BT 623: Research Methodology

Lecture 22: Poster

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P1-307 – P1-317
EPIGENETICS


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INTRODUCTION
Advanced ground-based telescopes rely on adaptive optics (AO) systems to correct the distortions in images caused by atmospheric turbulence. While an AO system is necessary to conduct diffraction-limited observations, each optical element in the AO system causes a small loss in throughput and adds thermal noise to the science images. For example, the Keck NIRC2 AO-system adds 7 extra reflections to the optical path and accounts for approximately one-half of the thermal background noise present in mid-IR images (4.7μm). Although small, this noise and throughput loss can prove detrimental to sensitive photon-limited science, especially in the infrared. Replacing the static secondary mirror with an adaptive secondary mirror (ASM) provides a method to integrate the AO system directly into the telescope, streamlining the optical path and reducing the thermal background and throughput loss.
AO
Keck-II AO photos of Uranus (2.2 μm)
We are testing deformable mirror prototypes made with HVR actuators
FLASH Deformable Mirror: Performance Testing Results
U of Hawaii 2.2m Telescope
UH 2.2m
ASM technology is becoming more accessible because of a new style of actuator
TNO Hybrid Variable Reluctance Actuators
SPIE
MEMBER
SESSION CHAIR
Elbow or Fist Bumps OK


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Research Poster:

A **research poster** is a visual and concise summary of a research project or study, typically presented at academic or professional conferences, seminars, or symposia. It provides an overview of the key aspects of the research, including the problem or question, methods, findings, and conclusions.

The poster format combines text, images, graphs, and tables in a layout that is easy to understand at a glance. Unlike full-length oral presentations, research posters allow researchers to summarize their work in a brief, visual form.

The main purposes of a research poster are:

Communication: A research poster serves as a tool to clearly convey complex research findings to a diverse audience. It distillS the essential components of any research into an easily digestible format, making it accessible to both experts and non-experts.

Networking: Poster presentations provide opportunities for researchers to connect with others in their field. By presenting their research visually, presenters can engage in conversations with conference attendees, peers, and professionals who share similar interests or may collaborate in the future.

Feedback: Presenting a poster allows researchers to gain constructive feedback from experts, peers, and attendees. This feedback can be invaluable for refining the research, addressing potential gaps, or generating new ideas for future projects.

Research posters are effective tools for communicating research findings, facilitating networking, and obtaining feedback in a visually engaging and concise manner.

Structure of an Effective Research Poster

Title: Clear, concise, and catchy. It should grab attention and give an idea of the research topic.

Introduction/Background: Briefly describe the research problem or question.

Objectives/Aims: State the research goals or hypothesis.

Methods: A concise description of the methodology used, often in bullet points.

Results: Present key findings visually (graphs, charts, or tables).

Conclusion: Summarize the research's key findings and implications.

References and Acknowledgments: List important references and acknowledge any collaborators.

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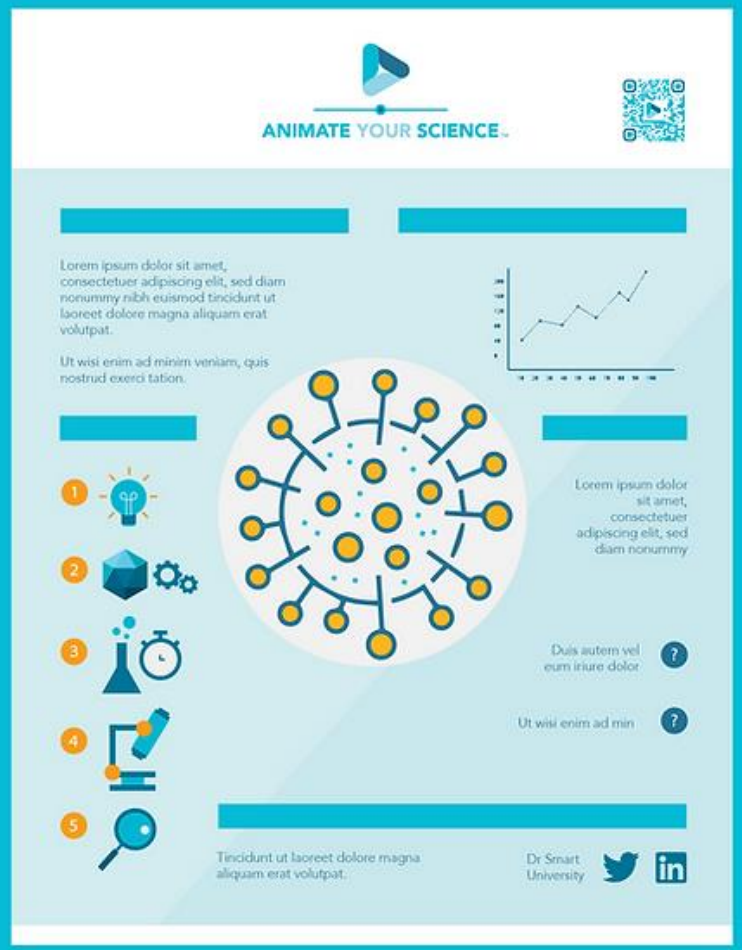
Ooh, that's pretty!



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

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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 O^6 position on guanine, forming mutagenic and highly cytotoxic interstrand DNA crosslinks. The DNA repair enzyme O^6-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 O^6 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.

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.

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.

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.

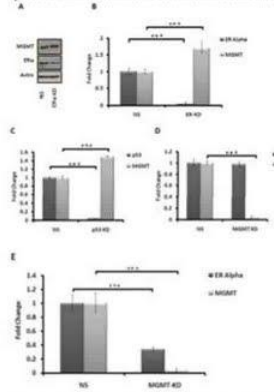


Figure 2. (A) Tamoxifen resistant MCF-7 cells were transfected with ERα siRNA (100nM) (ERα-KD) and NS siRNA (100nM) (NS), and cells were harvested 72h post transfection. Total proteins were isolated and ERα and MGMT expression was determined by western blot analysis. MGMT protein was significantly increased 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 (50 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).

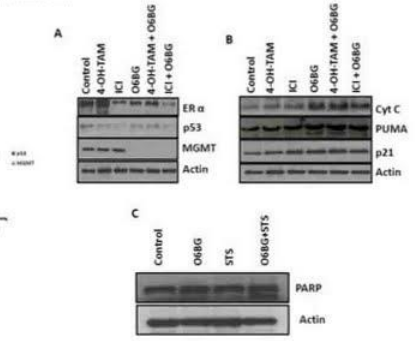


Figure 3. (A) Tamoxifen resistant MCF-7 breast cancer cells were treated in presence or absence of BG (50 μg) and 48h post treatment 4-OH-TAM (1μM), ICI (1μM) either alone or in combination with BG. 24h post treatment cells were harvested and proteins were isolated and western blot analysis was performed. (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 studi. 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).

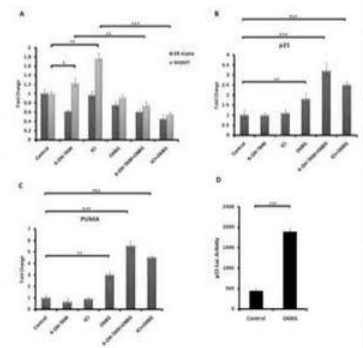


Figure 4. Tamoxifen resistant MCF-7 breast cancer cells were treated in presence or absence of BG (50 μg) for 48h and later 4-OH tamoxifen and ICI (1μM) was either alone or in combination with BG and 24h later cells were harvested and total RNA was isolated. (A) MGMT and ERα (B) p21 transcription (C) 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).

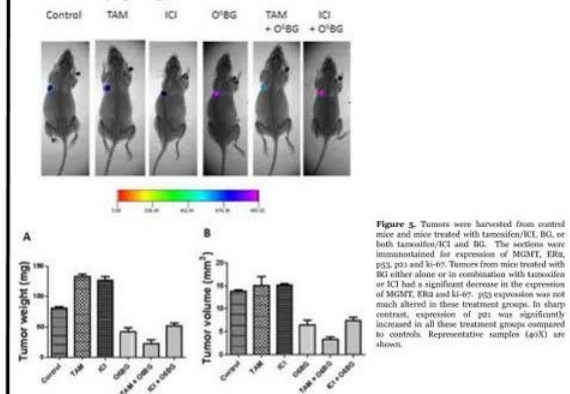
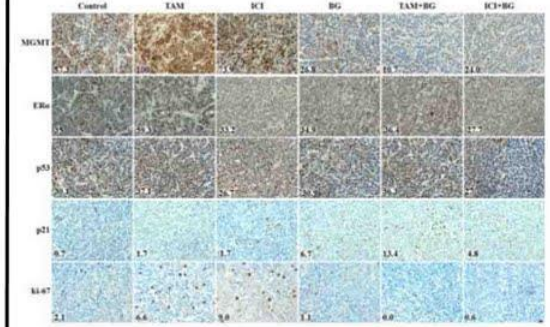


Figure 5. Tumors were harvested from control mice and mice treated with tamoxifen/ICI, BG, or both tamoxifen/ICI and BG. The sections were immunostained for expression of MGMT, ERα, p53, p21 and ki-67. Tumors from mice treated with BG either alone or in combination with tamoxifen or ICI had a significant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

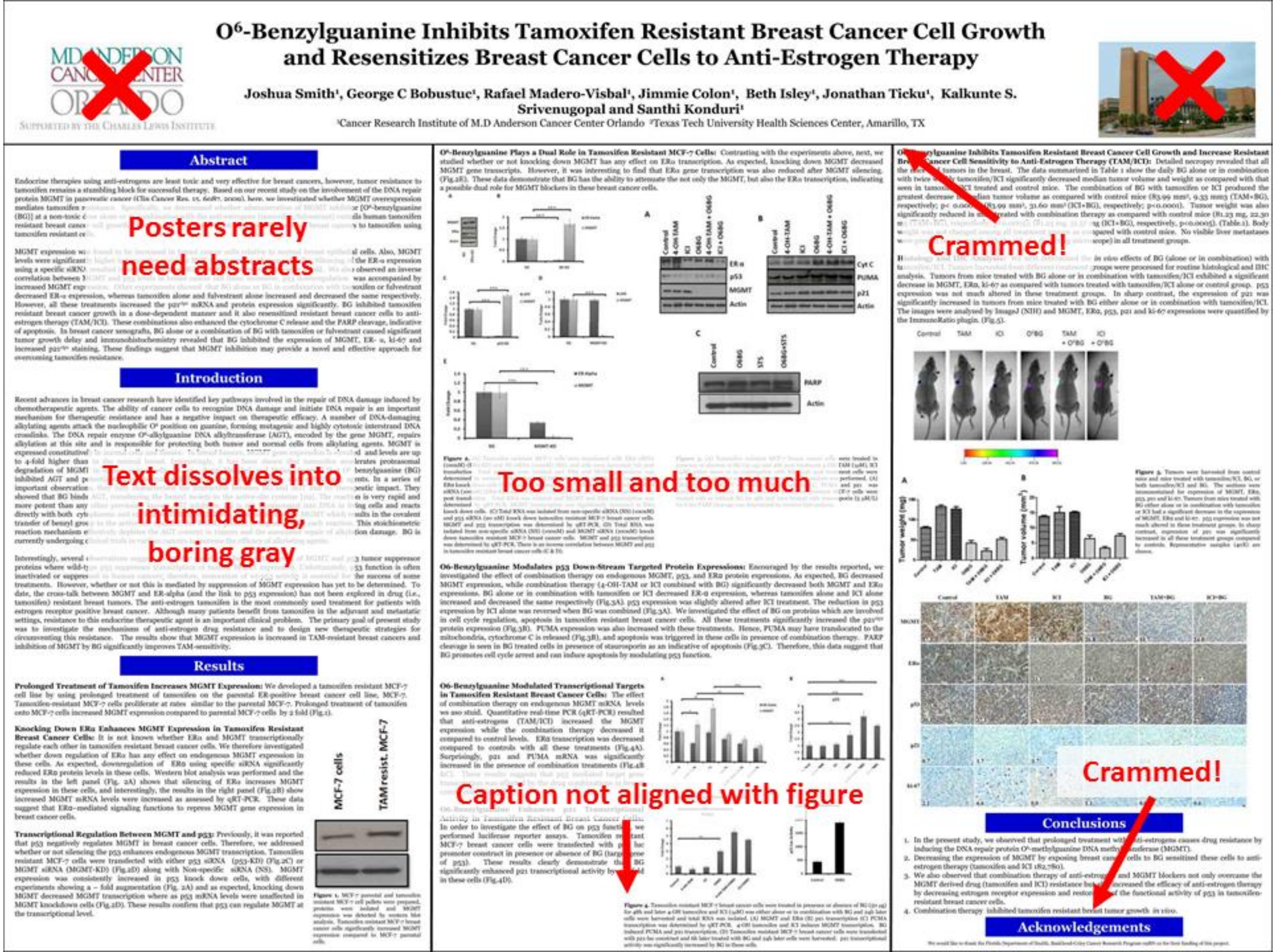


Conclusions

1. In the present study, we observed that prolonged treatment with anti-estrogens causes drug resistance by inducing the DNA repair protein O^6-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-10 for their funding of this project.



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A topical gel for biofilm-associated respiratory tract infections- translation from bench to bedside

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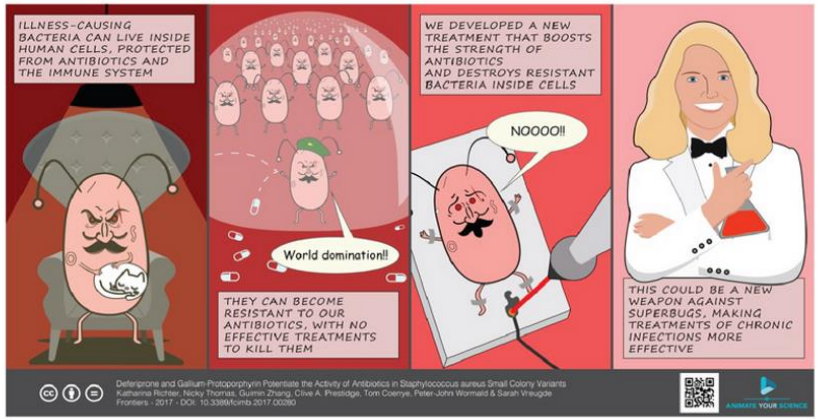
2 Adelaide Biofilm Test Facility, Sansom Institute for Health Research, University of South Australia, Adelaide, Australia
3 Laboratory of Pharmaceutical Microbiology, Ghent University, Gent, Belgium



Background

S. aureus forms biofilms and small colony variants (SCVs), which hide inside human cells, thereby surviving the immune attack and antibiotics[1]. Best medical care (long-term antibiotics, surgery) is ineffective resulting in recurring infections, significant healthcare costs and low quality-of-life[2].

Aim & Methods

Preclinical validation of a novel treatment comprising the iron-chelator deferiprone (Def) and the haem-analogue gallium-protoporphyrin (GaPP) against antibiotic-resistant S. aureus biofilms and SCVs[3].

Results

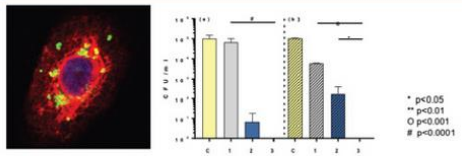


Fig. 1: Intracellular SCVs (green) in a human cell (red-blue). Infection assay: **Def-GaPP-Gentamicin eradicated intracellular (a) and extracellular (b) SCVs.** C: untreated control. Treatment 1: gentamicin (Gent), 2: Def-GaPP, 3: Def-GaPP-Gent.

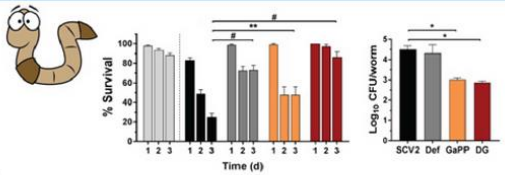


Fig. 2: C. elegans infection model: **Def-GaPP significantly increased the survival of worms infected with SCVs**. Uninfected controls (light grey). Worms infected with SCVs (black) and treated with Def (dark grey), GaPP (orange) or Def-GaPP (red). **GaPP and Def-GaPP significantly reduced the bacterial load per worm.**

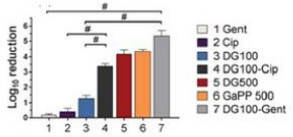


Fig. 3: Colony biofilm model: **Def-GaPP potentiated the antibiofilm activity of Gentamicin and Ciprofloxacin against resistant S. aureus.**

Conclusion

Def-GaPP showed significant activity against S. aureus biofilms and intracellular SCVs, and potentiated the potency of Cip and Gent against resistant strains[3]. **Delivered in a wound healing gel, Def-GaPP progressed to a first-in-human pilot study for the treatment of chronic rhinosinusitis at The Queen Elizabeth Hospital in Adelaide, Australia.**

References

1 Garcia LG, et al. J Antimicrob Chemother 2013;68(7):1455-64.
2 Chronic respiratory diseases in Australia. Australian Institute of Health and Welfare, 2015.
3 Richter K, et al. Front Cell Infect Microbiol 2017;7(280).

Acknowledgements

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KR and SV hold a patent on Def-GaPP for topical antimicrobial applications.

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Authors and affiliations

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Graphical abstract

the bigger the better!

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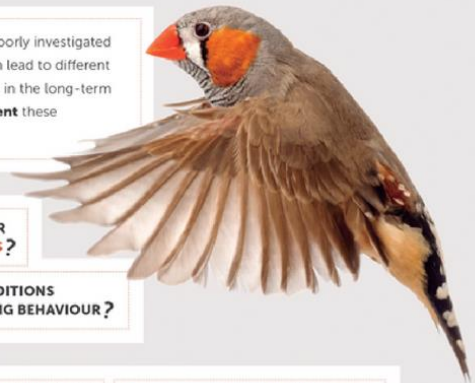
FLEXIBLE FORAGING BEHAVIOUR IN WILD ZEBRA FINCHES AND ITS RELATION WITH TEMPERATURE

CATERINA FUNGHI, LUKE MCCOWAN, WIEBKE SCHUETT AND SIMON GRIFFITH

INTRODUCTION

- **Foraging as behavioural trait** has been poorly investigated
- **Fluctuation** in enviromental contitions can lead to different behavioural strategies being equal in fitness in the long-term
- In **extreme and unpredictable environment** these fluctuations are more pronounced



QUESTIONS

IS INDIVIDUAL FORAGING BEHAVIOUR CONSISTENT IN WILD ZEBRA FINCHES?

HOW DO THE ENVIRONMENTAL CONDITIONS (TEMPERATURE) INFLUENCE FOREGING BEHAVIOUR?

MATERIALS AND METHODS

16 FEEDERS RANDOMLY LOCATED (I.E. 2 TRIALS)

Zebra finches were RFI - tagged and foraging behaviour monitored using a decoder-antenna-feeder system over 3 weeks.

PC ANALYSIS ON DAILY FORAGING BEHAVIOUR OF 72 ADULTS

52% variation	PC1 foraging
Visits	0.5
Distance travelled	0.58
Nb. feeders	0.6
Feeder fidelity	-0.46

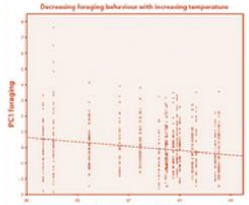
LOW PC1 foraging:
- visits per feeder
- distance travelled
- feeders visited
+ fidelity

HIGH PC1 foraging:
+ visits per feeder
+ distance travelled
+ feeders visited
- fidelity

Example of 2 individuals showing different foraging behaviour. Dots represent feeders located 800m around a dam

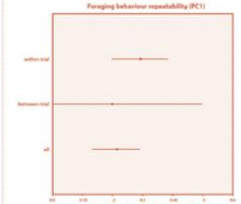
RESULTS AND DISCUSSION

IN EXTREME ENVIRONMENTS FORAGING BEHAVIOUR WAS INFLUENCED BY TEMPERATURE



GLMM: $F_{1,70.002}$, N=68. Negative relation between foraging behaviour (PC1) and Temperature (°C)

CHANGED ENVIRONMENT LED TO FLEXIBLE FORAGING BEHAVIOUR > OPPORTUNISM?



Repeatability (LMM) calculated considering all days of all trials, between and within trials.

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MACQUARIE University
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Margin

Large and impressive title

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Heading 1

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Heading 4

Figures

Figures

Logos and contacts

Design Principles of an Effective Research Poster

Simplicity: Less is more—use minimal text and focus on key information.

Visual Hierarchy: Use font sizes, headings, and layout to guide the viewer's eye from one section to the next.

Fonts and Colors: Choose easy-to-read fonts (e.g., Arial, Calibri) and use contrasting colors to make text stand out.

Graphs and Charts: Visual data should be clear, labeled, and easy to interpret.

White Space: Avoid clutter; white space helps the poster look clean and readable.

Visual Appeal of a Research Poster

Enhancing the visual appeal of a research poster is crucial for grabbing attention and making the information easy to understand. Here are key strategies to increase visual effectiveness:

Use a Clean and Simple Layout

1. Avoid clutter by keeping plenty of white space between sections.
2. Ensure that the poster has a clear flow, guiding the viewer's eye from one section to the next (usually top to bottom, left to right).
3. Use columns and align elements neatly to give the poster structure.

Limit Text and Focus on Key Points

1. Keep your text concise, using bullet points or short sentences. Posters should not be text-heavy; viewers should grasp the main idea quickly.
2. Use headers to organize sections clearly (e.g., "Introduction," "Methods," "Results," "Conclusion").

Use High-Quality Images, Graphs, and Charts

1. Use well-designed charts, graphs, and images to present your data visually. Make sure they are clear, labeled, and easy to interpret.
2. Avoid overly complex or crowded visuals—each graphic should have a purpose.
3. Ensure images are high resolution to prevent pixelation when printed.

Use Readable Fonts and Font Sizes

1. Use legible fonts like Arial, Calibri, or Helvetica. Ensure your font size is large enough to be easily readable from a few feet away (e.g., title 72pt, headings 40pt, body text 24-32pt).
2. Keep font styles consistent, using bold or different sizes to differentiate sections, not multiple font types.

Use a Harmonious Color Scheme

1. Choose a limited palette of 2-3 complementary colors to keep the design cohesive. Avoid overly bright or clashing colors that strain the eyes.
2. Use color to highlight key points or sections, but ensure there’s enough contrast between text and background to maintain readability (e.g., dark text on a light background).

Make Good Use of Icons and Symbols

1. Icons or symbols can help represent ideas or break up large sections of text. Make sure they are relevant to the research and enhance understanding.
2. Ensure consistency in style and size for all symbols or icons used.

Incorporate Visual Hierarchy

1. Use larger fonts, bold headings, and color highlights to create a clear visual hierarchy that directs attention to the most important information first.
2. Make the title and main findings stand out, ensuring these grab attention from a distance.

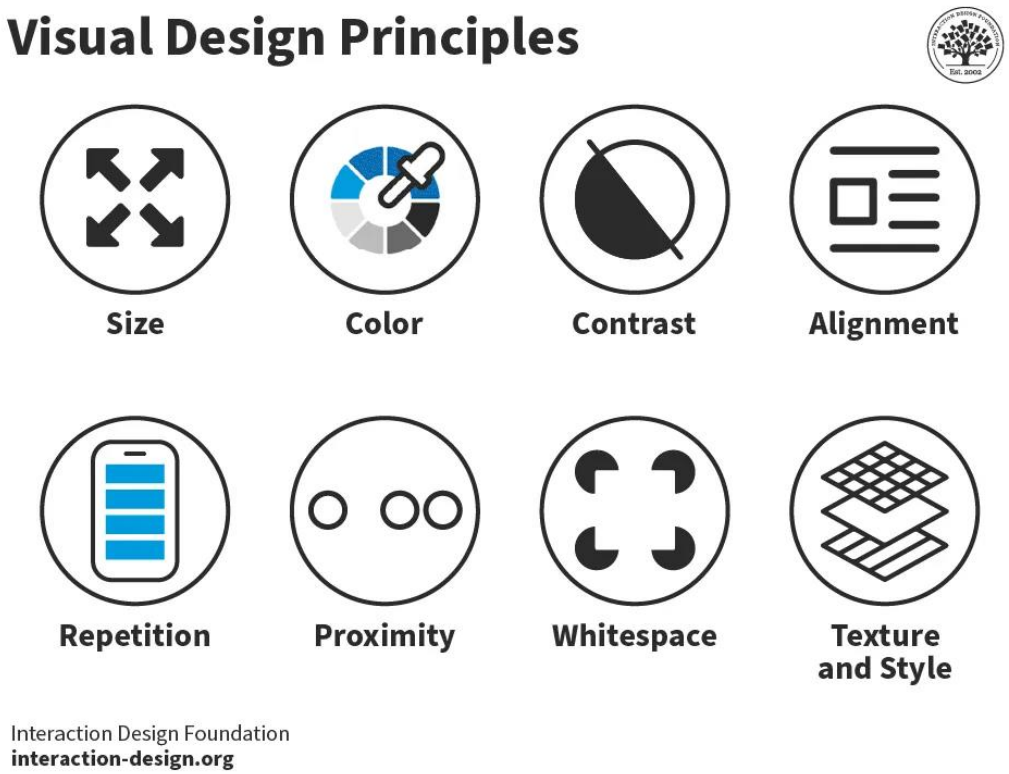
Visual hierarchy is the design principle of organizing and arranging elements in a way that demonstrates their relative importance. By using visual cues such as size, color, contrast, and positioning, designers structure content so that viewers can quickly understand and navigate the information.

Key aspects of visual hierarchy include:

Guiding Perception: Designers arrange elements (e.g., text, icons, images) to naturally lead the viewer's eye through the content in a logical sequence, ensuring that the most important information stands out first.

Strategic Layout: Elements are placed intentionally to guide users toward specific actions or insights. For instance, larger or bold titles catch attention first, while smaller details follow, helping the viewer focus on what matters most.

Enhanced Usability: Effective visual hierarchy makes it easier for users to process and understand information, reducing cognitive load and improving user experience. This is essential in areas like web design, marketing, or visual communication.



Size: Larger elements naturally draw more attention.

Color: Bright or vibrant colors capture focus over muted tones.

Contrast: Strong contrasts between colors make elements more eye-catching.

Alignment: Misaligned elements stand out, while aligned ones create harmony.

Repetition: Consistent styles signal related content.

Proximity: Elements placed close together are perceived as related.

Whitespace: Surrounding space emphasizes and draws attention to elements.

Texture and Style: Textured or detailed elements are more noticeable than flat designs.


BEFORE


AFTER

Avoid Common Mistakes/Pitfalls

Too Much Text: Avoid cramming your poster with text. Keep it concise.

Overcomplicated Graphics: Use simple, clear visuals—don't overwhelm viewers with complex figures.

Cluttered Layout: Poor organization makes it difficult for viewers to follow the flow of information.

Ignoring Visual Design: Bad color schemes, small fonts, and poor image quality reduce readability.

Recap: Key elements of creating an effective research poster.

Structure your poster with clear sections.

Design it for quick readability—use simple visuals and concise text.

Practice your summary and be ready to engage with your audience.

Avoid clutter and overly complex designs.

Use design tools like PowerPoint, Canva, or Adobe Illustrator.

Presentation Skill: Engaging with Your Audience

Prepare a Pitch: Have a 2-3 minute summary ready. Practice explaining your research concisely to a variety of audiences (experts, peers, or non-specialists).

Be Interactive: Stand by your poster, answer questions, and ask for feedback. Don’t just repeat what's on the poster—expand on it.

Body Language: Positive attitude, Smile, make eye contact, and be approachable.

Prepare for Questions: Be ready to explain details in your methods, results, and conclusions.

Prepare a poster on your favourite topic either using primary or secondary data

Poster Presentation date: Starting 17 October. Roster will be circulated soon.