A Presentation Primer

- General guidelines for posters
- General guidelines for “PowerPoint”

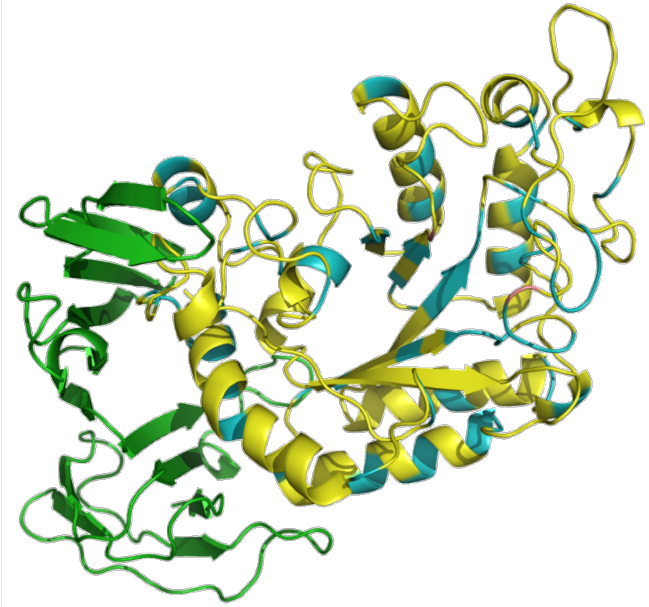
Presentation skills are required no matter what profession you end up in.

- You need to be able to communicate information
- You need to be able to convince people of your data

Poster Presentations

A poster *SHOWS*

it does not *TELL*



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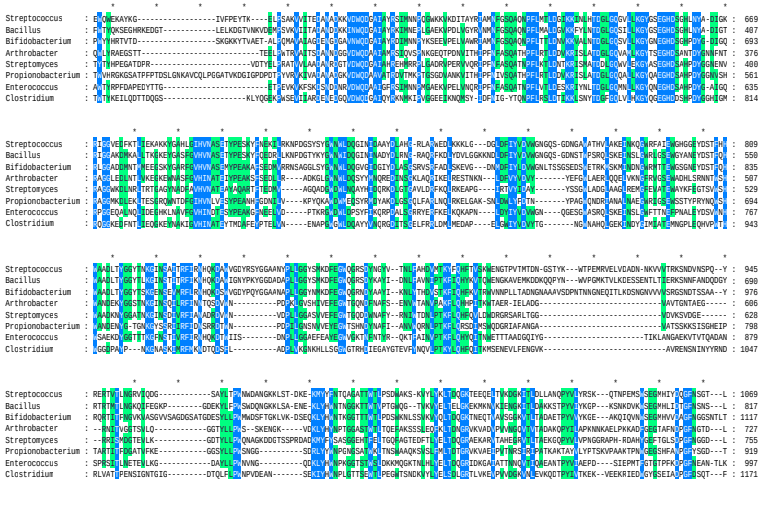


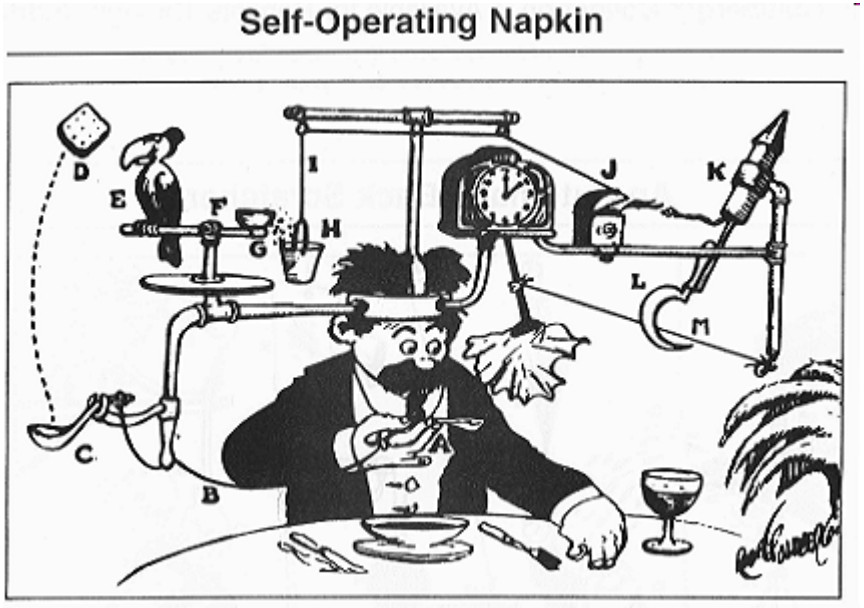
Advantages of “Visual” Presentations

Increased audience interest (catch it, then educate)

Increased understanding

Increased retention: images remembered longer than words

Increased efficiency: message communicated faster by images



Design Specifications

In general, self-explanatory graphics should DOMINATE.

A minimal amount of verbiage should supplement the graphics. 500 - 800 words total, max!

Edit ruthlessly! Simplify!

Recognise that readers use visual grammar…

They read L to R, top to bottom. This includes left justification. They find the active voice more comfortable to read. Always consider topic/stress.

More Design

- Aim for 20-25% text; 40-45%graphics; 30-40% white space
  (If your advisor says put more in the white space, nod sagely and ignore it)

- No abstract! With the text being so focused and tight, an abstract is superfluous.

- Bullets help to make a point - easier to follow

- Double space the text - easier to read

The Crux

Make sure there is a clear take-home message

Make sure there is *one central question* clearly stated

Canada *IS* the best!

Housekeeping

Font size implies importance

Biggest Title

Big Section headings

Smaller Supporting material

Smallest Details

Go EASY on colour

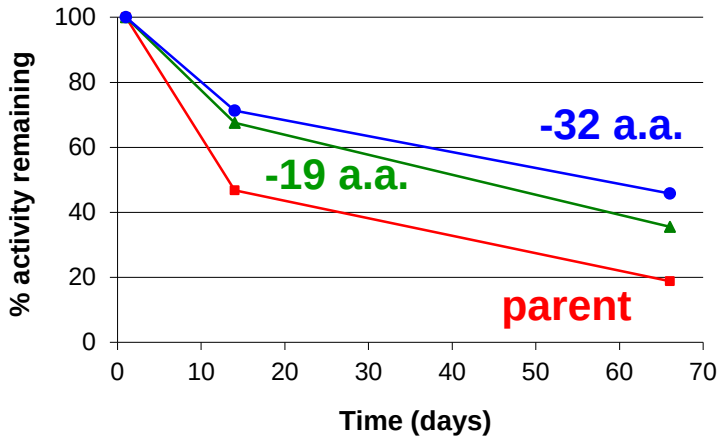
Subtle emphasis

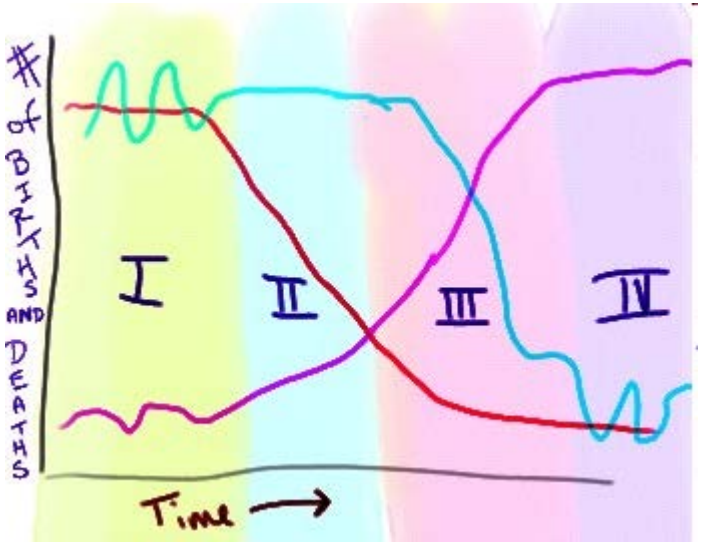
Figures

Label data lines directly instead of using a key, thereby eliminating keys and shortening legends!

Use short sentences; simple words if possible

Don't use "Figure 1" etc. Let the sequence of figures tell the story…although this is a minor point.





Don’t be afraid to use your own graph based on the data

Introduction (~200 words)

- Have minimal background and definitions

- Relate your problem to the primary literature

- Describe and justify the experimental approach

Give a clear hypothesis

- If you have an illustration of some sort that communicates some aspect of the problem find a way to include it.

Materials & Methods (~200 words)

Briefly describe methods & special equipment,
in less detail than a manuscript

Use figures, tables, flow charts wherever possible

Results (~200 words + fig legends)

The largest section and includes the Discussion, which is built in

- First paragraph should hit the spot - did it work?

Also, qualitative & descriptive results, if appropriate

- Then the more specific data analysis
- Use lots of figures, images, *graphs*. Convert tables to graphs if you can
- Make the legends informative and interesting and include some M & M to make M & M section shorter and save the reader from going back and forth

Conclusions (~300 words)

Last two paragraphs…

- Restate hypothesis and result, and state whether hypothesis was supported

- Try to convince the reader/listener why the results are interesting (and other good points)

- How the results are relevant to published work?

- What you plan to do next

Very bad!

The beginning of the end for chimpanzee experiments?

Philosophy, Ethics and Humanities in Medicine 2008; 3:16. http://www.peh-med.com/content/3/1/16.

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Ending US chimpanzee experimentation

On 17th April, 2008, *The Great Ape Protection Act* was tabled in the US Congress. The bill proposed to end invasive research and testing on an estimated 1,200 chimpanzees confined within US laboratories — some for over 40 years [1]. This followed a 2007 decision by the NIH National Center for Research Resources to permanently implement a chimpanzee breeding moratorium, which is expected to result in a major decline in numbers over the next 30 years, as most are retired or die [2-3]. Finances played a significant role. The lifetime costs of supporting captive chimpanzees are $300,000 to $500,000 [3], and the federally-funded population will cost $325 million [2]. These steps could signal the beginning of the end for invasive chimpanzee experiments within the US.

International bans on great ape experimentation

In the UK, special justifications for great ape (chimpanzees, bonobos, gorillas and orang-utans) experimentation became necessary under the *Animals (Scientific Procedures) Act 1986*, and in 1997 a policy ban was implemented by the Home Office [7-9]. Great ape experimentation has also been banned in Sweden (regulatory restrictions since 2003, with the exception of non-invasive behavioral studies), and Austria (since 2006, unless conducted in the interests of the individual animal). The Netherlands was the last European country to conduct invasive chimpanzee research, and outlawed great ape experimentation from 2004 [3, 9].

In countries such as Italy and Norway, great apes have not been used for years, although national bans have yet to be passed. Since 1992 great apes have not been subjected to invasive research within Germany, although non-invasive cognitive and behavioral studies do occur. In 2002, the Belgian minister responsible for animal welfare announced that Belgium would be working toward a ban on all primate experiments, and a Swiss state ethics commission recently demanded that the Swiss government ban great ape experimentation [3, 6, 9-14].

Japan ceased invasive chimpanzee research in 2006 [15]. In Australia and New Zealand, great ape experimentation is restricted by policy (Australia) [16], or legislation (New Zealand, since 1999) [9, 17]; unless in the best interests of the individual animal or species.

In late 2007, 433 Members of the Members of the European Parliament signed *Parliamentary Written Declaration 40/2007*, calling for urgent action to end the use of great apes and wild-caught monkeys in experiments, and for the establishment of a timetable for the cessation of all European primate experiments. By mid 2008, such changes were under consideration during a formal revision of *European Directive 86/609/EEC on the Protection of Animals used for Experimental and Other Scientific Purposes*, which governs animal use within EU member states.

Advancements in biomedical knowledge?

To assess the utility of chimpanzee experimentation, I recently surveyed three major biomedical bibliographic databases, locating 749 published invasive chimpanzee studies conducted from 1995 - 2004 (Figure 1) [51]. However, of 95 randomly-selected experiments, 49.5% (47/95; 95% CI = 39.6 – 59.4%) were not cited by any subsequent papers (Figure 2). The year of publication did not appear to significantly affect this outcome, as citation frequencies were similar across the decade.

Given that much research of lesser significance is not published, these published chimpanzee experiments can generally be assumed to be those with the greatest potential for advancing biomedical knowledge. Consequently, these results indicate that the majority of invasive chimpanzee studies generate data of questionable value, which makes little obvious contribution toward the advancement of biomedical knowledge.

Almost all of these experiments would have been approved by at least one institutional ethics committee entrusted with ensuring that their expected benefits were reasonably likely to exceed their welfare-related, bioethical and financial costs. The approval of large numbers of these experiments therefore indicates a widespread failure of the ethics committee system.

Advancements in human healthcare?

Only 14.7% (14/95; 95% CI = 8.9 – 23.4%) of these randomly-selected chimpanzee studies were cited by a total of 27 papers describing well-developed diagnostic, therapeutic or prophylactic methods for combating human diseases (Figure 2). However, detailed examination of these medical papers revealed that *in vitro* studies, human clinical and epidemiological studies, molecular assays and methods, and genomic studies, contributed most to their development.

The randomly-selected chimpanzee studies proved to be of peripheral importance to most of these medical papers, for a variety of reasons. 63.0% (17/27) were determined to be wide-ranging reviews of 26-300 (median 104) references, to which the cited chimpanzee study made a very small contribution. In 12 cases the chimpanzee studies appeared redundant, as humans or human sera were studied concurrently, or because they served only to confirm previous human observations. In seven cases the method explored in the cited chimpanzee study was not developed further, sometimes because later clinical trials in humans failed to demonstrate safety or efficacy, contrary to positive chimpanzee results. In most of the remaining cases the chimpanzee study examined a disease or method peripheral to the medical method described, or yielded results inconsistent with other human or primate data, or merely illustrated historical findings, or was cited only to discuss concurrent human outcomes within the cited chimpanzee study. In fact, none of these cited chimpanzee studies demonstrated an essential contribution, or — in most cases — a significant contribution of any kind, toward the development of the medical method described.

Bioethical considerations

Achieving a reasonable and rational balance between the interests of people and those of laboratory animals requires balanced consideration of the interests of both groups: primarily, the likely benefits accruing to humans, and the probable costs incurred by animal experimental subjects. Invasive chimpanzee experimentation allows investigation of a virtually limitless number of scientific questions. However, the majority of such experiments appear to generate data of questionable value, which makes little obvious contribution toward the advancement of biomedical knowledge. Additionally, such studies rarely — if ever — make significant contributions toward the development of methods efficacious in combating human diseases [51]. The resource and financial burdens incurred by such research are also considerable.

The costs to chimpanzees enrolled in such experiments include involuntary confinement within laboratory settings, social disruption, and participation within potentially-harmful research protocols. The effects of laboratory confinement and procedures, especially long-term, can be severe. Many captive great apes show gross behavioral abnormalities, such as stereotypies, self-mutilation or other self-injurious behavior, inappropriate aggression, fear or withdrawal [100-101], including among chimpanzees recently retired from US laboratories [102]. It is increasingly acknowledged that such abnormal behaviors resemble symptoms associated with human psychiatric disorders, such as depression, anxiety disorders, eating disorders, and post-traumatic stress disorder, and that pharmacological treatment modalities similar to those applied to human patients may be appropriate, and indeed, morally compelled, for severely disturbed animal patients [100, 103].

Although these highly sentient creatures are in no way responsible for any human grievance, such as the serious diseases we attempt to induce in them, we sometimes subject chimpanzees to conditions that would cause widespread social outrage if used to punish the most heinous of human criminals — for years on end, and in some cases, for decades. It is perhaps not unreasonable to assert that the lack of humanity highlighted by this difference in standards applies less to chimpanzees, than to ourselves.

The logic of Bradshaw and colleagues [102] is compelling: *"In human traumatology, the first step in treatment is to arrest its causes. This implies that prevention and treatment of chimpanzee psychopathology entails considering the factors and institutions that have brought chimpanzees to the point of irreversible distress: in simple terms, desisting from using apes as biomedical subjects in lieu of humans is compelled if trauma is not to be perpetuated."*

The unique biological characteristics of chimpanzees — which are rare in their own right — and their advanced sensory, psychological and social characteristics — which have some similarities with those of humans — all create a strong ethical basis for acknowledging the necessity of respecting at least the most basic and essential interests of chimpanzees, such as their interests in avoiding death, pain, suffering and captivity [104-105]. When according due consideration to the interests of both humans and chimpanzees, it cannot be concluded that invasive chimpanzee experimentation is ethically justifiable.

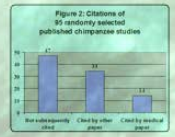
Conclusions

According due respect to such bioethical considerations does not require the termination of all chimpanzee research. Bioethical concerns are minimized within non-invasive observational, behavioral or psychological studies of free-living or sanctuary populations. Such limitations would inevitably restrict the range of scientific questions that might be investigated. It would, however, strike the correct ethical balance between satisfying the interests of chimpanzees, and those of human beings.

In the early 1990s around half a dozen countries conducted invasive chimpanzee experiments, but by 2008, the US was almost completely isolated internationally. Ending such research within the US would uphold the best interests of chimpanzees, and other fields presently deprived of research funding, and would also increase the compliance of US animal researchers with internationally-accepted animal welfare and bioethical standards. It could even result in the first global moratorium on invasive research, for any non-human species, unless conducted in the best interests of the individual or species.

References & Acknowledgements

References are taken from the *Philos, Ethics & Humanities in Med* 2008 article (see poster title). Figures 1-2 and the results of my systematic review of the human biomedical utility of invasive chimpanzee experiments were first published within the article: Knight A. The poor contribution of chimpanzee experiments to biomedical progress. *Journal of Applied Animal Welfare Science* 2007, 10(4):281-308. They are republished with permission of the publisher (Taylor & Francis Ltd, http://www.tandf.co.uk/journals).
Photo credits: World Society for the Protection of Animals (UK), People Against Chimpanzee Experiments (UK), www.Primates.com.








Too much text!

Diverging aspects of HDAC inhibitors: transcription and metabolism

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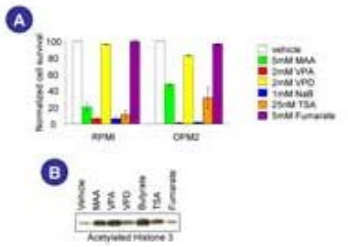
Abstract

Multiple myeloma is a hematological neoplasm caused by an expansion of malignant plasma B cells. Standard treatment includes corticosteroids, which induce apoptosis of the myeloma cells, but frequently results in resistance. Experimental alternative therapies for myeloma include histone deacetylase inhibitors (HDACi). We find that valproic acid (VPA), an HDACi widely used to treat seizures, efficiently induces apoptosis in myeloma cells. While HDACi can potentiate transcriptional activity of steroid hormone receptors (1), VPA affects myeloma cells independent of glucocorticoid receptor activity and efficiently induces apoptosis regardless of glucocorticoid resistance. HDACi are known to induce apoptosis in hematopoietic tumor cells concurrent with induction of p21 and TRAIL death ligand (2,4). In addition, HDACi's rapidly reduce mRNA and protein expression of growth factor receptors associated with growth and suppression of apoptosis in myeloma, including interleukin-6 receptor (IL-6R) α, fibroblast growth factor receptor (FGFR) 3, and B cell maturation antigen (BCMA) (2,3,5). However, HDACi have additional activities independent of their role in transcription. HDACi treatment reduces the available cellular pool of acetyl CoA. In response, the cells turn to protein degradation and metabolism of amino acids for energy, decreasing cellular levels of individual amino acids by up to ten fold. mRNAs encoding arginase II and carbamoyl phosphate synthase (CPS1), enzymes involved in amino acid metabolism and nitrogen clearance, are correspondingly induced after 24 hrs of HDACi treatment. Supplementation with additional amino acids increases the induction of apoptosis, suggesting that buildup of nitrogen metabolites of amino acid degradation contributes to HDACi mediated apoptosis. Organic acid analysis of cells following HDACi treatment indicates a significant drop in α-ketoglutarate, a key component of the TCA cycle that is also a required intermediate in the metabolism of amino acids and β-oxidation of fatty acids. These data together indicate that while HDACi can modulate transcription of select genes, an additional facet to their action is the profound effect on cellular metabolism initiated by a significant reduction in the cellular pool of acetyl CoA.

Results

Figure 1. HDACi induce apoptosis in myeloma cells regardless of dexamethasone sensitivity. (A) Multiple myeloma cells lines RPMI (dex sensitive) and OPM2 (dex resistant) were treated for 96hrs in complete media with the indicated compounds – methoxyacetic acid (MAA), valproic acid (VPA), valpromide (VPD), sodium butyrate (NaB), trichostatin A (TSA), or fumarate. Apoptosis was analyzed by annexin-PE and 7-AAD staining followed by flow cytometry. (B) Lysates of OPM2 cells treated for 24 hrs with the indicated compounds were analyzed for HDAC activity by Western blot analysis of acetylated histone 3.



Results

Figure 2. HDACi treatment rapidly down-regulates mRNA and protein expression of growth factor receptors previously demonstrated to participate in myeloma cell growth and resistance to apoptosis. Three indicated myeloma cell lines were treated 0-24 hrs with 2mM VPA followed by (A) Western blot or (B) real time qPCR analysis of lysates or RNA, respectively, analyzing expression of growth factor receptors demonstrated to be essential for each respective cell line.

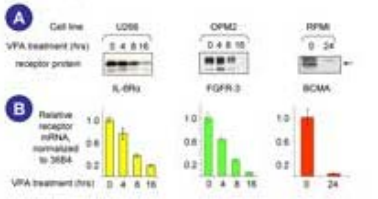


Figure 3. HDACi treatment reduces the cellular pool of available acetyl CoA. (A) Cellular processes that tightly regulate cellular levels of acetyl CoA through its contribution and utilization. (B) OPM2 cells were treated for 44hrs with VPA (2mM), MAA (5mM), butyrate (NaB – 1mM), suberoylanilide hydroxamic acid (SAHA – 5uM), or Dexamethasone (Dex - 100nM). Cells were lysed by sonication and MS/MS analysis was performed on clarified lysates to determine levels of acetyl carnitine (in equilibrium with acetyl CoA). The reduction of acetyl carnitine indicates a significant drop in the normally tightly regulated levels of acetyl CoA.

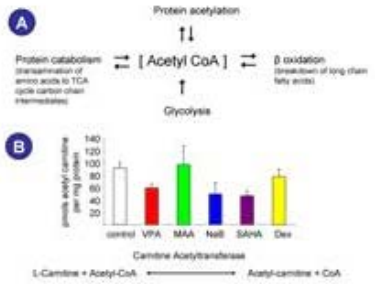
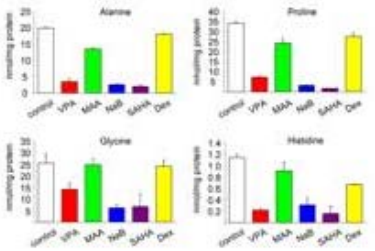


Figure 4. HDACi treatment increases metabolism of amino acids. Lysates from Figure 3B were examined using MS/MS analysis for amino acid content. No significant change in glycolytic intermediates or long chain fatty acids was observed, while a significant reduction in the levels of all 17 amino acids analyzed was evident, four of which are shown below. These finding indicate that OPM2 cells preferentially utilize transamination to replace acetyl CoA.



Results

Figure 5. Toxicity of amino acid metabolism in the presence of HDACi suggests a buildup of nitrogen intermediates. (A) Ammonia created through amino acid degradation is cleared physiologically through the indicated pathways. No significant production of urea was measured from HDACi-treated cells in culture (data not shown). (B and C) OPM2 cells were treated for 96hrs with VPA (2mM) in the presence of absence of the indicated compounds. Apoptosis was analyzed as in Figure 1. Increased apoptosis in the presence of VPA and supplemental amino acids suggests a buildup of a toxic nitrogen product. (D) OPM2 cells were treated 24 hrs with the indicated compounds as in Figure 3, and expression of arginase II and carbamoyl synthetase 1 (enzymes involved in nitrogen disposal) were analyzed by real time qPCR.

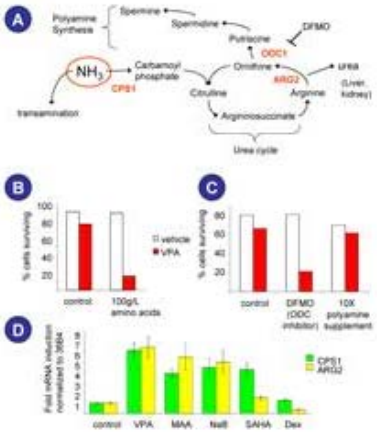
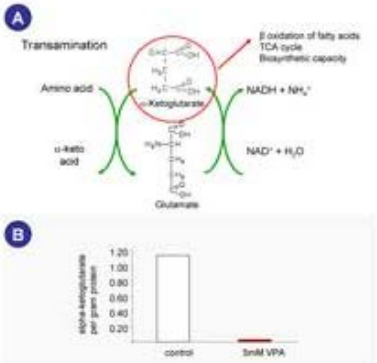
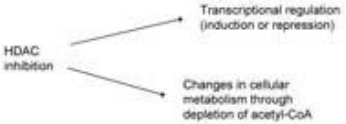


Figure 6. Toxicity of amino acid metabolism in the presence of HDACi suggests a buildup of nitrogen intermediates. (A) Model of transamination, the process by which amino groups are removed from amino acids to allow metabolism of the carbon skeleton. (B) NB4 cells were treated 24 hours with or without 1mM VPA prior to acidic extraction of the cells followed analysis of organic acids.



Conclusion



- HDAC inhibitors effectively induce apoptosis in multiple myeloma cell lines as well as myeloma patient isolates (not shown), and their ability to induce apoptosis appears to be proportional to their activity as HDAC inhibitors.
- In addition, HDAC inhibitors rapidly down-regulate growth factor receptors important for myeloma cell growth and survival, at both the mRNA and protein levels.
- HDAC inhibitor treatment reduces levels of acetyl carnitine, suggesting a corresponding reduction in the available cellular pool of acetyl CoA that may result in stalling of the TCA cycle and utilization of amino acids by the cell as an energy source.
- Breakdown of amino acids to salvage the carbon chains for energy forces the cell to dispose of ammonia released by deamination of the amino acids. Myeloma cells utilize both transamination and production of polyamines to sequester the released nitrogen.
- The contribution of the polyamine pathway may be small, because addition of excess polyamines does not significantly affect cell survival itself or the apoptotic activity of VPA. However, inhibition of the pathway increases the apoptotic potential of VPA, likely because of additional use of transamination.
- While transamination potentially sequesters the ammonia produced, it depletes the cell of α-ketoglutarate, further crippling the TCA cycle and ultimately preventing transamination.
- Physiologically, amino groups would be incorporated into arginine, glutamine, or alanine, and ultimately converted to urea in the liver. However, the rate of ammonia production that may be occuring in myeloma cells may contribute to the clinical effectiveness observed for HDAC inhibitors.
- Because cancer cells, as opposed to normal cells, rely primarily on glycolysis for energy and do not significantly utilize β-oxidation of fatty acids even in the presence of oxygen, the effect of HDACi on metabolism may ultimately be specific to cancer cells, accounting for the low toxicity observed clinically.

References

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Functional trait correlations as indicators of drought resistance strategies

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Introduction

Plant functional traits are attributes that allow plants to survive under certain environmental conditions, for example the stressful conditions associated with drought.[1] However, independent traits usually do not single handedly determine levels of drought resistance; it is more likely that **a suite of functional traits determines how plants survive drought**.[2] In order to assess the generality of trait correlations across a rainfall gradient, we measured four traits related to drought resistance.

Fig. 1: Collection of leaf, stem, and wood samples

Methods

The traits that were measured included **specific leaf area** (SLA), which is an index of sclerophylly,[3] **stem density** (SD), which is correlated with xylem cavitation resistance,[4] and **wood density** (WD), which is negatively correlated with cavitation resistance and negatively correlated with water storage.[5] In addition, **leaf phenology** plays an important role in drought resistance, as deciduous trees are able to reduce water loss by dropping leaves, while evergreen trees must resist drought.[6] Leaf and stem samples were collected using a leaf pruner, while trunk samples were collected using an increment borer (Fig. 1). ImageJ software was used to measure the surface area of leaves (Fig. 2). Regression analysis were performed using the computer software program Statview. Correlations between these traits were examined in trees of the dry semi-evergreen vine thicket (750-800 mm rainfall pa) and wet Type 5b 'Mabi' rainforest (1300-1600 mm rainfall pa) of northeast Queensland, Australia.

Fig. 2: Leaf sample photographed for processing in ImageJ

Results

SLA was significantly negatively correlated with SD regardless of leaf phenology ($F_{1,143}$=29.577, p<0.0001, Fig. 3). When examining **SLA and WD**, a significant **positive correlation** was found in **deciduous species** ($F_{1,50}$=8.922, p=0.0044, R^2=0.151), while a significant **negative correlation** was found in **evergreens** ($F_{1,69}$=9.593, p=0.0028, R^2=0.122, Fig. 4).

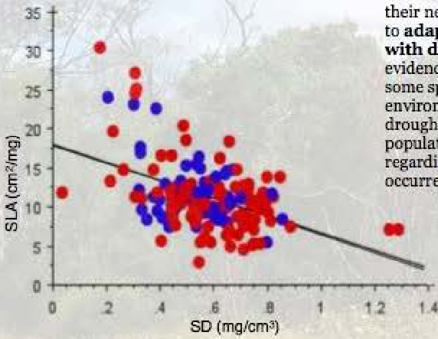
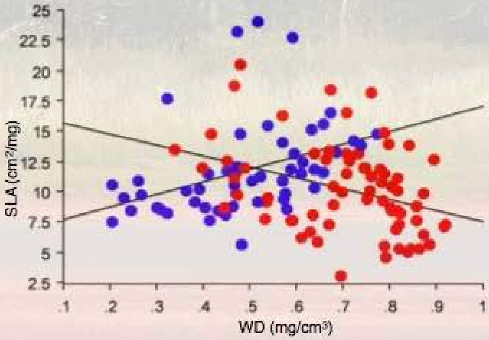


Fig. 3: Specific Leaf Area v. Stem Density

Conclusions

Deciduous species are able to survive with only one drought resistant trait due to the **flexibility that deciduousness allows**, while **evergreen** species have developed **multiple drought tolerant traits** in order to survive. Because of this, evergreen trees in the wet rainforest with low SLA and high WD, and all deciduous trees, would be most likely to survive the increasing aridity that may be associated with global warming, while high-SLA, low-WD wet rainforest evergreens would be the least likely to successfully survive in their new environments, unless they are able to **adapt by shifting to traits associated with drought resistance**. There is some evidence of such adaptation having occurred; some species which occur in both environments have traits indicative of greater drought resistance in their low rainfall populations, although there is no evidence regarding the timeframe over which this occurred.

Fig. 4: Specific Leaf Area v. Water Density



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6. Eamus, D. 1999. Ecophysiological traits of deciduous and evergreen woody species in the seasonally dry tropics. Tree 14: 11-16.

Acknowledgments

The School for Field Studies provided funding for food, transport, and other logistical needs. QPWS permits [illegible] and [illegible] and Forestry Plantations Queensland permit [illegible] to Tim Curran were necessary to conduct work on the state-owned land that we sampled from. We would also like to thank the landowners that granted us access to their land for our research purposes. In addition, R. Fensham of Queensland Herbarium provided valuable floristic data for Undara Volcanic and Forty Mile Scrub National Parks.

We would like to thank E. Reid for her constant and indispensable help throughout the project, and her positive attitude and support were always a welcome break during crunchtime.

Finally, we would like to thank T. Parkes, J. Iacarella, L. Phillips, and E. Silver for their camaraderie and moral support; we really were the greatest DR group ever, and none of this would have been possible without each one of you.

Simpler style, big images, good effort.

CHARACTERIZATION OF A THERMOSTABLE GH6 ENDOGLUCANASE FROM *CELLULOMONAS FIMI*

Marc de Asis[1], Marie Couturier[2], Emma Master[2] and Warren Wakarchuk[1,3]

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[2] Department of Chemical Engineering, University of Toronto, Toronto, ON

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Chemical Engineering & Applied Chemistry
UNIVERSITY OF TORONTO

WATERLOO SCIENCE

NSERC CRSNG

Background

Lignocellulose is one of the most abundant carbon sources in nature. This naturally occuring substance is an underutilized source of bioenergy. A major bottleneck in biofuel processing is the enzymatic hydrolysis of lignocellulose into its ultimate fermentable product, glucose. *Cellulomonas fimi* is a well-studied soil organism known for its capabilities to efficiently hydrolyze cellulose. Recently, *C. fimi*'s genome was sequenced, which revealed uncharacterized cellulases. One of these enzymes was Celf_1230, a putative cellulase from the glycoside hydrolase family 6. Using various cellulosic derivatives as substrates, we sought to characterize Celf_1230 and investigate possible synergistic effects with other known cellulases. Our results have shown Celf_1230 to be a thermostable enzyme with endoglucanase activity.

Glycoside Hydrolases in *Cellulomonas*

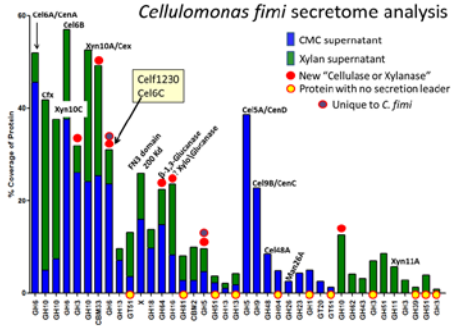
Species	GH	GT	PL	CE	CBM
Cellulomonas fimi ATCC 484 (DOE-JGI)	109	47	6	10	55
Cellulomonas flavigena DSM 20109 (DOE-JGI)	89	50	2	14	74

	GH											
Organism	1	2	3	5	6	9	10	11	13	42	43	Total
C fimi	1	1	11	3	4	4	5	1	18	6	7	109
C flavigena	1	1	3	3	3	5	16	3	14	2	5	89
T fusca	2	1	2	3	2	2	2	1	6	1	1	43
S coelicolor	5	7	9	5	3	2	2	1	18	2	6	159
C japonicus U107	0	3	3	15	1	3	4	2	17	0	14	125

So what is new in *C. fimi* then , based on cellulase/xylanase?

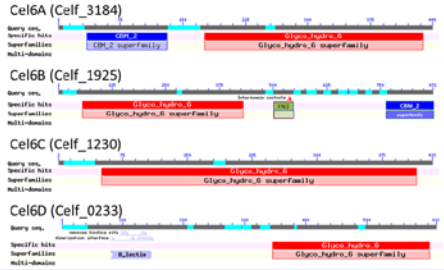
10 GH-3	β-Glucosidase/Xylosidase
2 GH-5	Cellulase/Mannase/Xylanase
2 GH-6	Cellulase
2 GH-9	Cellulase
2 GH-10	Xylanase
1 GH-74	(Xylo)glucanase

Cellulomonas fimi secretome analysis

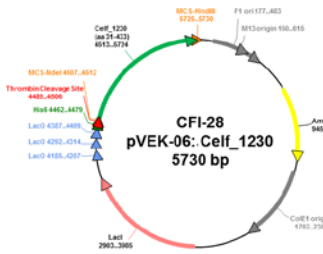


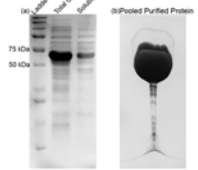
Supernatant proteins were precipitated with TCA, and the proteins were then solubilized for 1D SDS-PAGE analysis. For GelLC, each of the sample containing lanes were cut into 25 equal bands using a gel cutter from about 150kDa to just above the dark smear at the bottom of the gel.Tryptic fragments were then analyzed by LC-MS analysis. The protein ID was performed using MASCOT, and proteins with a score over 50 were used for the diagrams shown above.

GH6 Enzymes from *C. fimi*

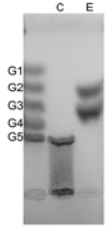


Purification and Characterization of Celf_1230 (Cel6C)

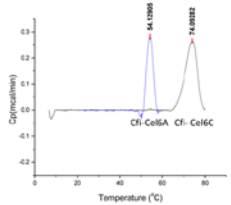




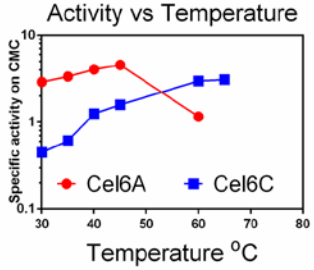
Purification of Cfi-Cel6C by IMAC



TLC analysis of G5 hydrolysis



Differential scanning calorimetry of Cfi-Cel6A and Cfi- Cel6C



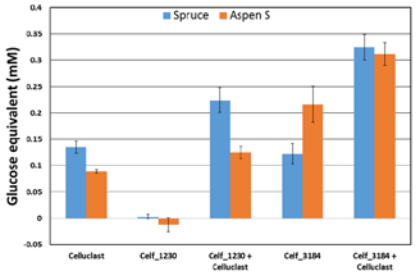
Activity Assay Data

	Celf_3184			Celf_1230		
Substrate	kcat (s^{-1})	±	(%)*	kcat (s^{-1})	±	(%)
CMC	5.86	0.11	100%	5.28	0.31	90%
PASC	2.02	0.25	34%	3.93	0.33	67%

	Relative activity	
Substrate	Celf_3184	Celf_1230
AZCL-Hydroxyethyl Cellulose	100%	12%
Azo-Avicel	-	-
AZCL-Xylan (birchwood)	-	-

*Relative activity based on Celf_3184 on CMC substrate using the HBAH assay.

Potential Synergy with Celluclast on Biomass Substrates



Celluclast® (a commercial blend of *Trichoderma* cellulases) was used to assess synergistic effects of two *C. fimi* endoglucanases on biomass substrates, aspen (hardwood) and spruce (softwood). Celf_1230 was observed to have no direct effect on these substrates. However, with the addition of Celluclast®, there is an apparent synergistic effect. Celf_3184 was able to hydrolyze these substrates on its own and only had an additive effect with Celluclast®. Hydrolysis was assessed based on reducing sugars released using the DNS assay. The assay was performed at an incubation temperature of 45°C.

Discussion and Future Directions

Celf_1230 (Cel6C) is a thermostable endoglucanase that has better activity on amorphous non-substituted cellulose. Its activity is unlike that of Cel6A and it shows considerable thermostability, and activity at elevated temperatures. Cel6C appears to require pre-digestion on cellulose to provide a synergistic activity. The reason for this is not known at present. This enzyme has no associated carbohydrate binding domain, unlike Celf_3184 (Cel6A) and other known GH6 cellulases. Homologs of Celf_1230, determined by BLAST, revealed putative cellulases that are yet uncharacterized.

The molecular structure of Celf_1230 has to be determined to fully understand both its thermostability and its activity. Furthermore, it would be interesting to construct a chimeric enzyme with thermostable CBMs to see if this would improve the hydrolytic activity.

Acknowledgements

This work is supported by an NSERC Discovery Grant to Warren Wakarchuk.
We thank Dr. John Kelly, Simon Foote and Denis Brochu at the NRC Ottawa for supernatant proteomic data; Helen Stubbs for her assistance with the DSC; and Dr. Anthony Clarke at the University of Guelph for his generous gift of the Celf_3184 plasmid.

A void commen misteaks

- Make it long, or dense. 500 - 800 words. EDIT!
- Put colons in titles. Usually takes longer to read
- Put title in 'Title case' or ALL CAPS
- Sometimes this is specified by the meeting!

'Sentence case' is easier to read

DON'T

- Use bullets,etc, for section headers
  - size & bold is enough
- Use dark backgrounds
- Use red & blue near each other (green too)

- With Graphs -

use grid lines (this could be used….)
use coloured backgrounds
use boxes (well…there are exceptions here too)

For the Final Poster Presentation

- A ppt template will be provided with rough dimensions
- Print it in black & white
  - You can add colour by hand if you want
  - You won't lose marks if there is no colour
  - B&W is a lot cheaper - >$20. ($100 or more in colour)
    - Check Kinko's in the Plaza. Might be cheaper than on campus
  - See me if the $ is a problem. There are other options.
- Remember it can take a couple of days to get it printed so don't leave it until the last minute.

Powerpoint Presentations

Some of the same principles apply here, that we just went through for posters.

Think simple!

Oral presentation basics:

- Know your audience, and design the talk accordingly. Frequently you will have a mixed audience.

- Practice the timing, ***nobody likes to run out of time!***
  In general I plan on ~1 minute per slide

- Avoid putting lab jargon anywhere in your talk
  e.g. Names of stocks from the lab don't mean anything, rename things for the slides!

- Try to avoid presentation templates which set font size and type.

I generally avoid all templates for PowerPoint.

Things to avoid in your presentation.

- Make carefull use of colour. 2 is good
- Don't mix fonts.
- Don't use **weird fonts** (or things like shadows)
- Don't use coloured backgrounds (gradients etc..)
- Don't use slide transitions (they get really irritating)
- Don't use templates – they don't add to your data!

Things to put into your presentations

High quality images:

- use pictures at as high a resolution as you can get (try for larger than 640 x 480)
- images off the web are frequently too low res to look good

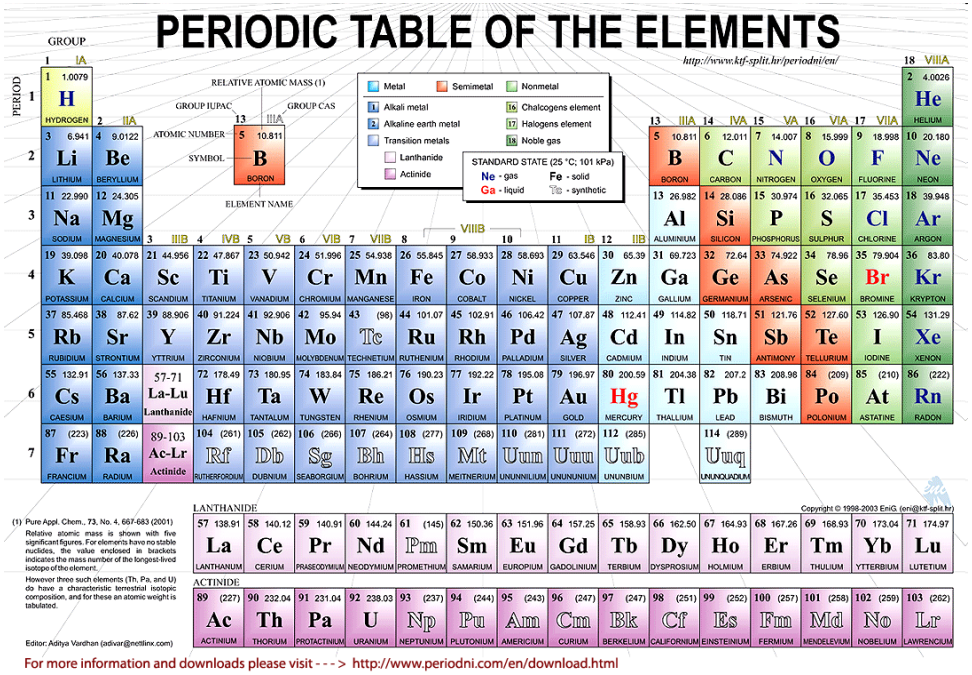
Things to put into your presentations 2

High quality images:

- images off the web are frequently copyrighted, make sure you quote the source, and obtain permission if possible.

Data slides: Tables

Symbol	Closing NAV	DIV	NAV Change	NAV Change %	Offer Price	10:00	11:00	12:00
FSAIX	27.13		+0.05	+0.18	27.97	27.05	27.06	27.01
FSAVX	25.90		+0.61	+2.41	26.70	25.62	25.65	25.66
FSRBX	34.63		+0.27	+0.79	35.70	34.51	34.44	34.44
FBIOX	48.08		+1.11	+2.36	49.57	47.72	47.81	47.94
FSLBX	43.65		+0.31	+0.72	45.00	43.53	43.42	43.38
FBSOX	12.50		+0.13	+1.05	12.89	12.43	12.42	12.42
FSCHX	40.81		+0.48	+1.19	42.07	40.55	40.61	40.61
FDCPX	29.42		+0.93	+3.26	30.33	29.02	29.07	29.10
FSHOX	27.93		+0.35	+1.27	28.79	27.69	27.64	27.78
FSCPX	21.52		+0.22	+1.03	22.19	21.40	21.38	21.38
FCYIX	13.00		+0.12	+0.93	13.40	12.92	12.92	12.94
FSDAX	43.68		+0.49	+1.13	45.03	43.30	43.34	43.37
FSDCX	12.95		+0.33	+2.61	13.35	12.76	12.78	12.79
FSELX	32.25		+0.92	+2.94	33.25	31.92	31.93	31.94



- Be careful with tables…remember our “keep it simple” model
- It is really easy to overload a slide with a table
- Isolate the data you want to show and only show those entries you will talk about
- Convert tables to graphs if you can

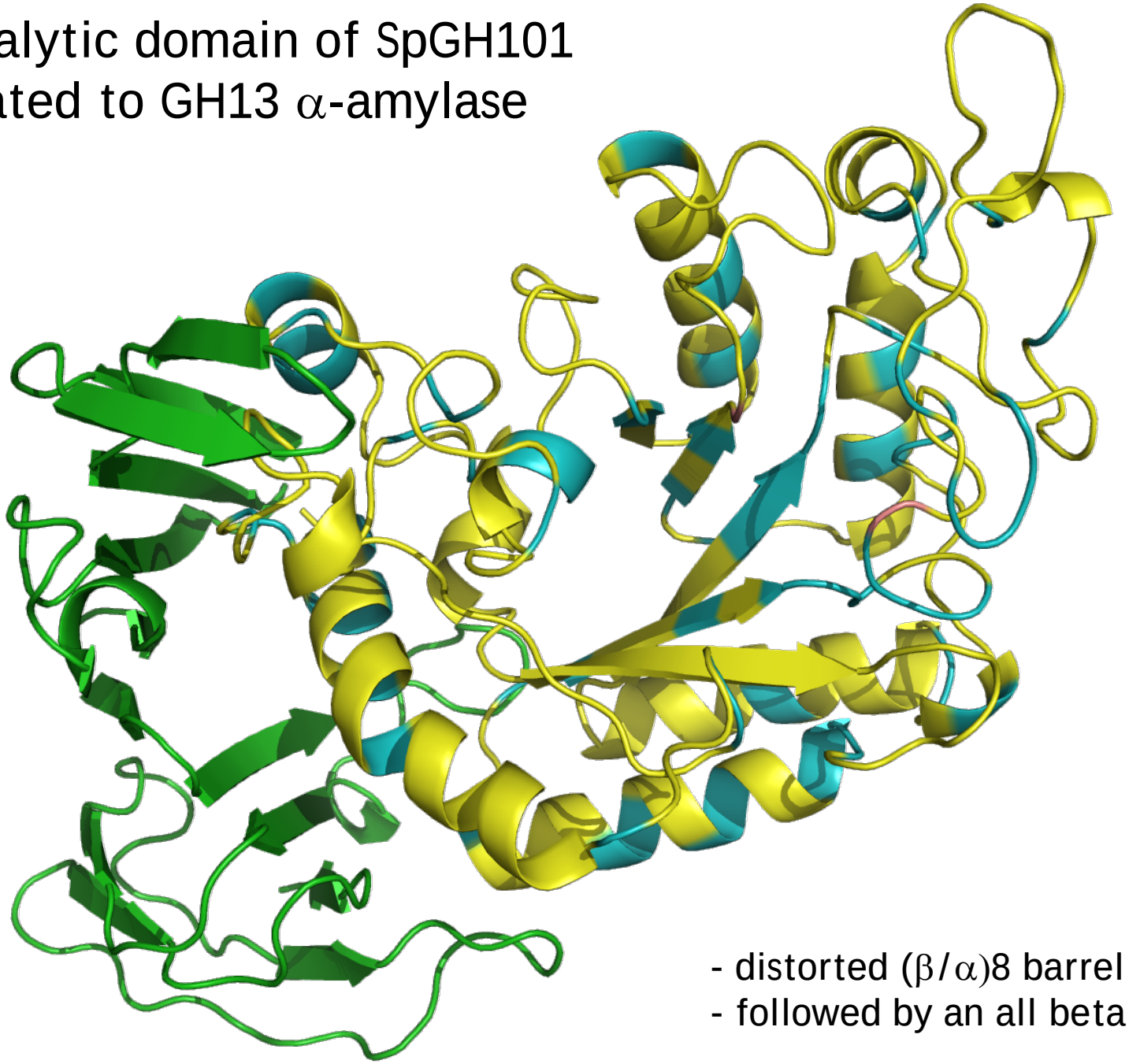
Getting more out of a slide with animations

- Use simple animations, this is like a slide transition the big motion, zooming or flashing ones are cool, but for a science presentation subtle is best.

- Use this to pace the introduction of data, to show a pathway, to build a structure

- Movies are very popular, but they present problems when your talk has to be on another computer (slow CPU, different version of PowerPoint, foreign OS, etc.)

The catalytic domain of SpGH101 is related to GH13 α-amylase



- distorted (β/α)8 barrel
- followed by an all beta domain

On-Virus Construction of Polyvalent Glycan Ligands for Cell-Surface Receptors

Eiton Kaltgrad, Mary K. O'Reilly, Liang Liao, Shoufa Han, James C. Paulson, and M. G. Finn

Presented by XXXX

Suppression of Tumour Growth and Metastasis in Mgat-5 Deficient Mice

Overview

•Malignant transformation is accompanied by increased β1, 6 GlcNAc branching of N-glycans in mature glycoproteins

•Mgat 5 catalyzes the addition of the β1, 6-linked GlcNAc

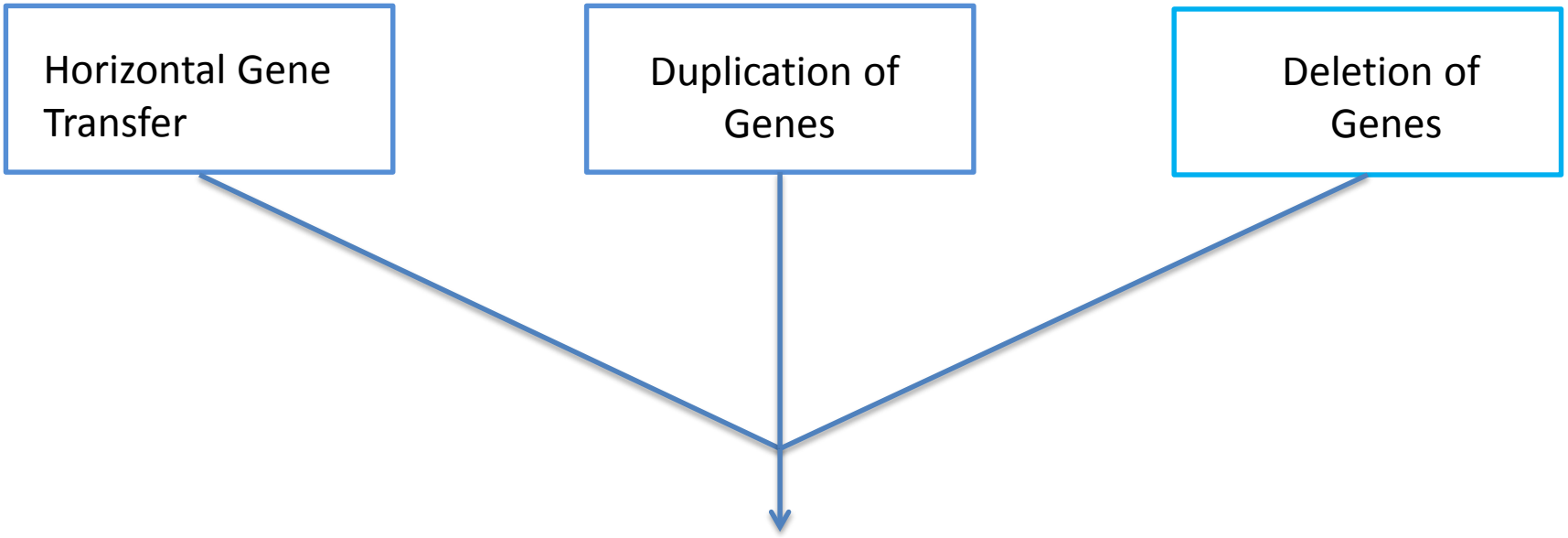
•Mgat 5 activity increases in fibroblasts and epithelial cell lines, WITH the expression of oncogenes such as *v-src,* and in cells infected with polyomavirus middle T antigen (PyMT) oncogene

•Mgat targeting vector created to replace the coding portion of the first exon of Mgat 5 with *LacZ* reporter gene

•Using Lectin p-HA western blot probing no Mgat 5 activity was detected in Mgat 5 -/- tissues, indicating that the mutation of the Mgat 5 locus had eliminated all the catalytic activity and Mgat 5 products in Mgat5 -/- mice

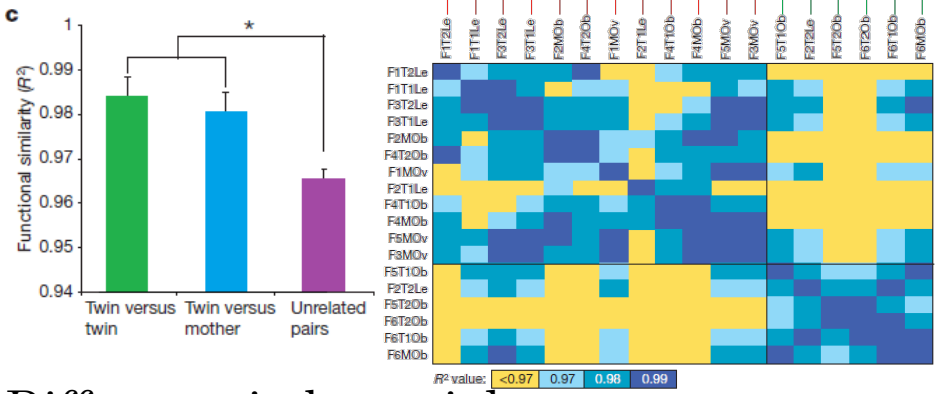
- Gene/Enzyme of interest: LgtK, an α-1,2-N-acetylglucosaminyltransferase.
- Previously, it was thought that the addition of an ethanolamine phosphate group at the O3 position on Hep(II) is necessary for the proper transfer of a GlcNAc residue to Hep(II).
- Experiments were conducted on LgtK knockouts and wildtype cells. Expressed and purified LgtK was used in an in vitro activity assay against the inner glycosylation core of LOS. Furthermore, synthetic substrates were investigated as it is very difficult to isolate pure, undistorted inner cores, and the LipidA component must be removed to prevent aggregation.

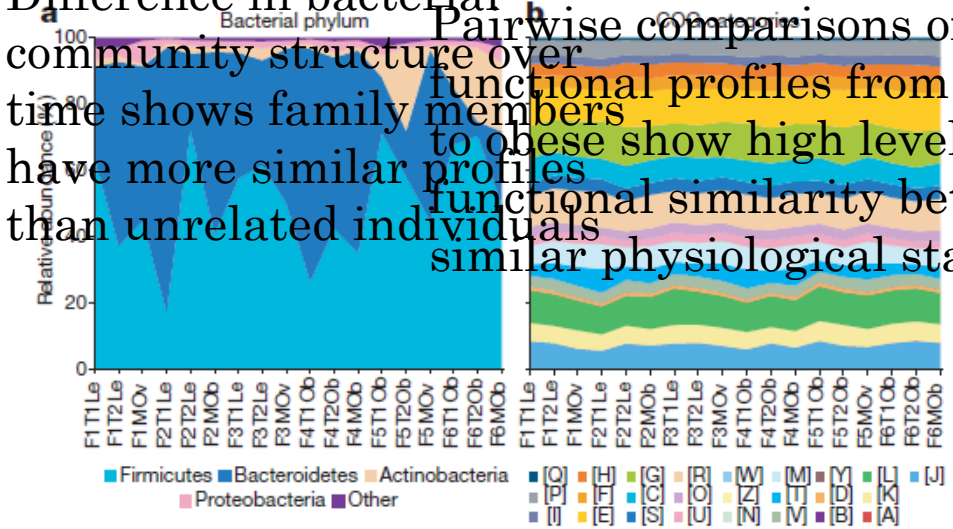
Gene Content Plasticity



Adaption to Environment = SURVIVAL

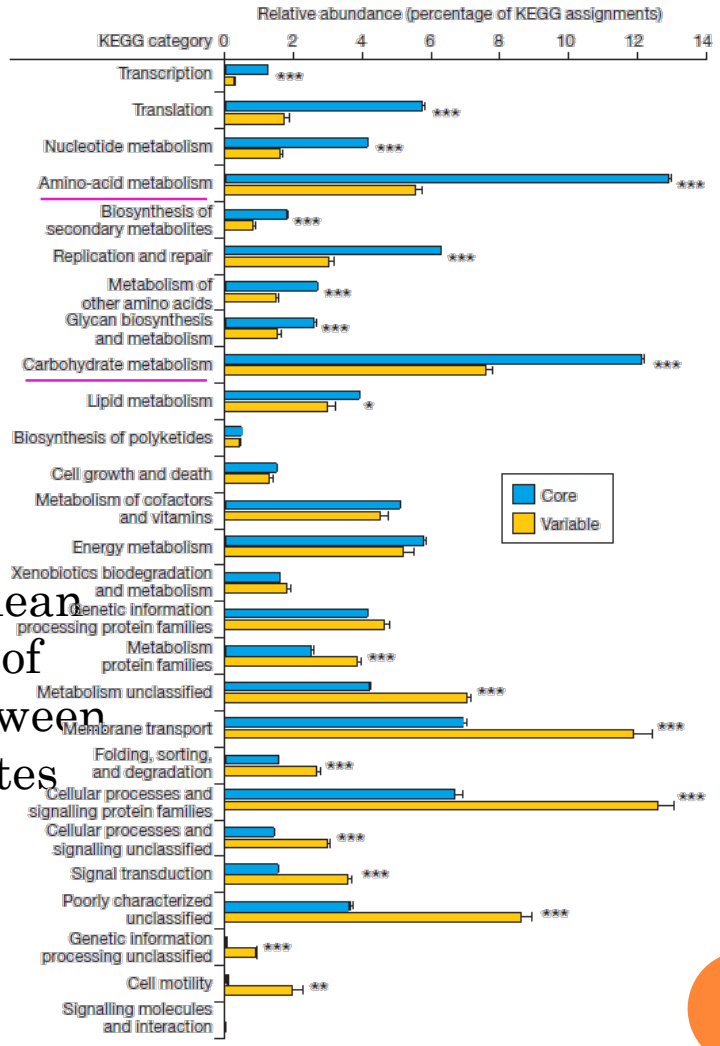
RESULTS





Difference in bacterial community structure over time shows family members have more similar profiles than unrelated individuals

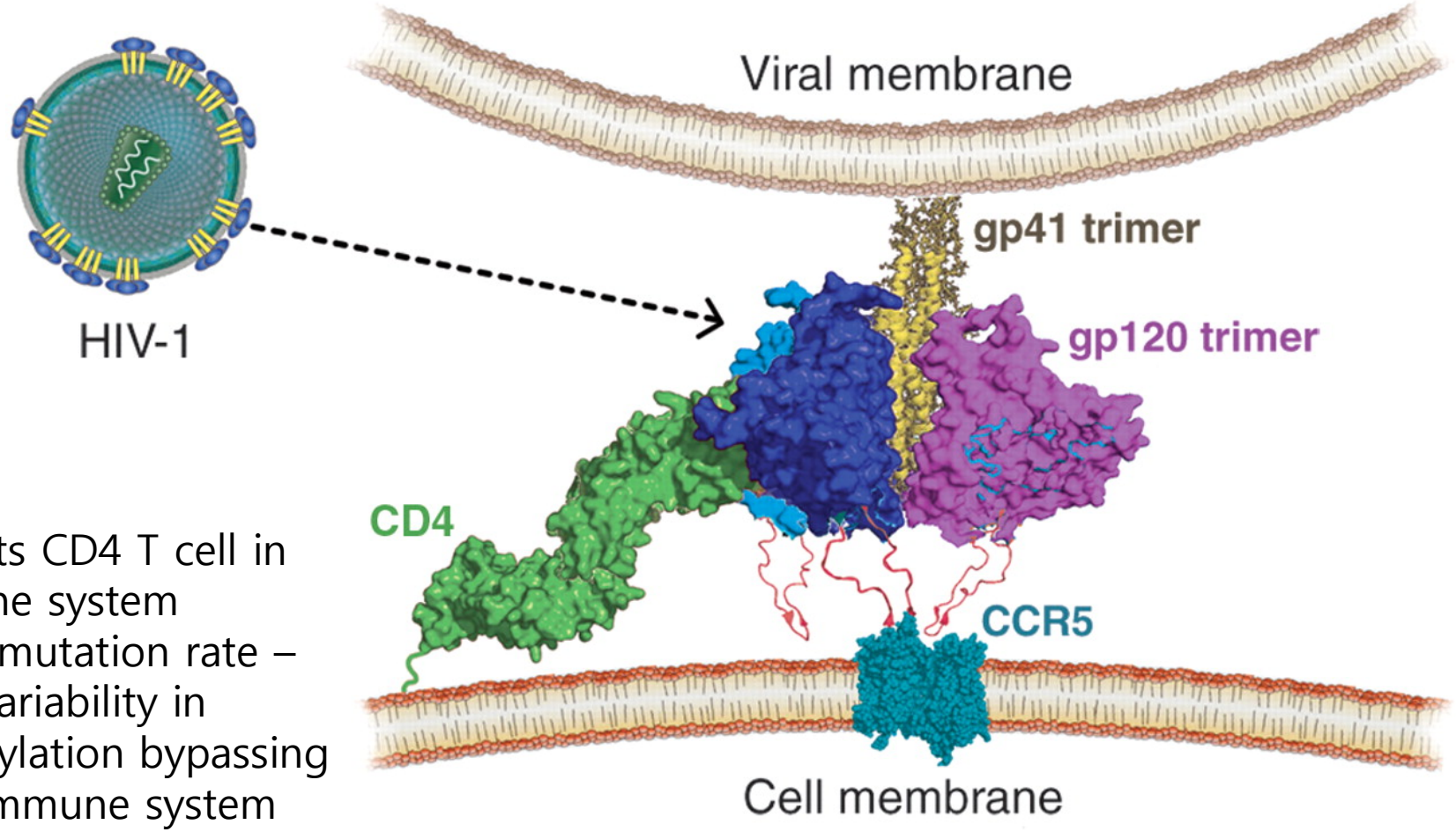
Pairwise comparisons of functional profiles from lean to obese show high level of functional similarity between similar physiological states



Relative abundance of phyla (a) and gene categories (b) across gut microbiomes

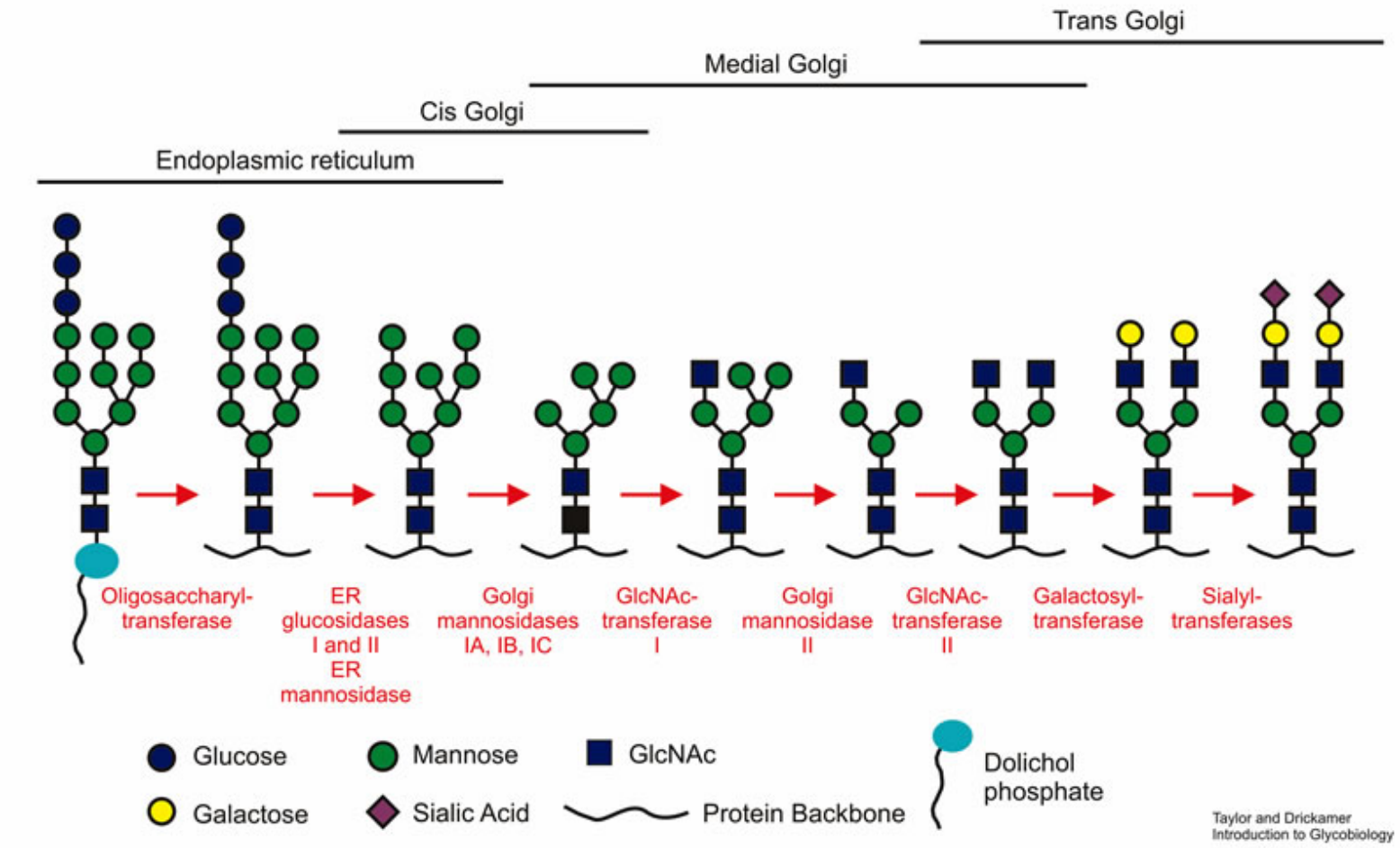
Core microbiome enriched for carbohydrate metabolism

HIV-1 Envelope spike



- Targets CD4 T cell in immune system
- High mutation rate – high variability in glycosylation bypassing host immune system "GLYCAN SHIELD"

Figure 3.6 Processing of an initial high mannose N-linked glycan to generate a complex glycan



BCH550/BIOL486 What is wrong with using this slide?

Peer Review

- Part of class participation grade

Peer review 1

BCH550 F2013 **Glycobiology**

ORAL PRESENTATION EVALUATION FORM

Presentation given by: _____ Date: 23/10-13

Preparation:
- Organization
- Understanding of material
  - Good Class
  - Explanation of cellulosome, Background too long!
  - Upregulated in crystalline cellulose.

+/- Subsites.

-Explanation of cellulosome, Background too long!
-Upregulated in crystalline cellulose

Presentation:
- Explained techniques and/or concepts clearly
- Clear visuals, speech, and grammar
  - Nice figures, good analogy, swiss army knives.
  - Pg.

-nice figures, good analogy, swiss army knives

General comments/impression
- ~~Needed to get into tal~~
- Went over time. Focus less on intro!
- Great speaking style + pace

-went overtime, focus less on intro
-great speaking style and pace

[redacted] On what basis?

Grade (%): A- A Name of evaluator: _____

Peer review 2

BCH550 F2013 **Glycobiology**

ORAL PRESENTATION EVALUATION FORM

Presentation given by: [redacted] Date: Oct 23

Structural insights into unique cellulase fold and.

Preparation:
- Organization
- Understanding of material

The pictures / figures of the slides probab
Good organization of slides although
slides dont have to be as lengthy. Two of
the same thing.

- Pictures needed references
- Introduction slides don't need to be so lengthy
- Two figures said the same thing

Presentation:
- Explained techniques and/or concepts clearly
- Clear visuals, speech, and grammar

verywell-explained introduction and objective / sig
work.
visuals were good athough complex protein nam
Mat. and Methods / Results were rushed and dif
Conclusions summed up the paper very well.
very well spoken and slides did not have grammer mistakes

- Very well explained into
- Visuals were good, although complex protein names were confusing
- Results were rushed and difficult to understand

General comments/impression

unfortunately went overtime. perhaps a bit
before final presentation
overall interesting worked, it seemed you knew
and background information very well

- Unfortunately went overtime – perhaps a bit more practice before presenting
- Overall interesting worked, it seemed you knew the paper and background information very well

Grade (%): 75 Name of evaluator: [redacted]

BCH550 F2013 **Glycobiology**

POSTER PRESENTATION EVALUATION FORM

Poster given by: [redacted] Date: Nov 20, 2013

Layout:

- Information content?
- Graphics?

- good info
- many graphics present

-good info
-many graphics present

Presentation:

- Clear story?
- Different from midterm presentation?
- Ability to answer questions?

- somewhat of a story
- some difficulty answering questions

-somewhat of a story
-some difficulty answering questions

General comments/impression

- What would have made this better?

- speak louder & enunciate

- speak louder and enunciate

On what basis ?

Grade (%): 80 Name of evaluator: [redacted]

BCH550 F2013 **Glycobiology**

POSTER PRESENTATION EVALUATION FORM

Poster given by: [redacted] : Nov 20th, 2013

Layout:
- Information content?
- Graphics?

- good use of graphics to explain concepts
- point form notes simplify information
- good descriptions of diagrams / explanations
- flow of information was OK
  - seemed to jump from topic to topic at time

- Good use of graphics
- Point form notes simplify info
- Good description of diagrams
- Flow of information was Ok – seemed to jump from topic to topic at times

Presentation:
- Clear story?
- Different from midterm presentation?
- Ability to answer questions?

- able to get basic idea of poster accross
  - finer points of experimental / background not
- seemed very similar to midterm presentation
  - not a lot of new information, though info pr
- able to answer a majority of questions
  - attempted to make connections to answer questio

- Able to get the basic idea across fairly easily – finer points…not conveyed
- Seemed very similar to midterm – not a lot of new information
- Able to answer a majority of questions
- Attempted to make connections to answer questions not known/covered

General comments/impression

- What would have made this better?

- new information covering a broader scope or more de
- more connections between findings/conclusions, backg applications/research would be interesting
  - more "why this is important" and "what does impact of presentation.

- New information covering a broader scope or more details on important info
- More connections between findings/conclusions, background information and future applications/research would be interesting
- More "why this is important" and what does it mean/signify would increase impact.

Grade (%): 75% Name of evaluator: [redacted]

Final thoughts:

- There are many examples of good data being lost because the presentation was bad.

- Good posters and PowerPoint talks are things that people will remember. (Think job interview!)

- You will get better with experience, but keeping presentations simple and to the point is never a bad idea. Resist the temptation to show <u>everything</u>!

Less can be more!