Designing Effective Research Posters

DR. VIRGINIA STORMER

The Basics

- Microsoft Power Point
- Adobe Illustrator, Photoshop, or InDesign
- Make sure your page size is set to the correct poster size (36x42 or 42x36)
  - In Powerpoint, enter the dimensions for the poster
    - Design > Slide Size > Custom Slide Size…or…
    - File > Page Set Up
- Print posters through The Studio (https://www.lib.utk.edu/studio/print-form/)
  - Print on coated or glossy paper—not plain
  - Pick up available at Hodges or Pendergrass Library

The Purpose

- Your poster should answer the following questions:
  - Why did you do this?
  - What are you adding to current knowledge?
  - What were your methods?
  - What did you find?
  - What do you recommend?

Formatting Titles and Subheadings

- Aim for around
  - 80-120 pt. font size for your title
  - 48-68 pt. for subheadings
  - 32-42 pt. for text
- Make sure to include group author names and affiliations
- Use generic/easy-to-follow subheadings
- Don’t allow your title to be too lengthy. Avoid colons if possible
  - Know your audience!

Formatting Text

- Use a serif font type for body text
  - SERIF
  - SANS-SERIF
- Keep your text to no more than 900 total words
- Use bullets, highlighting, headings, etc. to present information in a visually appealing and easily digestible manner
- Use lists instead of paragraphs

Formatting Graphs and Images

- LABEL EVERYTHING!
- Y-axis graphs should be horizontally aligned, if possible
- ALWAYS have a caption
- Make graphs easy to read
- Do not use colored backgrounds, grid lines, or boxes

Organizing the Poster

- Be aware of reader-gravity: order things top to bottom, left to right.
- Use a 3 or 4 column format and 3 or 4 row format
- Avoid hard to read colors or distracting color schemes
- White space is ok! Work to achieve balance
- Highlight the “takeaway” message
- Make sure to include your references & acknowledgements

O⁶-Benzylguanine Inhibits Tamoxifen Resistant Breast Cancer Cell Growth and Resensitizes Breast Cancer Cells to Anti-Estrogen Therapy

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MD ANDERSON CANCER CENTER ORLANDO

SUPPORTED BY THE CHARLES LEWIS INSTITUTE

Abstract

Endocrine therapies using anti-estrogens are least toxic and very effective for breast cancers, however, tumor resistance to tamoxifen remains a stumbling block for successful therapy. Based on our recent study on the involvement of the DNA repair protein MGMT in pancreatic cancer (Clin Cancer Res. 15, 6087, 2009), here, we investigated whether MGMT overexpression mediates tamoxifen resistance. Specifically, we determined whether administration of MGMT inhibitor [O⁶-benzylguanine (BG)] at a non-toxic dose alone or in combination with the anti-estrogens (tamoxifen/fulvestrant) curtails human tamoxifen resistant breast cancer cell growth. Further, we also determined whether BG sensitizes breast cancers to tamoxifen using tamoxifen resistant cells.

MGMT expression was found to be increased in breast cancer cells relative to normal breast epithelial cells. Also, MGMT levels were significantly higher in tamoxifen resistant MCF-7 compared to the parent cells. Silencing of the ER-α expression using a specific siRNA resulted in augmentation of MGMT mRNA and protein levels by 2 fold. We also observed an inverse correlation between MGMT and p53 levels in breast cancer cell lines; moreover, p53 downregulation was accompanied by increased MGMT expression. Other experiments showed that BG alone or BG in combination with tamoxifen or fulvestrant decreased ER-α expression, whereas tamoxifen alone and fulvestrant alone increased and decreased the same respectively. However, all these treatments increased the $p21^{cip1}$ mRNA and protein expression significantly. BG inhibited tamoxifen resistant breast cancer growth in a dose-dependent manner and it also resensitized resistant breast cancer cells to anti-estrogen therapy (TAM/ICI). These combinations also enhanced the cytochrome C release and the PARP cleavage, indicative of apoptosis. In breast cancer xenografts, BG alone or a combination of BG with tamoxifen or fulvestrant caused significant tumor growth delay and immunohistochemistry revealed that BG inhibited the expression of MGMT, ER- α, ki-67 and increased $p21^{cip1}$ staining. These findings suggest that MGMT inhibition may provide a novel and effective approach for overcoming tamoxifen resistance.

Introduction

Recent advances in breast cancer research have identified key pathways involved in the repair of DNA damage induced by chemotherapeutic agents. The ability of cancer cells to recognize DNA damage and initiate DNA repair is an important mechanism for therapeutic resistance and has a negative impact on therapeutic efficacy. A number of DNA-damaging alkylating agents attack the nucleophilic O⁶ position on guanine, forming mutagenic and highly cytotoxic interstrand DNA crosslinks. The DNA repair enzyme O⁶-alkylguanine DNA alkyltransferase (AGT), encoded by the gene MGMT, repairs alkylation at this site and is responsible for protecting both tumor and normal cells from alkylating agents. MGMT is expressed constitutively in normal cells and tissues. In breast tumors, MGMT gene expression is elevated and levels are up to 4-fold higher than in the normal breast. Interestingly, it has been shown that tamoxifen accelerates proteasomal degradation of MGMT in human cancer cells. In 1991, Pegg, Moschel, and Dolan observed that O⁶ benzylguanine (BG) inhibited AGT and potentiated the cytotoxicity of both chloroethylating agents and methylating agents. In a series of important observations, they fully characterized the interaction between BG and AGT and its therapeutic impact. They showed that BG binds AGT, transferring the benzyl moiety to the active-site cysteine [29]. The reaction is very rapid and more potent than any other previously known AGT inhibitor. BG is not incorporated into DNA in living cells and reacts directly with both cytoplasmic and nuclear AGT. Because BG is a psuedosubstrate for MGMT which results in the covalent transfer of benzyl group to the active site cysteine, the MGMT protein is degraded after each reaction. This stoichiometric reaction mechanism effectively depletes the AGT content in tumors and the associated repair of alkylation damage. BG is currently undergoing clinical trials in various cancers to increase the efficacy of alkylating agents.

Interestingly, several observations suggest an inverse correlation between the levels of MGMT and p53 tumor suppressor proteins where wild-type p53 suppresses transcription of human MGMT expression. Unfortunately, p53 function is often inactivated or suppressed in human cancers; therefore, restoration of wt-p53 activity is essential for the success of some treatments. However, whether or not this is mediated by suppression of MGMT expression has yet to be determined. To date, the cross-talk between MGMT and ER-alpha (and the link to p53 expression) has not been explored in drug (i.e., tamoxifen) resistant breast tumors. The anti-estrogen tamoxifen is the most commonly used treatment for patients with estrogen receptor positive breast cancer. Although many patients benefit from tamoxifen in the adjuvant and metastatic settings, resistance to this endocrine therapeutic agent is an important clinical problem. The primary goal of present study was to investigate the mechanisms of anti-estrogen drug resistance and to design new therapeutic strategies for circumventing this resistance. The results show that MGMT expression is increased in TAM-resistant breast cancers and inhibition of MGMT by BG significantly improves TAM-sensitivity.

Results

Prolonged Treatment of Tamoxifen Increases MGMT Expression: We developed a tamoxifen resistant MCF-7 cell line by using prolonged treatment of tamoxifen on the parental ER-positive breast cancer cell line, MCF-7. Tamoxifen-resistant MCF-7 cells proliferate at rates similar to the parental MCF-7. Prolonged treatment of tamoxifen onto MCF-7 cells increased MGMT expression compared to parental MCF-7 cells by 2 fold (Fig.1).

Knocking Down ERα Enhances MGMT Expression in Tamoxifen Resistant Breast Cancer Cells: It is not known whether ERα and MGMT transcriptionally regulate each other in tamoxifen resistant breast cancer cells. We therefore investigated whether down regulation of ERα has any effect on endogenous MGMT expression in these cells. As expected, downregulation of ERα using specific siRNA significantly reduced ERα protein levels in these cells. Western blot analysis was performed and the results in the left panel (Fig. 2A) shows that silencing of ERα increases MGMT expression in these cells, and interestingly, the results in the right panel (Fig.2B) show increased MGMT mRNA levels were increased as assessed by qRT-PCR. These data suggest that ERα–mediated signaling functions to repress MGMT gene expression in breast cancer cells.

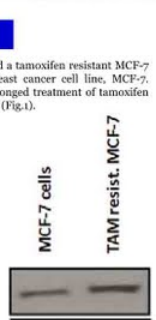


Figure 1. MCF-7 parental and tamoxifen resistant MCF-7 cell pellets were prepared, proteins were isolated and MGMT expression was detected by western blot analysis. Tamoxifen resistant MCF-7 breast cancer cells significantly increased MGMT expression compared to MCF-7 parental cells.

Transcriptional Regulation Between MGMT and p53: Previously, it was reported that p53 negatively regulates MGMT in breast cancer cells. Therefore, we addressed whether or not silencing the p53 enhances endogenous MGMT transcription. Tamoxifen resistant MCF-7 cells were transfected with either p53 siRNA (p53-KD) (Fig.2C) or MGMT siRNA (MGMT-KD) (Fig.2D) along with Non-specific siRNA (NS). MGMT expression was consistently increased in p53 knock down cells, with different experiments showing a – fold augmentation (Fig. 2A) and as expected, knocking down MGMT decreased MGMT transcription where as p53 mRNA levels were unaffected in MGMT knockdown cells (Fig.2D). These results confirm that p53 can regulate MGMT at the transcriptional level.

O⁶-Benzylguanine Plays a Dual Role in Tamoxifen Resistant MCF-7 Cells: Contrasting with the experiments above, next, we studied whether or not knocking down MGMT has any effect on ERα transcription. As expected, knocking down MGMT decreased MGMT gene transcripts. However, it was interesting to find that ERα gene transcription was also reduced after MGMT silencing. (Fig.2E). These data demonstrate that BG has the ability to attenuate the not only the MGMT, but also the ERα transcription, indicating a possible dual role for MGMT blockers in these breast cancer cells.

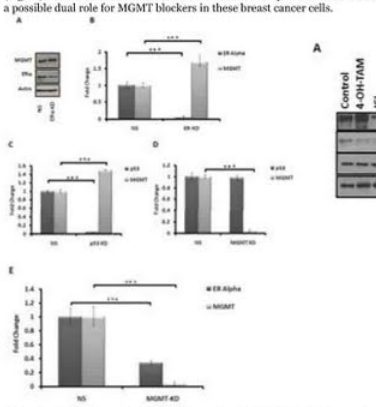


Figure 2. (A) Tamoxifen resistant MCF-7 cells were transfected with ERα siRNA (100nM) (ERα-KD) and NS siRNA (100nM) (NS), and cells were harvested 72h post transfection. Total proteins were isolated and ERα and MGMT expression was determined by western blot analysis. MGMT protein was significantly increased in ERα knock down cells (B) Tamoxifen resistant MCF-7 cells were transfected with ERα siRNA (100nM) (ERα-KD) and NS siRNA (100nM) (NS), and cells were harvested 72h post transfection. Total RNA was isolated and MGMT and ERα transcription was determined by qRT-PCR. MGMT transcription was significantly increased in ERα knock down cells. (C) Total RNA was isolated from non-specific siRNA (NS) (100nM) and p53 siRNA (20 nM) knock down tamoxifen resistant MCF-7 breast cancer cells. MGMT and p53 transcription was determined by qRT-PCR. (D) Total RNA was isolated from non-specific siRNA (NS) (100nM) and MGMT siRNA (100nM) knock down tamoxifen resistant MCF-7 breast cancer cells. MGMT and p53 transcription was determined by qRT-PCR. There is an inverse correlation between MGMT and p53 in tamoxifen resistant breast cancer cells (C & D).

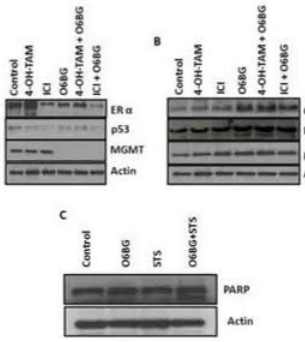


Figure 3. (A) Tamoxifen resistant MCF-7 breast cancer cells were treated in presence or absence of BG (50 μg) and 48h post treatment 4-OH-TAM (1μM), ICI (1μM) either alone or in combination with BG. 24h post treatment cells were harvested and proteins were isolated and western blot analysis was performed. (A) ERα, p53 and MGMT expressions (B) Cytochrome C, PUMA and p21 was determined by western blot analysis (C) tamoxifen resistant MCF-7 cells were treated with or without BG for 48h and later treated with staurosporin (5 μM/L) for 6 hrs PARP cleavage was determined by western blot analysis.

O6-Benzylguanine Modulates p53 Down-Stream Targeted Protein Expressions: Encouraged by the results reported, we investigated the effect of combination therapy on endogenous MGMT, p53, and ERα protein expressions. As expected, BG decreased MGMT expression, while combination therapy (4-OH-TAM or ICI combined with BG) significantly decreased both MGMT and ERα expressions. BG alone or in combination with tamoxifen or ICI decreased ER-α expression, whereas tamoxifen alone and ICI alone increased and decreased the same respectively (Fig.3A). p53 expression was slightly altered after ICI treatment. The reduction in p53 expression by ICI alone was reversed when BG was combined (Fig.3A). We investigated the effect of BG on proteins which are involved in cell cycle regulation, apoptosis in tamoxifen resistant breast cancer cells. All these treatments significantly increased the $p21^{cip1}$ protein expression (Fig.3B). PUMA expression was also increased with these treatments. Hence, PUMA may have translocated to the mitochondria, cytochrome C is released (Fig.3B), and apoptosis was triggered in these cells in presence of combination therapy. PARP cleavage is seen in BG treated cells in presence of staurosporin as an indicative of apoptosis (Fig.3C). Therefore, this data suggest that BG promotes cell cycle arrest and can induce apoptosis by modulating p53 function.

O6-Benzylguanine Modulated Transcriptional Targets in Tamoxifen Resistant Breast Cancer Cells: The effect of combination therapy on endogenous MGMT mRNA levels ws aso stuid. Quantitative real-time PCR (qRT-PCR) resulted that anti-estrogens (TAM/ICI) increased the MGMT expression while the combination therapy decreased it compared to control levels. ERα transcription was decreased compared to controls with all these treatments (Fig.4A). Surprisingly, p21 and PUMA mRNA was significantly increased in the presence of combination treatments (Fig.4B &C). These results suggests that p53 mediated target gene transcription was affected by the drug combinations in breast cancer cells (Fig. 3 & 4).

O6-Benzylguanine Enhances p21 Transcriptional Activity in Tamoxifen Resistant Breast Cancer Cells: In order to investigate the effect of BG on p53 function, we performed luciferase reporter assays. Tamoxifen resistant MCF-7 breast cancer cells were transfected with p21 luc promoter construct in presence or absence of BG (target gene of p53). These results clearly demonstrate that BG significantly enhanced p21 transcriptional activity by 4-5 fold in these cells (Fig.4D).

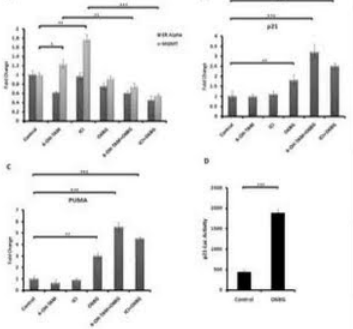
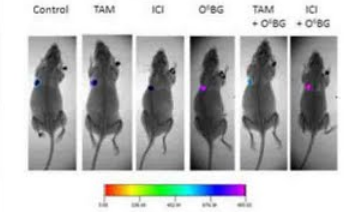


Figure 4. Tamoxifen resistant MCF-7 breast cancer cells were treated in presence or absence of BG (50 μg) for 48h and later 4-OH tamoxifen and ICI (1μM) was either alone or in combination with BG and 24h later cells were harvested and total RNA was isolated. (A) MGMT and ERα (B) p21 transcription (C) PUMA transcription was determined by qRT-PCR. 4-OH tamoxifen and ICI induces MGMT transcription. BG induced PUMA and p21 transcription. (D) Tamoxifen resistant MCF-7 breast cancer cells were transfected with p21-luc construct and 6h later treated with BG and 24h later cells were harvested. p21 transcriptional activity was significantly increased by BG in these cells.

O6-Benzylguanine Inhibits Tamoxifen Resistant Breast Cancer Cell Growth and Increase Resistant Breast Cancer Cell Sensitivity to Anti-Estrogen Therapy (TAM/ICI): Detailed necropsy revealed that all the mice had tumors in the breast. The data summarized in Table 1 show the daily BG alone or in combination with twice weekly tamoxifen/ICI significantly decreased median tumor volume and weight as compared with that seen in tamoxifen/ICI treated and control mice. The combination of BG with tamoxifen or ICI produced the greatest decrease in median tumor volume as compared with control mice (83.99 mm^3, 9.33 mm3 (TAM+BG), respectively; p< 0.0001); (83.99 mm^3, 31.60 mm^3 (ICI+BG), respectively; p<0.0001). Tumor weight was also significantly reduced in mice treated with combination therapy as compared with control mice (81.23 mg, 22.30 mg (TAM+BG), respectively, p<0.0005); (81.23 mg, 51.57 mg (ICI+BG), respectively, p<0.0005). (Table.1). Body weight was not changed among all treatment groups as compared with control mice. No visible liver metastases were present (enumerated with the aid of a dissecting microscope) in all treatment groups.

Histology and IHC Analysis: We next determined the *in vivo* effects of BG (alone or in combination) with tamoxifen/ICI. Tumors harvested from different treatment groups were processed for routine histological and IHC analysis. Tumors from mice treated with BG alone or in combination with tamoxifen/ICI exhibited a significant decrease in MGMT, ERα, ki-67 as compared with tumors treated with tamoxifen/ICI alone or control group. p53 expression was not much altered in these treatment groups. In sharp contrast, the expression of p21 was significantly increased in tumors from mice treated with BG either alone or in combination with tamoxifen/ICI. The images were analyzed by ImageJ (NIH) and MGMT, ERα, p53, p21 and ki-67 expressions were quantified by the ImmunoRatio plugin. (Fig.5).



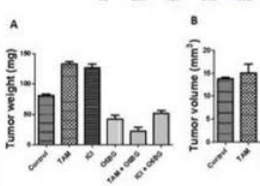
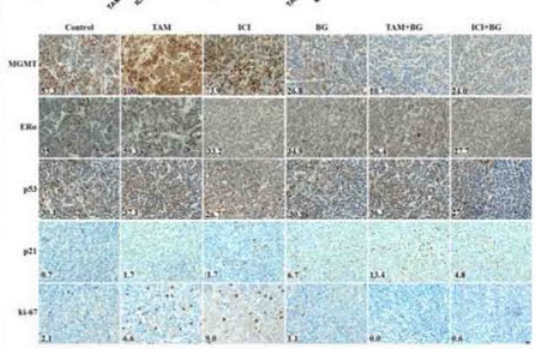


Figure 5. Tumors were harvested from control mice and mice treated with tamoxifen/ICI, BG, or both tamoxifen/ICI and BG. The sections were immunostained for expression of MGMT, ERα, p53, p21 and ki-67. Tumors from mice treated with BG either alone or in combination with tamoxifen or ICI had a significant decrease in the expression of MGMT, ERα and ki-67. p53 expression was not much altered in these treatment groups. In sharp contrast, expression of p21 was significantly increased in all these treatment groups compared to controls. Representative samples (40X) are shown.



Conclusions

1. In the present study, we observed that prolonged treatment with anti-estrogens causes drug resistance by inducing the DNA repair protein O⁶-methylguanine DNA methyltransferase (MGMT).
2. Decreasing the expression of MGMT by exposing breast cancer cells to BG sensitized these cells to anti-estrogen therapy (tamoxifen and ICI 182,780).
3. We also observed that combination therapy of anti-estrogens and MGMT blockers not only overcame the MGMT derived drug (tamoxifen and ICI) resistance but also increased the efficacy of anti-estrogen therapy by decreasing estrogen receptor expression and restoration of the functional activity of p53 in tamoxifen-resistant breast cancer cells.
4. Combination therapy inhibited tamoxifen resistant breast tumor growth *in vivo*.

Acknowledgements

We would like to thank the Florida Department of Health, Bankhead-Coley Cancer Research Program 09BN-09 for their funding of this project.

Parasite Spillback Effects on Native Communities in New Zealand Streams and Lakes

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Bridging Disciplines Program
Environmental Studies

[redacted]
The University of Texas at Austin Marine Science Institute

What is parasite spillback?

Parasite spillback is a process that describes the feedback of native parasites from new host species to native hosts.

- First, native parasites infect introduced or invasive host species.
- With a new host, parasites flourish.
- Now, parasites return to native species with increased infection and disease rates.

Salmonids Brown trout *Salmo trutta* (originating form Europe) and rainbow trout *Oncorhunchus mykiss* (North America) were first introduced to New Zealand waters in the late 19th century. Their effects on local and native stream communities as a non-indigenous species include lesser-studied effects such as parasite spillback and dilution.

Unpublished: Kelly, D.W., Paterson, R.A., Townshend, C.R., Poulin, R. & Tompkins, D.M. "Parasite spillback: a neglected concept in invasion ecology?"

Could parasite spillback be a cause of native species loss and local level extinction?

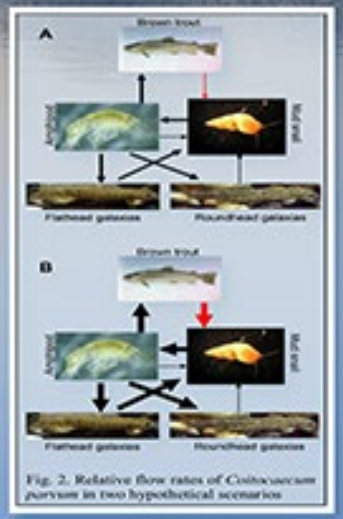


Fig. 2. Relative flow rates of *Coitocaecum parvum* in two hypothetical scenarios

Objectives

1. Test whether the presence of brown trout *Salmo trutta* and their parasite abundance is correlated to increased infection rates in four native species fish.
2. Identify for native fish and brown trout seasonal variations in infection intensity.
3. Understand the impact of parasites on host's condition, survival, and reproductive potential through captivity experimentation for all five host species. Parasite transmission to, establishment in, and mortality in different host species will also be identified.
4. Use multi-host and shared-parasite stochastic simulation models.
5. Consider global implications of this model by applying it to an Argentine system and conducting a literature survey of the abundance of shared parasites in native and exotic freshwater fish.

Unpublished: Kelly, D.W., Paterson, R.A., Townshend, C.R., Poulin, R. & Tompkins, D.M. "Is parasite spillback a cause of local extinction in native communities?"

Methods

- Analyze freshwater fish communities in lakes and streams
- Field surveys
- Host autopsies
- Infection trials
- Mathematical modeling

Presented at the Bridging Disciplines Program Poster Session and Reception
April 16, 2009

My Experience

I spent five months interning with this project, conducting various lab and field tasks. In the laboratory, I counted the invertebrates from lake benthic sediment samples. I also conducted lipid analysis on galaxids, brown trout, and bullys. In the field, I helped as we set nets and traps for fish. We also collected benthic sediment and zooplankton samples.

Discussion

Native species loss is a critical issue throughout the world in many different environments. This map from Conservation International shows biodiversity hotspots where over at least 70 percent of native species are already lost. The most biodiverse regions, including New Zealand, are also the ones most at risk.

Competition and predation are the traditional impacts of invasive species on native species, but disease driven impacts are becoming more widely recognized and researched. Whereas parasite spillover is already an accepted form of disease driven impact, parasite spillback can potentially be more widely used as a tool for describing and understanding impacts of invasive species and native species loss.

A parallel study with similar methods is currently being conducted by the same team of researchers in Argentina. Other areas of the world where parasite spillback has been researched include a study of competing native and invasive grasshopper populations in California. (Settle and Wilson 1990) With more awareness of this issue, more research and studies will hopefully begin and consider parasite spillback as a potential cause for native species loss, potentially helping reverse the trends in global hotspots.

Settle, W. H., and L. T. Wilson. 1990. Invasion by the variegated leafhopper and biotic interactions: parasitism, competition, and apparent competition. Ecology 71:1461-1474.

Acknowledgements

Professor [redacted] of the Evolutionary and Ecological Parasitology Group.
[redacted] of Landcare Research
Funded by The Royal Society of New Zealand Marsden Fund.

Assessing and Treating Speech Disorders in Children with Autism Spectrum Disorder (ASD)

[redacted]

Department of Communication Sciences and Disorders, The University of Texas at Austin

What is Autism?

According to the Centers for Disease Control and Prevention (CDC), "Autism Spectrum Disorders (ASD) are a group of developmental disabilities that can cause significant social, communication, and behavioral challenges." The CDC recently announced that 1 out of every 68 children and 1 out of every 42 boys has ASD.

Background and Introduction

Recent estimates suggest that 24-33% of verbal children with ASD demonstrate a speech disorder (SD) (Cleland et al., 2010), which is higher than total population estimates (4% by age 8) (Wren et al., 2009). However, treatment protocols remain understudied. This study compares the effects of three different approaches to intervention for treating SD in children with ASD in the following order:

- Explicitly targeting speech *(The Cycles Approach)*
- Targeting speech through language contexts *(Milieu Teaching)*
- Help the child build an awareness of phonological skills *(Metaphonological Awareness)*

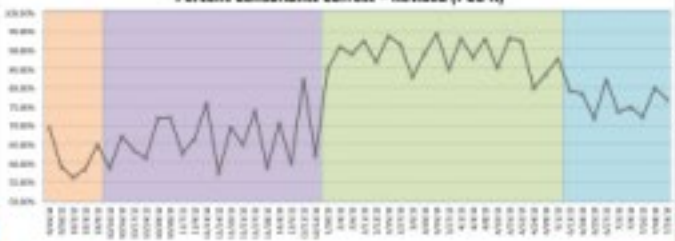
Methodology

This study uses a single subject experimental design: a combination of multiple baseline across participants and alternating treatments. Speech sample data will be collected and broadly transcribed using the International Phonetic Alphabet after each speech therapy session. Analysis of data will be based on calculations of:

- Percent Consonants Correct – Revised (PCC-R)
- Phonological Mean Length of Utterance (PMLU)
- Word Complexity Measure (WCM)

Alex: A Case Study

Percent Consonants Correct – Revised (PCC-R)



Phonological Mean Length of Utterance (PMLU)

Word Complexity Measure (WCM)

Baseline

Explicitly targeting speech **(The Cycles Approach)**

Targeting speech through language contexts **(Milieu Teaching)**

Targeting speech through awareness of sounds, syllables, and words **(Metaphonological Awareness)**

PCC-R, WCM, and PMLU scores show modest improvements when the Cycles Approach was implemented, significant improvements when Milieu Teaching was implemented, and significant regression when Metaphonological Awareness was implemented.

Results

- Modest improvements seen in Cycles compared to Baseline
- Significant improvements were evident in Language context condition compared to Cycles
- Significant regression in phonological skills in Meta-awareness condition

Statistical analysis showed a TauU of the following contrasts:

	Cycles Compared to Baseline	Milieu Compared to Cycles	Metaphonological Awareness Compared to Milieu
PCC	44% improvement	99% improvement	98% regression
PMLU	4% improvement	100% improvement	*Data not yet collected*
WCM	33% improvement	85% improvement	92% regression

Discussion

- Supports previous results by Koegel et al. (1998) suggesting that a natural environment/play is best
- Milieu seems most efficacious: clear effect and regression when this condition started and ended.
- Never expected scores to go down once they went up. Therefore, there is concern about the generalization of increased skills.
- Traits of autism is a topic of interest.
- Scores can be vulnerable to topic variations day by day. More participants are needed.

Acknowledgements

- Funding for advanced file comes from the Texas Speech and Hearing Foundation.
- Thank you to [redacted] for mentoring me throughout this project. Thank you to all of the undergraduate research assistants helping with this study.

Conflict Minerals: Should Businesses Avoid or Develop?

[redacted]

Supply Chain Management, McCombs School of Business

Abstract

- Conflict minerals are those that are sourced under armed forces
- Militia sell the minerals to smelters to be used
- In countries like the Democratic Republic of Congo (DRC), roughly 5 million civilians and workers have died working in these mines
- Armed militia in the DRC generate $180 million annually from exporting conflict minerals to smelters
- Profits are used to continue their supply of weapons

Research Objective

- Analyze the effects companies have on countries that source conflict minerals
- Determine the extent to which companies are held responsible for the treatment of workers in these mines
- Make recommendations for how businesses can create a more transparent supply chain

Background/Introduction

- In 2010, the US passed the Dodd-Frank Act
- This act required that companies list and audit all of their suppliers
- Businesses were prohibited from sourcing from countries that sold conflict minerals
- 2014 is the first year that companies are required to provide their first audit showing improvements in their supply chain towards less use of these minerals

Source: Source Intelligence

Source: Amnesty International

Benefits of Dodd-Frank Act

- Serves as guiding point for companies to base their plan of action against conflict minerals
- Forces companies to disclose what their suppliers use
- Eliminates a major source of income in countries, like DRC, that rely on profits from conflict minerals
- Creates more transparency in businesses

Negatives of Dodd-Frank Act

- Focus of economy should be on reforming financial and capital markets, not human rights
- Adds another layer of bureaucracy to business
- Shifts the balance in the supply and demand of companies
- Estimated initial compliance costs of $3 to $4 billion USD, and another $200 million annually thereafter

Methods for Verifying Conflict-Free

- Auditing: required of each supplier within a company's supply chain
- Supply certification: third-party auditors evaluate suppliers under standards that the company has already set out
- Bag and tag labeling: minerals are bagged and tagged "conflict free" immediately upon extraction
- Analytical fingerprinting: a method determined by a group of German scientists in which the region in which the minerals were extracted from can be determined

Recommendation

- Source majority of minerals from places that are not in conflicted regions
- Find political support from international amnesty to bring reform to the business process in these regions

Vices, Scapegoats, and Evil Forces:
Magic in the Works of Miguel de Cervantes y Saavedra, Juan Ruiz de Alarcón, and María de Zayas y Sotomayor

XXX
Department of Romance Languages and Literatures
Washington University in St. Louis

Background

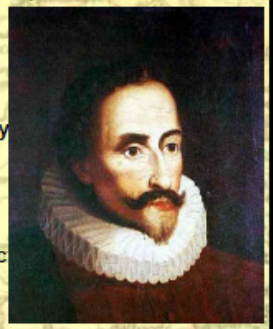
- The Golden Age of Spanish Literature ("Los siglos de oro") lasted from approximately 1492 until 1681.
- Magic was a part of daily Spanish life, especially in certain regions of the country.
- The Spanish Inquisition sought to eradicate popular practices of magic in order to assert control, adopting methods of fear.
- Moors were expelled from Spain by 1614.
- There was censorship of all published works in an attempt to control beliefs that contradicted Church doctrine.
- In order to publish works involving magic, authors had to reshape their ideas and present them in ways to evade censorship.

Focus of Research

- I explored works that were aimed toward two different audiences
- Cervantes and Zayas wrote prose for more educated audiences, while Alarcón wrote plays seen by the common man
- How did the depiction of magic differ between authors and modes of representation?
- How did censorship shape the authors' writing and depiction of certain groups?
- I focused on three main groups of people:
  - Innocent women
  - Moorish men
  - Real magic: witches, magicians, and the devil

Miguel de Cervantes y Saavedra

- Major satirist of the Golden Age
- Author of "El coloquio de los perros" as part of his Novelas ejemplares (1613)
- Evaded censorship by writing about magic in psychological terms
- The witches interact with the devil only under the influence of hallucinogens
- Despite not openly depicting magic, Cervantes utilizes talking dogs
- The high morals of the dogs counterac the supernatural aspect of their existence



Miguel de Cervantes y Saavedra

Juan Ruiz de Alarcón

- Born in Mexico, but spent the majority of his life in Spain
- Wrote many *comedias* (comedies), including:
  - *Quien mal anda en mal acaba* (1620)
  - *El prueba de las promesas* (1634)
- Stereotypes females as foolish and Moors as partners of the Devil
- In line with the morals of the Catholic Church in his writing
- Yet, the use of magic is justified in the case of a noble, concerned father

Juan Ruiz de Alarcón

María de Zayas y Sotomayor

- Early feminist writer of the Golden Age
- Examination of parts of her two major works:
  - *Novelas amorosas y ejemplares* (1637): "El jardín engañoso"
  - *Desengaños amorosos* (1647): "La inocencia castigada"
- Uses magic to denounce most men
- Empowers the virtuous, independent women in her works

María de Zayas y Sotomayor

Conclusions

- Cervantes appears to share the same views of magic as the Inquisition, but shows the humanity of the witches in his work.
- Zayas and Alarcón appear to share society's stereotypical views of Moors.
- Zayas uses magic in a feminist manner: desperate men use magic to achieve their goals, but virtuous women triumph.
- Alarcón demonstrates opposing views: Moorish men have connections with the devil, and men can use magic for good reasons.
- It is clear that the Inquisition did not always view the use of magic in literature as a threat.

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SILICATE CYCLING IN WASTEWATER DIATOMS

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Introduction

- Algae are good sources of material for biofuel, especially cellulose and lipids
- Algae rich in cellulose (e.g. green macroalgae like *Cladophora*) and lipids (e.g. diatoms like *Aulacoseira*) grow plentifully in domestic wastewaters
- Algae growing in wastewaters may help remove critical nutrients (nitrogen and phosphorus), and so help treat wastewaters
- Thus, algae could be useful in both wastewater treatment and biofuel production at treatment facilities
- BUT: Do wastewaters have sufficient amounts of other key nutrients (such as silica, Si) to support such a plan?
- Aim 1: To understand the Silicate availability of diatoms grown as a consortium in wastewater at Milwaukee's Jones Island Plant.
- Aim 2: To refine an existing mathematical model developed on an earlier project

Experimental Results

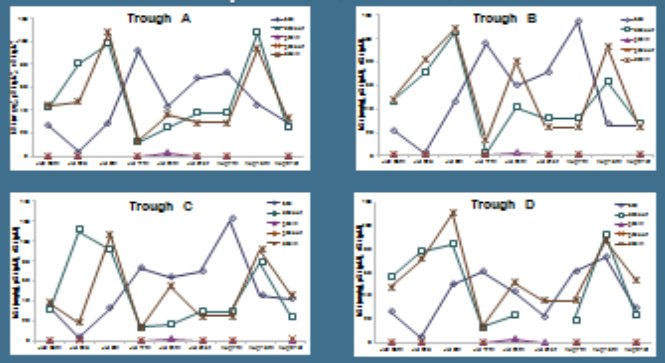

Figure 4: Troughs are denoted in Figure 1.

Model Implementation

- Model implementation used ordinary differential equations (Figure 2) and initial parameter estimates from previous work (Konjura et. al. 2015, unpublished)
- The base model was used to estimate the amount of Silicate expelled from algae
- Base model was designed to be a closed system of differential equations that followed parameter estimates from data analysis
- Both the original and new model was implemented through MATLAB
- New model flow diagram is displayed in Figure 3.
- New model is an open system of differential equations
- Initial parameter estimates were taken from original model
- The new model was designed to return the amount of Si that is contained in the algal bed given the amount of Silicate inflow and outflow.

Jones Island Trough System

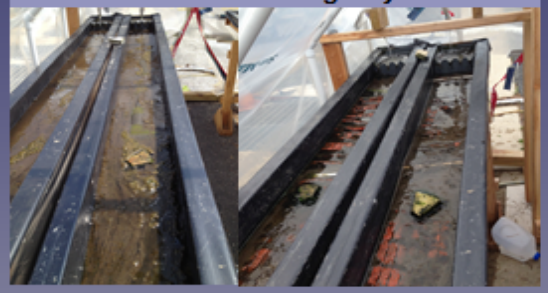

Figure 1. Trough system at Jones Island

Materials and methods

- Samples were collected ~weekly June 1-August 21st
- Water samples were taken from trough system (Fig 1.) from inflow of secondary-treated wastewater (in) and outflow at base of the trough (out)
- Water samples analyzed for: dissolved Si (dSi, measured with a molybdate color reaction, Parsons et. al. 1984) and particulate biogenic Si (pSi, digested in carbonate and analyzed as dSi, Paasche, 1980, Parsons et. al. 1984)
- Algal samples taken from troughs (completely cleaned weekly)
- Algal material used to measure biogenic Si (bSi, as for pSi)

Equations of the Mathematical Model

$$\frac{dDin}{dt} = -(1-\alpha) * \mu * b - \alpha * Dout$$

$$\frac{dDout}{dt} = Din * \alpha + \mu * b * \gamma$$

$$\frac{dPTin}{dt} = -\beta * \mu * b - (1-\beta) * PTout$$

$$\frac{dPTout}{dt} = (1-\beta) * PTin + \mu * b * k$$

$$\frac{dB}{dt} = \mu * b + \beta * PTin + (1-\alpha) * Din - \gamma * Dout - k * PTout$$

Figure 2: Silicate cycle model equations

Flow Diagram of the Mathematical Model

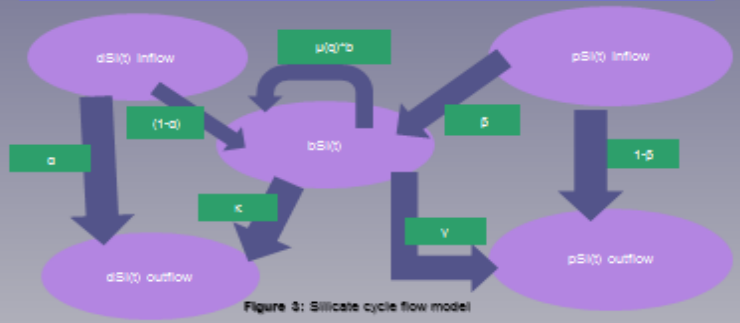

Figure 3: Silicate cycle flow model

Preliminary Data Results

- From Figure 4, it can be seen that in all of the troughs, there seems to be an inverse relationship between dissolved Silicate and biogenic Silicate.
- Particulate Silicate, both inflow and outflow had almost no impact on the system's flow, and growth of the organisms throughout the system.
- Diatoms are abundant in terms of biomass, and Si is an important nutrient in wastewater

Future Work and Conclusions

- Predictions are difficult because we do not have steady-state relations.
- Outflows are sometimes higher than inflow even though algae needs to take up Si to grow. It is unclear as to why this is, but it does make budgeting the Si difficult
- Multiple avenues have been thought of: Total Silicate, or Silicate scaled to other variables. Unfortunately these could add bias to our measurements.
- This trough system was designed in this fashion so that rural waste water treatment plants could implement this idea on a large scale.
- Algal growth and harvest on a large scale could significantly decrease the amount of farming and housing land used for the creation of biofuels, in fact, no additional land would be needed to grow and harvest the algae grown
- Biofuels are a clean alternative resource that necessarily replace fossil fuels

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Useful Websites

- Brand.utk.edu
- https://ugresearch.utk.edu/activities/eureca/
  - Be careful with other templates that may not be sized correctly!