Mini Proposal Bootcamp

Stanford BIOSCIENCES GRANT WRITING ACADEMY

Stanford MEDICINE | Stanford Biosciences Student Association

Organizers:


Crystal Botham, PhD


Courtney Peña, PhD


Lily Xu


Miles Tyner

Mini Bootcamp Activities:

Workshops - Wednesdays 1-2:30 PM


Orit Rapaport, PhD

- Getting Started on Your Proposal
- Writing Crystal Clear Specific Aims
- Crafting a Compelling Training Plan
- Addressing the Why, What, & How of the Research Strategy

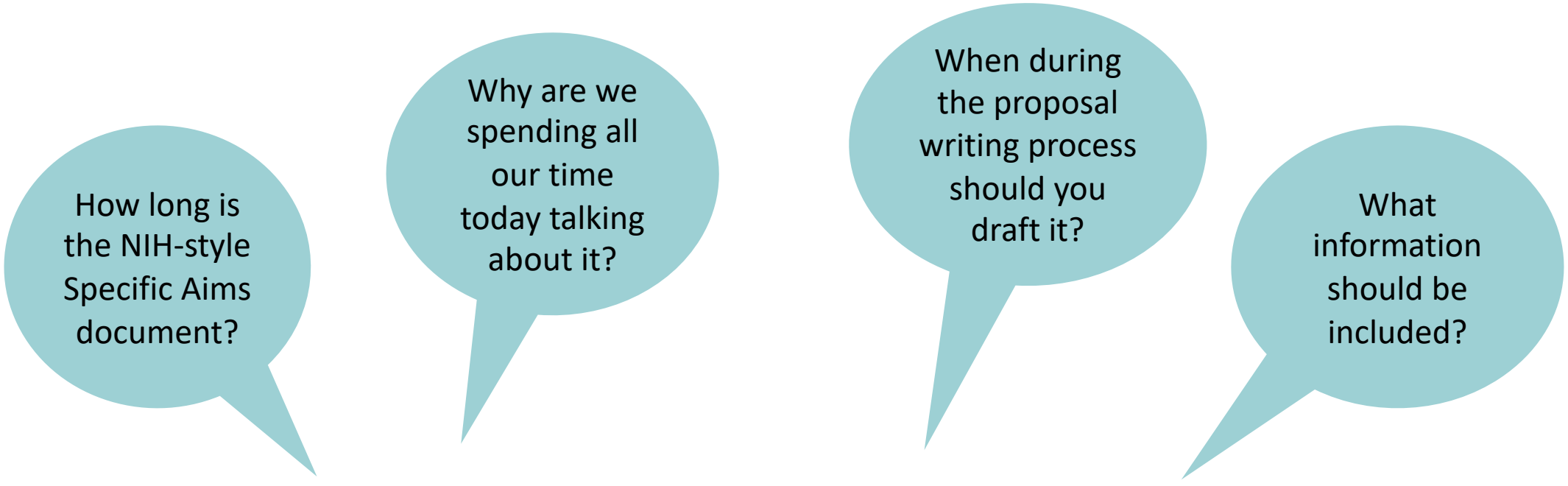
Time to Write - Thursdays 4-6 pm


Leah Guthrie, PhD

- A time to work on your proposal or other writing project
- Zoom or in-person (Lorry Lokey Stem Cell Research Building)

* In-person get $5 gift card to Peet's

Share what you know about the NIH-style Specific Aims document



Specific Aims Instructions

National Institutes of Health (NIH)
*** 1 page ***

State concisely the goals of the proposed research and summarize the expected outcome(s), including the impact that the results of the proposed research will exert on the research field(s) involved.

List succinctly the specific objectives of the research proposed, e.g., to test a stated hypothesis, create a novel design, solve a specific problem, challenge an existing paradigm or clinical practice, address a critical barrier to progress in the field, or develop new technology.

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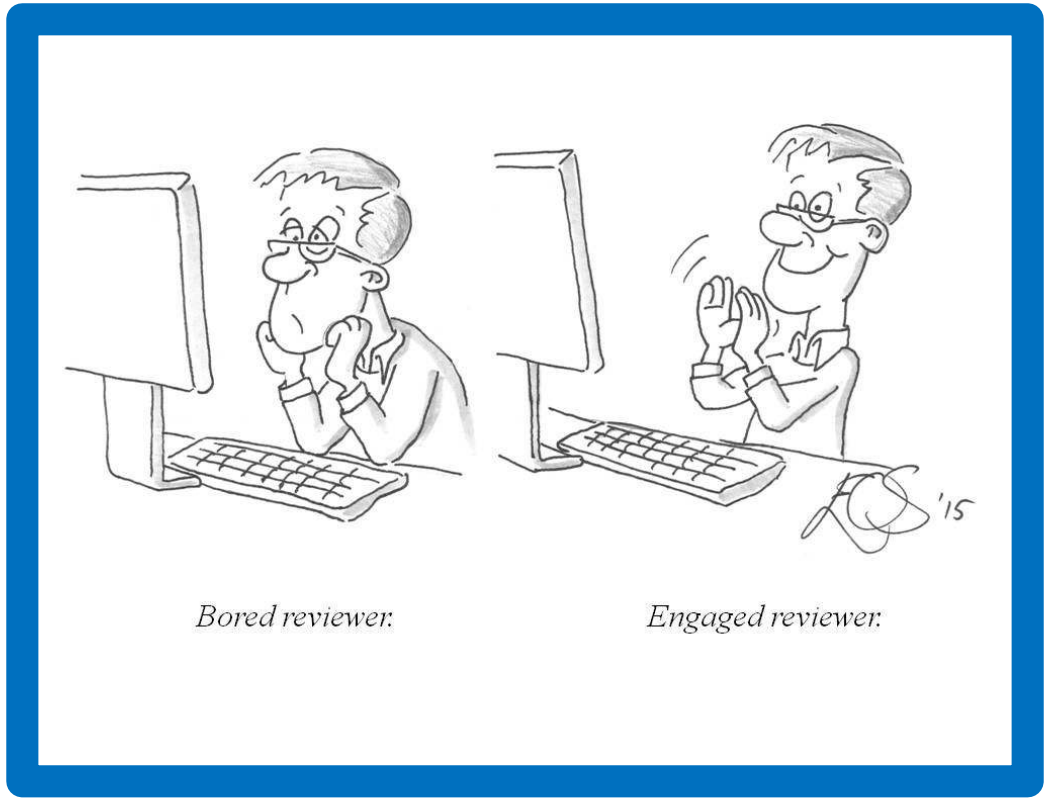
Specific Aims: First document Reviewers look at!



Why is the Specific Aims document so important?

Watch: NIH Peer Review Reviled to learn more about the review process.

Reviewers will read it!

1 page is perfect for eliciting feedback from mentors and colleagues!

Roadmap for the rest of the proposal!

Specific Aims Examples

Example 1

Contact PD/PI: Putnam, Nicole E

SPECIFIC AIMS

The impact of innate immune recognition of *Staphylococcus aureus* on bone homeostasis and skeletal immunity

Bone is constantly remodeled through the coordinated efforts of bone-forming osteoblasts (OBs) and bone-resorbing osteoclasts (OCs). This process is referred to as bone homeostasis and is tightly regulated by local and systemic factors, including cytokines, hormones, and growth factors. *Staphylococcus aureus* is the leading cause of invasive bone infection (osteomyelitis), during which inflammation leads to altered interactions between skeletal cells. Dysregulation in bone homeostasis triggers aberrant bone formation and bone destruction, which may result from changes in skeletal cell physiology during osteomyelitis that are distinct from cell death. Our preliminary data show that bacterial components modulate the differentiation of OCs (osteoclastogenesis) from myeloid cells with and without the canonical OC differentiation factor, receptor activator of nuclear factor κB-ligand (RANKL). Specifically, BM treatment with *S. aureus* supernatants induces OC differentiation without canonical RANKL signaling, and limits OC formation when pretreated with RANKL. The primary objective of this proposal is to define the mechanisms by which bacterial pathogens alter osteoclastogenesis to impact bone homeostasis and skeletal immunity.

Skeletal cells are known to express innate pattern recognition receptors (PRRs), but the contribution of innate sensing by OC PRRs, such as Toll-like receptors (TLRs) towards pathogen clearance and bone remodeling during *S. aureus* osteomyelitis has not yet been explored. In order to further define the contribution of skeletal cell PRRs to altered bone homeostasis and antibacterial immunity during osteomyelitis, we focused on the critical PRR signaling adaptor MyD88, which is required for TLR and IL-1 family cytokine signaling. In preliminary studies, data support a MyD88-mediated mechanism by which bacteria perturb OC differentiation, emphasizing the importance of innate signaling in modulating osteoclastogenesis. Overall, I hypothesize that *S. aureus* modulates OC precursor (pre-OC) cell biology and bone remodeling through ligation of OC PRRs and the induction of inflammation. To test this hypothesis, we propose two integrated Aims that will define how *S. aureus* perturbs the differentiation and functional ability of OC-like cells to resorb bone, and determine how innate activation of skeletal cells affects bacterial clearance and bone homeostasis in a powerful new osteomyelitis murine model that is capable of precise quantification of pathogen-induced changes in bone turnover. The Aims will elucidate bacterial-induced mechanisms of altered bone remodeling and further define the ability of skeletal cells to respond to *S. aureus*. These studies have the potential to significantly impact human health by identifying therapeutic targets to limit bone destruction during osteomyelitis. The Aims are:

Aim 1: Define the role of TLRs and IL-1R in *S. aureus*-mediated perturbation of osteoclastogenesis.

Based on preliminary studies that suggest a MyD88-mediated mechanism of OC perturbation by bacterial components *in vitro,* I hypothesize that *S. aureus* modulates pre-OC cell biology through TLR recognition or IL-1R signaling upstream of MyD88. To test this hypothesis, we will perform osteoclastogenesis assays on bone marrow (BM) cultures from wild-type and immune-deficient mouse strains, including TLR2, TLR9, and IL-1R-deficient mice, with and without RANKL stimulation, components of *S. aureus*, TLR agonists, or recombinant IL-1 to (i) identify changes in expression of TLRs and factors known to modulate osteoclastogenesis, (ii) define the activation status of intracellular signaling cascades and transcription factors, and (iii) investigate the functionality of OCs induced by bacterial components with bone resorption assays. Taken together, these data will detail how bacterial stimulation modulates OC differentiation and function through TLR and IL-1 signaling.

Aim 2: Elucidate the role of skeletal cell-specific MyD88 signaling on pathogen clearance and bone remodeling during *S. aureus* osteomyelitis.

Aim 1 will identify *in vitro* changes caused by *S. aureus* during osteoclast differentiation, including alterations in OC signaling and function. Our *in vitro* assays demonstrate that MyD88 in skeletal cell precursors could be responsible for downstream changes following *S. aureus* stimulation. Interestingly, preliminary data obtained in our *S. aureus* osteomyelitis model shows that MyD88 is also necessary to limit bacterial replication and dissemination to other organs. Based on these data, I hypothesize that innate sensing of *S. aureus* by skeletal cells *in vivo* impacts bacterial clearance and alters bone remodeling during osteomyelitis. To test this hypothesis we will induce osteomyelitis in wild-type mice and mice with skeletal cell-specific MyD88 deletion to (i) differentiate the kinetics of pathogen clearance from bone and bacterial dissemination to other organs, (ii) investigate bone remodeling alterations in cortical and trabecular bone using micro-computed tomography (microCT) analysis, and (iii) quantify osteoclast differentiation *in vivo* through histological assessment. Collectively, these Aims will investigate how innate immune activation of skeletal cells alters bone homeostasis, thereby elucidating fundamental mechanisms of osteo-immunologic crosstalk.

Example 2

Research Plan
2. Specific Aims

Eukaryotic innate immune systems act as effective barriers to infection by microorganisms. Understanding the mechanisms that bacterial pathogens employ to circumvent innate immune systems will improve our ability to control disease. Plants and animals use specific pattern recognition receptors (PRRs) to recognize conserved molecules of microorganisms (known as PAMPs). Plants have numerous PRRs that can recognize specific virulence proteins specifically present in pathogens (known as Avr proteins). Many Gram-negative bacteria use type III protein secretion systems to inject effector proteins into host eukaryotic cells. We have shown that a primary role for many *Pseudomonas syringae* type III effectors is to suppress innate immunity. However, the enzymatic activities and the mechanisms that type III effectors use to suppress innate immunity are not well understood. Identifying the enzymatic activities of type III effectors and their substrates is essential to identify important components of innate immunity and to improve strategies to control bacterial diseases.

Our *long-term goal* is to elucidate the molecular basis for suppression of innate immunity by type III effectors. The objective of this application is to identify targets of the *P. syringae* type III effector HopU1, a mono-ADP-ribosyltransferases (ADP-RTs), and to determine its roles in bacterial pathogenesis. *The central hypothesis of the proposed experiments is that the targets of the HopU1 ADP-RT type III effector will be components of innate immunity.* We formulated this hypothesis based on the literature and on our research on other type III effectors as well as our preliminary data showing that HopU1 suppresses outputs of innate immunity. Recently, we have shown that HopU1 can use several *Arabidopsis* RNA-binding proteins as high affinity substrates in *in vitro* ADP-RT assays. Based on our preliminary data, one of these proteins, *At*GRP7, plays a role in innate immunity. A major goal of this application is to elucidate the function of this protein as it relates to innate immunity. We are prepared to undertake the proposed research because we have extensive experience in manipulating type III systems, and we were among the first to report that certain type III effectors suppress innate immunity. In addition, our preliminary identification of HopU1's substrates has positioned us well to perform the experiments described in this application. Our research team includes experts in the following areas: type III secretion systems, proteomics and mass spectrometry, Affymetrix microarrays, plant glycine-rich RNA-binding proteins, and animal pathogen ADP-RTs. This qualified group of investigators will insure that our discoveries are linked to basic concepts of pathogenesis and immunity in both plants and animals.

The Specific Aims of this application are as follows:

***1.* Determine the molecular consequence of ADP-ribosylation on the function of *At*GRP7 and elucidate the role this protein plays in innate immunity.** Our *working hypothesis* of this aim is that *At*GRP7 binds to immunity-related RNAs to enhance the innate immune response and that ADP-ribosylation by HopU1 disrupts its function.

***2.* Identify additional substrates of HopU1 and verify their involvement in innate immunity.** Our *working hypothesis* is that the plant targets for the HopU1 ADP-RTs will be important components of plant innate immunity.

***3.* Analyze the affect that HopU1 has on host-microbe interactions.** Our *working hypothesis* of this aim is that HopU1 type III effector suppresses innate immunity. This is based on our preliminary data and in this aim we will determine to what extent this occurs with *HopU1*.

The proposed research is innovative because, to date, ADP-RTs have not been implicated in the suppression of innate immune surveillance systems. Moreover, RNA-binding proteins have not been described as substrates for ADP-RTs and, therefore, represent novel substrates for this important group of bacterial toxins. Collectively, we expect the outcomes of these experiments will greatly add to our understanding of the activities and roles of type III effectors, particularly in how they suppress innate immunity in eukaryotes.

Example 3

SPECIFIC AIMS

Epidemiologic studies suggest that obese breast cancer patients experience poorer outcomes than their normal-weight counterparts (1-3), with a recent meta-analysis of 82 studies reporting that obese breast cancer cases have 41% higher total mortality than normal-weight cases (95% CI: 29%-53%) (1). It has been suggested that obese women may experience poorer outcomes, in part, due to the greater likelihood of dose reduction in chemotherapy (4-6). For most cytotoxic drugs, dose is calculated using body surface area (BSA); therefore, obese women would be expected to receive a higher absolute dose than normal weight women. However, due to concern about inducing chemotherapy-associated toxicity, clinicians are more likely to reduce the dose administered to obese women than normal-weight women (7), a finding replicated in several studies (8-12). While dose reductions may be warranted for reasons such as certaincomorbidities and toxicities, in 2012, the American Society of Clinical Oncology (ASCO) released guidelines urging clinicians to end this practice on the grounds of obesity, given research showing that obese women dosed at their BSA-determined dose are no more likely to experience toxicity than their normal weight counterparts (5). The guidelines were met with some criticism, citing the need for further research on adequate dosing (13).
Understanding the drivers of dose reductions may help better inform our understanding of this practice and efforts to disseminate guidelines; however, we know little about factors driving dose intensity, and how these factors may vary by body size.

Two major questions pertaining to the ASCO guidelines remain unanswered. First, while these guidelines suggest that chemotherapy dose reductions among obese patients may, in part, explain the association between obesity and breast cancer survival (5), this question has not been evaluated. **Demonstrating that dose reductions contribute to the associations between obesity and adverse outcomes would strengthen the case for these guidelines, and would provide a clear point of intervention to improve prognosis for obese women.** Second, the guidelines acknowledge that data pertaining to risk of toxicity are extremely limited with regard to *more severe obesity*, and in the real-world context of the presence of obesity-associated *comorbid conditions* (5). **If women with larger body sizes receiving the BSA-determined dose of chemotherapy do not experience excess toxicity as compared to normal weight women, this would provide further evidence in favor of the ASCO guidelines.**

Our interdisciplinary team, with expertise in epidemiology, pharmacology, and breast medical oncology is uniquely positioned to address these questions. Taking advantage of the rich data of two integrated healthcare delivery systems, Kaiser Permanente Northern California (KPNC) and Group Health (GH), in nearly 34,000 early-stage (Stage I-IIIA) breast cancer patients, we will address the following **Specific Aims:**

1. Identify predictors of chemotherapy dose intensity, focusing on whether body size is a principal driver of dose reduction, and whether other predictors of dose reduction vary by body mass index. Factors to be considered include patient-level factors (e.g., age, race/ethnicity, level of obesity, comorbidities such as diabetes, kidney disease, or cardiovascular disease), disease characteristics (e.g., stage, nodal involvement, grade, hormone receptor status), treatment, and provider-level factors (e.g., practice size, gender, age, provider-patient racial concordance).
2. Evaluate associations between body size and breast cancer recurrence and survival, focusing on the role of chemotherapy dosing as a mediator of these associations.
3. Among women identified as receiving BSA-expected dosing of chemotherapy, evaluate the association between BMI and toxicity; this will reveal if women receiving BSA-determined dosing at higher BMIs experience more or less toxicity than their non-obese counterparts. Toxicities include neutropenia and neuropathy, cardiotoxicity, renal impairment, and hepatic impairment.

Understanding these points will better inform clinical management for the estimated 102,000 obese women diagnosed with breast cancer each year in the United States (14, 15). It is critical that we address this issue, given that approximately 40% of adult women in the United States are obese and the prevalence of obesity continues to rise among women (14). With detailed information on treatment, comorbid conditions, and toxicities from KPNC and GH, as well as follow-up for recurrence and survival, we are uniquely positioned to address these questions about which little is known, but which have direct clinical relevance. In fact, to our knowledge, this is only such setting in which such a large-scale study can take place. Given our experience, expertise, and access to this rich data source, our team is ideally suited to address this important, novel, and timely avenue of research.

Breakout Room - Activity #1 (30 min.)

- Introductions
  - Name, pronouns, department, favorite ice cream
- Pick a Specific Aims draft as a group
  - Read Specific Aims draft
  - How much, if any, of your hard-earned money do you want to fund the proposed research?

Specific Aims Examples

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Why?

Example 1

Contact PD/PI: Putnam, Nicole E

SPECIFIC AIMS

The impact of innate immune recognition of *Staphylococcus aureus* on bone homeostasis and skeletal immunity

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Example 2

Research Plan
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SPECIFIC AIMS

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I read lots of Specific Aims each year!

Specific Aims Examples

Proposed research is important

Goal is appropriate for PI / team

Aims address specific questions

Return on investment is expected

Contact PD/PI: Putnam, Nicole E

SPECIFIC AIMS

The impact of innate immune recognition of *Staphylococcus aureus* on bone homeostasis and skeletal immunity

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2. Specific Aims

Eukaryotic innate immune systems act as effective barriers to infection by microorganisms. Understanding the mechanisms that bacterial pathogens employ to circumvent innate immune systems will improve our ability to control disease. Plants and animals use specific pattern recognition receptors (PRRs) to recognize conserved molecules of microorganisms (known as PAMPs). Plants have numerous PRRs that can recognize specific virulence proteins specifically present in pathogens (known as Avr proteins). Many Gram-negative bacteria use type III protein secretion systems to inject effector proteins into host eukaryotic cells. We have shown that a primary role for many *Pseudomonas syringae* type III effectors is to suppress innate immunity. However, the enzymatic activities and the mechanisms that type III effectors use to suppress innate immunity are not well understood. Identifying the enzymatic activities of type III effectors and their substrates is essential to identify important components of innate immunity and to improve strategies to control bacterial diseases.

Our *long-term goal* is to elucidate the molecular basis for suppression of innate immunity by type III effectors. The objective of this application is to identify targets of the *P. syringae* type III effector HopU1, a mono-ADP-ribosyltransferases (ADP-RTs), and to determine its roles in bacterial pathogenesis. *The central hypothesis of the proposed experiments is that the targets of the HopU1 ADP-RT type III effector will be components of innate immunity.* We formulated this hypothesis based on the literature and on our research on other type III effectors as well as our preliminary data showing that HopU1 suppresses outputs of innate immunity. Recently, we have shown that HopU1 can use several *Arabidopsis* RNA-binding proteins as high affinity substrates in *in vitro* ADP-RT assays. Based on our preliminary data, one of these proteins, *At*GRP7, plays a role in innate immunity. A major goal of this application is to elucidate the function of this protein as it relates to innate immunity. We are prepared to undertake the proposed research because we have extensive experience in manipulating type III systems, and we were among the first to report that certain type III effectors suppress innate immunity. In addition, our preliminary identification of HopU1's substrates has positioned us well to perform the experiments described in this application. Our research team includes experts in the following areas: type III secretion systems, proteomics and mass spectrometry, Affymetrix microarrays, plant glycine-rich RNA-binding proteins, and animal pathogen ADP-RTs. This qualified group of investigators will insure that our discoveries are linked to basic concepts of pathogenesis and immunity in both plants and animals.

The Specific Aims of this application are as follows:

***1.* Determine the molecular consequence of ADP-ribosylation on the function of *At*GRP7 and elucidate the role this protein plays in innate immunity.** Our *working hypothesis* of this aim is that *At*GRP7 binds to immunity-related RNAs to enhance the innate immune response and that ADP-ribosylation by HopU1 disrupts its function.

***2.* Identify additional substrates of HopU1 and verify their involvement in innate immunity.** Our *working hypothesis* is that the plant targets for the HopU1 ADP-RTs will be important components of plant innate immunity.

***3.* Analyze the affect that HopU1 has on host-microbe interactions.** Our *working hypothesis* of this aim is that HopU1 type III effector suppresses innate immunity. This is based on our preliminary data and in this aim we will determine to what extent this occurs with *HopU1*.

The proposed research is innovative because, to date, ADP-RTs have not been implicated in the suppression of innate immune surveillance systems. Moreover, RNA-binding proteins have not been described as substrates for ADP-RTs and, therefore, represent novel substrates for this important group of bacterial toxins. Collectively, we expect the outcomes of these experiments will greatly add to our understanding of the activities and roles of type III effectors, particularly in how they suppress innate immunity in eukaryotes.

SPECIFIC AIMS

Epidemiologic studies suggest that obese breast cancer patients experience poorer outcomes than their normal-weight counterparts (1-3), with a recent meta-analysis of 82 studies reporting that obese breast cancer cases have 41% higher total mortality than normal-weight cases (95% CI: 29%-53%) (1). It has been suggested that obese women may experience poorer outcomes, in part, due to the greater likelihood of dose reduction in chemotherapy (4-6). For most cytotoxic drugs, dose is calculated using body surface area (BSA); therefore, obese women would be expected to receive a higher absolute dose than normal weight women. However, due to concern about inducing chemotherapy-associated toxicity, clinicians are more likely to reduce the dose administered to obese women than normal-weight women (7), a finding replicated in several studies (8-12). While dose reductions may be warranted for reasons such as certaincomorbidities and toxicities, in 2012, the American Society of Clinical Oncology (ASCO) released guidelines urging clinicians to end this practice on the grounds of obesity, given research showing that obese women dosed at their BSA-determined dose are no more likely to experience toxicity than their normal weight counterparts (5). The guidelines were met with some criticism, citing the need for further research on adequate dosing (13).
Understanding the drivers of dose reductions may help better inform our understanding of this practice and efforts to disseminate guidelines; however, we know little about factors driving dose intensity, and how these factors may vary by body size.

Two major questions pertaining to the ASCO guidelines remain unanswered. First, while these guidelines suggest that chemotherapy dose reductions among obese patients may, in part, explain the association between obesity and breast cancer survival (5), this question has not been evaluated. **Demonstrating that dose reductions contribute to the associations between obesity and adverse outcomes would strengthen the case for these guidelines, and would provide a clear point of intervention to improve prognosis for obese women.** Second, the guidelines acknowledge that data pertaining to risk of toxicity are extremely limited with regard to *more severe obesity*, and in the real-world context of the presence of obesity-associated *comorbid conditions* (5). **If women with larger body sizes receiving the BSA-determined dose of chemotherapy do not experience excess toxicity as compared to normal weight women, this would provide further evidence in favor of the ASCO guidelines.**

Our interdisciplinary team, with expertise in epidemiology, pharmacology, and breast medical oncology is uniquely positioned to address these questions. Taking advantage of the rich data of two integrated healthcare delivery systems, Kaiser Permanente Northern California (KPNC) and Group Health (GH), in nearly 34,000 [illegible]

1. Identify predictors of chemotherapy dose intensity, focusing on whether body size is a principal driver of dose reduction, and whether other predictors of dose reduction vary by body mass index. Factors to be considered include patient-level factors (e.g., age, race/ethnicity, level of obesity, comorbidities such as diabetes, kidney disease, or cardiovascular disease), disease characteristics (e.g., stage, nodal involvement, grade, hormone receptor status), treatment, and provider-level factors (e.g., practice size, gender, age, provider-patient racial concordance).
2. Evaluate associations between body size and breast cancer recurrence and survival, focusing on the role of chemotherapy dosing as a mediator of these associations.
3. Among women identified as receiving BSA-expected dosing of chemotherapy, evaluate the association between BMI and toxicity; this will reveal if women receiving BSA-determined dosing at higher BMIs experience more or less toxicity than their non-obese counterparts. Toxicities include neutropenia and neuropathy, cardiotoxicity, renal impairment, and hepatic impairment.

Understanding these points will better inform clinical management for the estimated 102,000 obese women diagnosed with breast cancer each year in the United States (14, 15). It is critical that we address this issue, given that approximately 40% of adult women in the United States are obese and the prevalence of obesity continues to rise among women (14). With detailed information on treatment, comorbid conditions, and toxicities from KPNC and GH, as well as follow-up for recurrence and survival, we are uniquely positioned to address these questions about which little is known, but which have direct clinical relevance. In fact, to our knowledge, this is only such setting in which such a large-scale study can take place. Given our experience, expertise, and access to this rich data source, our team is ideally suited to address this important, novel, and timely avenue of research.

Specific Aims: Answer key Questions

Is the question important?

What specifically will be done?

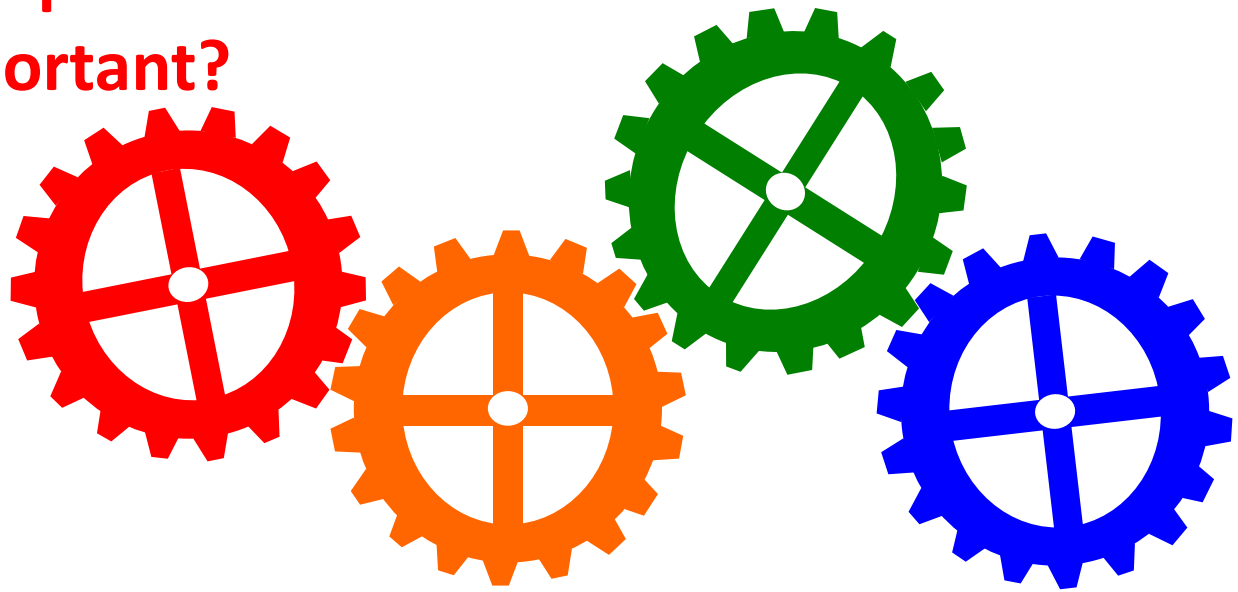
What is the overall goal?

What is the expected payoff?

Specific Aims: Gearing Up

Introductory Paragraph

Is the question important?



Introductory Paragraph

- **Attention grabbing first sentence**
  - Clearly relate to the institute's mission

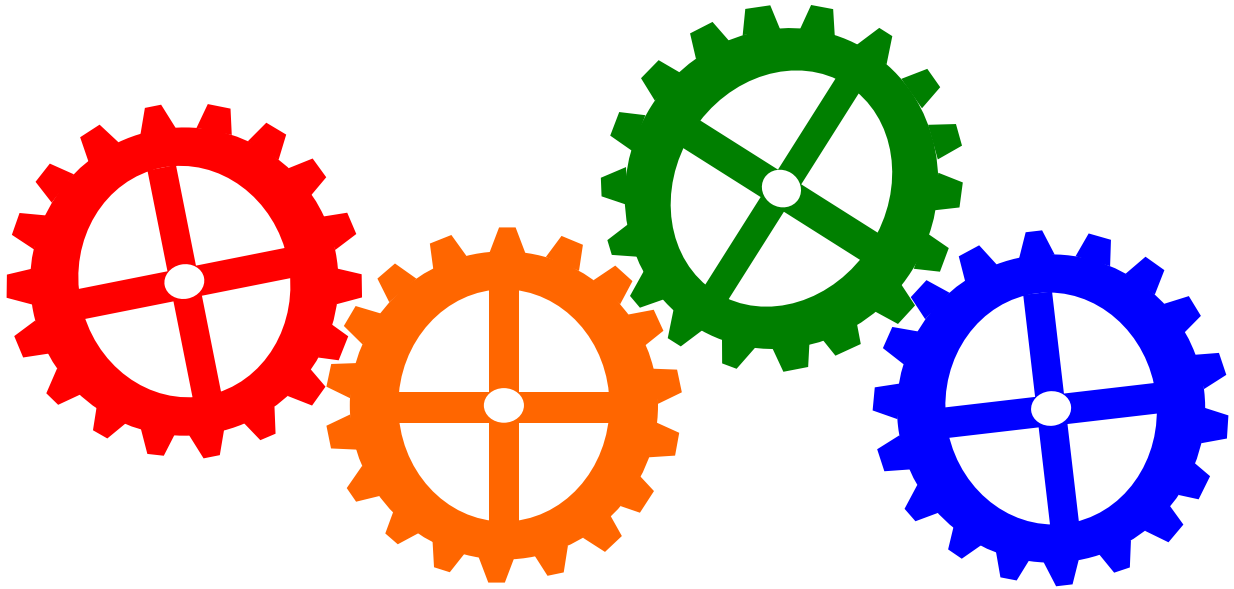
Introductory Paragraph

- **Attention grabbing first sentence**
- **Bring reviewers up to speed**
  - Summarize current knowledge
  - Lead the reviewer to what needs to be done next

Introductory Paragraph

- **Attention grabbing first sentence**
- **Bring reviewers up to speed**
- **Frame the knowledge gap or need**
  - Highlights what drives your application

Overall Goals Paragraph



What is the overall goal?

Overall Goals Paragraph

- **Big-picture goal**
  - ‘Long-term career goal’ that relates to the funding agency and your research niche

Overall Goals Paragraph

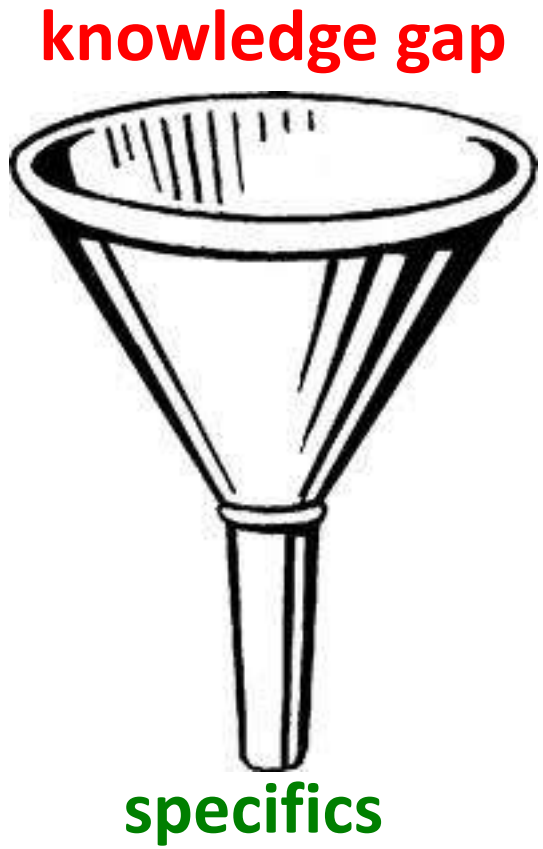
- **Big-picture goal**
- **Objective of this proposal**
  - Define the purpose of the proposal
  - Must fill the gap delineated above
  - Must be attainable, regardless of how the hypothesis tests
  - Emphasize the product of the research instead of the process

Overall Goals Paragraph

- **Big-picture goal**
- **Objective of this proposal**
- **Central hypothesis**
  - "Best bet"
  - Must be objectively testable
  - Gives focus to the research
  - Tightly linked to Specific Aims

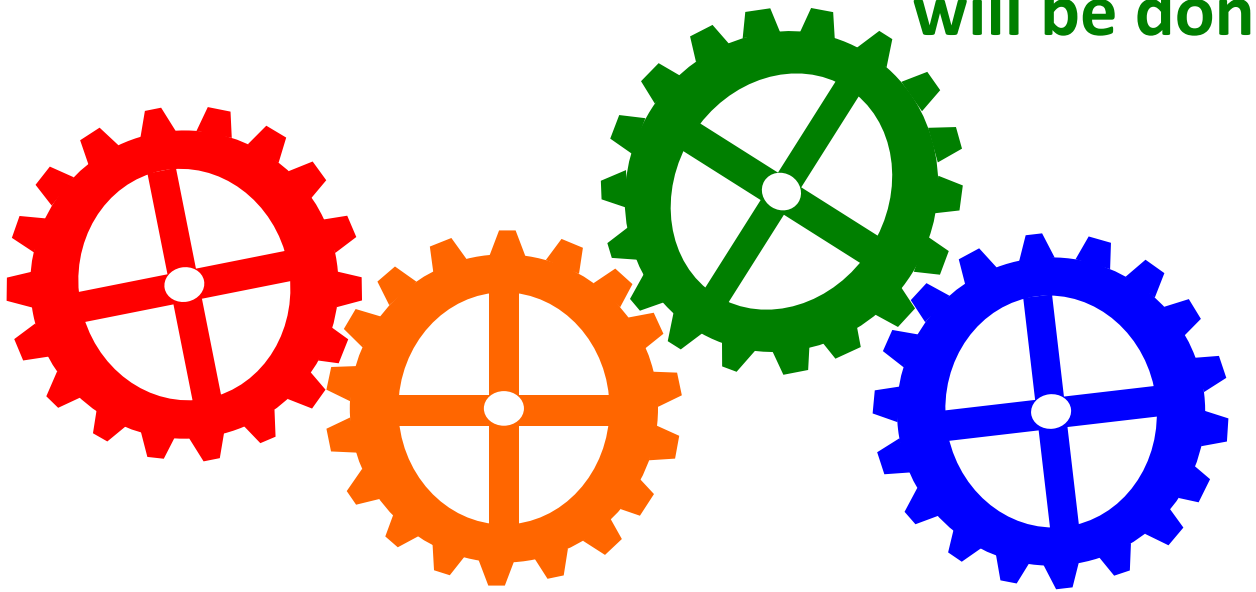
Overall Goals Paragraph

- Big-picture goal
- Objective of this proposal
- Central hypothesis
- Evidence in support of hypothesis



Specifics Paragraph

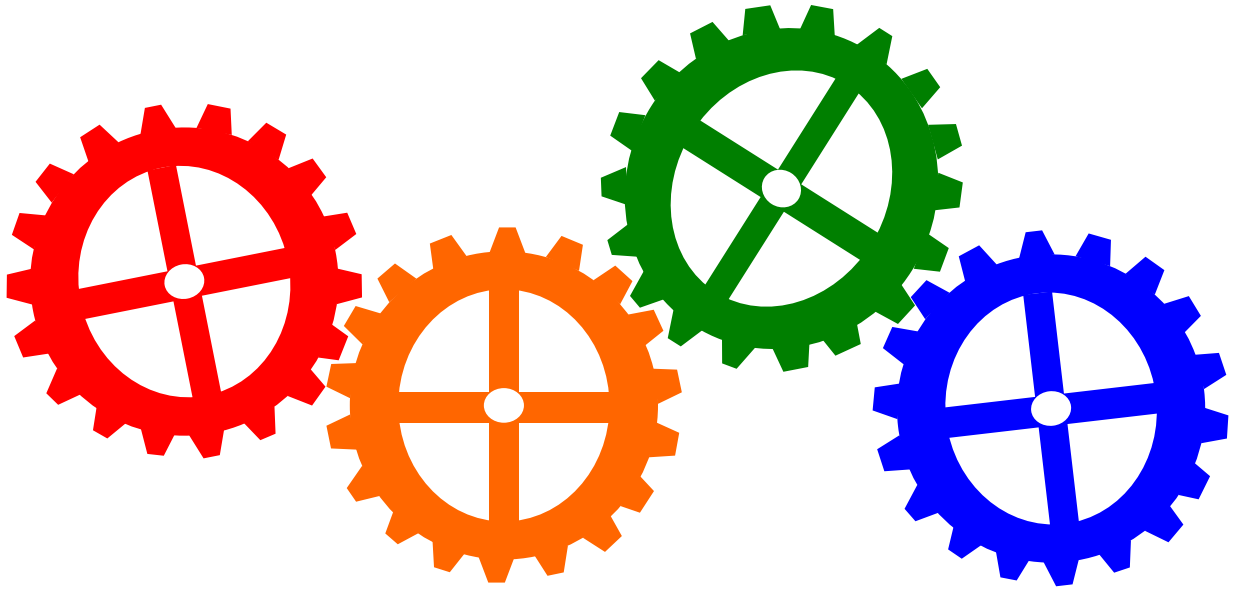
What specifically will be done?



Specifics Section

- Attention-getting headlines that will grab a reviewer's attention / interest
- NOT descriptive – no fishing expeditions
- Logical flow but not absolutely interdependent
- Written in a way that, no matter how the hypothesis tests, you will accomplish the aim's objective
- Deliberately broad and open-ended aim BUT then followed by a focused working hypothesis

Payoff Paragraph



What is the expected payoff?

Payoff Paragraph

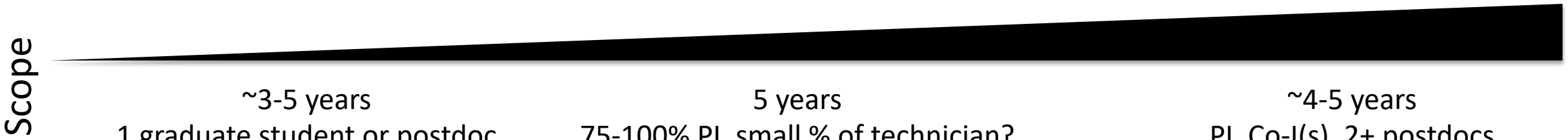
- Tells the reviewers what they can expect for a return on their investment
  - What are the expected products of the research?
  - What is the positive impact that contributes to the mission of the agency?
  - For Fellowships & Career Development Awards include your career progression too!

Gearing Up


Is the question important?
What specifically will be done?
What is the overall goal?
What is the expected payoff?

Comparing Different Types of Grants

Fellowships (e.g., NIH F31, F32)	Career Development Awards (e.g., NIH K01, K08, K99/R00)	Research Grants (e.g., NIH R01)
~3-5 years 1 graduate student or postdoc	5 years 75-100% PI, small % of technician? *(But R00 scope is like small R01)*	~4-5 years PI, Co-I(s), 2+ postdocs



Comparing Different Types of Grants

Fellowships (e.g., NIH F31, F32)	Career Development Awards (e.g., NIH K01, K08, K99/R00)	Research Grants (e.g., NIH R01)

Scope

Independence

Review Criteria

Candidate Career Development Plan Mentors Environment & Institutional Commitment Research Plan	Significance Investigators Innovation Approach Environment

Breakout Room - Activity #2 (6 min.)

- Discuss the questions you still have.
- Prioritize and pick one to ask today.

What questions do you still have about the NIH-style Specific Aims document?

Resources

Botham, Crystal, All About the NIH K Award Workbook, Chapter 8, https://grantwriting.stanford.edu/secured/k-award-workbook/

Hollenbach, Andrew. *A Practical Guide to Writing a Ruth L. Kirschstein NRSA Grant*. Amsterdam: Academic Press, 2014. [ISBN 978-0-12-420187-3]

Russell, Stephen W. and David C. Morrison. *The Grant Application Writer's Workbook: National Institutes of Health Version*. Los Olivos, CA: Grant Writers' Seminars and Workshops, LLC, 2016. <www.grantcentral.com>

Yang, Otto O. *Guide to Effective Grant Writing: How to Write an Effective NIH Grant Application*. New York: Springer US, 2012. [eBook ISBN 978-1-4614-1581-7]

Sample NIH applications and summary statements are available here: https://www.niaid.nih.gov/grants-contracts/sample-applications