CREATING POSTERS USING POWERPOINT

Ian Levstein, MS
Forensic Science Center
Updated, Fall 2013

Why a Poster?

- A poster is your opportunity to
  - make one point → loudly and clearly
  - stimulate interest and discussion
  - receive feedback on research
- A poster is not
  - a research paper stuck to a board

Poster Size

- Largest size slide that Powerpoint can create is 56″ x 56″

Display Board Size:	Optimum Powerpoint Slide Size:
None (free hanging)	56″ x 56″
72″ x 48″	56″ x 46″
48″ x 36″	46″ x 34″

Poster Size (cont.)

- Do not *ever* create a poster that exceeds the size of the slide
  - easy to do if you're not careful
- Doing so will initiate an unfortunate chain of events if/when converting to a PDF file

Transporting

- For auto/train:
  - poster tubes usually up to 36”
- For airplane:
  - PVC pipe with 2 end caps
- Don’t leave the poster in your car!
- Know the phone number of the last person leaving for the airport

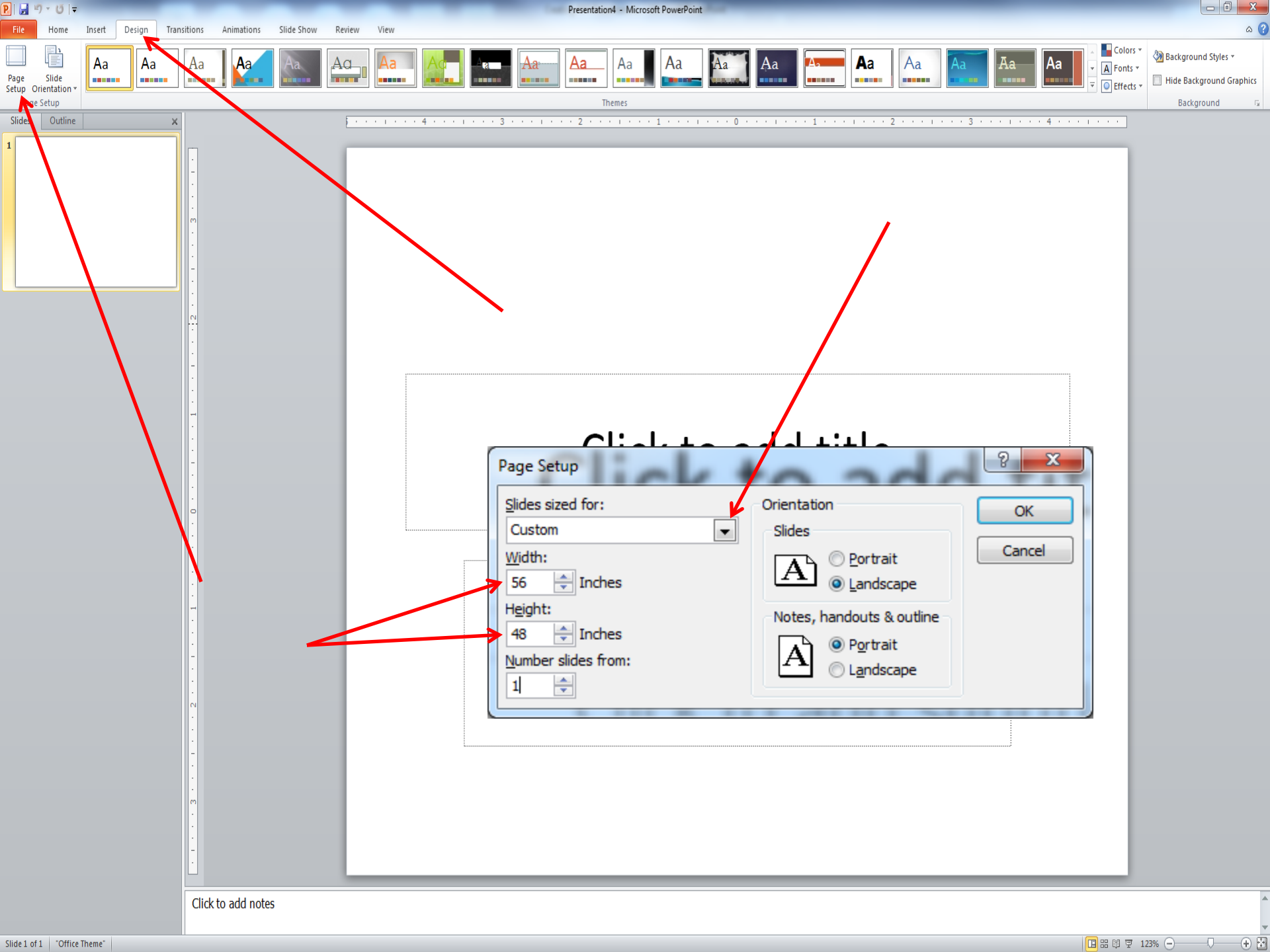
Administrative

- Find out in advance what the business needs in order to print successfully:
  - how much lead time do they need?
  - what version of Powerpoint do they use?
    - compatibility issues between versions?
    - what about reprints?
  - what are their business hours?
    - can they print on short notice if required?
  - do they trim the posters?

http://mimiandeunice.com/category/travel/

Getting Started

- You are going to create a single, large slide
- Start Powerpoint
  - create a new, blank slide
  - delete any existing text boxes
- Design → Page Setup
- Change width to 56" and height to 48" (or size of your choice), click OK


Presentation4 - Microsoft PowerPoint
File
Home
Insert
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Transitions
Animations
Slide Show
Review
View
Page Setup
Slide Orientation
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Colors
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Effects
Background Styles
Hide Background Graphics
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Slides
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Click to add title
Page Setup
Slides sized for:
Custom
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Number slides from:
1
Orientation
Slides
Portrait
Landscape
Notes, handouts & outline
Portrait
Landscape
OK
Cancel
Click to add notes
Slide 1 of 1
"Office Theme"
123%

Getting Started (cont.)

- Use built-in rulers/grids (View → Ruler)
- Most commercial large-format printers require a 2" margin on right hand side
- Allow lots of time to create

Text

- Text should already exist
  - don't try to edit and compose at the same time
  - several small text boxes are better than a single large one
  - can be resized
  - can be justified
- Be consistent
  - be consistent
    - be consistent

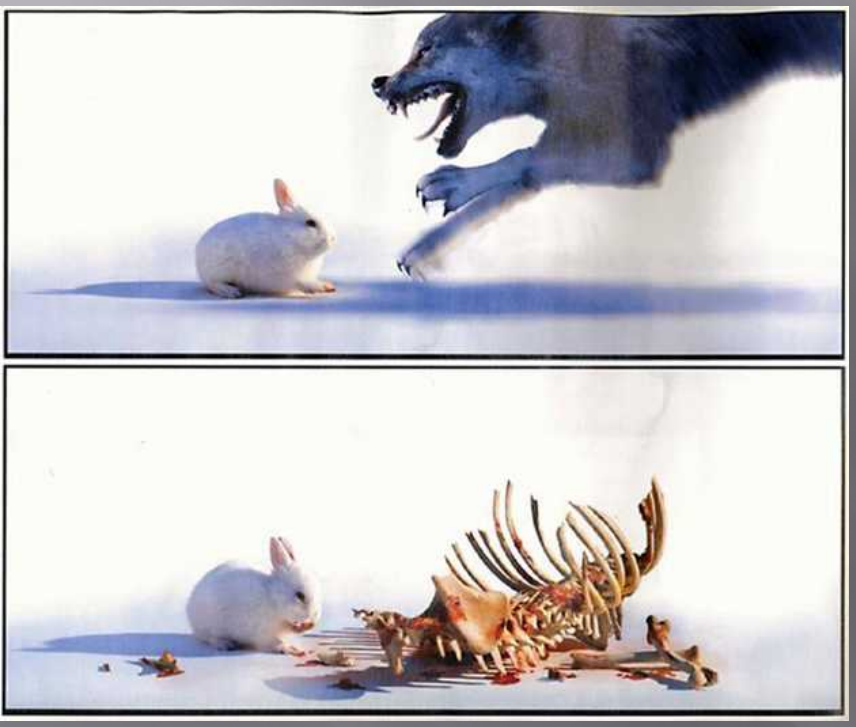
Images

- GIFs or TIFFs
  - uncompressed
  - even low quality images look good when large
- JPGs
  - compressed
  - low quality images look pixilated when enlarged
  - high resolution images may be okay

Images (cont.)

2″ margin on right is critical

Left margin is less critical



http://tvtropes.org/pmwiki/pmwiki.php/Main/KillerRabbit

This is fine:

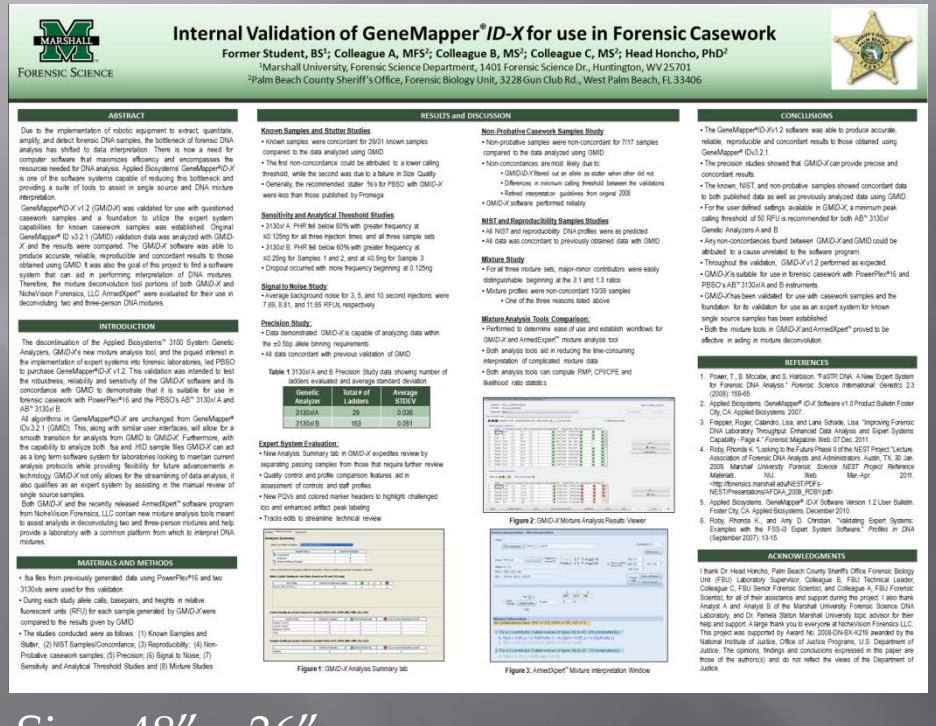
http://www.flickr.com/photos/obiwanjr/838737075/

Picture will compress and/or text will scroll

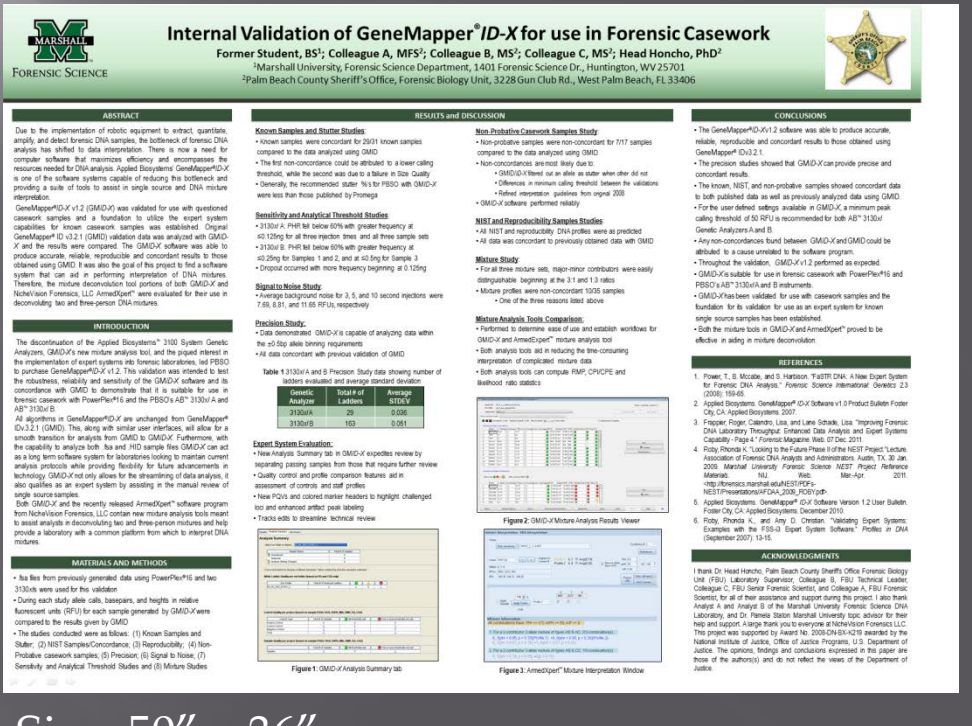
This is not:

Images (cont.)

- It's easy to add a 2" margin to your poster
  - create a new slide 2" wider
  - copy/paste from one slide to the other



Size: 48" x 36"



Size: 50" x 36"

Images (cont.)

- Always acknowledge your sources
- Images must already exist
  - beware of copyright issues
  - can be resized, rotated
  - high quality images
  - beware of screen captures
    - spelling mistakes (or big, scientific words) can not be changed

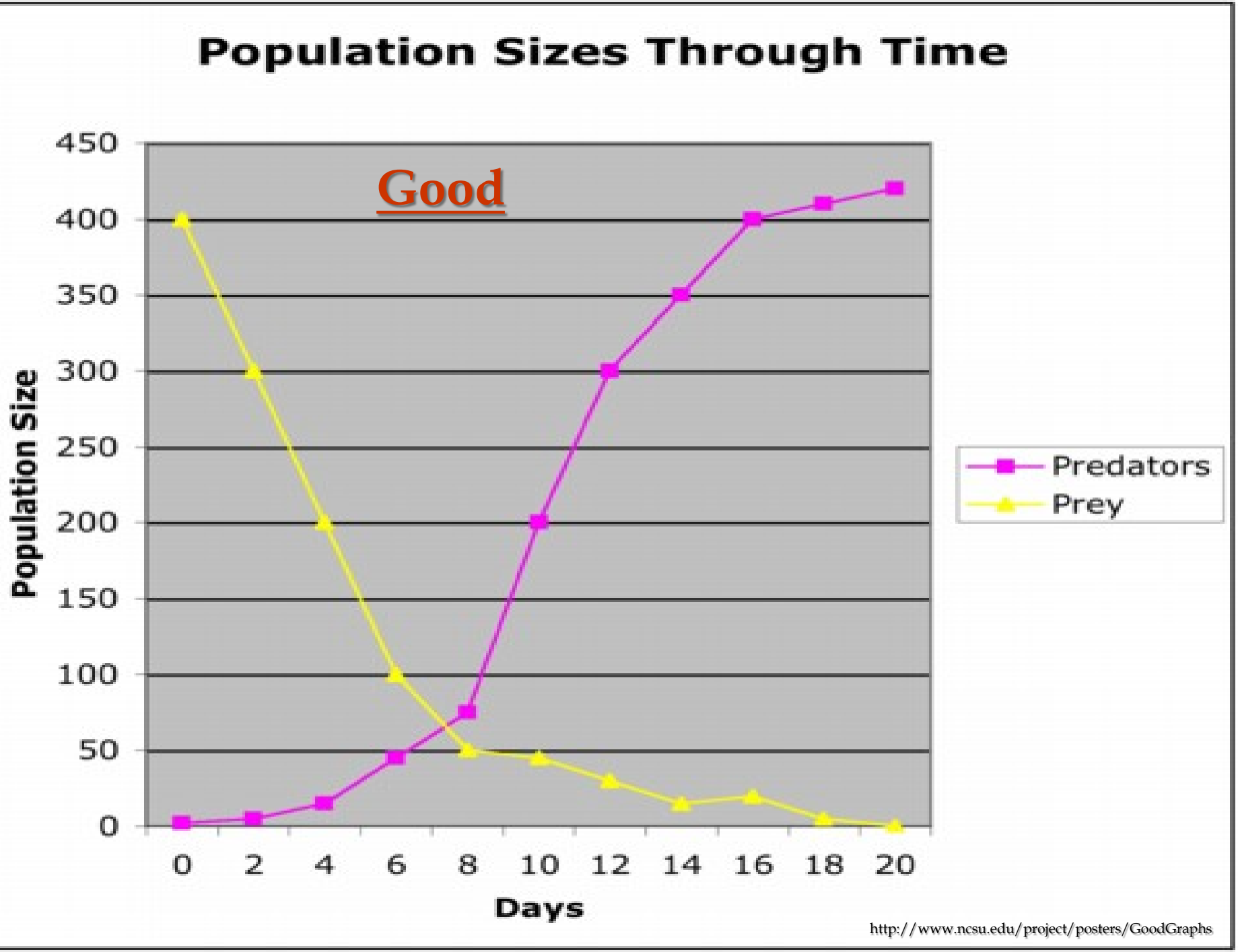
Spelling mistakes can't be changed when you copy/paste a screen capture

Candidate Gene Abbreviation and Name	SNP Frequency
AGT (angiotensinogen preprotein)	2
APOA (apolipoprotein A1, A4, and A5)	3
APOB (apolipoprotein B)	7
APOC2 (apolipoprotein C-II)	2
APOE (apolipoprotein E)	1
BHMT (betaine-homocysteine methyltransferase)	3
CBS (cystathionine beta-synthase)	3
CETP (cholesteryl ester transfer protein)	17
CRP (c-reactive protein)	1
F5 (coagulation factor V)	1
FOLH (folate hydrolase)	1
HDLBP (high density lipoprotein binding protein)	1
HNF4A (hepatocyte nuclear factor 4, alpha)	2
LCAT (lecithin: cholesterol acyltransferase)	1
LDLR (low density lipoprotein receptor)	1
LIPC (lipase, hepatic)	2
LIPG (lipase, endothelial)	1
LPL (lipoprotein lipase)	2
LRP1 (low density lipoprotein-related protein 1)	2
LRP1B (low density lipoprotein-related protein 1B)	7
LRP5 (low density lipoprotein receptor-related protein 5)	7
LPR8 (low density lipoprotein receptor-related protein 8)	4
MPO (myeloperoxidase)	2
MSR1 (macrophage scavenger receptor 1)	1
MTR (5-methyltetrahydrofolate-homocysteine)	1
MTRR (methionine synthase reductase)	1
NOS3 (nitric oxide synthase 3)	3
PLTP (phospholipid transfer protein)	2
PON (paraxonase)	8
PPARA (peroxisome proliferative activated receptor)	7
Total: 30	**96**

It will print exactly as it shows up…
red squiggly lines included

Graphs

- Focus on your data
  - keep graphs clean and simple
- Being exact and specific has its place
  - but it's better to get the message across
- No distractions
  - your message will come through loud and clear


Population Sizes Through Time
Good
Population Size
450
400
350
300
250
200
150
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0
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2
4
6
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12
14
16
18
20
Days
Predators
Prey
http://www.ncsu.edu/project/posters/GoodGraphs


Population Sizes Through Time
Better
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450
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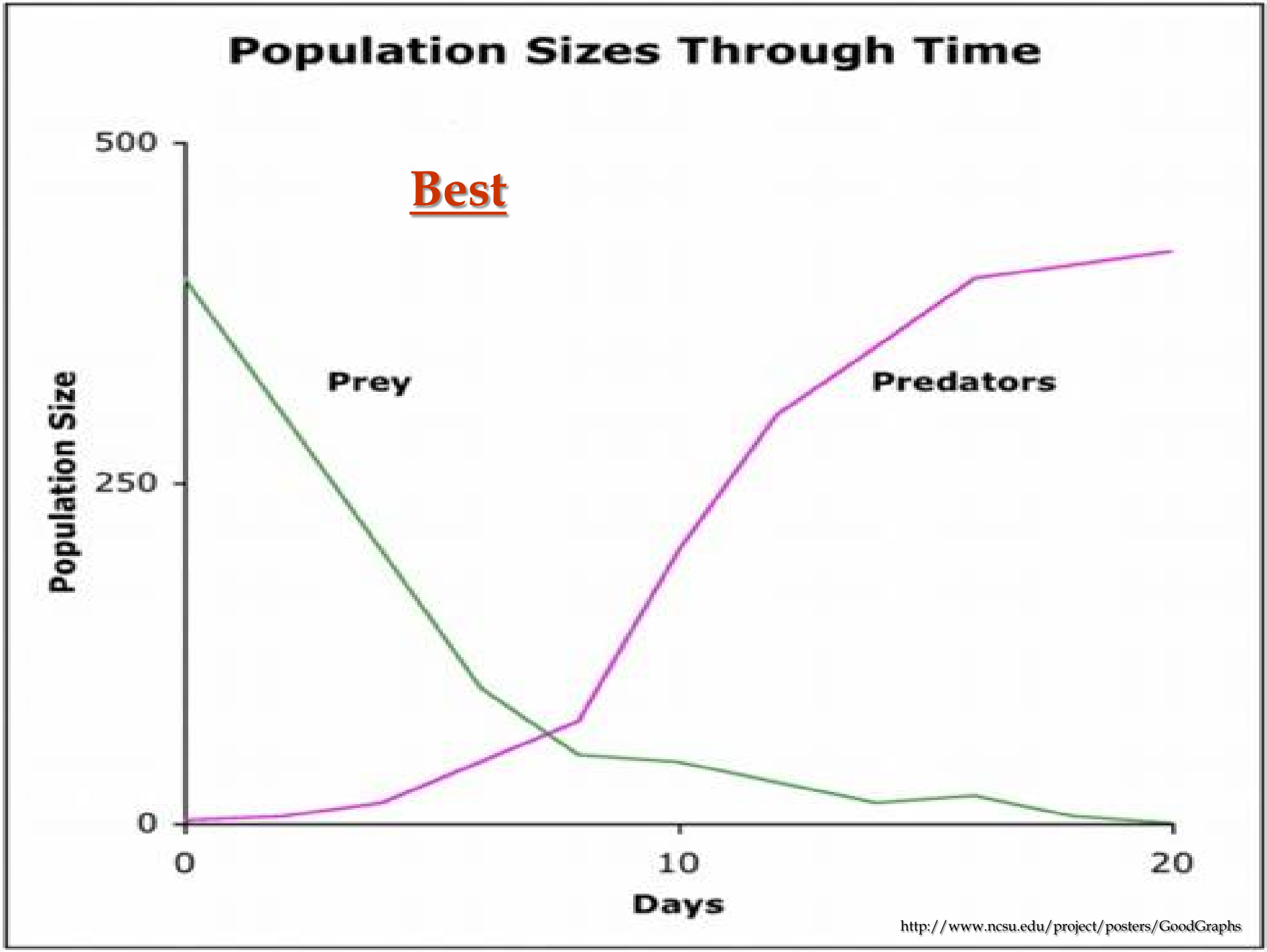

Population Sizes Through Time
Best
500
250
0
Population Size
Prey
Predators
0
10
20
Days
http://www.ncsu.edu/project/posters/GoodGraphs

Composition

- Font size is font size
  - 20 pt TNR is 20 pt TNR
  - don't be fooled by apparent size
    - on monitor
    - test print on desktop printer

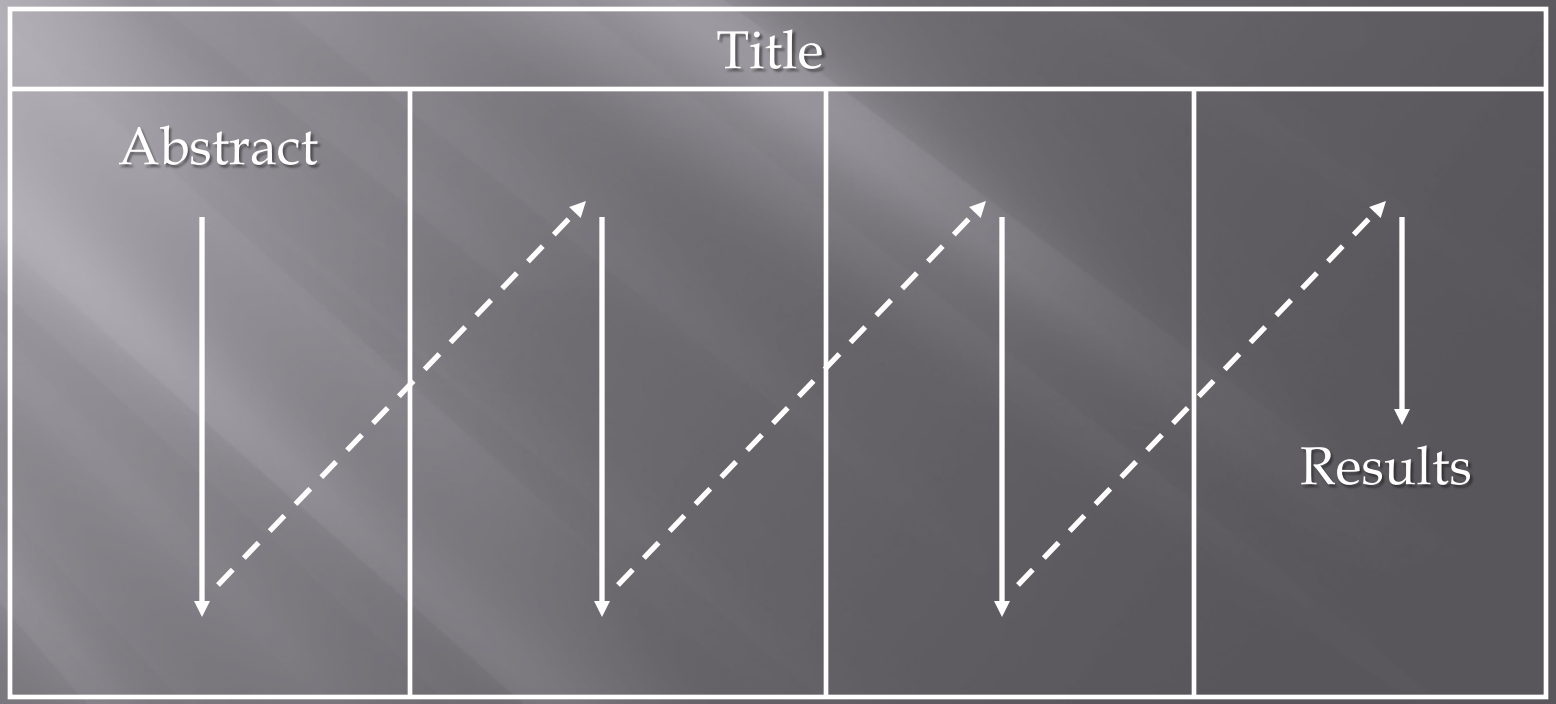
Composition (cont.)

- Calibri is better for computer monitor
  - Powerpoint default

- TNR is better for paper, therefore poster
  - Titles: 80 – 96 pt
  - Authors: 54 – 60 pt; Address: 40 – 48 pt
  - Section headings: 36 – 40 pt
  - Text, References: 28 – 32 pt
  - Acknowledgments: 18 – 24 pt

Composition (cont.)

- Sketch your layout before you start



Backgrounds

- Do:
  - use native PPT background
  - keep backgrounds simple
    - solid color or gentle graphic
- Do not:
  - use backgrounds that fade from light/dark
    - very distracting
  - use a photo
    - often creates unpredictable results

Weight Loss Outcomes With High Protein, Low Carbohydrate, Unlimited Calorie Diet

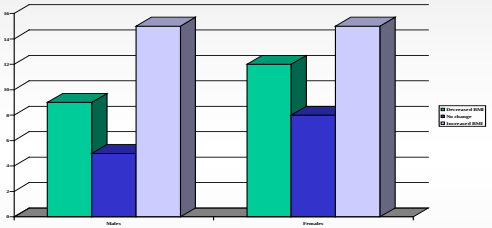
Department of Pediatrics, Joan C. Edwards School of Medicine, Huntington, West Virginia

Introduction: Childhood obesity is currently a national epidemic. The prevalence of childhood overweight (BMI>85%) and obesity (BMI>95%) has doubled in the last 20 years. Traditional interventions with a low fat diet and exercise have been unsuccessful for weight reduction.

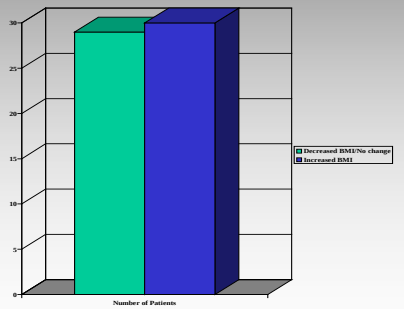
Objective: The purpose of our study was to determine the change in BMI in overweight children referred to the endocrine clinic managed with a high protein, low carbohydrate, unlimited calorie diet for six months.

Methods: A retrospective chart review was conducted of all patients referred to the pediatric endocrine clinic over a six year time period with the diagnosis of overweight and obesity. Initial dates of visit, height, and weight were recorded from each patient and on follow up visit six months later. Each patient was counseled on a high protein, low carbohydrate, unlimited calorie diet. BMI was calculated for the initial visit and the follow up visits. Patients with a diagnosis of diabetes mellitus or in which accurate weight could not be assessed were excluded.

Results: A total of 59 patients aged 6-18 years participated in the study. Patients were included in the study if follow up data was available 6 months after the initial visit. 20 (36%) of 59 patients had a decrease in BMI at the follow up visit. 8 (13%) patients had no change in BMI and 30 (51%) patients had an increase in their BMI. Average BMI decrease in those patients with weight loss was 2.76 points (SD 4.85). Average weight loss in the 21 patients with a drop in BMI was 10.3 pounds (SD 13.5).



Not Good



Discussion: The prevalence of overweight and obesity is increasing in the US. The prevalence of obesity has doubled in the past 20 years. Currently, about 15 % of children ages 6-19 are obese. This trend is seen in the US and other developed countries. Increased rates of obesity are also seen in minorities and disadvantaged children. Body mass index (BMI), the ratio of the weight in kilograms to the square of the height in meters, correlates well with body fatness and other obesity related conditions. Our study aimed to measure the weight loss and decrease in BMI in patients on a high protein, low carbohydrate, and unlimited calorie diet. Their primary care physicians referred patients for obesity. A retrospective chart review was done on over 200 chart of patients referred to the clinic. Follow-up data was available on 59 patients. More than one-third of patients decreased their BMI in this study. The average BMI decrease was 2.76 points (SD 4.85) and 10.3 pounds (SD13.5 pounds). This data is in comparison to the very poor success rates in traditional low calorie, low fat diets. In conjunction with exercise, patients on the high protein, low carbohydrate, unlimited calorie diet can successfully lose weight. Pediatric patients can then use this weight loss as a stepping-stone to a healthier more active lifestyle that includes better food choices for the long term.

Conclusion: We have demonstrated a high protein, low carbohydrate, unlimited calorie diet is effective in maintaining or reducing BMI and weight over a six month time period in 49% of overweight children. This approach has been more effective than the traditional low calorie diet with exercise at our institution.

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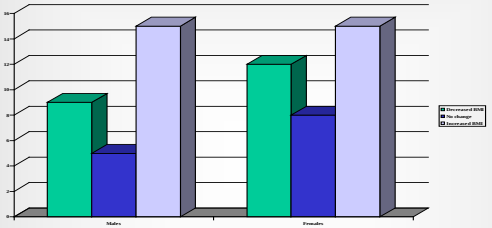

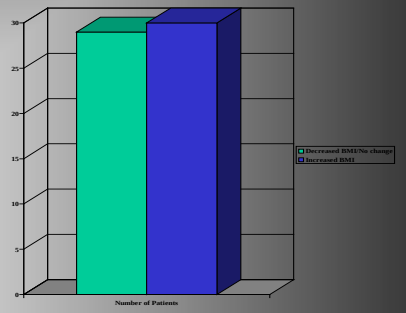

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Conclusion: We have demonstrated a high protein, low carbohydrate, unlimited calorie diet is effective in maintaining or reducing BMI and weight over a six month time period in 49% of overweight children. This approach has been more effective than the traditional low calorie diet with exercise at our institution.

Not Good

Chmp1, a Strabismus binding protein, plays multiple roles in vertebrate convergent extension movements and tumor formation

*Biochemistry and Microbiology Dept., School of Medicine, Marshall University, Huntington, WV,
#Pharmacology Dept., HHMI, University of Washington, Seattle WA, ^Zoology Dept., University of Washington, Seattle WA

Not Good

ABSTRACT

Strabismus (Stbm) modulates convergent extension movements during gastrulation in vertebrates as a component of the Planar Cell Polarity (PCP) pathway. Chromatin modifying protein 1 (Chmp1) was identified as a Stbm binding protein by using a yeast two-hybrid screen. Chmp1 protein is highly conserved throughout evolution and belongs to a family of vesicle trafficking proteins (Charged Multivesicular body Protein). However, the function of Chmp1 in vertebrate development as well as its signaling activities have barely been investigated.

We have confirmed the interaction between Stbm and Chmp1 and have shown the co-localization of Stbm and Chmp1. We also demonstrated that functionally Chmp1 regulated convergent extension movements in zebrafish similar to Stbm. Chmp1 function in gastrulation movements appears to be the consequence of the activation of AP-1 complex via PCP pathway.

In addition, we have shown that Chmp1 induced hyperplasia in zebrafish when its function was mis-regulated. In NIH 3T3 cells, exogenous Chmp1 induced the inhibition of cell growth. Subsequently, NIH 3T3 cells were transformed to grow anchorage-independently when Chmp1 was knocked-down by siRNA. The data from NIH 3T3 cells indicated that Chmp1 may function as a tumor suppressor.

We established the potential tumor suppressor function of Chmp1 by screening human cancer profiling arrays and human tumor tissue arrays. Chmp1 mRNA was specifically reduced in various pancreatic tumor samples compared to corresponding normal samples. Besides, Chmp1 protein was also absent in the acinar cells of pancreatic tumors.

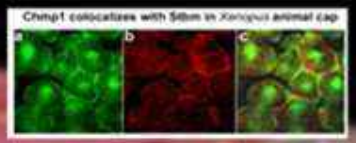
INTRODUCTION

Previously we have reported that Strabismus (Stbm), a component of PCP (Planar Cell Polarity) pathway, regulates convergent extension movements in vertebrates. We demonstrated that Stbm interfered with gastrulation movements by gain-of-and loss-of function assay. Mechanistically, Stbm was shown to inhibit β-Catenin mediated Wnt signaling activity. In addition, Stbm activated c-Jun mediated AP-1 reporter and phosphorylated c-Jun. Based on the data, we hypothesized that Stbm, by interacting with Dsh, activates c-Jun signaling pathway to regulate convergent extension movements.

Using a yeast two-hybrid approach we identified Chmp1 (Chromatin Modifying Protein 1/ Charged Multivesicular body Protein) as a Stbm associated protein. Chmp1 was reported to silence gene activation by interacting with a transcriptional repressor Polycomb-group (PcG) protein, Polycomblike (Pcl). Over-expression studies demonstrated that the gene silencing of Chmp1 was due to its effect on the chromatin structure and cell cycle progression. Chmp1 was also shown to physically associate with multivesicular sorting protein, SKD1/VPS4 (vacuolar protein-sorting 4). Maize homolog of Chmp1 named SAL1 (supernumerary aleurone layers 1) was also identified. SAL1 was shown to be involved in regulating the formation of aleurone cell layer in maize grains. Mutation in *sal1* results in more aleurone cell layers suggesting that SAL1 might play a role in growth. Taken together, these studies indicate that Chmp1 may play a role in vertebrate development by controlling cell growth and receptor activity.

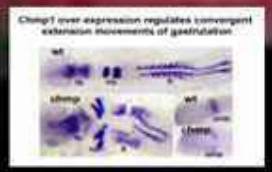
We cloned Chmp1 from zebrafish as a Stbm associated protein using a yeast two-hybrid screen. Similar to Stbm, Chmp1 regulated convergent extension movements of gastrulation by activating AP-1 complex, a down stream targets of the PCP pathway. Anti-sense Morpholino-mediated Chmp1 knockdown induced abnormal cell growth in zebrafish.

In addition, Chmp1 knockdown by siRNA rendered NIH 3T3 cells to grow in an anchorage independent manner. These knockdown assays indicated that Chmp1 might function in human tumor development. To test this hypothesis, we measured Chmp1 mRNA and protein level in cancer tissues samples and compared with its level in corresponding normal tissues. Chmp1 mRNA was strongly reduced in various pancreatic tumor tissues when compared with corresponding normal tissues. Furthermore, Chmp1 protein was nearly absent from the acinar cells of pancreatic tumor tissues. Taken together, our study indicates that Chmp1 function in convergent extension movements and tumor development in vertebrates.



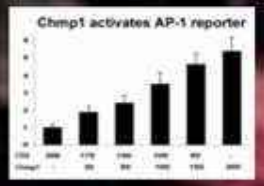
As seen in the third lane, Chmp1 associates with Stbm in vivo. Immunoprecipitation (IP) with HA-tagged Chmp1 and Western Blotting for Myc-tagged Stbm. First lane is a Stbm input, and second lane is a control.

GFP-tagged Chmp1 protein (green) is localized to the cytoplasm, membrane, and nucleus (a). Endogenous Stbm protein (red) is localized at the membrane (b). Chmp1 co-localizes with Stbm at the membrane (yellow) as can be seen in c.



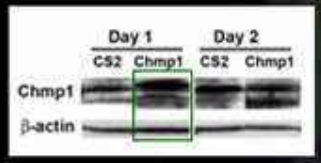
Flatmount zebrafish embryos, wild type (wt), and Chmp1 mRNA injected (chmp). Forebrain, hindbrain and somites are visualized with OTX-1, Krox-20, and MyoD respectively. Chmp1 over-expressed embryo has a shorter and wider body axis with open neural tube (*)

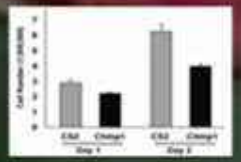
Shown at s1 and s2 are the same embryo, light and fluorescent (GFP) microscopic analysis respectively. GFP-tagged Chmp1 is expressed in the tissue (arrow). The hyperplasia found at the dorsal side of the embryo increases as the embryo grows older, shown in arrows (h2 is the same embryo in b1, a day after).



Luciferase reporter assay showing Chmp1 plasmid activates AP-1 in a dose dependent manner in NIH 3T3 cells.

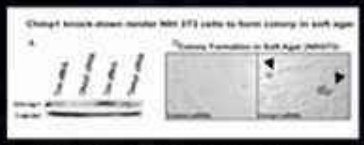
Chmp1 phosphorylates c-Jun at serine 73 in NIH 3T3 cells. GST vector is transfected as a control. The level of gapdh is blotted to monitor protein loading.



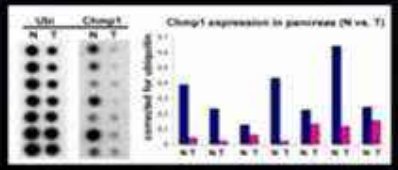


Chmp1 over-expression upon transfection is shown in NIH 3T3 cells (green box). CS2 vector is transfected as a control. β-actin is used as a loading control.

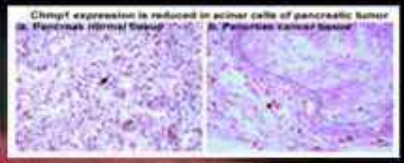
Chmp1 over-expression induces growth inhibition in NIH 3T3 cells. An average was taken from three separate experiments.



Chmp1 siRNA knocks-down Chmp1 protein and β-actin is used as a loading control (a). NIH 3T3 cells are transformed to form colonies (arrows) in soft agar when Chmp1 is knocked-down by siRNA (b). Control siRNA delivered NIH 3T3 cells do not form colonies.



Chmp1 mRNA is strongly reduced in pancreatic tumors. Clontech Cancer Profiling Array II hybridized with human ubiquitin (Ubi, control) and Chmp1 cDNA probe demonstrates the reduction of Chmp1 mRNA in various pancreatic tumors. Densitometric analysis of array exhibits 1.5 to 20 folds reduction of Chmp1 mRNA in pancreatic tumors.



Chmp1 protein is detected absent from the acinar cells of pancreatic tumors. Chmp1 protein is localized to the nucleus of the acinar cells of pancreas (arrow). In contrast, Chmp1 protein expression is detected absent from the acinar cells (dotted area) even though its expression in connective tissue is evident (*).

Conclusions

- Chmp1 binds and co-localizes with Stbm.
- Chmp1 regulates convergent extension movements.
- Chmp1 mis-regulation induces hyperplasia in zebrafish.
- Chmp1 over-expression inhibits growth in NIH 3T3 cells.
- Chmp1 knockdown results in anchorage-independent colony formation.
- Chmp1 mRNA and protein expression is highly reduced in pancreatic tumors.

This Research has been supported in part by NIH COBRE (Center for Biomedical Research Excellence, RR020180-02) and WV Bio Pointer Science and Technology Research Challenge Fund (EPS-01-MURC-3).

Backgrounds (cont.)

- Avoid:
  - burst images
  - strongly mottled images
  - wild colors
- Remember: simple can be striking!


Not Good

Program Number: 937.24

Nitric Oxide and Diet-Induced Hypertension in the Obese Zucker Rat

Departments of [a]Physiology and [d]Cardiovascular Services, Joan C. Edwards School of Medicine at Marshall University, [b]Wheeling Jesuit University, [c]West Liberty State College

Not Good

Abstract

We have shown that obese Zucker rats on a high fat diet develop hypertension when saline is added to their drinking water whereas lean Zucker rats do not. The objective of this study was to determine whether the production of nitric oxide (NO), a potent vasodilator involved in blood pressure regulation, is altered by similar dietary manipulation. Eight-week-old female lean and obese Zucker rats were fed a diet high in fat content or standard rat chow (control diet) for 10 weeks. From week 4 through week 10, 1 % NaCl was added to their drinking water. Urinary excretion of NO metabolites (NO_2^- and NO_3^-) was measured at weekly intervals. The excretion of NO metabolites was dramatically reduced within a week of placing the rats on a high fat diet and continued to decline over time, suggesting that the high fat diet may be suppressing NO production. We conclude that this suppression of NO production may be a factor in limiting the ability of the obese rat to maintain its blood pressure when challenged with a salt load.

Introduction

Obesity is a well-known risk factor for a wide variety of diseases including hypertension, type II diabetes, and coronary heart disease (Pi-Sunyer 1993). Consequently, the medical importance of this condition has rapidly escalated in tandem with its prevalence in developed countries throughout the world (World Health Organization 2000).

High dietary fat intake has also been independently associated with hypertension and other pathological conditions; however, the link between high fat diets and hypertension has received much less attention than the relation between obesity and hypertension. This disparity is due in part to a very close and confounding relationship between high fat diets and weight gain, which makes it difficult to isolate the direct hypertensive effects of a high fat diet from indirect effects that are mediated through the metabolic alterations of obesity per se. Nonetheless, high fat diets have been shown to induce insulin resistance independent of obesity in studies of metabolic syndrome X (Bernard 1998, Vessby 2003). Moreover, results of the DASH trials have indicated that low fat diets reduce blood pressure regardless of body weight, although the effect is more pronounced when accompanied by weight loss (Bacon 2004).

One objective of the current study was to determine whether a high fat diet directly promotes the development of hypertension in obese rats. This was done by assessing the effects of a high fat diet on the blood pressure of lean and obese rats before and after they were subjected to an increased salt load. Because NO plays an important role in blood pressure homeostasis, changes in NO production by the different experimental groups were assessed to determine whether alterations in NO production could be involved in the mechanism leading to blood pressure changes induced by the experimental design.

Table 1. Gram percentage of macronutrient components for the control and high fat diets

Diet	Control	High Fat
Protein	23.4	18.5
Carbohydrate	48.1	56.7
Fat	4.5	15.6

Figure 1. NO metabolite excretion in lean control (LC), lean high fat (LHF), obese control (OC), and obese high fat (OHF) experimental groups on week 8. N=9 for lean groups and N=8 for obese groups. *P < 0.05 versus phenotypic controls. +P < 0.05 versus dietary controls.

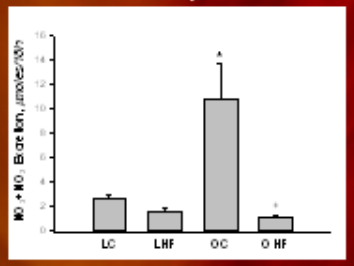


Figure 2. Blood pressures of obese (A) and lean (B) Zucker rats on either a high fat or control diet. The arrow just after week 0 indicates when the high fat diet was initiated in high fat groups. The arrow after week 4 indicates when the all groups began drinking 1% saline. N = 16 for obese groups and N=18 for lean groups. *P < 0.05 versus same week dietary controls.

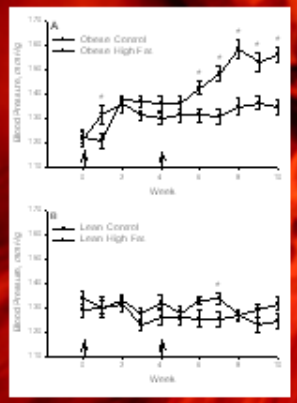


Figure 3. Plasma leptin (A) and insulin (B) concentrations in lean control (LC), lean high fat (LHF), obese control (OC), and obese high fat (OHF) experimental groups after 10 weeks. *P < 0.05 vs. phenotypic controls. N=9 for lean groups and N=8 for obese groups. +P < 0.05 vs. dietary control.

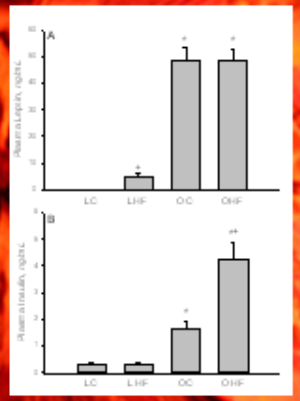


Table 2. Metabolic and cardiovascular characteristics of lean and obese Zucker rats fed a high fat or regular diet. For each lean and obese group, n=9 and n=8, respectively. Values are given in means ± SE. Lee's index of obesity* is the cube root of body weight (g) divided by nasoanal length (cm). Estimated cumulative energy intake* (CEI), changes in body weight, and energy efficiency ratio* (EER) express proclivity towards weight gain. Measurements are significant ($P<0.05$) when compared to phenotypic[#] and dietary[+] controls.

	Lean		Obese	
Parameter	High Fat	Regular	High Fat	Regular
Estimated CEI (MJ/10 wk)	24.1 ± 3.3	24.5 ± 2.0	32.5 ± 4.0[#]	35.1 ± 3.4[#]
Change in Body Weight (g/10 wk)	80.4 ± 8.4	66.1 ± 2.4	148.9 ± 17.5[#]	125.5 ± 11.3[#]
Estimated EER* (g/MJ)	3.31 ± 0.23	2.71 ± 0.12	4.50 ± 0.43[#]	3.53 ± 0.22[#]
Final Body Weight (g)	259 ± 10	247 ± 4	445 ± 18[#]	424 ± 21[#]
Nasoanal Length (cm)	21.2 ± 0.2	21.2 ± 0.2	21.9 ± 0.1[#]	21.7 ± 0.3
Lee's Index of Obesity*	.301 ± .003	.295 ± .002	.350 ± .005[#]	.347 ± .005[#]
Heart Weight (mg)	914 ± 22	872 ± 32	1367 ± 76[#+]	1094 ± 25[#]
Triglycerides (mg/dL)	141 ± 9	130 ± 12	2189 ± 588[#]	2200 ± 945[#]

Summary

- On a standard diet, NO production by obese rats is greater than by lean rats.
- When obese rats are fed a high fat diet, NO production is markedly suppressed compared to obese rats on the diet of standard rat chow.
- Obese Zucker rats on a high fat diet develop hypertension when subjected to a salt load. In contrast, obese rats do not develop hypertension when subjected to the same salt load.
- Lean rats do not develop hypertension when subjected to the same experimental conditions that promote salt-sensitive hypertension in obese rats.

Conclusions

- Obesity is a risk factor for the development of salt sensitive hypertension
- The suppression of NO production may promote the development of salt-sensitive hypertension in obese rats

Acknowledgments

This research was supported in part by grant # P20 RR016477 from the National Center for Research Resources awarded to the West Virginia Biomedical Research Infrastructure Network and by the Marshall University Joan C. Edwards School of Medicine Cardiovascular Research Support Fund.

References

Bacon SL, Sherwood A, Hinderliter A, Blumenthal JA. Effects of exercise, diet and weight loss on high blood pressure. Sports Med. 2004;34(5):307-16. Review. PMID: 15107009

Bernard RJ, Roberts CK, Varon SM, Berger JJ. Diet-induced insulin resistance precedes other aspects of the metabolic syndrome. J Appl Physiol. 1998 Apr;84(4):1311-5. PMID: 9516198

Pi-Sunyer FX: Medical Hazards of Obesity. *Ann Intern Med* 1993; 119:655-660.

Vessby B. Dietary fat, fatty acid composition in plasma and the metabolic syndrome. Curr Opin Lipidol. 2003 Feb;14(1):15-9. Review. No abstract. PMID: 12544695

World Health Organization. *Obesity: Preventing and managing the global epidemic.* Report of a WHO Consultation: Geneva, 2000 (World Health Organization Technical Report Series; 894)

Not Good

MARSHALL UNIVERSITY™

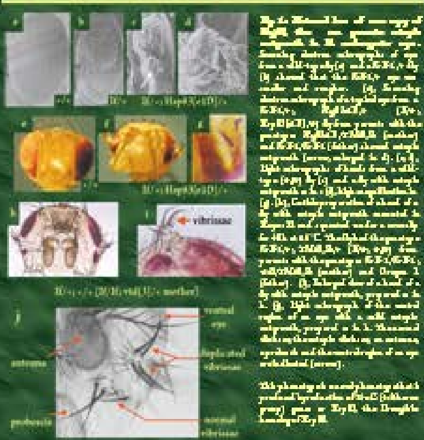
Department of Biochemistry and Microbiology
Joan C. Edwards School of Medicine
Huntington, WV

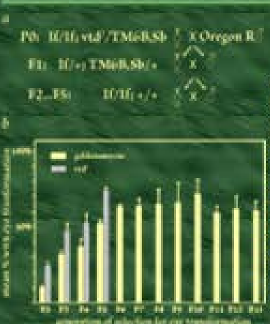
Not Good

HSP90 as an Epigenetic Enabling Mechanism in Leukemia

Abstract

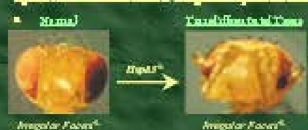
Studies in the Drosophila Model System

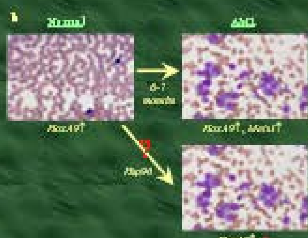




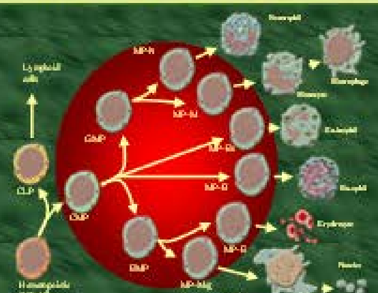
Acute Myelogenous Leukemia (AML)

Epigenetic Roles for Hsp90 in the Mammalian System

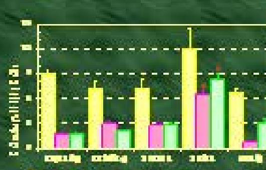


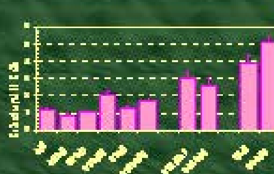


The Myeloid Progenitor Cell Compartment



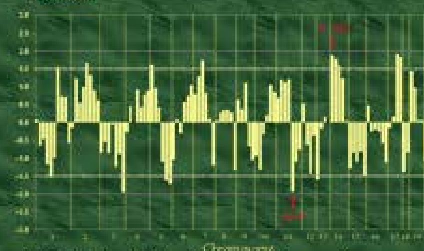
Quantifying the Myeloid Progenitor Cell Compartment





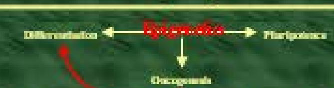
Inbred Strain Variation in the Hematopoietic System

"In Silico" Genetic Mapping



Conclusions

Research Perspective



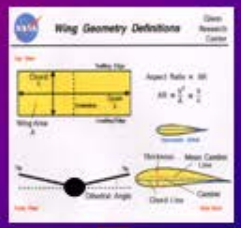
Not Good

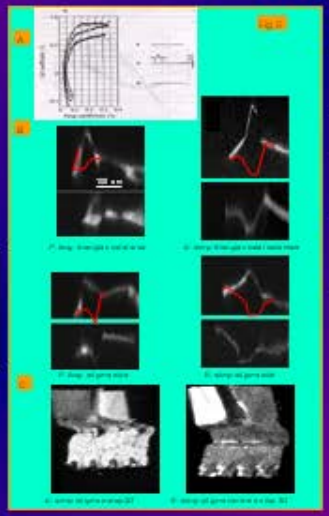
Survey of autofluorescence in the wings of dragonflies *Pachydiplax longipennis* and *Erythemis simplicicollis* with emphasis on costal morphology as related to flight mechanics

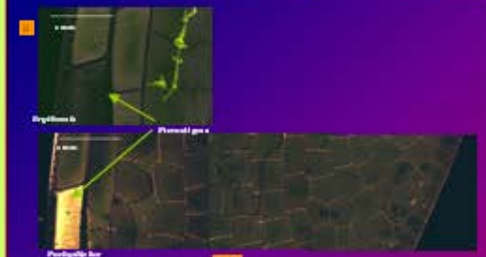
Department of Bio. Science, Marshall University

Not Good

ABSTRACT







MATERIALS AND METHODS

ACKNOWLEDGEMENTS

LITERATURE CITED

Good

Effective Posters

- Consider viewer impact
  - make it easy to read
  - make it easy to understand
  - people only have a few minutes per poster
- Keep it short, simple, and to the point

Effective Posters (cont.)

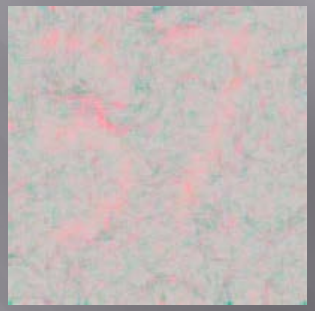
- Poster should be self-explanatory
  - remove all non-essential information
  - use verbal explanations to fill in the details
- Attract visual attention: use graphics
- Try for:
  - 25% text
  - 50% graphics
  - 25% empty space

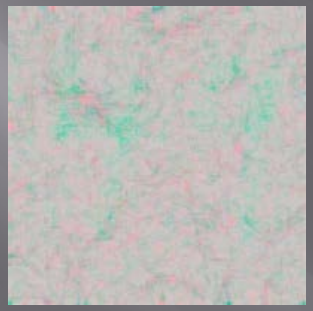
Effective Posters (cont.)

- Pick one font and stick to it
  - use larger, bold, or italics font for emphasis
- Use bullet points rather than paragraphs
- If the title is too long, shorten it
  - don't reduce the font size

Effective Posters (cont.)

- Avoid red/green combinations
  - red/green color blindness is common





- Biggest mistake: too much text
  - the most effective posters provide minimal text

http://en.wikipedia.org/wiki/Color_blindness

Effective Posters (cont.)

- A poster is not a research paper stuck to a board
  - avoid visual chaos
  - avoid shadowed text
  - make text easy to read
- View poster at 100%
  - text okay?
  - images pixilated?

Effective Posters (cont.)

- For poster approval…
  - be consistent!
    - not: Person #1, B.S., Person #2, PhD
    - not: "I would like to thank…"
    - ensure a 2" margin on the right hand side
    - one font throughout
    - paragraph indents – all the same
    - references in alphabetical order
  - be consistent!

Status of the West Virginia State Collection of Amphibians and Reptiles

Good

Marshall University, Department of Biological Sciences, Huntington, WV 25755

The West Virginia Academy of Science gave Neil D. Richmond $100 in 1935 to travel the state and collect amphibians and reptiles. These specimens and supplemental collections in 1937 and 1938 formed the nucleus for a state collection of amphibians and reptiles. Since Richmond was not associated with a museum or university, he lacked curatorial services and a building to hold the collections. To provide curatorial services, the collections were moved to Marshall College in 1939 under the care of N. Bayard Green. N.B. Green maintained the collections from 1939 to 1971. During this time the collections grew from approximately 1,000 to over 5,000. Michael Seidel served as the curator from 1971 to 1987 and Thomas K. Pauley assumed the curatorship in 1987 and continues to provide curatorial services for the collection today. Presently, there are over 12,000 specimens. The West Virginia Division of Natural Resources, United States Park Service, and United States Department of Agriculture-Forest Service have provided financial assistance for the maintenance of the collections.

Research involving N. Bayard Green Museum of Natural History

(West Virginia Biological Survey)

- 32 graduate student completed Master's theses
- 13 current graduate student working on Master's theses
- Many published research papers
- Published book: Amphibians and Reptiles in WV (Green and Pauley 1987)
- Work in progress: 2nd Edition of Amphibians and Reptiles in WV and WV Herpetological Atlas

Dr. N. Bayard Green

History of the West Virginia Biological Survey Collection

	Number of Known Species in West Virginia			Number of Specimens in Collection	
Amphibians	1948	2003		1948	2003
Salamanders	25	35		2,098	8,613
Frogs & Toads	13	15		483	2,748
Subtotal	38	50		2,581	11,361
Reptiles					
Snakes	20	22		385	969
Lizards	5	6		77	212
Turtles	11	14		64	239
Subtotal	36	42		526	1,420
Total	74	92		3,107	12,781

Selected West Virginia Amphibians

Northern Red Salamander
Pseudotriton r. ruber

Northern Cricket Frog
Acris c. crepitans

Green Salamander
Aneides aeneus

Cope's Grey Treefrog
Hyla chrysoscelis

Four-toed Salamander
Hemidactylium scutatum

Fowler's Toad
Bufo fowleri

Selected West Virginia Reptiles

Eastern Wormsnake
Carphophis a. amoenus

Common Snapping Turtle
Chelydra s. serpentina

Northern Red-bellied Snake
Storeria o. occipitomaculata

Wood Turtle
Glemmys insculpta

Black Ratsnake
Elaphe o. obsoleta

Northern Copperhead
Agkistrodon contortix mokasen

Acknowledgement

We thank the WVDNR and NPS for providing funding for the museum. Special thanks to all of the graduate and undergraduate student who have helped to build and maintain this museum. Photos by Mizuki Takahashi and Mark Watson.

Literature cited

Green, N.B. 1948. The Herpetological Collections of the West Virginia Biological Survey. West Virginia Academy of Science. 20:37-44.

PARENTAL PERCEPTION OF OVERWEIGHT/OBESE CHILDREN IN WV

Dept. of Pediatrics, Marshall University, Huntington, WV

Good

Abstract

Background: Twenty eight percent of low-income children between 2 and 5 years of age in West Virginia are overweight or at risk of becoming overweight (CDC, 2003). Previous studies have demonstrated that parents acknowledge the health risk of obesity, but medical interventions for obesity begin with the understanding of parental perception of the disease.
Objective: To determine parental perception of obesity in West Virginia.
Design/Methods: A questionnaire was given to parents in the pediatric outpatient clinic over a nine month period. Surveys were given to parents of children over 2 years of age. The survey contained questions about the appearance/appetite of their child and questions about the health related aspects of obesity. Overweight was defined as a BMI of 25 in adults and 85th percentile in children. Obesity was defined as a BMI 30 in adults and 95th percentile in children.
Results: Of 400 surveys given, 344 were returned, of which 209 surveys had adequate data to be included. Overall, 120 (55%) parents and 69 (33%) children were overweight/obese (OW/OB). Of the 69 OW/OB children, a total of 49 (71%) were described as normal weight, and 50 (72%) were described as having a normal appetite.
Conclusions: Most overweight/obese parents do not recognize obesity as a health problem, nor do they recognize obesity in their children. Future clinical guidelines to prevent obesity must acknowledge parental attitudes and perception of the problem.

Methods

SURVEY QUESTIONS

Do you think your child is:
underweight/normal weight/overweight

Do you think your child's appetite is:
too little/just right/too much

Do you think obesity is a health problem?

Do you think obesity is preventable?

Do you think that children who are obese grow up to be obese adults?

Rank how important these factors are to making someone overweight:
emotional stress
genetics
lack of exercise
overeating
type of foods eaten

Does your child's Doctor speak with you about nutrition?

If yes, how much?
too little/just right/too much

If no, would you like your child's Doctor to speak with you about nutrition at routine visits?

Rank these sources by information concerning nutrition:

books	newspaper
doctor	television
friends/family	WIC
internet	magazines
Other	

Do you struggle with your weight?

Results

Parents: Children:

Of the overweight/obese children, 71% were described by parents as being of normal weight and 72% were described as having a normal appetite.

Overweight/obese children (n = 69)

	OW/OB Parent	NW Parent	P-value
Perceived as NW	33 (69%)	16 (33%)	0.211
Appetite described as just right	34 (68%)	15 (30%)	0.211

Parents were asked to comment on the health related aspects of obesity:

Is obesity a health problem?
20% answered No (63% OW/OB)

Is obesity preventable?
8% answered No (73% OW/OB)

Do obese children grow up to be obese adults?
23% answered No (48% OW/OB)

Overeating ranked as #1 factor involved in making someone overweight

75% stated physician spoke with them about their child's nutrition

Doctor ranked as #1 source of nutrition information

54% stated they struggle with their weight

Conclusion

In our population, 55% of parents and 33% of children were obese or overweight

The majority of parents did not recognize obesity in their child

Obese/overweight parents are not more likely than normal weight parents to fail to recognize that their child is overweight/obese

However, most overweight/obese parents do not recognize obesity as a health problem

Future clinical guidelines in obesity prevention must acknowledge parental attitudes and perception of the problem

References

www.cdc.gov

SEM IMAGING OF THE SENSORY ORGAN, CAMPANIFORM SENSILLAE, OF WILD-TYPE AND MUTANTS OF THE FRUIT FLY, *Drosophila melanogaster*.

Dept. of Biological Sciences, Marshall University

Good

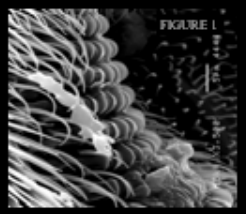
ABSTRACT

The sense organs are specialized epidermal structures for the reception of external stimuli such as contact vibrations, tastes, odors, and light. There are different types of sense organs that the fruit fly utilizes, however, this experiment focuses on just one type. The campaniform sensillae (CS), are dome shaped sense organs found on three different body parts of the Drosophila, the halteres, the legs, and the wings. The focus of the research was aimed at the halteres CS. Two different phenotypes of fruit flies were examined in this experiment: a wild type (Oregon R.) and a mutant (*prickle*). The CS on the halteres of both flies were examined by imaging halteres via the SEM microscope. The images were examined to determine if there was a difference in the orientation of the haltere CS between the wild type and *prickle* mutant fly. The images were viewed and studied and it was found that there was a few dissimilarities in orientations of the CS between the two types of flies. Continued research would be beneficial to further the findings in this area of emphasis.

The arrows show a point in the beat form of the haltere under the wings.

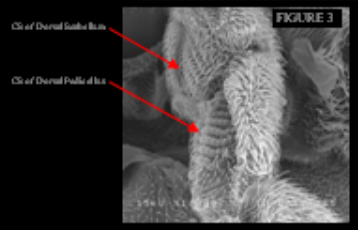
INTRODUCTION

The epidermis of the adult *Drosophila* is patterned with many sense organs including the light-sensitive ommatidia of the compound eye and the mechanosensory bristles. Both ommatidia and bristles have a specific orientation or polarity that is consistent from fly to fly and which is assumed to be functionally important. This polarity requires the activity of the Planar Cell Polarity (PCP) genes during the development of the sense organs, and mutations in these genes cause the irregular orientation of ommatidia and bristles. A third class of sense organs are the campaniform sensillae (CS) that are present at the specific locations on the halteres, legs, and wings. Although the *Drosophila* CS have previously been characterized by the Scanning Electron Microscope (SEM), little has been described about their underlying polarity and the genetic mechanisms required for their orientation. The aim of this study is to use the SEM to investigate differences in CS orientation between wild type flies and flies mutant for the PCP gene *prickle*.

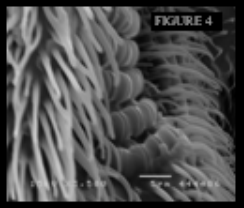


Wild type (Oregon R) ventral pedicellar CS

Oregon R CS of Dorsal Pedicellum and Scabellum of Haltere



plcp-1 CS of Dorsal Pedicellum and Scabellum of Haltere



Mutant (*prickle*) ventral pedicellar CS

Images obtained by Aaron Smith and David Neff.

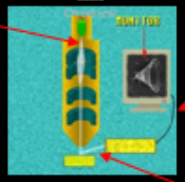
MATERIALS AND METHODS

During the course of this research, I was involved with using the SEM to obtain my images of the haltere CS. The SEM is a microscope that allows one to obtain very detailed images of microscopic materials by way of an electron beam scanning the surface of the specimen and projecting the image on a TV screen (below is a brief overview of how the SEM works). Preparation for my samples involved dissecting parts of the flies, including the legs and wings, to obtain unobstructed views of the halteres. Once the flies were mounted on metal stubs for SEM samples, sputter coating was essential before viewing through the SEM. This allows for the sample to be able to conduct electricity because the specimens are illuminated with electrons when being viewed. The use of a fixation method (described below, right) was necessary to decrease the shrinking that took place during the sputter coating and also within the vacuum of the SEM. This allowed for the images not to have a shriveled, imprecise appearance when viewing, helping to maintain the 3-D structure. Viewing the images was then done on the computer after pictures were taken of the images that were obtained on the microscope. Flies imaged were Oregon R. wild type and flies homozygous for the *prickle* gene allele *prickle*^{spiderO}

RESULTS AND DISCUSSION

The purpose of this research was to determine if the mutant fly CS orientation were different from the wild type fly. There are three different sections of the haltere, the scabellum (inner most section), the pedicellum (mid section), and the Capitellum (outer most section). The fields of CS that were studied in this experiment were located on the scabellum and pedicellum. I found that there was a lot of a difference in the CS on the dorsal pedicellum, which is apparent in Figure 2 and Figure 3. The lower row (1-9) of the CS on the mutant fly are disorientated and not in line as they should be (on Fig 2). The spacing between the rows of the pedicellar CS on the mutant fly also look wider than on the wild type fly. However, on the same figures (2 & 3), the dorsal CS on the scabellum look similar in orientation. Also, apparent in Figures 1 & 4, the ventral pedicellum CS are not altered as well. Some errors could have also occurred during the research as well. There could have also been a fault in the fixation methods since they were kept in separate vials when going through the process, which could have caused some error. Further research and imaging would be very helpful in making the determination that the *prickle* gene aids in altering orientation of the dorsal pedicellar CS.

SEM



Electron Beam

Electrons shoot down a tunnel through an array of magnetic lenses that focus the beam on a spot (specimen).

The detector then counts the electrons and the amplifier receives the signals that eventually are processed into an image on the monitor.

Secondary electrons are knocked loose from the specimen's surface and sent to the detector and amplifier.

Standard Glutaraldehyde Fixation

The fixative consisted of: 10 ml 2.5% glutaraldehyde
50 ml buffer stock solution (cacodylate buffer, pH 7.3-7.4)
Make up 100 ml with distilled water
Final Concentration: 2.5% glutaraldehyde in 0.1 M sodium cacodylate
During fixation I immersed the flies in fixative for approximately 8-12 hrs, rotating the flies in vials. I then rinsed the flies overnight in the buffer solution. I then rinsed flies in water and proceeded on to the first step in the method, the dehydration alcohol series. The alcohol series consisted of first up the flies in different percentages of ethanol (5 minutes in each solution). The series consisted of 30% (EtOH), 50%, 70%, 85%, 95%, 100% & 100% (Final Concentration is used twice and also for approximately 30 min).

CHARACTERIZATION OF PLANT CELL BIOMASS AND BIOACTIVE COMPOUND PRODUCTION IN A MICROGRAVITY-BASED HYDRODYNAMIC FOCUSING BIOREACTOR (HFB)

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Not Good

ABSTRACT

INTRODUCTION

MATERIALS AND METHODS

RESULTS

DISCUSSION

CONCLUSIONS

LITERATURE CITED

ACKNOWLEDGEMENTS

NASA Scholarship Recipient

PROGESTIN STIMULATION OF MANGANESE SUPEROXIDE DISMUTASE IN T47D HUMAN BREAST CANCER CELLS

Departments of Biochemistry and Microbiology and Pharmacology, Physiology, and Toxicology,
Joan C. Edwards School of Medicine, Marshall University, Huntington, WV

Not Good

Abstract

Introduction

$$2O_2^{\cdot -} + 2H^+ \rightleftarrows H_2O_2 + O_2 \quad A$$

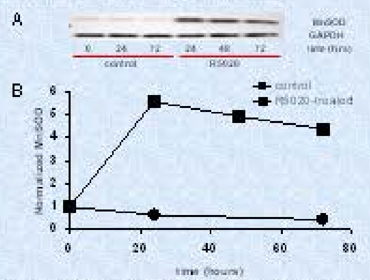
$$2H_2O_2 \rightleftarrows O_2 + 2H_2O \quad B$$

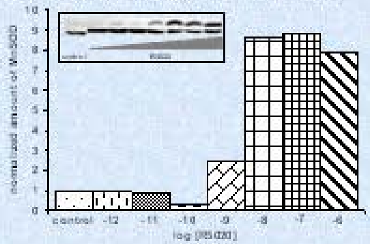
$$GSH + ROOH \rightleftarrows GSSG + H_2O + ROH \quad C$$

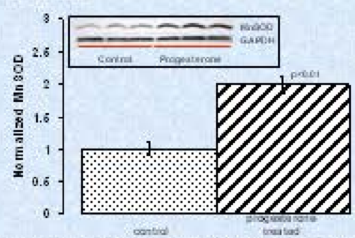
Experimental

Experimental (continued)

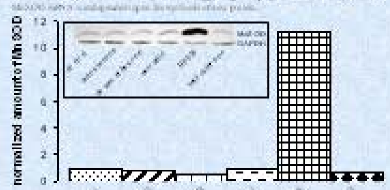
Results

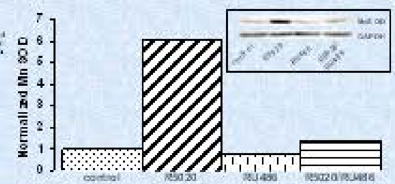






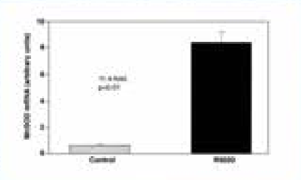
Results (continued)

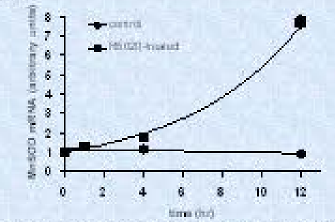


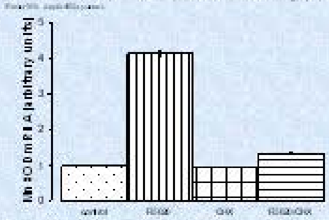


Discussion

Discussion (continued)







Conclusions

Acknowledgements

References

Questions?

http://www.dvwgroup.com/DVW_Group_Overview.htm

References

- http://www.biology.lsa.umich.edu/research/labs/ktosney/file/PostersHome.html
- http://www.ncsu.edu/project/posters/GoodGraphs
- http://projects.cs.dal.ca/DCSI/present.ppt#1
- http://www.cmer.wsu.edu/~yonge/ce465/poster.pdf
- http://depts.washington.edu/mphpract/perfect%20Poster.ppt#1
- http://www.asp.org/education/howto_onPosters.html