UNIVERSITY LOGO

September 16th, 2015

Meriadoc Brandybuck, PhD
Search Committee Chair
Department of Physiology and Biophysics, School of Medicine
R1 University A, USA, 00000-0000

Dear Dr. Brandybuck,

I am writing to express my interest in a tenured faculty position at the level of assistant professor in the Department of Physiology and Biophysics. Currently, I am completing my postdoctoral research training in the laboratory of Samwise Gamgee at the R1 University B (RUB).

My research interests are largely direct towards understanding how physiological systems interact during human disease. I am particularly interested in learning how the immune system interacts with the system a during disease d and how this interplay influences the development and progression of disease. As a predoctoral student in the laboratory of Farmer Maggot at the R1 University C (RUC), I was trained in the fundamental methodologies of tissue type a research that aided me in investigating the role of cell type ds in tissue type a injury and repair.

During the initial years of my postdoctoral training, I investigated the pathogenic interactions of the immune system with the tissue d and its vascular network in the mouse model of disease A. Through this work I established a firm conceptual and technical framework in immunology that I used to develop an independent line of investigation focused on defining the role of Cell type F and other lymphoid cell regulators in controlling tissue type a inflammation. My research has led to important discoveries that have advanced our understanding of immune regulation during non-autoimmune diseases such as disease d. My work has shown that Cell type F are elevated in both human and mouse disease d tissue type a and display an activated phenotype, suggesting a tissue type a antigen-driven activation of Cell type F. Importantly, depleting Cell type F in disease d mice exacerbated tissue type a inflammation and Cytokine 1 production in tissue type a effector Cell type i, leading to increased cell type e injury. The therapeutic implications of this work were illustrated by the reduction of tissue type a inflammation and cell type e injury when disease d mice are treated with recombinant PQR: anti-PQR antibody immune complexes that increase Cell type F and Cell type G.

My diverse training in tissue type a physiology and immunology has placed me in a unique position to run an independent research program aimed at unravelling the cellular and molecular basis of tissue type a immunity and immune regulation during disease d. Moreover, the findings of my research may open new therapeutic avenues that lead to the development of novel treatments for disease d. In my lab I, will continue to study the role of Cell type F during disease d, with an emphasis in defining the mechanism/s of Cell type F-mediated suppression of tissue type a inflammation. In addition to their

role in controlling tissue type a immunity, my lab will examine the capacity of Cell type F to directly promote tissue type a regeneration through their production of specialized mediators of tissue repair. A second arm of research will be focused on defining the role of Cell type G in disease d. Through unpublished work I have found that Cell type G are activated in disease d tissue type a, and I have generated novel genetic tools to ablate this population in vivo, allowing me to examine their capacity to modulate the severity of disease d. By coupling studies involving preclinical animal models with the analysis of human tissue type a biopsies my laboratory will be well positioned to examine the therapeutic potential of targeting immune system-derived factors that modulate tissue type a disease.

In addition to my research, I have had the rewarding experience of teaching and mentoring high school and undergraduate students, and have participated in programs that aim to increase the participation of underrepresented minorities in the sciences. My participation in such programs has provided me with the opportunity to speak at various colleges and high schools throughout the Location Here about career paths in biomedical research. I invite you to please read my teaching statement that will provide you with more details of my mentoring and teaching experiences.

Please find enclosed my curriculum vitae and a list of references. In addition, you will find my research and teaching statement that provide further details of the summary above. I am looking forward to hearing from you, and I thank you in advance for your consideration.

Sincerely,

Bilbo Baggins, Ph.D.
Postdoctoral Fellow,
Laboratory of Samwise Gamgee
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Bilbo Baggins

R1 University B. Disease A Center
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Education

2010 **Doctor of Philosophy**
Molecular, Cellular and Integrative Physiology
R1 University C
Laboratory of Dr. Farmer Maggot

2003 **Bachelor of Science**
Biochemistry and Molecular Biology
R1 University D
Laboratory of Dr. Grima Wormtongue

1999 **Associate in Arts**
Liberal Arts
Community College 1

Research and Training Experience

2010-present **Postdoctoral Fellow**
R1 University B
Disease A Center
Laboratory of Dr. Samwise Gamgee

Project: My work focuses on addressing how explanation here

2004-2010 **Graduate Student Researcher**
R1 University C
Science Department A
Laboratory of Dr. Farmer Maggot

Thesis project: My dissertation work examined the effect of explanation here.

2003-2004 **Staff Research Associate**
R1 University B
Cardiovascular Research Institute
Laboratory of Dr. Peregrin Took

I participated in investigations that explored the role of explanation here.

Summer 2002 **Summer Undergraduate Research Fellowship Program**
Isildur Medical School
Department of Microbiology and Immunology
Laboratory of Dr. Fredegar Bolger

Summer project: I was involved in a study that examined the selective delivery of explanation here.

		I was responsible for explanation here.
	2002-2003	**Very Competitive Research (VCR) Fellow** R1 University D Molecular, Cell and Developmental Biology Laboratory of Dr. Grima Wormtongue The goal of my project was to explanation here.
	Summer 2002	**Another Competitive Research Program** R1 University D Summer Internship Weekly rotations in various disciplines of biomedical research.
Funding	2014-Present	**Gandolf's Very Excellent Research Fellowship (GVER)** Postdoctoral Fellowship
	2011-2014	**American Disease A Association** Boromir Postdoctoral Fellowship Award
	2010-2011	**Gandolf's Very Excellent Research Fellowship (GVER)** Predoctoral Fellowship
	2007-2010	**Gandolf's Very Excellent Research Fellowship (GVER)** Predoctoral Fellowship
Honors and Awards	July 2015	Young Investigator Award: Conference A Winner
	March 2009	Best poster presentation award at the annual Science Department A retreat
	March 2008	Best poster presentation award at the annual Science Department A retreat
	2002-2003	Very Competitive Research (VCR) undergraduate Fellowship
	Spring 2002	Academic Excellence Honors Program letter of recognition
	Fall 2001	Academic Excellence Honors Program letter of recognition

Publications

1. **Baggins B,** name, name, name, name, Maggot F, name, name, Gamgee S. *Title here.* Science Translational Medicine. In press.
2. Name, name, name, name, **Baggins B**, name, name, name. *Title here*. Science Translational Medicine. In review.
3. **Baggins B**, name, name, name, name, name, name, name, name, name, name, Gamgee S. *Title here.* Disease A. 2014.

4. name, name, **Baggins B**, name, Maggot F. Title here. J Immunol. 2013.

5. **Baggins B**, name, name, name, Maggot F. Title here. J Immunol. 2012.

6. **Baggins B**, name, name, name, name, Maggot F. Title here. Hum Mol Genet. 2011.

7. Maggot F, **Baggins B**. Title here. Am J Physiol Regul Integr Comp Physiol. 2011.

8. Maggot F, **Baggins B**. Title here. Nature Medicine. 2010.

9. **Baggins B**, name, name, name, Maggot F. Title here. Hum Mol Genet. 2010.

10. name, name, name, **Baggins B**, and Took P. Title here. J Immunol. 2009.

11. name, name, name, **Baggins B**, Maggot F. Title here. Hum Mol Genet. 2009.

12. name, **Baggins B**, name, name, name, name, name, name, name, name, name, name, name, name, name, Maggot F, name, name. Title here. J Clin Invest. 2008.

13. Took P, name, name, **Baggins B**, name, name, name. Title here. Clin Exp Allergy. 2006.

14. name, name, **Baggins B**, name, Took P. Title here. J Clin Invest. 2005.

Scientific Presentations

1. **Baggins B,** name, name, name, name, Maggot F, name, name, Gamgee S. *Title here. Conference A*. Location USA, 2015

2. **Baggins B**, name, name, name, name, name, name, name, name, name, name, Gamgee S. *Title here.* Conference B. Location USA. May 2014.

3. **Baggins B**, name, name, name, name, name, name, name, name, name, name, Gamgee S. *Title here.* R1 University, Disease A Center retreat. Location USA. May 2013.

4. **Baggins B**, name, name, name, name, name, name, name, name, name, name, Gamgee S. *Title here. R1 University E. May 2012*

5. **Baggins B**, name, name, name, name, name, name, Gamgee S. *Title here.* Conference B. Location USA. May 2012.

6. **Baggins B**, name, name, name, name, name, name, Gamgee S. *Title here.* Conference C. Location USA. April 2012.

7. **Baggins B**, name, name, name, name, Gamgee S. *Title here.* Conferences D and E. September 2011.

8. **Baggins B**, name, Maggot F. *Title here.* Science Department A retreat. Location USA. March, 2009. Best poster presentation award.

9. **Baggins B** and Maggot F. *Title here.* Science Department A student seminar. Location USA. June, 2008.

10. **Baggins B** and Maggot F. *Title here.* Conference F. Location USA. April, 2008.

11. **Baggins B** and Maggot F. *Title here.* Science Department A program retreat. Location USA. March, 2008. Best poster presentation award.

12. **Baggins B,** name, name, Maggot F. *Title here.* Science Department A student seminar. Location USA. June, 2007.

13. **Baggins B,** name, name, Maggot F. *Title here.* Invited speaker by the Very Competitive Research Careers program. R1 University D. June 2007.

14. **Baggins B** and Maggot F. *Title here.* Science Department A program retreat. Location USA. March, 2007.

15. **Baggins B** and Maggot F. *Title here.* Science Department A program retreat. Location USA. March, 2006.

16. **Baggins B** and Grima Wormtongue. *Title here.* Very Competitive Research Career student seminar. R1 University D. May, 2003.

Teaching and Mentoring Experience	Summer 2015	High School Summer Intern Mentored Gollum Summer High School Intern Program
	2013-2015	Undergraduate Student Researcher Mentored Gimli

	2011-2013	Undergraduate Student Researcher Mentored Galadriel
	Summer 2010	High School Summer Intern Mentored Faramir Summer High School Intern Program
	2007-2008	Undergraduate Student Researcher Mentored Gamling
	2006-2007	Undergraduate Student Researcher Mentored Elrond
	Winter 2007	Teaching Assistantship at RUC Course name: Name here
	Fall 2005	Teaching Assistantship at RUC Course name: Name here
Career Development	Pending: November 2015	**Seminar: Faculty Job Offer Negotiation** Hosted by the Career Services Office
	August 2015	**Seminar: Faculty application materials creation** Hosted by the Career Services Office
	September 2015	**Panel: Faculty perspectives** Hosted by the Career Services Office Multiple RUB faculty members were brought together to share their experience in applying for faculty positions or hiring other faculty members
	April 2015	**Applying for NIH Grants** Hosted by the Career Services Office
	June 2008	**RUB Postdoc Preparation Colloquium**
	September 2007	**Institute on Postdoc preparation**
	June 2005	**Fellows Program A. Biotechnology Institute**
	March 2003	**Seminar A**
	September 2003	**The Society for A and B Americans in Science**
	September 2003	**The Society for A and B Americans in Science**
Extracurricular Activity	March 2008- June 2010	**Activity 1** Explanation here
	March 2006-June 2008	**Council Representative** The Council provides representation and promotes the interests of *all graduate students within the biological sciences at RUC.*

References

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Research Statement: Immune-mediated regulation of disease B.

Disease CD (DCD) is a lethal childhood disease, which is caused by mutations of the legolas gene[1]. Legolas deficiency renders the cell type a membrane susceptible to contraction-induced injury[2], leads to loss of ambulation by adolescence and death by the 2nd-3rd decade of life. Currently, no cure exists for DCD and therapies are limited to corticosteroids that broadly suppress the immune response to injured tissue type a, implicating chronic inflammation as an important determinant of disease severity. Perturbations of the immune system in abc mice, a mouse model of DCD, have shown that the immune system contributes to the pathogenesis of DCD by exacerbating tissue type adamage[3]. However, the inflammatory response to injury is also critical in mediating tissue type a regeneration[4]. This dichotomous role for the immune system can be partly explained by distinct subsets of immune cells that either exacerbate tissue injury or promote repair. For instance, Cell type b (CTB) that are induced by pro-inflammatory cytokines such as Cytokine 1 and Cytokine 2 promote cell type e injury[5]. In contrast, other type cytokines such CYTOKINE 3, CYT 4 and CYT 5 induce Cell type c (CTC), which antagonize the action of Cell type b via an arginase-dependent mechanism[5]. Recent studies indicate that Cell type F and Cell type G (CTG) both have the capacity to modulate the activation status of Cell type d[6, 7]. Moreover, we found that modulation of these lymphoid populations in abc mice regulates the severity of disease B. However, whether these populations directly regulate tissue type a injury and repair, or indirectly through the modulation of tissue type a cell type d activation during disease d remains to be addressed.

Research strategy: During the first 3-5 years of operation, my lab we will focus on investigating the functional role of Cell type F and Cell type G during disease d using a combined expertise in immunology and tissue type a physiology. We will use mouse genetics, histological assays, cellular and molecular techniques, gene expression profiling methods, single cell analysis using various flow cytometry platforms, and tissue type a performance tests to study Cell type F and Cell type G in tissue type a disease. We will modulate Cell type F and CTG numbers or functionality at various stages of disease using mouse genetics and pharmacological approaches to test the hypothesis that Cell type F and CTG function cooperatively to suppress the pro-inflammatory response to injured tissue type a, decrease cell type e injury and promote tissue type a regeneration. To examine the translational implications of our basic research we will examine human tissue type a biopsies using histological and biochemical assays to examine the activation and functional state of tissue type a Cell type F and Cell type G in DCD and healthy patients. In later years my lab will begin to address whether the preliminary observations regarding Cell type F and CTG function in DCD are specific to this disease or are a generalized inflammatory response to injured tissue type a that extend to other tissue type a diseases. This would allow us to assess whether the therapeutic implications of our work are applicable to a broader class of tissue type a disease. We will define the direct and indirect mechanisms by which Cell type F and Cell type G regulate tissue type a injury and repair during tissue type a diseases by addressing the following specific aims:

Figure 1 here

Fig. 1. The depletion of Cell type F exacerbates tissue type a inflammation and injury in abc mice.

Representative images of hematoxylin-stained tissue b sections of control - (**A**) and DEPLETION-treated (**B**) abc mice. DEPLETION was used to deplete Cell type F for a 3 week period. (**C**) Tissue type a injury expressed as the percent albumin^{+} tissue type a area over total tissue type a area. (**D**) Quantification of the frequency of tissue type a Cell type h Cytokine 1^{+} cells following depletion of Cell type F in abc^{deleter} mice. (**E**) The expression of effector molecules associated with a Other other type response was measured by RT-qPCR. The levels of statistical significance are defined as: ♦= p< 0.01; *= p< 0.05. White bars= control abc mice; black bars= Cell type F-depleted abc mice.

Aim 1. To test the hypothesis that Cell type F-derived CYT 5 suppresses tissue type a inflammation.

The discovery that Cell type F suppress tissue type a inflammation and injury, and the observation that they also promote tissue type a repair[8], suggests that they play a critical role in the pathogenesis of disease d. We used a Cell type F-depleting antibody (DEPLETION) or diphtheria toxin (DT)-mediated depletion of Cell type F in abc^{DELETER} mice to show that Cell type F ablation exacerbated tissue type a inflammation (Fig. 1A-B), injury (Fig. 1C) and the expression of inflammatory mediators (Fig. 1D-E) in disease d mice. A key focus in my research will be to examine the communication between the immune and system a and understand how Cell type F act as liaison between both systems. My future research will be directed at unraveling the molecular and cellular mechanisms of Cell type F-mediated regulation of tissue type a inflammation and injury, and examining their capacity to promote tissue type a regeneration during disease d. These studies promise to advance our understanding of immune regulation in tissue type a diseases. We will examine the capacity of Cell type F to delay the development and progression of disease d by reducing tissue type a injury through an CYT 5 mediated suppression of tissue type a inflammation.

The indirect pathway: enhancement of tissue type a repair through the modulation of the inflammatory response to injured tissue type a. It has become increasingly evident that an imbalance of inflammatory CTB and Cell type c, which promote damage and regeneration, respectively, contributes to the immune-mediated exacerbation of disease B[9]. However, the mechanisms that regulate transitions between CTB and CTC activation of Cell type d during disease d remain ill-defined. Our previous work has revealed that CYT 5 is a key regulatory cytokine that controls cell type d activation during disease d. We showed that total ablation of CYT 5 in abc mice caused an increase in cell type e injury and decrements in tissue type a function that was attributed to enhanced cytotoxic activity of Cell type b[10]. Moreover, CYT 5 treatment of tissue type a Cell type d reduced protein a and increased protein a expression, markers of CTB and Cell type c, respectively. Collectively, these data suggest that CYT 5 controls the CTB to CTC transition of cell type d activation to reduce immune cell-mediated damage of disease d tissue type a. Although CYT 5 clearly plays an important role in suppressing immune-mediated damage the important cellular sources are not as of yet defined. Based on the observation that tissue type a Cell type F are a rich sources of CYT 5[8], we hypothesized that Cell type F modulate cell type d activation. Indeed, we found that depletion of Cell type F during the acute stage of disease increased the activation of Cell type b and cell type e injury, and that pharmacological methods to increase tissue type a Cell type F increased CYT 5 expression in abc tissue type a. To test the hypothesis that Cell type F are a critical source of CYT 5 that suppress tissue type a inflammation by promoting CTB to CTC transitions we are crossing mice containing a floxed CYT 5 allele (CYT $5^{fl/fl}$)[11] with an abc mouse expressing a GFP-Cre fusion protein that is transcriptionally regulated by the Protein c promoter, allowing the specific deletion of CYT 5 in Cell type F. A comparative genetic analysis using complementary abc-Cre systems, including the Protein D-Cre, Protein e-Cre and total CYT 5 null mutant abc mice and bone marrow chimeras, will allow us to simultaneously examine the role of CYT 5 in other immune and non-immune cell compartments. We will use flow cytometry and histological methods to assess whether Cell type F interact functionally with tissue type a Cell type d in an CYT 5-dependent manner by examining phenotypic markers of CTB and CTC activation and the localization of Cell type F and cell type ds in tissue type a of Cell type F-specific, CYT 5-deficient abc mice and controls. These mouse models will provide indispensable tools to examine the CYT 5-expressing immune cell types that promote the resolution of tissue type a inflammation.

Aim 2: To test the hypothesis that Cell type F produce factors that modulate tissue type a regeneration.

In this aim, we will examine the contribution of Cell type F to tissue type a regeneration by testing the hypothesis that Cell type F express molecules that directly act on cell type as to enhance cell type a proliferation and/or differentiation.

Figure 2 here

Fig. 2. Protein ef reduces the severity of muscular dystrophy. RT-qPCR measurements of protein ef expression in whole tissue type a (**A**) and fractionated cellular compartments of abc tissue type aaccording to their expression of MNO, Nop, PROTEIN D and Protein c (GFP) (**B**). The expression of protein ef receptor, Receptor a, in whole tissue type a was measured by RT-qPCR (**C**). Histological analysis of tissue type a sections revealed that Receptor a is expressed by regenerating cell type k in abc mice (**E**) but absent in WT tissue type a(**D**). H&E-stained tissue b sections from abc mice treated with PBS (**F**) or α-protein ef antibody (**G**).

The direct pathway: a direct line of communication between Cell type F and regenerating cell type e's that promotes tissue type a regeneration. Through gene profiling studies we found that Cell type F express candidate molecules that may directly regulate tissue type regeneration. For example, a novel protein f-like family member, protein ef, was highly expressed in Cell type F compared to effector or naive Cell type i in wild type mice. In addition to binding to distinct receptors, protein g and protein ef also bind a common receptor (RECEPTOR A[12]), which is expressed in tissue type a and cell type js, and treatment of injured tissue type a with protein g enhanced tissue c type arepair[12, 13, 14]. However, whether protein ef has a similar function and whether Cell type F can promote cell type a proliferation or differentiation via protein ef is not known. In preliminary work, we found that protein ef expression is increased in abc tissue type a (Fig. 2A) and its expression in the tissue type a is predominantly confined to tissue type a Cell type F (Fig. 2B). Moreover, we have found that the protein ef receptor, Receptor a, is upregulated in disease d tissue type a (Fig. 2C). Interestingly, Receptor a is expressed by regenerating fibers in abc mice (Fig. 2E), but absent in WT tissue type a (Fig. 2D), suggesting a direct communication between regenerating tissue type a and Cell type F via the protein ef/Receptor a

signaling axis. We tested the hypothesis that protein ef suppresses the severity of disease B by treating abc mice with an anti-protein ef antibody and found that tissue type a inflammation and injury was exacerbated in abc mice treated with α-protein ef (Fig. 2G) relative to control-treated abc mice (Fig. 2F).

We will use genetic and adoptive transfer methods to investigate the mechanism of protein ef-mediated regulation of disease B. We will cross protein ef-deficient mice (protein $ef^{-/-}$)[15] with abc mice and perform a histological analysis to examine the effect of protein ef ablation on tissue type a inflammation, injury and regeneration. We will also generate bone marrow chimeras in which sublethally irradiated abc mice or abc.protein $ef^{-/-}$ will be adoptively transferred with WT or protein $ef^{-/-}$bone marrow, allowing us to determine whether the critical source of protein ef is confined to the hematopoietic compartment or extends to the stromal compartment as well. Similar experiment will be conducted with WT or protein $ef^{-/-}$Cell type F that will be adoptively transferred in to abc.$OPQ^{-/-}$ (abc mice lacking Cell type i) and abc controls to directly examine the role of Cell type F-derived protein $ef^{-/-}$ in disease d. Using these model systems histological, cellular and biochemical assays will be used to interrogate the relationship between protein ef and tissue type a inflammation and regeneration. Moreover, we will perform functional tests to examine the effect of protein ef deficiency on tissue c type a performance and strength. In vitro coculture system consisting of wild-type or protein ef-deficient Cell type F and cell type js will also be used to examine whether Cell type F directly regulate cell type j proliferation and differentiation . We anticipate that Cell type F will enhance tissue type a regeneration through their secretion of protein ef (and possibly other putative factor that we are examining) and subsequent activation of protein g receptors on tissue type a fibers.

Aim 3: to test the hypothesis that Cell type G regulate tissue type a regeneration.

We found that the treatment of abc mice with recombinant PQR/anti-PQR antibody immune complexes (PQRc) increased the number of tissue type aCell type G (Fig. 3A). Moreover, PQRc treatment reduced the prevalence of injured fibers (Fig. 3B) suggesting that Cell type B reduce cell type e injury or are capable or promoting cell type e repair and tissue type a regeneration. To examine the functional importance of Cell type G in disease d we generated genetic tools that allow us to ablate CTG in abc mice (abc.CYT 6-DTA mice) by using the CYT 6 promoter to drive the expression of the Cre enzyme. Cre subsequently drives the excision of stop codon inserted in a diphtheria toxin transgene; thus, liberating the expression of DTA and ablation of the DTA-expressing cells (Fig. 3C). We will interrogate these mice using the assays described above to test they hypothesis that Cell type G promote tissue type a regeneration during disease d. Specifically, we will examine the effect of CTG deletion on tissue type a inflammation, injury and regeneration, and perform global gene expression analysis on purified tissue type a Cell type G using next-generation sequencing to reveal mechanistic insights on how these cells contribute to tissue type a regeneration.

Figure 3 here

Fig. 3. Increases in CTG are associated with reduced cell type e injury. (**A**) PQRc treatment increases the number of Cell type G in abc tissue type a. (**B**) PQRc reduces cell type e injury in abc tissue b (**C**) DTA expression in abc.CYT 6-DTA mice reduces Cell type G by 90%. The levels of statistical significance are defined as: *= p< 0.05.

Impact: Understanding the functional outcome of immune cell and disease d tissue type a interactions that promote injury may lead to the development of novel treatments that prevent or delay disease progression. Corticosteroids delay the deterioration of tissue type a associated with chronic inflammation, but may also silence protective mechanisms that promote tissue type a repair. It is, therefore, imperative that the mechanisms that promote cytotoxic inflammatory responses are delineated from those that promote repair. Our preliminary studies are beginning to reveal that the contribution of regulatory lymphoid populations to the immune-mediated modulation of disease d is likely multi-faceted, involving direct and indirect cooperative efforts between Cell type F, CTG and CTC cell type ds in suppressing tissue type a inflammation and promoting regeneration. As shown in our preliminary findings we have demonstrated that Cell type F depletion in abc mice exacerbated tissue type a inflammation and cell type e degeneration and increases in Cell type G reduced cell type e injury. Moreover, gene profiling experiments showed that Cell type F expressed candidate molecules that have been implicated in tissue type a repair, including but not limited to protein ef and CYT 5. The results of our investigation will provide a greater understanding of the physiological role of Cell type F and Cell type G during tissue type a injury and repair, and mechanistic details on how immune tolerance is regulated in tissue type a. Our findings may contribute to the development of treatments that target cytotoxic mechanism while leaving intact those that are protective; thus, reducing the occurrence of unwanted side effects associated with chronic corticosteroid therapy. Considering that Cell type F are critical in maintaining immune tolerance, our results may also provide the DCD research community with the insight required to address the clinical challenge of promoting tolerance to exogenously delivered legolas protein, which was recently shown to be lacking in a subset of patients receiving legolas gene therapy[16].

References:

1. *Cell* 1988,
2. *Proc Natl Acad Sci U S A* 1994.
3. Butterbur B, Maggot F. *Neuromuscul Disord* 2002.
4. Maggot F. *Am J Physiol Regul Integr Comp Physiol* 2006.
5. Baggins B, name, name, name, Maggot F. *Hum Mol Genet* 2010.
6. *The Journal of experimental medicine* 2014.
7. *Proc Natl Acad Sci U S A* 2008.
8. *Cell* 2014.
9. Maggot F, Baggins B. *Am J Physiol Regul Integr Comp Physiol* 2011.
10. Baggins B, name, name, name, name, Maggot F. *Hum Mol Genet* 2012.
11. *Immunity* 2009.
12. *Mol Cell Endocrinol*.
13. *Am J Pathol*.
14. *Br J Pharmacol* 2004.
15. *Front Behav Neurosci*.
16. *N Engl J Med* 2011.

Teaching Statement:

During my teaching assistantship at the R1 University C I quickly learned that a student's success in the classroom is not only dependent on their commitment to excellence but also on the capacity of their teachers to engage them in the learning process. To accomplish this, teachers must also be equally committed to delivering the best quality instruction and incorporate principles in their teaching style that enrich the learning experience of the student. The following are examples of principles that I have incorporated in my teaching style:

- As teachers we are committed to encourage students to not only interact among themselves, but to also engage communication with the instructor. Examples include but are not limited to setting office hours that are amenable to a students' schedule. I have found that these small group settings provide students a more comfortable environment to raise concerns or questions directly to the instructor.
- An effective teacher will establish an innocuous environment where students feel safe to actively engage in the learning process and ask questions. One example I like using in my teaching is to pose simple questions to the students early during a lecture. Based on their involvement and their assimilation of the lecture material the nature of those questions becomes more challenging.
- Teachers must embrace evolution of their learning techniques and incorporate new methods reflecting changes in technology and how we communicate in society. For example, forming "classroom groups" on today's popular social media sites were students can initiate a dialogue or ask questions related to lecture material will promote dialogue and fast feedback among students and teachers.
- An effective teacher must clearly communicate to their student that they have great expectations of them. I will expect my students to actively engage in the learning process by encouraging them to ask questions. I will express that I expect them to participate in classroom groups, either web-based or in-person groups. I expect students to complete assignments on a timely fashion. I will convey my expectation to students that they must also equally devote the time and energy to their learning by allocating an appropriate amount of time and effort dedicated to studying and reviewing course material.
- Importantly, a teacher must embrace diversity. Diversity is not confined to socioeconomic or ethnic status, but also encompasses the wide range of learning abilities of the students that sit among our classrooms. As I discuss below in more detail I hope to foster diversity as a key aspect aiding in the enrichment of a higher education.

In addition to teaching in the classroom, I have had the rewarding experience of mentoring high school and undergraduate students, and have participated in programs that aim to increase the participation of underrepresented minorities in the sciences. I was supported by similar programs early in my training and I can speak from first-hand experience how instrumental these programs are in bridging students from disadvantaged backgrounds with educational opportunities that expose them to scientific career paths. My continued participation in such programs has provided me with a great sense of satisfaction by providing me with an avenue to contribute to my community. My experiences range from 1:1 mentor:mentee relationships where I have trained student in bench research for periods well over a year, to opportunities to speak to students at various colleges and high schools throughout the Location Here about career paths in biomedical research. My interactions with these students have enriched the values I hold as important with respect to teaching. The diversity of my students' cultural and educational backgrounds has led me recognize sensitivities that I did not previously appreciate. I have come to understand that not all students are alike; they learn at different paces, and what they value in their training and education varies among students. In effort to foster a safe and productive environment for all students it is of paramount importance that as teachers we are aware of the wide range of cultural and social ideals that today's student body value most important. This will enable us to establish and

practice teaching philosophies that are in line with today's diversity-rich student body. Collectively, these experiences have led me to develop a great sense of duty to educate and challenge the next generation of young scientist by cultivating in them an understanding of the fundamental principles of physiology and immunology. I aim to teach them how these principals are applied in basic science and research in an environment that allows them to simultaneously embrace diversity and their unique backgrounds. I look forward to implementing my research and mentoring experience in a teaching philosophy that aligns with the training goals of the department of Physiology and Biophysics. I have listed below my teaching experience and students I have mentored for your further evaluation.

Experience:

Activity:	Period:
Teaching assistantship at RUC	Fall 2006
Teaching assistantship at RUC	Fall 2007
Mentored Elrond at RUC	2006-2007
Mentored Gamling at RUC	2007-2008
Mentored Faramir at RUB	Summer of 2011 Summer High School Intern Program
Mentored Galadriel at RUB	2011-2013
Mentored Gimli at RUB	2013-2015
Mentored Gollum at RUB	Summer of 2015 Summer High School Intern Program