
Grace Under Fire: Oral and Poster Research Presentations

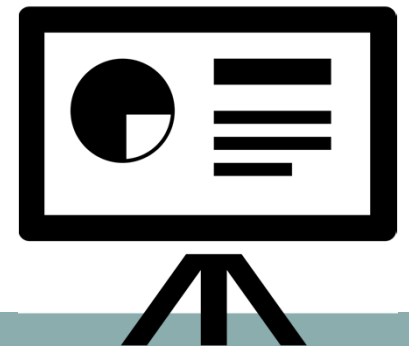
Alison Huang, MD, MAS
Publishing & Presenting Clinical Research
Spring, 2017

A solid teal horizontal bar spanning the width of the slide at the bottom.

Both oral and poster presentations



- Involve giving a focused summary of your research
- Use visual aids that should support but not dominate the presentation
- Are a prelude to interactive questioning by the audience



Before preparing an oral presentation



- What is your audience (scientists or clinicians, generalists or specialists)?
- How much time will you have (20 minutes, 12 minutes, 6-7 minutes)?
- What is the presentation session? What talks come before and after yours?
- What ideas, methods, or results will be the most challenging to communicate?

Basic principles of oral presentation slides



- Slides should complement your presentation but not replace it
- Use slides to provide structure to your talk and illustrate spoken points
- Remember that you yourself are your most important visual aid

Nuts and bolts of creating slides



- Keep it simple- avoid complex graphics, cluttered layouts, multiple visuals per slide
- Don't crowd your text- avoid more than 8 words per line, or 8 lines per slide
- Use the space you have, but retain white space between and around text
- Plain dark backgrounds with light text, or light backgrounds with dark text

Basics of font sizes and styles

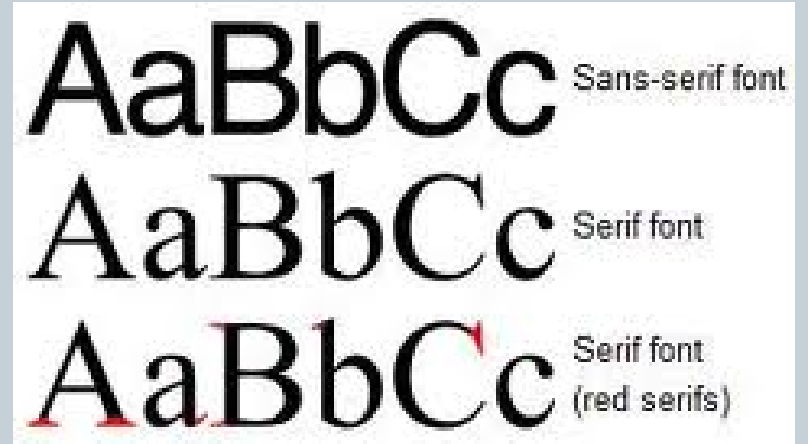


Minimum sizes (this is 28 point)

- This is 24
 - ✦ This is 20
 - This is 18

Serif vs. Sans serif fonts

- Is this easy for you to read?
 - ✦ How about this ?
 - Or this?



Bottom line for fonts



- 1-2 styles for the whole presentation (unless you're highlighting special text)
- Minimum 24 points font for main text, 32 points for slide titles
- Personal font style preference: sans serif font like Arial or Helvetica

But the best font choice is one where people don't notice the font but the message



US-CERT



Amit Yoran

US-CERT and the Multi-State ISAC are working together on a number of programs, including this webcast series, to help enhance our Nation's cyber security readiness and response. The Multi-State ISAC has recently become a member of the US-CERT portal, which provides a secure mechanism for sharing information between and among partners, improving cyber preparedness, readiness and response capabilities.

US-CERT also hosts a public website, at www.us-cert.gov, which provides a wealth of information regarding cyber security – helpful tips for protecting against cyber security threats; cyber security alerts and bulletins, as well as the ability to sign up to receive free cyber security alerts via email.

National Webcast Initiative



What was wrong with that slide?



- Avoid including multiple graphics or logos on a single slide
- Use plain backgrounds rather than distracting textured ones
- Don't include full paragraphs unless including a quote for emphasis

Avoid distracting animation



- Are you listening to the presenter or just watching this shimmer?
- Transition animations generally take too long
- Repeated animation forces attention away from speaker
- Too much eye candy suggests not enough substance

Guidelines for using animation



- Use only “appear” if you decide use any animation at all
- Don’t annoy your audience by forcing them to wait for each point to appear
- Be sure to practice your animations to avoid forgetting them on the day

Clues that something is wrong



- You have 15 minutes to talk, but you have 30 slides
- It takes you >10 minutes to make a non-graphic slide
- No results presented, but you're $3/4$ through your slides

Organizing your presentation



- Title and Credentials
- ~~• Conflicts and Disclosure~~
- Introduction or Rationale
- Design and Methods
- Research Results
- ~~• Limitations/Caveats~~
- ~~• Discussion/Implications~~
- Take-Home Points

How to introduce your research



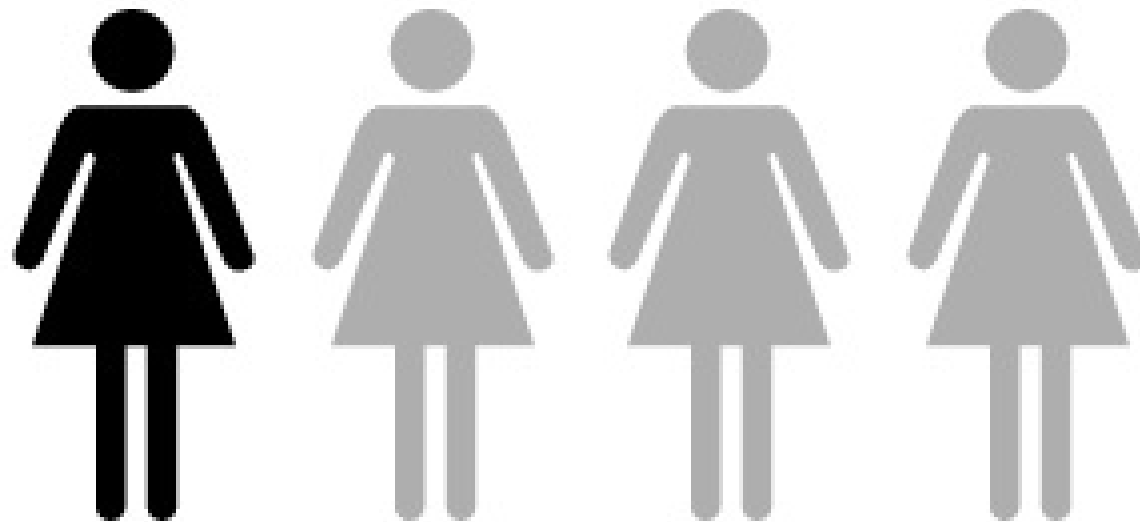
- Goal is to explain why the study you're about to present is important and relevant
- Consider starting with an attention-getting question, statement, or anecdote
- Opening slides should not only inform, but convey the motivation for the work

Types of openers



- Emphasize the gravity— “Every 15 minutes, someone dies of an alcohol-related motor vehicle collision”
- Pose a problem— “Four out of five menopausal women in the U.S. experience hot flashes, but to this day, we do not know why”
- Mark a transition— “You’ve been hearing about diabetes late in life; now I’m going to talk about diabetes in children for whom life is just beginning”

Use diagrams or pictures



One in four women will experience abuse in their lifetime

Tips for text-based introductions



- Stick with bullet points rather than complete statements
- Use short, strong statements in the active voice (no breaths needed)
- Continue as you begin– use a consistent style throughout the presentation – font, size, etc.

Should you read what's on your slides?



- Reading every single word on every slide can be boring and turn your audience off
- Most people can read faster than someone can speak (can't you?)
- But don't ignore what you've written (slide-speech dissonance)

Tables and graphics



- Use graphics judiciously to make your points
- Minimize extra data, decimal places, acronyms
- Always use data labels on graphs/figures
- Label percentages (%), standard deviations ($\pm$ SD)
- Make it easy to grasp the main point

Table 1: Contraceptive Methods in Family PACT Program by Demographic Characteristics, 2007 (%)

	Total	Oral Contra- ceptives	Injection	Ring	Patch	IUD	Barrier and/or EC	Total ^s (%)
Total	1,046,447	45.1	13.5	6.4	6.3	3.0	25.4	100
Race/ Ethnicity								
White (reference)	243,826 <i>*p<.05</i>	57.6	9.1	10.6	3.1	1.7	17.8	100
Latina	644,263	39.8***	15.9***	4.5***	7.5***	3.9***	27.9***	100
Black	53,212	35.8***	15.5***	7.3***	4.9***	1.1***	35.2***	100
Asian/ Pacific Islander	72,449	54.8***	7.0***	6.8***	7.9***	1.4***	22.1***	100
Other	32,697	50.3***	9.5*	9.0***	4.6***	2.3***	24.3***	100
Age (years)								
<20	213,047	47.6***	13.3***	6.5***	5.4***	1.1***	26.1***	100
20-29 (reference)	542,106	46.9	14.1	7.8	6.8	3.4	20.9	100
30-39	219,772	41.2***	13.5***	4.1***	6.8	4.1***	29.3***	100
>39	71,522	35.9***	9.7***	2.1***	3.6***	2.5***	45.6***	100
Parity								
0 (reference)	533,579	52.2	9.2	8.3	5.1	0.9	24.1	100
1	190,799	38.2***	18.9***	5.6***	7.7***	4.5***	24.9***	100
2 or More	322,069	37.0***	17.4***	3.6***	7.4***	5.7***	27.9***	100

Baseline sexual function scores did not differ by treatment assignment

Sexual function domain	Scale Range (worst to best)	Estradiol (mean $\pm$ SD)	Placebo (mean $\pm$ SD)	<i>P</i>*
Desire	0 to 100	35 $\pm$ 31	37 $\pm$ 29	.38
Satisfaction	0 to 100	65 $\pm$ 25	64 $\pm$ 24	.93
Orgasm	0 to 100	61 $\pm$ 27	65 $\pm$ 24	.53
Dryness	0 to 100	67 $\pm$ 27	66 $\pm$ 27	.80

*P-values derived from t-tests comparing mean scores between groups

Interpersonal Violence and PTSD are Associated with Genitourinary Dysfunction

Intimate Partner Violence

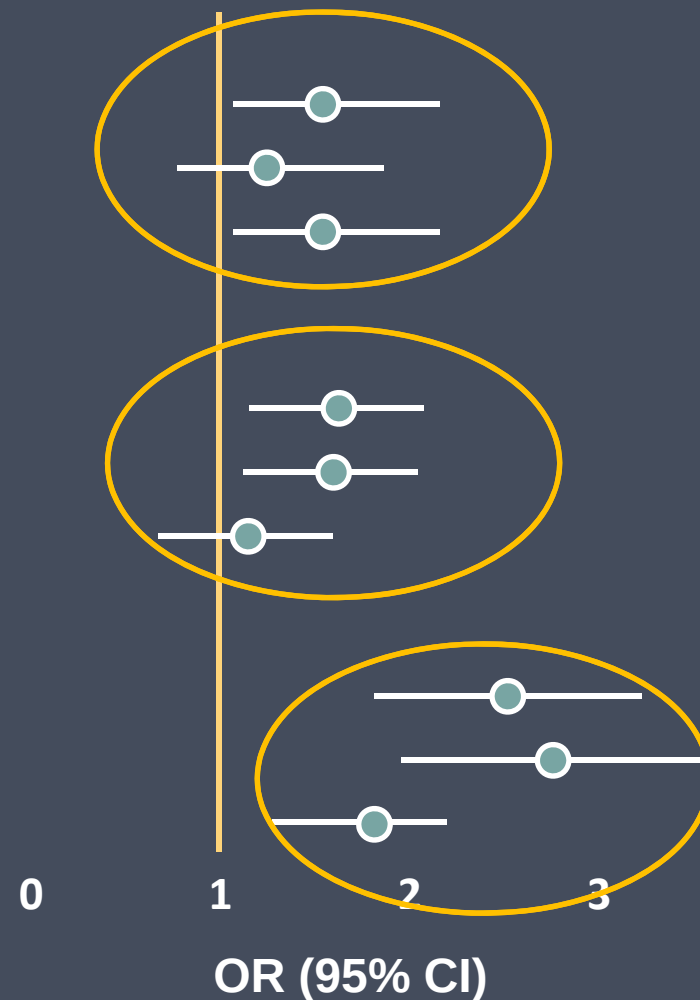
Vaginal pain with intercourse
Vaginal irritation
Urinary incontinence

Sexual Assault

Vaginal pain with intercourse
Vaginal irritation
Urinary incontinence

Posttraumatic Stress Disorder

Vaginal pain with intercourse
Vaginal irritation
Urinary incontinence



Proportion of participants who reported being at least “moderately” sexually satisfied

Total participants (N=1943)

P for trend

45 to 54 yrs old

54%

55 to 64 yrs old

27%

65 yrs or older

19%

<.01

Sexually active women (N=1161)

P for trend

45 to 54 yrs old

60%

55 to 64 yrs old

26%

65 yrs or older

14%

< .05

Sexually inactive women (N=742)

P for trend

45 to 54 yrs old

32%

55 to 64 yrs old

28%

65 yrs or older

40%

< .05

Explanatory results titles



- Life is Tough
- Life is Really Tough for Fellows
- Life is Even Tougher for Junior Faculty
- Life is a Piece of Cake for Senior Faculty

What are the most annoying things about the bad presentations you've seen?

- The speaker reads the slides to us 60%
- Text so small I couldn't read it 51%
- Complex sentences that I couldn't follow 48%
- Slides hard to see because of color 37%
- Moving or flying text or graphics 25%
- Overly complex diagrams or charts 22%

The wrap-up



- Focus on just a few concluding points (your audience won't remember more than one or two)
- Consider two-part wrap-ups— first summary then implications, conclusions then next steps,
- Prioritize synergy between spoken and written word in this section

Practicing the presentation



- First aim for overall message and time
 - Remember to speak slower than normal
- Then seek input about your slides
 - Fix your slides as you do so
- Then seek suggestions about the talk
 - Get “style” comments in private

Don't get spontaneous until you get good



- For short (<15 minute) presentations, ad libbing is dangerous
- Pay attention to transitions, both between slides and between concepts
- Memorize at least your opening statement, and possibly your entire talk

The presentation itself



- Arrive ~10 minutes early
- Introduce yourself to the chairs
- Position friends in the front
- Bring a glass of water
- Chuck the laser pointer

Prepare for opening variations



- Practice an opening that doesn't involve repeating your name and presentation title
- Gently correct any mistakes in your name, position, or institution
- Save your acknowledgments for the end of your presentation

If the unexpected happens, roll with it



- Technical powerpoint glitches are common
- “Presenter view” may not be available
- Alarms go off, and construction happens
- If you don’t mind, neither will your audience

Responding to hostile questioners



- Multi-part questions:
 - Pick the question you like best to answer first
 - Then say “What was the other question?”
- Hostile or argumentative:
 - “That’s an interesting point that requires in-depth discussion. Perhaps we can talk later. Next question?”

Responding to questions as ESL



- ESL: “Thanks for your attention. I will try to respond to your questions, but English is not my first language. Please speak slowly and simply.”
- All: “I'm sorry; I don't understand your question. Perhaps we can discuss this after the session.”

Research posters



- Usually contain WAY too much data and too little useful information
- Find out the poster board dimensions and make your poster as big as you can
- Put the key material at eye level in big fonts or simple tables and figures
- Your first draft will almost always include too much text

[illegible]

Author, Author, and Author
Address(es)

Introduction

Blah, blah, blah

Results

Blah, blah, blah

Conclusions

Blah, blah, blah

Materials and methods

Blah, blah, blah

Literature cited

Blah, blah, and blah. 2012. Blahing, blahing, and more blahing. *Journal of Blahology* 1:1-2.

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Journal of Blahology 1:1-2.
Blah, blah, and blah. 2012. Blahing, blahing, and more blahing.
Journal of Blahology 1:1-2.

Acknowledgments

Blah, blah, blah

Further information

Blah, blah, blah

Poster font sizes



- Poster title = 85 point
- Your name = 56 point
- Sub-headers = 36 point
- Main text = 24 point
- Legends, tables = 18 point

Always use upper and lower case and left-justify

Do's and Don'ts for poster text



- Do use bullet points rather than full sentences and paragraphs
- Don't use text if a graphic will work instead (even in the methods)
- Don't provide extra decimal places, duplicate N's and %'s
- Don't incorporate an abstract directly into your poster even if you're told to do so

Visual strategies for posters



- At least one figure on every poster– even if a table would make more sense
- Consider your color palette (2 to 4 contrasting but complementary colors)
- Rule of thumb– details should be clearly visible from six feet away

Visual appeal- color, graphics, space

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

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¹Cancer Research Institute of M.D Anderson Cancer Center Orlando ²Texas Tech University Health Sciences Center, Amarillo, TX



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 tamoxifen resistant breast cancer (J Clin Oncol. 2010; 28:6487), we investigated whether MGMT overexpression modulates tamoxifen resistance. Specifically, we demonstrated selective silencing of MGMT inhibiting O⁶-benzylguanine (BG) at a non-toxic dose alone or in combination with tamoxifen or 4-OH-tamoxifen (4-OH-TAM) resensitized tamoxifen resistant breast cancer cells to tamoxifen treatment.

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 breast cancer cells. Silencing of the ER-α expression using a specific siRNA resulted in a significant increase in tamoxifen sensitivity. We also observed an inverse correlation between MGMT and ER-α expression. ER-α knockdown in tamoxifen resistant cells was accompanied by increased MGMT expression. Other experiments showed that BG alone or BG in combination with tamoxifen or 4-OH-TAM decreased ER-α expression, whereas tamoxifen alone and 4-OH-TAM alone increased and decreased the same respectively. However, all these treatments increased the p53 mRNA and protein expression significantly. BG inhibited tamoxifen resistant breast cancer growth in a dose-dependent manner and it also resensitized tamoxifen resistant breast cancer cells to anti-estrogen therapy (TAM/ICI). These combinations also enhanced the cyclinase C release and the PARP cleavage, indicative of apoptosis. In breast cancer xenografts, BG alone or a combination of BG with tamoxifen or 4-OH-TAM caused significant tumor growth delay and immunohistochemistry revealed that BG inhibited the expression of MGMT, ER-α, Ki-67 and increased p53 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 all mammalian cells, and its levels are up to 4-fold higher than in normal breast cells. Interestingly, it has been shown that MGMT overexpression accelerates proteasomal degradation of MGMT and p53 tumor suppressor proteins where wild-type p53 suppresses transcription of MGMT mRNA. Unfortunately, p53 function is often inactivated or suppressed in breast cancers, therefore, p53 regulation is essential for the success of cancer 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-α (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.

Interestingly, several observations suggest that MGMT and p53 tumor suppressor proteins where wild-type p53 suppresses transcription of MGMT mRNA. Unfortunately, p53 function is often inactivated or suppressed in breast cancers, therefore, p53 regulation is essential for the success of cancer 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-α (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 a 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.

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 4-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. 2E). These results confirm that p53 can regulate MGMT at the transcriptional level.

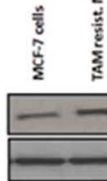


Figure 1. MCF-7 parental and tamoxifen resistant MCF-7 cell lines were treated with tamoxifen (100 nM) for 72 hours. MGMT protein levels were detected by Western blot analysis. Tamoxifen resistant MCF-7 breast cancer cells significantly increased MGMT expression compared to MCF-7 parental cells.

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. 3A). 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.

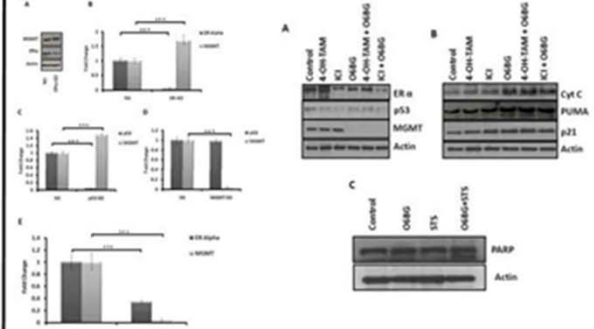


Figure 3. (A) Tamoxifen resistant MCF-7 breast cancer cells were treated with BG (100 nM) alone or in combination with 4-OH-TAM (100 nM) or ICI (100 nM) for 72 hours. MGMT and ERα mRNA levels were determined by qRT-PCR. (B) Total RNA was isolated from non-specific siRNA (NS) (control) and p53 siRNA (p53-KD) knock down cells. MGMT and ERα mRNA levels were determined by qRT-PCR. (C) Total RNA was isolated from non-specific siRNA (NS) (control) and MGMT siRNA (MGMT-KD) knock down cells. MGMT and ERα mRNA levels were determined by qRT-PCR. There is an inverse correlation between MGMT and p53 in tamoxifen resistant breast cancer cells (Fig. 3).

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^{ras} 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 was also studied. Quantitative real-time PCR (qRT-PCR) revealed 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 suggest that p53 mediated target gene expression.

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 p53 luciferase reporter construct in presence or absence of BG (target gene of p53). These results clearly demonstrate that BG significantly enhanced p21 transcriptional activity by 4-fold in these cells (Fig. 4D).

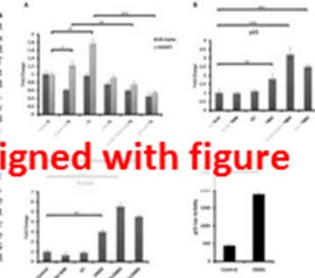


Figure 4. Tamoxifen resistant MCF-7 breast cancer cells were treated in presence or absence of BG (100 nM) for 72 hours and then 4-OH-TAM (100 nM) or ICI (100 nM) for 72 hours. MGMT and ERα mRNA levels were determined by qRT-PCR. (B) Total RNA was isolated from non-specific siRNA (NS) (control) and p53 siRNA (p53-KD) knock down cells. MGMT and ERα mRNA levels were determined by qRT-PCR. (C) Total RNA was isolated from non-specific siRNA (NS) (control) and MGMT siRNA (MGMT-KD) knock down cells. MGMT and ERα mRNA levels were determined by qRT-PCR. (D) Total RNA was isolated from non-specific siRNA (NS) (control) and p53 siRNA (p53-KD) knock down cells. MGMT and ERα mRNA levels were determined by qRT-PCR. There is an inverse correlation between MGMT and p53 in tamoxifen resistant breast cancer cells (Fig. 3).

O⁶-Benzylguanine Inhibits Tamoxifen Resistant Breast Cancer Cell Growth and Increases Resistant to Anti-Estrogen Therapy (TAM/ICI): Detailed xenograft revealed that all the tamoxifen resistant 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 mm³, 9.33 mm³ (TAM+BG), respectively; p<0.0001; 83.99 mm³, 31.60 mm³ (ICI+BG), respectively; p<0.0001). Tumor weight was also significantly reduced in mice treated with combination therapy as compared with control mice (83.23 mg, 22.30 mg; TAM+BG, respectively; p<0.0001; 83.23 mg, 22.30 mg; ICI+BG, respectively; p<0.0001). Body weight was not changed among all treatment groups as compared with control mice. No visible liver metastases were present in any of the treatment groups (microscope) in all treatment groups.

Histological Analysis: Tumor sections from 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 groups. 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 Immunohistochemistry plugin (Fig. 5).

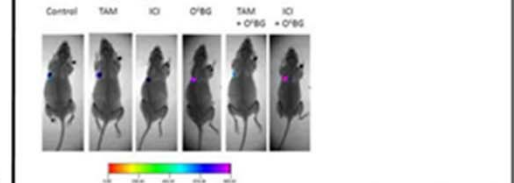
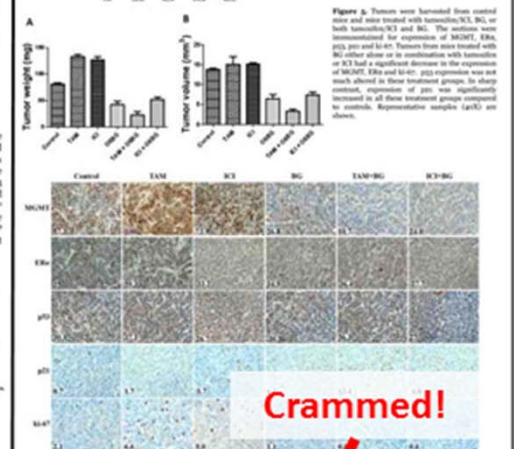


Figure 5. Tumors were harvested from control mice and mice treated with tamoxifen/ICI, BG, or both tamoxifen/ICI and BG. The sections were immunostained for expression of MGMT, ERα, p53, p21 and Ki-67. Tumors from mice treated with BG either alone or in combination with tamoxifen/ICI had a significant 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.



Crammed!

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) (Fig. 2A).
3. We also observed that combination therapy of anti-estrogen 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 restoring 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, Breast Cancer Research Program grants to the first author of this paper.

Visual appeal– color, graphics, space



PREFERENCES FOR HUMAN PAPILLOMAVIRUS (HPV) TESTING IN DIVERSE OLDER WOMEN

Alison Huang, Eliseo Perez-Stable, Sue Kim, Sabrina Wong, Celia Kaplan, Judith Walsh, A. Yuri Iwaoka-Scott, George Sawaya

BACKGROUND

- HPV testing is increasingly being used to determine the optimal Pap testing interval in older women
- Little is known about women's attitudes toward HPV testing or how attitudes influence screening decisions

OBJECTIVE

To examine preferences for HPV and concomitant Pap testing in diverse older women with access to screening

METHODS

- Women age 50 to 80 years were recruited from continuity practices near San Francisco from Oct 2002 to Jan 2006
- Women were given a brief introduction to the role of HPV in cervical cancer and the principles of HPV testing:

"A new cervical cancer screening test is available to see if you have a virus known as HPV (Human Papilloma Virus). This virus is known to cause cervical cancer. Here are some facts about HPV:

- The HPV test involves a similar process to getting a Pap test
- If your HPV test is abnormal, you have the virus and are at a higher risk than average for getting cervical cancer.
- If your HPV test is normal, you do not have the virus and are at a lower risk than average for getting cervical cancer.
- Most women will be exposed to HPV over their lifetime through sexual intercourse (it is a sexually-transmitted disease).
- There is no treatment for HPV. Testing for HPV is only used for assessing your risk of getting cervical cancer."

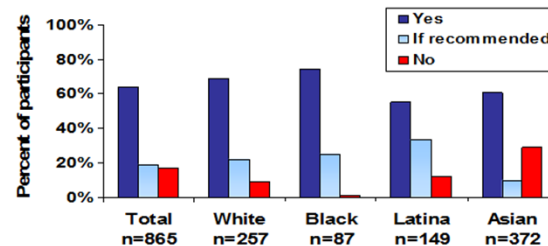
- Preferences for HPV & Pap testing were assessed in face-to-face interviews in English, Spanish, Mandarin, or Cantonese
- Logistic regression was used to identify predictors of HPV and Pap testing preferences

RESULTS

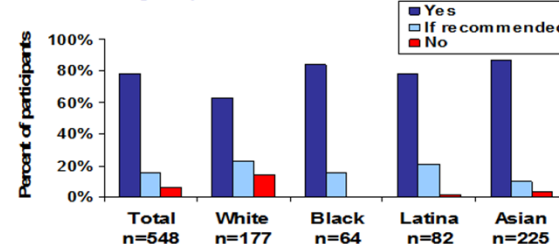
Demographic and Clinical Characteristics of Participants

Characteristic	Total N = 865	White N = 257	Black N = 87	Latina N = 149	Asian N = 372
Age in years (mean ± SD)	61 ± 8	60 ± 7	60 ± 8	63 ± 9	61 ± 7
Currently married	57%	54%	29%	42%	71%
College graduate	34%	70%	33%	12%	19%
Private health insurance	61%	82%	55%	40%	51%
Poor or fair overall health	45%	16%	43%	60%	59%
Income ≤ \$15,000/year	30%	12%	29%	45%	38%
Born in the United States	42%	89%	100%	10%	6%
Family history of cancer	39%	58%	54%	39%	22%
Personal history of cancer	15%	23%	16%	15%	9%

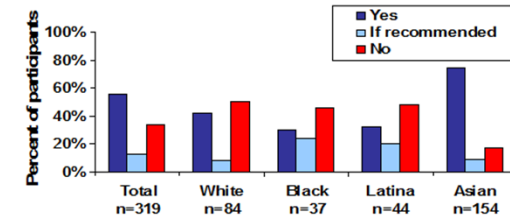
If the HPV test were available to you, would you want to be tested?



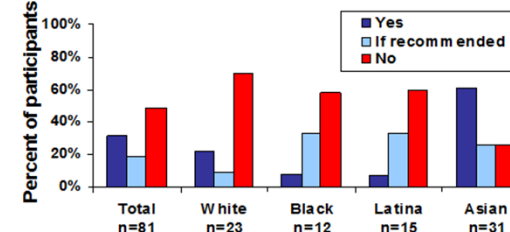
All women wanting testing: If you had an abnormal HPV test, would you want to get Pap tests more often than once a year?



Women < age 65 wanting testing: If you had a normal HPV and Pap test, would you be willing to get Pap tests every 3 years instead of every year?



Women ≥ age 65 wanting testing: If you had a normal HPV and Pap test, would you be willing to stop getting Pap tests?



Predictors of Testing Preferences in Multivariable Analysis

- Younger age was associated with interest in being tested for HPV
- With an abnormal HPV test, women were more likely to want more-frequent-than-annual Paps if they were Black or had a cancer history
- With a normal HPV & Pap test, women < age 65 were more likely to want annual Paps if they were Latina or U.S. born or had a Pap < 1 year ago
- No characteristics predicted willingness to stop Paps over age 65

CONCLUSIONS

- Among women age 50 to 80, preferences for testing vary significantly by race/ethnicity, place of birth, and cancer history.
- The majority of older women would want more frequent Pap tests if they had an abnormal HPV test with a normal Pap test
- A substantial minority of women would insist on at least annual Pap testing if they had a normal HPV test and normal Pap result



Center for Aging in Diverse Communities, University of California San Francisco

The “presentation” part of the poster



- Like oral slides, the poster complements the presentation but doesn't replace it
- Rehearse a one-line opener plus a 5- and 10-minute talk to give to passers by
- Visitors are more likely to remember you than your poster, so make yourself easy to remember

Tips on giving a poster talk



- Speak to your viewers as you explain your poster (don't talk to your poster)
- Point to relevant parts of your poster as you talk (“As you can see from this graph...”)
- If more viewers arrive in the middle of your talk, finish it for the first viewers first

What to do at a poster session



- Meet and greet your neighbors
- Invite (famous) strangers to discuss
- Greet your guests cheerfully
- It's your party– pass the drinks
- Hang in there – it's only 2 hours

Building an audience



- Ask your friends to come by in advance
- Volunteer to answer questions
- Offer to tell your story (but don't be pushy)
- If you can attract 1-2 people, more will come

Consider having on hand:



- A black pen and correction fluid in case you find a last-minute embarrassing typo
- Extra copies of your abstract (better than putting this in your poster)
- Your business cards or miniature versions of your poster
- A photograph of yourself to tack up at the side of the poster

Poster travel logistics



If you're going to junk it afterwards:

- Use the cheapest paper available (not glossy)
- Have it shipped directly to your conference

If you need to re-use it:

- Consider a cloth-based poster you can roll up or fold
- Go the old-fashioned way (separate pieces of paper)

Questions?

Final Project



Choose one of the following:
(due by 5 pm on June 5)

1) a research poster or oral presentation slide set appropriate for presenting a research project at a scientific or professional meeting

or

2) a draft full-length manuscript based on a recently completed research project, structured according to the guidelines of the expected target journal

Session 5 (June 6)



Running the Gauntlet: the Manuscript Review Process

and

**Journal Editor-in-Chief and
Senior Editor Roundtable**