How to develop and write an NIH grant

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How to become funded

- Idea
- Commitment
- Grant writing skills

Idea: how to develop one

- Be knowledgable
  - Extensively read existing literature
  - Where is the current cutting edge of knowledge?
- Be thoughtful
  - Devote time to “just” thinking
  - Think in question format: formally write out every question you’d like to ask that’s even remotely related to your project
  - Think in experiment format: formally write out every possible experiment you should do or you dream about doing – with no consideration of money, expertise or equpiment
  - Think in hypothesis format: formally write out all of the hypotheses related to your project
- Be creative
  - Borrow tools and approaches from other fields
  - Combine these in new and compelling ways
- Be open to feedback and criticism
  - Share your ideas with colleagues before you start writing
  - Learn to accept criticism – it’s not personal

Commitment

- Passion
- Attitude
  - I can't → - I can and I will
  - I don't have time → - I will reorder my priorities
  - There's too much competition → - I welcome the chance to compete
  - It's good as it is now → - It can always be better
  - I'll submit now and "get in line" → - I won't submit until it's the very best grant I can write
- Time
  - Lead time – *how much time do you think it takes to prepare a 25 page grant submission?*
  - Quality time

Grant writing skills

- There’s one and only one key point
  - You have to sell your ideas to reviewers
  - You have to make the Reviewer your advocate in the Study Section (more on that later)
- How?

How to sell your ideas to the Reviewer

A successful salesperson

- Has something special to offer (significance and importance of work to the field)
- Makes a good first impression (Specific Aims Page)
- Is well prepared and knowledgable (B+S section)
- Has appropriate credentials (BioSketch)
- Provides supporting documentation (Preliminary data; published papers)
- Delivers a clear message than can be understood by a knowledgable person without specialized background (Research plan)
- Has appropriate endorsements (Letters of Collaboration from colleagues)
- Is persistent!!!

Abstract

- A concise summary of the question/hypothesis, aims and their SIGNIFICANCE

- Text boxes taken from Dr. Erfei Bi, Associate Professor, Dept. of Cell Biology and Development, Univ. Penn SOM

Signaling mechanisms in cell polarity in yeast

My long-term objective is to use the genetically tractable eukaryote Saccharomyces cerevisiae to determine how Cdc42p, an evolutionarily conserved GTPase, controls the organization of the actin cytoskeleton and of the septins. Recent work suggests that Cdc42p controls the actin organization by two parallel pathways: one involving Msb3p, Msb4p, and Bni1p; the other involving Gic1p, Gic2p, and Bem4p. In the present studies, these pathways will be explored further, focusing initially on the roles of Msb3p and Msb4p.

Msb3p and Msb4p, a pair of highly homologous proteins in yeast, play a redundant role in linking Cdc42p to the actin cytoskeleton. They belong to a family of proteins that includes the Drosophila cell adhesion molecule, Pollux, and the human oncoprotein, Tre17. Thus, studying the function of Msb3p and Msb4p will shed significant light on the function of this family of proteins. Specifically, research in this proposal will address the following questions: What proteins link Msb3p and Msb4p to the upstream Cdc42p, and to the downstream actin cytoskeleton? How are the proteins in the Msb-mediated pathways organized at the molecular level? What are the functions of the putative domains in Msb3p and Msb4p (the highly conserved PTM domain, the membrane-spanning domains, and a lipid modification site)? How are the localization of Msb3p and Msb4p regulated in the cell cycle? These questions will be answered with a combination of genetic, cytological, and biochemical approaches. To expand our hypothesis, additional genes that are involved in regulating Cdc42p activity or a specific effector pathway will be identified by genetic screens, and affinity chromatography coupled with tandem mass spectrometry.

Homologs of Cdc42p are involved in diverse cellular processes, such as cell polarity, cell migration, and cell growth control. In addition, deregulation of Cdc42p activity in mammals is associated with serious diseases, such as cancer. Thus, studying the signaling mechanisms of Cdc42p in yeast represents an important bridge between basic and clinical sciences.

Specific Aims section

- The single most important section in the grant
  - It's the master plan for the rest of the proposal
  - You engage or lose the Reviewer on this page
- It's the most difficult section to write
  - The logic of each aim must be compelling
  - The answers must be important to the field
- Write Aims that you are excited about!

Specific Aims section

- Whenever possible – test a hypothesis in the specific aim title
  - You want the Reviewer to know that your work is hypothesis driven
  - Don't make the Reviewer work to figure out what the hypothesis is
- The goal of the aim should be to understand mechanism – even if the experiments are largely descriptive
- 3 – 4 Specific Aims for a 4 to 5 year grant – each aim is a paper, or is a significant part of a paper
- The Specific Aims should be detailed but far reaching – the Aims should not be a list of experiments

Specific Aims - Examples

Okay:

Specific Aim 1: To test the hypothesis that neurons in the GluR1 knockout mouse will have delayed dendritic maturation.

Better:

Specific Aim 1: To test the hypothesis that GluR1 signaling is necessary for dendritic maturation.

(or is sufficient).

TITLE: Signaling mechanisms in cell polarity in yeast

Research Plan

A. Specific Aims

My long-term objective is to use the genetically tractable eukaryote *Saccharomyces cerevisiae* to determine how Cdc42p activity is regulated and how it controls the organization of the actin cytoskeleton and of the septins. Recent work has led to our model that *S. cerevisiae* Cdc42p controls actin organization by two parallel pathways, one involving Msb3p, Msb4p, and Bni1p, and the other involving Gic1p, Gic2p, and Bem4p. In the studies proposed here, these pathways will be explored further, focusing initially on the roles of Msb3p and Msb4p. *MSB3* and *MSB4* can function independently as multicopy suppressors of *cdc24* and/or *cdc42* mutations. Both proteins have a cell cycle-regulated, cell cortex-associated localization that depends on Cdc42p function. Deletion of *MSB3* and *MSB4* together produces a strong defect in actin organization but not in septin organization. These data suggest that Msb3p and Msb4p function downstream of Cdc42p and are specifically involved in linking Cdc42p to the actin cytoskeleton. This function will now be explored further through the following specific aims.

1. Establish molecular linkages among Msb3p, Msb4p, and other proteins involved in controlling actin organization

We will (*a*) determine whether Msb3p and Msb4p interact directly with Cdc42p or other proteins that are known to affect actin organization by two-hybrid, co-immunoprecipitation, and *in vitro* protein binding experiments; (*b*) identify additional Msb3p- and/or Msb4p-interacting proteins by two-hybrid, affinity chromatography, and genetic screens; and (*c*) determine the functional order of proteins involved in the Msb3p/4p-mediated pathway by *in vivo* and *in vitro* protein binding assays.

2. Define functional domains of Msb3p and Msb4p

Both Msb3p and Msb4p are predicted to have the following molecular domains at corresponding positions: an evolutionarily conserved PTM domain, two potential membrane-spanning domains, and a motif similar to the prokaryotic membrane lipoprotein lipid attachment site. To define the role of each domain in Msb3p/4p protein function, we will (*a*) perform structure-function analyses on Msb3p and Msb4p; and (*b*) determine to what extent related proteins from other organisms are functional homologues of Msb3p and/or Msb4p. In addition, we will (*c*) determine how Msb3p and Msb4p localization is regulated during the cell cycle.

3. Identify additional genes involved in Cdc42p signaling pathways

We will use both (*a*) genetic and (*b*) biochemical approaches. In the genetic approach, we will exploit a set of 82 temperature-sensitive (ts) mutations in *CDC42* that have been generated by random PCR mutagenesis. These mutations have been classified into three groups according to their terminal morphologies. Representative mutations will be used to identify additional genes involved in Cdc42p signaling by isolating overexpression or extragenic suppressors. In the biochemical approach, we will use a previously constructed set of three *GST-CDC42* fusions that contain different Cdc42p moieties. These fusions will be used to identify proteins that are specifically associated with different forms of Cdc42p by affinity chromatography coupled with tandem mass spectrometry. We will then determine whether and how the newly identified genes/proteins fit into our parallel-pathway model.

Specific Aims: Dos

- Write your Aims early – some may fall apart as you design a plan to test them or discuss them with colleagues
- Try to limit this section to one page – it's a roadmap to the rest of the proposal and it must include the logic behind your aims.
- Don't assume your Reviewer is an expert in your particular area – so write Aims for a non-expert compared to the rest of the proposal

Specific Aims: Don'ts

- Don't state a hypothesis that you cannot actually test with the experiments you are proposing
- Avoid using phrases like: To correlate… To describe… To develop; these help get your grant pegged as "too descriptive"
- Avoid wishy-washy, passive tense, or flowery language – instead write your aims in active form with strong meaningful verbs
- Don't write aims that can be viewed as "a fishing expedition" – microarray experiments, expression cloning, etc.

Background and Significance

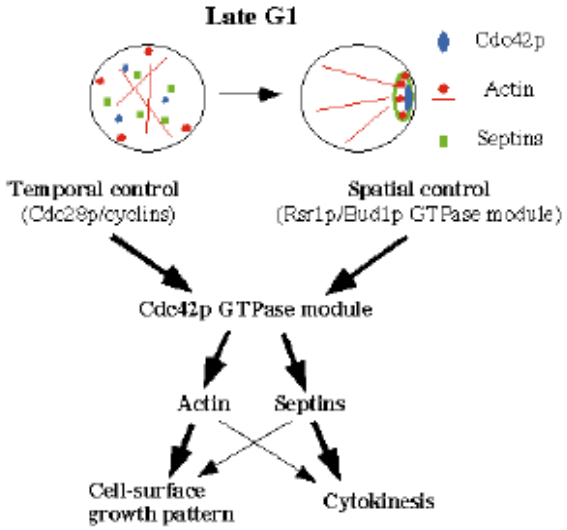
- Background
  - Should lead the reader to each question or hypothesis that you're testing in each aim
- Significance
  - State this explicitly
  - This section must explain why the Study Section should fund your proposal rather than the next one
  - What is the "value added" to your field if you're able to do the work?

B. Background and Significance

Cell polarity can be simply defined as the asymmetric organization of cellular components, including plasma membrane proteins, organelles, and cytoskeletal elements. The generation of asymmetry or functional domains within the cell plays a crucial role in many biological processes and is essential for development and differentiation (20, 30). However, the molecular mechanisms for establishing the axis of cell polarity are not well understood in any system. We are using the budding yeast *Saccharomyces cerevisiae* to study how the actin cytoskeleton is polarized at the molecular level because of the many experimental advantages of this organism, including its genetic tractability. In addition, the behavior of the actin cytoskeleton during the *S. cerevisiae* cell cycle has been well characterized and can easily be monitored (2, 44, 50), thus providing a convenient assay for examining the effects of mutations on actin organization.

Parallel studies in many systems have firmly established that Cdc42p, an evolutionarily conserved GTPase, plays a central role in the polarized organization of the actin cytoskeleton in diverse organisms ranging from yeast to humans. During the last few years, accumulating evidence has suggested that Cdc42p also plays a role in other cellular processes. In this section, I will briefly review what we know and what we do not know about the function of Cdc42p in yeast and other systems; then, I will discuss the significance of my proposed studies.

The diagram below summarizes our current understanding of the morphogenetic pathway in *S. cerevisiae* (Fig. 1).



Cell polarity in yeast

In budding yeast, newly divided cells grow isotropically in G1 until the cell reaches a critical cell size, then the master cell cycle control system, comprising Cdc28p kinase and the G1 cyclins, is activated

Preliminary Studies

- In order of Specific Aims
- You don’t have to know the outcome of each experiment before the grant is submitted
- You DO have to:
  - Show that you can perform all of the necessary techniques and methods (Letters of Collaboration)
  - You are committed to this area of research and are off and running
  - New techniques are feasible, reliable and yield interpretable data

C. Preliminary Studies

Introduction. Most of the studies described below were performed by me, with some help from two undergraduate students, while I was a postdoctoral fellow in John Pringle's laboratory at the University of North Carolina at Chapel Hill. The initial identification and characterization of *MSB3* and *MSB4* are described in the accompanying paper (9). Since January, 1998, I have established my own laboratory at the University of Pennsylvania School of Medicine. I am continuing to explore the molecular role of Msb3p and Msb4p in linking Cdc42p to the actin cytoskeleton. In addition, we are undertaking genetic and biochemical approaches to identify additional genes involved in Cdc42p signaling. This section contains three sets of results that form the basis for the three Specific Aims.

1. Msb3p and Msb4p are specifically involved in linking Cdc42p to the actin cytoskeleton

Genetic interaction between *MSB3* or *MSB4* and *CDC42*. *MSB3* was isolated as a gene that can serve as a multicopy suppressor of a particular *cdc24* allele (*cdc24-11*) in conjunction with multicopy *CDC42*. In addition, multicopy *MSB3* by itself suppresses a *cdc42*^{ts} mutation, suggesting that Msb3p positively regulates Cdc42p function. A close homolog, *MSB4*, was identified by database searching. Msb3p and Msb4p are 50% identical in amino acid sequence over the entire proteins. Like *MSB3*, multicopy *MSB4* and *CDC42* together suppressed *cdc24-11*, suggesting that Msb3p and Msb4p are functional homologs.

Msb3p and Msb4p are functionally redundant and are specifically involved in actin organization. Deletion of *MSB3* or *MSB4* alone did not produce any obvious phenotype. However, deletion of both genes together resulted in slow cell growth, particularly at lower temperatures. In addition, a large proportion of the double mutant cells became larger and rounder than normal (Fig. 3), with F-actin randomly distributed in the cell cortex; this resembles the phenotype of a *cdc42*^{ts} mutant at the nonpermissive temperature. These data suggest that Msb3p and Msb4p play a redundant role in actin organization. In addition, they support the genetic conclusion that Msb3p and Msb4p positively regulate Cdc42p function.

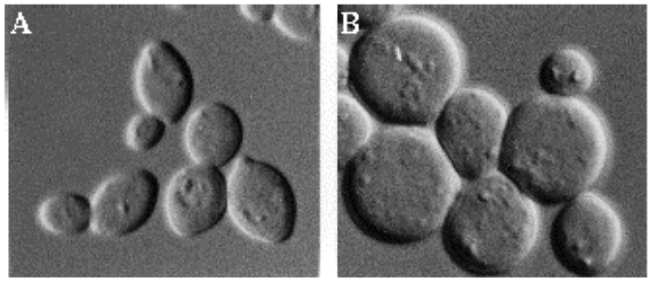


Fig. 3. Morphological defects caused by *msb3* and *msb4* deletions. A wild-type strain (A) and an isogenic *msb3 msb4* double mutant strain (B) were grown exponentially in rich medium at 23°C, then processed for Differential-interference-contrast (DIC) microscopy.

To ask whether Msb3p and Msb4p function as general regulators of Cdc42p (regulate the ratio of GTP/GDP-bound Cdc42p) or as specific effectors involved in actin organization, the following logic was applied: Cdc42p function is required for the polarized organization both of the actin cytoskeleton and of the septins at the beginning of the cell cycle. However, the organization of these two cytoskeletal elements is independent of each other (3, 35). Thus, if Msb3p and Msb4p are general regulators of Cdc42p, deletion of these genes should affect the organization of both cytoskeletal elements; if Msb3p and Msb4p function as a part of the effector pathway that links Cdc42p to the actin cytoskeleton, deletion of both genes should not affect septin organization. The latter result was obtained: the septins were able to organize normally in the fraction of *msb3 msb4* double mutant cells that had lost actin organization (a representative cell is shown in Fig. 4), suggesting that Msb3p and Msb4p function downstream of Cdc42p and are specifically involved in linking Cdc42p to the actin cytoskeleton.

Experimental Plan

- Specific Aims are fleshed out with the actual experimental approach
  - Rationale (1 paragraph) – logic- why are you doing it? Two possible things: why are you asking this question in the first place? v/s why are you doing THESE SPECIFIIC EXPERIMENTS?
  - Experiments – how- how are you doing it?
    - CONTROLS (positive and negative)
  - Analysis and Interpretation – what will results mean?
  - Pitfalls and Alternative Approaches
  - Detailed Methods

D. Research Design and Methods

Training of the applicant. I have extensive training in genetics, cell biology, and molecular biology from my prior work in Joe Lutkenhaus's laboratory at the University of Kansas Medical Center and in John Pringle's laboratory at the University of North Carolina. When I was a graduate student, I used a combination of approaches to study bacterial cell division, including co-immunoprecipitation, cross-linking, and immunoelectronmicroscopy. During my postdoctoral training, I have been highly experienced in yeast genetics and cell biology. With specific questions in mind, I have performed several successful genetic screens. In addition, I have used a variety of microscopy techniques including video microscopy, and I have also done affinity purification and protein extraction experiments. Thus, I believe that the experiments proposed here can be efficiently carried out in my laboratory, which is already well equipped by virtue of my start-up package from the University of Pennsylvania.

Aim 1. Establish molecular linkages among Msb3p, Msb4p, and other proteins involved in controlling actin organization

One of the central issues in polarity establishment is how positional information is communicated from Cdc42p in the cell cortex to the actin cytoskeleton. As discussed above, genetic and cytological evidence suggests that *S. cerevisiae* Cdc42p controls actin organization through two parallel pathways, one involving Msb3p, Msb4p, and Bni1p, and the other involving Gic1p, Gic2p, and Bem4p. However, many important questions remain unanswered: How do Msb3p and Msb4p interact with other components in the same pathway biochemically? Are there additional factors that function upstream or downstream of Msb3p and Msb4p to link Cdc42p to the actin cytoskeleton? What is the functional order among the components in each pathway? How are the parallel pathways organized at the molecular level? Given the power of yeast genetics and the well characterized actin behavior during the yeast cell cycle, the functional output of each physical interaction can easily be assessed by monitoring the effects of alterations in that particular interaction on actin organization *in vivo*. Answers to all the questions above are essential for elucidation of the molecular mechanisms by which Cdc42p controls actin organization.

Question #1: Do Msb3p and Msb4p interact directly with Cdc42p and/or with other proteins that are known to affect actin organization? Based on the parallel-pathway model, the simplest prediction is that Msb3p and Msb4p interact directly with the GTP-bound form of Cdc42p, and perhaps with Bni1p, but do not interact with Gic1p, Gic2p, or Bem4p. It is also possible that Msb3p and Msb4p are linked to Cdc42p through other proteins that are known to affect actin organization, such as Bem1p, Bem2p, Boi1p, Boi2p, Rho3p, or Rho4p.

Methods. (i) Two-hybrid: We have cloned full-length Msb3p and Msb4p into the bait plasmid pEG202 (87), as well as into a prey plasmid, a modified version of pJG4-5 (29, 36). These constructs will be assayed for their interactions with various forms of Cdc42p (the wild-type, constitutively active, and constitutively inactive forms), Bni1p, Gic1p, Gic2p, Bem4p, Bem1p, Bem2p, Boi1p, Boi2p, Rho3p, and Rho4p. Most of the relevant constructs are already available, and preliminary assays have shown that full-length Msb3p and Msb4p have a weak interaction with the constitutively activated forms of Cdc42p [$Cdc42p^{G12V, C188S}$, and $Cdc42p^{Q61L, C188S}$ (the purpose of the C188S mutation in the CAAX box is to prevent the membrane localization of Cdc42p so that a two-hybrid interaction in the nucleus is possible)]. These weak interactions were observed only when *MSB3* or *MSB4* was under inducible promoter control in the prey plasmid. No interaction was observed between full-length Msb3p or Msb4p and full-length Bem1p. The other genes listed above have not yet been tested. The full matrix of potential two-hybrid interactions among these genes will be thoroughly examined and the results will be integrated with those from other approaches in an attempt to define the proteins with which Msb3p and Msb4p directly interact.

Anticipated Results/Pitfalls: Both Msb3p and Msb4p are predicted to contain at least one membrane-spanning domain, which may explain why a weak interaction between the full-length Msb3p or Msb4p and the constitutively active form of Cdc42p was observed only when the expression of *MSB3* or *MSB4* was induced to a high level in the prey plasmid (perhaps, a small amount of the full-length protein can get into the nucleus under such conditions). Even if Msb3p and Msb4p are indeed membrane proteins (to be investigated under Specific Aim 2), the two-hybrid approach can still be applied by using fragments of Msb3p or Msb4p that do not contain the possible membrane-spanning domain. Even for nonmembrane proteins, it has been well documented that in some cases, a fragment of the protein shows stronger interaction with the partner than does the full-length protein. The advantage of the two-hybrid approach is that many potential interactions can be assayed easily in a short time. However, at best, the results from two-hybrid assays can only indicate that two proteins are capable of interacting with each other (directly or through a bridging protein) in the nucleus when overexpressed.

Other grant parts

E. Human Subjects

F. Vertebrate Animals

G. Literature Cited

H. Consortium/Contractual Arrangements

I. Consultants

You have a draft…now what?

- Rewrite.
  - Read each sentence ALOUD. Can it be made simpler? Less wordy? More compelling?
  - The only good writing is REWRITING.
- Get feedback from other scientists – in and somewhat tangential to your field
  - Timing
  - Accepting criticism
  - “Pay it forward” principle
- Repeat above.

Other Important Issues

- Page requirements
- Font size and line spacing
- SPACING OF TEXT SECTIONS
- Embed figures into the text. Include a brief, clear legend.
- Figure must be absolutely clear/visible to the Reviewer – include color pages and mark these copies as “Color Figures for Reviewer.”
- Learn how to use MS Word
- Spelling and grammar – ZERO TOLERANCE for sloppy mistakes.

DR. PAMELA MARINO
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SUMMARY STATEMENT
(Privileged Communication)

Application Number: 1 R01 GM59216-01

Review Group: MBC-1
MICROBIAL PHYSIOL & GENETICS SS SUBCOM 1

Meeting Dates: IRG: OCT/NOV 1998 COUNCIL: JAN/FEB 1999 C103AM
Requested Start Date: 04/01/1999

BI, ERFEI, PHD
UNIV OF PENNSYLVANIA SCH OF MED
ANATOMY
36TH & HAMILTON WALK
PHILADELPHIA, PA 19104-6058

Project Title: SIGNALING MECHANISMS IN CELL POLARITY IN YEAST

IRG Action: Priority Score: 155 Percentile: 6.0
Human Subjects: 10-NO HUMAN SUBJECTS INVOLVED
Animal Subjects: 10-NO LIVE VERTEBRATE ANIMALS INVOLVED

GENDER, MINORITY, & CLINICAL TRIAL CODES NOT ASSIGNED

PROJECT YEAR	DIRECT COSTS REQUESTED	DIRECT COSTS RECOMMENDED	ESTIMATED TOTAL COST
01	193,719	166,219	258,939
02	170,516	170,516	265,633
03	174,941	174,941	272,526
04	179,499	179,499	279,627
05	184,195	184,195	286,942
TOTAL	902,870	875,370	1,363,667

NOTE TO APPLICANT FOLLOWS SUMMARY STATEMENT.

RESUME AND SUMMARY OF DISCUSSION

This is an outstanding proposal Dr. Bi to explore the bifurcated pathways controlled by Cdc42. Strengths of the application include the investigator, the importance of the questions being addressed, and the care with which the research plan was presented. Various possibilities for pathway organization were clearly laid out, and experiments were designed that should distinguish among them. Anticipated results and potential pitfalls, and their resolutions follow each experimental section. The experimental plan has been carefully thought out. No major weaknesses were identified.

DESCRIPTION:

Dr. Bi's long term objective is to use budding yeast to determine how Cdc42, an evolutionarily conserved GTPase, controls the organization of the actin cytoskeleton and of the septins. Recent work suggests that Cdc42 controls the actic organization by two parallel pathways: one involing Msb1, Msb4, and Bni1; the other involving Gic1, Gic2, and Bem4. In the present studies, these pathways will be explored further, focusing initially on the roles of Msb3 and Msb4.

Date Released: 12/23/1998 Date Printed: 12/23/1998

Responding to the Reviews

- Read the reviews.
- Get over your disappointment and anger.
- Don’t take it personally.
- Respect the Reviewers, their Reviews, and the process.
- Take them seriously – often they are right.
- Make a list of the major and minor issues and respond first to the major ones.
- Directly respond to the criticisms with positive responses. If the Reviewer misunderstood and is thus wrong -- it’s your fault, not theirs!
- So, show them why, using facts, logic, additional explanation, references, etc.
- Do not NOT address one of the issues, even the most minor one, that is raised by a Reviewer – especially if more than one Reviewer mentions it.
- Don’t send the same grant back. You must show progress, evolution of your thinking, etc.
- Don’t include anything so far out that can raise new questions if your score is close.

NIH's new electronic grant application process

Date: Tue, 13 Dec 2005 17:00:00 -0500
Reply-To: PennERA@POBOX.UPENN.EDU
Sender: PennERA Proposal Tracking Investigators <ERA_PT_INVESTIGATORS@LISTS.UPENN.EDU>
From: PennERA@POBOX.UPENN.EDU
Subject: NIH/Grants.gov Webcast Update
To: ERA_PT_INVESTIGATORS@LISTS.UPENN.EDU
X-Spam-Checker-Version: SpamAssassin 3.0.0 (2004-09-13) on pobox.upenn.edu
X-Spam-Status: No, score=-10.6 required=5.0 tests=ALL_TRUSTED,HTML_30_40,
HTML_MESSAGE,MIME_HTML_ONLY,NO_REAL_NAME autolearn=disabled
version=3.0.0
X-Spam-Level:

TITLE: NIH's New Electronic Grant Application Process and the SF424 (R&R)

PURPOSE: By May 2007 all research grant applications for NIH will have to be submitted electronically through Grants.gov using the SF424 Research & Related (R&R) form set. This training session, geared toward the applicant community, will provide an overview of NIH's transition plans, the submission process and the new form set. A question and answer session will follow the formal presentations.

WHEN & WHERE: 2 Sessions Available DUNLOP AUDITORIUM, ground floor, Stemmler Hall
Wednesday, January 11, 2006, 8:30 AM to 12:00 PM EST
Wednesday, January 11, 2006, 12:30 PM to 4:00 PM EST

Both the morning and afternoon sessions will also be available for remote viewing via VideoCast, NIH's streaming video service. For more information OR to register to view this program on your desktop:

http://era.nih.gov/training/ElectronicSubmission/

IF YOU ARE PLANNING TO VIEW THE WEBCAST IN DUNLOP AUDITORIUM, IT IS NOT NECESSARY TO REGISTER.

On line resources for grant writing

- Visit the *Advance* faculty professional development web site at www.med.upenn.edu/fapd/advance and view the following materials on the research page:
  - All About Grants tutorial on developing R01 grant applications produced by the NIAID at the NIH http://www.niaid.nih.gov/ncn/grants/default.htm

    CHECKLIST – very helpful
  - Common Pitfalls of Grant Preparation PowerPoint with synchronized voice by Dr. Ann Kennedy, Professor of Research Oncology at Penn School of Medicine

- Some information taken from “Grantsmanship workshop: how to develop a fundable research proposal,” T. Bray, Ph.D., Dean, Oregon State Univ. College of Health and Human Sciences