

Academic Poster

Yesoda Bhargava

February 6, 2021

Introduction

- Posters are widely used in the academic community, and most conferences include poster presentations in their program.
- Research posters summarize information or research concisely and attractively to help publicize it and generate discussion.
- The poster is usually a mixture of a brief text mixed with tables, graphs, pictures, and other presentation formats.
- At a conference, the researcher stands by the poster display while other participants can come and view the presentation and interact with the author.

What makes a good poster?

- Important information should be readable from about 10 feet away
- Title is short and draws interest
- Word count of about 300 to 800 words
- Text is clear and to the point
- Use of bullets, numbering, and headlines make it easy to read
- Effective use of graphics, color and fonts
- Consistent and clean layout
- Includes acknowledgements, your name and institutional affiliation

Where to begin?

- Answer these three questions:
 - What is the most important/interesting/astounding finding from my research project?
 - How can I visually share my research with conference attendees? Should I use charts, graphs, photos, images?
 - What kind of information can I convey during my talk that will complement my poster?
- How to make an academic poster

What software can I use to make a poster?

- PowerPoint
- Adobe Illustrator, Photoshop and InDesign
- Open Source Alternatives : OpenOffice is the free alternative to MS Office (Impress is its PowerPoint alternative). Inkscape and Gimp are alternatives to Adobe products. For charts and diagrams try Gliffy or Lovely Charts. A complete list of free graphics software.

SUPPORTED BY THE CHARLES LEWIS INSTITUTE



*Cancer Research Institute of M.D. Anderson Cancer Center Orlando. †Texas Tech University Health Sciences Center, Amarillo, TX.

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 paucicentric cancer (Clin Cancer Res, 15, 6087, 2009), here, we investigated whether MGMT overexpression mediates tamoxifen resistance. Specifically, we determined whether administration of MGMT inhibitor or [O⁶-benzylguanine (BG)] at a non-toxic dose alone or in combination with the anti-estrogen (tamoxifen) could sensitize human tamoxifen-resistant breast cancer cell growth. Our results showed that the combination of tamoxifen with BG or MGMT inhibitor is more effective than tamoxifen alone in inhibiting growth of tamoxifen-resistant breast cancer cells.

new abstracts

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-damage-inducing 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

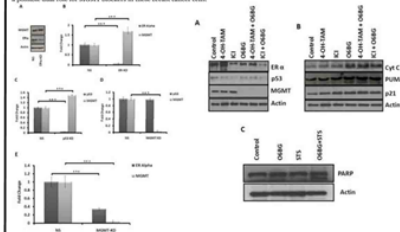
intrinsically, several of
proteins where wild-type
inactivated or suppress
function. However, whether or
the result, the crucial link
drug, the crucial link
tamoxifen) resistant breast cancer. The anti-estrogen tamoxifen is the most commonly used treatment for patients with
estrogen receptor positive breast cancer. Although many patients benefit from tamoxifen in the adjuvant and metastatic
disease, a subset of patients do not respond to tamoxifen. The primary aim of this study was to investigate the mechanisms of anti-estrogen drug resistance and to design new therapeutic strategies
circumventing this resistance. The results show that MGMT expression is increased in TAM-resistant breast cancers and

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 a fold (Fig. 1).

Knocking Down ERK1 Enhances MGMT Expression in Taxol/Resistant Breast Cancer Cells: It is not known whether ERK and MGMT transcriptionally regulate each other in taxol-resistant breast cancer cells. We therefore investigated whether down regulation of ERK has any effect on endogenous MGMT expression in these cells. As expected, downregulation of ERK using specific siRNA significantly reduced ERK protein levels in these cells. Western blot analysis was performed and the results in the left panel (Fig. 2A) shows that silencing of ERK 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 ERK-mediated signaling functions to repress MGMT gene expression in drug-resistant 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. 2A) or MGMT siRNA (MGMT-KD) (Fig. 2B) along with Non-specific siRNA (NS). MGMT expression was consistently decreased in p53 knock down cells, with different experiments showing a fold-silencing (Fig. 2A) and as expected, knocking down MGMT decreased MGMT transcription where as p53 mRNA levels were unaffected in MGMT knockdown cells (Fig. 2B). These results confirm that p53 can regulate MGMT at the transcriptional level.

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

[illegible]

06-Biotherapy/Signaling Modulators **5353 Down-Stream Targeted Protein Expressions.** Encouraged by the results reported by Bergström et al., we investigated the effect of combination therapy on endogenous MGMT, p53, and ERK protein expressions. As expected, BG decreased MGMT expression, while combination therapy (4-OH-TAM or ICI) combined with BG significantly decreased both MGMT and ERK expression above or comparable with control group. In contrast, p53 expression was increased after tamoxifen treatment and ICI treatment and down-regulated after combination therapy (Fig.3A). Surprisingly, p53 levels were also down-regulated after BG treatment alone. The reduction in p53 expression by BG 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 pro-apoptotic protein Bax expression and decreased anti-apoptotic protein Bcl-2 expression. However, no significant change in caspase-9, cytochrome C released (Fig.3B), and apoptosis was triggered in these cells in presence of combination therapy. TAM treatment alone in BG treated cells in presence of staurosporin as an indicative of apoptosis (Fig.3C). Therefore, this data suggest that

06-Benzylguanine Modulated Transcriptional Targets in Tamoxifen Resistant Breast Cancer Cells: The effect of combination therapy on endogenous MGMT mRNA levels

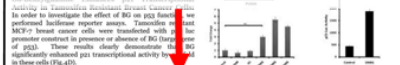
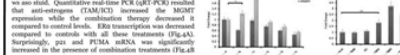
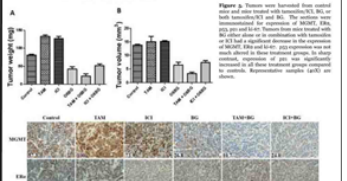


Figure 8. MCF-7 parental and tamoxifen-resistant MCF-7 cell pellets were prepared and total protein extracts were isolated and MG expression was detected by western blot analysis. Tamoxifen-resistant MCF-7 breast cancer cells significantly increased MG expression compared to MCF-7 parental cells.

Background: Polyamine Induced Tamoxifen Resistant Breast Cancer Cell Growth and Increase Resistance to Tamoxifen

Conclusion: Breast Cancer Sensitivity to Anti-Estrogon Therapy (TAM/ICI): Detailed necropsy revealed that all breast tumors in the breast. The data summarized in Table 1 show the tamoxifen alone or in combination with ICI treatment significantly reduced tumor size and weight compared to control group. This was even in tamoxifen treated and control mice. The combination of BG with tamoxifen or ICI produced the greatest decrease in tumor mass as compared with control mice (8.9g mean ± 0.33 mg/mg TAM/ICI, respectively; p < 0.0001). Tumor volume ± 31.66 mm³ (BG/ICI, respectively; p < 0.0001). Tumor weight was also significantly reduced with combination therapy mice (8.9g mean ± 0.33 mg/mg TAM/ICI, respectively; p < 0.0001). Body weight was not significantly different between groups compared with control mice. No visible metastases were seen in all treatments.

Crammed!



Conclusions

- 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).
- Decreasing the expression of MGMT by exposing breast cancer cells to BG sensitized these cells to anti-estrogen therapy (tamoxifen and TCI-86-96).
- We also observed that combination therapy of anti-estrogen and MGMT blockers not only overcame the

Acknowledgements

A SHARED PROPENSITY TOWARDS FOOD AND ALCOHOL

BACKGROUND

Overeating and binge drinking are two of the most common health problems among college students:



48%

report binge eating problems



63%

of females report binge drinking episodes



85%

of males report binge drinking episodes

ALCOHOL IS LIKE FOOD



- Alcohol is derived from sugar - similar chemical bases as food.
- Both eating and drinking alcohol activate dopaminergic pathways in the human brain.
- Addiction models have been applied to both food and alcohol use, as well as correlations between food and alcohol intake conducted in animal studies.

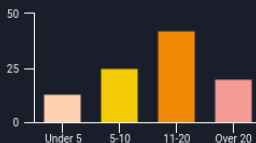


METHOD & MEASURES

200 UCLA Undergraduates (76% females, Mean Age = 22.1) filled out an online survey in one-sitting as part of a longer experimental study with the following exclusionary criteria:

- Less than 21 years old
- Self-reported history of eating disorders or substance abuse
- Abstinence from drinking beer
- A strict diet and food allergies to experimental stimuli

of times alcohol was consumed with a meal / month:



Alcohol Expectancy Questionnaire (AEQ)

A questionnaire measuring one's anticipatory effects of drinking and consuming alcohol.

Dutch Eating Behavior Questionnaire (DEBQ)

A questionnaire assessing one's eating behaviors (since expectancies predict consumption, food expectancies are implicitly implied).

RESULTS

Overeating and binge drinking are two of the most common health problems among college students:

AEQ / DEBQ	External	Emotional
Relaxation & Tension Reduction	320	240
Arousal & Aggression	240	220
Increased Social Assertiveness	250	169
Physical & Social Pleasure	210	114



CONCLUSION

The results support our hypothesis that Food expectancy is positively correlated to alcohol expectancy.

DEBQ External eating scale correlated to all AEQ scales, while DEBQ Emotional Eating scale only correlated to some AEQ scales.

External eating had a more consistent relationship with alcohol expectancies, where Emotional eating had a less consistent relationship.



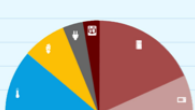
University of New York,
New York

STUDY CONDUCTED BY: Carla Ramirez, Francis R. Griffon and Elena Takiyama, UNIVERSITY OF NEW YORK
RESOURCES: Walsh, Kathy., Chan, Anthony. "The psychology of consumption and stress" The Pearson Journal, 2017. // Satoda, Mariana., Termitope, Janet., "Neuroscience of addiction and pleasure" The Science Review, 2016. // Jurgen, Hans., Lee, Penelope., "Consumption, Pleasure and Empowerment", The Arch Journal, 2020.

Figure 3: Sample poster for critique

Feedback

How you are doing: January 2012-February 2012



ENGAGE Website: Real-Time Energy Feedback down to Appliance-Level

ENGAGE is a real-time energy monitoring study at UCLA. It is currently among the largest behavioral studies in energy conservation in the U.S.

Residents at UCLA's University Village Apartments are currently having their apartments outfitted with a system to measure their electricity usage down to the appliance level. We take a behavioral science approach combined with enabling technology to test the effectiveness of financial and non-financial incentives.

<http://engage.environment.ucla.edu>



University Village Apartments

The Technology

Figure 2: Information Flow



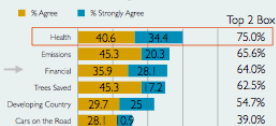
Social Comparisons



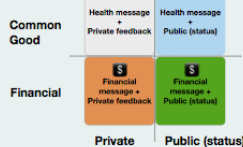
Public Information



Behavioral Science Messages: Pre-Test



2x2 Research Design



Econometric Model

$$Usage_{it} = \alpha + \beta \cdot P_{it} + \gamma \cdot I_{it} + \delta (P_{it} \cdot T_{it}) + \mu + \eta + \psi + \epsilon_{it}$$

Fixed treatment effect on population | Common time trend of treatment | Household controls | Psychological controls | Weather controls

Sample Messages

FINANCIAL "Last month you used 66% more electricity than your efficient neighbors. In one year, this will cost you \$34 dollars extra.

HEALTH "Last month you used 66% more electricity than your efficient neighbors. This results in 609 pounds of additional CO₂ emissions and air pollution that is known to contribute to health impacts such as childhood asthma."

How can we motivate or "nudge" people to conserve energy?

Figure 4: Sample poster for critique



Emily Anne Vaughn, Alfredo Cruz Ramírez, Luis Herrera Estrella
Departamento de Ingeniería Genética, Centro de Investigación y Estudios Avanzados del Instituto Politécnico Nacional,
Irapuato, Guanajuato, México
IHIMI International Summer Research Fellow, 2005



Our experiment examined the role that two different phospholipases play in the development of *Arabidopsis thaliana* root apical meristem architecture. The phospholipase (PLC and PLD) both synthesize phosphatidic acid (PA), a lipid second messenger, but go about it differently. We were interested in whether the PA derived from either PLC or PLD is more important for proper meristem development. We observed that certain PLC and certain PLD treatments make a difference in transcription factor levels (and thus cell identity) and root architecture. However, certain doubts about the specificity of the treatments mean that we cannot necessarily attribute our observations to our proposed pathway. The data collected suggest many interesting future projects.

The principal focus of the Herrera lab (Departamento de Ingeniería Genética, CINVESTAV-IPN) in Irapuato, Mexico, is how plants acquire phosphorus and use it efficiently. The lab uses *Arabidopsis thaliana* mutants and marker lines to study the physiological mechanisms by which plants obtain sufficient phosphorus (P) when P is

Interestingly, the abundance of phospholipids decreases during P-stimulation, while the abundance of other lipids increases. We hypothesize that phospholipids are broken down to phosphatidic acid (PA) during P-stimulation to make phosphorus available for metabolic functions, and/or to trigger stress-response processes. PA can be derived from the phospholipase C (PLC) or phospholipase D (PLD) pathway (Fig. 1, Fig. 2). Our project examines the effect of manipulating PA levels synthesized by different pathways on a group of mitotically inactive cells in the root apical meristem, called the quiescent center (QC) (Fig. 5).

Since this was a "fishing" experiment, rather than predicting which pathway will prove more important in conferring cell identity and organization, we hoped to disprove the null hypothesis that the pathways are equally

- ◆ What adaptive responses occur in the root meristem as a result of changes in FA biosynthesis, specifically in the quorum-sensing center (QCC)?
- ◆ Is the FA stat. transition an lipid secondary messenger to the QCC primarily derived from the PLC or the PLD pathway?

- Findings will contribute to the general understanding of iron nutrition metabolism and function, and the effects of CP-stimulation on *Artemia* hatching.
- Results may be useful for the control of iron nutrition.

We exposed 5–7 day-old *Arabidopsis* seedlings to four different treatments (Fig. 2) for 24 hours to visualize the differential effects of manipulating aspects of the two routes of PA synthesis. Effects were assessed based on QC identity, general root architecture (Fig. 5), and expression of a transcription factor marker called GUS. We used two promoter trap lines to stain specific regions of the root with GUS: *pLTT7-GUS* and *pQC46-GUS*. The *PLT* protein is expressed throughout the root apical meristem, most densely in the QC and columella. QC46 is a

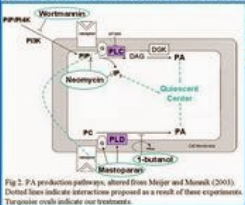
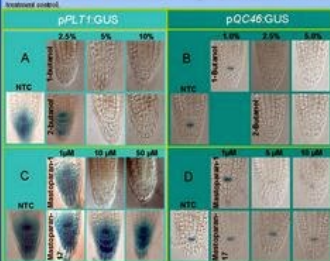
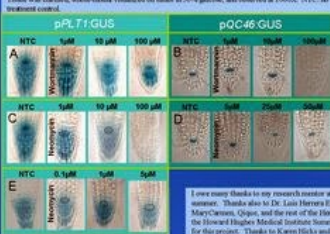
[illegible]

Fig 4. Results of treatment with various PLD pathway-manipulating reagents at different concentrations. Tissue was clarified, whole-mount visualized in slides in 50% glucose, and observed at 1000X. NTC: no



Abstract: GUS expression in *Arabidopsis thaliana* was greatly reduced at even small concentrations of butanol, yet roots did not display developmental alterations in other marker line (Fig. 1, A, B). Measurements above 2.5% are not reliable, as changes were observed in the 2-butanol control. **Key words:** Decreases in GUS expression were observed at the same concentrations in both marker lines, suggesting that MP1 similarly affects the transcription factors of both lines (Fig. 3, C, D). The effect of butanol on GUS expression was not observed in the 2-butanol control. In some roots, we observed loss of GUS identity, and use GUS expression in initial cells. **Key words:** **Wortmannine:** There was a more pronounced decrease in GUS expression in pGUS-GUS than in pGUS-GUS (Fig. 4, A, B). No disruption of meristem architecture was observed. **Key words:** A comparison of the effect of butanol on GUS expression in the two marker lines at higher concentrations than decreased GUS expression (Fig. 4, C, D). In pGUS-GUS we observed division of GUS cells (Fig. 4, C, D). This experiment was replicated with a five concentration gradient and

Fig. 5. Results of treatment with various PLC pathway-manipulating reagents at different concentrations. Time was clarified, whole-mount visualized on slides in 50% alcohol, and observed at 1000X. NTC, no



◆ Neomycin induces QC cells to divide. QC cells are mitotically inactive; they do not normally divide. This result inspired us to investigate the ways in which Neomycin might induce cells to move from G_0 to G_1 . Neomycin may influence cell cycle transitions by

◆ **GUS is expressed in the cortex/epidermal initials in pQC46;GUS, QC46**
transcription factors are only found in QC cells: the presence of GUS in initials, and not in the QC, suggests that QC identity was transferred to initials. This is unexpected, QC cell fate was determined by *W* expression in the QC. (Hirose et al., 2007)

◆ Diminished expression of GUS in the QC implies loss of QC identity, but not necessarily change in architecture. The QC is essential for conferring identity to surrounding cells, thereby regulating general root architecture. However, sometimes we observe a loss of QC identity without a change in GUS expression. Not unusual for a specific marker

◆ Treatments are non-specific. Most of the treatments have a variety of effects on trophoblasts. For example, Ebuhakabe et al. (2001) propose a relationship between P.D.

and the cytoskeleton. Therefore, our observations are not necessarily the result of manipulation of one of the PLC and PLD pathways. While we have not fully answered our

original questions, through these experiments we have synthesized better questions, and generated ideas for future experiments.

◆ Concentrations may not have been optimal. The literature on several of the treatments report conflicting ideal maximum and minimum concentrations.

◆ There may be cross-talk between the PLC and PLD pathways. MP-1 (which we used as a PLD treatment) increases IP₃ (a product of the PLC pathway) levels in soy cells (Legende et al., 1993). Also, there is evidence that MP-1 acts on all heterotrimeric G proteins, not only those that feed into the PLD pathway.

Fig. 5. A. Whole-mount analysis of Arabidopsis root apical meristem (modified from Scientific American, 2004). B. Root apical meristem analysis (modified from Asan et al., 2004). C. Typical pTET:GUS variation (in image) 1000X. D. Typical pC46:GUS variation (in image) 1000X.

◆ **Test confidence in Mastoparan.** We had assumed that MP-1 exclusively targets the PLD pathway, but there is some evidence that MP-1 non-specifically induces genes through heterotrimeric G proteins (Legender et al., 1993). To test this, we will cross a

If division of QC cells is observed in the absence of functional G proteins, then the MP-1 that induced the division is not introduced to the cell via G proteins. If division of QC cells is not observed, then the MP-1 is delivered by G proteins. The next step is to identify

◆ **Does Neomycin act by inhibiting Inositol 1,4,5 Phosphate (IP₃)?** We will treat QC46 GUS roots with Neomycin and IP₃. If the double-treatment restores the QC46 GUS phenotype, then the alteration was due to a decrease in IP₃ rather than a decrease in PLC.

derived PA. If the results of the previous experiment find that MP-1 acts indiscriminately upon heterotrimeric G proteins, then we would expect treatment of QC46-GUS with IP₃ and MP-1 to yield similar phenotypes.

I owe many thanks to my research mentor at CINVESTAV, Dr. Alfredo Cruz Ramirez, for his guidance and support in and out of the lab throughout the summer. Thanks also to Dr. Luis Herrera Estrella, Alejandra Chacón-López, Francisco Razo, Dr. Andrés Cruz Hernández, Anahí Pérez, Krutina and Mary Carmen, Qique, and the rest of the Hóvora lab and Iaqueto crew for their various and invaluable contributions. Thank you to Dr. Joan Szwedowski at the Howard Hughes Medical Institute Summer Research Fellows program, and the Kenyon Biology Department for giving me the opportunity and funding for this research. Thanks to Karen Hicks and Sam Shoshinski for assistance in the editing of this report and poster.



Creando Nuestra Salud: Findings from the 2012 Promotora Campaign Increasing Early Breast Cancer Detection Among Hispanic Women

Authors: Robin Lewy, MA, Francine Ricardo, BA, Diana P. Viviescas Vargas, MPH, and Angela Bakidis, MPH
Rural Women's Health Project, Gainesville, FL



Introduction

Breast cancer is the most commonly diagnosed cancer among Hispanic women in the United States and the leading cause of cancer death within this population. According to the American Cancer Society, one in ten Hispanic women in the United States will develop breast cancer during their lifetime (ACS, Cancer Facts & Figures for Hispanics/Latinos, 2012).

Additionally, breast cancer is more aggressive among Hispanics than non-Hispanic whites due to late entry into cancer care. Many Hispanic women are neither taught, nor practice the breast self-exam. This population faces additional barriers to accessing early detection screenings: language, transportation, socio-economic factors, lack of insurance and immigration status. (WHOIC, 2006; Nation's Health, 2006)

In 2008, to impact the factors that lead to the late detection of breast cancer among Hispanic immigrants, the Rural Women's Health Project (RWHP) developed the *Creando Nuestra Salud* program (Creating Our Health). The goal of *Creando Nuestra Salud* (CNS) has been to increase the health literacy of Hispanic women living in North Florida, through culturally-relevant education.

Objectives

The core objectives of the *Creando Nuestra Salud* program in serving Hispanic women are to:

- Influence the practice of monthly breast self-exams as a critical strategy for detecting breast irregularities
- Inform about early breast cancer detection screening guidelines
- Link women to clinics which offer clinical breast exams and mammography referrals

Methodology

The RWHP has collaborated with five partner organizations in six counties of North Florida: Alachua, Lake, Levy, Orange, Putnam and Volusia. The collaborators include two churches, one farmworker women's organization, five medical facilities and a county outreach program.

The RWHP trained 46 promotoras (Hispanic, Spanish-speaking health-care workers) in two, four-hour workshops held in each county. The promotoras were initially trained on "The Role of the Promotora," confidentiality & trust, breast cancer basics, breast self-exam (BSE), breast health screening recommendations, and accessing local clinical care and services for women with cancer. The promotoras were then trained to use the CNS tools, protocol and use of the teaching guide/contact sheet.



For the "orientaciones," promotoras utilized a teaching outline and contact sheet to gather data, the folioleto, a breast-teaching model and a necktie to educate on tumor sizes. Serving as a companion to the materials was the distribution of a county-specific Resource Guide to medical and social services.

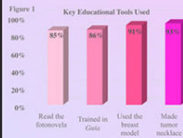
The promotoras spent two months educating their peers (10 contacts per month) and in their third month they focused on follow-up calls/visits "reminder notes" with each of the twenty women they oriented. All data was collected, coded and analyzed with SPSS.

Findings

In 2012, with the critical assistance of the promotoras, over 1,200 Hispanic women were educated in early breast cancer detection. Table 1 outlines the demographic characteristics of the women served.



Promotoras were trained in the use of the educational tools listed in the figure below (Figure 1). Using their teaching guides as a resource, the promotoras trained women with the tools they feel most appropriate to meet the woman's need. As seen above, the tools most commonly utilized were the breast model as well as the tumor necktie, used with 91% and 93% of women, respectively.



Prior to the CNS training, 54% of the participants had practiced a breast self-exam, however only 28% practiced the BSE monthly (Figure 2).

Post training, 97% of women reported that they felt more capable of performing the BSE, having learned steps and approaches previously not mastered, and they were committed to practicing the BSE monthly.

In the CNS follow-up, 80% of women reported having performed BSE within the three months following the CNS educational sessions (Figure 3).

Also in follow-up, commitments were made by participants to be screened for clinical breast exams (87%) and mammography (59%) within the next three months.

Prior to the CNS training, women said they faced obstacles to receiving a clinical breast exam or mammography.

The critical obstacles to accessing these services were cost (73%), transportation (22%), fear (12%) and embarrassment (10%). This was a multiple response question.

Post training, 20% of women needed additional assistance (navigation) with areas such as the cost of CBE or mammography, transport and translation. The CNS program assisted in navigating these women to the appropriate resources.

Table 1. Participant Characteristics

Demographics (n=1243)	
Age	
Under 40 years of age	23%
40 years and older	77%
Country of origin	
Mexico	64%
U.S.	7%
Central America	8%
Caribbean	16%
South America	4%
Years in the Country	
0-3 years	22%
4-10 years	24%
>10 years	54%
Insurance coverage	
No	66%
Family history of cancer	
Yes	14%

Figure 2. Breast Self-Exam Outcomes



Figure 3. Outcomes for Clinical Exams and Mammography



*Participants not screened in the previous year. Results from follow-up training group 47%.

Conclusions

The CNS findings demonstrate an increase in women's consciousness about the importance of early breast cancer detection.

Based on the obstacles faced by the women participants, due to their high rates of uninsured status and reported low screening practices prior to CNS, the breast self-exam is an achievable and vital detection method for this vulnerable population.

However, the program recognizes that the BSE is not enough. Therefore CNS dynamic education tools, which are left with each woman participant, promote the comprehensive screening recommendations and the program's linkage of women to clinical services. These materials can assist in their ability to enact their screenings.

CNS' lay-health worker strategy, and the inclusion of non-health community members, reduces the stigma around breast cancer, encourages women to prioritize their own health and elevates women's confidence in seeking services. These elements, when combined, increase women's probability of linking to care when breast irregularities are found.

Recommendations

The CNS campaign is a holistic intervention that can be replicated to increase participation in early cancer screening and early entry to care in other communities with similar characteristics.

Continued work with grassroots organizations and promotoras strengthens the community's social network. Additionally, distribution of community-specific Guide (Resource Guides) helps bridge women to locating a medical home, which is key as well as empowering a woman with a list of social services useful to her family priorities.

Understanding that prevention of cancer is related to one's diet, bleeding CNS with nutrition programs which emphasize the importance of weight control, physical activity and healthy dietary patterns, will assist these communities to reduce their risk of cancer as well as lowering their risk for other chronic diseases.

Acknowledgments

Project Lead Partners:

- Ana Botello, Alamos de Mujeres Activas
- Maria Granados, BS, Lake County Community Health Worker Program
- Meneles de Salud Alamos Health Care
- Community Health Initiative
- Iglesia Shalom
- Community Health Initiative Clinics
- Shands Mobile Outreach Clinic, UF
- Levy County Health Department
- Family Medical and Dental Center



Funders:

- American Cancer Society, Florida Division, Inc.
- Florida Breast Cancer Foundation
- Susan G. Komen, Central Florida Affiliate
- Wal-Mart Foundation, Inc.

References

- American Cancer Society. *Cancer Facts & Figures for Hispanics/Latinos 2012-2014*. Atlanta: American Cancer Society, 2012.
- Srigal C, Jermal A, West E, Cookman J, Smith R, Home H L, and Thun M (2006). Trends in Breast Cancer by Race and Ethnicity. Update 2006. CA Cancer J Clin. 56:165-163.
- Salazar, Mary K. "Hispanic Women's Beliefs about Breast Cancer and Mammography" Cancer Nursing. 19:6 (1996) 437-446

September, 2013

Figure 6: Sample poster for critique

Conclusion

- There is no absolute RIGHT or WRONG in Research Poster preparation. It is simply a matter of good or not-so-good and attractive or not-so-attractive. Thus, you can design your poster whatever you like, But please remember, you will beat the purpose of creating a Research Poster if nobody can understand or even interested to read it.
- Best way to learn is to make your research posters for MTP Project.
- Please write your queries at yesodabhargava@gmail.com. Thank you.