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2014-2015 Colloquium Schedule

September 12: **Strategies for designing and delivering a scientific presentation** (by Matt Carter, Assistant Professor of Biology and Author of "Designing Science Presentations: A Visual Guide to Figures, Papers, Slides, Posters, and More")
Delivering a clear, engaging scientific presentation that is able to be understood by others requires multiple design and communication skills. In this one-hour talk/workshop, we will discuss strategies for organizing complex scientific information into a simple narrative, using Powerpoint or Keynote software to visually communicate scientific concepts, and improving verbal and nonverbal delivery during a scientific talk. Open to anyone, particularly important for senior biology honors thesis students.

September 19: Connecticut Valley Zebrafish Meeting in Wege Auditorium
13 speakers - Faculty, Graduate and Undergraduate students will give 10-30 min talks about a wide range of work on zebrafish. Topics include neurogenesis, regeneration, gastrointestinal physiology, toxicology, melanoma and developmental biology. Demonstrations of fluorescence microscopy in zebrafish (or your fluorescent samples) by Charles Mazel of Nightsea in MSL 124 throughout the afternoon. Click the link in the title for the complete schedule.

September 26 (BIMO Class of 60s Scholar): Dianne Newman, CalTech
"Why changing color matters to *Pseudomonas aeruginosa*"
One of the defining attributes of *Pseudomonas aeruginosa* is its striking blue-green color. While microbiologists and clinicians have long used color to identify the organism, why it is colored in the first place—and why its color changes with aeration—is a question that not many have considered. We now know that phenazines, a class of redox-active pigments, are responsible not only for the blue-green color of *P. aeruginosa* in the presence of oxygen, but also for different colors displayed by other *Pseudomonas* species. Phenazines came to be known as "secondary metabolites", molecules produced at late stages of microbial growth in laboratory cultures whose function was thought to be to protect *Pseudomonas* species from competitors. While the antibiotic activity of phenazines has been elegantly shown in a variety of contexts, labeling phenazines as "secondary metabolites" suggests that they are not essential to the growth or survival of their producers. I will discuss a variety of important physiological functions phenazines play for *P. aeruginosa* under anoxic conditions that transcend their antibiotic activity, including controlling carbon flux through central metabolic pathways, redox homeostasis, iron

acquisition, survival in multicellular communities, and cell-cell signaling including the implications of these findings for treating cystic fibrosis infections.

October 10 and 17: Thesis Talks

October 24: Jose Andres, University of Saskatchewan, Canada
"Social interactions, relatedness and population structure in cervids: implications for pathogen transmission"
Host populations frequently exist in patchy and isolated environments that create a continuum of genetic and social familiarity. Such variability has an important effect on pathogen spread. Moreover, pathogens are not species-specific and pathogen transmission is a multi-host community-level phenomenon. Thus, there is a clear need to identify the relative contributions of different host species, their interactions, and environmental characteristics in the overall transmission heterogeneity of pathogenic diseases. Using a combination of landscape population genetics and telemetry data I have investigated the landscape-scale potential for bovine tuberculosis (bTB) transmission within a non-migratory white-tailed deer (*Odocoileus virginianus*) and elk (*Cervus canadensis*) community. Our study suggests that the role of white-tailed deer in bTB transmission is likely to be more critical than previously appreciated. This has important applications for ongoing intervention programs that so far have been largely elk-biased.

October 30: Sigma Xi Lecture by **Matt Carter**, Assistant Professor of Biology
"The Neuroscience Behind a Good Night's Sleep"
What happens in our brains and bodies during sleep, and what does it mean to "sleep well"? Recent research suggests that sleep *quality* is about much more than sleep *quantity*. This talk will provide a fundamental understanding of what happens when we sleep andoffer a neuroscientist's definition of "a good night's sleep." We will examine recent studies (from the Carter laboratory and other sleep labs) that elucidate the neuronal basis of sleep and wakefulness. Finally, we will survey simple lifestyle changes that anyone can employ to get a better night's sleep.

November 7: Anne Farewell, STINT Fellow, Sweden
"Global Gene Expression in Stationary Phase *E. coli"*
When gram negative bacteria encounter conditions which do not allow cell growth and proliferation, a massive shift in gene expression occurs. Generally speaking, genes and proteins involved in cell growth are repressed and those involved in survival are induced. This shift is mediated by an overlapping set of global regulatory pathways involving not only traditional activators and repressors, but modifications to RNA polymerase itself. In this talk I will summarize my work and others on the overlapping regulatory pathways of the sigma factor RpoS, the stringent control factor ppGpp, as well as passive regulation of the levels of active RNA polymerase. Finally, I will highlight the importance of these systems in the clinically relevant problem of persistence.

November 14: Mariana Wolfner, Cornell University
"Battles and ballets: Reproductive functions and evolution of seminal proteins in Drosophila"
Male animals transfer seminal proteins to their mates, along with sperm. These proteins improve reproduction by causing physiological and other changes in females. Genetic, genomic and

reproductive methodologies make Drosophila a particularly good system with which to dissect how seminal proteins affect females. Results are of relevance to all animals, including to humans and also to insects that transmit diseases. Seminal proteins act in networks or pathways that include both male and female proteins. For example, the Drosophila seminal protein "ovulin" stimulates females to ovulate by increasing females' neural signaling. Ovulin and other mating components regulate the distribution and levels of several neural signals in females. This likely optimizes the movement of gametes through the reproductive tract. Despite this molecular cooperation, the overall reproductive strategies of males and females differ. This leads to interesting conflicts that affect the evolution of seminal proteins' sequences and levels. In turn, evolutionary information can help to dissect the pathways by which seminal proteins interact with the female.

February 6: Claiming Williams

February 13: Jonathan Gitlin, Marine Biological Laboratory CANCELLED due to weather
"Human disease and the trafficking of transition metals"
Transition metals play an essential role in the metabolism of all living organisms serving as cofactors in specific proteins to catalyze facile electron exchange, bind substrates and stabilize protein structure. These metals are required for nitrogen fixation, oxygenic photosynthesis and aerobic respiration, linking all of life through maintenance of the global electron exchange that sustains our biosphere. As such, the availability of these elements, and their unique chemistries, has served as a fundamental driving force for the evolution of life on Earth. Molecular genetic analyses of human diseases, as well as numerous studies in model organisms, are now defining the mechanisms involved in the uptake and trafficking of these transition metals.

February 20: Winter Carnival

February 27 (BIMO Class of 60s Scholar): Todd Golub, Broad Institute of MIT and Harvard
"Cancer and the Human Genome"

March 13: Saul Villeda, University of California, San Francisco
"A Systemic Approach for Rejuvenating the Aging Brain"
Cognitive decline continues to be one of greatest health threats affecting the elderly. When considering, the rate at which the human population is aging, it becomes imperative to identify means by which to maintain cognitive integrity by protecting against, or even counteracting, the effects of aging. Presupposed dogma holds that the old brain is unable to combat the effects of aging due to a lack of inherent plasticity that facilitates permanent age-related functional impairments. However, our work using heterochronic parabiosis (in which the circulatory systems of young and old animals are connected) has begun to challenge such dogma by showing that systemic exposure of an old animal to young blood can improve adult stem cell function in the old brain. The question now arises whether enhancements of young blood extend beyond regeneration, to reverse molecular and cellular changes underlying age-related cognitive dysfunction. I will talk about ongoing research in my lab that is investigating the role of both pro-aging and pro-youthful blood-borne factors, and the potential of targeting these factors for promoting cognitive rejuvenation.

April 10: Ethan Graf, Amherst College
"Activating the Active Zone: Control of Synaptic Structure and Function by Rab3"
The main goal of my laboratory is to investigate how neuronal synapses form and function. We utilize the *Drosophila* larval neuromuscular junction (NMJ) as a model synapse to explore the molecular mechanisms that control synapse development. In particular, we are interested in mechanisms that influence the formation of the presynaptic release apparatus, the macromolecular machine that regulates neurotransmitter release. We previously identified the protein Rab3 as playing a novel role that controls the localization of the presynaptic release machine to release sites, or active zones. However, the mechanism by which Rab3 regulates synapse formation is not understood. Thus, current work in my laboratory revolves around an exploration of how Rab3 functions at the *Drosophila* NMJ to control active zone development.

April 17 (Biology Class of 60s Scholar): Dean Li, University of Utah
"Using rare human genetic diseases to uncover new signaling cascades in biology"

April 23 (Public Health Class of 60s Scholar): Ruth Faden, Johns Hopkins University
"Henrietta Lacks: Ethics at the Intersection of Health Care and Biomedical Science"
I will begin by summarizing, very briefly, what happened to Mrs. Lacks, her children and her cells. Much of the commentary about the modern lessons of the Lacks experience has focused on the questions raised by their story for the ethics of using human tissues in biomedical science. I will briefly review these questions that have been primarily about consent and compensation. I will spend most of my time, however, arguing that, while these questions are important and remain largely unresolved, they are not independent of wider considerations of social justice and access to medical care. Here I will use our theory of social justice to illustrate why so much of what is most compelling about the experience of Mrs. Lacks and her family has little to do with the science itself.

April 24: Thesis Workshop with Professors Carter, Engel, Williams, and Edwards
To help you prepare and present your poster for the thesis poster presentations (on May 8th), the Biology Department will be having a poster design workshop during our normally scheduled seminar time on Friday, April 24 at 1:10pm in TBL 112. This workshop will provide advice on how to prepare, design, print, and present your poster, and will allow you to share your own thoughts and ideas as well.

Sigma Xi: Marek Demianski, Astronomy

May 8: Thesis Poster Presentations, TBL Lobby 1:00 - 2:30

May 19: Yves Chabu, Howard Hughes Medical Institute at Yale University
"Mechanisms Governing Tumor Overgrowth: A Fly's Eye View"
Activating mutations of the oncogenic *RAS* gene are highly prevalent in human cancers. However, there are no successful targeted therapy strategies and the available chemotherapy regimens are not effective at treating the most aggressive Ras cancers. Developing effective therapies against Ras cancers has been challenging partly because oncogenic Ras signaling is complex and involves a network of cooperating pathways. How oncogenic Ras elicits these growth-promoting signaling pathways and how these pathways are integrated to achieve

oncogenic synergy is not fully understood. Genetic screens aimed at isolating mutations that synthetically suppress overgrowth in animal models of oncogenic Ras will not only broaden our understanding of cancer biology, but can also potentially lead to the discovery of novel therapeutic targets. Using a fly tumor model, we performed a genetic screen aimed at identifying mutations that can suppress oncogenic Ras-driven tumor overgrowth and identified exocytosis and *Egfr* mutants. The talk will be centered on the characterization of novel molecular