Writing and Presenting a Scientific Conference Abstract

DATE: December 9, 21 PRESENTED BY: Allison Fryer, PhD and Cynthia Morris, PhD
How to Write an Abstract

Allison Fryer, PhD
Associate Dean Graduate Studies
Abstracts are IMPORTANT INVITATION to read the rest of the paper.
After the title, the abstract is the second-most-read part of your article.
Think if it as a mini paper

Introduction

Purpose/Objectives/Why do the study?

Methods

Data/Results

Conclusion
It must stand alone
Antigen Sensitization Influences Organophosphorus Pesticide–Induced Airway Hyperreactivity

Bakry J. Proskocić,1 Donald A. Bruun,2 Jesse K. Lorton,3 Kirsten C. Blensly,1 David B. Jacoby,1,2 Pamela J. Lehn,2
and Allison D. Fryer1

1Department of Physiology and Pharmacology, 2Center for Research on Occupational and Environmental Toxicology, and 3Division of Pulmonary and Critical Care Medicine, Oregon Health & Science University, Portland, Oregon, USA

Introduction

Background: Recent epidemiologic studies have identified organophosphorus pesticides (OPs) as environmental factors potentially contributing to the increase in asthma prevalence over the last 25 years. In support of this hypothesis, we have demonstrated that environmentally relevant concentrations of OPs induce airway hyperreactivity in guinea pigs.

Objectives: Sensitization to allergen is a significant contributing factor in asthmatic patients, and recent studies have shown that sensitization changes virus-induced airway hyperreactivity from an eosinophil-independent mechanism to one mediated by eosinophils. Here, we determine whether sensitization similarly influences OP–induced airway hyperreactivity.

Methods: Non-sensitized and ovalbumin-sensitized guinea pigs were injected subcutaneously with the OP parathion (0.001–1.0 mg/kg). Twenty-four hours later, animals were aerosolized and ventilated, and bronchoconstriction was measured in response to exercise and substance-induced in vivo release of acetylcholine. Inflammatory cells and acetylcholine were collected immediately after physiologic measurements.

Results: Ovalbumin sensitization increased the threshold dose for parathion-induced airway hyperreactivity and exacerbated parathion effects on vagally induced bronchoconstriction in sensitized guinea pigs. Parathion did not increase the number of eosinophils in airways or the number of eosinophils associated with airway nerves nor did it alter eosinophil activation as assessed by major basic protein deposition.

Conclusions: Antigen sensitization increases vulnerability to parathion-induced airway hyperreactivity and changes the mechanism to one that is dependent on OP–IL-5. Because sensitization to allergen is characteristic of 50% of the general population and 80% of asthmatics (including children), these findings have significant implications for OP risk assessment, intervention, and treatment strategies.

Keywords: airway hyperreactivity, asthma, atopy, eosinophils, organophosphorus pesticides, parathion, sensitization.

Purpose/Why did you do the study?

Methods

Data

Conclusion
Dissecting an Abstract

Antigen Sensitization Underlies Organophosphorus Pesticide–Induced Airway Hyperreactivity

Becky J. Proskocil,1 Donald A. Black,2,3,4,5,6,7 Lorton,1 Kirsten C. Blensky,1 David B. Jacoby,1,8 Pamela J. Lehning,1,7,8

1Department of Physiology and Pharmacology, 2Center for Research on Occupational and Environmental Toxicology, and 3Division of Pulmonary and Critical Care Medicine, Oregon Health & Science University, Portland, Oregon, USA

BACKGROUND: Recent epidemiologic studies have identified organophosphorus pesticides (OPs) as potential environmental factors potentially contributing to the increase in asthma prevalence over the last 25 years. In support of this hypothesis, we have demonstrated that environmentally relevant concentrations of OPs induce airway hyperreactivity in guinea pigs.

OBJECTIVES: Sensitization to allergens is a significant contributing factor in asthma, and we have shown that sensitization changes virus-induced airway hyperreactivity from an eosinophil-independent mechanism to one mediated by eosinophils. Here, we determine whether sensitization similarly influences OP-induced airway hyperreactivity.

METHODS: Non-sensitized and ovalbumin-sensitized guinea pigs were injected subcutaneously with the OP parathion (0.001–1.0 mg/kg). Twenty-four hours later, animals were pretreated with antibody to interleukin (IL)-5, and hyperreactivity was measured in response to either vasoconstriction or intravenous acetylcholine. Inflammatory cells and acetylcholinesterase activity were assessed in tissues collected immediately after physiologic measurements.

RESULTS: Ovalbumin sensitization decreased the threshold dose for parathion-induced airway hyperreactivity and decreased the activity of acetylcholinesterase. Pretreatment with antibody to interleukin (IL)-5 prevented parathion-induced hyperreactivity in sensitized but not in non-sensitized guinea pigs. Parathion did not increase the number of eosinophils in alveoli or the number of eosinophils associated with airway nerves nor did it alter eosinophil activation as assessed by major basic protein deposition.

CONCLUSIONS: Antigen sensitization increases airway hyperreactivity by inducing eosinophils. The increase in airway hyperreactivity is dependent on the presence of IL-5, which activates eosinophils and increases the number of eosinophils in alveoli. These findings have significant implications for OP risk assessment, intervention, and treatment strategies.

KEY WORDS: airway hyperreactivity, asthma, vasoconstriction, eosinophils, parathion, sensitization, Environmental Health Perspect 116:381–388 (2008)
One goal, one point

state exactly what you did and how you did it

what do you want them to remember?
A Morphological and Genetic Analysis of *Polistes versicolor*: the Paper Wasp Invading the Galápagos Islands

- OHSU Student, OHSU Student, Faculty member
  OHSU, Portland Oregon

*Polistes versicolor*, a wasp native to Ecuador, has only recently invaded the Galápagos Islands. This invasion may have put us in position to explore evolution as it occurs, but only if we collect data as the invasion progresses. With preliminary evidence suggesting *P. versicolor* body characteristics vary with elevation, we gathered ecological, morphological, and genetic data during the early phase of this invasion. Individuals (n = 714) from the Ecuador mainland and six different island regions were collected in 2007 and 2008. Head, wing, and leg measurements were gathered. DNA was extracted and cataloged for each animal, PCR was performed on mainland and select island individuals, and loci were examined by polyacrylamide gel electrophoresis. We found significant morphological differences in relation to elevation. Data suggests that larger heads, smaller wings, and smaller legs are seen at higher elevations. Highly polymorphic loci have also been isolated for mainland individuals. Preliminary genetic data suggests that island-specific reductions in genetic diversity may have occurred and such limited variation supports morphological plasticity. These data will serve as a reference in morphological and genetic analyses over time to decipher whether plasticity or evolution is driving such differences.
Introduction

This covers what you were trying to achieve, e.g. to address an ongoing debate or problem, or some gap you found in the literature,

Keep in mind:

WHY should READER CARE?
Polistes versicolor, a wasp native to Ecuador, has only recently invaded the Galápagos Islands. This invasion may have put us in position to explore evolution as it occurs, but only if we collect data as the invasion progresses. With preliminary evidence suggesting P. versicolor body characteristics vary with elevation, we gathered ecological, morphological, and genetic data during the early phase of this invasion. Individuals (n = 714) from the Ecuador mainland and six different island regions were in 2007 and 2008. Head, wing, and leg measurements were gathered. DNA was extracted and cataloged for each animal, PCR was performed on mainland and select island individuals, and loci were examined by polyacrylamide gel electrophoresis. We found significant morphological differences in relation to elevation. Data suggests that larger heads, smaller wings, and smaller legs are seen at higher elevations. Highly polymorphic loci have also been isolated for mainland individuals. Preliminary genetic data suggests that island-specific reductions in genetic diversity may have occurred and such limited variation supports morphological plasticity. These data will serve as a reference in morphological and genetic analyses over time to decipher whether plasticity or evolution is driving such differences.
Objective

This is where you say what YOUR research covered.

This will be a narrower focus than what was covered in the background.

What specific question did YOU answer.
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Methods

Results
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Conclusion

This is what you concluded from your research, ie your ideas on what was happening or why it happened or how it relates to other research in the area.

*Note: not plural conclusions*
Conclusion

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One or two sentences providing a basic introduction to the field, comprehensible to a scientist in any discipline.

Two to three sentences of more detailed background, comprehensible to scientists in related disciplines.

One sentence clearly stating the general problem being addressed by this particular study.

One sentence summarising the main result (with the words “here we show” or their equivalent).

Two or three sentences explaining what the main result reveals in direct comparison to what was thought to be the case previously, or how the main result adds to previous knowledge.

One or two sentences to put the results into a more general context.

Two or three sentences to provide a broader perspective, readily comprehensible to a scientist in any discipline, may be included in the first paragraph if the editor considers that the accessibility of the paper is significantly enhanced by their inclusion. Under these circumstances, the length of the paragraph can be up to 300 words. (The above example is 190 words without the final section, and 250 words with it).
Am J Respir Cell Mol Biol. 2013 Feb 28. [Epub ahead of print]

TLR2/6 and TLR9 Agonists Suppress Viral Replication but not Airway Hyperreactivity in Guinea Pigs.

Drake MG, Evans SE, Dickey BF, Fryer AD, Jacoby DB.
Pulmonary and Critical Care, Oregon Health and Science University, Portland, Oregon, United States.

Abstract
Respiratory virus infections cause airway hyperreactivity (AHR). Preventative strategies for virus-induced AHR remain limited. Toll-like receptors (TLRs) have been suggested as a therapeutic target due to their central role in triggering antiviral immune responses. Prior studies have shown concurrent treatment with TLR2/6 and TLR9 agonists reduces lethality and microbial burden in murine models of bacterial and viral pneumonia. This study investigated the effects of TLR2/6 and TLR9 agonist pretreatment on parainfluenza virus pneumonia and virus-induced AHR in guinea pigs in vivo. Synthetic TLR2/6 lipopeptide agonist Pam2CSK4 and class C oligodeoxynucleotide TLR9 agonist ODN2395 given in combination 24 hours before virus infection significantly reduced viral replication in the lung. Despite a 5-fold reduction in viral titers, concurrent TLR2/6 and TLR9 agonist pretreatment did not prevent virus-induced AHR or virus-induced inhibitory M2 muscarinic receptor dysfunction. Interestingly, the TLR agonists independently caused non-M2-dependent AHR. These data confirm the therapeutic antiviral potential of TLR agonists, while suggesting virus inhibition may be insufficient to prevent virus-induced airway pathophysiology. Furthermore, TLR agonists independently cause AHR, albeit through a distinctly different mechanism from parainfluenza virus.
Follow Directions

every journal/meeting is different

Limits by space, words, characters, tables, figures text, references?

Is presenting author first, underlined?

Paragraphs indented, justified, headings?
Font ± serif and size?
76 M-Cholinoreceptors Mediate the Pressure Responses to Electrical and Chemical Stimulation of Aplysia in Conscious Saline

5. Oktay, N. Aslan, Z. Garcia, U. Oktay, F. Oktay. Departments of Pharmacology & Physiology, Marmara University School of Medicine, Haydarpasa, Istanbul, TURKEY.

The effects of electrical and chemical stimulation of the central nervous system of the amphipod crustacean Aplysia dorvillei (CNS) on mean arterial pressure (MAP) were investigated in conscious, unrestrained Aplysia dorvillei. The electrical effect of electrical stimulation of the central nervous system of the CNS on mean arterial pressure (MAP) was investigated in conscious, unrestrained Aplysia dorvillei. The electrical effect of electrical stimulation of the CNS on mean arterial pressure (MAP) was investigated in conscious, unrestrained Aplysia dorvillei. The electrical effect of electrical stimulation of the CNS on mean arterial pressure (MAP) was investigated in conscious, unrestrained Aplysia dorvillei. The electrical effect of electrical stimulation of the CNS on mean arterial pressure (MAP) was investigated in conscious, unrestrained Aplysia dorvillei. The electrical effect of electrical stimulation of the CNS on mean arterial pressure (MAP) was investigated in conscious, unrestrained Aplysia dorvillei. The electrical effect of electrical stimulation of the CNS on mean arterial pressure (MAP) was investigated in conscious, unrestrained Aplysia dorvillei. The electrical effect of electrical stimulation of the CNS on mean arterial pressure (MAP) was investigated in conscious, unrestrained Aplysia dorvillei. The electrical effect of electrical stimulation of the CNS on mean arterial pressure (MAP) was investigate...
Eosinophil Increase Neuron Branching in Human and Murine Skin and In Vitro

Erie L. Foster1, Eric L. Simpson2, Lorna J. Fredriksson3, James J. Lee4, Nancy A. Lee5, Allison D. Frye6, David B. Jacoby7

1Department of Molecular Microbiology and Immunology, Oregon Health & Science University, Portland, Oregon, United States of America; 2Department of Dermatology, Oregon Health & Science University, Portland, Oregon, United States of America; 3Department of Biochemistry, Mayo Clinic, Scottsdale, Arizona, United States of America; 4Division of Pulmonary and Critical Care, Department of Medicine, Oregon Health & Science University, Portland, Oregon, United States of America

Abstract
Cutaneous nerves are increased in atopic dermatitis, and itch is a prominent symptom. We studied the functional interactions between eosinophils and nerves in human and murine skin and in culture. We demonstrated that human atopic dermatitis skin has eosinophil granule proteins present in the same region as increased nerves. Transcriptomic microarray studies revealed that the number of genes was also significantly increased in the epidermis. In co-cultures, eosinophilic inflammatory pathways and increased branching of sensory nerves isolated from the dorsal root ganglia (DRG) of mice. This effect did not occur in DRG nerves cocultured with mast cells or with dead eosinophils. Physical contact of the eosinophil with the neurones was required, and the effect was not blocked by an antibody to nerve growth factor (NGF), eosinophil cationic protein (ECP), or IL-1β, which may be important in the recruitment, binding, and activation of eosinophils in the region of cutaneous nerves. These data indicate a pathophysiological role for eosinophils in cutaneous nerve growth in atopic dermatitis, and suggest they may present a therapeutic target in atopic dermatitis and other eosinophilic skin conditions with neuronal symptoms such as itch.

Key words: Cutaneous nerves, Neuron, Eosinophils, Itch, Atopic dermatitis

Introduction
Atopic dermatitis is a chronic inflammatory skin diseases characterized by erythema, hyperkeratosis, and pruritus. When aesthetic skin conditions are accompanied by symptoms of itch, the resultant skin diseases are referred to as atopic dermatitis (AD) or eczema. AD is a chronic inflammatory skin disease that affects a large proportion of the population, with an estimated prevalence ranging from 5% to 20% in Western countries and 1% to 3% in non-Western countries. The pathogenesis of AD is complex and involves a combination of genetic and environmental factors. Recent studies have shown that eosinophils play a role in the pathogenesis of AD.

Eosinophils are a type of immune cell that are primarily involved in the response to parasitic infections and allergic reactions. In the context of AD, eosinophils infiltrate the skin and produce a range of cytokines and chemokines that can contribute to the induction and maintenance of inflammation. Eosinophils are also known to release granule proteins that can damage skin cells and induce the release of histamine, a potent mediator of itching.

The role of eosinophils in the pathogenesis of AD has been extensively studied, and it has been shown that eosinophils are activated in the skin of patients with AD. The activation of eosinophils in AD is thought to be mediated by the release of cytokines, such as IL-33, which is produced by keratinocytes in response to damage or infection. Eosinophils are also known to be activated by the release of histamine, which is produced by mast cells in response to allergens.

The role of eosinophils in the pathogenesis of AD is likely to be multifaceted, and further research is needed to fully understand the mechanisms involved. However, the role of eosinophils in the pathogenesis of AD is an important area of research that has the potential to lead to new treatments for this common and chronic skin disease.


2. Copyright © 2017 Foster et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

3. Funding: This work was supported by NIH T23GM008510 (EB) and an OHSU T32 Fellowship to EB, and the May Foundation for Medical Education and Research, as well as National Institutes of Health Grant GM055226 (EB).

4. The authors have no competing interests to declare.

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References


2. Copyright © 2017 Foster et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

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Introduction
Atopic dermatitis is characterized by itching, which greatly affects the quality of life of patients [1, 2]. The itch often begins as early as infancy and may last throughout life. It can be a significant problem for patients with atopic dermatitis. Atopic dermatitis is a chronic inflammatory skin disease that affects a large proportion of the population, with an estimated prevalence ranging from 5% to 20% in Western countries and 1% to 3% in non-Western countries. The pathogenesis of atopic dermatitis is complex and involves a combination of genetic and environmental factors. Recent studies have shown that eosinophils play a role in the pathogenesis of atopic dermatitis.

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The role of eosinophils in the pathogenesis of atopic dermatitis has been extensively studied, and it has been shown that eosinophils are activated in the skin of patients with atopic dermatitis. The activation of eosinophils in atopic dermatitis is thought to be mediated by the release of cytokines, such as IL-33, which is produced by keratinocytes in response to damage or infection. Eosinophils are also known to be activated by the release of histamine, which is produced by mast cells in response to allergens.

The role of eosinophils in the pathogenesis of atopic dermatitis is likely to be multifaceted, and further research is needed to fully understand the mechanisms involved. However, the role of eosinophils in the pathogenesis of atopic dermatitis is an important area of research that has the potential to lead to new treatments for this common and chronic skin disease.
Special Considerations for Meeting/Abstracts.

- limit points to...1/ONE/a single.

-method include enough details to allay ethical concerns (IRB approval).
3. Steps to an Abstract
1. Write a lot

It is easiest to write a lot at the start.

Overcome ‘blank paper syndrome’ - write everything you want to say.
Hint: Start with Method and Data
Use sticky notes to list points you want to make, and then move them around.
Hint: TITLE
Write key words then shuffle them

PCR
ABSTRACT
research
TALK

How to write an Abstract: Important talk for Research Week
2. EDIT

Then edit harshly...very harshly. Do NOT be wed to your prose.

"Whatever you do, please don't edit yourself."
‘Wordiness’

O2 uptake was examined and found to vary considerably. O2 uptake varied considerably.

Scissors were sterilized prior to use. Scissors were sterilized, or Sterilized scissors were used.

We found a higher response in children compared to adults. Response was higher in children than in adults.
‘Dear Dr. Fryer,

We loved your paper containing 11 figures and 63 references. Unfortunately we have a limit of 6,000 words/paper and yours is currently over 13,000 words including figure legends. If you could please shorten the text ...we would be happy to publish it in the Journal of Irreproducible Results....’
Word Choice

• The cells were exposed to serum for 2 hours.
• After cutdown to an artery we removed the spleen.
• Clotting was changed in response to heparin.
• Serotonin was associated with an increase in the blood pressure.
• The change in CO2 was observed.
The Word Choice

• **The** cells were incubated with serum for 2 hours.
• After exposing an artery we removed **the** spleen.
• Clotting was decreased by heparin.
• Serotonin increased **the** blood pressure.
• **The** change in CO2 was measured.
No, no, if you make the paper too easy to read, everyone will know how you got the results!
The power of ‘but’

BUT -implies a difference or contrast:

Cardiac output was decreased BUT blood pressure was unchanged.

Growth factors caused all cells BUT nerve cells, to divide.
The power of ‘and’

AND does not imply difference.... it could be similar

Cardiac output AND blood pressure were decreased

Growth factors caused fibroblasts AND nerve cells, to divide.
“In SCID and RAG-2/- mice NK cells mediate a CHS response. Cutaneous hapten sensitization primes 2 distinct populations of CD8 T cells, to produce IFN-γ and IL-17, and elicit CHS. DNFB and Ox requires activation of both CD8 T cell populations within the AG challenge site. CD8 prime hapten-specific T cells in the vasculature of challenged sites are followed by activation that produces IL-17 and IFN-γ. Endo cells that have acquired and present hapten/MHC, activate CD8 T cells to produce cytokines after AG challenge, but it is important to note CD8 T cells do not penetrate the vascular barrier to infiltrate parenchymal tissue 1 and 8 hrs later. Both IL-17 and IFN-γ are required to stimulate Endo cells to produce CXCL1 and CXCL2, that direct
3. Seek an Outside Reader

IF YOU FOUND A MISTAKE, I DON'T WANT TO KNOW.

YOU MISSPELLED YOUR NAME.

NOT LISTENING! NOT LISTENING!

UH, OK.
Suggested Reading

**Essentials of Writing Biomedical Research Papers**
Second Edition

**The Elements of Style**
Fourth Edition

*“...still a little book, small enough and important enough to carry in your pocket, as I carry mine.”*
—Charles Osgood

Foreword by Roger Angell
Questions and Comments

WHAT IS IT?

YOU SAID TO DO AN ABSTRACT
You have 10 minutes.....

don’t screw it up
The challenge…

- This presentation is the culmination of years of hard work
- You represent your research group, department, university, practice, and most importantly, yourself.
- Don’t leave your presentation until you are on the plane en route to the meeting.
Step 1: Know your audience

- What do they know?
- What do you need to tell them?
- What do they expect?
- What will be interesting?
- What will keep them focused?
Step 2: Know your time limit

- 10 minutes with 5 minutes for questions
- Does not mean 15 minutes
Step 3: Know your message

- Every presentation should have one main message
- Be able to state your message concisely
- Know the exact take home point you want to get across
Step 4: Know yourself

- Ask yourself the tough questions
  - Does speaking in public make me nervous?
  - Do questions fluster me?
  - Am I easily distracted?

Preparation will help.
Tell the story of your data

- Every good story has a beginning (background, hypothesis), middle (methods), and end (results, conclusion).
- Do not take the audience down the same tortuous path your research has taken.
- Keep it simple.
Presentation philosophy

- Your slides should support your talk
- Your slides are not your talk
How many slides? One slide per minute

- Introduction - 1
- Hypothesis - 1
- Methods – 1-2
- Results – 4
- Conclusion – 1
- Limitations or future direction – 1
Title slide?

- On screen during your introduction
- Most meetings require disclaimers or conflict of interest slide
Introduction

- Your audience has general subject knowledge but summarize your area of research.
- Do not regurgitate everything you know about the topic.
- Reference other studies to place this in proper context.
Methods

- A schematic of your study is helpful.
- Give sufficient details to allow understanding of results.
- Define your population or model.
Results

- Show baseline or descriptive data.
- Make tables simple, highlight data.
- Bar graphs, pie charts, scatterplots are useful. Use basic colors.
- If you present numeric data in a bar graph, either state the means or note on bar.
Conclusions

- What message do you most want the audience to remember after the meeting?
- List 3 major conclusions at most
What next?

- Fend off your critics. Acknowledge the major limitations of the study.
- What is the likely consequence of this study?
- Make sure your audience knows when you are done.
Answering questions

- Be prepared for questions you anticipate
- Ask for clarification
- If you don’t know, be honest
- Answers should be short, to the point
- Inconspicuously jot down the question topic if you forget under stress.
Avoid audiovisual disasters

- Compose your slides
- Do not let design overpower message
- Do not copy/paste slides from different presentations
- Use a common simple background, common font
- Minimize animation
- Solid background, avoid the rainbow.
- Maximize contrast
  - Tiny print cannot be read.
- Use no smaller than 32 pt font.
- Spllllcheck.
- Use the 6x6 rule. No more than 6 lines, 6 words per line, 6 lines or bars on a graph.
- Create simple tables, graphs
Other suggestions

- No full sentences!
- Use bullet points
- Do not read slides
- Minimize UOA
- Do not crowd the slide
Write your talk, make your slides, practice

- Finish slides 2 weeks in advance.
- Write presentation word for word, craft concise sentences.
- Practice in front of colleagues who are not familiar with your study.
- Take criticism to heart and revise.
- Keep practicing. You cannot overprepare.
Get your timing down

- If you run overtime, your message is lost.
- Speak slowly. Pause on every slide to orient the audience.
- Too many words detract from your slide.
- Nothing is more distracting than gesturing wildly and talking rapidly.
On the day of your talk…

- Get to the meeting site early.
- Beware of software version mismatch, file size
- Familiarize yourself with the equipment.
- Arrive at the meeting room early, introduce yourself to the moderators, and watch the speakers before you.
Relax!

- You are well prepared and you know your study better than anyone in the audience.
- Talk to the audience, not your slides.
- Make eye contact
- If you are nervous, limit your use of the pointer.