TRAINING AND CAREER DEVELOPMENT PLAN

My career path has unfolded in an unlikely way that involved stops at a community college, a pharmaceutical start-up, and an agricultural biotechnology company. Each stop along the way has helped crystallize my strong desire to understand the molecular mechanism of plant disease. In pursuit of this goal I am pursuing my Ph.D. in plant-microbe biology at Cornell University. My goal is to finish my Ph.D. training in the spring of 2019, and, after completing a post-doc at a major plant research institute, begin leading my own research group at a public university. The AFRI pre-doctoral fellowship will allow me to pursue the topic that I am passionate about, **translating knowledge of resistance gene diversity into products with increased disease resistance**. It is becoming increasingly important for Ph.D. students to acquire a broad skill-set and I am particularly excited about the opportunity that this fellowship would provide for me to travel to The Sainsbury Institute in the UK in order to expand my network and build a solid computational biology foundation to manage large datasets.

A large component of my Ph.D. training has focused on becoming a better mentor. I have been fortunate to have had excellent mentors at all stages of my training, and it is important for me to provide mentorship whenever possible. It is undeniable that there are traits that make people naturally good mentors, however it is a skill that requires training and practice. I have had the opportunity to expand my mentorship skills by working with students in the Research Experience for Undergraduates (REU) and CienciAmerica programs in each of my summers as a graduate student. I had to develop an entirely different strategy when I was not mentoring a single student, but instead acting as a teaching assistant for a group of 30 introductory plant pathology students. Faculty positions at academic institutions are becoming increasingly competitive and in order to secure one of these highly desired positions **it is important that I not only develop scientific expertise, but also develop a professional network and expand my teaching, mentoring, and management skills.**

MENTORING PLAN

My primary mentor for the proposed research is Prof. Adam Bogdanove. Prof. Bogdanove has demonstrated a commitment to well-rounded graduate student training as evidenced by his former students being placed in diverse positions including academic post-docs in research laboratories, faculty teaching positions, and positions in industry. His appointment as the Director of Graduate Studies demonstrates a commitment to mentoring graduate students across the entire section of Plant Pathology and Plant-Microbe Biology. Paper discussions at weekly 'Boglab' meetings strengthen lab members' critical thinking skills in a supportive environment and ensure that we stay up-to-date on current literature in our field. Bi-annual lab research presentations allow the synthesis of several months of data into a format that is clear and places our research in the context of national and global plant protection and food security. Boglab members are expected to contribute to the scientific community by volunteering to peer review scientific literature, mentor junior scientists, and present at national and international conferences. My professional network has expanded greatly due to Prof. Bogdanove inviting me to meetings with visiting scientists and introducing me to colleagues at conferences.

PROJECT PLAN

Rationale and Significance

Bacteria from the genus *Xanthomonas* are responsible for devastating diseases in diverse crops including citrus canker, wheat bacterial leaf streak, and bacterial leaf streak and blight of rice[1]. In many cases, *Xanthomonas* virulence is increased due to the deployment of type III secreted transcription activator-like effectors (TALEs)[1-3]. Due to strict conservation of several domains, TALEs represent a large target for direct or indirect recognition by plant defense proteins. However, until recently, canonical resistance to TALEs has been limited to a single example, the *Bs4* gene from tomato[4]. Two recent reports of resistance to TALEs in an activation-domain independent manner provide an exciting potential source of broad-spectrum resistance to TALE-deploying bacteria. Due to the conserved nature of the downstream defense pathways in plants[5] transfer of a resistance proteins to distinct plant families is possible and has been demonstrated by the movement of functional *Rxo1* from maize to rice[6]. **The goal of this project is to leverage knowledge of TALE-dependent resistance to inform translational approaches and the creation of crops resistant to TALE-deploying pathogens.** A broad-spectrum resistance to TALEs would reduce crop losses and increase food security domestically and internationally.

Introduction and Preliminary Data

TALEs contribute to virulence by binding host DNA and up-regulating downstream susceptibility genes[2,3]. Due to their DNA binding and transcriptional regulation properties, resistance to TALE-deploying bacteria can be accomplished via non-canonical resistance mechanisms including mutation/loss of a TALE binding site or by activation of an executor resistance gene[2,3]. These resistance mechanisms are generally specific to a single TALE so do not provide broad-spectrum resistance and are outside the scope of this proposal.

Plants and animals share a common defense gene family, the NLRs, characterized by a conserved nucleotide-binding domain fused to a variable leucine-rich repeat (LRR) domain. In plants, NLRs are responsible for canonical resistance and can be divided into two subclasses based on the inclusion of the Toll-Interleukin-1 (TIR) or coiled-coil (CC) domain and are referred to as TIR-NLRs and CC-NLRs respectively[5]. NLRs must evolve quickly in order to respond to changing biotic stresses. The presence of many NLRs within a genome enables this rapid evolution via recombination between paralogs[7]. As a result of tandem duplication events, NLRs are often found in complex clusters within the genome[8-10]. Additionally, NLRs are enriched in presence-absence variation [11-13]. Taken together these features facilitate rapid evolution of NLRs but make this protein family particularly challenging to study with available sequencing technologies.

Xanthomonas oryzae is the most important bacterial pathogen of rice and pathovars *oryzae* (*Xoo*) and *oryzicola* (*Xoc*) cause bacterial blight and leaf streak respectively[14]. In the mid 1990s the first *Xanthomonas* resistane gene, *Xa1,* was cloned from rice and shown to encode a CC-NLR[15]. In 2002, the chromosome four sequence of rice cultivar Nipponbare was published and six *Xa1*-like homologs were identified. The authors speculated that diversity of the gene family may provide resistance to different races of *Xanthomonas oryzae*[16]. Since this publication, we have learned that *Xa1* is triggered by a variety of TALEs in an activation-domain independent manner[17]. The American heirloom rice cultivar Carolina Gold Select is resistant to some strains of *Xoc* and encodes a similar TALE-dependent resistance, *Xo1*, which maps to a 1.09 Mb region of chromosome four that overlaps the Xa1 locus[18]. These are important findings, however virtually nothing has been done to follow up on the now 15-year-old hypothesis that allelic diversity of the *Xa1*-like resistance gene family may provide

different resistance spectra. This has been, at least in part, due to the suppression of *Xa1* and *Xo1* resistance by truncated TALEs from *Xoo* and *Xoc*, a phenomenon recently described by Ji et al. and Read et al.[17,19]. These advances, taken together, provide the foundation and toolkit for probing *Xa1*-like resistance allowing researchers to tackle long-standing questions in these pathosystems.

TALE-Dependent Plant Resistance

Xa1, the first resistance to *Xanthomonas* (*Xa*) gene was identified in 1967 and shown to provide resistance to Japanese Race I of *Xoo*[20]. *Xa1* resistance is conferred by a CC-NLR on the long arm of chromosome four[15] and contains a characteristic zinc finger BED domain which has been speculated to act as an integrated decoy[21]. The reference genome for the *xa1* cultivar Nipponbare shows six homologs at this location, indicating expansion of the family[16]. The first resistance to *Xoc, Xo1*, was recently reported and, interestingly, maps to a region that overlaps the *Xa1* genomic location[18]. In addition to both being encoded on similar regions of chromosome 4, both Xa1 and Xo1 recognize TALEs in an activation-domain independent manner[17,18], a phenomenon previously only observed for the tomato TIR-NLR Bs4[4]. Interestingly, several additional uncharacterized *Xa* genes map to a similar region of chromosome four (*Xa2*, *Xa12*, *Xa14*, *Xa17*, *Xa31(t)*, and *Xa38*), raising the possibility that these genes may be part of the *Xa1*-gene family. The avirulence determinant(s) for these genes have yet to be determined, however similarities to *Xa1* activity indicate a possibility that they will also respond to TALEs.

There is evidence in the literature of diversity at the *Xa1*-locus. Allelic diversity of other rice NLRs has been shown to result in different resistance spectra as demonstrated by the rice blast resistance gene *Pi-ta*[22]. *Xa17* (previously *Xa1*-as(t)) was initially reported as an allele of *Xa1* from the rice cultivar Asominori[23]. This cultivar shows a nearly identical resistance profile to *Xa1*, however, while *Xa1* resistance has broken down, the resistance provided by *Xa17* has not[24]. The *Xa*38 resistance has been mapped to a 38kb interval that overlaps *Xa1* and provides resistance to many strains of *Xoo* in India[25,26]. Mining sequence data reveals the presence of an *Xa1*-like gene in the rice cultivar Jamaica, this gene was sequenced and can be distinguished from *Xa1* by 14 amino acid differences, 12 of which occur in the LRR[27]. My preliminary results indicate that an *Xa1*-like candidate from the *Xo1* locus is nearly identical to the Jamaica allele, and that both display differences in resistance phenotypes when compared with *Xa1*. **Aim 1 formally characterizes the TALE recognition spectra of a panel of resistant rice cultivars.**

Truncated TALEs Can Suppress Resistance

The delay in identifying the avirulence determinant for *Xa1* can at least partially be explained by the recent demonstration that most Asian *Xoo* and *Xoc* strains encode truncTALEs that are sufficient to suppress *Xa1* and *Xo1* resistance. The *Xa1* and *Xo1* resistance is only able to manifest when the truncTALEs of a strain are artificially deleted[17] or against strains with non-functional truncTALEs[17,19]. Based on our results, it appears that truncTALEs cannot bind DNA, leading us to believe that they may suppress resistance via protein-protein interactions[19]. My analysis of available sequence data shows that truncTALEs are unique to *Xoo* and *Xoc* lineages.

There is evidence of differential resistance spectra within the chromosome 4 *Xa* genes. Particularly promising is the observation that *Xa38* provides resistance to most Indian pathovars of *Xoo*, while Xa1 does not[25,26]. Our data indicate the presence of truncTALEs in Indian *Xoo* suggesting that they are not sufficient to suppress Xa38. Similar observations

have been made for the *Xa31*(t) encoding cultivar Zhachanglong from China[28], which almost certainly encounters *Xoo* and *Xoc* with functional truncTALEs. Identification of an *Xa1*-like gene that is immune to truncTALEs would have an immediate impact in rice producing areas where *Xoo* and *Xoc* are endemic. An immune allele would be a top candidate for transfer to other crop systems to protect against the possibility of other pathogens acquiring or evolving a functional truncTALE. **Aim 2 tests Tal2h suppression of resistance in a panel of rice cultivars.**

Sequence diversity of TALE-dependent R-genes

A novel method for analyzing sequence of NLRs, R-gene enrichment sequencing (RenSeq), was developed in the lab of Dr. Jonathan Jones at The Sainsbury Institute[29]. This method leverages the conserved sequence features found in all NLRs by shearing genomic DNA and utilizing bead-bound probes to pull down fragments that contain NLR sequence. The original iteration of RenSeq utilized short-read Illumina technology and was unable to resolve similar NLR gene family members. The most recent version of RenSeq combines the sequence capture step with long-read PacBio sequencing to generate reads up to 7kb. Reads of this length allow the assembly of entire NLR clusters and include important intragenic and regulatory regions[30,31].

The *Xa1* locus in the *xa1* reference sequence for Nipponbare contains a cluster of six *Xa1*-like genes[16], however orthologs have been identified in other genomic regions. *Xa1*-like genes belong to plant model organism orthologous group APK_OrthoMCL716[32]. In addition to the six orthogroup members on chromosome four, Nipponbare also encodes orthologs on chromosomes two and eleven. The presence of orthologs on other chromosomes adds a layer of complexity to the expansion of the gene family, indicating that more than tandem duplication is responsible for the expansion. The complex genomic landscape and diversity of the *Xa1*-like family limit the usefulness of the reference genome and present a challenge to traditional amplification based sequencing techniques. **Aim 3 utilizes the most recent version of RenSeq to determine the diversity of the *Xa1*-like family from diverse rice cultivars.**

Approach

Aim 1:

H01: Rice TALE-dependent resistance genes have different recognition spectra.

Rationale: NLRs are under selective pressure to evolve rapidly in response to biotic stress. Because many extant *Xoo* and *Xoc* strains encode truncTALEs, it is possible that many rice cultivars encode functional TALE-dependent R-genes with different recognition spectra.

Approach: Triplett et al. used a panel of TALEs delivered by the TALE-free Xo strain X11-5A to determine the resistance profile of *Xo1*[18]. This panel will be expanded to include TALEs from diverse *Xanthomonas* species, with and without the activation domain (see Table 1). Inclusion of TALEs from outside of the *Xoo*/*Xoc* lineage provides the opportunity to discover an *Xa1*-like resistance that could provide protection for diverse crops. Leaf clip inoculation will be carried out on a rice cultivar panel selected based on: known resistance to *Xoo*/*Xoc*, sequence data indicating a close homolog of *Xa1*, or to represent rice diversity (see Table 2). Expected Outcomes: I expect to see differential lesion lengths for rice cultivars challenged by the TALE panel indicating differences in recognition spectra. Resistance to TALEs without the activation domain is evidence for an *Xa1*-like recognition, while any observed resistance that is dependent on the activation domain is more likely to be specific and due to activation of an executor R-gene[33]. Activation domain dependent resistance will be noted and pursued by other lab members.

			Reaction on CarGold	
TALE	**source**	**host plant**		**ΔAD**
Pth*Xo1*	*X. oryzae pv. oryzae*	rice	R	R
Tal1c	*X. oryzae pv. oryzicola*	rice	R	nt
Tal2g	*X. oryzae pv. oryzicola*	rice	R/S	nt
Tale1	*X. axonipodis pv. manihotis*	cassava	S	nt
AvrHah1	*X. gardneri*	pepper	S	nt
AvrBs3	*X. vesicatoria*	tomato/pepper	nt	nt
AvrXg1	*X. axonipodis pv. glycines*	soy	nt	nt
PthA4	*X. citri pv. citri*	citrus	nt	nt
Tal2	*X. translucens pv. undulosa*	wheat	nt	nt

Table1: *TALE genes to express in TALE-free X11-5A. Modified from [18]. ΔAD – activation domain deletion variant, R- resistant, S-susceptible, nt – not tested*

Cultivar	**Justification**	**Ref**
Asominori	Contains *Xa17* (formerly *Xa1*-as(t)) - distinct resistance spectra	23,24
Carolina Gold Select	*Xo1*-locus provides TALE-dependent resistance	18
Jamaica	Encodes an *Xa1*-like gene and resistance to Japanese Race I	27
Java14	In addition to *Xa1*, reported to encode *Xa1*2 at *Xa1*-region of chr4	34
Kasalath	Resistant to Japanese Race 1 -sequence data shows an *Xa1* candidate with an expanded LRR region	27,35
Kogyoku	Source of *Xa1* in NIL IRBB1	20
***O. nivara* IRGC 81825**	Source of *Xa*38 from the *Xa1*-region of chr4 - distinct resistance spectra	25,26
Taichung Native 1 (TN1)	Source of *Xa1*4 in the *Xa1*-region of chr4 - distinct resistance spectra	36
Tetep	Encodes *Xa*2 which maps to the *Xa1*-region of chr4 and provides resistance to JXO Race 2	37
Zhachanglong	*Xa*31(t) - maps to *Xa1* region - distinct resistance profile	28

Table 2: *Rice panel for use in Aims 1-3. Cultivars in bold will be prioritized for RenSeq based on resistance characteristics*

Aim 2:

H02: truncTALE, Tal2h, is not sufficient to suppress all TALE-dependent resistance. Rationale: The observation that truncTALEs from *Xoo* and *Xoc* are able to suppress *Xa1* and *Xo1* resistance respectively[17,19] demonstrates a bacterial counter-defense mechanism and may limit the utility of deployed TALE-dependent resistance genes. However, the ability of truncTALEs to suppress resistance has not been characterized for other resistance genes mapped to chromosome 4 and it is possible that some *Xa1*-like genes cannot be suppressed by truncTALEs. Approach: Our group has demonstrated that Tal2h works to suppress resistance when delivered in trans via co-inoculation of a bacterial mixture[19]. I will utilize this technique to co-infiltrate the TALE-free strain X11-5A expressing Tal2h along with X11-5A expressing individual TALE panel proteins from Table 1. Syringe infiltration experiments will provide qualitative resistance suppression data, while leaf clipping and lesion measurement will be employed to measure quantitative suppression of resistance. Expected Outcome: I expect to see differential responses to the infiltration and leaf-clipping treatments for the chromosome 4 *Xa* genes indicating partial or full immunity to Tal2h-based suppression. Potential Pitfall: It is possible that a TALE will not be an avirulence determinant for some of the resistance genes that map to the *Xa1*-locus. In these cases, in leiu of co-inoculation, *tal2h* will be transformed to an avirulent *Xoo* strain matching each *Xa* gene and screened for suppression of resistance in the manner described. This will provide valuable information demonstrating the specificity of Tal2h based defense suppression.

Aim 3:

H03: The *Xa*-1 resistance gene family shows signs of rapid evolution. Rationale: NLRs are rapidly evolving and often present in gene families that may be present in clusters or

spread throughout the genome. Utilizing RenSeq to pull down and sequence the NLRs from representative rice cultivars with known *Xanthomonas* resistance phenotypes will allow a comprehensive study of the diversity of the *Xa1*-like gene family. The diversity data can be combined with resistance and suppression data generated in Aim 1 and 2 to provide insights into *Xa1*-like function. Approach: A rice RenSeq probe-set is currently being constructed by another group in collaboration with The Sainsbury Lab (Jones personal communication). I will travel to the lab of Dr. Jonathan Jones and train on sample preparation and computational methods required to generate and analyze RenSeq results (see Documentation of Collaboration). Upon returning to Cornell, I will submit samples from the six rice cultivars noted in Table 2 for RenSeq and determine the diversity if the *Xa1*-like family. Observations for the *Xa1* family will be compared with the extensive rice phylogenetic resources to find evidence of rapid evolution. If time and resources allow, additional rice cultivars will be selected for RenSeq. Results will be stored at NCBI and will be accessible to the international rice research community.

EVALUATION PLAN

Career Development Plan

Mentorship: I will mentor one undergraduate researcher over the two summers covered by the fellowship.
Teaching: A Get Set Workshop Certificate of Participation will be earned in year one by attending three workshops offered by the Cornell Center for Teaching Excellence.
Networking: The proposed training at The Sainsbury Institute will provide an opportunity to expand my connections with the international plant-microbe community. Presenting research at ICPP and XGC conferences will allow exposure to the broader research community.
Publishing Research: In addition to presenting at conferences, the research conducted as part of this fellowship will be reported in a one to two papers in appropriate journals.
Career Advancement: At the conclusion of the fellowship I will transition to a postdoctoral position at a facility with a strong plant-microbe interaction focus.

Mentoring Plan

I will continue to meet annually with my Ph.D. committee consisting of my advisor, Prof. Adam Bogdanove, as well as Prof. Alan Collmer and Prof. Adrienne Roeder. By drafting SMART goals[38] I will be able to quantitatively measure my growth as a researcher at regular one-on-one meetings with Prof. Bogdanove.

Project Plan and Timeline

Assembly of rice and TALE panels – in progress complete by **Y1 Q2**
Aim 1 – resistance spectra assay – **Y1 Q2 - Y2 Q1**
RenSeq training at The Sainsbury Laboratory – **Y1 Q3**
Aim 2 – suppression spectra assay – **Y1 Q4 - Y2 Q2**
Aim 3 – diversity of *Xa1*-family – RenSeq – **Y1 Q4**
Aim 3 – data analysis – **Y2 Q2 - Y2 Q4**

Dissemination Plan: Results of this research will be presented at the International Congress of Plant Pathology and the *Xanthomonas* Genomics Conference. Oral presentations will be made at Cornell symposia and graduate student research seminars and published in peer-reviewed journals. RenSeq data will be stored at NCBI and made available to the public for analysis.

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