunity to Influenza Viruses CD8 T Cell Dependent and Independent Immunity Against Plasmodium Following Vaccination** *Vaccines and Immunostimulants for Finfish* **Characterization of Host Protective Immunity Against Influenza Infection in Ferrets and Mice** *Diet and Immune Function* **T Cell Immunity to Hepatitis C Virus: Lessons for a Prophylactic Vaccine**

In times of globalization and the resulting contact to ever-new pathogens, it becomes even more important to understand how exactly protective immunity is induced to be able to generate more potent vaccinations. CD8+ T cells are crucial for protection against intracellular pathogens. Until today, vaccines capable to induce long lasting CD8+ T cell responses are usually based on living intracellular organisms. However, live vaccines can be potentially harmful, especially for immunocompromised individuals. In order to improve the safety of vaccines, the goal of many laboratories is to dissect the components derived from live vaccines, which are responsible to effectively induce long lasting protective immunity. Our aim of this study was to use purified Listeria monocytogenes (L.m.)-derived antigens and to modulate the immune system to promote the development of preferentially CD8+ effector memory T cells (TEM), which have recently shown to be the major population mediating protective immunity against L.m. We chose the L.m.-derived protein LLO and the LLO91-99 peptide as targets, because LLO-specific T cell responses have been demonstrated to confer efficient protection in this model. Furthermore, strong LLO91-99- specific immune responses are induced, increasing the chances to analyze also more subtle changes within the induced cell populations. While immunization with peptide alone or in combination with adjuvant

did not lead to measurable CD8+ immune response, the full protein LLO induced readily detectable antigen specific CD8+ T cell numbers. Thereby, the route of application played a crucial role, since LLO91-99-specific immune responses were much higher after local application of LLO and almost absent after systemic administration. Priming of LLO91-99- specific CD8+ T-cell responses upon immunization with the purified protein requires access of antigen to the MHC class I cross-presentation pathway. As TLR9 ligands like bacterial CpG DNA are believed to be able t. Abstract: There is consensus that HCV-specific T cells play a central role in the outcome (clearance vs. persistence) of acute infection and that they contribute to protection against the establishment of persistence after reinfection. However, these T cells often fail and the virus can persist, largely as a result of T cell exhaustion and the emergence of viral escape mutations. Importantly, HCV cure by direct-acting antivirals does not lead to a complete reversion of T cell exhaustion and thus HCV reinfections can occur. The current lack of detailed knowledge about the immunological determinants of viral clearance, persistence and protective immunity is a major roadblock to the development of a prophylactic T cell vaccine. This minireview highlights the basic concepts of successful T cell immunity, major mechanisms of T cell failure and how our understanding of these concepts can be translated into a prophylactic vaccine It has been more than three decades since HIV (Human Immunodeficiency virus)1 has been identified as the causative agent of AIDS, but an effective vaccine is still underway. Various vaccine vector delivery systems have been developed to enhance CD8 T cell mediated immunity against HIV-1. In our laboratory, heterologous mucosal systemic HIV-1 poxvirus prime-boost immunization have shown to induce high avidity HIV-specific CD8 T cell with excellent protective immunity. These studies also revealed that mucosal immunization induced lower numbers of IL-4 and IL-13 expressing HIV-specific CD8 T cells compared to pure systemic delivery. Data indicated that the route of immunization can determine the quality or avidity of CD8 T cell immunity and this is mainly governed by Th2 cytokines IL-4 and IL-13. Th17 cells are a newly discovered subset of T cells that specifically produce cytokines IL-17A to F. Various studies have shown that both Th1 and Th2 cytokines negatively regulate IL-17A producing CD4 T cells in order to mediate their effector immune response. As Ranasinghe et al. have shown that IL-4 and IL-13 modulate the quality of CD8 T cells, this study aimed to establish whether the expression of IL-17A by HIV-specific CD8 T cells is dependent on Th2 cytokines IL-4, IL-13 or Th1 cytokine IFN-gamma. Wild type BALBc, IL-4, IL-13 and STAT6 KO mice were prime-boost immunized with control vaccine and the expression of IL-17A in spleen and lung were evaluated. Data indicated that the expression of IL-17A was significantly enhanced in HIV specific CD8 T cells obtained from all KO mice tested compared to WT BALBc control mice. But IFN-gamma did not have any effect on the IL-17A expression. To further investigate these findings and better understand the transcriptional regulation of IL-17A; wild type BALBc, IL-4, IL-13 and STAT6 KO mice were prime-boost immunized with control vaccine and RT-PCR was performed to evaluate the IL-17A regulatory factors in CD8 T cells following HIV-specific peptide stimulation. Data showed that IL-17A, TGF-beta, IL-6 and ROR-gamma t mRNA levels were highly elevated in CD8 T cells obtained from IL-4 KO mice compared to the other groups tested. This data further confirmed that IL-4 played a predominant role in down regulating IL-17A induction, and TGF-beta, IL-6 and ROR-gamma t (not IL-23a) were involved in this regulation. As previous studies in our laboratory have shown that IL-13 can significantly modulate the avidity of HIV-specific CD8 T cells, recently, Ranasinghe et al. have developed a vaccine that can temporarily inhibit IL-13 at the vaccination site. Since, IL-4 and IL-13 have shown to modulate IL-17A expression, in this study, the expression of IL-17A in HIV specific CD8 T cells was also evaluated using control vaccine and novel IL-13 inhibitor vaccine at 3 days, 14 days, 8 weeks and 10 days post-challenge. Data indicated that compared to the control vaccine, IL-13 inhibitor vaccine showed enhanced IL-17A expression by HIV-specific CD8 T cells at 14 days post booster vaccination and following surrogate influenza-HIV mucosal challenge in both spleen and lung. Collectively, the data indicate that out of the two Th2 cytokines, IL-4 mainly regulate the IL-7A expression in HIV-specific CD8 T cells. As IL-4 and IL-13 are involved in regulating the avidity of CTLs, data suggest that IL-17A plays an indirect role in modulating CD8 T cell avidity and

protective immunity. The type 2 immune response that develops during infectious disease has undergone major paradigm shifts in the last several years as new cell types and pathways have been identified. It is now clear that the type 2 immune response, characterized by elevations in specific cytokines, including IL-4, IL-5 and IL-13, is associated with helminth infections in both humans and mice. This response is complex and includes effector functions that mediate resistance, contributing to expulsion and in some cases destruction, of the parasite. But just as importantly, the type 2 immune response can also mediate tolerance mechanisms, which can mitigate tissue injury as these large multicellular parasites transit through vital organs. The tolerance mechanisms include both tissue repair and immune regulatory effects. These latter aspects of the helminth-induced type 2 immune response are increasingly recognized as a potential resource that can be mined for the development of novel immunotherapies that may enhance wound healing, control of autoimmune and inflammatory diseases and regulation of metabolic homeostasis. In this book, leading researchers in this exciting and dynamic field discuss the latest findings and emerging concepts, providing an intellectual framework that can be used as a basis for new discoveries and potentially new treatments for diseases associated with inflammation. Persistent Viral Infections Edited by Rafi Ahmed Emory Vaccine Center, Atlanta, USA and Irvin S. Y. Chen UCLA School of Medicine, Los Angeles, USA During the past decade much of our attention has focused on diseases associated with viral persistence. Major breakthroughs in immunology, and the advent of molecular approaches to study pathogenesis have increased our understanding of the complex virus-host interactions that occur during viral persistence. Persistent Viral Infections focuses on: * The pathogenesis and immunology of chronic infections * Animal models that provide, or have the potential to provide, major insights This volume will be essential reading for virologists, immunologists, oncologists and neurologists. It has been said that the development of vaccines against a variety of infectious diseases is among the greatest triumphs of immunology. Indeed, several pathogens have lost their horror through the availability of effective vaccination measures. Unfortunately, this does not hold true for the pathogens dealt within this volume. Malaria, schistosomiasis, leishmaniasis, leprosy, and tuberculosis together are prevalent in more than 100 countries, and over 400 million persons suffer from these diseases. It is becoming increasingly clear that the failure to control these infections in a satisfactory way is directly related to the complexity oftheir interactions with the immune system. These agents have lived with their hosts for long enough to give both-host and parasite-ample opportunity to develop a highly sophisticated interrelationship. The central role of T lymphocytes both in acquired resistance to and pathogenesis of these microbes is well appreciated. In the beginning it may have been thought that acq uired resistance against infectious agents is nothing but another aspect of the immune response, studied with soluble and particulate antigens. This simple concept has gradually changed, and it has become clear that the viability not only of the immune cells but also of the 'antigens' adds another dimension to the game. Several achievements in cellular immunology and molecular biology have now made it possible to better understand at least some mechanisms in this intricate interplay. Vaccination remains one of the most effective means for preventing infectious diseases. During viral infection, activated CD8 T cells differentiate into cytotoxic effector cells that directly kill infected cells and produce anti-viral cytokines. Further T cell differentiation results in a population of memory CD8 T cells that have the ability to self-renew and rapidly proliferate into effector cells during secondary infections. However during persistent viral infection, T cell differentiation is disrupted due to sustained antigen stimulation resulting in a loss of T cell effector function. Despite the development of vaccines for a wide range of viral diseases, efficacious vaccines for persistent viral infections have been challenging to design. Immunization against virus T cell epitopes has been proposed as an alternative vaccination strategy for persistent viral infections, such as HIV. However, vaccines that selectively engage T cell responses can result in inappropriate immune responses that increase, rather than prevent, disease. Quantitative models of virus infection and immune response were used to investigate how virus and immune system variables influence pathogenic versus protective T cell responses generated during persistent viral infection. It was determined that an intermediate precursor frequency of virus-specific memory CD8

T cells prior to LCMV infection resulted in maximum T cell mediated pathology. Increased pathology was independent of antigen sensitivity or the diversity of TCR in the CD8 T cell response, but was dependent on CD8 T cell production of TNF and the magnitude of initial virus exposure. The threshold for exhaustion of responding CD8 T cells ultimately influences the precursor frequency that causes enhanced disease. In addition, viral infection can occur in the context of co-infection by heterologous pathogens that modulate immune responses and/or disease. Co-infection of two unrelated viruses in their natural host, Ectromelia virus (ECTV) and Lymphocytic Choriomeningitis virus (LCMV) infection in mice, were studied. ECTV infection can be a lethal infection in mice due in part to the blockade of antiviral cytokines, including Type I Interferons (IFN-I). It was determined that ECTV/LCMV co-infection results in decreased ECTV viral load and amelioration of ECTV-induced disease, presumably due to IFN-I induction by LCMV. However, immune responses to LCMV in ECTV co-infected mice were also lower compared to mice infected with LCMV alone and biased toward effector-memory cell generation. Thus, providing evidence for bi-directional effects of viral co-infection that modulate disease and immunity. Together the results suggest heterogeneity in T cell responses during vaccination with viral vectors may be in part due to heterologous virus infection or vaccine usage and that TNF-blockade may be useful for minimizing pathology while maintaining protection during virus infection. Lastly, quantitative mathematical models of virus and T cell immunity can be useful to generate predictions regarding which molecular and cellular pathways mediate T cell protection versus pathology. Infection with Plasmodium species leads to nearly 400,000 deaths a year despite widespread use of mosquito bed nets, insecticides, and anti-malarial drugs. To date, there is not a licensed vaccine capable of providing complete protection from Plasmodium infection to vaccinees. Whole parasite vaccination of humans and rodents can achieve complete protection in vaccines, but the dose of sporozoites, number of administrations, and production concerns in generating these types of vaccines will likely prevent these approaches from achieving worldwide use. However, the protective immunological responses against Plasmodium parasites engendered by these vaccination approaches can be studied and aid in the development of advanced subunit vaccines against Plasmodium. Using rodent models of malaria to elucidate the features of protective immunity engendered by whole parasite vaccination, it has been repeatedly shown that CD8 T cell responses directed against liver-stage parasite antigens can provide complete protection with some contribution by CD4 T cells and antibody responses depending on the model system studied. However, the quantatitive and qualitative requirements for CD8 T cell immunity against Plasmodium remains largely undefined. To enhance our understanding of how to generate protective immunity against Plasmodium, I have utilized rodent models of malaria to study the superior protection afforded from single-dose vaccination with virulent sporozoites administered under prophylatic chloroquine-cover, referred to as chemoprophylaxis sporozoites (CPS) vaccination, compared to the well-studied approach of administering radiation-attenuated Plasmodium sporozoites (RAS). RAS vaccination has long been considered the “gold standard” in vaccination due the ability of RAS vaccination to engender complete protection following sporozoite challenge of vaccinated humans and rodents. Leading researchers review the activation of the mammalian immune system by bacterial DNA and its immunostimulatory sequences (ISS), and consider the applications of ISS in clinical medicine. The authors survey the latest findings concerning the receptor-recognition and signaling pathways triggered by ISS , the process of cell activation, and the potential vaccination strategies using ISS. Specific pharmaceutical applications discussed include infectious disease (Hepatitis B, HIV, and mycobacterial infections), allergy (asthma and conjunctivitis), cancer (lymphoma), and inflammation and autoimmunity (arthritis and colitis). Fc receptor (FcR)-dependent effector functions of antibodies contribute significantly to protective immunity against microbial pathogens and tumors. Therefore, FcR-mediated immunological processes constitute a key component of the immune system’s defense armamentaria for maintaining the biological and physiological integrity of the mammalian host who is yoked with frequent encounters with infections and neoplasia. The direct effector functions that result from FcR triggering are phagocytosis, antibody-dependent cellular cytotoxicity, and induction of inflammation;

also, FcR-mediated processes provide immunoregulation and immunomodulation that augment T-cell immunity and fine-tune immune responses against antigens. This plasticity of effector and immunoregulatory functions provides unique opportunities to apply FcR-based platforms and immunotherapeutic regimens for vaccine delivery and drug targeting against infectious and non-infectious diseases. This chapter focuses on the protective immunological processes resulting from antibody or immune complex binding to FcRs on effector cells (i.e., NK cells, macrophages, dendritic cells, PMNs, and eosinophils), as well as innovative strategies to apply these mechanisms in immunotherapy, vaccine, and drug delivery against infectious and non-infectious diseases. Deleterious immune reactivity associated with FcR engagement, including immune complex diseases, allergic reactions due to IgE-mediated activation of mast cells and basophils, or facilitation of microbial infectivity, such as antibody-mediated enhancement of infections, are outside the focus of this review. This book contains twelve chapters contributed by prestigious international experts who are at the forefront of B cell research, and aims to provide a cutting-edge and comprehensive overview of all aspects of B cells, including B cell development, maturation and activation, germinal center reaction, memory and plasma cell differentiation, and antibody-mediated positive and negative regulation of humoral immune responses. There are also three chapters describing human diseases caused by B cell abnormalities, including primary antibody deficiencies, autoimmune diseases, and B cell malignancies. We hope that this book will become a standard and routine reference for both basic researchers and clinicians. Avian Immunology, Third Edition contains a detailed description of the avian innate immune system, encompassing the mucosal, enteric, respiratory and reproductive systems. The diseases and disorders it covers, include immunodepressive diseases and immune evasion, autoimmune diseases, and tumors of the immune system. Practical aspects of vaccination are examined as well. Extensive appendices summarize resources for scientists including cell lines, inbred chicken lines, cytokines, chemokines, and monoclonal antibodies. With contributions from the foremost international experts in the field, Avian Immunology 3rd, provides the most up-to-date crucial information not only for poultry health professionals and avian biologists, but also for comparative and veterinary immunologists, graduate students and veterinary students with an interest in avian immunology. Avian Immunology, Third Edition, is a fascinating and growing field and surely provides new and exciting insights for mainstream immunology in the future. Reflects significant advances in the field since the second edition, particularly the explosion of knowledge on genomics including work on the chicken, turkey and zebra finch genomes Provides a single source reference ranging from the basic science to cutting edge research Provides practical information for veterinarians particularly those specialised in poultry or companion bird medicine New chapters on the impact of the microbiome on the immune system, defence mechanisms in the egg and embryo and emerging transgene technologies Influenza A viruses continue to pose a threat to human health worldwide and virusspecific CD4 T cells are key to protective immunity. The goal of the experiments within this thesis was to provide novel insight into elements within CD4 T cell responses that influence CD4 T cell specificity and function. Using a combination of novel reporter viruses, intravascular labeling in vivo, multi-parameter flow cytometry, and cytokine ELISpot procedures in two mammalian models, this work examined various stages of the developing immune response and the relationship between antigen acquisition, CD4 T cell fate choice, trafficking and compartmentalization within the inflamed lung, antigen specificity, and cytokine functionality. We found that pandemic 2009 H1N1 virus infection resulted in a broad pattern of viral antigen acquisition in the lung. Complex subpopulations of MHC class II-positive cells from both the hematopoietic and non-hematopoietic lineages were identified, with dynamic shifts over time. Our results demonstrated that antigen access occurs by uptake of exogenous antigen as well as infection. Finally, our results confirmed that many different cell types displayed peptide:MHC class II complexes in situ and possessed the capacity to stimulate influenza-specific CD4 T cells. These results correlated with differences in infection status, cell lineage, and MHC class II expression. These data suggest that there are many different types of antigen presenting cells in the lung that may impose additional checkpoints of CD4 T cell functionality and

regulation. At the peak of the immune response, we found that lung CD4 T cells compartmentalized to vasculature and tissue. Antigen-experienced CD4 T cells were enriched in the tissue and expressed higher levels of molecules involved in trafficking to sites of pulmonary infection, damage, and repair. Surprisingly, we found similar representation in the fine specificity, immunodominance hierarchy, and polyfunctional potential of effector CD4 T cells in the two compartments. These results suggest that imprinting of CD4 effector programs in the priming lymph node plays a strong deterministic influence on CD4 function and fate. Also, we provide evidence of cellular intermediates in CD4 T cell trafficking in the lung. Finally, this thesis includes the first description of the CD4 and CD8 T cell repertoires in response to infection, a key step needed to advance the ferret model forward for future transmission and vaccine studies. Mechanistic studies of infection and vaccination in domestic ferrets may reveal novel links between antigen specificity and effector function, cellular correlates of protection, and the merits, consequences, and cellular heterogeneity of complex immunological memory. This comprehensive, authoritative treatise covers all aspects of mucosal vaccines including their development, mechanisms of action, molecular/cellular aspects, and practical applications. The contributing authors and editors of this one-of-a-kind book are very well known in their respective fields. Mucosal Vaccines is organized in a unique format in which basic, clinical, and practical aspects of the mucosal immune system for vaccine development are described and discussed. This project is endorsed by the Society for Mucosal Immunology. Provides the latest views on mucosal vaccines Applies basic principles to the development of new vaccines Links basic, clinical, and practical aspects of mucosal vaccines to different infectious diseases Unique and user-friendly organization The hepatitis C virus (HCV) chronically infects 71 million people worldwide and is major cause of severe progressive liver diseases such as cirrhosis and hepatocellular carcinoma. Persistence of HCV in most infected persons is thought to be a consequence of failed virus-specific T cell immunity. Indeed, the only HCV vaccine candidate to progress to phase II clinical efficacy testing in humans is a virally vectored prime-boost regimen designed to induce virus-specific CD4 and CD8 T cell responses. However, there is still uncertainty about the role of T cells in acute HCV control, why they tend to fail, and whether their induction via vaccine could prevent virus chronicity. Addressing these fundamental questions has been exceedingly difficult in the absence of relevant HCV animal models. Chimpanzees, the only species besides humans fully permissive to HCV infection, are no longer available for research, and current mouse models are immune- compromised and therefore unsuitable for detailed immunological studies. In recent years, a number of closely-related viral homologs of HCV have been discovered in diverse animal hosts, opening new possibilities for in vivo modeling. The most promising of these is a rodent hepacivirus (RHV) discovered in wild Norway rats (Rattus norvegicus). RHV shares important virological features with HCV and, most notably, causes chronic hepatotropic infections in immune- competent laboratory rats. The central objective of these studies was to elucidate the importance of T cells during rat RHV infection and to identify mechanisms leading to their failure to eliminate persistent virus, in both naïve and immunized contexts. Using a recombinant adenovirus vaccine expressing RHV non-structural proteins as an experimental tool, we demonstrate that T cells are a critical component of immunity to persistent infection. Vaccination prevented RHV persistence in most animals and transient depletion of CD8 or CD4 T cells undermined protective immunity. We also describe the nature and fate of RHV-specific CD8 T cells induced during persistent infection and reveal early viral load as a key factor underlying their inability to mediate protective immunity in this model. Finally, using an RHV strain containing immune escape mutations within dominant CD8 T cell epitopes, we highlight antigen mismatch as an important determinant of RHV vaccine efficacy and reveal correlates of successful immunity to heterologous virus. Overall, our data provide direct evidence that T cells are vital for hepacivirus control and that vaccines that stimulate cellular immunity without parallel antibody induction can be effective in preventing virus persistence. Furthermore, our studies highlight the ease and utility of rat RHV infection for investigating mechanisms of HCV immunity, persistence, and vaccine protection. This book illustrates the intimate relationship between alveolar macrophages and Mycobacterium tuberculosis (M.tb.), and the former’s role in

both innate and adaptive immunity against M.tb. It covers research done over the last decade. It also explores the role of macrophage death following infection with M.tb. in determining whether successful immunity is stimulated, or whether clinical disease develops; furthermore, the function of host lipid mediators in macrophage death modality are addressed. The book also illustrates how the balance between prostaglandins and lipoxins determines whether infected macrophages undergo apoptosis or necrosis, which is the ultimate factor in the outcome of infection. Finally, it is a synthesis of the authors' recent studies and the studies of others to offer a new understanding of immunity to tuberculosis. T-cell Activation in Health and Disease is a collection of papers presented at the "T-cell Activation in Health and Disease—Disorders of Immune Regulation—Infection and Autoimmunity" workshop held in Oxford on September 25-29, 1988. This book discusses the progress occurring in T-cell immunity research. One paper discusses the effects of two interaction clones of T-cells that can define the T-cell immunoregulatory network. Another paper discusses the relationship between connectivity and tolerance of the immune network. This paper then suggests the possibility that autoimmunity arises because self-reactive clones are inadequately connected to the network. Another paper reviews the cell-mediated responses in the synovial fluids, as well as the interaction of rheumatoid arthritis synovial fluid dendritic cells and T lymphocytes. The book also examines why attempts for protective immunity to the HIV virus have not been successful. One article then discusses the goals of immunologic intervention in autoimmune disease by using an approach involving the cellular and cytokine targets and their deployment. This text can prove significant for scientists in the field of pharmacology, cellular biology, and researchers in the field of immunology and infectious diseases. Dengue is the most important mosquito-transmitted viral disease in humans. Half of the world population is at risk of infection, mostly in tropical and sub-tropical areas. The World Health Organization (WHO) estimates that 50 to 100 million infections occur yearly, with 50,000 to 100,000 deaths related to dengue, mainly in children. Recent estimates show higher numbers, up to three times more, with 390 million estimated dengue infections per year, among which 96 million apparent infections (Bhatt et al. 2013). Initially localized to South-East Asia, dengue virus (DENV) started its spread in Latin America in the 80's. Little is known about DENV spread in Africa, but multiple seroprevalence surveys over several years are now clearly showing endemic areas in East and West Africa (Brady et al. 2013). Finally, due to global warming and intense traveling there is a risk of global spread towards more temperate regions, and both US Key islands (FL) and southern Europe recently faced DENV outbreaks. There are currently no specific treatments or vaccines available. Even though several dengue vaccines are in the pipeline, clear correlates of protection are still lacking. The recent failure of the live-attenuated Sanofi vaccine Phase 2b trial (Sabchareon et al. 2013) and the lack of correlation between clinical protection and in vitro neutralization assays, clearly underlines the necessity to better understand the role of the different components of the immune system in protection against dengue virus infection and the requirement for the development of additional and/or improved predictive assays. The aim of this research topic is to provide novel data, opinions and literature reviews on the best immune correlates of protection and recent advances in the immune response to DENV infection that can allow rapid progress of dengue vaccines. Authors can choose to submit original research papers, reviews or opinions on pre-clinical or clinical observations that will help unify the field, with perspectives from epidemiology, virology, immunology and vaccine developers. This research topic will discuss different aspects of the protective immune response to DENV that can influence vaccine development. It will include a review of epidemiological data generated in the field, which will address spatio-temporal diversity of DENV epidemics, the importance of cross-reactive protection and of the time-interval between infections as a predictor of disease. It will further include a review of the role of both the innate and adaptive immunity in DENV infection control, and discuss the usefulness of new improved animal models in dissecting the role of each immunological compartment, which will help define new correlate of immune protection. New data concerning the DENV structure and anti-dengue antibody structure will address the necessity of improved neutralization assays. The ultimate test to prove vaccine efficacy and study immune correlates of

protection in humans before large trials will open up the discussion on human DENV challenges using controlled attenuated viral strains. Finally, the role of vaccines, administered in flavi-immune populations, in the modification of future epidemics will also be approached and will include novel studies on mosquitoes infection thresholds. Antibody Fc is the first single text to synthesize the literature on the mechanisms underlying the dramatic variability of antibodies to influence the immune response. The book demonstrates the importance of the Fc domain, including protective mechanisms, effector cell types, genetic data, and variability in Fc domain function. This volume is a critical single-source reference for researchers in vaccine discovery, immunologists, microbiologists, oncologists and protein engineers as well as graduate students in immunology and vaccinology. Antibodies represent the correlate of protection for numerous vaccines and are the most rapidly growing class of drugs, with applications ranging from cancer and infectious disease to autoimmunity. Researchers have long understood the variable domain of antibodies, which are responsible for antigen recognition, and can provide protection by blocking the function of their target antigen. However, recent developments in our understanding of the protection mediated by antibodies have highlighted the critical nature of the antibody constant, or Fc domain, in the biological activity of antibodies. The Fc domain allows antibodies to link the adaptive and innate immune systems, providing specificity to a wide range of innate effector cells. In addition, they provide a feedback loop to regulate the character of the immune response via interactions with B cells and antigen-presenting cells. Clarifies the different mechanisms of IgG activity at the level of the different model systems used, including human genetic, mouse, and in vitro Covers the role of antibodies in cancer, infectious disease, and autoimmunity and in the setting of monoclonal antibody therapy as well as naturally raised antibodies Color illustrations enhance explanations of the immune system Histoplasma capsulatum (Hc) is a dimorphic fungus that causes a wide spectrum of disease. The interaction of T cells with macrophages and the subsequent production of cytokines are essential for protection. This thesis examines the importance of the T cell receptor (TCR) repertoire during the generation of immunity to infection with Hc and vaccination with a protective antigen (Ag). During primary infection, V 4 + T cells expand in the lungs and depletion of these cells increases the fungal burden in mice. To examine the Ag-specificity of lung V 4 + cells, they were isolated from infected mice and used to generate hybridomas. These cells were screened for Ag reactivity using an extract derived from the cell wall and cell membrane of Hc yeast. T cell immunoblotting with hybridomas indicate that the majority of lung-derived cells respond with an antigen at 110-130 kDA. Mass spectrometry identified this antigen as a homolog of Sec31 from Saccharomyces cerevisiae . In parallel, we examined the TCR repertoire following immunization with the protective Ag, Hsp60. A majority of T cell clones derived from Hsp60 immunized mice expressed V 8 .1/8.2. Depletion of V 8 .1/8.2 + cells abrogated the efficacy of Hsp60 immunization. Among these cells, those that generate IFN-? and reacted to F3 were able to confer protection in IFN-? -/- and TCR a/ -/- mice. The protective response to Hsp60 also relies on the presence of IL-10, IL-12, and IFN-?. To determine if the absence of IL-10 and IFN-? disrupt the generation of immunity to Hsp60 by altering the TCR repertoire, T cells were isolated from IL-10 and IFN-? deficient mice following immunization with Hsp60. The IL-10 -/- -derived TCR repertoire resembled that previously observed in wildtype animals, while IFN-? -/- repertoire had a smaller proportion of V 8 .18/2 + cells. Together, the data presented here demonstrate that specific subsets of Ag-specific T cells mediate the protective response to Hc during both the respond to primary infection as well as the challenge subsequent to vaccination. The activity of these cells is associated with production of IFN-?, which also influences the repertoire during initial stages of the immune response. Supporting initiation, development and resolution of appropriate immune responses is key to survival. Many nutrients and dietary components have been purported to have a role in supporting optimal immune function. This is vital throughout the life course, from the development and programming of the immune system in early life, to supporting immunity and reducing chronic inflammation in older people. In this special issue of Nutrients, we examine the evidence for the role of diet and dietary components in promoting protective immunity. Live, attenuated Plasmodium vaccines against liver stage infection have shown

great promise in the mouse model and are being optimized in preliminary human clinical trials. While CD8+ T cells play a key role in mediating immunity conferred by attenuated parasites, it is unclear what signals CD8+ T cells receive from antigen-presenting cells (APC) or CD4+ T helper cells during priming that induce an effective memory response. The late-arresting vaccine strain fabb/f- induces powerful, CD4+ T cell-dependent, sterilizing immunity and can be used to determine what mechanisms must be triggered during immunization to generate a protective and long-lasting response that prevents malaria. As a route of CD4+ T cell help, the co-stimulatory molecule CD40 enhances immunity through several distinct roles in T cell activation memory formation. CD40 was critical for the full maturation of liver dendritic cells, clonal expansion of CD8+ T cells in the liver, and protective immunity immunization with the P. yoelii fabb/f- genetically attenuated parasite (GAP). We evaluated the contributions to CD8+ T cell immunity of CD40 expressed on APC as compared to CD40 expressed on CD8+ T cells. Most of the effects of CD40 could be accounted for by expression in the T cells' environment, but CD40 on the CD8+ T cells themselves had a distinctive role in limiting effector cell differentiation and controlling the expression of exhaustion-associated markers. Thus CD40 on APC drives clonal-expansion of effector cells, but unopposed this signal discriminates against the formation of memory. CD40 expressed on the CD8+ T cells themselves balances this, allowing the formation of long-lived memory cells. We conclude that protective immunity conferred by late-arresting parasite vaccines relies heavily on CD4+ T cell help, APC licensing and strong co-stimulatory signals via CD40 to prime both effector CD8+ T cells and long-lived memory T cells. Explore the premier text for immunology at the advanced undergraduate, graduate, and medical school levels. Beginning students appreciate the bookÕs clear writing and informative illustrations, while advanced students and working immunologists value its comprehensive scope and depth. This edition is thoroughly revised and up to date with significant developments in the field, especially on the topic of innate immunity. The Janeway's Immunobiology CD-ROM, Immunobiology Interactive, is included with each book, and can be purchased separately. It contains animations and videos with voiceover narration, as well as the figures from the text for presentation purposes. RAG1-/- mutant zebrafish lack T and B lymphocytes. However, when re-exposed to homologous bacteria, these fish mount a response that provides specific protection. To further define this response, we utilized microarray analyses to determine the mechanisms underlying innate immune system memory in zebrafish. We also analyzed interferon (IFN) gamma by qRT-PCR. It is produced by activated NK cells and could indicate if this cell mediates the protective response seen in lymphocyte deficient zebrafish. Pathological studies and in situ hybridizations were performed to observe tissue changes and location of the cells that produced IFN gamma. Following bacterial re-exposure, zebrafish transcripts in cell receptor activation, cell proliferation and cytotoxic function categories were differentially expressed. We found high expression of IFN gamma in the lymphocyte like cell population after bacterial exposure and this was induced to a higher level in fish that had been vaccinated. The phagocytic cell population showed no induction of INF gamma. Over-all, the pathological response was much less severe in the vaccinated (48 hps) fish. Our microarray and pathological findings indicate that the primary immune response of mutant zebrafish is not impaired, and they demonstrate an enhanced innate immune response following secondary bacteria exposure. Following homologous secondary exposure, mutant zebrafish have a cell population that is undergoing upregulated cell receptor activation, cell cytotoxic functions and cell proliferation. This cell population expresses INF gamma. Activated T cells, NK-T cells and NK cells express INF gamma. Since RAG1 deficient zebrafish do not have T or NK-T cells, this cell population is most likely NK cells. This eBook is a collection of articles from a Frontiers Research Topic. Frontiers Research Topics are very popular trademarks of the Frontiers Journals Series: they are collections of at least ten articles, all centered on a particular subject. With their unique mix of varied contributions from Original Research to Review Articles, Frontiers Research Topics unify the most influential researchers, the latest key findings and historical advances in a hot research area! Find out more on how to host your own Frontiers Research Topic or contribute to one as an author by contacting the Frontiers Editorial Office: frontiersin.org/about/contact. "A subject collection from

the Cold Spring Harbor perspectives in biology." Early studies recognized the unique phenotype and attributes of T cells found in mucosal tissues, such as the intestines, skin, lung and female reproductive tract. This special topic issue will cover many aspects of mucosal-resident T cell biology during infection and disease and is dedicated to Leo Lefrancois, a pioneer in this field who recently passed away. A major proportion of these mucosal T cells are memory T cells, now recognized as a major constituent of memory T cells referred to as tissue-resident memory T cells. Unlike central and effector memory T cell subsets, tissue-resident memory T cells exhibit tissue specificity with minimal systemic migration. Nonetheless, tissue-resident memory T cells share a similar origin and display some overlapping phenotypes with their other memory T cell counterparts. Articles in this issue will describe the different types of memory T cells residing in mucosal tissues, their origins and functions as well as how they vary among discrete mucosal sites. Manuscripts will consider the unique physiological environments and cellular constituents which facilitate tissue residency while preserving tissue function. Additionally, there will be descriptions of the various mechanisms responsible for the migration and segregation of tissue resident memory CD8 T cells from the peripheral T cell pool. Although the mechanisms facilitating the sequestration of tissue-resident memory T cells within a respective tissue has not well characterized, various theories will also be discussed. Lastly, how these T cells contribute to immunity to pathogens, cancer, and autoimmunity and could be modified through vaccination or therapeutic intervention will be described. As mucosal tissues are the major portals of pathogen entry and frequent transformation, the activities and persistence of tissue resident memory T cells is crucial for mediating protection at these sites.

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