Strategies and Career Resources for Postdocs

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Strategies for a Successful Postdoctoral Traineeship

1. Publication
2. Oral Communication
3. Getting Funded

Strategies for a Successful Postdoctoral Traineeship

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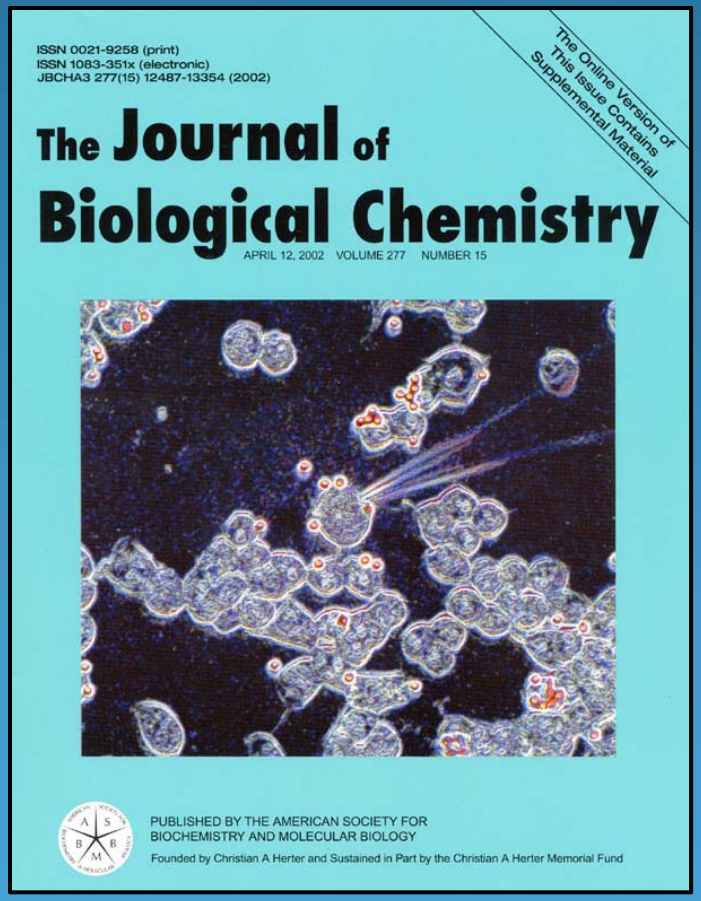
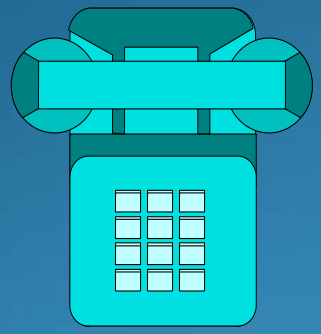
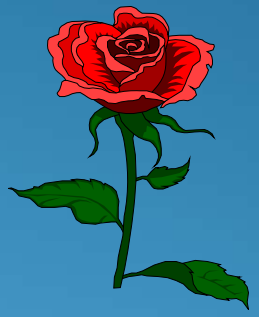
Publication without losing sight of the joy of discovery



The Idea





Read the literature AND talk to your colleagues

Discovery Favors the Prepared Mind

SUCHEN:

Erde | Mensch & Technik

06. Februar 2004

...hten Supernasen

...al mehr, wie Gentechniker ...usen erkennen mussten. Die ...n Nagern ein Protein - und ... Supernasen.

...rdünnte ...esser ...elassenen ...n Debi ...sity of ...nd ihre ...itschrift "Neuron" (Bd. 41, S. 389 - 404). Den Nagern fehlt das Protein mit der Bezeichnung Kv1.3, das bei der Übertragung der Informationen von der Nase zum Gehirn eine wichtige Rolle spielt.

DPA

Maus mit Käse: Ohne Kv1.3-Protein duftet der Happen noch besser

SCHLAGZEILEN
FORUM
LÄNDERLEXIKON
LESERBRIEFE
NEWSLETTER
ARCHIV
DOSSIERS

Exklusiv

"Opportunity": Mars-Wasser bot Raum für Leben

- Falscher Asteroiden-Alarm: "Rufen wir den Präsidenten an?"
- Forschungsbetrug: Daten-Trickser behält Professorentitel
- Krebsvorsorge: "Wir brauchen einen McCancer"
- Umstrittene Tests: US-Mediziner befürworten Menschenversuche mit Giften

What are the logistics of publication for a postdoc?

1. Need first author publication on 2 or more papers. After the second year of your postdoctoral fellowship, you need periodic consistency.

2. You need a foundation of quality publication in your own field that will support feasibility for a fundable project.

3. Careful, planned experiments drawn together for a full-length manuscript (cohesive story).

4. Need to have multiple pots on the stove.

Creating an Integrated Research Plan

"DREAM PAPER" vs. "Set of fragmentary observations"

1. Think graphically (pencil then software)
2. Sketch out an encompassing hypothesis
3. Generate questions as you analyze your results
4. Additional clarifying experiments
5. Figures >> Methods >> Introduction >> Results >> Discussion >> Abstract >> Title >> Cover Letter
6. First authors write the first draft
7. First authors send to colleagues and revise

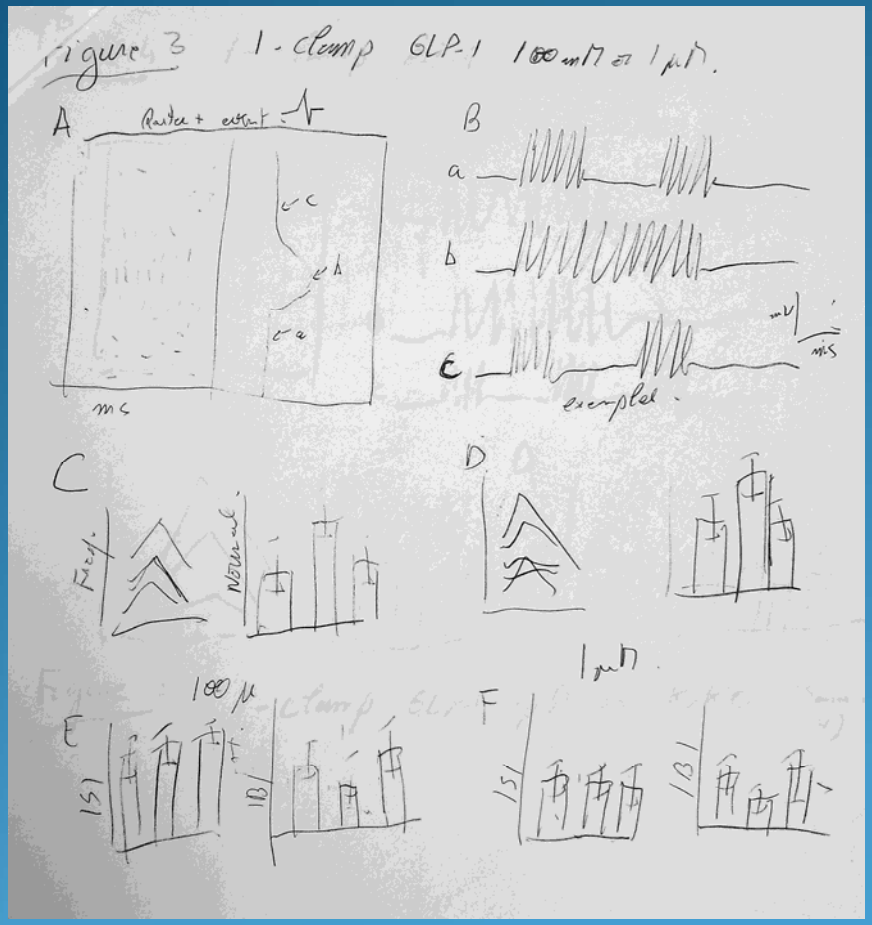
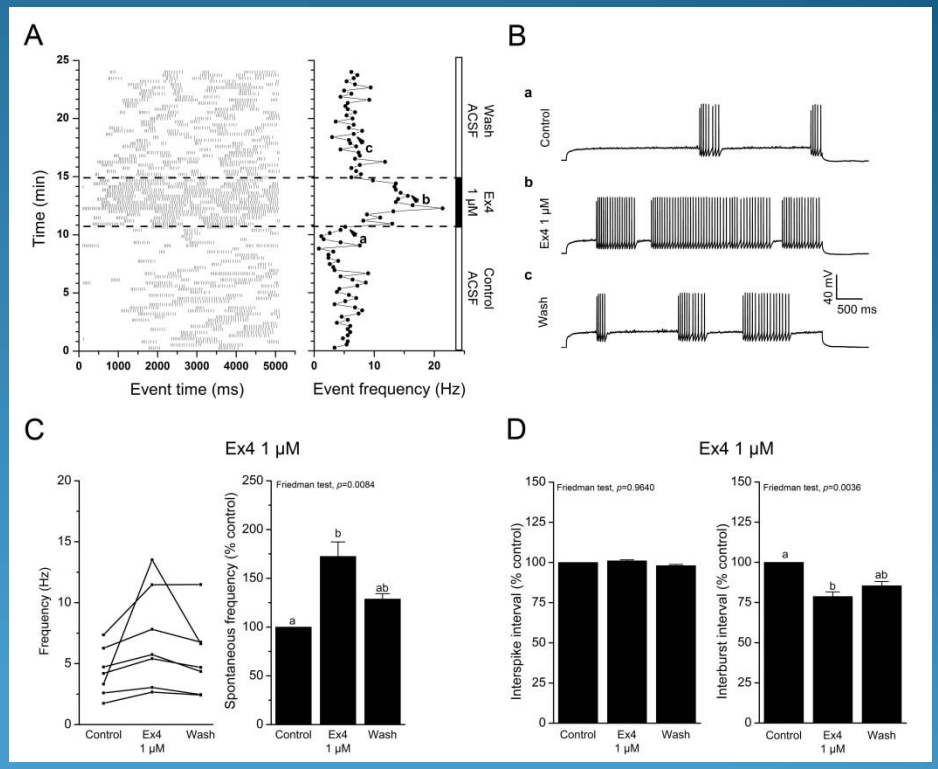
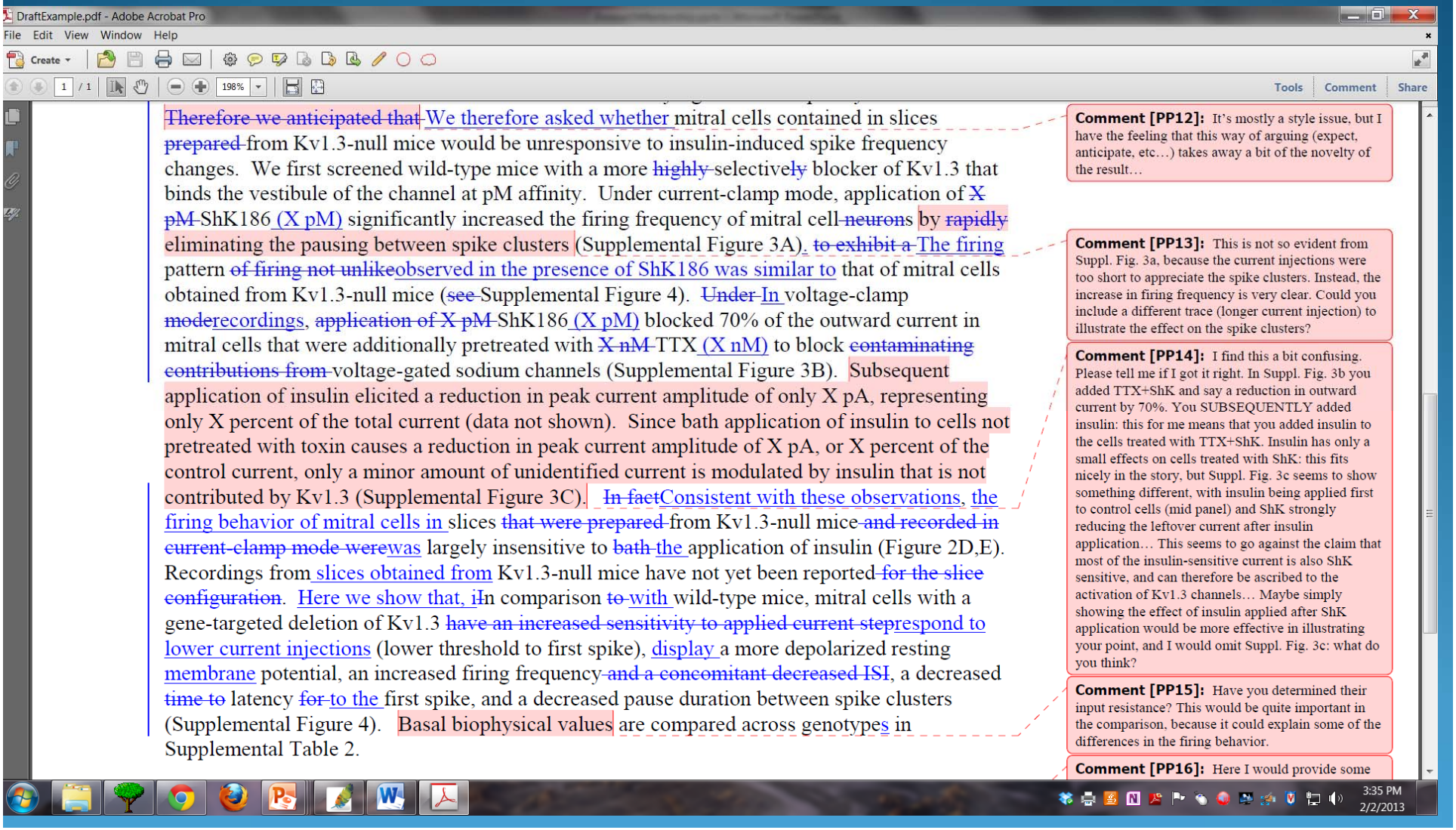


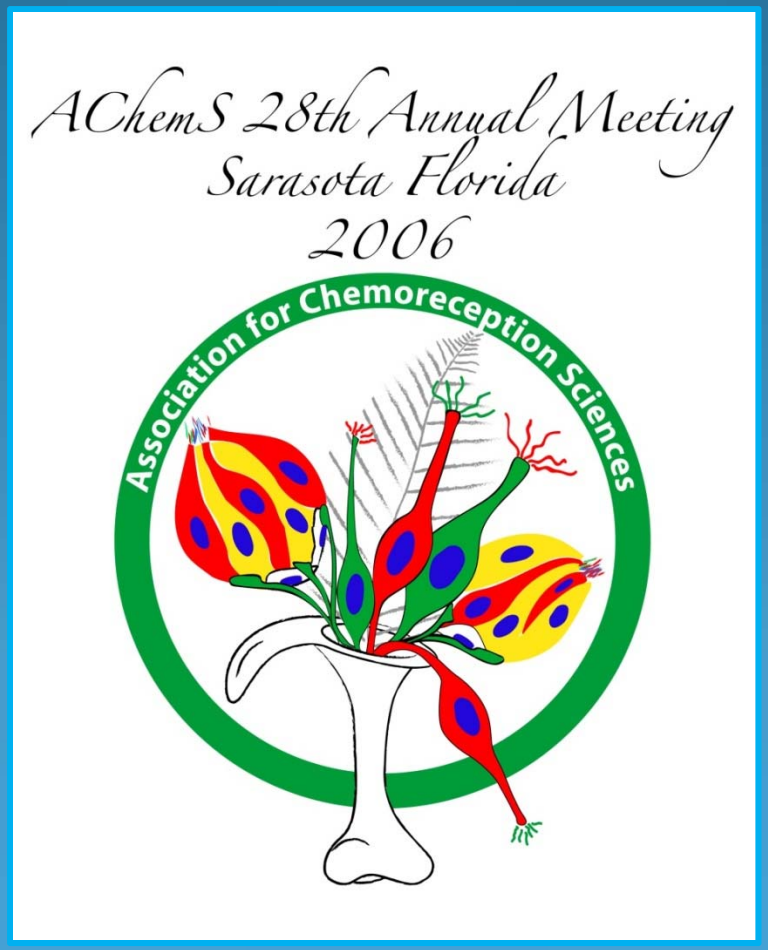
figure 3 I-clamp GLP-1 100 nM or 1 µM.
A
Raster + event
B
a
b
c
mV
ms
example
C
Freq.
Normal
D
100 µ
1 µM
E
F
1S
1B


A
Time (min)
Event time (ms)
Event frequency (Hz)
Wash ACSF
Ex4 1 µM
Control ACSF
B
Control
Ex4 1 µM
Wash
40 mV
500 ms
C
Ex4 1 µM
Frequency (Hz)
Spontaneous frequency (% control)
Friedman test, p=0.0084
Control
Ex4 1 µM
Wash
D
Ex4 1 µM
Interspike interval (% control)
Friedman test, p=0.9640
Interburst interval (% control)
Friedman test, p=0.0036

An internal review will increase strength of the external review



How can you generate support and refine your planned publication?



Submitting and Resubmitting

1. What is a pre-submission inquiry?
    a. abstract
    b. 500 word cover letter
    c. meeting
2. Navigating the review and comments to reviewers
3. What is a “cost-benefit analysis”?
4. What does it mean to take the “high road”?
5. What if you are asked to be a reviewer?

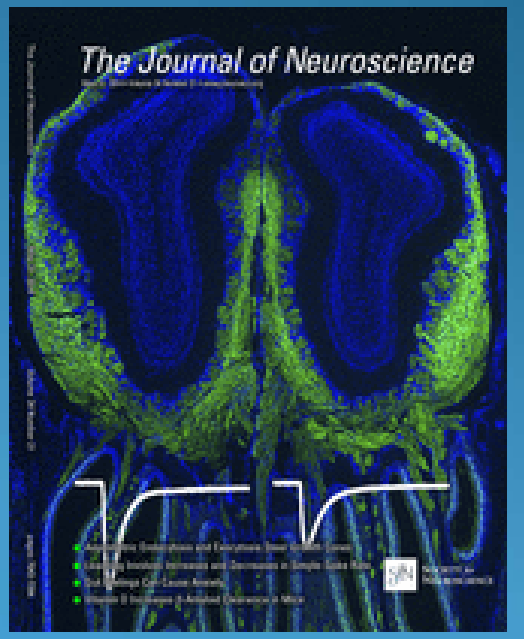
2. **Comment:** Why *serial sections are not performed to determine the exact location the costal-caudal and in latero-median axes?*
Response: From both our whole mount and cryosections, it is evident that the location of the glomerular projection is not grossly modified. There are very refined software programs that are now available to perform the determination of the precise costal-caudal and latero-median axes (Diego Restrepo Laboratory). These determinations are extremely time consuming and we judge pragmatically that we would be confirming a non effect. It was decided that this degree of quantification would not be a good time investment.

3. ***Comment:*** *[for the M72 glomerulus in sections on page 11] The description of quantification analysis is uncomplete. It is necessary to give: cryosection number/animal like in page 12 for immunodetection of Ki67 protein.*
Response: We disagree. When we are immunodetecting Ki67 protein, then we are making our statistical metric by using a sampling of the population. Therefore the range of the sections sampled per animal is necessary to indicate depth and quantity of the measurement. When we are sampling the number and cross sectional area of the M72 glomerulus, there is not a range of sections. We are locating that particular M72 glomerulus (genetically identified) within a single section and then measuring it in that particular animal. Only the number of animals or half bulbs sampled is necessary.

4. ***Comment:*** *It seems that the SDS-PAGE Western blot analysis of the two proteins Golf and MOR28 are not extracted from the same sample tissue, since number of mice is different for the two proteins.*
Response: This is correct. We sampled Golf in 12 membrane preparations (6 CF, 6 MHF) and MOR28 in 28 preparations (14 CF, 14 MHF). MOR28 was sampled more frequently because its small size (we could cut the nitrocellulose when screening for higher molecular weight proteins).

Increasing your visibility after publication

1. Symposia or talks at conferences
2. University media site
3. Laboratory website
4. Cover art
5. Why such self-promotion?
    a. Federal funding agencies
    b. Next reviewer
    c. Potential collaborators
    d. Future laboratory members
    e. Reverse invitations



Thiebaud et al., 2014

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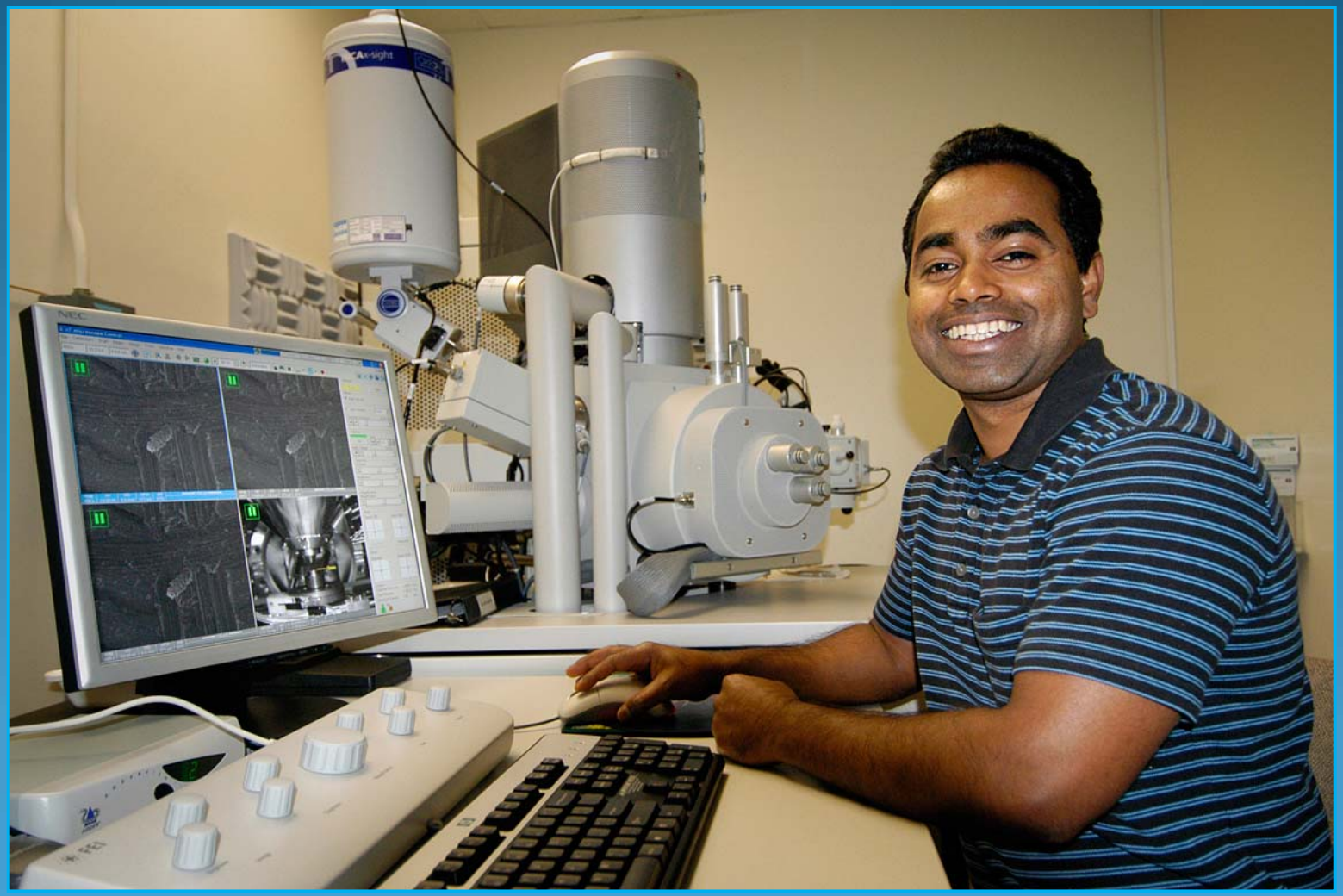
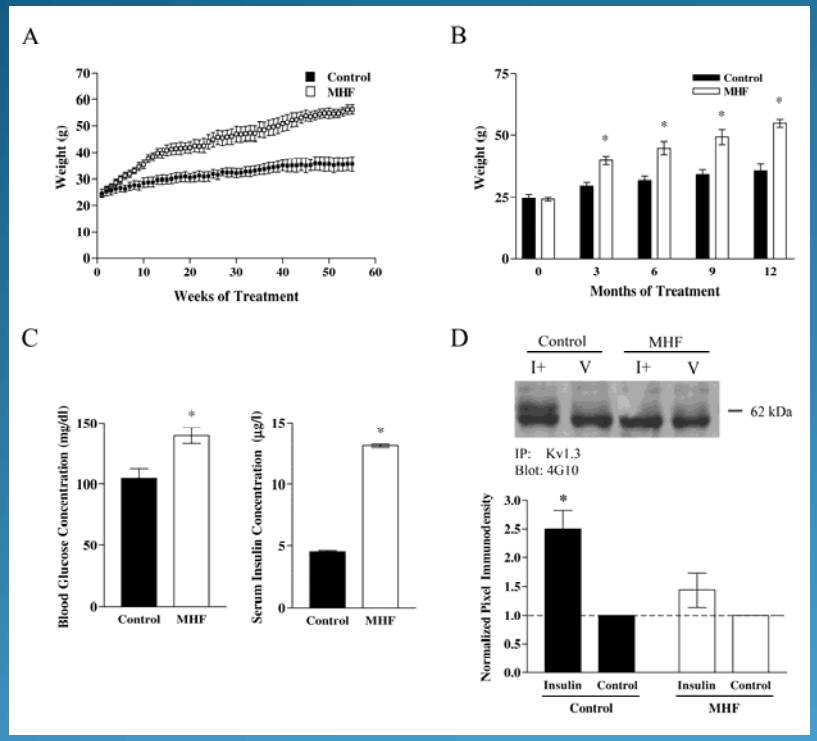
Oral Communication

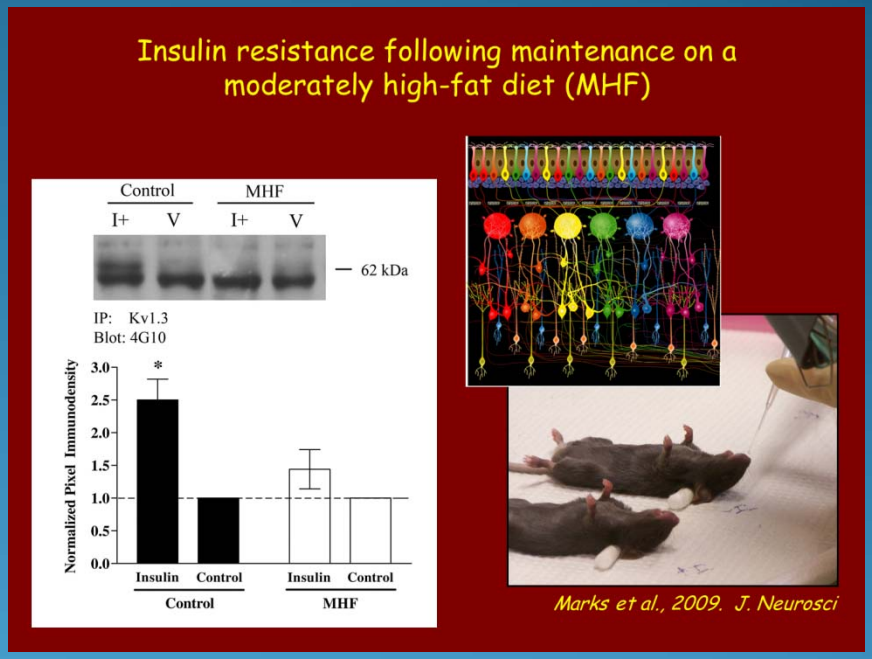


Oral Communication Strategies

1. Know your audience
2. Guide the intelligent people in front of you
3. There are many types of talks – know what is expected
    a. 12 minutes
    b. 50 minute research
    c. classroom teaching
    d. chalk talk
    e. International talks
4. How should you prepare?
5. How to you make transitions?
6. Never go over time
7. Best format for answering questions



Manuscript "Slide"



Presentation "Slide"

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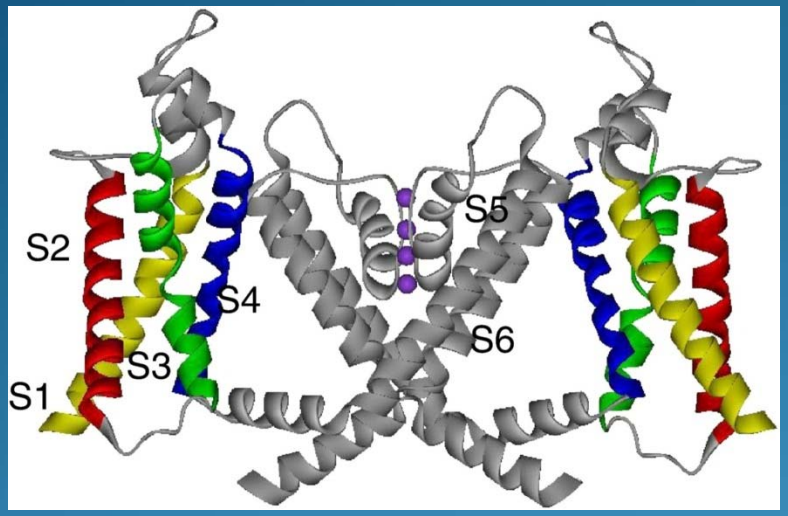
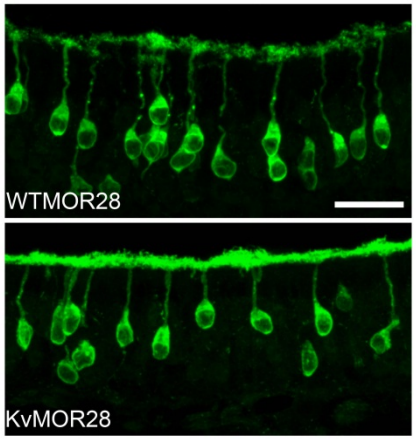
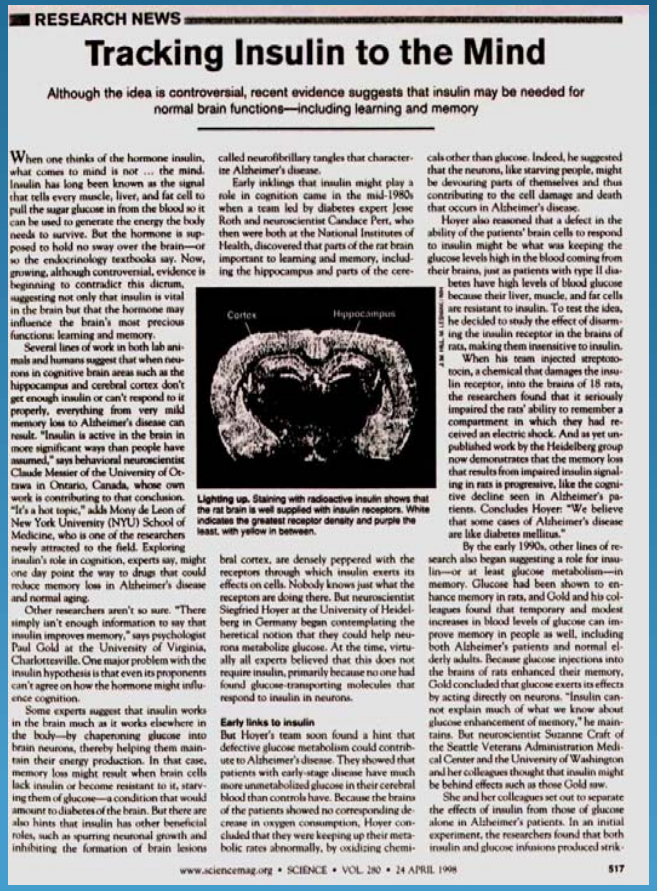
Find Your Niche



Potassium Ion Channel Camp



Chemical Senses Camp



RESEARCH NEWS

Tracking Insulin to the Mind

Although the idea is controversial, recent evidence suggests that insulin may be needed for normal brain functions—including learning and memory

When one thinks of the hormone insulin, what comes to mind is not ... the mind. Insulin has long been known as the signal that tells every muscle, liver, and fat cell to pull the sugar glucose in from the blood so it can be used to generate the energy the body needs to survive. But the hormone is supposed to hold no sway over the brain—or so the endocrinology textbooks say. Now, growing, although controversial, evidence is beginning to contradict this dictum, suggesting not only that insulin is vital in the brain but that the hormone may influence the brain's most precious functions: learning and memory.

Several lines of work in both lab animals and humans suggest that when neurons in cognitive brain areas such as the hippocampus and cerebral cortex don't get enough insulin or can't respond to it properly, everything from very mild memory loss to Alzheimer's disease can result. "Insulin is active in the brain in more significant ways than people have assumed," says behavioral neuroscientist Claude Messier of the University of Ottawa in Ontario, Canada, whose own work is contributing to that conclusion. "It's a hot topic," adds Mony de Leon of New York University (NYU) School of Medicine, who is one of the researchers newly attracted to the field. Exploring insulin's role in cognition, experts say, might one day point the way to drugs that could reduce memory loss in Alzheimer's disease and normal aging.

Other researchers aren't so sure. "There simply isn't enough information to say that insulin improves memory," says psychologist Paul Gold at the University of Virginia, Charlottesville. One major problem with the insulin hypothesis is that even its proponents can't agree on how the hormone might influence cognition.

Some experts suggest that insulin works in the brain much as it works elsewhere in the body—by chaperoning glucose into brain neurons, thereby helping them maintain their energy production. In that case, memory loss might result when brain cells lack insulin or become resistant to it, starving them of glucose—a condition that would amount to diabetes of the brain. But there are also hints that insulin has other beneficial roles, such as spurring neuronal growth and inhibiting the formation of brain lesions called neurofibrillary tangles that characterize Alzheimer's disease.

Early inklings that insulin might play a role in cognition came in the mid-1980s when a team led by diabetes expert Jesse Roth and neuroscientist Candace Pert, who then were both at the National Institutes of Health, discovered that parts of the rat brain important to learning and memory, including the hippocampus and parts of the cerebral cortex, are densely peppered with the receptors through which insulin exerts its effects on cells. Nobody knows just what the receptors are doing there. But neuroscientist Siegfried Hoyer at the University of Heidelberg in Germany began contemplating the heretical notion that they could help neurons metabolize glucose. At the time, virtually all experts believed that this does not require insulin, primarily because no one had found glucose-transporting molecules that respond to insulin in neurons.

Lighting up. Staining with radioactive insulin shows that the rat brain is well supplied with insulin receptors. White indicates the greatest receptor density and purple the least, with yellow in between.

Early links to insulin

But Hoyer's team soon found a hint that defective glucose metabolism could contribute to Alzheimer's disease. They showed that patients with early-stage disease have much more unmetabolized glucose in their cerebral blood than controls have. Because the brains of the patients showed no corresponding decrease in oxygen consumption, Hoyer concluded that they were keeping up their metabolic rates abnormally, by oxidizing chemicals other than glucose. Indeed, he suggested that the neurons, like starving people, might be devouring parts of themselves and thus contributing to the cell damage and death that occurs in Alzheimer's disease.

Hoyer also reasoned that a defect in the ability of the patients' brain cells to respond to insulin might be what was keeping the glucose levels high in the blood coming from their brains, just as patients with type II diabetes have high levels of blood glucose because their liver, muscle, and fat cells are resistant to insulin. To test the idea, he decided to study the effect of disarming the insulin receptor in the brains of rats, making them insensitive to insulin.

When his team injected streptozotocin, a chemical that damages the insulin receptor, into the brains of 18 rats, the researchers found that it seriously impaired the rats' ability to remember a compartment in which they had received an electric shock. And as yet unpublished work by the Heidelberg group now demonstrates that the memory loss that results from impaired insulin signaling in rats is progressive, like the cognitive decline seen in Alzheimer's patients. Concludes Hoyer: "We believe that some cases of Alzheimer's disease are like diabetes mellitus."

By the early 1990s, other lines of research also began suggesting a role for insulin—or at least glucose metabolism—in memory. Glucose had been shown to enhance memory in rats, and Gold and his colleagues found that temporary and modest increases in blood levels of glucose can improve memory in people as well, including both Alzheimer's patients and normal elderly adults. Because glucose injections into the brains of rats enhanced their memory, Gold concluded that glucose exerts its effects by acting directly on neurons. "Insulin cannot explain much of what we know about glucose enhancement of memory," he maintains. But neuroscientist Suzanne Craft of the Seattle Veterans Administration Medical Center and the University of Washington and her colleagues thought that insulin might be behind effects such as those Gold saw.

She and her colleagues set out to separate the effects of insulin from those of glucose alone in Alzheimer's patients. In an initial experiment, the researchers found that both insulin and glucose infusions produced strik-

Bridged via Neuromodulation
Clinical Health Impact

Getting Funded

1. Finding the best mechanism

NIH = R03 (7 years); K99R00 (5 years), F32/T32 (3 years total), R15 (AREA award).

Co-investigator or Co-PI

AHA , NSF, private foundations

2. Attend workshops on campus and professional conferences (Travel Awards July 1 and Jan 1)

3. Get a copy of a funded proposal and an unfunded proposal in your field for the mechanism for which you are applying.

4. A grant is 3x the work load (minimally) than a manuscript.

Getting Funded

5. When is the best time to submit?

6. Criteria for rating a grant application are generally published

7. Read the grant guidelines for good grantsmanship and new regulations

8. Clear strategies, good scientific writing, innovation of both thought, technologies, and analysis. What are the 4 Cs?

9. Same guidelines as a manuscript – to get pre-submission inquiry (Contact Program Officer!), to achieve internal feedback (What is a Mock Study Section?), and how to respond to reviewer comments (but cannot change "journals").

Thank you!

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http://opda.fsu.edu/

A. Travel Awards July 1 and Jan 1
B. Weekly Digest
C. Free memberships to NPA and versatile PhD

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