

# Immunity Pogil Continued...

- Model 3 and 4 40 minutes
- Allergy Video w/Questions

# Pogil Immune In Modela Pathogen

**Tomer Zohar**



## **Pogil Immune In Modela Pathogen:**

**Recent Advances on Model Hosts** Eleftherios Mylonakis, Frederick M. Ausubel, Michael Gilmore, Arturo

Casadevall, 2016-08-23 Most studies of bacterial or fungal infectious diseases focus separately on the pathogenic microbe the host response or the characterization of therapeutic compounds Compartmentalization of pathogenesis related research into an analysis of the pathogen the host or the antimicrobial compound has largely been dictated by the lack of model systems in which all of these approaches can be used simultaneously as well as by the traditional view that microbiology immunology and chemical biology and pharmacology are separate disciplines An increasing number of workers from different fields have turned to insects fish worms and other model hosts as facile ethically expedient relatively simple and inexpensive hosts to model a variety of human infectious diseases and to study host responses and innate immunity Because many of these hosts are genetically tractable they can be used in conjunction with an appropriate pathogen to facilitate the discovery of novel features of the host innate immune response This book provides a series of reports from the 1st International Conference on Model Hosts This first of its kind meeting focused on invertebrate vertebrate and amoeboid systems used for the study of host pathogen interactions virulence and immunity as well as on the relevance of these pathogenesis systems and mammalian models Importantly a common fundamental set of molecular mechanisms is employed by a significant number of microbial pathogens against a widely divergent array of metazoan hosts Moreover the evolutionarily conserved immune responses of these model hosts have contributed important insights to our understanding of the innate immune response of mammals This book provides a series of reports from the 1st International Conference on Model Hosts This first of its kind meeting focused on invertebrate vertebrate and amoeboid systems used for the study of host pathogen interactions virulence and immunity as well as on the relevance of these pathogenesis systems and mammalian models Importantly a common fundamental set of molecular mechanisms is employed by a significant number of microbial pathogens against a widely divergent array of metazoan hosts Moreover the evolutionarily conserved immune responses of these model hosts have contributed important insights to our understanding of the innate immune response of mammals *Unveiling Pathogen Interactions with Immune Cells and Model Biomembranes* Mengchi Jiao, 2024 The overall goal of my dissertation research is to unveil the pathogen immune cell interactions through the endocytosis pathway by fluorescence microscopy enabled biochemical sensors and biophysical assays Endocytosis is a critical cellular function that clears foreign materials and protects the host from pathogen attack However as can be brought into the intracellular environment of the host cell by endocytosis pathogens have developed a variety of mechanisms to hijack the endosome maturation so that they can somehow manage to escape leading to access to replication machinery and successful infection Therefore understanding how pathogens escape from endosomes and bypass the innate immune system is critical to preventing infection and developing medical therapeutics My graduate research provided insights into addressing three fundamental questions regarding

pathogen hijacking the endocytosis pathway 1 How does the anisotropic ligand presentation on pathogens modulate the innate immune response of the host cell during phagosome maturation 2 How does the interaction between non enveloped viruses and the lipid membrane result in the virus endosomal escape and host infection 3 What are the specific roles of capsid protein released peptides and lipids during the dynamic virus membrane interactions The anisotropic arrangement of cell wall components is ubiquitous among pathogens but how this functional asymmetry affects interactions between microbes and host immune cells is not known In the first part of my thesis we asked how ligand anisotropy on pathogens modulates phagosome maturation the process used by host immune cells to degrade internalized microbes Building on our previous research we developed two faced Moon particles as model pathogens that not only display ligands on solely one hemisphere but also simultaneously function as fluorogenic sensors for probing biochemical reactions inside phagosomes during maturation We show that the anisotropic presentation of ligands on particles delays the start of acidification and proteolysis in phagosomes but does not affect their degradative capacity Importantly our work suggests that functional asymmetry provides pathogens with a longer time in the neutral phagosomal environment making it easier for the pathogens to escape The second part of my thesis focuses on virus host membrane interactions Non enveloped viruses without a lipid coating enter host cells primarily through the endocytosis pathway The virus escape requires re arrangement of the viral capsids and conformational change of capsid proteins However how the conformational changes enable the endosomal membrane penetration remains unknown To tackle this question we have focused on using reovirus as a model to understand how non enveloped viruses penetrate across the host membrane Reoviruses virions are digested by extracellular and endosomal proteases to generate entry intermediate called the infectious subvirion particles ISVPs We sought to investigate the dynamic interactions between ISVPs and lipid membranes by using the giant unilamellar vesicle GUV model system Our work reveals a previously undocumented role of virus particles in inducing local membrane perturbation at the site of dynamic interaction Specifically we demonstrate the formation of relatively large pores that approach the size of particles that are delivered across the membrane Importantly the large pore formation requires the presence of ISVPs and the virus induced membrane rupture depends on cholesterol content in the membrane We further investigated the three way interactions of viral proteins released peptides and lipid membranes during the membrane entry We introduced the planar supported lipid bilayer as a model membrane system and applied the single virus tracking technique to dissect the interactions between lipids and viruses By trajectory analysis we unveiled the dual roles of the u1N peptides which not only drove the diffusion of ISVPs on the membrane but also served as receptors to recruit and confine new ISVPs In addition we found u1 protein also affected the ISVP lipid interaction From our results we established the burnt bridge mechanism for the initiation of infection consisting of virus attachment and endosomal escape Our studies highlighted a well orchestrated and coherent self propagating mechanism underlying reovirus membrane interaction which is critical to understanding the

infection of other lethal non enveloped viruses and the development of anti viral strategies In summary my doctorate research gave insights into the mechanisms by which pathogens hijack the endocytosis pathway to infect the host cell Learning the infection mechanism will enable us to understand more about complex diseases facilitating the development of anti pathogen therapies and promoting public health

**The Mononuclear Phagocyte System in Infectious Disease**

Geanncarlo Lugo-Villarino,Céline Cougoule,Etienne Meunier,Yoann Rombouts,Christel Vérollet,Luciana Balboa,2019-10-04

The Mononuclear Phagocyte System MPS of vertebrates is composed of monocytes macrophages and dendritic cells Together they form part of the first line of immune defense against a variety of pathogens bacteria fungi parasites and viruses and thus play an important role in maintaining organism homeostasis The mode of transmission type of replication and mechanism of disease causing differ significantly for each pathogen eliciting a unique immune response in the host Within this context the MPS acts as both the sentinel and tailor of the immune system As sentinels MPS cells are found in blood and within tissues throughout the body to patrol against pathogenic insult The strategy to detect microbial non self relies on MPS to recognize conserved microbial products known as pathogen associated molecular pattern PAMPs PAMPs recognition represents a checkpoint in the response to pathogens and relies on conserved pattern recognition receptors PRRs Upon PRR engagement MPS mount a cell autonomous attack that includes the internalization and compartmentalization of intracellular pathogens into toxic compartments that promote destruction In parallel MPS cells launch an inflammatory response composed of a cellular arm and soluble factors to control extracellular pathogens In cases when innate immunity fails to eliminate the invading microbe MPS serves as a tailor to generate adaptive immunity for pathogen eradication and generation of memory cells thus ensuring enhanced protection against re infection Indeed MPS cell functions comprise the capture process migration and delivery of antigenic information to lymphoid organs where type 1 immunity is tailored against intracellular microbes and type 2 immunity against extracellular pathogens However this potent adaptive immunity is also a double edge sword that can cause aberrant inflammatory disorders like autoimmunity or chronic inflammation For this reason MPS also tailors tolerance immunity against unwanted inflammation Successful clearance of the microbe results in its destruction and proper collection of debris resolution of inflammation and tissue healing for which MPS is essential Reciprocally as part of the evolutionary process taking place in all organisms microbes evolved strategies to circumvent the actions bestowed by MPS cells Multiple pathogens modulate the differentiation maturation and activation programs of the MPS as an efficient strategy to avoid a dedicated immune response Among the most common evasion strategies are the subversion of phagocytosis inhibition of PRR mediated immunity resistance to intracellular killing by reactive oxygen and nitrogen species restriction of phagosome maturation modulation of cellular metabolism and nutrient acquisition regulation of cell death and autophagy and modulation of pro inflammatory responses and hijacking of tolerance mechanisms among others The tenet of this eBook is that a better understanding of MPS in infection will yield insights for development of therapeutics to enhance

antimicrobial processes or dampen detrimental inflammation for the host's benefit. We believe that contributions to this topic will serve as a platform for discussion and debate about relevant issues and themes in this field. Our aim is to bring expert junior and senior scientists to address recent progress, highlight critical knowledge gaps, foment scientific exchange, and establish conceptual frameworks for future MPS investigation in the context of infectious disease.

### **Mathematical Modeling of the Immune System in Homeostasis, Infection and Disease** Gennady Bocharov, Burkhard

Ludewig, Andreas Meyerhans, Vitaly Volpert, 2020-02-24 The immune system provides the host organism with defense mechanisms against invading pathogens and tumor development and it plays an active role in tissue and organ regeneration. Deviations from the normal physiological functioning of the immune system can lead to the development of diseases with various pathologies including autoimmune diseases and cancer. Modern research in immunology is characterized by an unprecedented level of detail that has progressed towards viewing the immune system as numerous components that function together as a whole network. Currently, we are facing significant difficulties in analyzing the data being generated from high-throughput technologies for understanding immune system dynamics and functions, a problem known as the curse of dimensionality. As the mainstream research in mathematical immunology is based on low-resolution models, a fundamental question is how complex the mathematical models should be. To respond to this challenging issue, we advocate a hypothesis-driven approach to formulate and apply available mathematical modelling technologies for understanding the complexity of the immune system. Moreover, pure empirical analyses of immune system behavior and the system's response to external perturbations can only produce a static description of the individual components of the immune system and the interactions between them. Shifting our view of the immune system from a static schematic perception to a dynamic multi-level system is a daunting task. It requires the development of appropriate mathematical methodologies for the holistic and quantitative analysis of multi-level molecular and cellular networks. Their coordinated behavior is dynamically controlled via distributed feedback and feedforward mechanisms which altogether orchestrate immune system functions. The molecular regulatory loops inherent to the immune system that mediate cellular behaviors, e.g., exhaustion, suppression, activation, and tuning, can be analyzed using mathematical categories such as multi-stability, switches, ultra-sensitivity, distributed system, graph dynamics, or hierarchical control. GB is supported by the Russian Science Foundation grant 18-11-00171. AM is also supported by grants from the Spanish Ministry of Economy, Industry and Competitiveness and FEDER grant no. SAF2016-75505-R. The Mar a de Maeztu Programme for Units of Excellence in R+D MDM-2014-0370 and the Russian Science Foundation grant 18-11-00171.

### **Methods, Models, and Machine Learning Approaches for Understanding Pathogen-specific Humoral Immunity**

Tomer Zohar, 2022 The humoral immune response is comprised of vast libraries of polyclonal antibodies capable of recognizing a myriad of targets and directing a spectrum of innate immune functions. The complex heterogeneity in antibody profiles across both populations and diseases makes defining mechanisms of protection difficult. Understanding these

mechanisms and the factors that influence them is essential to defining immunity and helps inform the design of vaccines and therapeutics. Thus in this thesis I describe five studies that present the development of experimental and computational methods and machine learning approaches for investigating the mechanisms, dynamics and determinants of pathogen specific humoral immunity. The first study introduces an assay for probing antigen specific antibody mediated primary monocyte phagocytosis that is capable of capturing subsequent downstream functions. The second study describes a machine learning approach for defining the correlates of upper and lower respiratory protection against RSV and methods for evaluating vaccine designs. The third study uses machine learning methods to uncover signatures of humoral protection against SARS CoV 2. The fourth study presents a method for longitudinally modelling humoral immunity that was used to investigate the temporal dynamics of antibody features across individuals with varying COVID 19 severity. Finally the last study describes a genome wide association screen of pathogen specific polyclonal antibody characteristics and functions that was then validated with transcriptomics data. Ultimately the methods described in this thesis present new approaches for investigating underlying phenomena related to pathogen specific humoral immunity.

**Macromolecular Structure Underlying Recognition in Innate Immunity** Uttara SenGupta, Uday Kishore, Maha Ahmed Al-Mozaini, 2018-07-09 *Building a Robust Immune Response* Moria Cairns Chambers, 2012. How does your immune system prepare for all of the potential pathogens it might face over the course of a lifetime? Trade offs occur when you invest in responses that are beneficial when fighting one pathogen but are actively detrimental for fighting another. Due to the diversity of pathogens, immunity is potentially rife with this type of antagonism. To appreciate the full scope of potential trade offs, we must think about all of the possible immune responses a host can bring to bear on a pathogen. I propose that an immune response is any response to infection that influences the outcome of that infection. This includes processes that affect either resistance (the ability to clear pathogen) or tolerance (the ability to cope with pathology induced by infection). This broad definition of immunology will bring research of classically non immune physiologies (metabolism, circadian rhythm and mating) into the immune arena. Throughout this thesis I will explore using *Drosophila melanogaster* as a model a number of different trade offs in immunity from the antagonism inherent in a resistance response to the benefits and consequences of energy expenditure during infection. First I demonstrate that there is an inherent trade off due to investment in phagocytosis when flies encounter two different infections: *Listeria monocytogenes* and *Streptococcus pneumoniae*. *L. monocytogenes* is a facultative intracellular pathogen that harnesses the additional phagocytosis increasing entry into a desirable niche. *S. pneumoniae* is an extracellular pathogen that is better cleared by increased phagocytosis. I discovered the trade off by comparing and contrasting the phenotypes caused by mutants in two *Drosophila* immunity genes: *ets21c* (a putative transcription factor) and *wntD* (a negative regulator of immunity). Further exploration of the immune phenotypes of the *ets21c* mutant revealed that these mutants have a range of phenotypes during infection, suggesting a complex picture. *Ets21c* affects both tolerance and resistance to infection and the

class of phenotype observed in *ets21c* mutants cannot be predicted solely by the intracellular versus extracellular nature of the infecting pathogen. *Ets21c* mutants also have a strikingly altered basal metabolic state resembling sick wild type flies and have a muted change in transcript levels in response to infection. This thesis also deepens our understanding of developmental immune pleiotropy in the *wntD* pathway. Pleiotropy itself causes trade offs, while pleiotropy promotes efficiency in the genome, it also restricts the ability to evolve. *WntD*, a negative regulator of the toll pathway, impacts both immunity and dorsal ventral development. Recent work with the developmental phenotypes led to the discovery of components in the *wntD* signaling pathway. I show that these developmental mediators are also involved in immunity and impact survival during *L. monocytogenes* infection. *L. monocytogenes* infection causes infection-induced anorexia in *Drosophila*, and this thesis shows that infection with *L. monocytogenes* affects a number of metabolic pathways at both the transcript and metabolite level. This metabolic and transcriptome data generated a number of more specific and mechanistic hypotheses concerning additional potential trade offs. First, energy stores, metabolic intermediates, and transcripts for beta oxidation and glycolysis decrease during infection. This reduction of available energy can both negatively impact the host when it runs out of energy for essential processes and positively impact the host by restricting the nutrients available to the pathogen. By infecting mutants with either initially low energy stores or an inability to access stores, we show that access to energy stores is important to the host during infection, although the flip side of this trade off remains untested. A second potential trade off seen through our metabolomics are changes in the level of an anti-oxidant, uric acid. The flies enzymatically reduce levels of uric acid during *L. monocytogenes* infection. A reduction in an anti-oxidant should cause the reactive oxygen species to have additional potency. This would be helpful in combating the bacterial load but potentially detrimental due to an increase of damage to the host itself. However, mutants in *uricase*, which fail to lower uric acid levels during infection, do not have such easily explainable phenotypes, potentially due to compensation through other anti-oxidants. While not conclusive, these data suggest that the flies regulate their anti-oxidant levels during infection and that this complexly affects immunity. To address the dilemma of how to build a robust immune response, I contend that one must consider many different variables: diversity of pathogens, genetic efficiency, and the energetic cost. Years of evolution have honed the immune responses with many potential solutions. I found that *Drosophila* immune systems are likely constrained by a variety of tradeoffs, antagonistic abilities of resistance responses, metabolic links with immunity, and developmental immune pleiotropy. We still need to better understand how these tradeoffs are regulated and their downstream implications. Understanding these antagonistic relationships will help us manipulate them to develop more effective treatment, as we can tailor medicine to the individual pathogen and the individual person's physiology.

Host-pathogen Interaction Using the Whole Blood Models Sravya Sreekantapuram, 2022\*

In order to investigate the complex interactions between the immune cells and pathogens, the aim of this thesis was to establish an *ex vivo* whole blood model using murine and avian blood and to investigate how the model pathogens *Candida*



albicans Escherichia coli Staphylococcus aureus Salmonella Enteritidis and Salmonella Gallinarum interact with various host components in this environment In the first part of our study the established whole blood model was used to understand the immune responses in the peripheral blood of two chicken lines differing in egg laying performance to infection with either C albicans S aureus or E coli Our results demonstrated chicken line and pathogen dependent differences in pathogen survival immune cells viability and their interactions with the pathogens Comparing different avian leukocyte subsets the bacterial pathogens were found to be most associated with monocytes followed by the granulocytes In contrast C albicans more frequently interacted with granulocytes and at a lower rate with monocytes C albicans was observed to have stronger impact on immune cell viability in chicken than to the bacterial species These studies were furthermore extended to Salmonella enterica sp which are important and widely studied pathogens in chickens The second part of the study provided insights into interactions between murine blood and C albicans E coli and S aureus Our results indicated relatively low pathogen clearance and pathogen dependent differences regarding rates of association with immune cells Using a filament deficient C albicans mutant it was shown that increased filamentation does not explain the reduced killing Likewise the lower absolute number of neutrophils in murine blood could not fully explain higher fungal survival Lack of prior exposure to pathogens and absence of adaptive responses such as antibodies appear to contribute to low pathogen clearance

### **Innate Immune Cell Recruitment and Host Defense in Response to Intracellular Bacterial Infection** William Jerome Bunce Vincent, 2017

The interactions between invading pathogens and host immune cells that respond to infection is a long and involved relationship In particular a class of pathogens have evolved to not only evade clearance by the cells of the innate immune system but do so by manipulation of the host cytosol This proximity has led to the intricate co evolution of clearance and evasion mechanisms of the host and pathogen respectively How these interactions are carried out in the cellular immune response is the subject of this dissertation In Chapter 2 I build a localized infection model for one such pathogen Listeria monocytogenes using the transparent zebrafish larval host Using this model I demonstrate the functional presence of a conserved innate immune sensing pathway the inflammasome that surveils the cytosol for the presence of bacterial flagellin Upon optimal inflammasome activation the host is protected from lethal infection and I show that macrophages are the crucial host cell population in this defense In Chapter 3 I characterize L monocytogenes utilization of the host actin network demonstrating live imaging of this interaction for the first time in vivo Once L monocytogenes is localized within the cytosol it slows down macrophage motility although in an actin independent manner In Chapter 4 I develop a model to study the recruitment and resolution of leukocytes during concurrent wounding and infection This model demonstrates both beneficial and detrimental leukocyte responses and provides evidence that the immune responses to bacterial infection and wounding are separable programs during early stages of immune response Taken together I have shown that macrophages are a crucial part of host defense downstream of inflammasome activation that macrophages activate other cell autonomous responses

during their interaction with intracellular pathogens and that macrophage neutrophil interactions can be critical in determining a beneficial or detrimental response to infection These findings highlight the intricate and close evolution of host immune cells and intracellular pathogens as well as the critical nature of inflammatory balance during immune responses

*How the Immune System Learns from Infections* Hongda Jiang, 2022 The immune system is a complex system of cells and molecules that work cooperatively to protect us against pathogenic organisms It can perform complicated tasks such as pattern recognition learning and memory all of which require dynamical coordination among a large number of components across multiple scales Nevertheless the multitude of different components makes it challenging to unveil the mechanistic principles that give rise to these remarkable functions My thesis focuses on how our immune system learns from infections and improves specificity of pathogens recognition on the fly This process is known as affinity maturation where the affinity of B cell receptor improves through Darwinian evolution Although recent progresses in experiments revealed many details what remains is a first principle and quantitative understanding of how different elements come together to achieve the goal Using statistical physics tools and computational modeling I study various aspects of the maturation process including molecular interactions information extraction and evolutionary dynamics To understand how B cells with different affinities are discriminated during affinity maturation we investigate the process of antigen extraction where B cells use cytoskeleton forces to extract antigen molecules from other presenting cell surface We show this process allows a B cell to infer its receptor affinity by measuring the number of extracted antigens Our model highlights the regulatory role of mechanical force Application of a constant force with proper magnitude can enhance discrimination fidelity and usage of a dynamical force that introduces negative feedback can improve discrimination robustness with respect to fluctuations in antigen concentration To illustrate how molecular interactions influence cellular evolution we couple the physical theory of antigen extraction to a minimal model of affinity maturation and simulate ensembles of cell populations under different conditions The multiscale model predicts that the affinity ceiling stems from the physical limit of antigen tether strength and identifies strategies to alleviate the constraint Lastly we present a study on the long term coevolution between evolving pathogen and adaptive immune response Our work reveals that the asymmetric reaction range between immunogenicity the ability of pathogens to induce an immune response and antigenicity the ability of pathogens to interact with antibodies is critical in determining the dynamics of coevolution

**Crossroads Between Innate and Adaptive Immunity IV** Peter D.

Katsikis, Stephen P. Schoenberger, Bali Pulendran, 2013-02-28 This volume presents a collection of reviews derived from work presented at the Aegean Conference 4th Crossroads between innate and adaptive immunity This meeting was the fourth in a series and assembled a team of scientists working on mechanisms by which the innate immune system of the host senses pathogens the cellular and signaling networks that orchestrate the innate response and antigen presentation and adaptive immunity The importance of the crosstalk between innate immunity and the adaptive immune response has only recently

started to be appreciated Although it is well recognized that dendritic cells NK cells NK T cells and T cells are all critical for the host response to pathogens the respective fields that study the biology of these immune cells tend to exist in parallel worlds with minimum exchange of information and ideas This fragmentation hinders the integration of these fields towards a unified theory of host response The Aegean Conference Crossroads between Innate and Adaptive Immunity brought together leading international scientists and experts to address critical areas of Innate and Adaptive immunity something necessary for the development of more efficient scientific exchange and crosspollination between these fields This conference attracted scientists from all over the world to discuss their latest findings on the various aspects of Innate and Adaptive immunity The conference had limited participation and a scientific and social program that maximized scientific interchange through lecture presentations poster sessions and informal discussions

### **Orchestration of an Immune Response to Respiratory**

**Pathogens** Andrea Sant, Steven Varga, 2019 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](mailto:frontiersin.org) about contact

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