# 2nd International Conference on Cancer Health Disparities

AT THE

# 2024 SCHOOL OF MEDICINE RESEARCH SYMPOSIUM

UTRGV | UT Health Rio Grande Valley

School of Medicine

Cancer Prevention & Research Institute of Texas

# MISSION EVENT CENTER

## 200 N Shary Rd, Mission, TX 78572


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*Event Sponsored by the School of Medicine Research Office,*
*The University of Texas Rio Grande Valley*

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**Dr. Jennifer Cahn,** ***Director of Research Administration***

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*Thank you to all our committees!*

# WELCOME TO
# THE 7th ANNUAL UTRGV
# SOM Research Symposium


On behalf of our faculty, staff, and students, I am pleased to welcome you to the UTRGV School of Medicine's Seventh Annual Research Symposium. We are excited to bring this program to the Valley and to showcase the outstanding research done by investigators at the University as well as our national and international partners. The oral and poster presentations that you will experience today are examples of the excellent work that these researchers have completed. They provide an expansion of knowledge in these key disciplines and demonstrate the diligence and commitment of these individuals in their pursuit of science. With the theme of "International Conference on Cancer Health Disparities" this symposium aims to showcase the work done by our researchers from a broad array of disciplines (academia, community, health care) to identify gaps and/or solutions to respond to multi-faceted heath and health disparity issues impacting minority and underserved populations across the Nation and Worldwide.

One of the key missions of a medical school is the sponsorship and conduct of research activities, including basic, translational, and clinical research. It is through research that we engage our students in critical thinking and in enhancing scientific curiosity. Research serves as the basis for evidence on the quality and efficacy of clinical care and for enhancing patient safety. Discoveries made in the laboratories of our basic scientists assist in the understanding of mechanisms in both health and disease and offer the foundation for translating these findings into clinical interventions. Research provides public visibility for a medical school and contributes to its reputation as an institution of higher learning.

It is with these key principles in mind that I once again welcome you to this Research Symposium. Thank you for attending and for participating with us in this important scholarly activity. Please enjoy the day and the program.

**Michael B. Hocker, MD, MHS**
Dean, School of Medicine

# PROGRAM SCHEDULE

## FRIDAY, FEBRUARY 9, 2024

**7:45-8:30 AM**
**LOBBY**

**REGISTRATION + NETWORKING BREAKFAST**

**8:30-8:45 AM**
**RUBY RED BALLROOM**

**WELCOME & OPENING REMARKS**
**Subhash C. Chauhan, Ph.D.**, Director, South Texas Center of Excellence in Cancer Research, UTRGV SOM
**Abby Guillory, MLIS, CRA,** Assistant VP for Research Enhancement, UTRGV
**Michael Hocker, M.D., MHS,** Dean, School of Medicine, Sr. Vice President UT Health RGV

**8:45-10:15 AM**
**RUBY RED BALLROOM**

**Symposium Session 1 - Keynote Lecture**
8:45- 9:15 AM- **Keshav K. Singh, Ph.D.**
*Decoding MitoGenomics in African Genomes to Decipher Cancer Health Disparities*
**Invited Talks**
9:15- 9:35 AM- **Erin H. Seeley, Ph.D.**
*Mass Spectrometry Imaging Enables Multiplexed Multi-omics Analysis of Tissue Sections*
9:35- 9:55 AM- **Platinum Sponsor**
**Conor Mullens, Ph.D. -** *Bruker Presentation*
9:55-10:15 AM- **Platinum Sponsor**
**Kara Wendel, Ph.D. -** *Scintica Presentation*

**8:45-10:15 AM**
**MANDARIN HALL**

**Oral Presentations Session 1**
8:45- 9:00 AM **Mohammad S. Hussain, Ph.D.**
9:00- 9:15 AM **Ryan P. Coll, Ph.D.**
9:15- 9:30 AM **Lin Wang, Ph.D.**

**9:00 AM-12:30 PM**
**TANGERINE HALL**

**CPRIT EAB Meeting- Closed Session**

**10:15-10:30 AM**
**LOBBY**

**BREAK**

# FRIDAY, FEBRUARY 9, 2024

**10:30 AM- 12:00 PM**
**RUBY RED BALLROOM**

**Symposium Session 2 - Keynote Lecture**

10:35- 11:00 AM- **James Alaro, Ph.D.**
*Embracing the Complexity: Transdisciplinary Approaches to Advance the Science of Cancer Health Disparities*

**Invited Talks**

11:00- 11:20 AM **Ajaikumar B. Kunnumakkara, Ph.D., FRSM**
*Tumor necrosis factor-α induced protein 8 family as a novel molecular target for oral cancer*

11:20-11:40 AM **Manal Hassan, M.D., Ph.D.**
*Genome-wide association study identifies high-impact susceptibility loci for hepatocellular carcinoma in North America*

11:40 AM-12:00 PM **Robert Tsai, M.D., Ph.D.**
*Interrogating Genome-Wide DNA Methylation Changes in NAFLD Progression from Simple Steatosis to Advanced Fibrosis or NASH".*

**12:00 -12:45PM**
**VALENCIA HALL**

**POSTER SESSION #1**

*Judges will score posters from*
***Undergraduate, Graduate, Staff, and Faculty Categories.***

**12:45-1:30 PM**
**RUBY RED BALLROOM**

**NETWORKING LUNCH sponsored by Bruker Scientific**

**1:30-3:00 PM**
**RUBY RED BALLROOM**

**Symposium Session 3 - Keynote Lecture**

1:30- 2:00 PM **Michelle M. Le Beau, PhD***
*CPRIT: Catalyzing the Fight Against Cancer in Texas*

**Invited Talks**

2:00- 2:20 PM **Eduardo "Eddie" Olivarez**
*Infectious Disease Awareness in Cancer Treatment*

2:40- 3:00 PM **Subhash C. Chauhan, Ph.D. / Prasun Jalal, M.D.**
*Clinical Aspects of Liver Cancer*

*indicates this speaker will be presenting live through Zoom, streaming into the Hall.

# FRIDAY, FEBRUARY 9, 2024

**1:30-3:00 PM**
**LEMON HALL**

**Oral Presentations Session 2**
1:30- 1:45 PM **Arathi Radhadkrishnan***
1:45- 2:00 PM **Anupam Dhasmana, Ph.D.**
2:00- 2:15 PM **Anusmita Shekher, Ph.D.***
2:15- 2:30 PM **Ankit Srivastava, Ph.D.***
2:30- 2:45 PM **Andrew Kolodziej**
2:45- 3:00 PM ***OPEN***

**1:30-3:00 PM**
**MANDARIN HALL**

**Keynote Lecture:**
1:30- 2.00 PM **Murali Yallapu, Ph.D.**
*Nanomedicine: Basics to Cancer Therapeutics*
**Oral Presentations Session 3**
2:00- 2:15 PM **Melissa Cruz**
2:15- 2:30 PM **Mohammed Sikander, Ph.D.**
2:30- 2:45 PM **Sheema Khan, Ph.D.**
2:45- 3:00 PM **Sheema Khan, Ph.D.**

**3:00-3:15 PM**
**LOBBY**

**BREAK**

**3:15-4:45 PM**
**RUBY RED**
**BALLROOM**

**Symposium Session 4- Keynote Lecture**
3:15-3:45 PM **Jose Torres Ruiz, PhD**
*Cancer Health Disparities Addressed by the Comprehensive Cancer Center-UPR in San Juan, PR*
**Invited Talks**
3:45-4:05 PM **Manoj K. Mishra, Ph.D.**
*Understanding the Role of Socioeconomic Factors in Prostate Cancer Health Disparities in Alabama*
4:05-4:25 PM *Cancer Seminar Series Speaker:*
**Tuula Klaavuniemi, M.D., Ph.D.**
*From Basic Science to Real World Clinical Practice - The Way to Provide the Best Care to Our Cancer Patients*
4:25-4:45 PM **Rajiv Saini, Ph.D.-CSO, H2Ocean**
*Oral Care in Cancer Survivorship: Discover the Healing Power of Sea Salt*

*indicates this speaker will be presenting live through Zoom, streaming into the Hall.

# FRIDAY, FEBRUARY 9, 2024

**3:15-4:45 PM**
**LEMON HALL**

**Keynote Lecture**
3:15- 3:45 PM **Dev Karan, Ph.D.***
*Analysis of serum cytokines-chemokines in association with prostate cancer disparity*
**Oral Presentations Session 4**
3:45- 4:00 PM **Nirakar Sahoo, Ph.D.**
4:15- 4:30 PM **Silvia Mejia-Arango, Ph.D.**
4:30- 4:45 PM **Miroslava Gomez-Garza, M.D., OBGYN, & Noe Garza, DDS, MPH**

**3:15-4:45 PM**
**MANDARIN HALL**

**Special Topics Sessions**
3:15-3:45 PM- **John Ronnau, M.D.**
*Understanding the Impacts of Social Context on Health*
Accompanied by:
Xavier Duran- Medical Office Manager
Lizette Ingle- Program Coordinator
Miguel Jimenez Mejia- Program Manager
Karla Soberanis- Program Specialist
3:45-4:15 PM **Kelsey Baker, Ph.D.**
*Neuromodulation for Cancer: Where can we go?*
4:15- 4:45 PM **Sue Ann Chew, Ph.D.**
*Biomaterial-Based Strategies for the Treatment of Cancer*

**4:00-5:15 PM**
**VALENCIA HALL**

**POSTER SESSION #2**
*Judges will score posters from*
***Medical School Students, Fellows/ Post-Docs, Medical Residents, and High School Students***

**5:15- 8:00 PM**
**RUBY RED BALLROOM/LOBBY**

**NETWORKING RECEPTION**
Complimentary appetizers, refreshments, and dinner provided!
Sponsored by Bruker Scientific, highlighting Purple Night for Pancreatic Cancer Awareness.

# SATURDAY, FEBRUARY 10, 2024

**7:45- 8:30 AM**
**RUBY RED BALLROOM**

**REGISTRATION + NETWORKING BREAKFAST**

**8:30-8:45 AM**
**RUBY RED BALLROOM**

**OPENING REMARKS**
**Subhash C. Chauhan, Ph.D.,** *Scientific Chair*

**8:45-10:15 AM**
**RUBY RED BALLROOM**

**Symposium Session 5 -Keynote Lecture**
8:45- 9:15 AM **Brett Spear, Ph.D.**
*Tumor Promoting Activity of the Transcription Factor Zhx2 in Hepatocellular Carcinoma*
**Invited Talks**
9:15- 9:35 AM **Michael X. Zhu, Ph.D.**
*Dysfunctional calcium signaling in cutaneous T-cell lymphoma*
9:35- 9:55 AM **Edward James Kruse, DO**
*Cancer in the Rio Grande Valley – Screening Programs and Opportunities*
9:55-10:15 AM **Subash C. Gupta, Ph.D.***
*Modulation of prohibitin, nuclear factor-κB, and long non-coding RNAs by an isothiocyanate in breast cancer cells*

**8:45-10:15 AM**
**LEMON HALL**

**Keynote Lecture**
8:45- 9:15 AM **Shrikanth S. Gadad, Ph.D.**
*Epigenetic Regulation of Breast Cancer by Nuclear Non-Coding*
**Oral Presentations Session 5**
9:15- 9:30 AM **Varsha Gupta, Ph.D.**
9:30- 9:45 AM **Shabia Shabir Khan, Ph.D.**
9:45- 10:00 AM **Avtar Meena, Ph.D.**
10:00- 10:15 AM **Vivek K. Kashyap, Ph.D.**

**8:45-10:15 AM**
**MANDARIN HALL**

**Oral Presentations Session 6**
8:45- 9:00 AM **Yossef Alsabawi**
9:00- 9:15 AM **Miguel Lopez**
9:15- 9:30 AM **Miguel Lopez**
9:30- 9:45 AM **Lisa Salinas, Ph.D.**
9:45- 10:00 AM **Shikha Sharma, Ph.D.***

*indicates this speaker will be presenting live through Zoom, streaming into the Hall.

# SATURDAY, FEBRUARY 10, 2024

**10:15- 10:30 AM**
**LOBBY**

**BREAK**

**10:30 AM-12:00 PM**
**RUBY RED BALLROOM**

**Symposium Session 6 - Keynote Lecture**
10:30- 11:00 AM- **Henry C. Manning, Ph.D.**
*Access to Radiopharmaceuticals for Imaging and Therapy: the need, current challenges, and prospects for a bright future*
**Invited Talks**
11:00- 11:20 AM **Everardo Cobos, M.D.**
*New developments in Hepatocellular and Cholangiocarcinoma*
11:20- 11:40 AM **Varaprasad Kokkarachedu, Ph.D.**
*Biocidal activity of hybrid nanomaterials for next-generation applications*
11:40 AM- 12:00 PM
*Cancer Seminar Series Speaker:*
**Kanchan Chauhan, Ph.D.**
*Biocatalytic nanoreactors towards therapeutic nanofactories*

**10:30 AM-12:00 PM**
**LEMON HALL**

**Oral Presentations Session 7**
10:30- 10:45 AM **Manish Tripathi, Ph.D.**
10:45- 11:00 AM **Bilal Bin Hafeez, Ph.D.**
11:00- 11:15 AM **Martin Ekoumou, M.D., MPH, PH.D-C**
11:15- 11:30 AM **Tomas Gomez Jr.**
11:30-11:45 AM **Noah Al-Hassan & Taha Al-Hassan**

**10:30 AM-12:00 PM**
**MANDARIN HALL**

**Oral Presentations Session 8**
10:30- 10:45 AM **Barbara Malaga-Espinoza, M.D.**
10:45- 11:00 AM **Shreel Patel, M.D.**
11:00- 11:15 AM **Shreel Patel, M.D.**
11:15- 11:30 AM **Shreel Patel, M.D.**

**12:00- 1:00 PM**
**RUBY RED BALLROOM**

**Networking Lunch sponsored by Scintica**

Scintica:

# SATURDAY, FEBRUARY 10, 2024

**1:00- 2:30 PM**
**RUBY RED BALLROOM**

**Symposium Session 7- Keynote Lecture**
1:00- 1:30 PM **Subhash C. Chauhan, Ph.D.**
*Prolonged Socio-Physiological Stress and Cancer*
**Invited Talks**
1:30- 1:50 PM **Diana Resendez Perez, Ph.D.**
*Novel interaction complexes of homeoproteins and transcriptional factors in the genetic control of development in Drosophila melanogaster*
1:50- 2:10 PM **Ms. Cassandra Perez**
*Diverse Impact: Empowering Inclusive Community Involvement in Cancer Policy*
2:10- 2:30 PM **Mehdi Shakibaei, Ph.D.**
*Resveratrol and p53: How do they affect the plasticity and apoptosis of CRC?*

**1:00- 2:30 PM**
**LEMON HALL**

**Oral Presentations Session 9**
1:00- 1:15 PM **Dikshanta V. Luitel**
1:15- 1:30 PM **Melina J. Sedano**
1:30- 1:45 PM **Sana Shabir Khan**
1:45- 2:00 PM **Andrea Dorado Baeza**
2:00- 2:15 PM **Barbara Yang**
2:15- 2:30 PM **Nayarah Shabir**

**1:00- 2:30 PM**
**TANGERINE HALL**

**Trainee Meet & Greet- Closed Session**
1:00-1:45 PM **Kanchan Chauhan, Ph.D.**
1:45- 2:30PM **Tuula Klaavuniemi, M.D., Ph.D.**

**1:00- 2:30 PM**
**MANDARIN HALL**

**Oral Presentations Session 10**
1:00- 1:15 PM **Jessica Daza, M.D.**
1:15- 1:30 PM **Jessica Daza, M.D.**
1:30- 1:45 PM **Lois Akpati**
1:45- 2:00 PM **Liza Salloum M.D.**

**2:30-2:45 PM**
**LOBBY**

**BREAK**

# SATURDAY, FEBRUARY 10, 2024

**2:45-3:45 PM**
**RUBY RED BALLROOM**

**Special Topics Session**

***Grant Funding: Current Trends and Best Practices***

**Organizer/ Moderator: Dr. Jennifer Cahn**

**Dr. James Alaro-** *Program Director*, National Cancer Institute, Center for Global Health

**Dr. Michelle Le Beau-** *Chief Scientific Officer*, Cancer Prevention & Research Institute of Texas

**Dr. Brett Spear-** *Professor/ Director*, SuRE Resource Center, University of Kentucky College of Medicine

**Ms. Tribbie Grimm-** *Director of Sponsored Programs*, UTRGV

**3:45- 4:00 PM**
**LOBBY**

**BREAK**

**4:00-4:30 PM**
**RUBY RED BALLROOM**

**AWARD CEREMONY sponsored by Scintica**

Scintica:

# EXHIBIT HALL- Valencia Hall + Lobby

## List of Exhibitors/ Sponsors in attendance

**Lobby**

Bruker Scientific
H2Ocean
Scintica
Shimadzu
Subhash Bose
ThermoFisher Scientific

**Valencia Hall**

BioTek
Eppendorf
Miltenyi Biotec
Nikon
UTRGV SOM- Institute of Neuroscience
UTRGV- Lab Animal Resources

# POSTER EXHIBIT- Valencia Hall

## FRIDAY, FEBRUARY 9, 2024

| | |
|---|---|
| 8:00 AM- 5:00 PM | **Viewing available for Poster Exhibit** |
| 12:00- 12:45 PM | **POSTER SESSION 1**<br>***Judging for all Undergraduate Students, Graduate Students, Staff, and Faculty*** |
| 4:00- 5:15 PM | **POSTER SESSION 2**<br>***Judging for all Medical Students, Medical Residents, Fellows/ Post-Docs, and High School Students*** |

## SATURDAY, FEBRUARY 10, 2024

| | |
|---|---|
| 8:00 AM- 4:30 PM | **Viewing available for Poster Exhibit/ Exhibit Hall** |

# List of Oral Presentations

## Oral Presentations- Session 1

## February 9th, 8:45 AM-10:15 AM, MANDARIN HALL

**Moderator: Ms. Staci Eaton**

| | |
|---|---|
| **Mohammad Shabir Hussain, Ph.D.** *Post- Doc* | *Proteomic analysis of stress associated factor overexpression in Hepatocellular carcinoma* |
| **Ryan P. Coll, Ph.D.** *Post-Doc* | *Assessment of Mucin 13 (MUC13) as an Imaging Target for Guiding Colorectal Cancer Treatment: A Pathway Towards Theranostic Development* |
| **Lin Wang, Ph.D.** *Faculty* | *Assessing the Reliability, Internal Consistency, and Sensitivity of a Nutrition Knowledge Questionnaire for Four-Year-Old Pre-K Children.* |

## Oral Presentations- Session 2

## February 9th, 1:30-3:00 PM, LEMON HALL

**Moderator: Dr. Bilal Hafeez**

| | |
|---|---|
| **Arathi Radhakrishnan** *Graduate Student* | *Targeting mycobacterial efflux system for combating anti-microbial resistance* |
| **Anupam Dhasmana, Ph.D.** *Staff* | *CEACAM7 emerges as a promising early detection biomarker in pancreatic cancer.* |
| **Anusmita Shekher, Ph.D.** *Post-Doc* | *Metabolic reprogramming in breast cancer patients as revealed by 1H NMR spectroscopy* |
| **Ankit Srivastava, Ph.D.** *Post-Doc* | *Moringin, an isothiocyanate improves the susceptibility of breast cancer cells to doxorubicin* |
| **Andrew Kolodziej** *Medical Student* | *A Rare Encounter: Extracranial Meningioma Mimicking Musculoskeletal Neoplasms* |

## Oral Presentations- Session 3

## February 9th, 1:30 -3:00 PM, MANDARIN HALL

**Moderator: Dr. Manish Tripathi**

| | |
|---|---|
| **Melissa Cruz**<br>*Medical Student* | *Comparative Effectiveness of Endovascular vs Surgical Arteriovenous Fistulas: A Preliminary Analysis* |
| **Mohammed Sikander, Ph.D.**<br>*Faculty* | *Novel therapeutic strategy for cervical cancer treatment* |
| **Sheema Khan, Ph.D.**<br>*Faculty* | *Antibody mediated Targeted Drug Delivery approach for Pancreatic Tumors* |
| **Sheema Khan, Ph.D.**<br>*Faculty* | *Novel strategy to make KRAS targeted therapies more effective for PDAC treatment* |

## Oral Presentations- Session 4

## February 9th, 3:15- 4:45 PM, LEMON HALL

**Moderator: Dr. Anupam Dhasmana**

| | |
|---|---|
| **Nirakar Sahoo, Ph.D.**<br>*Faculty* | *Targeting Ion Channels in Liver Cancer Cells: Stimulating Lysosomal TRPML1 and Inhibiting hEAG1 Potently Reduce Cell Viability* |
| **Silvia Mejia-Arango, Ph.D.**<br>*Faculty* | *Effect of Alcohol Consumption on Cognitive Decline among Mexican Adults* |
| **Miroslava Gomez-Garza, M.D., OBGYN, & Noe Garza, DDS, MPH**<br>*Staff* | *Human Papilloma Viruses Infection and Pre-Malignant Lesions in Women on the Texas Mexico Border* |

## Oral Presentations- Session 5

## February 10th, 8:45-10:15 AM, LEMON HALL

**Moderator: Dr. Neeraj Chauhan**

**Varsha Gupta, Ph.D.**
*Faculty*
*Role of Gut Microbiome in Rheumatoid Arthritis*

**Shabia Shabir Khan, Ph.D.**
*Faculty*
*Revolutionizing Feature Selection: A Breakthrough Approach for Enhanced Accuracy and Reduced Dimensions, with Implications for Early Medical Diagnostics*

**Avtar Meena, Ph.D.**
*Faculty*
*Role of TRPV6 in Mitigating Alcohol-Induced Disruption of Tight Junctions, Barrier Function, and Hepatic Injury*

**Vivek K. Kashyap, Ph.D.**
*Faculty*
*Piperlongumine nanoformulation attenuates pancreatic tumor desmoplasia and alter tumor immune responses*

## Oral Presentations- Session 6

## February 10th, 8:45-10:15 AM, MANDARIN HALL

**Moderator: Dr. Mohammed Sikander**

**Yossef Alsabawi**
*Medical Student*
*The Increasing Prevalence of Cleft Lip with or without Cleft Palate in the Rio Grande Valley of Texas*

**Miguel Lopez**
*Medical Student*
*Birth defect trends within Texas Public Health Region 11, 2000-2019: an analysis of Texas Department of State Health Services public data.*

**Miguel Lopez**
*Medical Student*
*Inflammatory Breast Carcinoma in the Rio Grande Valley: A Case Report*

**Lisa Salinas, Ph.D.**
*Faculty*
*Risk of Food Insecurity Reflects Health Status in Adult Relatives of PreK Children – Data from the STEPS Snap-Ed Research Study*

**Shikha Sharma, Ph.D.**
*Staff*
*Circular Gene Mapping of Identified AMR Genes in Multi-Drug Resistant Escherichia coli Isolated from Potable Water*

# SPECIAL THANKS!

*Abstract Reviewers*

*Presentation Judges*

*Oral Session Moderators*

*Staff Volunteers*

*Student Volunteers*

On behalf of the 2023-24 UTRGV SOM Research Symposium Scientific and Event Planning committees, we thank each of you who attended the conference. A special thanks to The School of Medicine Research Office, National Cancer Institute, UTRGV SOM Medicine & Oncology ISU, The City of Mission, and all our donors and sponsors.

Funding for this conference was made possible (in part) by 1R13CA278054-01 from the National Cancer Institute. The views expressed in written conference materials or publications and by speakers and moderators do not necessarily reflect the official policies of the Department of Health and Human Services; nor does mention by trade names, commercial practices, or organizations imply endorsement by the U.S. Government.

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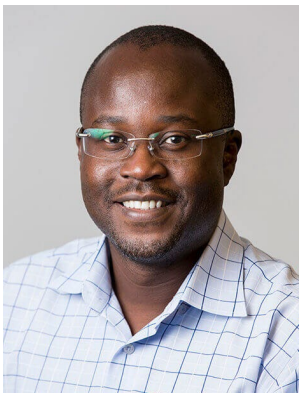
# 2024 KEYNOTE SPEAKERS



## James Alaro, PhD

**Program Director, Center for Global Health, National Cancer Institute.**

A global health expert committed to advancing research to accelerate progress towards achieving health equity for all populations. He envisions a more trans-disciplinary future of cancer health disparities research and progress towards cancer health equity for all populations, both locally and globally.

## Subhash C. Chauhan, PhD

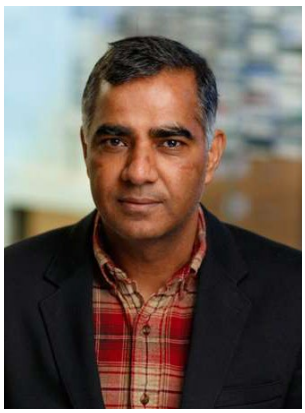
**Professor, Department of Immunology and Microbiology and Director, Institute of Cancer Immunotherapy School of Medicine.**

Primary research interest of Dr. Chauhan's lab is to identify and characterize the diagnostic and therapeutic targets for cancer. This research is aimed for the identification and characterization of biomarkers that aberrantly express or localize in cancer cells to develop newer tools for early disease diagnosis. We are utilizing genomics and proteomics approach for identification of novel early diagnostic markers. Recently we have identified a novel trans-membrane mucin MUC13 which is highly over-expressed ovarian and pancreatic and colon cancers. This may be potential biomarker for early cancer diagnosis as well as a good target for antibody guided targeted therapy.

## Shrikanth Gadad, PhD

**Assistant Professor, Department of Biomedical Sciences, Texas Tech University Health Sciences Center at El Paso**

Now a molecular biologist at Texas Tech University Health Sciences Center at El Paso, Dr. Gadad is studying the role of non-coding regions in the onset, progression, and metastasis of many different types of cancer. Gadad appreciates the support from CPRIT, which has enabled him to hire talented researchers for his laboratory and employ cutting-edge technologies to conduct innovative research to understand and combat cancer.



## Dev Karan, PhD

**Associate professor, Department of Pathology at the Medical College of Wisconsin**

Dr. Karan's research focuses on cancer immunology and immunotherapy, and the understanding of the role of inflammation in the development and progression of prostate cancer.

# 2024 KEYNOTE SPEAKERS

## Michelle M. Le Beau, PhD

**Chief Scientific Officer, Cancer Prevention & Research Institute of Texas (CPRIT)**

Dr. Le Beau leads CPRIT's academic research program in supporting innovative cancer research and recruiting world-class cancer researchers to Texas institutions. She is recognized for her work identifying recurring chromosomal abnormalities and defining the clinical and genetic subsets of leukemia, characterizing the genetic pathways that lead to hematological malignancies, and the application of fluorescence in situ hybridization for clinical diagnostics and gene mapping.

## Henry C Manning, PhD

**Professor, Department of Cancer Systems Imaging, Division of Diagnostic Imaging, The University of Texas MD Anderson Cancer Center**

Since 2006, his laboratory has focused on the discovery, translation, and validation of chemical and molecular probes for cancer imaging and therapy. In addition, his laboratory discovers and translates novel radiopharmaceuticals and chemical probes, with emphasis on positron emission tomography (PET) imaging.

## Keshav K. Singh, PhD

**Professor, Genetics Research Division, University of Alabama-Birmingham School of Medicine**

Since 2011, he is Joy and Bill Harbert Endowed Chair and Director of Cancer Genetics Program at UAB Comprehensive Cancer Center. He is a Professor of Genetics, Pathology and Environmental Health. He is also a member of Center for Free Radical Biology and Center for Aging at UAB. Dr. Singh is the author of more than 100 research publications and three books.

## Brett Spear, PhD

**Professor and Director of Graduate Studies, Microbiology, Immunology & Molecular Genetics, University of Kentucky College of Medicine**

His research interests are in the area of gene regulation; in particular, we are interested in transcriptional regulation in the liver during development and disease. We are currently interested in the role of Zhx2 in lipid homeostasis, liver function in response to injury, developmental and sex-biased gene expression and hepatocellular carcinoma.

# 2024 KEYNOTE SPEAKERS

## Jose Torres Ruiz, PhD

**Director, Office of Research Administration, Comprehensive Cancer Center-University of Puerto Rico**

Dr. Torres-Ruiz served as the Provost/ Vice President for Academic Affairs for the Ponce Health Sciences University from 2014 to 2018. In 2018 he was named Chancellor of the PHSU system until he took his current position at CCC-UPR in 2023.

## Murali Yallapu, PhD

**Associate Professor, Department of Immunology and Microbiology, School of Medicine, University of Texas Rio Grande Valley**

His research focus is to study the fate of nanoformulations that leads to novel insights of various biological factors and properties responsible for effective and targeted delivery and treatment. The overall goal of his research is to use these studied materials to devise advanced delivery and immunotherapy systems that can be tailored to meet the needs of individual cancer patient.

# INVITED SPEAKERS

**Kelsey Baker, PhD**
*Assistant Dean for Pre-Clerkships, UTRGV School of Medicine*

**Kanchan Chauhan, PhD**
*Associate Professor, Centre of Nanoscience and Nanotechnology, National Autonomous University of Mexico*

**Sue Ann Chew**
*Associate Professor, Department of Health and Biomedical Sciences, UTRGV*

**Everardo Cobos, MD**
*Chair of the Integrated Service Unit in Medicine and Oncology at UTRGV School of Medicine*

**Subash C. Gupta**
*Professor and Head, Department of Biochemistry, Associate Dean (Research), All India Institute of Medical Sciences, Guwahati, India*

**Manal Hassan, MD, PhD**
*Associate Professor, Department of Epidemiology, The University of Texas MD Anderson Cancer Center, Houston, TX*

**Prasun Jalal, MD**
*Assistant Professor in Medicine and Surgery, Baylor College of Medicine*

**Tuula Klaavuniemi, MD, PhD**
*Chief Oncologist, Mikkeli Central Hospital, Finland*

**Varaprasad Kokkarachedu, PhD**
*Associate Professor, Universidad San Sebastián, Chile*

**Edward Kruse**
*Surgical Oncologist, UTRGV School of Medicine*

**Ajai Kunnumakkara, PhD**
*Professor, Department of Biosciences and Bioengineering, IIT Guwahati*

**Manoj K. Mishra, PhD**
*Professor of Biology Director, Cancer Biology Research and Training Program Director, Freshman Biology Program*

**Conor Mullens, MS**
*Technical Sales Representative, Bruker Daltonics*

**Eduardo Olivarez**
*Administrative Officer, Hidalgo County Health and Human Services*

**Cassandra Perez, MPA**
*Grassroots Manager at the American Cancer Society Cancer Action Network based in the Rio Grande Valley*

**Diana Resendez Perez, PhD**
*Professor, Department of Biological Sciences, Autonomous University of Nuevo León*

# INVITED SPEAKERS

**John Ronnau, M.D., PhD**
*Senior Associate Dean, Community Health Partnerships, UTRGV School of Medicine*

**Rajiv Saini, PhD**
Associate Professor in department of Periodontology and Oral Implantology of Pravara Institute of Medical sciences-Loni, Maharashtra, India.

**Erin H. Seeley, PhD**
*Mass Spectrometry Imaging Facility Director, University of Texas at Austin*

**Mehdi Shakibaei, PhD**
*Professor, Anotamical Institute, Faculty of Medicine, Ludwig Maximillian University of Munich, Munich, Germany*

**Robert Tsai, MD, PhD**
*Texas A&M Health Science Center*

**Kara Wendel, PhD**
*Product Manager at Scintica Instrumentation*

**Michael X. Zhu, PhD**
*Professor of Integrative Biology and Pharmacology, McGovern Medical School, The University of Texas*

# SCHOLAR TRAINEE SPEAKERS

**Ryan P. Coll, PhD**
*Postdoctoral Fellow at MD Anderson Cancer Center, Department of Cancer Systems Imaging*

**Andrea Dorado Baeza**
*BS.Nanotechnology | Bionanotechnology Laboratory at Universidad Nacional Autónoma de México*

**Gustavo Jiménez Mejía, PhD**
*Faculty of Biological Sciences,Universidad Autónoma de Nuevo León*

**Ville Paappanen**
*PhD Student and Medical student at University of Eastern Finland*

**Melina J. Sedano**
*HSC Sr Research Scientist at Texas Tech University Health Sciences Center El Paso, Department of Molecular and Translational Med*

**Barbara Yang**
*Research Associate at Texas Tech University Health Sciences Center*

# ORAL PRESENTATIONS

## FACULTY CATEGORY

**Analysis of serum cytokines-chemokines in association with prostate cancer disparity.**

*Dev Karan*
*Department of Pathology, MCW Cancer Center and Prostate Cancer Center of Excellence, Medical College of Wisconsin, 8701 Watertown Plank Road, Milwaukee, WI 53226, USA.*

Background: Cancer health disparities represent a significant public health concern in the United States. Prostate cancer is one such disease with a higher incidence and death rate in African American (AA) men than in Whites. This study aimed to understand the role of systemic inflammation in association with prostate cancer disparity among AA and White men.
Methods: We used a 40-plex human chemokine assay kit to determine the differential level of cytokines-chemokines associated with prostate cancer disparity among AA and White men. A total of 211 serum samples from prostate cancer patients and healthy donors were tested. Differential expression of the selected chemokines was also analyzed using IHC (immunohistochemistry) in a representative group of prostate tumor tissues of two races.
Results: Race-specific differences were observed for significantly higher serum levels of CXCL2, CXCL5, and IL-6 and lower levels of CCL23 and CCL27 in AA prostate cancer patients (n = 14) compared to Whites (n = 14). Further extension of the analysis to 211 serum samples showed significantly higher levels of CXCL2 and CXCL5 in AA men than in Whites, whereas CCL23 differed significantly within and between the races with a lower level in AA cancer cases than healthy donors. Patient age, PSA, or Gleason scores were not significantly associated with these chemokines. IHC for CXCL5 and CCL23 in prostate tissues displayed higher CXCL5 in prostate tumors than in benign tissues, while CCL23 was non-detectable in tumor tissues. In contrast, independent of racial composition, serum levels of C-C chemokines CCL1, CCL7, CCL8, CCL11, CCL13, CXCL9, CXCL10, and the cytokine IL-2, and TNF-$\alpha$ were significantly lower in prostate cancer patients (n = 28) compared to healthy controls (n = 11).
Conclusions: This study observed that serum chemokines CXCL2, CXCL5, and CCL23 are the most distinguished chemokines differentially expressed in men of AA and White races. The impact of such chemokines contributing to the disparity in prostate cancer biology among races needs further investigation. The establishment of such disproportionate levels of serum chemokines may help guide the therapeutic approaches in targeting aggressive prostate cancer as often diagnosed in men of AA race.

**Antibody mediated Targeted Drug Delivery approach for Pancreatic Tumors**

*Nirnoy Dan[1], Saini Setua[1], Poornima Shaji[2], Sonam Kumari[1], Murali M. Yallapu[1, 2, 3], Stephen Behrman[4], Subhash C. Chauhan[1, 2, 3] Sheema Khan[1,2,3]*
*[1]Department of Immunology and Microbiology, School of Medicine, University of Texas Rio Grande Valley, McAllen, TX 78504, United States [2] South Texas Center of Excellence in Cancer Research, School of Medicine, the University of Texas Rio Grande Valley, McAllen, TX, United States [3] Department of Pharmaceutical Sciences, College of Pharmacy, University of Tennessee Health Science Center; Memphis, TN, USA [4] Department of Surgery, Baptist Memorial Hospital and Medical Education, Memphis, TN 38120, USA*

Background: Pancreatic ductal adenocarcinomas, originating from the epithelial cell lining of ducts, account for approximately 95% of tumors in this category, showcasing a survival rate of less than 5-7%. Unfortunately, little progress has been seen in the outcomes of patients with PDAC as tumor develops high desmoplasia and chemo-resistance to chemotherapeutic drugs, such as gemcitabine (Gem). The therapies are unable to penetrate to the fibrotic tumors leading to insufficient availability of the therapeutic drugs at the tumor site. We and others have shown that MUC13 is aberrantly expressed in pancreatic tumors but not in normal pancreas, which makes MUC13 as an excellent protein for specifically targeting pancreatic tumors. Herein, we demonstrate a unique ability of our in-house generated mouse and humanized monoclonal antibody of MUC13 to penetrate and target pancreatic cancer.
Methods: These antibodies have been conjugated with our recently developed novel patented superparamagnetic iron oxide nanoparticles (SPIONS) to deliver therapeutics specifically to pancreatic tumors. In this study, we are using curcumin that depletes tumor microenvironment and gemcitabine to investigate the efficacy of the MUC13 conjugated SPION in delivery of therapeutic drugs.
Results: Our results demonstrate that enhanced uptake of MUC13-SPION formulation in MUC13 positive (MUC13+) PanCa cells, compared with MUC13 null (MUC13-) cells as demonstrated by immunofluorescence, Prussian blue staining and flow cytometry experiments. Interestingly, the formulation resulted in sustained delivery of curcumin (CUR), enhanced inhibition of cell proliferation, migration and invasion in MUC13+ cells as compared with MUC13- cells, which suggests the targeting efficacy of the formulation. In PanCa orthotopic mice model, MUC13-SPION efficiently targeted pancreatic tumors resulting in significant tumor accumulation.

We observed inhibition of tumor volume, metastasis, gem resistance and improved survival in mice treated with the formulation. Additionally, the tumor tissues from treated mice showed extensive downregulation of PCNA and expression of key proteins in SHH pathway, such as SHH, Gli-1, Gli-2, Patched 1, SMO, which has been associated with cancer progression and drug resistance. Conclusion: In conclusion, the results indicate high therapeutic significance of MUC13-SPIONS for achieving pancreatic tumor specific delivery of drugs.

**Antidiabetic drug Jardiance (Empagliflozin) effectively attenuated the weight gain induced by the antipsychotic drug Zyprexa (Olanzapine) in female Wistar rats**

*Ghulam Md Ashraf*
*Department of Medical Laboratory Sciences, College of Health Sciences, and Sharjah Institute for Medical Research, University of Sharjah, Sharjah 27272, United Arab Emirates*
*Email: gashraf@sharjah.ac.ae*
*Academic status of Presenting author: Faculty (Associate Professor)*
*Mode of presentation: Oral (online)*
*Discipline: Neuropsychopharmacology*

Atypical antipsychotic drugs are commonly associated with undesirable side effects including body weight gain (BWG) and metabolic deficits. Many pharmacological interventions have been tested to minimize or prevent these side effects. Preliminary evidence suggested that antidiabetic drugs may be effective in attenuating the BWG induced by antipsychotic drugs. In the first phase, we carried out a 28-day study to standardize the correlated effective dosage of the antidiabetic drug empagliflozin (EMPA) and the antipsychotic drug olanzapine (Ola). Rats were divided into control (vehicle), Ola-4 and Ola-8 (4 and 8 mg/kg/OD, IP, respectively), and EMPA-10 and EMPA-20 (10 and 20 mg/kg/OD, IG, respectively) groups. Both doses of Ola produced a significant increase in the percentage of BWG, however, Ola-4 produced a higher BWG. Also, both the doses of EMPA were able to reverse the effect of Ola-induced BWG; however, EMPA-20 produced a higher reversal in BWG and normalized the rat's body weight. So, we concluded that Ola-4 and EMPA-20 were the most effective dosage for experimental purposes in female Wistar rats. In the second phase of the study, we examined the effect of EMPA-20 on BWG induced by Ola-4 in female as well as male Wistar rats. Rats were divided into six groups based on the dosage they received: group 1 (female control), group 2 (female EMPA-20), group 3 (female Ola-4), group 4 (female Ola-4 + EMPA-20), group 5 (male control), and group 6 (male Ola-4). Ola induced sustained increase in BWG. The subsequent treatment of Group 3 and 4 with EMPA attenuated the Ola-induced BWG in female Wistar rats. In terms of the gender difference, the male control group 5 and male Ola group 6 gained more weight throughout the study as compared to the female control group 1 and female Ola group 3, respectively. However, Ola did not cause any weight difference between male rats treated with Ola in comparison with male control group, thus showing a significant gender difference regarding body weight between male and female Wistar rats regardless of Ola administration. In addition, the present findings showed that EMPA effectively attenuated the Ola-induced BWG in female Wistar rats. These novel findings should help to better understand the underlying molecular and behavioral mechanisms contributing to the observed increase in body weight after treatment.

**Assessing the Reliability, Internal Consistency, and Sensitivity of a Nutrition Knowledge Questionnaire for Four-Year-Old Pre-K Children**

*Wang L,[1] Cortez K,[2] Rodriguez B,[1] Reyes J,[1] Cisneros M,[1] Alaniz E,[1] Romero Z,[1] Salinas L,[1] Lopez-Alvarenga JC, [1]Trevino R[1]*
*1 University of Texas Rio Grande Valley*
*2 Social and Health Research Center*

Background: Assessing nutrition knowledge in four-year-olds, a developmental stage marked by limited attention spans and varying comprehension abilities, is challenging with traditional methods. A reliable test is pivotal for establishing a foundation for future health interventions.
Purpose & Hypothesis: We evaluated a 13-question nutrition and healthy habits test in Pre-K4 children to assess reliability and sensitivity to detect differences. Null hypothesis was used.
Methods & Results: Calculations included Cronbach's alpha, kappa coefficient, McNemar analysis by item, and Bland-Altman plots for test-retest differences. Mixed model regression assessed the questionnaire's sensitivity by sex and association with age. Item response theory (IRT) models were employed, generating latent abilities for students and individual scores using Bayesian modeling.
Conclusion: The Pre-K questionnaire exhibited consistency and validity. This evaluation is crucial for appraising the effectiveness of educational programs, fostering an improved understanding of nutrition, and promoting healthier dietary habits in young children.

**Clinical significance of targeting ribosome biogenesis in pancreatic cancer therapy**

*Mudassier Ahmad[1,2], Sahir Sultan Alvi[1,2], Haider Ahsan[1], Carlos Perez[1,3], Muhammad Bangash[1], Andrew Massey[4], Emmanuel Anning[1,3], Aun Bangash[1], Manu Sebastian[5], Manish Tripathi[1,2], Dae Joon Kim[1,2], Subhash C Chauhan[1,2], Bilal Bin Hafeez[1,2,3]*

*[1]Department of Medicine and Oncology ISU, [2]South Texas Center of Excellence in Cancer Research, School of Medicine, UTRGV, McAllen, TX 78504, [3]Graduate Program in Biochemistry and Molecular Biology, University of Texas Rio Grande Valley, Edinburg, TX [4]National Institute of Biomedical Imaging and Bioengineering, Section on Mechanobiology, Bethesda, MD, 20892, [5]School of Mathematical and Statistical Sciences, UTRGV, Edinburg TX*

Pancreatic cancer (PanCa) is the third leading cause of cancer-related deaths in the United States with limited therapeutic options available. Gemcitabine (GEM), a deoxycytidine nucleoside analog is currently considered the most effective therapy for PanCa. However, it shows only a marginal survival benefit of six months. Aberrant ribosome biogenesis occurs in most tumor types. We observed that PanCa cells are addicted to ribosome biogenesis (RiBi), which supports their highly aggressive metastatic phenotypes. Thus, strategically targeting RiBi process could be one of the ideal strategies for the prevention and treatment of PanCa. In this study, we elucidated the molecular mechanisms of POLR1A (RPA194) overexpression and how its targeting along with p53 status impacts RNA polymerase I inhibitor therapy against PanCa. The expression level of RPA194 was significantly elevated in pancreatic tumor tissues when compared with adjacent normal pancreatic tissues. BMH-21 is a potent pharmacological inhibitor of RNA Pol I, which is known to degrade RPA194 protein. Our results demonstrated that BMH-21 can selectively induce apoptosis in various PanCa cells but not in HPNE cells. We also found that the cytotoxic effect of BMH-21 was dependent on the expression pattern of RPA-194 and p53 status. We further examined the therapeutic efficacy of BMH-21 in orthotopic xenograft mouse models by using two different PanCa cells, AsPC1 which contains non-functional p53 and MIA PaCa-2 which contains functional mutant p53. We observed that BMH-21 significantly inhibited the growth of tumors derived from both cancer cell lines. Interestingly, BMH-21-mediated inhibition of tumor growth was more significant in tumors derived from MIA PaCa-2 cells compared to AsPC1 cells. Further examination revealed that the inhibition of tumor growth was correlated with RPA194 degradation followed by inhibition of cell proliferation. Overall, our results strongly suggest that BMH-21 is a promising non-toxic agent for the treatment of advanced PanCa and its therapeutic potential depends on RPA194 expression and p53 status in PanCa cells.

**Effect of Alcohol Consumption on Cognitive Decline among Mexican Adults**

*Mejia-Arango S., Salloum I., & Maestre, G.*

Background. Studies on the association between alcohol use and cognitive impairment have yielded controversial results suggesting a reduced risk of dementia in drinkers vs. nondrinkers. We aimed to examine the effect of alcohol use on cognitive trajectories among Mexican adults aged 50 and over.

Methods. Data are from 5,898 cognitively normal individuals (2,512 men and 3,386 women) from the Mexican Health and Aging Study (MHAS) with a mean age of 59 years (50-90 years) at baseline (2001) and followed up after 11 years (2012). The Cross Cultural Cognitive Examination was the cognitive battery measuring verbal memory, visual memory, attention, and constructional praxis. Impairment was defined if scores in two or more functions were 1.5 standard deviations below the mean based on norms by age and education. Using self-reported data on frequency and quantity of alcohol consumption, as well as scores on the CAGE questionnaire, a 4-question screening tool for the detection of alcoholism, we constructed a comprehensive measure to classify alcohol use into five categories: never, mild, moderate, and heavy drinking. Those who reported they had never drunk in their life were classified as never drinkers, mild drinkers included those drinking less than one day per week, and those who had drunk before and scored zero in the CAGE questionnaire, moderate drinkers included those who reported having one or two drinks each day, and as heavy drinkers, we classified those who reported having three or more drinks per day and those who drunk before and scored one or more in the CAGE questionnaire. Multivariate logistic regression models were used to analyze the predictive role of alcohol consumption in developing cognitive impairment. Sociodemographic and health variables known to increase the risk of cognitive decline (age, education, gender, locality, cardiovascular risk factors, smoking, and depressive symptoms) were included as covariates.

Results. At follow-up, 4,844 participants (82.1%) remained cognitively normal, while 1,054 participants (18%) showed impaired cognition. When the more generally classified drinkers were compared to never-drinkers, they had a reduced risk of cognitive impairment (OR 0.78, 95% CI 0.64-0.95). However, when we analyzed mild, moderate, and heavy drinkers compared to never-drinkers, only mild drinkers had a significantly reduced risk of cognitive decline (OR 0.76, 95% CI 0.62-0.93). The reduced risk of cognitive impairment in moderate drinkers (OR 0.72, 95% CI; 0.49-1.05) and heavy drinkers (OR 0.90, 95%CI .70-1.16) was not significant. Conclusions: Our results suggest that among drinkers, only mild drinking has a protective effect on cognitive decline in Mexican adults aged 50 years and older

**Healthcare Professional's Attitudes Towards Interprofessional Collaboration**

*Martin Ekoumou. MD, MPH, PhD-C*
*Clinical Assistant Professor / Clinical Coordinator: UTRGV Physician Assistant Department*
*Jerome Fisher, PhD*
*Ret. Professor, School of Rehabilitation Services and Counseling / UTRGV COHP*

Background: Traditional healthcare has been characterized as a field where professionals work independently, creating an environment with multiple providers in multiple locations duplicating services, adding to the ineffectiveness and waste of resources. The fragmentation of healthcare provision promotes isolation, competition and attempts to preserve power among professions. Accordingly, patient care is less than optimum. To begin to ameliorate this situation Interprofessional Education has been developed. However, a significant part of this education is effective interprofessional collaboration (IPC) which creates higher patient satisfaction, better coordination of patient care, efficient use of healthcare services and higher professional job satisfaction. The purpose of this research was to determine attitudes of Healthcare Professionals towards IPC.
Methods: An online survey, the PINCOMQ, was distributed to healthcare professionals including doctors, nurses, physician assistants, social workers, and rehabilitation counselors. A total of 193 useable responses were received.
Results: On a seven-point scale the overall rating for the 48 items was mean of 4.99 (SD: 64) which was as slightly positive attitude towards IPC. The lowest rated factor of 12 factors was Personality as a part of IPC with a mean of 3.78 (SD: .98) which was slightly negative. The highest rated factor was Professional Power with a mean of 6.34 (SD: .84) which was positive.
Conclusions: Healthcare Professionals need more training in IPC. Although they have some positive attitudes about factors of IPC, many factors can be improved.
Keywords: Interprofessional collaboration, interprofessional education, global, healthcare, fragmentation, healthcare ethic

**LncRNA Malat1 as a novel stress-related factor in Hepatocellular Carcinoma**

*Amayrani Sanchez, Kyle Doxtater, Md. Shabir Hussain, Sophia Leslie, Subhash C. Chauhan, Elias George, Manish K. Tripathi*

As a result of the fast life pace, stress, and a major dietary shift toward preserved food, hepatocellular carcinoma (HCC) incidence and mortality are on rising trend, thus posing severe concerns. Texas is expected to have the second highest number of deaths related to liver cancer, with Hispanics having the highest mortality rate. HCC accounts for 85% to 90% of liver cancers. The Rio Grande Valley (RGV) region, where a predominantly (~90%) Latino/Hispanic population resides, has ~4-fold higher prevalence of liver cirrhosis and is a major hotspot of HCC in the nation. In addition, it was found to be positively correlated with diabetes and obesity. Thus, the RGV region is severely affected by the disproportionate burden of HCC incidence and mortality. As per recent SEER data, the five-year survival rate in this disease drops from 35% to 2% of patients diagnosed with regional and distant stages. Unfortunately, no adequate and specific molecular markers are available that can detect HCC at the early onset of disease. Additionally, how different socio-behavioral, dietary, and stress factors influence the molecular drivers of HCC are not fully understood. It has been demonstrated that, apart from family history, different socio-behavioral factors, certain diets, alcohol, and smoking are associated with *dysregulated functioning of a major endocrine* (HPA; Hypothalamus-Pituitary-Adrenal) axis and higher levels of biochemical stressors (cortisol, cytokines, leptin). These are active areas of research in the HCC field to aid the discovery of new early diagnostic molecular markers and define molecular triggers of the disease. *Recently, we have observed that a Long noncoding RNA (LncRNA), Metastasis Associated Lung Adenocarcinoma Transcript 1 (LncRNA MALAT1), is involved in HCC pathogenesis. It* is regulated by the transcription factor *Nuclear Factor of Activated T cell 1* (NFATc1)*, a poor survival indicator of cancer patients.* We have developed a novel, clinically applicable Z-Probe-based RNAScope Technology for detecting LncRNAs (such as MALAT1) on tumor tissues just like immunohistochemistry. This will allow us to investigate MALAT1 expression about NFATc1 and establish the association of these two distinct class of molecular markers (LncRNA and NFATC1 protein) in HCC samples. We are investigating how biochemical stress factors can influence the expression of these two oncogenic drivers.

**Novel strategy to make KRAS targeted therapies more effective for PDAC treatment**

*Ana I. Martinez Bulnes[1], Orlando Garcia[1], Poornima Shaji[1,2], Swathi Holla[1,3], Nirnoy Dan[4], Anupam Dhasmana[1], Shabnam Malik[1], Murali Yallapu[1], Stephen Behrman[5], Subhash C. Chauhan[1] and* ***Sheema Khan***[1]
[1]*Department of Immunology and Microbiology, School of Medicine, University of Texas Rio Grande Valley, TX 78539, USA*
[2]*Department of Otolaryngology, UT Southwestern Medical Center, 5323 Harry Hines Blvd, Dallas, TX 75390*
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[4]*University of Connecticut, 55 N. Eagleville Road, Connecticut 06269-3060*
[5]*University of Tennessee Health Science Center, 800 Madison Ave, Memphis, TN 38103, and Baptist Memorial Hospital, 8970 Winchester Rd, Memphis, TN 38125*

Introduction: Pancreatic Ductal Adenocarcinoma (PDAC) patients exhibit extremely poor prognosis. KRAS mutation on codon-12 is present in 70–95% of PDAC cases and it drives PDAC growth and progression. Galectin-1 (Gal-1) is present in both PDAC and stromal cells, being involved in tumor microenvironment, immune cell activation and metastasis. Therefore, this study discusses the efficiency of combined inhibition of mutated $KRAS^{G12D}$ and Gal-1 inhibition to effectively suppress PDAC growth and progression. For this we have delivered $KRAS^{G12D}$ inhibiting siRNA ($siKRAS^{G12D}$) using a superparamagnetic iron oxide nanoparticle (SPION) and a galectin inhibitor.
Methods: SPION nano-formulation was used to deliver $siKRAS^{G12D}$ and investigate in conjunction with Gal-1 inhibitor for its anticancer efficacy. Particles were investigated for size, physico-chemical characterization (Dynamic light scattering), hemocompatibility (hemolysis assay) and the complexation of siKRAS (gel retardation assay). Cellular internalization and uptake of the particles were investigated. Anti-cancer efficacy was determined using *in vitro* functional assays for cell viability (MTT), migration (Boyden chambers), invasion (Matrigel), clonogenicity, tumor spheroid formation, and in a *KrasG12D;LSL-Trp53R172H* syngeneic mouse model.
Results: Our results demonstrate that SP-siKRAS efficiently internalized in PDAC cells and suppressed $KRAS^{G12D}$ as well as its downstream targets, YAP and PDL-1. Combined targeting of siKRAS and Gal-1 inhibited cell proliferation, clonogenicity, migration, and invasion of PDAC cells and tumor spheroid growth in 3D cell models, which recapitulate the heterogeneity and pathophysiology of PDAC. We have used -*KrasG12D;LSL-Trp53R172H* syngeneic mouse model of PDAC for investigating efficacy of combined SP-siKRAS formulation and galectin-1 inhibitor. Our results showed that the combination treatment inhibited the fibrotic tumor growth and increased survival rate. The combined treatment increased infiltration of total T cell population and CD8+T cells, reduced the population of myeloid-derived suppressor cells (MDSCs) by 50% (CD45+, CD3-, CD11b+, Ly6C high, Ly6G-) and T-Regulatory cells (Treg) by 57% (FoxP3+CD25+CD45+CD3+) and increased memory T cells by 34% in mice.
Conclusion: This gene therapy targeting KRAS G12D mutation with a Gal-1 inhibition has a potential to modulate the oncogenic network and tumor microenvironment resulting in the repression of growth, metastasis, chemoresistance, and improvement in patient survival. This study will develop a novel sustainable therapeutic approach to target PDAC growth and improve patient survivability.

**Novel therapeutic strategy for cervical cancer treatment**

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Abstract
Aberrant activation of β-catenin signaling is strongly associated with cancer proliferation, invasion, migration, and metastasis, thus small molecules that can inhibit this pathway might have great clinical significance. Our molecular modeling studies suggest that Ormeloxifene (ORM), a triphenylethylene molecule docks with β-catenin, and its brominated analogue (Br-ORM) bind more effectively with relatively less energy (-7.6 kcal/mol) to the active site of β-catenin as compared to parent ORM. Herein, we report the synthesis and characterization of a Br-ORM by NMR and FTIR, as well as its anti-cancer potential in cervical cancer models *in vitro* and *in vivo*.Br-ORM treatment effectively inhibited tumorigenic features (cell proliferation and colony forming ability, etc.) and induced apoptotic death as evident by pronounced PARP-cleavage and arrest of cells in G1-S phase of cell cycle. Further, mechanistic investigations revealed that Br-ORM targets the key proteins involved in promoting epithelial mesenchymal transition (EMT) as demonstrated by upregulation of E-cadherin expression and repression of N-cadherin, Vimentin, Snail, MMP-2, -9 expression. Br-ORM also represses the expression and nuclear subcellular localization of β-catenin. Consequently, Br-ORM treatment effectively inhibited tumor growth in orthotopic cervical cancer xenograft mouse model along with EMT associated changes as compared to vehicle control treated mice. Altogether, *our in vitro* and pre-clinical *in vivo* findings suggest that Br-ORM is a novel, promising β-catenin inhibitor, therefore can be harnessed as a potent anti-cancer small molecule for the treatment of aberrant wnt/β-catenin signaling induced cancers, including cervical cancer.

Keywords: Cervical cancer, β-catenin, EMT; Epithelial to Mesenchymal Transition, Bromo-ormeloxifene

**Piperlongumine nanoformulation attenuates pancreatic tumor desmoplasia and alter tumor immune responses**

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Abstract:
Pancreatic cancer (PanCa) is characterized by lack of early diagnosis, poor response to available therapeutic modalities and chemoresistance. Gemcitabine (GEM) is currently considered the most effective therapy for PanCa; however, it shows only a marginal survival benefit of 6 months. This poor drug response has been attributed to desmoplasia, causes suboptimal drug delivery, alters tumor microenvironment (TME), which includes tumor surrounding blood vessels, fibroblasts, immune cells, extracellular matrix, and other signaling molecules and induces chemo-resistance in tumors. To overcome these existing issues associated with chemotherapy, identification and development of novel therapeutic modalities are a pressing need. Piperlongumine (PL) is a natural alkaloid isolated from the long pepper, Piper longum L., and has shown substantial cancer-preventive and therapeutic efficacy against a variety of cancers. However, delivering its effective concentration in pancreatic tumors has been challenging. We have recently engineered a multi-layered Pluronic F127 and polyvinyl alcohol stabilized, and poly-L-lysine coated piperlongumine loaded poly(lactic-coglycolic acid) nanoparticle formulation (PLGA-PL), which effectively inhibits the growth of PanCa cells. In this study, we demonstrate that PLGA-PL effectively sensitizes tumor cells to GEM via decreased desmoplasia, altered TME, SHH/CXCL12/CXCR4 and immune surveillance. Our finding show that PLGA-PL synergizes with GEM in inhibiting PanCa cell (HPAF-II and Panc-1) growth, migration, and invasion compared to free PL. Mechanistically, PLGA-PL targets the TME via inhibition of sonic hedgehog (SHH) pathway and oncogenic CXCR4/CXCL12 signaling axis that inhibits bidirectional tumor-stromal cells interaction. We have also found that PLGA-PL alone and in combination with GEM targets cancer stem cells by inhibiting pluripotency maintaining stemness factors (Nanog, Sox2, c-Myc, CD133, and Oct-4) as determined by qRT-PCR, Western blotting, and immunofluorescence analysis, and further confirmed by restricting tumor sphere formation. Furthermore, PLGA-PL also effectively targets tumor-associated macrophages (TAM) by repolarizing M2 into M1 phenotype via inhibiting expression of M2 markers and an increase in M1 markers in mouse macrophage cell line RAW264.7. M2 polarization of RAW264.7 cells were induced by culture with IL-4 (20 ng/mL) in presence of PLGA-PL or vehicle control. In addition, PLGA-PL effectively increases phagocytic capacity in murine macrophages as determined by phagocytosis assay (Vybrant Phagocytosis Assay Kit). In conclusion, we observed that PLGAPL effectively targets TME, facilitates GEM uptake by inhibiting the activation of CXCR4/CXCL12/SHH signaling, and reprograming the tumor immune surveillance. This study suggests that PLGA-PL has great potential for future clinical use in management of PanCa.
Keywords: Pancreatic cancer, Gemcitabine, nanoformulation, tumor-associated Macrophages

**Revolutionizing Feature Selection: A Breakthrough Approach for Enhanced Accuracy and Reduced Dimensions, with Implications for Early Medical Diagnostics**

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BACKGROUND: The system's performance may be impacted by the high-dimensional feature Feature Selector (Fs) serves as an operator, transforming an m-dimensional feature set into an n-dimensional feature set. This process aims to generate a filtered dataset with reduced dimensions, enhancing the algorithm's efficiency.
METHODS: This paper introduces an innovative feature selection approach utilizing a genetic algorithm with an ensemble crossover operation to generate the most robust offsprings, i.e., feature set solutions. The selection process is driven by computed minimum objective function values (OFVs). Experimental evaluations were conducted across diverse datasets sourced from various repositories, all processed through a standardized classifier. A comparative analysis was performed, contrasting the proposed feature selection system with various traditional counterparts.
RESULTS: Our innovative approach yielded superior results compared to conventional feature selection techniques in terms of accuracy and the reduction in the number of features. This holds significant promise as a valuable tool for the diagnosis of lung cancer and pancreatic cancer.
CONCLUSION: In addition to surpassing traditional feature selection techniques in accuracy and feature reduction, our novel approach holds great potential across diverse fields, with a particularly promising impact in the medical domain for early diagnostics. This advancement could contribute significantly to the timely and effective identification of medical conditions, enhancing the overall capabilities of diagnostic processes.

**Risk of Food Insecurity Reflects Health Status in Adult Relatives of PreK Children – Data from the STEPS Snap-Ed Research Study**

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Background. Past research shows a correlation between household food insecurity and chronic medical conditions such as diabetes in the general U.S. population as well as metabolic syndrome-related cardiometabolic markers in Hispanic/Latino youth. Furthermore, minority status, including Hispanic/Latino identity, shows an association with low food security and diabetes. The Hunger Vital Sign (HVS) is a validated screener designed to assess risk of food insecurity within households. This study analyzes associated factors related to the screener's 2 questions, specifically targeting PreK 4-year-old children and their household relatives. The first question assesses concern about food running out before obtaining more due to financial constraints, while the second question evaluates instances when food did not last and there was insufficient money to purchase more.
Methods. The participating preschools were primarily Hispanic/Latino and nested within schools and school districts. Data were collected through household questionnaires. The frequency of food insecurity (FI) risk was measured as an ordinal variable (never, sometimes, often) and dichotomized (never vs sometimes + often). Agreement between questions was assessed using the kappa coefficient. Mixed models were employed to predict the risk of children's BMI and the risk of relatives developing chronic disease, adjusting for age and sex.
Results. The study included 828 families, with 74% reporting never experiencing food insecurity, 22% sometimes, and 4% often. The agreement between the 2 survey questions was substantial (kappa 0.80, $p < 0.001$). Probability of FI decreased as income level and education level of parents increased. No significant associations were found between FI and children's age, BMI, height, or the ratio of children to adults in the household. However, a positive screen using the HVS correlated with hypertension and/or diabetes in the family.
Conclusion. The HVS screens for food insecurity risk and is associated with health issues in adults rather than children's body weight or height. Responses to the survey's 2 questions can provide insights into the overall health status of a household, emphasizing the intricate relationship between food insecurity, malnutrition, and various health outcomes. The presented results support planning and resource allocation strategies to reduce food insecurity in the Hispanic/Latino population.

**Role of TRPV6 in Mitigating Alcohol-Induced Disruption of Tight Junctions, Barrier Function, and Hepatic Injury**

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Background: Persistent alcohol consumption is widely recognized as a precursor to alcoholic liver disease. However, the intriguing observation persists that only a minority, approximately 20%, of individuals with alcohol use disorder succumb to this liver ailment. The factors contributing to this variability remain elusive. Studies indicate that individuals with alcoholic liver disease exhibit endotoxemia, with endotoxins primarily originating from colonic microflora. Moreover, these patients manifest disruptions in epithelial tight junctions, leading to compromised barrier function in the gastrointestinal tract. In this context, the transient receptor vanilloid receptor 6 (TRPV6) emerges as a crucial regulator of calcium absorption and transport, particularly in epithelial cells within the gastrointestinal tract. Research strongly suggests that suppressing the TRPV6 channel in Caco-2 cells can alleviate alcohol-induced disruption of tight junctions and barrier function.
Methods, Results, and Conclusions: A scientific study subjected adult wild-type and $Trpv6^{-/-}$ mice to chronic alcohol feeding. Barrier function was assessed through *in vivo* measurement of inulin permeability, while tight junctions (TJ) and adherens junctions (AJ) integrity were evaluated using immunofluorescence microscopy. Systemic responses were analysed by assessing endotoxemia, systemic inflammation, and liver damage. Our findings highlight that alcohol induces the redistribution of tight junctions and adherens junctions, closely associated with the presence of TRPV6. Crucially, experiments with murine models reveal that the absence of TRPV6 mitigates alcohol-induced disruption of tight junctions, adherens junctions, gut barrier integrity, endotoxin absorption, and subsequent liver damage. Additionally, enteroids and colonoids generated from mice demonstrate that alcohol and its metabolite, acetaldehyde, increase the permeability of these organoids. Interestingly, organoids derived from TRPV6 knockout animals exhibit resistance to heightened permeability. Collectively, these findings suggest a pivotal role for the TRPV6 channel in mediating alcohol-induced damage to the gastrointestinal tract and liver. Our research provides valuable insights into potential mechanisms underlying alcohol-induced liver disease and emphasizes the significance of TRPV6 as a promising target for further exploration and potential therapeutic interventions.
Keywords: TRPV6,

**Targeting Ion Channels in Liver Cancer Cells: Stimulating Lysosomal TRPML1 and Inhibiting hEAG1 Potently Reduce Cell Viability**

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Abstract:
Background: Liver cancer has high mortality with few treatment options. This demands new therapies. Ion channels represent attractive targets as these membrane pores regulate ion flux, altering cancer cell behavior. While channels enabling liver cancer are recognized, the functional roles of lysosomal Zn2+ release channels and oncogenic plasma membrane K+ channels in driving pathogenesis remain unclear. We investigated if purposefully modulating their activities negatively impacts cancer cell survival.
Methods: We employed organelle electrophysiology, Zn2+/ROS imaging, voltage imaging, electron microscopy and small-molecule channel modulators to directly activate lysosomal TRPML1 channels and inhibit hEAG1 oncogenic K+ channels in liver cancer cells. We comprehensively characterized downstream cytotoxic effects from disrupting ion homeostasis and channel activities.
Results: Pharmacologically stimulating TRPML1 Zn2+channels triggered lysosomal Zn2+ release, elevating ROS and blocking autophagic flux, ultimately causing widespread cell death. Additionally, we found the endogenous hEAG1 channel inhibitor heme at nanomolar concentrations suppressed hEAG1 activity, depolarized plasma membrane potential, and dramatically reduced cell survival.
Conclusions: By activating lysosomal TRPML1 channels or inhibiting hEAG1 oncogenic K+ channel activity uniformly disrupted ion homeostasis, channel function and potently reduced liver cancer cell growth/viability. These discoveries reveal targeting these channels presents a compelling therapeutic strategy for combating liver cancer progression

# POST-DOC/FELLOW CATEGORY

**Assessment of Mucin 13 (MUC13) as an Imaging Target for Guiding Colorectal Cancer Treatment: A Pathway Towards Theranostic Development**

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Background: A theranostic strategy combining diagnostic imaging and targeted therapy in a single regimen is proposed for improved management and treatment of colorectal cancer (CRC). Increased specificity in detection by the noninvasive imaging technique positron emission tomography (PET) can be achieved by radiolabeling antibodies (Abs) designed to target tumor-associated antigens with increased expression post-translational modifications present in cancer cells. In this study, an Ab designed to target the transmembrane glycoprotein mucin 13 (MUC13) was radiolabeled with the positron-emitting radionuclide zirconium-89 (89Zr) for PET imaging of a xenograft mouse model of CRC. Specified uptake of this radioimmunoconjugate was observed in the presence of increased MUC13 expression was observed through imaging along with in vitro and ex vivo analyses.
Methods: Radiochemistry: The MUC13-targeting Ab C14 conjugated with desferrioxamine (DFO) was radiolabeled with 89Zr alongside isotype control Ab MOPC-21 (IgG) at a 59 kBq/µg (1.6 µCi/µg) ratio, producing [ 89Zr]Zr-DFO-C14 and [89Zr]Zr-DFO-IgG. Radiochemical purity (RCP) was determined using radio-iTLC and radio-SEC. Radiochemical yield (RCY) was determined with a well-type dose calibrator. Cellular Binding and Internalization: Cultured human CRC cell lines T84 (MUC13+) and SW480 (MUC13- ) were incubated with either [89Zr]Zr-DFO-C14 or [89Zr]Zr-DFO-IgG. At 2 and 24h, cell membranes were separated and radioactivity measured to compare membrane-bound and cell-internalized activity. To determine binding specificity of radiolabeled C14, cells were co-incubated with excess unmodified Ab. µPET/CT Imaging: T84 and SW480 cells were introduced subcutaneously in athymic nude mice. Once palpable tumors were detected, mice were placed in the following treatment groups for 1.9 MBq (50 µCi) injection: T84+[89Zr]Zr-DFO-C14 (n=5), T84+[89Zr]Zr-DFO-C14 with 350 µg C14 (n=2), SW480+[89Zr]Zr-DFO-C14 (n=5), and T84+[89Zr]Zr-DFO-IgG (n=4). PET imaging was performed 24, 48, and 120h post-injection (p.i.) alongside computational tomography (CT) imaging to provide anatomical context. After 120h, mice were euthanized and blood, organs, and tissues were collected to measure radioactivity biodistribution and radioimmunoconjugate distribution in tumor tissue.
Results: Radiolabeled C14 and IgG were successfully produced with RCY>83% (n.d.c.) and RCP>95%. Reflecting rapid internalization observed in vitro (57.9±13% [89Zr]Zr-DFO-C14 uptake in T84 at 2h compared to 6.57±0.6% uptake in SW480 ($p<0.0001$) and 0.39±0.1% [89Zr]Zr-DFO-IgG uptake ($p<0.0001$)), mice bearing T84 xenografts displayed greater signal intensity from [ 89Zr]Zr-DFO-C14 at 24h p.i. through 120h p.i. compared to that measured in SW480 xenografts (5.5±0.7% ID/cc vs. 2.8±0.5% ID/cc at 24h p.i., $p<0.0001$) as well as that in T84-bearing mice injected with [ 89Zr]Zr-DFO-IgG (1.9±0.2% ID/cc at 24h p.i., $p<0.0001$). Autoradiography revealed high, homogeneous distribution of [ 89Zr]Zr-DFOC14 within the tumor. Furthermore, co-injection with excess C14 resulted in reduced PET signal (2.7±0.1% ID/cc, $p=0.0002$), supporting the targeting specificity of [89Zr]Zr-DFO-C14. Ex vivo biodistribution comparison confirmed high, persistent [89Zr]Zr-DFO-C14 uptake in T84-derived tumor (18.5% ID/g at 120h p.i.).
Conclusion: MUC13 expression was clearly represented by PET/CT imaging in a xenograft mouse model of CRC using a 89Zr-labeled MUC13-targeting Ab, which also demonstrated target specificity both in vitro and ex vivo. These promising results justify further exploration into developing a theranostic platform for CRC treatment. Future work will test the therapeutic efficacy of the MUC13-targeting Ab radiolabeled with a beta particle-emitting radionuclide.

**Metabolic reprogramming in breast cancer patients as revealed by $^{1}$H NMR spectroscopy**

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Background: Breast cancer is a global health concern among women. Several metabolic pathways are dysregulated in breast cancer cells, including alterations in energy metabolism, amino acid metabolism, and lipid metabolism. Reprogramming of metabolic pathways may facilitate inappropriate proliferation of cancer cells and adaptation to the tumor microenvironment. Long non-coding RNAs (lncRNAs) have emerged as important regulatory targets in the process of tumorigenesis. However, the role of lncRNAs in the process of metabolic reprogramming is not properly known. Exploring metabolic alterations and its association with lncRNAs expression might be helpful for developing new biomarkers and therapeutic targets for cancer management.

Objectives: Serum from 43 breast cancer patients and 13 healthy individuals were used for the analysis of metabolic profile.
Methods: For the identification and quantification of metabolites, $^{1}$H NMR spectroscopy was used while for lncRNAs expression, q-RT-PCR was used.
Results: Metabolites such as amino acids, lipids, membrane metabolites, lipoproteins, and energy metabolites were observed in the serum of both patients and healthy individuals. The serum of patients and healthy individuals produced measurable amounts of metabolites related to lipoproteins, amino acids, membrane, lipids, and energy. The unsupervised PCA, supervised PLS-DA, supervised OPLS-DA, and random forest classification analyses revealed alterations in more than 25 metabolites. Further analysis of metabolites with AUC value >0.9 revealed significant elevation in the levels of LPR, glycerol, and lactate, while the levels of succinate, glucose, and isobutyrate was reduced in comparison to healthy control. The advanced stage breast cancer patients revealed alterations in these metabolites (except LPR) in comparison to early breast cancer patients. Over 25 metabolic signaling pathways were associated with altered metabolites. Further, a dysregulation in MEG3, H19, and GAS5 lncRNAs were observed in the breast tumor tissue in comparison to normal adjacent tissue.
Conclusion: The study reveals that metabolic pathways are altered in breast cancer patients. The study also opens a window for examining the association of lncRNAs with metabolic patterns in patients.

**Moringin, an isothiocyanate improves the susceptibility of breast cancer cells to doxorubicin**

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Background: Breast cancer has become the most frequent tumor worldwide and is expected to have a large impact on cancer fatalities globally. Thus, the development of precise molecular diagnosis and prognosis is essential for the effective treatment of breast cancer patients. Doxorubicin (DOXO) is the widely accepted chemotherapeutic drug for the treatment of breast cancer. However, the primary challenge with current chemotherapy drugs is their toxic effects on healthy tissues. Moringin (MG), an Isothiocyanate (ITC) is produced from the seeds of *Moringa oleifera*. It is produced from the myrosinase-catalyzed hydrolysis of glucomoringin. Chemosensitization is a strategy used in cancer treatment to make cancerous cells more responsive to chemotherapeutic drugs. A chemosensitizing drug is used in therapy to reduce the doses of potential anticancer drugs, tackle the resistance of cancer cells to these treatments, prevent healthy cells from toxicity, and decrease side effects for patients. In this study, we investigated the potential of MG alone and in conjunction with DOXO against breast cancer cells.
Methods: MCF-7 and MDA-MB-231 breast cancer cell lines were used in the investigation. The chemo-sensitization experiments of MG, both alone and in combination with DOXO, were investigated by using the MTT and colony formation assay. Apoptosis was examined by using the AO/PI dual staining, cell cycle analysis, and mitochondrial membrane potential measurement. Expression of cell survival proteins was assessed through semi-quantitative PCR and western blotting. Expression of long non-coding RNAs was performed by qRT-PCR.
Results: The MTT and clonogenic assay results revealed the anti-proliferative potential of MG both alone and in combination of DOXO. The expression of proteins involved in cell survival (PARP, Bcl-2, Bcl-xL and Survivin) is also downregulated in combination compared to individual drug. We observed an increase in the sub-G1 and S-phase population in MG and DOXO (both individual as well in combination). However, the G1 and G2/M population was slightly decreased with MG and DOXO (both individual as well in combination). The expression of oncogenic lncRNAs such as H19, HOTAIR and NKILA, were reduced and the expression of tumor suppressor genes such as MEG3, GAS5 and MALAT1 was increased.
Conclusions: Taken together, results of this study provide evidence that, MG in breast cancer cell lines increases the susceptibility of breast cancer cells to DOXO.

**Proteomic analysis of stress associated factor overexpression in Hepatocellular carcinoma**

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Abstract:
Background: Hepatocellular carcinoma (HCC) constitutes a substantial portion, accounting for 85% to 90% of liver cancers worldwide. Notably, within the Hispanic population, liver cancer mortality rates are notably higher, particularly evident in regions like the South Texas Rio Grande Valley (RGV), where nearly 90% of the populace is Latino/Hispanic. This region grapples with poverty affecting nearly 30% of its residents, coupled with elevated rates of obesity, diabetes, and low-income households, thereby fostering a prevalent environment of stress. Stress can profoundly impact cancer outcomes by compromising immune functionality and triggering inflammatory responses, potentially impairing surveillance against oncogenic triggers.

The activation of the Hypothalamic-Pituitary-Adrenal (HPA) axis during stress results in the release of stress hormones like leptin and cortisol, possibly influencing cancer tumor progression and growth. The cumulative effects of stress, anxiety, and depression can detrimentally affect liver health, impairing hepatic cell function and regeneration, ultimately expediting HCC progression. This study has identified the Long Non-coding RNA (LncRNA) MALAT1 as overexpressed under stress conditions. The Cancer Genome Atlas (TCGA) database, we've analyzed lncRNA expression in HCC patient cohorts. We want to comprehend the proteins and pathways influenced by stressassociated lncRNA MALAT1 expression in HCC, thereby illuminating potential mechanisms driving HCC progression under stressful conditions.

Methods: We engineered a stable cell line (SK-Hep1_Malat1) overexpressing LncRNA Malat1, utilizing transient transfection, puromycin selection, and FACS enrichment for GFP expression. Additionally, a vector-only control cell line was established for comparative analysis. TCGA data encompassing HCC patient tumors was analyzed to explore LncRNA Malat1's influence. Subsequently, High Throughput Nano-Liquid Chromatography Mass Spectrometry (LC-MS) was employed to identify proteins and pathways modulated in stable LncRNA Malat1- expressing cell lines. Further analyses included utilizing the Human Protein Atlas, Mechanism, Pathway Enrichment analyses, and STRING pathway analysis.

Results: The Cancer Genome Atlas (TCGA) database revealed heigh LncRNA Malat1 levels in HCC patient tumors compared to control tissues. Additionally, LncRNA Malat1 expression exhibited significant correlations with HCC progression, metastasis, and unfavorable clinical outcomes. We observed an upregulation of LncRNA MALAT1, ZNF384, and YB-1 genes to varying degrees, influenced by exposure to stress hormones (cortisol, leptin), and treatment duration. Furthermore, we identified the proteins and pathways modulated by lncRNA Malat1 overexpression in the SK-Hep1 cell line. The comprehensive results detailing these molecular alterations and pathway modulations will be presented.

Conclusion: The identified differential proteins will undergo rigorous validation using quantitative techniques such as western blotting, immunoprecipitation, co-immunoprecipitation, and immune-fluorescence assays. Future investigations will encompass skimmed assessments to deliver deeper into the modulated pathways. Our findings illuminate the upregulation of oncogenic long non-coding RNA and transcription factors, outlining a signaling pathway model influenced by stress factors in HCC cell lines. Understanding the pivotal role of stress factors in oncogenicity and HCC progression holds promise in developing therapeutics, particularly for populations where stress contributes significantly to cancer disparities. These insights offer potential avenues for targeted interventions aimed at mitigating the impact of stress on HCC, potentially improving outcomes for affected populations.

# MEDICAL RESIDENT CATEGORY

**An Unusual Presentation of the Severe Hypothyroidism Presenting As Shortness Of Breath**

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Background
Hypothyroidism is defined as failure of the thyroid gland to produce sufficient thyroid hormone to meet the metabolic demands of the body. Untreated hypothyroidism can contribute to hypertension, dyslipidemia, infertility, cognitive impairment, and neuromuscular dysfunction. It may occur as a result of primary gland failure or insufficient thyroid gland stimulation by the hypothalamus or pituitary gland. Primary gland failure can result from the congenital abnormalities, autoimmune destruction, iodine deficiency, and infiltrative diseases. Patients can clinically present with weight gain, cold intolerance, depression, muscle fatigue, poor concentration, and menstrual irregularities. The best laboratory test for the thyroid assessment is serum TSH test. If the TSH is elevated the serum free thyroxine (T4) should be done. Therefore, here we present a case of the young lady with severe hypothyroidism presenting with shortness of breath.
Case Discussion
A 31 year old lady with no known significant past medical history presented to the emergency department complaining of the shortness of breath which has been ongoing from last 6 months. She also mentioned that she has been more constipated than usual and attributed her symptoms to constipation. On further evaluation, vitals on admission were HR of 50s and BP 90/50s. The orthostatic vital signs were performed and were negative. The physical examination was positive for hair loss, dry skin, muffled heart sound and pallor of conjunctiva was noted. Further the labs were drawn and significant labs showed hemoglobin of 5.4 g/dl, CK of 344 u/L, LDL of 182 mg/dl. 1 unit of packed red blood cells was given and simultaneously Chest X Ray was done which showed boot shaped heart and EKG was done which showed sinus bradycardia. Another set of blood was collected for the lactic acid, troponin and TSH. All were normal except TSH was 257.34 uIU/ml. The patient was diagnosed with severe hypothyroidism and further free T4 was 0.4ng/dl which was significantly low. Started on the Levothyroxine 1.6 micrograms/kg/day which rounded up as 100 micrograms daily for her. As soon as the levothyroxine was given, within 1 hour her heart rate and blood pressure and her symptoms started resolving. Bedside ultrasound was done which showed the moderate pericardial effusion, which was cause of the dyspnea. The patient was worked up for the autoimmune hypothyroidism and everything was negative, therefore was patient was safely discharged home with Levothyroxine 100 micrograms and advised to follow up with the PCP for the dose titration.
Conclusion
The main learning point from this is that severe hypothyroidism can disguise in different ways and present differently. The key point is to have sharp suspicion for the hypothyroidism if chest xray shows boot shaped heart and bradycardia. The pericardial effusions can occur in patients with severe untreated hypothyroidism and can be just treated with the oral levothyroxine and pericardial effusion will resolve eventually.

**Case of Rosai Dorfman Disease in a Patient with Newly Diagnosed Hodgkin Lymphoma**

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Background: Rosai Dorfman Disease (RDD, also known as sinus histiocytosis with massive lymphadenopathy) is a rare non-Langerhans cell histiocytosis, classified into nodal (classic) and extranodal disease. Most patients present with non-tender cervical lymphadenopathy, although other sites including the inguinal and axillary regions have been reported. Extranodal disease (~43% of cases) can involve any organ system. RDD is more common in children, but cases have been reported in patients in their 70s. The prognosis of nodal RDD can correlate with the number of nodal groups involved. Many cases can be managed only with observation, although surgery, chemotherapy, corticosteroids, and immunomodulators have been used in more extensive disease processes or in cases of relapse. However, there is no consensus on the duration and type of treatment due to limited data.
Case Presentation: A 38-year-old male with HIV (CD4 count 754) presented with progressively worsening dyspnea, productive cough, chest pain, multiple painless and indurated palpable lumps on the right side of his neck and right inguinal area, and unintentional 25-pound weight loss over 5 months. Physical exam showed diffuse rhonchi in the right lobe and non-tender indurated right cervical and inguinal lymphadenopathy. CT chest showed multifocal bilateral pneumonia and reactive mediastinal lymph nodes. Right cervical lymph node biopsy revealed Reed-Sternberg cells weakly positive for PAX5, CD20, CD30, CD15, MUM1, findings consistent with mixed cellularitytype Hodgkin lymphoma. Tumor cells also stained positive for EBERish, consistent with EpsteinBarr virus. No infiltration by the lymphoma was seen on bone marrow biopsy. He received one cycle of ABVD chemotherapy and coverage with vancomycin, meropenem, and fluconazole.Right inguinal node excisional biopsy was positive for CD68, BCL1 (cyclin D1), and S100, highlighting sinus histiocytes. No definitive emperipolesis was identified and clinical correlation with additional lymph node sampling was recommended if Rosai-Dorfman disease is clinically suspected.

Given his coagulopathic state and limited ambulation, he developed RLE proximal and distal DVT, necessitating IVC filter placement and anticoagulation with rivaroxaban. He also briefly required supplemental oxygen as well as furosemide due to new onset pleural effusions. Dyspnea improved and he did not require oxygen upon discharge. He was discharged on rivaroxaban, Bactrim, and instructions to follow up with his oncologist and HIV clinic.
Conclusion: Our patient did not follow up with his oncologist after discharge. Additionally, the rivaroxaban he needed has a prohibitively high cost, creating a barrier to care for many patients who need treatment for DVT. This is a complex case in a rather young patient whose disease progression will be negatively impacted by a lack of regular follow-up and lack of access to medications due to cost. Additionally, RDD is rare and disease progression is not well known, creating another barrier as this patient requires extensive follow up for monitoring.

**Chasing incessant urinary tract infections results in an intriguing case of myeloma kidney.**
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Background: Light chain cast nephropathy, formerly known as myeloma kidney, is the primary cause of renal failure in multiple myeloma (MM). At the time of presentation, about 50% of patients had kidney involvement, strongly associated with higher mortality rates.
Case presentation: A 68-year-old white female with a history of osteoarthritis on chronic NSAIDs and recurrent UTIs, proteinuria treated with long-term antibiotics, presented to the Emergency Department with the chief complaint of lightheadedness, described as episodic, accompanied by gait instability, polyuria, polydipsia, back pain, and constipation. Negative for urinary symptoms or weight loss. Medical records obtained from Primary Care Physician proven history of proteinuria, pyuria, recurrent UTIs with no significant UFC, and normal kidney function. Upon presentation unremarkable physical examination. Laboratory showed leukocytosis, anemia, and severely impaired kidney function. Further work-up such as ionized calcium, parathyroid hormone, and
vitamin D compatible with malignant hypercalcemia. Urinalysis showed bacteriuria and proteinuria. Therapy was started with isotonic fluids, calcitonin, and bisphosphonates avoided in the setting of severe kidney impairment, and nephrotoxin agents were removed. Due to high suspicion of underlying MM Bence-jones protein urine showed M spike 78.5%. To confirm the diagnosis a bone marrow biopsy was done and showed plasma cell myeloma (95% lambdarestricted plasma cells). Due to new onset kidney disease, aimed to high suspicion for nephropathy, a kidney biopsy was done showing light chain cast nephropathy, and chronic active interstitial nephritis. Renal replacement therapy was contemplated if there was no improvement. The oncologist and Nephrologist were consulted, and the patient successfully started on pulse steroids and received the first dose of bortezomib. There was a remarkable improvement in kidney function, and hypercalcemia was resolved. The patient was discharged safely to continue to be followed by the Oncologist.
Conclusions: Light chain cast nephropathy should be suspected in patients who do not carry a diagnosis of MM but have evidence of a monoclonal protein (M-protein) in the urine. More testing is warranted to evaluate MM. Clinical features include acute kidney injury, proteinuria, electrolyte abnormalities, and evidence of tubular dysfunction. This can occur as the first manifestation or develop later during myeloma. The key is found if a monoclonal protein is involved in the pathogenesis of kidney disease; in most cases, kidney biopsy is needed. The treatment consists of anti-myeloma therapy, fluid management, and, in patients with severe acute kidney injury, dialysis. In terms of prognosis, patients with significant kidney dysfunction at presentation tend to have worse outcomes than those without. Reported rates of kidney function improvement in patients with newly diagnosed myeloma treated with bortezomib-based chemotherapy range between 50-80%. This case has clinical relevance in patients with persistent proteinuria, UTI´s, risk factors, new onset of kidney impairment, these features could be masking an underlying serious and deadly pathology. Whenfacing these cases, a thorough workup is needed.

**Emergency total proctocolectomy in an uninsured Hispanic Man with Colorectal Adenocarcinoma Secondary to Familial Adenomatous Polyposis**
*Authors: Barbara Malaga-Espinoza, Diana Othon, Yilen K. Ng-Wong, Vamsikalyan Borra, Aramide Tijani, Fatimah Bello*

Background: FAP is a rare genetic disorder classically inherited in an autosomal dominant pattern, which affects about 1 in 8 300 individuals. The Hispanic population has limited data regarding the spectrum of FAP mutation and clinical manifestation, although there is significant anecdotal evidence that the prevalence might be higher, with one only known Hispanic familial cancer registry in Puerto Rico.
Case Presentation: We are reporting the case of a 25-year-old Hispanic gentleman with a strong family history of Familial Adenomatous Polyposis (FAP) and Colorectal Cancer (CRC) who presented for evaluation of abdominal pain, recurrent bloody stools, and profound weight loss. Initial Hb was 7.2 g/dL, and abdominal examination showed generalized rigidity and tenderness worse in the left lower quadrant. Colonoscopy revealed multiple large, non-bleeding polyps in the entire colon and up to the dentate line. The pathology report was positive for tubulovillous adenoma, while the surgical pathology report showed grade 2 moderately differentiated adenocarcinoma.

Immunohistochemical stains were positive for the expression of MLH1, PMS2, MSH2, and MSH6 mismatch repair proteins. The patient subsequently had laparoscopic-assisted proctocolectomy with Brooke ileostomy. His hospital course was uneventful, and he was discharged home to follow up with medical oncology, surgery, and primary care.
Conclusion: To reduce the associated healthcare costs and morbidity and mortality of cancer in general, especially those with associated strong risk factors such as FAP and CRC, early genetic counseling, timely screening, appropriate risk-reducing medical and surgical interventions, and regular lifetime follow-up of index cases are crucial. It is also imperative to promote health literacy, especially in communities with low socio-economic status who are often at a disadvantage. Lastly, there is a need for continued research on FAP, especially in minority populations, with increased promotion and use of familial cancer registries to reduce the overall burden.
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**Little bit about Liddle: Big improvement in blood pressure**
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Background: Liddle syndrome is a rare autosomal dominant disorder associated with abnormalities in function of the collecting tubule sodium channel, also called the epithelial sodium channel(ENaC). ENaC function is increased in the Liddle's syndrome leading to the manifestations of mineralocorticoid excess symptoms, such as hypertension, hypokalemia, and metabolic alkalosis. The diagnosis is made when these features are seen with low renin and low plasma or urinary aldosterone. Genetic testing is not required for the diagnosis of Liddle syndrome. Liddle's syndrome has an excellent response to amiloride.
Case Presentation: A 56-year-old gentleman, with longstanding uncontrolled hypertension was seen for functional constipation and lower extremity weakness. He had no other symptoms. His initial blood pressure was 210/122. His physical exam was remarkable for a Grade 3/6 decrescendo diastolic murmur. He had normal strength and reflexes. His potassium was 2.7mEq/L bicarbonate 33.3 mEq/L and Ph 7.49. EKG shows QT prolongation and patient was tachycardic. The patient was admitted to the hospital for treatment of his blood pressure and potassium replacement. Despite treatment, the potassium remained low and blood pressure uncontrolled. The patient described a 17-year history of poorly controlled blood pressure. He had seen multiple physicians for blood pressure management and had taken numerous medications. An evaluation for secondary causes of hypertension was initiated. His plasma renin was low at <0.167 ng/ml/hr, and aldosterone was low at 11ng/ml. The 24-hour urine metanephrine was normal. He started on amiloride 5 mg with improvement of blood pressure to 150/70 and potassium of 3.4 meq Therefore, the patient was diagnosed with Liddle syndrome at age of 56-year-old.
Conclusion: This case report demonstrates the important concept of reviewing previous treatment and wondering why the patient has a poorly controlled medical problem. Secondary causes of hypertension are less common but often improve with specific treatment In this patient, thinking about why he had resistant hypertension, hypokalemia and metabolic alkalosis led to further work-up. The low renin and low aldosterone added to the expanded history led to better blood pressure control by selecting the correct treatment. Thus, A little bit of thinking about Liddle's can lead to big improvement in blood pressure.

**Too Yellow: An Idiopathic Case Of Autoimmune Hemolytic Anemia**
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*Co-Authors: Donnel Dockery-Joseph,MD, Kashif Ali, MD, Taha Al Hassan,DDS, Aramide Tijani,MD, Sri Harsha Koneru,MD, Elimar Gonzalez Morales,MD, Eunbee Cho,MD*

Background: Autoimmune hemolytic anemia is caused by autoantibodies that react with self red blood cells and cause them to be destroyed. Warm AIHA, due to antibodies that are active at body temperature, is the most common type of the AIHA. It is mostly caused by the underlying disease like infections, autoimmune disorders, lymphoproliferative disorders, immunodeficiency or physiologic state like pregnancy. Rarely it happens that there is no underlying cause. Therefore, here we present a case of idiopathic autoimmune hemolytic anemia without any underlying cause.

Case Discussion: A 77 year old lady with known history of the diabetes mellitus type 2, essential hypertension and dyslipidemia presented to the emergency department complaining of generalized body weakness and recently started noticing yellowish-red urine and yellowish discoloration of the skin from last 1 week. On asking her further she denies any recent change in medications which were metformin and losartan, no recent sick contacts or any infections, any weight loss orchest pain or abdominal pain, near syncope or loss of consciousness, any change in bladder or bowel habits other than the change in the color of urine. Denies any positive family history or prior blood transfusions. On admission her labs showed Hb of 5 g/dl, total bilirubin of 8.9 mg/dl, conjugated bilirubin was 0.43 mg/dl, LDH was 1572 u/L, MCV was 107.5 fl, haptoglobin less than 10 mg/dl, total reticulocyte count was 20.3 %. Urine only showed few RBC and 4+ blood. Her significant vitals were only tachycardia with the heart rate ranging from 110-130s. Further investigations were done including the CT abdomen pelvis with oral and IV contrast which was negative for inflammation or any tumor and CXR and EKG were not significant. Blood cultures and urine culture also showed no growth after 48 hours. Other infections like Hepatitis and HIV were ruled out.Direct antiglobulin test(Coombs test) came out positive. Her physical exam was only positive for sinus tachycardia and severe yellowish discoloration of skin and conjunctiva. Therefore, everything looked consistent with autoimmune hemolytic anemia on basis of the positive DAT test, high LDH, low haptoglobin, low HB, high indirect bilirubin and the peripheral smear showed polychromasia and nucleated RBC. The patient was started on the prednisone 1mg/kg/day and emergency release of O neg blood was done for transfusion to keep Hb above 7 g/dl. She was eventually started on the rituximab 375 mg per square meter once weekly for 4 weeks. She felt better after the prednisone and was discharged home with follow up with the Hematologist for rituximab.
Conclusion: The main learning point is that even without underlying factors the patient can have the autoimmune hemolytic anemia. The main treatment for this kind of scenario is oral or intravenous glucocorticoids and rapid blood transfusion. The second line therapy is Rituximab and other immunosuppressive agents and chemotherapy agents.

**What time is it? It is 8 and 1/2 time. A rare case about right medial pontine stroke, right INO, right facial colliculus ischemic stroke consistent with eight-and-a-half syndrome.**

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Background: Eight-and-a-half syndrome (EHS) is a rare neuro-ophthalmologic condition characterized by conjugated horizontal gaze palsy, internuclear ophthalmoplegia (INO), and ipsilateral fascicular seventh cranial nerve palsy. Clinical features are attributed to the involvement of the lower pontine, including the abducens nucleus, ipsilateral medial longitudinal fasciculus (MLF), and adjacent facial colliculus. Etiology includes vascular diseases such as pontine infarction or ischemia, demyelinating conditions at the level of the pons, and rarely space-occupying lesions.
Case report: A 49-year-old Hispanic white male with a past medical history of essential hypertension, uncontrolled diabetes mellitus type II and family history of strokes in a first-degree relatives came to the emergency department with the chief complaint of right-sided facial droop and dysarthria for three days, associated with double vision for two weeks. No other symptoms reported. Upon physical examination, vital signs were stable, alert, oriented to person, time, and place. Mild dysarthria was noticed. Cranial nerves (CN): II: Pupils 4 mm in size, equal, round, and reactive to light. Visual fields binocular diplopia. CN III, IV, VI: Primary gaze disconjugate, bilateral exotropia. Upgaze and downgaze intact, lateral gaze and medial gaze were incomplete OD, medial gaze incomplete OS with full lateral gaze and nystagmus on lateral gaze. This is consistent with a right internuclear ophthalmoplegia (INO) and a partial left INO. CN V: Facial sensation symmetrically preserved. CN VII: Right facial droop at rest, there was incomplete heavy facial activation that extended to the forehead; indicative of lower motor neuron involvement. CN VIII: Preserved. CN IX, X: Palate and uvula asymmetric, no dysphagia. The remaining neurological examination was unremarkable. Blood work-up, EKG, echocardiogram, CT head unremarkable. The neurologist was consulted and recommended further imagining studies such as brain MRI (Magnetic Resonance Imaging) showed subacute ischemic lesion at the level of the right pons and facial colliculus, also involving the medial longitudinal fasciculus. CT angiogram head and neck remarkable for atherosclerotic disease, with decreased caliber of the basilar at the level of the pons. In the setting of family history of strokes and at an early age, autoimmune workup was ordered with no abnormalities found. Therapy started with dual antiplatelet agents, high dose statin, glucose, and blood pressure control. The patient was discharged home safely with Neurologist follow-up and physical therapy.
Conclusion: Eight-and-a-half syndrome should be suspected in a patient with features such as facial palsy associated with INO, however, more testing is warranted to evaluate the syndrome. The key is identifying the disorder since in most cases pontine infarction or ischemia is the primary cause. Pontine stroke mortality rate ranges from 30%-50%. A prompt diagnosis determines the prognosis and depends on the progression of the underlying disease entity. Treatment involves managing the underlying cause and addressing associated symptoms. Rehabilitation and physical therapy play a crucial role in recovery. Cases involving infarcts, recovery is contingent upon the affected area's ability to recover. Isolated cases presenting with diplopia and facial palsy have a good Prognosis.

# STAFF CATEGORY

**CEACAM7 emerges as a promising early detection biomarker in pancreatic cancer.**

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Background: According to the key statistics from the American Cancer Society in 2021, Pancreatic Cancer (PanCa) impacts approximately 60,430 individuals annually in the U.S., affecting 31,950 men and 28,480 women. Diagnosis and treatment of PanCa pose significant challenges. It stands as the fourth leading cause of cancer-related deaths, boasting a mere 9% 5-year survival rate and an overall grim prognosis. Adenocarcinomas, particularly PDAC, constitute the majority (around 85-90%) of PanCa cases, contributing to its highly aggressive nature and low survival rates. The absence of an early tumor-specific biomarker for PDAC underscores the urgent need for a novel strategy to enhance the limited diagnostic options available for PanCa. Recognizing this critical situation, our research group has identified a promising oncogenic protein, Carcinoembryonic antigen-related cell adhesion molecule 7 (CEACAM7). Our studies indicate elevated CEACAM7 expression in pancreatic ductal adenocarcinoma (PDAC) tumors and its correlation with patient survival.
Methodology: The research commenced with bioinformatics screening, involving the assessment of CEACAM7 expression across various cancer types, overall survival analysis, correlation with genes, association analysis, spot prediction, and evaluation of immune cell infiltration capability in the context of pancreatic ductal adenocarcinoma (PDAC). Subsequently, guided by the insights gained from the bioinformatics approach, molecular biology techniques were employed to meticulously examine the progressive cell line panel of PDAC (HPNE, HPAF-2, SU86.86/BxPc3, and Panc-1) in terms of both mRNA and protein expression levels of CEACAM7. Confocal microscopy was utilized to recognize the intensity and localization of CEACAM7 protein expression in various cell lines. Additionally, immunohistochemistry (IHC) analysis was conducted to identify protein expressions in human tissue microarray (TMA) cores, along with relevant location and grading data.
Results: Bioinformatic results clearly cited the relevance of CEACAM7 as a potential prognostic biomarker of PDAC, followed by molecular biology approaches revealed the positioning of CEACAM7 as an early detection biomarker.
Conclusion: Our observations clearly cited that CEACAM7 can be investigated as an early detection biomarker for PDAC.
Keywords: Pancreatic cancer, PDAC, CEACAM7, Early detection biomarker, Tumor grading & Bioinformatic

**"Circular Gene Mapping of Identified AMR Genes in Multi-Drug Resistant *Escherichia coli* Isolated from Potable Water"**

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ABSTRACT:
Background: *Escherichia coli* is a Gram-negative & facultative anaerobe bacterium ubiquitously found in all environments. It is a waterborne and foodborne pathogen associated with many diarrhoea and GI tract diseases inclusive of UTIs in humans. The rise in the number of bacteria resistant to antimicrobial drugs has a major impact on disease control efforts. Antibiotic-resistant bacteria can be found in a variety of water sources. As a result, the objective of the current study is to construct a circular AMR gene map based on the assessment of the antibiotic susceptibility profiling of *E. coli* isolated from twenty various potable water sources.
Methodology: *Escherichia coli* was isolated by using IS 5887 (Part-1) 1976. The antibiotic susceptibility was assessed using the Agar Well Diffusion Assay against ten therapeutically significant antibiotics. AMR genes were identified by using Comprehensive Antibiotic Resistance Database (CARD). The whole genome sequencing was also used to construct circular AMR gene map using Proksee tool.
Results: Our research clearly shows that the *E. coli* isolates are resistant to antibiotics. The most resistant drugs for isolated *E. coli*. were found to be azithromycin, ampicillin, and metronidazole, to which every isolate showed 100% resistance. Out of 20 water sources, 13 *E. coli* isolates showed 100% susceptibility to ciprofloxacin, doxycycline, and meropenem and 100% resistance to azithromycin, ampicillin, and metronidazole. Tetracycline and norfloxacin showed intermediate susceptibility in 78% of *Escherichia coli* species. 15% of the isolates in the research were sensitive to cefixime, and 38% to co-trimoxazole. A circular gene map was created using identified 59 AMR genes, including those that encode antibiotic efflux pumps for small multidrug resistance (SMR), ATP binding cassette (ABC) antibiotic efflux pumps, and resistant nodulation cell division (RND) antibiotic efflux pumps etc.

Conclusion: Antibiotic overuse and misuse have caused microorganisms to become less susceptible and more resistant. As a result, treating bacterial and other infections with antibiotics is no longer as effective. The present investigation assessed the antibiotic susceptibility profiles of ten commonly given antibiotics against isolates of *Escherichia coli spp*. Among the other medications, ciprofloxacin and meropenem were found to be 100% vulnerable to *Escherichia coli spp*. Unlike meropenem and ciprofloxacin, which were the most promising antibiotics, metronidazole, ampicillin, and azithromycin were determined to be the most failed antibiotics since they were ineffective against all discovered isolated strains.
Keywords: *E. coli*, Antibiotic resistance, Antibiotic susceptibility, AMR gene, multi drug resistant bacteria, circular gene

**Human Papilloma Viruses Infection and Pre-Malignant Lesions in Women on the Texas Mexico Border.**
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Background: In United States, 24-40 million women have had at least one Human Papillomavirus (HPV) infection. While HPV infection is responsible for over 93% of cervical cancer cases worldwide, it is relatively uncommon in US given screening programs and the introduction of the Papanicolaou test. In Texas, where cervical screening and uptake of the HPV vaccine is lower, the incidence of cervical cancer is 12 cases per 100,000 women. Along the border with Mexico the incidence of cervical cancer rises to 16 cases per 100,000 women, mirroring Mexico's public health problem: cervical cancer is the leading cause of death in women. Despite successful screening programs and declining incidence of cervical cancer in the US and other high-income countries, HPV infection and cervical cancer are still very formidable public health issues in the lower income countries such as Mexico and the US-Mexico border.
Methods: Women were recruited from a specialty clinic in Valle Hermoso, Tamaulipas, a border city in Mexico. Women were eligible to participate in the study if were over the age of 18, reported being sexually active, were not pregnant and had never had a hysterectomy or cervical cancer (n=212). Each participant was given a self-administered questionnaire to be completed prior to having the conventional Pap smear examination. The questionnaire obtained demographic, lifestyle and reproductive histories for participants and their partners. History of diabetes mellitus, tobacco, alcohol use. Examinations were performed using the standard method by placing the woman on an examination table in the lithotomy position.
Results: Participants median age was 38 years old (91%), had greater than or equal to a high school education (54%), were married (84%), reported only having had only one sexual partner (93%), reported using at least one contraceptive method (96%), and approximately 14% reported having had a history of a sexually transmitted infection. For those women whose specimens were available for analyses (n=200), sixty-four percent were found to be HPV infected and 17% were found to have multiple types of HPV. Tested positive for an HPV infection, 39% were infected with high risk types 16 (n=45) and 18 (n=33). When including other HPV high-risk types associated with cancer, the percentage of women infected with a high-risk HPV jumped to 66%.
Conclusion: These data raise important issues about the needs for cervical cancer screening and HPV vaccination Mexican women; particularly those living along the US-Mexico border. Data collected during the same time period among women working in a nearby maquiladora found that only 7% of employed woman were infected with a high-risk HPV type despite similar demographic, lifestyle and reproductive histories. Another area of concern is that high-risk HPV infections may be just as high among Hispanic women living along the US-Mexico border. More studies are needed to document the burden of HPV among these underserved populations.

# MEDICAL STUDENT CATEGORY

**Title: A Rare Encounter: Extracranial Meningioma Mimicking Musculoskeletal Neoplasms**

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Background: Meningiomas are the most common primary brain tumor in adults. While commonly encountered intracranially, 2% manifest extracranially. Although usually benign, 10% of meningiomas can become malignant. Despite their relatively high incidence, they are often difficult to diagnose due to long asymptomatic periods, often diagnosed after mass effect symptoms occur. This case explores the intricacies of diagnosing and managing an extracranial meningioma that mimicked musculoskeletal neoplasms.
Case Presentation: A 70-year-old female, with a history of hypertension and dyslipidemia, presented with diplopia, blurry vision, and intermittent right orbital pain. A recent CT had identified a slow-growing right temporal mass, yet she remained asymptomatic, and she did not have any additional follow-up due to financial burden. She denied any personal or immediate family history of malignancy or extensive radiation exposure. Vitals and labs were unremarkable. Physical examination revealed a fixed, non-tender soft tissue mass extending from the right frontal to the temporal region, causing significant right eye proptosis. Imaging studies confirmed a large soft tissue mass in the right maxillofacial area with adjacent bone destruction, infiltrating nearby musculature. A multidisciplinary team, including neurosurgery, otolaryngology, and hematology/oncology, collaborated for surgical planning and outpatient follow-up for treatment. A right temporalis biopsy disclosed an extracranial meningioma involving skeletal muscle bundles. Immunohistochemical stains showed tumor cells positive for EMA and SSTR2a and negative for pancytokeratin, PR, GFAP, SOX10, desmin, SMA, CD34, synaptophysin, chromogranin, and GATA3. No features of atypical or malignancy were reported. Despite negative metastatic workup, the extensive osteolytic and skeletal muscle invasion presented challenges. The patient was discharged for follow-up with oncology and otolaryngology, emphasizing the need for individualized treatment.
Conclusions: This case demonstrates a rare but aggressive extracranial meningioma in a patient who did not undergo evaluation and treatment early on due to not being referred to the appropriate specialists as well as the broad differential associated with her initial symptoms. This patient did not undergo an MRI early on due to the large financial barrier associated with obtaining this imaging, likely contributing to the increased invasion of the tumor when she presented to the hospital. She requires extensive care and follow-up from a multidisciplinary team including oncologists, otolaryngologists, radiation oncologists, and neurosurgery.
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**Baclofen Induced excessive perspiration; a case report**

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Abstract
Background: This case merits presentation due to the unexpected adverse effect of Baclofen, a long-established medication with a well-documented safety profile. The novelty lies in the rare manifestation of an excessive perspiration condition that is not commonly associated with Baclofen use. While the drug's adverse effects typically include sedation, confusion, and muscle weakness, excessive perspiration is not a frequently reported outcome. Understanding and documenting this adverse reaction is crucial for healthcare professionals, as it sheds light on Baclofen's lesser-known side effect profile. Exploring this case enhances our understanding of its pharmacological impact and informs future patient care and drug development endeavors.

Case Presentation: This case involves a 63-year-old woman with a complex medical history, including diabetes mellitus, essential hypertension, Hashimoto's thyroiditis, and chronic back pain caused by a motor vehicle accident. She has two spinal stimulators and has utilized long-term muscle relaxant use. The patient presented with a complaint of excessive sweating that worsened over the period of a year, describing a constant state of being drenched. Initial assessment attributed symptoms to the postmenopausal state, leading to the prescription of Venlafaxine. However, subsequent follow-ups showed persistent sweating despite dosage adjustments. In January 2023, extensive testing revealed mildly elevated cortisol, low serotonin, and normal thyroid function. Despite discontinuing thyroid medications, the sweating persisted, prompting the discontinuation of Baclofen, a previously well-tolerated medication. The patient reported a resolution of symptoms in February 2023 after the withdrawal of Baclofen. Notably, the excessive sweating linked to Baclofen was unprecedented after five years of use. This case emphasizes the importance of considering medication-related side effects, even with established drugs, and highlights the challenges in diagnosing and managing unusual adverse reactions.
Conclusions: This case report illustrates the clinical impact of Baclofen-induced excessive sweating, showcasing the infrequency of this adverse effect and underscoring its potential underreporting in existing literature. By presenting a diagnostic challenge and the subsequent resolution upon Baclofen withdrawal, the case emphasizes the need for increased awareness among healthcare professionals regarding unusual side effects associated with commonly prescribed medications. The study's significance lies in filling the knowledge gap regarding the incidence and prevalence of excessive sweating linked to Baclofen, a critical step toward understanding the full spectrum of its effects. This research sheds light on the profound impact of excessive sweating on patients' lives and advocates for increased awareness, accessibility to treatment, destigmatization, and further investigation into the prevalence of medication-induced sweating to improve patient outcomes and quality of life.

**Birth defect trends within Texas Public Health Region 11, 2000-2019: an analysis of Texas Department of State Health Services public data.**

*Miguel Lopez*

South Texas is a predominantly Hispanic region with high rates of chronic illness, poor healthcare access, and a history of birth defect clusters. Between 1986 and 1991, 47 cases of anencephaly in Cameron County were linked to elevated fumonisins in the region's corn-based diet, prompting a series of ongoing public health efforts. This paper aims to identify changes in prevalence for CNS defects, in addition to cardiac, circulatory, gastrointestinal, and genitourinary defects in South Texas within the last two decades. Public data on 20 birth defects from the Texas Department of State HealthServices were obtained for decades 2000-2010 and 2010-2019 in Texas Public Health Region 11 and the remaining regions of Texas. We report that Region 11 saw larger birth defect prevalences compared to the remainder of Texas in both decades studied. When looking at single regions between decades, there was an increase in the prevalences of microcephaly, ASD, pulmonary valve atresia or stenosis, PDA, and hypospadias within Region 11 in 2010-2019; the prevalences of these defects also increased in the remaining regions of Texas in 2010-2019, with the addition of 8 more: hydrocephaly, double outlet right ventricle, tetralogy of Fallot, VSD, tricuspid valve atresia or stenosis, coarctation of the aorta, stenosis or atresia of the small intestine, and renal agenesis/dysgenesis. Pyloric stenosis alone saw a significant decrease in prevalence in 2010-2019 for both regions in this study. Furthermore, it was found that the prevalences of anencephaly and spina bifida without anencephaly were unchanged in both regions.

**Comparative Effectiveness of Endovascular vs Surgical Arteriovenous Fistulas: A Preliminary Analysis**

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Abstract
Purpose: The objective of this study was to compare the efficacy and safety of endovascular arteriovenous fistula (endoAVF) creation versus open surgical AVF (openAVF) for hemodialysis access across centers participating in the Dialysis Access Learning and Innovation Collaborative (DiAL-In Collaborative) in the United States. In this preliminary analysis, we report the baseline characteristics and clinical profile of patients enrolled at a single center.
Materials and Methods: A retrospective cohort study was conducted in chronic kidney disease patients who underwent creation of an upper arm autogenous vascular access for hemodialysis (2018-2022) at the Brigham and Women's Hospital in Boston, MA. Data were gathered from the Vascular Quality Initiative dataset and supplemented with chart adjudication. All patients were followed for a minimum of one year with a maximum follow-up until July 2023.
Results: A total of 145 patients were enrolled (13 endoAVF and 132 openAVF). Overall, 67% of patients were hemodialysis dependent and 2% were peritoneal dialysis dependent at the time of fistula creation. Patients with an endoAVF were older (75 vs. 67 years; p= 0.073), and more likely to be male (62% vs. 48%; p= 0.047 ). The openAVF cohort had a higher proportion of patients with medical comorbidities such as congestive heart failure (44% vs. 31%; p= 0.057), history of percutaneous coronary intervention (19% vs. 8%; p= 0.022), and diabetes (58% vs. 46%; p=.089).
Conclusions: In this single-center preliminary analysis, we found differences in the baseline demographic and clinical profile of patients undergoing endoAVF and openAVF for hemodialysis. A thoughtful analytical approach will need to be employed to account for these important baseline differences to compare outcomes of endoAVF versus openAVF in the multicenter DiAL-In Collaborative.

***Inflammatory Breast Carcinoma in the Rio Grande Valley: A Case Report***
*Miguel Lopez*

Inflammatory breast carcinoma (IBC) is a rare and aggressive subtype of invasive breast cancer found in as small percentage of patients in the United States. 1 The Rio Grande Valley is a region of Texas with pockets of low socioeconomic status and increased rates of obesity, a large risk factor for IBC. 2,3 The cutaneous presentation of IBC is a tender, erythematous patch or plaque that surrounds hair follicles (referred to as peau d'orange) and is frequently edematous. Diagnosis requires a high clinical index of suspicion supported by a thorough patient history and pathology consistent with invasive carcinoma. 4 This case study discusses a 59 year old overweight female patient with a sudden eruption of a rash that was clinically suspicious for inflammatory breast carcinoma and a biopsy which demonstrated breast adenocarcinoma confined to intravascular spaces. While rare, IBC has a high mortality rate and a higher likelihood of metastasis if not detected and treated in early stages of a patient's presentation.
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**The Increasing Prevalence of Cleft Lip with or without Cleft Palate in the Rio Grande Valley of Texas**
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Background: Orofacial clefts are a subset of birth defects that include cleft lip with or without cleft palate (CLP) and cleft palate alone (CP). The treatment for orofacial clefts is surgical repair, ideally within the first six months of life. Their impacts on patients and families are various and substantial. Babies with orofacial clefts can have trouble with breastfeeding, speech, recurrent
ear infections, and hearing loss as they age. Additionally, there is a significant economic burden, with the average repair costing nearly $20,000, not including the costs of medical devices, postoperative care, and rehabilitation. Additionally, children with orofacial clefts face a high incidence of teasing and ostracization by peers and even family. These issues can be especially difficult in medically underserved and socioeconomically disadvantaged populations such as the Rio Grande Valley of Texas (RGV). This paper explores trends in the prevalence of orofacial clefts in the RGV.
Methods: Aggregated data for orofacial clefts from 1997-2018 was acquired from the Texas Department of State Health Services Birth Defects Epidemiology and Surveillance Branch. All birth outcomes were included, but only definite diagnoses of orofacial clefts are included in this study. No distinction between syndromic and non-syndromic orofacial clefts was made. Populations studied included children born to Hispanic mothers in the RGV (Cameron, Hidalgo, Starr, and Willacy counties) and non-border counties of Texas. Statistical analysis was conducted through two-tailed z-score analysis.
Results: Overall, the prevalence of CP between 1997-2018 was lower among children born to Hispanic mothers in the RGV than in non-border counties, but not significantly (p=0.059). The prevalence of CP in the RGV was higher between 2008 and 2018 than in 1997 and 2007 but not significantly (p=-.105). Non-Border counties saw a similar non-statistically significant increase in CP prevalence (p=.177). Overall, the prevalence of CLP between 1997-2008 was higher among children born to Hispanic mothers in the RGV than in non-border counties, but not significantly (p=0.26). For the years 1997-2007, the prevalence of CLP was lower, but not significantly, in the RGV than in nonborder counties (p=.258). For 2008-2018, the prevalence of CLP was significantly higher than in
non-border counties (p=.007). The RGV saw a non-statistically significant increase in the prevalence of CLP between the decades of 1997-2007 and 2008-2018 (p=0.063). Contrarily, the non-border counties of Texas saw a non-statistically significant decrease in the prevalence of CLP between the decades of 1997-2007 and 2008-2018 (p=0.155).
Conclusion: From 2008-2018, children born to Hispanic mothers in the RGV had a significantly higher prevalence of CLP than those born to Hispanic mothers in non-border counties in Texas. Additionally, the RGV saw an increase in the prevalence of CLP, while non-border counties saw a decrease. This is concerning, given that many parts of the RGV are designated as healthcare shortage areas. Travel to other cities of Texas for care can be difficult or impossible for undocumented and disadvantaged residents of the RGV. Investment is required to evaluate local reporting measures, meet healthcare demands, and increase affordable care for these conditions among RGV Residents

# GRADUATE STUDENT CATEGORY

**Exploring Xenophagy: How Colon Epithelial Cells Combat Intracellular Bacterial Threats**

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Background: The immune system utilizes macrophages and other specialized cells to counteract invading pathogens; however, the mechanisms of defense utilized by non-immune cells remain a subject of interest for research. The gastrointestinal tract, specifically the colon, is a location where host cells and pathogenic microorganisms interact frequently. Methods: This study uses Listeria innocua, as a model system to investigate how colon epithelial cells fight these invading bacteria, specifically focusing on xenophagy as a potential protective mechanism. By using immunocytochemistry, confocal microscopy and western blotting, we assessed the potential impact of L. innocua invasion on colon epithelial cells. Results: Our results indicate that L. innocua invasion stimulates cellular xenophagy machinery, which increases autophagosome formation, a double-membraned structure that works to capture and contain intracellular bacteria. This result demonstrates that colon epithelial cells produce autophagosomes responding to bacterial invasion. Our research also shows that L. innocuainvasion increases lysosome formation in colon epithelial cells. We also found that lysosomal calcium Ion channels play a role in this xenophagy-mediated bacterial clearance.
While activation of the channel potentiated, inhibition of the channel suppressed the activity of xenophagy machinery. Furthermore, through cell infection assay, we found that the colon epithelial cells could clear off more L. innocua when the calcium channel was activated. This finding suggests that colon epithelial cells facilitate the lysosomal-mediated calcium dependent degradation and clearance of intracellular L. innocua. Conclusions: In summary, our study showed that non-immune colon epithelial cells use xenophagy machinery to fight bacterial invasion. This study improves our understanding of host-pathogen interactions in the gastrointestinal tract and may help design novel therapeutic strategies to combat bacterial infections.

**Improving Health and Sustainability in Construction through the Use of AI-Based Models for Prediction and Mitigation of environmental cancer risks**

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Introduction: Sustainable construction practices face significant challenges in eliminating toxic building materials, driven by a lack of awareness among professionals and economic influences shaping regulatory decisions. This study proposes a solution to address these challenges through the implementation of artificial intelligence (AI) models capable of analyzing vast datasets to predict material toxicity, with a particular focus on identifying carcinogenic materials. The objective is to provide real-time insights to architects and civil engineers, fostering environments free from harmful building materials and preventing potential health hazards, including cancer. The presence of carcinogenic materials in construction processes poses severe health risks. Asbestos, a commonly used insulation material, is notorious for causing lung cancer. Silica dust, generated during the manipulation of materials like concrete and stone, is associated with lung cancer and respiratory diseases. Benzene, found in certain adhesives, and formaldehyde, present in paints and coatings, are volatile organic compounds classified as known human carcinogens, with links to various cancers.
Methods: To comprehensively understand toxic building materials, this paper utilizes existing data to train AI models and create algorithms for material toxicity prediction. These models leverage advanced algorithms, trained on existing datasets, to analyze extensive information. We particularly focus on volatile organic compounds from paints, impregnating agents, fire-induced toxicity, asbestos, radioactive materials, lead plumbing, silica dust, benzene, and formaldehyde. A comparative analysis is employed to determine the most effective model for predicting material toxicity and identifying carcinogenic elements.
Results: Our AI-driven models not only predict carcinogenic emissions and environmental cancer risk but also identify alternative substances that align with safety standards and economic considerations. This dual functionality aims to guide professionals in making informed decisions during the construction process, with a heightened emphasis on cancer prevention. The integration of AI-driven approaches will mark a paradigm shift in sustainable construction practices, facilitating informed decision-making and empowering professionals to effectively eliminate toxic building materials, thereby preventing cancer risks.
Conclusion: In conclusion, the implementation of AI-driven models for prediction and recommendation of alternate strategies, presents a transformative solution to the challenges faced in sustainable construction. This study underscores the significance of informed decision-making, promoting the elimination of carcinogenic building materials and fostering a safer and more sustainable construction industry. This study not only contributes to creating environments that prioritize safety and sustainability but also plays a pivotal role in preventing potential health risks, including cancer. This marks a crucial advancement in the field of sustainable construction practices with a profound impact on public health.

**Optimization and characterization of one-step multi-functionalization of virus-like particles for multimodality nanoplatforms**

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Background: Multimodality platforms integrating targeting, therapeutic and diagnostic modalities are gaining attention in the area of smart nanomedicines for cancers. However, their multi-step synthesis strategies are the major bottleneck for large-scale production with high reproducibility. Isocyanide-based multi-component reactions (IMCRs) have become popular in modern chemistry and have offered effective access to highly diverse and complex final products in a single step. Herein, isocyanide-based 4-component (4CRs) UGI reaction is optimized on virus-like particles (VLPs) to develop multimodal nanoplatforms more efficiently.
Methods: In this work, biocatalytic P22CYP nanoreactors were multifunctionalized with: (i) enzyme Glucose Oxidase (GOx) to obtain a cascade enzymatic activity for prodrug transformation, (ii) a photosensitizer, Indocyanine green (ICG) for NIR activated photodynamic and photothermal therapy, and (iii) a targeting ligand, 2-Deoxy-d-glucose (2DG). The VLPs were exposed to UGI reaction at room temperature and variable ionic strength, reagent concentrations, and times. They were later characterized through enzymatic activity, photosensitizer quantification and electron microscopy.
Results: There was no significant difference in the extent of surface functionalization of GOx and ICG when the concentration of UGI reagents, *t*-butylisocyanide and formaldehyde were varied. TEM analysis revealed the preserved integrity of VLP under all reaction conditions suggesting no significant affect of UGI reaction on proteinaceous nanostructure. The functionalized ICG generated reactive oxygen species and raised the temperature of medium up to 3.6°C after excitation at 808 nm. The synergy of photothermal and photodynamic property on the glucose mediated prodrug transformation was also evaluated by HPLC and displayed higher transformation rates. The functionalization of 2-DG could not be characterized in this work, therefore, future work will focus on the identification of targeting ability of nanoreactors by cell internalization assay.
Conclusions: Our preliminary analysis shows that multimodal P22CYP-GOx-IcG-2DG could be a potential candidate for quadruple combination therapy and NIR imaging of cancer. These results highlight the potential of 4CR UGI reaction for one-step multifunctionalization of protein based nanoparticles under ambient conditions for the development of cancer therapy nanoplatforms.

**Targeting mycobacterial efflux system for combating anti-microbial resistance**

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Background: The drug resistance in the microbes is a serious concern in medicine. Along with intrinsic factors, extrinsic factors like unprescribed usage of drugs are the contributing factors. The drug tolerance has led to the emergence of superbugs. Mycobacterial species utilize an array of multidrug efflux mechanisms linked to intrinsic and acquired antibiotic resistance. Understanding molecular mechanisms regulating efflux could reveal new therapeutic targets and strategies. Our study is aimed to target regulators of efflux Mycobacterial transporter.
Methods: Using the reference mycobacterial strain, antibiotic sensitivity was first profiled by minimal inhibitory concentration assays across a panel of antimicrobials, followed by mutagenesis analyses of transporter-deficient mutants. To uncover regulators, real-time transcriptional induction in the presence of selected antimicrobials is being done.
Results: Measurements of antibiotic susceptibility in mycobacterial strains are anticipated to demonstrate reduced drug tolerance. There are dose-dependent associations of transcriptional activation of efflux genes paving ways for elucidating mechanisms governing anti-microbial efflux. This work would provide candidate targets to block resistance emergence strains. Conclusions: Identifying and targeting regulators of mycobacterial efflux systems may enable novel therapeutic approaches countering antimicrobial resistance, with wider applicability across nosocomial and community-acquired infections. It leads to drug development strategies against mycobacterial infections.
Keywords: Antimicrobial Resistance, Mycobacterium, Stress Tolerance, Drug Development

**The role of a cancer testis-antigen in regulating tumor growth and oncogenic pathways in triple-negative breast cancer**

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Breast cancer is the second leading cause of cancer-related deaths in women, after lung cancer. Unfortunately, it is the primary cause of death among Hispanic women. Approximately 10-20% of breast cancers are classified as basal (triple-negative) subtypes, which are challenging to treat due to the absence of specific targets.

Triple-negative breast cancer is a highly aggressive and lethal type of tumor, particularly among Hispanic women, with a poor prognosis compared to other breast cancer subtypes. Therefore, it is crucial to identify new molecules with therapeutic and prognostic values to treat this evolving disease. Multiple studies have demonstrated that X-linked genes are associated with cancer. Although these genes are tightly regulated and expressed only in immune-privileged organs, many become abnormally expressed in tumors.

Our genomic analysis has revealed that newly annotated X-linked genes previously thought to be gene deserts in the human genome are highly antigenic and cancer testis-antigens (CT genes). However, little information exists about their expression and role in triple-negative breast cancer. To fill this gap, we have used an integrated genomic approach to identify testis-tissue-specific genes differentially expressed in this type of breast cancer. Additionally, we have discovered a highly immunogenic cancer-testis gene using cutting-edge technology-driven (Global Run-On Sequencing) gene expression analysis. With cell- , mouse-based, and genomic approaches, we have mechanistically found that this CT gene modulates gene expression, immune response, and tumor growth in triple-negative breast cancer. This finding can potentially revolutionize the diagnosis and treatment of this type of breast cancer.

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**The role of divergent noncoding gene in triple-negative breast Cancer**

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Breast cancer is the second leading cause of cancer mortality in women after lung cancer. Breast cancer is grouped into different molecular subtypes; triple-negative breast cancer (TNBC) accounts for 20%. TNBC is a highly aggressive histologic subtype; the lack of unique therapeutic targets makes it harder to treat. Recent studies have implicated genes involved in cancers that are transcribed using a process known as divergent transcription. The expression of these genes is tightly regulated; however, many escape regulation and become aberrantly expressed in tumors. Interestingly, our genomic analysis suggests that several of these genes encode noncoding transcripts. Analysis of transcriptome from different tissue types, including cancer and normal, revealed that many are upregulated in various cancers. Selected divergent transcript, such as lncRNA67, has been completely characterized (transcription start and stop site, 5’ cap, polyA tail, and exon/intron structure) and cloned. In functional assays, overexpression of lncRNA67 regulates gene expression, cell proliferation, and tumor growth. Our molecular analyses indicate that lncRNA67 plays a vital role in triple-negative breast cancer biology. Collectively, our results suggest that divergent transcripts are an integral component of cancer biology and present a new repertoire of diagnostic and potential therapeutic targets.

S.S.G. is supported by grants from a) The Cancer Prevention and Research Institute of Texas (CPRIT; RR170020); b) Lizanell and Colbert Coldwell foundation; c) The Edward N. and Margaret G. Marsh Foundation; d) The American Cancer Society

# UNDERGRADUATE STUDENT CATEGORY

**Exploring Neuroplasticity Changes in Neurotoxin-induced Parkinson's Disease: A Preliminary Analysis using Transcranial Magnetic Stimulation**

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Background: Parkinson's disease (PD) is a neurodegenerative condition that affects movement, cognition, gait, and significantly impacts one's quality of life. Studies have suggested that neurotoxin pre-exposure is related to PD pathology and progressive motor/non-motor deficits, though it remains unclear how neurotoxin exposure affects neuroplasticity. The present study aimed to examine neurotoxin–induced PD-associated neuroplasticity changes in relationship to mental acuity and PD motor functionalities.
Methods: 7 voluntary participants experiencing early-stage PD symptoms with self-reported neurotoxin pre-exposure were enrolled in the longitudinal, repeated-measures clinical study; 2sex-matched, age-matched, and occupation-matched healthy subjects were recruited for controlled comparative analysis (n=9). UTRGV's Institute of Neuroscience (HION) served as study host, and its' facilities aided in data capture for both sessions, baseline and post-2 months. During the baseline session participants self-disclosed neurotoxin pre-exposure (e.g.pesticides, Agent Orange, heavy metals, insecticides). Study staff then collected outcomes related to mental acuity (SLUMS), PD-associated gait abnormalities (HY Scale), nonmotor/motor experiences burdening daily life (MDS-UPDRS), and arm motor functionalities. Corticospinal excitability and neuroplasticity were evaluated using Transcranial Magnetic Stimulation (TMS) in both groups. Specifically, we applied TMS at varying intensities to the area of the brain dedicated to the first dorsal interosseus (FDI) to evaluate neuroplasticity. Motor evoked potentials (MEPs) were recorded from the FDI at each assessed TMS intensity.
Results: Multivariate Analysis of Covariance revealed statistically significant mean differences in %MEP for Amplitude MEP and Area MEP after controlling for age, gender, mental status, HY ratings, motor function, and pre-stimuli EMG activity, [Pillai's Trace = 0.24, F(18, 1358) =10.6, partial eta2 = 12%, p < .001]. Post-hoc ANOVA's resulted statistically significant % MEP mean differences for EMG Area MEP, [F(9, 676) =18.0, partial eta2 = 19%, p < .001], and for EMG Amplitude MEP, F(9, 676) =19.0, partial eta2 = 20%, p < .001. HY ratings alone did not reveal statistical differences in mean EMG amplitude, p > .05, however, mean EMG G Amplitude for %
MEP 70-180 statistically fit a sigmoid model curve, F(1, 681) = 651.2, p < .001. The sigmoid model follows the specified equation, $y$ = 1 1+$e$−(0.374+0.989$x$).
Conclusions: Our findings suggest potential clinical implications in PD conditions related to motor function, with specific relationships between HY ratings and sigmoid model insights into physiologically observed differences. Identified differences in Amplitude MEP and Area MEP highlight the importance of multivariate approaches to understanding MEP. Application of the present study can improve a variety of areas: e.g., physical therapy, neurotoxin regulation, even PD treatment. It can be speculated that variables such as age, gender, mental status and prestimuli EMG activity should be carefully considered in future research on %MEP. Researchers should explore underlying mechanisms behind observed effects, interactions between variables, and clinical relevance of these findings. Specific implications may vary depending on the context of future research, e.g. characteristics of investigated populations, field of research (e.g., neurology, motor control, clinical rehabilitation), but nevertheless researchers should consider these conclusions in the broader context of existing literature and specific goals of investigation.

**The Role of Oral Microbiota in Periodontitis and Alzheimer's Disease**

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Description of Presentation
Porphyromonas gingivalis and Treponema denticola are major pathogens in adult periodontal disease, demonstrating a unique synergy that exacerbates some of the major symptoms and risk factors associated with AD. Both pathogens help each other metabolize, co-aggregate and cooperate, which further promotes disease progression in other parts of the body. Another way they work together is observed in with the blood brain barrier (BBB), as P. gingivalis enables T. denticola to regain its motility and move along the cells located in the neural region. By doing so, T. denticola can begin the inflammatory process in the brain, causing neurodegradation and norepinephrine imbalance, resulting in a cytokine cascade. The cytokine cascade then allows P. gingivalis to enter the bloodstream through the inflamed periodontal tissues, then into the brain further contributing to the neurodegenerative process. The pathogens moving from the oral cavity and into the cranial cavity not only causes inflammation in the brain, but also may cause neurodegeneration. In one instance, a study observed that mice who were affected by the T. denticola pathogen experienced a down regulation in specific proteins that protect neurons from AB-induced neuronal apoptosis. In a separate study, mice that were treated with P. gingivalis were observed to have a higher number of neurofibrillary tangles (NFTs), lower amount of intact neuronal cells, and an overall lower amount of gene expression. As seen through multiple studies, compared to the control, most mice affected with either of the pathogens show an increase in AB plaque buildup, NFTs, and neuronal loss, all of which being major risk factors and determinants of AD.

Therefore, the pathogens entering the cranial cavity is worrisome as both P. gingivalis and T. denticola can induce multiple strains of AB accumulation and NFT lesions in the hippocampi, leading to the hypothesis that both pathogens exacerbate Alzheimer's disease.

Background and Introduction

Periodontal disease (PD) is characterized by biofilm-induced inflammation of the soft and hard tissues that support dentition within the oral cavity and affects about 20-50% of the global population. Chronic PD results in inflammatory-mediated destruction of the periodontal pocket epithelium, leading to potential hematogenous dissemination of oral microbes that manifest systemic sequelae secondary to endotoxin and exotoxin damage. Notably, literature has linked elevated inflammatory states and transient bacteremia from chronic PD with coronary heart disease, heart failure, peripheral artery disease, stroke, osteoporosis, preterm birth, respiratory disease and even Alzheimer's disease. Red complex microbes, Porphyromonas gingivalis, Treponema denticola, and Tannerella forsythia, are recognized as the major pathogens in adult periodontal disease, with the former two species demonstrating unique synergy that enables their penetration through the blood brain barrier and subsequent neurodegeneration. Alzheimer's disease (AD) is an irreversible neurodegenerative disorder that clinically presents progressive memory loss and cognitive and behavioral decline. It remains the fifth leading cause of death among Americans aged 65 and older and impacts over 50 million lives worldwide. Pathogenesis is linked with aberrant cleavage of amyloid precursor protein resulting in oligomerization, diffusion into synaptic clefts, and interference with synaptic signaling. Literature supports the possibility of a microbiologic component to AD progression with some studies demonstrating the presence of gram-positive cocci, and rod-shaped bacterial genera Porphyromonas, Actinomyces, and Treponema within autopsied AD brains. This is significant given the potential neuroanatomic pathway between the trigeminal nuclei and locus coeruleus, connection between periodontal nerve endings and central nervous system, and the enhanced neurotropism exhibited by T. denticola with P. gingivalis. Overall, considering the burden both PD and AD have on the global population, investigation of their microbiologic link can be significant in highlighting clinical points of intervention to reduce progression and severity of both chronic conditions.

Methods

The systematic and extensive search was conducted in electronic databases including PubMed, PLOS ONE, Nature, Springer, and Sage covering from 2010 to 2023. The strategy of the search utilized both keywords and phrases, ranging from variations of "Porphyromonas gingivalis," "Treponema denticola," "periodontal disease," and "oral pathogens found in neurocognitive disorders." For the inclusion criteria, any study that was published in peer-reviewed journals, were in English, and specifically investigated either the pathogens themselves or the relationship between P. gingivalis and T. denticola in the context of AD were included. Both in vitro and in vivo research was accepted. Excluded studies included studies that were not peer-reviewed, case reports, reviews, commentaries, and non-English language publications. The reviewed articles were first screened by two independent reviewers who looked at the titles and abstracts of potential articles that were relevant to the research, then any article deemed relevant was given a full-text review. Data extracted from the studies were meticulously selected, this includes authors, year of publication, sample size, methods, key findings, tables & figures, and conclusions. After extraction, data were then synthesized through a narrative style approach, summarizing key findings and categorizing information based on the primary focus of the study. Ethical considerations were not required for this study, as it is a literature review that relied on publicly available data from previously published studies. The systematic review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines, allowing for transparency and comprehensiveness when it comes to the reporting of the research process and its findings.

Results

Multiple clinical studies have proven a link between periodontitis and AD. Two of the red complex pathogens, P. gingivalis and T. denticola, have the ability to mobilize from the oral cavity and into the brain through inflammation. T. denticola is associated with an increased neuronal apoptosis rate due to its ability to down regulate important proteins that help combat AB accumulation on the hippocampi. In addition, T. denticola might not only induce AB accumulation but also may cause neuronal apoptosis alone by similar pathways of mitochondrial apoptosis. However, more research is needed to prove this. On the other hand, P. gingivalis may enter the brain and further cause inflammation, which later leads to the degeneration of neurons. It is important to note that both P. gingivalis and T. denticola have been observed and thought to increase AB accumulation in the hippocampi, however the one difference is that P. gingivalis has shown to also induce an increased production of NFT's and neuron loss. This could be a reason to why multiple studies have shown that the degree of cognitive impairment in patients with severe periodontitis is 3 times greater compared to individuals with mild or no periodontitis. With these results alone it can be observed how both pathogens can exacerbate and worsen the development of AD. In conjunction, these results alone show how the synergistic work of both pathogens can exacerbate inflammation and thus, enhance the development of AD

# POSTER PRESENTATIONS

## FACULTY CATEGORY

**A Novel Exo-Glow Nano-system for Bioimaging**

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Background: Milk exosomes are widely used to improve the performance of various small macromolecules, oligonucleotides, and imaging agents for delivery and imaging applications. Indocyanine green (ICG) based Near-Infrared (NIR) fluorescent imaging is an attractive and safer technique used for number of clinical applications. However, ICG tend to have poor photostability, short half-life, non-specific proteins binding, and concentration-dependent aggregation. Therefore, there is an unmet clinical need to develop newer modalities to package and deliver ICG. Bovine milk exosomes are natural, biocompatible, safe, and feasible nanocarriers that facilitate the delivery of micro and macro molecules. Herein, we developed a novel exosomes based ICG nano imaging system that offers improved solubility and photostability of ICG.
Methods: Following acetic acid based extracellular vesicles (EV) extraction method, we extracted the bovine milk exosomes from a variety of pasteurized fat-free milks. The EVs were screened for their physicochemical properties such as particle size and concentration, and zeta potential. Stability of these exosomes was also determined under different conditions including storage temperatures, pH, and salt concentrations. Next, ICG dye was loaded into these exosomes (Exo-Glow) *via* sonication method and further assessed for its fluorescence intensity and photostability using an IVIS imaging system.
Results: Initial screening suggested that size of the selected bovine milk exosomes was from 100 - 135 nm with an average particle concentration of $5.8x10^2$ particles/mL. Exo-Glow (ICG loaded exosomes) further showed higher fluorescence intensity of ~ $2x10^{10}$ MFI compared to free ICG (~ $8.1x10^9$ MFI).
Conclusions: These results showed that Exo-Glow has the potential to improve solubility, photostability, and biocompatibility of ICG and may serve as a safer NIR imaging tool for cells/tissues.

**A TRANSLATIONAL APPROACH TOWARDS MORE COST-EFFECTIVE LUNG CANCER TREATMENTS**

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Lung cancer, globally the second most common cancer, causes 1.8 million deaths annually with 2 million new cases. It's also one of Finland's deadliest cancers. Chemotherapeutic agents, targeted drugs and radiation therapy have been the mainstay in oncologic treatment for lung cancer patients for decades. Checkpoint inhibitors (e.g. nivolumab, pembrolizumab, atezolizumab) have changed the landscape also in lung cancer. These drugs awake patients own immune system to attack cancer cells. These drugs are widely used in different cancers and they have efficacy also in lung cancer, but only in a minority of patients. Checkpoint inhibitors also induce autoimmune side effects. Majority are mild, but often irreversible, such as hypothyroidism or diabetes. Life-threatening and fatal adverse events are rare, but they also occur. Neither treatment response nor adverse effects can be predicted individually. In Finland, these drugs cost app. 10 000€ per month per one patient increasing the economic burden to our society. Thus, we need better understanding of lung cancer biology to be able to offer individual therapies in patient-centered manner, but also within the limits of the carrying capacity of our society. In 10-15% of lung adenocarcinomas there is a driver EGFR-mutation. Checkpoint inhibitor are ineffective in this type. However, EGFR pathway targeted agents, such as osimertinib, deliver often sustainable responses, but unfortunately resistance mechanism arise in this subtype as well. We need to better understand also the resistance mechanism in EGFR mutated subtypes to better tailor the best optimal treatment sequences to our patients.

Translational research, that combines the basic science of lung cancer biology with patient characteristics and treatment outcomes, is a crucial tool in responding to these demands. Tumor microenvironment plays an important role in carcinogenesis, as well as in treatment response, especially acidic environment is immunosuppressive and promotes cancer cell survival. Mucins are a large family of transmembrane glycoproteins expressed on epithelial membranes, including airways. They form the protective immunogenic glycocalyx against microbes as well as pollutants, e.g., carcinogens in tobacco smoke. They are involved in lung cancer formation and in drug resistance forming an important compartment of the tumor microenvironment protecting cancer cells from immune system. Mucins function also as a signaling platform orchestrating cell proliferation, migration and metastasis. Our hypothesis is that dysfunctional regulation and expression of mucin 13, a member of mucin family, provides an immunosuppressive environment and associates with poor response to checkpoint inhibition and poor survival. Mucin 13 is also involved in EGFR signaling. Analyzing mucin 13 with advanced biomolecular techniques using tumor and serum samples of Finnish lung cancer patients combined with clinical characteristics and treatment outcomes will elucidate the biological mechanisms and improve patient selection for optimal treatments.

**Assessment of Anti-cancerous properties of Nyctanthes arbor-tristis Linn natural compounds**

*Qazi Mohammad Sajid Jamal, Varish Ahmad*

Abstract

Purpose: Nyctanthes arbor-tristis Linn., the lovely and fragrant flowering tree, sometimes called night-blooming jasmine or parijat, is native to Southeast and South Asia. For ages, this has been utilized in traditional medicine. There are claims that its leaves and blossoms have anti-inflammatory, antimicrobial, and anti-diabetic qualities. Plants contain anti-cancer capabilities; however, this has not yet been investigated. Therefore, we propose to explore mechanistic interaction with different cancer targets and active chemicals of this plant through computational methods, which will be further subjected to in vitro/in vivo experimental validation to show its anti-cancerous potentialities.

Description: *Nyctanthes arbor-tristis Linn.* plant contains several natural compounds like flavonoids, lignans, saponins, and napthoquinones. *Nyctanthes arbor-tristis Linn.* has been used for generations to treat various illnesses, including certain malignancies, in ancient medical systems like Ayurveda. These uses, however, require substantial scientific support as they are predicated on anecdotal evidence. One of the most important factors in carcinogenesis and the success of anticancer treatments is inflammation. Our latest research on anti-inflammatory activities reveals that natural chemicals from *Nyctanthes arbor-tristis Linn.* can interact with cyclooxygenase-2 (COX-2) and prostaglandin E2 (PGE2).Cyclooxygenase-2, or COX-2, is a multifunctional and intricate protein that frequently aids in the growth and metastasis of cancer. COX-2 catalyzes arachidonic acid to produce prostaglandins, the main by-product of which is PGE2.

PGE2 is a signaling molecule that affects the behavior of tumor cells in several ways. PGE2 promotes cell division in cancer cells and prevents apoptosis or programmed cell death. PGE2 encourages the creation of new blood vessels, which gives tumors the oxygen and nourishment they require for survival. PGE2 facilitates metastasis, or the spread of cancer cells to other organs, by improving their capacity to move and infiltrate surrounding tissues. PGE2 inhibits the immune system's ability to fight cancer, which makes it easier for cancer cells to avoid being found and destroyed. The overexpression of COX-2 stimulates many signaling pathways in cancer cells, augmenting their proliferation and endurance. COX-2 can become constitutively activated in cancer cells due to mutations in the genes controlling its expression. Natural chemicals derived from this plant may target cancer genes such as TP53 (tumor suppressor), KRAS (oncogene), BRCA1, BRCA2, and HER2 (breast cancer) to express their anti-cancer capabilities. Thus, the explored interaction of active chemicals of this plant with cyclooxygenase and other cancer targets will help to suggest alternative anticancer molecules from this plant.

Looking Ahead: The proposed computational analysis led by molecular interaction and molecular dynamics simulation will provide new insight into alternative anticancer potentialities of natural resources, which could prevent life-threatening diseases and avoid the initiation of tumors.

**Diosgenin prevents breast cancer metastasis via the inhibition of epithelial-mesenchymal transition.**

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Background: Globally, breast cancer (BrCa) is the primary cause of cancer-related morbidity and death in women. Despite significant changes in healthcare activities like screening and early detection over the past few decades, African Americans (AA) continue to experience cancer health disparities. Many studies have been done on BrCa treatments, but AA patients have had less success than other racial or ethnic groups. Therefore, novel strategies are required to improve survival rates, lower BrCa mortality, and ultimately enhance the health of racial/ethnic minorities. Current treatment regimens, such as chemotherapeutic agents, are showing less effectiveness since they are linked to drug resistance, side effects, and the recurrence of the disease. Thus, the need for more potent therapeutic agents for BrCa is rising; these agents could be natural compounds that have been shown to have several targets, are less toxic and have fewer side effects. In this study, we investigated the effect of Diosgenin (DG), a natural compound derived from the plant Dioscorea villosa, on the BrCa cells metastasis using the disparity cell lines MDA-MB-468 (AA) and MDA-MB-231 (Caucasian American, CA).

Methods: Cell viability and cytotoxicity of DG were estimated by live/ dead cell assay; scratch assay and clonogenic assays were carried out following treatment with DG with various concentrations (5uM to 40uM) for different time intervals. RT-qPCR and western blots were used to assess the expressions of EMT (epithelial to mesenchymal transition) and apoptotic markers.
Results: Our results show DG considerably and dose-dependently reduced the number of colonies and the cell migration in both BrCa cell lines. Immunoblots and RT-PCR analysis showed a noticeable decrease in antiapoptotic (BCL-xL) and increases in pro- (BAK, BAX) and -apoptotic (PARP) markers. Furthermore, high doses of DG increased EMT markers expression, such as E-Cadherin, which, in turn, decreased ZEB-1 expression. Therefore, it stops BrCa cells from undergoing EMT. In addition, the effects of DG on blocking EMT pathways were verified using Nanostring technology.
Conclusions: Altogether, DG dramatically raised cytotoxicity and apoptosis and inhibited metastasis in both cell lines compared to control cells. These findings suggest that DG might be a therapeutic agent to treat BrCa metastases

**Integrative transcriptomics data analysis of TRPV1 in hepatocellular carcinoma**

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Background: Hepatocellular carcinoma (HCC) is the most common type of liver cancer. Transient receptor potential vanilloid 1 (TRPV1) is a nonselective $Ca^{2+}$ channel protein that is widely expressed and plays a significant role in cancer initiation and progression. However, the biological significance of TRPV1 in HCC has not been systematically and comprehensively investigated. Using deep data mining and transcriptomics analyses, in this study, we described the significance of TRPV1 expression and its association with HCC prognosis.
Methods: TRPV1 mRNA expression in HCC was examined using the Cancer Genome Atlas (TCGA), the Genotype Tissue Expression Atlas (GTEx), the Tumor Immune Estimation Resource (TIMER), and the UALCAN databases to examine the relationship between the expression of TRPV1 and the clinicopathological characteristics of HCC. The genetic alterations and frequency of TRPV1 were analyzed using the cBioPortal and COSMIC databases. The correlations between TRPV1 and tumor-infiltrating immune cells were examined using the TIMER 2.0, TISIDB, and GEPIA databases. The data processing analysis is based on the R language. LinkedOmics was used for TRPV1 co-expression network analysis.
Results: TRPV1 mRNA expression was upregulated in HCC samples as compared to normal liver tissues. Kaplan-Meier analysis demonstrated that high expression of TRPV1 is associated with better prognostic significance for overall survival (OS) and disease-free survival (DFS) in HCC patients. The mutation landscape analysis confirms that TRPV1 genetic alterations reached 6%, of which missense substitutions accounted for the highest proportion of 16.16%. The findings of the TIMER analysis indicated a correlation between immune cell infiltration and TRPV1 copy number alterations (CNA). The expression level of TRPV1 was positively correlated with the infiltration level of CD4+ T cells but negatively correlated with CD8+ T cells, B cells, macrophages, and dendritic cell infiltration. Additionally, TRPV1 expression was also found to be associated with certain immunosuppressive cells, chemokines, and receptors. Through Gene Ontology (GO) and Kyoto Encyclopedia of Genes and Genomes (KEGG) enrichment analysis, TRPV1 expression was found to be closely related to some immune pathways, including drug metabolism, PPAR signaling pathway, and chemical carcinogenesis.
Conclusion: Our observation demonstrates that TRPV1 was highly expressed in HCC tissues and is linked to prognosis of HCC patients and tumor immune responses.
Key words: TRPV1, Hepatocellular carcinoma (HCC), tumor-infiltrating immune cells, $Ca^{2+}$ channel protein

**Metagenomic Analysis Unveils the Microbial Landscape of Pancreatic Metagenomic Analysis Unveils the Microbial Landscape of Pancreatic Tumors**

*Sheema Khan, Anupam Dhasmana*

Pancreatic adenocarcinoma (PDAC) has a very expressed in pancreatic cancer. poor survival rate due to late diagnosis. Therefore, identification approaches that aid in early diagnosis are highly desirable. The composition of resident microbes in the human body is linked to various diseases and their treatment outcomes. Studies have identified bacterial communities associated with pancreatic ductal adenocarcinoma (PDAC), mainly in oral and gut samples. However, the prevalence of microbiota in pancreatic tumor tissues compared to their adjacent normal appearing tissues from the same patient can provide meaningful insights to develop newer diagnostic/prognostic molecular signatures. So, this study presents a comparative profiling for bacterial inhabitance in cancer tissues with that of matched adjacent normal tissues. This association will enable discovery of potential microbial biomarker(s) for PDAC. Additionally, we have correlated the alterations in selected microbial genera with PDL-1, which is a checkpoint being aberrantly

**MUC13- A Novel Cancer Biomarker for Early Pancreatic Cancer Diagnosis MUC13- A Novel Cancer Biomarker for Early Pancreatic Cancer Diagnosis and Imagingand Imaging**
*Sheema Khan, Anupam Dhasmana*

Pancreatic adenocarcinoma (PDAC) has a very poor survival rate due to late diagnosis. Therefore, identification approaches that aid in early diagnosis are highly desirable. MUC13 is a recently identified high molecular weight glycoprotein that is aberrantly expressed in PDAC and allows its progression via alterations of multiple tumor signaling pathways. Overexpression of MUC13 in PDAC cells leads to enhanced tumorigenic and metastatic phenotypes. These characteristics of PDAC cells are mediated by physical interactions between MUC13 and HER2/Neu. This study elucidates significance of MUC13, as a diagnostic and imaging marker of PDAC. Additionally, a collaborative effort has commenced to investigate the targeting ability of a novel anti-MUC13 monoclonal antibody (MAb) "C14" by radiolabeling with 89Zr for in vivo microPET/CT imaging.

**Novel nanoformulation of Sabizabulin (VERU-111) for pancreatic cancer therapy**
*Vivek Kashyap*

Background: Pancreatic cancer (PanCa) is one of the leading causes of cancer-related mortality in the United States due to very limited therapeutic options. Thus, developing novel therapeutic strategies will help for the management of this disease. We recently identified VERU-111, a novel synthetic molecule which showed potent anti-cancer effect against PanCa via targeting clinically important βIII and βIV tubulin isoforms. In this study, we synthesized and characterized its novel nanoformulation (MNP-VERU) and evaluated its therapeutic effects in vitro and xenograft mouse model.
Methods: MNPs were prepared by chemical precipitation method and loaded with VERU-111 using diffusion method. This formulation was characterized for particle size, chemical composition, and drug loading efficiency, using various physico-chemical methods (TEM, FT-IR, DSC, TGA, and HPLC). The internalization of MNP-VERU was achieved after 6 hours incubation with MNP-VERU in PanCa cells. To determine therapeutic efficacy of MNP-VERU, we performed various in vitro (MTS, wound healing, boyden chamber real-time xCELLigence, and apoptosis assays) and in vivo (mouse tumor xenograft) studies using PanCa. Effect of MNP-VERU on various key oncogenic signaling pathways, and miRNAs was evaluated by Western blot, immunohistochemistry (IHC), confocal microscopy, qRT-PCR and in situ hybridization (ISH) analyses respectively.
Results: Our novel MNP-VERU formulation provided an average size of 110 nm in dynamic light scattering (DLS) and exhibited -8.23 to -11.65 mV zeta potential with an outstanding loading efficiency (94%). Cellular uptake and internalization studies demonstrate that MNP-VERU escapes lysosomal degradation, providing efficient endosomal release to cytosol. MNP-VERU showed remarkable anti-cancer potential in various PanCa cells (Panc-1, AsPC-1, HPAF-II, BxPC-3, MiaPaca) and more effectively repressed βIII and βIV tubulin isoforms via restoring the expression of miR-200c. MNP-VERU more effectively suppressed AsPC-1 cells derived xenograft tumors in athymic nude mice.
Conclusions: Taken together, our results suggest that MNP-VERU has more anti-cancer potential than free VERU-111 against PanCa. MNP-VERU may reduce the toxicity and improve the bioavailability of free VERU-111 and could be used for the management of PanCa and health disparity.
Keywords: Pancreatic cancer; Sabizabulin;VERU-111; Tubulin inhibitor; MNPs

**Smoking and Drinking Activate NF-κB /IL-6 Axis to Promote Inflammation During Cervical Carcinogenesis**
*Vivek Kashyap*

Background: High-risk strains of HPV are known to cause cervical cancer. Multiple clinical studies have emphasized that smoking and drinking are critical risk factors for cervical cancer and its high-grade precursors. In this study, we investigated the molecular mechanisms involved in the interplay of smoking and/or drinking with HPV infectivity and defined a systematic therapeutic approach for their attenuation in cervical cancer.
Methods: The impact of benzo[a]pyrene (B[a]P) and/or ethanol (EtOH) exposure on cervical cancer cells was assessed by measuring changes in their biophysical, cell migration, and invasion characteristics. Expression of HPV16 E6/E7, NF-κB, cytokines, and inflammation mediators was determined using qRT-PCR, immunoblotting, ELISA, luciferase reporter assay, and confocal microscopy.
Results: Treatments with B[a]P and/or EtOH altered the expression of HPV16 E6/E7 oncogenes and EMT markers in cervical cancer cells; it also enhanced migration and invasion. In addition, B[a]P and/or EtOH exposure promoted inflammation pathways through TNF-α and NF-κB signaling, leading to IL-6 upregulation and activation of VEGF. The molecular effects caused by B[a]P and/or EtOH exposure were effectively attenuated by curcumin (Cur)/PLGA-Cur treatment.
Conclusions: These data suggest a molecular link between smoking, drinking, and HPV infectivity in cervical carcinogenesis. In addition, attenuation of these effects by treatment with Cur/PLGA-Cur treatment implies the role of curcumin in cervical cancer prevention and treatment.
Keywords: Cervical cancer; HPV16 E6/E7; Cigarette smoking and drinking; Benzo[a]pyrene; NF-κB; Cur/PLGA-Cur; Nanoformulation

# POST-DOC/FELLOW CATEGORY

**A Potential Role of Urinary p75ecd as a Biomarker for Amyotrophic Lateral Sclerosis in an American Cohort**
*Swati Dhasmana*

Background: Neurological disorders present a unique complexity compared to other diseases, involving multiple risk factors, causes, treatments, and outcomes. These disorders often exhibit various molecular and morphological changes indicative of disruptions in cellular plasticity and resilience. The pathogenesis of many neurological disorders remains unclear, necessitating ongoing investigations. Amyotrophic lateral sclerosis (ALS) exemplifies an idiopathic and fatal neurodegenerative disease marked by the degeneration of upper and lower motor neurons. The average life expectancy post-diagnosis is a mere 36 months, primarily attributed to respiratory muscle denervation.The persistent challenges in ALS clinical trials and the absence of effective therapeutic options have intensified interest in the potential role of biomarkers in advancing therapy development. Notably, neurofilament light (NfL) and phosphorylated neurofilament heavy (pNfH), cytoskeletal proteins in biological fluids, emerge as promising prognostic markers and potential pharmacodynamic biomarkers. However, their relatively stable levels over time limit their utility in reflecting disease progression. Consequently, a significant gap exists in identifying biological fluid-based biomarkers for monitoring disease progression. In response to this gap, our focus turns to the common neurotrophin receptor, p75, as a potential biomarker for motor neuron degeneration. Building on existing literature revealing elevated levels of the extracellular domain of p75 (p75ecd) in the urine of ALS patients compared to healthy individuals, we explore the potential of urinary p75ecd as a novel biomarker for disease progression and prognosis within an American cohort.
Methods: The study included samples from ALS patients and healthy controls. The urine samples of 60 confirmed ALS patients were purchased from 'National ALS biorepository'. The urine samples of 19 healthy controls were collected from friends, family, and colleagues on volunteer basis. The samples were collected and procured according to the IBC and IRB guidelines respectively. Each sample was tested in triplicate, to quantify p75ecd levels by sandwich ELISA and to quantify creatinine by colorimetric enzymatic assay. Levels of urinary p75ecd were standardised to urinary creatinine and data comparisons between two groups were performed using the Unpaired t test test for two independent groups.
Result: p75ecd was higher in patients with ALS (9.229 ± 1.198 ng/mg creatinine; N=60) compared to controls (3.979 ± 0.2891 ng/mg creatinine; N=19, p value: 0.0083).
Conclusion: The assay for urinary p75ecd demonstrates strong analytical robustness, signaling its potential as a promising biomarker for Amyotrophic Lateral Sclerosis (ALS) with applications in prognosis, disease progression monitoring, and potential pharmacodynamic assessments. Notably, urinary p75ecd stands out as a biomarker offering valuable prognostic insights and holds the unique distinction of being the sole biological fluid-based indicator of disease progression in ALS.

**Assessment of cytochrome P450 isoforms in colorectal cancer and utilization of phytochemicals for their modulation.**
*sudha .*

Cytochrome P450 (CYPs) belongs to Phase I enzymes and exists in multiple isoforms. CYPs are present in all tissues with the highest concentrations in the liver and small intestine. These are mainly involved in drug detoxification, cellular homeostasis, metabolism of endogenous & exogenous substrates and in the inactivation/ activation of several anticancer drugs. As these CYPs are selectively and differentially expressed in CRC, which could not only provide a mechanism for drug resistance, but also insights into future therapies that may use these enzymes as therapeutic targets. This study is designed to evaluate the differential expression of various CYPs isoforms in colorectal cancer (CRC) and their further modulation via phytochemicals using *in silico* approach. CRC differential expression data was downloaded from TCGA database, using the Broad Institute's firehose_get data-retrieval utility. DEApp tool, was used to carry out differential expression analysis. Three CYPs were found to be differentially expressed in colorectal cancer. Isoforms with downregulated expressions were CYP3A4, and CYP4B1 having fold change values of -6.1262, and -4.8293 respectively. However, the expression of CYP2W1 was upregulated with a fold change value of 8.39. Phytochemicals are known modulators of cytochrome P450's expression and activity. The present study was focused on modulating CYPs activity using phytochemicals. Molecular docking studies were conducted using Autodock vina. Phytochemicals were taken from three repositories: PDTDB, Serpentina, and Phytochemica and a list of phytochemicals was screened using *in silico* approach based on their binding affinity. The phytochemicals with binding affinity >-7 were taken into account. The common phytochemicals that were discovered to modulate all three cytochrome P450' s were rutin, swertiaside, and astragalin.

**Cucurbitacin B: A novel agent for inducing tumor-immune response.**

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Abstract:

Despite the emergence of immunotherapy as a potential breakthrough in cancer treatment, it showed only a marginal response in pancreatic and liver cancers. Thus, novel strategies are highly desirable to take full advantage of immunotherapy in the treatment of these cancers. One of the critical factors that influence the efficacy of immunotherapy is the increased infiltration of myeloid-derived suppressor cells (MDSCs) and tumor-associated macrophages (TAM) into tumors that alter the immune landscape and serve as facilitators of tumor proliferation, metastatic growth, and immunotherapy resistance. Thus, we believe that selecting a potent molecule that has the ability to suppress the function or revert the phenotypes of TAM and MDSCs will have a more significant impact in enhancing tumor immunotherapy response. Cucurbitacin B (Cuc B) is a potent inhibitor of Stat3, CSF-1R, and PI3Kγ and has shown its chemopreventive and therapeutic effects against various cancers but is limitedly explored for its application in modulating tumor immune response. In this study, we investigated the molecular effects and underlying molecular mechanisms of Cuc B on TAM and MDSCs. Cuc B significantly ($P<0.01$) decreased the expression of M2 markers (Arginase I, YM1 FIZZ1, PPARγ and TGFβ in M2 polarized BMDMs and increased M1 markers (NOS2, IL-6, and CD11C) compared to IL-4 alone treatment group. It has been demonstrated that TAMs secrete PDL-1 which neutralizes the function of T-cells. Cuc B treatment significantly ($P<0.001$) decreased PDL-1 expression in IL-4-treated RAW264.7 cells. Surprisingly, we observed that Cuc B treatment abolished the protein levels of PI3Kγ in IL-4 treated macrophages as determined by confocal microscopy and Western blot analysis. Cuc B treatment of bone marrow-derived MDSCs significantly ($P<0.01$) decreased the expression of Arginase-1, IL-10, PDL-1, and Stat3. We observed that IL-4-treated BMDMs inhibited phagocytic capacity which was significantly restored upon Cuc B treatment. We observed that Cuc B is a more potent molecule than a pharmacological inhibitor of PI3Kγ (IPI-549) in suppressing key signaling components of TAM and MDSCs. We are performing in vivo study to investigate Cuc B potential to enhance checkpoint blockade immunotherapy response in clinically relevant mouse models of cancer. These results suggest that Cuc B is a novel therapeutic agent which has the potential to suppress or revert TAMs and MDSCs phenotypes. Cuc B may be used as an adjuvant drug molecule in combination with PD1 or CTLA-4 antibodies for improving immunotherapy response against less responsive tumors.

**Development of miRNA mimics targeting tumor necrosis factor-α induced protein eight (TNFAIP8/TIPE) in oral cancer**

*Aviral Kumar*

Abstract

*Background:* The prevalence of oral cancer worldwide is on the rise, with India accounting for almost a third of all documented cases. Despite significant progress in therapeutic approaches, elevated mortality rates and recurrent disease incidences pose a considerable challenge to India's emerging healthcare sector. Hence, there is an urgent need to comprehend the underlying pathophysiology of oral cancer and devise innovative and effective therapeutic interventions for both prevention and treatment of this lethal ailment. TNFAIP8 is an apoptosis regulator proven to have an important function in the proliferation, invasion, metastasis, and progression of various malignancies. Therefore, targeting TNFAIP8 protein might be a crucial step in circumventing the oral carcinogenesis. MicroRNAs (miRNAs) being the master regulators of gene expression, are known to modulate the various hallmarks of oral tumorigenesis. Thus, the proposed study aims to identify significant miRNAs regulating TNFAIP8 protein and develop miRNA therapeutics for oral cancer treatment.

*Methods:* A comprehensive bioinformatics analysis for TNFAIP8 and the candidate miRNAs was undertaken using head and neck cancer (HNSC) datasets from different databases associating gene expression with various clinco-pathological attributes. Further, selected miRNAs were transfected in oral cancer cell lines and the functionality of the miRNA was assessed using different assays for establishing its therapeutic relevance.

*Results:* It was revealed that the expression of TNFAIP8 was significantly higher in HNSC patients in various databases, including GEO-HNSC (Gene Expression Omnibus-HNSC), TCGA-HNSC (The Cancer Genome Atlas-HNSC), and CPTAC-HNSC (Clinical Proteomic Tumor Analysis Consortium-HNSC). Moreover, utilizing web-based computational tools, seven potential miRNAs targeting TNFAIP8 were identified and subsequent analysis unveiled that the expression levels of five among these miRNAs exhibited significant downregulation in both TCGA-HNSC and CPTAC-HNSC datasets. Furthermore, qRT-PCR findings in different oral cancer cell lines were consistent with the in-silico analysis. Overexpression of hsa-miR-299-5p resulted in knockdown of TNFAIP8 with reduced proliferation, colony formation ability and autophagy with increased apoptosis in oral cancer cells. Further, miRNA-mediated knockdown of TNFAIP8 led to induction of S-phase arrest with increased levels of p-53 expression. It was also demonstrated that upregulation of hsa-miR-299-5p led to decreased invasion, migration and EMT of oral cancer cells. Moreover, mimics treatment inhibited the phosphorylation of NF-B and AKT, key signaling pathways in oral cancer development and progression.

However, further studies are ongoing to elucidate the effect of hsa-miR-299-5p mimics AKT/mTOR and STAT3 pathways.
*Conclusion:* In various HNSC datasets, TNFAIP8 exhibited upregulation, while hsa-miR-299-5p was downregulated when compared with appropriate controls. Hsa-miR-299-5p, identified as a putative regulator of TNFAIP8, functioned as a tumor suppressor gene, suppressing various cancer hallmarks like proliferation, migration, invasion and autophagy. Hence, targeting the TNFAIP8/hsa-miR-299-5p axis presents a potential strategy to inhibit oral tumorigenesis.
Keywords: miRNAs, oral cancer, TNFAIP8, hsa-miR-299-5p

**Development of miRNA therapeutics targeting ATP citrate lyase (ACLY) for the treatment of oral cancer**
*Uzini Devi Daimary*

Background: The global incidence of oral cancer (OC) has seen a consistent rise, with India accounting for nearly one-third of reported cases. Thus, there is an urgent need to gain a comprehensive understanding of the underlying pathophysiology of OC and develop effective and novel therapeutic interventions for the prevention and treatment of this deadly disease. Lipogenesis, a primary metabolic alteration in cancer cells, is linked to ACLY, a key enzyme in lipid biogenesis making it a promising target for OC treatment. Aberrant expression of ACLY regulates vital signaling pathways in tumorigenesis, indicating its potential as a novel therapeutic strategy against OC. MicroRNAs (miRNAs) are recognized as primary regulators of gene expression for their ability to modulate the diverse hallmarks of tumorigenesis. The proposed study aims to investigate the role of microRNAs in the regulation of ACLY and to develop microRNA therapeutics for the treatment of OC. The therapeutic potential of the candidate microRNA mimics that regulate ACLY expression will be tested in OC models.
Methods: *In silico* studies were conducted to determine the expression levels of ACLY mRNA, protein, and phospho(p)-protein in HNSCC patients obtained from GEO-HNSC (Gene Expression Omnibus-HNSC), TCGA-HNSC (The Cancer Genome Atlas-HNSC), and CPTAC-HNSC (Clinical Proteomic Tumor Analysis Consortium-HNSC) datasets. Moreover, ACLY expression was examined in OC cell lines via immunoblotting technique. Further, the microRNAs targeting ACLY were identified using various online web-based computational tools and further validated via the qRT-PCR method. To determine the functional effect of miR-655-3p on several hallmarks of OC, different assays were carried out to determine its therapeutic relevance.
Results: The analysis revealed that the expression of ACLY was significantly high in HNSC patients in various and had lower median survival compared to those with low ACLY-expressing groups. Moreover, ACLY is highly expressed in OSCC cell lines when compared to normal cells. ACLY targeting microRNA mimic i.e. miR-655-3p exhibited a downregulation of ACLY in oral cancer cells. As anticipated, the knockdown of ACLY via miR-655-3p resulted in a considerable inhibition of oral cancer cell proliferation, decreased oral cancer cell migration, and caused S-phase arrest. It also led to the modulation of various proteins implicated in critical cellular processes such as growth, survival, autophagy, EMT, etc. Furthermore, the silencing of ACLY by mir-655-3p resulted in the dysregulation of various components of the Akt/mTOR signaling pathway. The Akt/mTOR signaling pathway is a crucial regulator of cellular growth, and its constitutive activation is a well-known contributor to the development and progression of various cancers, including OC, through the activation of survival and proliferative genes.
Conclusion: In light of these findings, our results suggest that ACLY is highly expressed in OC and miR-655-3p might play an important role in suppressing the different hallmarks of OC. Thus, targeting ACLY via a-miR-655-3p serves as a potential strategy to inhibit oral carcinogenesis.
Keywords: miRNAs, oral cancer, ACLY, miR-655-3p

**Evaluation of ergosterol and its metabolites as LXR agonists and their anticancer potential in colon cancer**
*Yogain Taank*

Purpose: Aberrant cholesterol homeostasis is a well-recognized hallmark of cancer and implicated in metastasis and chemotherapeutic resistance, the two major causes of cancer associated mortality. Liver X receptors (LXRs) are the key transcription factors that induce cholesterol efflux via enhancing the expression of ABCA1 and ABCG1.
Methods: Molecular docking and dynamic simulation studies were done to assess the binding affinity and stability of the receptor ligand complexes. Activation of LXRs was evaluated using the luciferase reporter assay. qRT-PCR and western blotting was done to analyse the mRNA and protein expression of cholesterol homeostasis genes. Flow cytometric analysis was carried out to evaluate the surface expression of ABCA1. The effect of selected sterols on viability of three cancer cell lines and one normal epithelial cell line was assessed using MTT assay.
Results: Ergosterol (Erg), ergosta-7,22,24(28)-trien-3β-ol (Erg1), ergosta-5,22,25-trien-3-ol (Erg2), ergosta-5,7,22,24(28)-tetraen-3β-ol (Erg3), and ergosta-7,22-dien-3β-ol (Erg4) displayed good binding affinities and formed stable complexes with both isoforms of LXRs. Treatment with Erg led to 2.5 fold while Erg2 and Erg4 led to 1.7 fold increase in LXR activation. Furthermore, a significant increase in mRNA expression of *NR1H2*, *ABCA1*, *ABCG1* and *ApoE* was observed upon Erg treatment and it also led to a 25 fold increase in cell surface expression of ABCA1. All of the sterol were selectively toxicity toxic towards colorectal cancer cells but not towards normal epithelial cells.
Conclusion: Our findings suggests that ergosterol activates LXRβ and have significant anticancer activity and thus it could be a likely candidate to manage aberrant cholesterol homeostasis associated with colorectal cancer.

**Graphene nanoparticles for microRNA delivery**
*Eswara Naga Hanuma Kumar Ghali*

Background: Graphene (G) has been established as an exciting prospect for a broad range of applications owing to its remarkable properties. As the molecular structure of G itself is achiral thus introducing chirality in G by simple attachment of a functional group (a chiral ligand) on the G nanosheet may result in more diverse applications. The recent innovations of G chiral nanosystems have been extended to drug delivery. Herein, we have developed a novel and facile synthesis method for producing chiral G for its application in chirality-dependent microRNA delivery.
Methods: L-graphene and D-graphene were produced in a single step by using chiral L-tyrosine and D-tyrosine as stabilizing and chiral-inducing agents and applying high-temperature sonication. The chirality of the exfoliated L-graphene and D-graphene was assessed with circular dichroism (CD) spectroscopy and their structural, morphological, and surface evaluations were studied using Raman spectroscopy, transmission electron microscopy (TEM), and X-ray photoelectron spectroscopy (XPS), respectively. In addition, an attempt has been made to explore cell viability, hemocompatibility, cellular uptake, internalization pathway, chirality-mediated interaction, and microRNA (hsa-miR-205-5p) transfection with C4-2B prostate cancer cells.
Results: The CD spectra confirmed the chirality present in the exfoliated L(D)-Graphene. Moreover, the Raman spectrum and TEM data confirmed the formation of multi-layer graphene with asymmetric morphology and a large aspect ratio. L-graphene and D-graphene show cellular compatibility. Chiral preferential binding occurring between miR-205 and D-graphene makes them an exciting prospect for gene delivery. D-graphene exhibits superior hemocompatibility compared to commercially available transfection reagents (Lipofectamine). Cellular uptake is clearly shown by the internalization of D-graphene into C4-2B prostate cancer cells. miR-205 efficient delivery utilizing D-graphene was confirmed by transfection efficiency and MTT assay.
Conclusions: Our results demonstrated a direct approach- one-step liquid phase exfoliation-induced chirality in graphene and their selective chirality-mediated microRNA delivery.

**ICG-loaded mesoporous nanohybrid (Nd-doped Hydroxyapatite/$Fe_3O_4$) for photonic/magnetic hyperthermia and photodynamic therapy**
*Prakhar Sengar*

Background: Multimodality nanoplatforms play a crucial role in advancing medical interventions by integrating multiple functionalities into a single system. However, issues like intricate production processes and biocompatibility persist. Herein, a facile synthesis of a biomaterial-based mesoporous nanocarrier, HAp:Nd+SPIONs@$mSiO_2$, loaded with the near-infrared (NIR) emitting dye indocyanine green (ICG) is reported.
Methods: HAp:Nd nanoparticles were synthesized via combustion methods. Thereafter, commercial SPIONs and HAp:Nd were integrated within a mesoporous silica via a modified Stöber approach. HAp:Nd+SPIONs@$mSiO_2$ nanoplatform was characterized for particle size, porosity, and luminescence using TEM, BET, and luminescence spectroscopy. The synthesized nanoplatform was further loaded with ICG dye and the loading efficiency was analyzed via UV-Vis spectroscopy. Photonic and magnetic thermal heating of the ICG-loaded nanoplatform was also analyzed along with the photo-stimulated ROS generation ability. Finally, cytotoxicity and therapeutic analysis was performed in vitro using triple-negative breast cancer cells (MDA-MB-231).
Results: The nanohybrid with ~100 nm average size, comprised of Nd-doped hydroxyapatite (HAp), $Fe_3O_4$ superparamagnetic iron oxide nanoparticles (SPIONs), and mesoporous silica, exhibiting magneto-luminescent properties. The mesoporous structure was loaded with ICG as a model drug (4.3 µg/mg of nanoparticles) where a pH-dependent release was observed. The nanocarrier demonstrated dual functionality by generating heat through magnetic and photonic stimulation, as well as producing reactive oxygen species (ROS) upon excitation with 808 nm light. In vitro bioevaluation on aggressive triple-negative breast cancer cells (MDA-MB-231) showed the high biocompatibility of nanohybrid with and without ICG and exhibited significant toxicity after irradiation of NIR light. Noticeably, the nanohybrids also exhibit the ability to monitor temperature changes via $Nd^{3+}$ associated NIR luminescence.
Conclusions: The nanoplatform integrates clinically relevant components, highlighting its potential for translation from the laboratory to clinical applications. The developed nanohybrids, with combined NIR-mediated photothermal and photodynamic effects, magnetic photothermal capabilities, and NIR/MR imaging, offer promise in addressing cancer heterogeneity and improving conventional treatments with reduced side effects.

**Nebulization based Inhalation Nanomedicine for Lung Cancer Treatments**
*Rahul Tiwari*

Background: Lung cancer is reported to have a high incidence rate and first leading cause of cancer-related morbidity and mortality across the world including in the United States. Noninvasive nebulized inhalation is a promising delivery strategy for lung, which can enhance the targeting efficiency and detention time interval of nanoparticles in the lung tissue, thus elevating the therapeutic index of therapeutic agent(s) at lower dosages. The aim of this study is to develop inhalable nanoparticles (INPs) for effective delivery of therapeutic agents in lung cancer cell lines and ex vivo models.

Methods: The inhalation nanoparticles (INPs) were prepared by solvent evaporation and self-assembly approach. The INPs formulations were characterized by particle size, chemical composition, and drug loading efficiency using various analytical methods including FT-IR, DSC, SEM, and DSC/TGA. Cellular uptake of INPs was evaluated in 2D and 3D models of lung cancer cell lines (A549 and NCI-H1299) using fluorescence microscopy and flow cytometry analysis. Additionally, the therapeutic evaluation of gambogic acid and gemcitabine encapsulated INPs was performed by basic in vitro biological assays using proliferation (CCK-8), mucoadhesion Boyden chamber, and apoptosis assays using lung cancer (A549 and NCI-H1299) monolayers, spheroids, and xenograft tumors.
Results: The developed INPs exhibited an average size of ~110 nm in dynamic light scattering measurements. INPs formulation showed a remarkable mucoadhesion and mucopenetration potential in-vitro model(s). Cellular uptake studies demonstrated that INPs formulation facilitates an effective endosomal release into the cytosol. The in vitro study confirms that INPs release the drugs in a sustained manner. Additionally, the INPs formulation showed superior in vitro anti-cancer activity in lung cancer cell lines, spheroids and xenograft tumor.
Conclusions: Altogether this study confirms that INPs formulation demonstrates an improved therapeutic benefit over free drug against lung cancer cell lines, spheroids and xenograft tumor. This study could lead as an innovative therapeutic modality for the treatment of lung cancer.

**Translational impact of UBTF in developing new therapeutic strategy for pancreatic cancer treatment**

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Pancreatic cancer (PanCa) is the third leading cause of cancer-related deaths in the United States. It shows only a marginal survival benefits of 6 months with the available therapeutics. Thus, to understand the biology of PanCa and discovery of new molecular targets will help in designing the new therapeutic strategies for the management of advanced PanCa. Aberrant ribosome biogenesis is the hallmark of many cancers and has been associated with poor prognosis. Cancer cells show significantly high demand of protein synthesis and to meet this demand modify ribosomal machinery at genetic and epigenetic level. The *UBTF* (upstream binding transcription factor, RNA polymerase I) gene encodes a member of the HMG-box DNA-binding protein family, which plays a critical role in ribosomal RNA (rRNA) transcription. Recent studies provide strong clues of UBTF involvement in cancer cells survival and chemoresistance. However, no study defines its role in pancreatic cancer. Our results demonstrated that UBTF expression was significantly higher in pancreatic tumors than normal pancreatic tissues as analyzed in tissue microarray. We also observed differential overexpression of total and phospho-UBTF in various human PanCa cells compared to normal pancreatic ductal epithelial cells. Next, we generated UBTF knockdown clones of HPAF-II and MiaPaCa-2 cells and characterized by analyzing UBTF and pre-rRNA expression. We observed 80% inhibition of UBTF and pre-rRNA complex expression which indicates the role of UBTF in functioning of RNA Polymerase I (RNA Pol I). kinase array results showed a significant ($P<0.01$) decrease expression of mutant p53 in UBTF knockdown MiaPaCa-2 cells (express mutant p53). We also observed that targeted inhibition of UBTF sensitizes the effect of gemcitabine and RNA Pol I inhibitors (BMH-21 and CX-5461) in PanCa cells. Targeted knockdown of UBTF significantly ($P<0.01$) inhibited the growth of pancreatic tumor in orthotopic xenograft mouse model as analyzed by tumor volume and excised tumor weight. We observed decrease expression of various cell proliferative (PCNA and ki67) and ribosome biogenesis markers (UBTF, RPA-194, pre-rRNA) compared to WT HPAF-II cells xenograft tumors. Taken together, our results suggest that UBTF could be developed as a potential molecular target for pancreatic cancer therapy.

**TRIP13's crucial role in pancreatic cancer progression**

*Swati Dhasmana, Anupam Dhasmana, Stella Rios, Sheema Khan, Farrukh Afaq, Upender Manne , Murali M Yallapu , Subhash Chauhan*

Background: Pancreatic cancer, characterized by its high mortality rate, stands as one of the most aggressive cancer forms. The projected surge in pancreatic cancer-related deaths, making it the second leading cause in the United States by 2030, underscores the urgency for effective early screening tools. This study employs data mining methods to scrutinize bioinformatic data surrounding TRIP13. Examining differential expression across various cancers, correlating TRIP13 expression with pancreatic cancer stages, exploring associations with common cancer genes, and analyzing overall survival rates constitute the core investigations. Integrated with molecular biology techniques, the study further quantifies TRIP13 expression in progressive pancreatic cancer cell lines and human pancreatic tissues. The research unveils TRIP13's role at both transcriptional and translational levels, suggesting its potential as a specific biomarker for early pancreatic cancer detection, with implications for patient prognosis and targeted therapies in clinical settings.
Methods: Utilizing extensive transcriptomic data analysis, the study employs bioinformatics tools such as ConSurf, GTEx, GEPIA2, and LinkedOmics. Molecular biology techniques including qPCR, western blotting, and IHC are applied to validate and integrate bioinformatics findings.

Result: ConSurf analysis identifies highly conserved amino acids within the AAA+ ATPase domain of TRIP13. Increased TRIP13 expression correlates with lower disease-free survival in pancreatic cancer, displaying positive associations with CEACAM5 and S1004A. Isoform analysis reveals seven TRIP13 transcripts, with two coding transcripts. Multiple phosphorylation sites further characterize TRIP13. mRNA expression analysis in disease-free conditions indicates minimal TRIP13 expression, notably higher in pancreatic cancer than in normal tissues. Molecular biology techniques confirm elevated TRIP13 expression in moderately differentiated cell lines and tumor grades. Functional enrichment analysis links higher TRIP13 expression to modulation of crucial pathways such as DNA repair, cellular senescence, and viral carcinogenesis.
Conclusion: This study positions TRIP13 as a potential early diagnostic biomarker for pancreatic cancer, offering prospects for enhancing current biomarker panels. The integrated biology approach holds promise for identifying specific biomarkers not only for pancreatic cancer but also for other malignancies.

# MEDICAL RESIDENT CATEGORY

**Chemical-induced Pancreatitis in a Peritoneal Dialysis Patient at South Texas: A Case Report**
*Yareli Durazo*

Acute pancreatitis is the leading cause of gastrointestinal-related hospitalization in the United States. Pancreatitis is often linked to excessive alcohol consumption or gallstones, accounting for 80% of all cases. However, in some cases, it can be drug-induced acute pancreatitis (DIP) or via chemical injury.
Icodextrin (Extraneal) is a high-molecular-weight glucose polymer developed as an alternative osmotic agent to dextrose in peritoneal dialysis (PD). It is regarded as biocompatible due to its iso-osmolarity, making it generally safe and well-tolerated. While rare, cases have been reported associating icodextrin with pancreatitis; nevertheless, specific rates of icodextrin-induced pancreatitis are unknown. In this case, we present a long-term PD patient recently exposed to Icodextrin and presented with acute pancreatitis.

**Cocaine Inducing QT Prolongation**
*Jian Garcia Cruz, Elimar Gonzalez Morales, Alberto Pena*

Introduction
The Q-T interval represents ventricular repolarization of the heart. Prolongation of this interval is called Long QT syndrome (LQTS) and can lead to deadly arrythmias such as Torsades de Pointes. (1) According to the AHA, a Q-T interval is prolonged when it is >450ms in males and >460ms in females. LQTS has different etiologies (not limited to, but including): idiopathic, congenital, electrolyte abnormalities, and drug-induced. (2) However, recreational drugs such as cocaine may often be overlooked by some. A study by Magnano et al. found that cocaine can increase a QTc interval by 23 points (+/- 25) (P Case: 42-year-old gentleman with a known history of hypertension not on medications and polysubstance abuse who presented to the ED with complaints of fever, hypertension, sore throat, nausea, bilateral upper and lower extremity joint pains and stiffness and was admitted for sepsis due to influenza. Patient was taking ibuprofen and acetaminophen since the symptoms started. UDS was positive for cocaine, cannabinoids and opioids. EKG on admission was remarkable for sinus tachycardia with qtc of 472. While on the hospital his blood pressure remained persistently elevated with systolic blood pressure in the 200s after nifedipine and hydralazine were administered at the ED. Enalapril injection 1.25 mg q6h PRN was commenced but patient's blood pressure remained elevated with systolic blood pressure in the 200s. Patient blood pressure continued to be elevated regarding treatment; therefore, the patient was transferred to the ICU for the management of hypertensive urgency with Cardizem drip. EKG on the second day showed sinus tachycardia with qtc 452. No medication that affects the qtc was given or taken by the patient that is known to cause qtc prolongation except for cocaine. Conclusion: Cocaine use has been associated with QTc prolongation, which can increase the risk of serious cardiac arrhythmias. Cocaine can interfere with the normal functioning of ion channels in the heart, leading to disruptions in the electrical signals. This disturbance can prolong the QTc interval, potentially causing torsades de pointes or other arrhythmias. Prolongation of the QTc interval can lead to an increased risk of this abnormal heart rhythm, which may result in fainting, seizures, or even sudden cardiac death. Healthcare providers need this information to make informed decisions about prescribing medications, avoiding potentially harmful drug interactions, and ensuring patient safety. It's crucial for healthcare professionals to be aware of these risks when treating individuals who use or have a history of cocaine use, as it can impact decisions regarding medication and overall patient care.

**Exploring the Long-Term Consequences of Neglected Hypothyroidism Patient at South Texas**
*Elimar Gonzalez*

Hypothyroidism is a condition caused by the thyroid gland's insufficient production of thyroid hormones. This affects 13.95% of the population according to data from this year. Long-term untreated hypothyroidism, which is not frequently seen, can lead to serious health concerns as well as apparent physical signs like fatigue, cognitive difficulties, dry skin, hair loss, and constipation. While most of the people in the US may be able to receive treatment for this debilitating disease, there are specific vulnerable populations that due to socioeconomic difficulties, may not have access to medications.

**Jungle Fever: Navigating Malaria in Immigrant Travelers Patient at South Texas: A Case Report**
*Elimar Gonzalez*

Malaria is a severe mosquito-borne illness that can be life-threatening. This infection continues to be a significant global health concern, most prominent in tropical and subtropical regions. Malaria's clinical manifestations exhibit a striking diversity, influenced by geographical variables, the individual's immunity, and age. Diagnosing and treating malaria early is crucial to preventing severe complications and fatalities. This heterogeneity poses unique challenges to its diagnosis and treatment, making it a subject of continuous study and innovation. In this case, we present a case of a patient traveling from an endemic area to a non-endemic area.

**METHOTREXATE-INDUCED PANCYTOPENIA: A CALL FOR MINDFUL MEDICATION PRACTICES**

*Eunbee Cho*

Introduction

Methotrexate(MTX) is a widely used disease-modifying antirheumatic drug for various conditions including autoimmune diseases or malignancies. Pancytopenia as an adverse effect of methotrexate is rarely reported but sometimes fatal, and requires more attention. This case report shows a 76-year-old woman with a history of rheumatoid arthritis(RA) on MTX therapy, presenting with severe pancytopenia and oral lesions, and highlights the importance of safe medication practices especially in elderly patients with poor health literacy and compliance.

Case Presentation

A 76-year-old female with rheumatoid arthritis presented to the emergency department with a worsening oral lesion. She had been on an unspecified maintenance dose of MTX for the management of RA for more than 2 years without adequate follow-ups. On initial evaluation, the patient had multiple ulcerative lesions in her oral cavity involving buccal mucosa and soft palate, and severe gingivitis in the upper and lower gumline, associated odynophagia. She also had similar ulcerative lesions in the genitalia with vaginal bleeding. On initial labs, she had severe pancytopenia with hemoglobin(Hb) 8.6 g/dL, white blood cell(WBC) count 670/uL with absolute neutrophil count(ANC) 300/uL, and platelet(PLT) 12,000/uL. It was a dramatic change compared to the lab 3 months ago showing Hb 10.7g/dL, WBC 4,710/uL, and PLT 142,000/uL. As an initial investigation of pancytopenia and newly developed mouth ulcers, broad differentials are discussed including primary bone marrow(BM) disorder, medication-induced pancytopenia, nutritional deficiencies, infectious disease, and autoimmune disease. Autoimmune panels including ANA and ANCA came out negative. Vitamin B12 and Folic acid levels were above normal limits, and infectious workup with HIV antibody and RPR tests came out negative. Peripheral blood smear revealed pancytopenia but didn't reveal myeloid or blast cells. BM biopsy revealed markedly hypocellular BM with 2~3% cellularity. Flow cytometry did not reveal any abnormalities. The methotrexate level that was drawn on day 2 of admission was unremarkable. With the ambiguous diagnosis of methotrexate-induced pancytopenia versus aplastic anemia, the patient was started on eltrombopag, which was discontinued on day 5 with dramatic improvement in all cell lineages. Upon discharge, the patient was put off the methotrexate and was closely monitored in the outpatient setting without treatment for pancytopenia. The patient continued to maintain the recovered state of all cell lineages, which is more suggestive of drug-induced pancytopenia

Discussion

MTX-induced pancytopenia is a rare but severe complication observed in patients undergoing high-dose infusion therapy or long-term maintenance therapy with a possible pathophysiology being interference of DNA synthesis by inhibition of dihydrofolate reductase, leading to BM suppression. Although MTX is a widely used medication, safety education for physicians and patients has fallen short. For patients with rheumatologic diseases, the initial and the maintenance dose and duration of this medication differ greatly and toxicities should be closely monitored with proper education. Especially in elderly patients with a poor understanding of the disease, adverse effects can be seen more frequently and safety education is warranted.

**Newly diagnosed Ulcerative Colitis in a Young Hispanic Mexican Female at South Texas: A Case Report**

*Elimar Gonzalez*

Ulcerative colitis (UC) is a chronic inflammatory bowel disease (IBD) characterized by recurring episodes of inflammation limited to the mucosal layer of the colon. Traditionally, this disease has been associated mostly with white Americans, however in recent years, an increase incidence has been reported in minorities in the US and around the world. In Hispanic population, cases are frequently underdiagnosed, and literature is scarse. In this case, we present a young patient without past medical history who presented with what appeared to be an infectious gastroenteritis and found to have a severe UC.

**SICK SINUS SYNDROME AND TAKOTSUBO CARDIOMYOPATHY**

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INTRODUCTION

Takotsubo cardiomyopathy (TSCM) or broken heart syndrome was first described in Japan in the 1990s, the syndrome has gained worldwide attention within the scientific community in the past few decades. The disease manifests predominantly in postmenopausal females in the presence of stressful triggers such as severe physical or emotional stress. Initially thought to be a benign condition, recent reports have demonstrated that TSCM may be associated with severe complications and mortality similar to acute coronary syndrome. Concerted efforts have been made to define various pathophysiologic aspects of TSCM; however, the precise etiologic understanding remains unclear. Some of the mechanisms proposed for the development of Takotsubo syndrome include elevated levels of circulating plasma catecholamines and their metabolites, microvascular dysfunction, inflammation, estrogen deficiency, spasms of the epicardial coronary vessels, and aborted myocardial infarction. In our patient, there was no acute stressor factor identified.

CASE REPORT
A 69-year-old lady with OSA, Vit D deficiency, venous insufficiency, and sick sinus syndrome (s/p pacemaker in 2006). The patient presented to the ED referred by her PCP for evaluation of chest pain and dyspnea on exertion that started while she was dancing on a Saturday night. Initially, the pain was severe 9/10, accompanied by chest pressure and diaphoresis, with no radiation. She denied stressing factors. Her symptoms improved with rest, and she went to bed, the next morning the patient woke up with chest pain and mild dyspnea, so she decided to consult with her PCP. On her way to the ER, she was given nitroglycerin by EMS which subsided her chest pain. In the ER her symptoms were mild, with no respiratory distress, on room air, and speaking in full sentences, her MAP was around 66-70, her HR was in the low 60's, and her RR was 18. On physical exam no JVD, clear lungs, and unremarkable S1 and S2 without pedal edema. Her EKG showed new T wave inversion in anterolateral leads, and elevated HS troponin (186). Negative UDS. 2D Echocardiogram showed LVEF 50% with apical akinesis and basal septal hyperkinesis. Left heart catheterization was done and revealed widely patent LAD, LCX, and RCA (dominant). The patient's symptoms resolved and was started on DGMT before discharge.
DISCUSSION
The modified Mayo Clinic criteria are used to make the diagnosis of Takotsubo cardiomyopathy and include the following: 1. Absence of coronary artery disease on angiography 2. Transient dyskinesis, hypokinesis, or akinesis of the left ventricle midsegments with or without apical involvement 3. ECG evidence of ST-segment elevation and/or T wave inversion 4. Modest elevation of troponin levels 5. Absence of myocarditis or pheochromocytoma Management of TSCM mainly involves supportive therapy. Approach to acute management can be based on the suspected underlying cause, although this is difficult to achieve as patients presenting are usually worked up for other diseases such as acute MI, and the diagnosis is not made until after catheterization and left ventriculogram. Heart failure can occur, especially in patients aged >70 years, and those with LVEF <40% and physical stressors. Mild cases (stable vitals, minimal troponin leak, no cardiogenic shock) can be treated with classic heart failure guideline-directed therapy including ACE-I/ARB/ARNI, betablockers, mineralocorticoid receptor antagonists, and/or another diuretic[1]. If patients are asymptomatic and hemodynamically stable, pharmacotherapy should be carefully chosen to avoid inflicting iatrogenic harm on an otherwise naturally recovering process [1]. Short-course anticoagulation can be added especially if there is a concern for thrombus formation related to cardiac akinesia/dyskinesia with ballooning [1]. Patients with large areas of hypo/akinesia and/or LVEF <30% need to be observed more closely for heart failure, atrial/ventricular arrhythmias, cardiac thrombi, and cardiogenic shock [2].
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**The Cardiac Cost of Cancer Care: Atezolizumab-Induced Dilated Cardiomyopathy**
*Jian Garcia Cruz, Elimar Gonzalez Morales, Conrad Chouinard, Martha Solis*

Atezolizumab is a humanized immunoglobulin G1 (IgG1) monoclonal antibody that binds to programmed cell death-ligand 1 (PD-L1) and blocks interactions with the PD-1 and B7.1 receptors on activated T cells. It activates PD-L1/PD-1 pathway-mediated inhibition of the immune response, leading to activation of an anti-tumor effect. This drug is commonly used for urothelial cancer, non-small cell lung cancer (NSCLC), small cell lung cancer (SCLC), breast cancer, and hepatocellular cancer. A recent trial called First-Line Atezolizumab plus Chemotherapy in Extensive-Stage Small-Cell Lung Cancer demonstrated the addition of atezolizumab to chemotherapy in the first-line treatment of extensive-stage small-cell lung cancer resulted in significantly longer overall survival and progression-free survival than chemotherapy alone, for which this medication has been included as part of treatment. Many side effects of this medication have been listed, but the most common cardiovascular effects are arrhythmia (1%), hypotension (5%), myocarditis (rare), and venous thromboembolism (4%). We present a case of new-onset, heart failure secondary to dilated cardiomyopathy secondary to atezolizumab in a patient with stage IV small cell lung cancer.

**The Dog That Broke My Heart**
*Eunbee Cho*

Introduction
Takotsubo cardiomyopathy, also known as stress-induced cardiomyopathy or "broken hearts yndrome" is a reversible form of acute heart failure characterized by temporary left ventricular dysfunction, typically precipitated by intense emotional or physical distress. Its clinical presentation mimics that of acute coronary syndrome with chest pain, shortness of breath, elevated troponins, and sometimes with electrocardiogram(EKG) changes, which makes initial diagnosis challenging. This case report presents a Takotsubo cardiomyopathy in an 81-year-old female, which developed after the loss of her beloved Basenji puppy.

Case Report

An 81-year-old female presented to the emergency department(ED) with worsening dyspnea for one day. On the day before admission, she had a long car journey to return the Basenji puppy that she bought 3 months ago. The patient reports she loved her puppy but had to return the puppy due to the amount of mass it was causing at home. After the trip, she felt extreme fatigue and dyspnea with walking from a room to a bathroom, prompting her ED visit. On admission, she revealed a pulse rate of 92, blood pressure within normal range, and had signs of respiratory distress. Chest X-ray showed characteristic COPD changes without signs of cardiomegaly or pulmonary edema. A CT angiogram ruled out pulmonary embolism. Laboratory investigations revealed an elevated BNP at 1136 and elevated high-sensitivity cardiac troponins peaking at 546, followed by subsequent readings at 351 and 313. An EKG demonstrated normal sinus rhythm with occasional premature ventricular contractions. An echocardiogram(ECHO) displayed severe LV dysfunction, estimating a LVEF of 25-30%. This was characterized by akinesis of multiple walls (anterior, anteroseptal, apical lateral, mid inferior, and inferior apical), limited apical dyskinesia, and grade 3 diastolic dysfunction. Left heart catheterization revealed LV apical ballooning extending beyond the Left Anterior Descending artery's territory, consistent with Takotsubo cardiomyopathy. Minor non-obstructive coronary artery disease with 30% stenosis at multiple locations was observed (mid LAD, ostial first diagonal, ostial RCA, and midRCA). After establishing the diagnosis of Takotsubo cardiomyopathy, recommendations were made to start a short course of diuretics for management of acute systolic congestive heart failure. A plan was set for a repeat ECHO before discharge to reassess LVEF, with consideration for a LifeVest if LVEF remained at or below 35%. The patient's clinical status improved with her repeat ECHO on day 3 demonstrated a slight improvement in LVEF to 36-40%. She was discharged with recommendations for close outpatient follow-up, optimization of medical therapy for heart failure with short-term beta blocker and diuretics therapy, and counseling for coping with emotional stressors.

Discussion:

Takotsubo cardiomyopathy is typically precipitated by intense psychological stress and primarily occurs in postmenopausal women. The characteristic finding of apical ballooning is seen in left ventriculography or ECHO. Despite frequent hemodynamic compromise, most of the patients recover completely within the next few weeks. This case emphasizes the potential development of Takotsubo cardiomyopathy secondary to severe emotional stress, which can differ among individuals. Identifying stressors and adjusting medications based on patient history is crucial for optimal management.

# STAFF CATEGORY

**Assessing Gait Metrics for Early Parkinson's Disease Prediction**
*Daniel Salinas*

BACKGROUND: Parkinson's Disease (PD) is characterized by both motor and non-motor symptoms, and its diagnosis primarily relies on clinical presentation. There is a growing need for diagnostic tools to identify the early signs of PD, particularly the initial motor impairments often manifested as gait abnormalities. Here we seek to present preliminary findings to address this need. Our study focuses on using Machine Learning techniques (ML) to predict the PD clinical stage most efficiently and accurately. Specifically, we have sought to evaluate how spatiotemporal characteristics and other locomotor performance variables obtained on a walkway system can be utilized to identify the Hoehn and Yahr (HY) score in PD.
METHODS: Six individuals with PD and 6 Healthy individuals participated in the study. PD patients were classified on the HY scale by a physician (score range 0-5). Participants completed eight passes on the Zeno Walkway while Protokinetics Movement Analysis Software recorded and calculated the temporal, spatial, and pressure measurements of within-step recordings. Data preprocessing and predictive modeling were analyzed using R and the caret package. Multiple regression, utilizing predictors such as gait speed, left and right steps, and walking methods (socks, shoes, and barefoot), were employed to normalize the data. The data was split using 80% for training and 20% for testing and used three repeated 10-fold cross-validations. Models included Random Forest, Neural Networks, Naive Bayes, Support Vector Machines with Linear Kernel (SVM), Penalized Multinomial Logistic Regression, and eXtreme Gradient Boosting. Models were compared based on a weighted rank system, prioritizing model accuracy, interclass balanced accuracy, computational efficiency, kappa, weighted averages of the area under the curve (AUC) for HY ratings, and successful participant prediction.
RESULTS: The eXtreme Gradient Boosting algorithm demonstrated the best ability to predict HY scores, achieving an overall model accuracy of 97.4%, interclass balanced accuracy of 97.3%, Kappa of 95.9%, weighted AUC of 52.6%, and data partition learning of 2.7 seconds. The top four gait patterns in PD were Stride Time, Stride Velocity, Integrated Pressure, and Stance Center of Pressure Distance.
CONCLUSIONS: Our data highlights the need to test the accuracy and efficiency of multiple models to provide real-time feedback in clinical populations. Furthermore, the success of the deployed ML algorithms in this study motivates further exploration to identify the economic feasibility of early detection of PD. The Gait mat and programmed software may assist patients in accessing affordable, validated, and reliable clinical assessment for early-stage Parkinson's Disease.

**Engaging the South Texas Community: Utilizing Community Member Feedback to Inform Research, Services, and Community Engagement**
*Dolores Garcia*

BACKGROUND
The UT Health San Antonio Mays Cancer Center Community Outreach and Engagement Core is identifying how South Texas Latino residents navigate cancer-related health needs. Our team explored these from a class and place perspective.
METHODS
Listening sessions were conducted across South Texas. Sessions lasted 1.5 hours, were in English and Spanish, and recorded. Recordings were transcribed, and a thematic analysis was completed.
RESULTS
Seventy-four South Texas residents participated in the sessions. Insurance status and healthcare access were key factors impacting communities' ability to navigate cancer-related health needs. Analyses were explored from an urban, rural, inland, and border perspective.
*Urban*
· For inland participants, access to services was a barrier in the context of inconvenient clinic hours, commute time, and distance to facilities.
· For border participants, access to services was a barrier in the context of lack of diagnostic/treatment services and making travel arrangements to services in inland cities.
· A shared perspective was the negative impact of healthcare expensiveness, and insurance not covering full costs of treatment/screenings.
*Rural*
· For inland participants, access to services was a barrier in the context of few or no urgent care and hospital facilities and unreliable ambulance services.
· For border participants, traveling to Mexico for affordable services is common.
· A shared perspective was insufficient access to primary, specialty, and cancer care.
CONCLUSION
This study uncovered the nuanced factors associated with the cancer burden for South Texas Latinos. Findings will be used to contextualize how Political Determinants of Health contribute to cancer-related health inequities in South Texas.

**Exploring Imageability through Architecture to Study Neuroscience: Preliminary Results of a Systemic Review**
*Cristian Maestre*

Background: Neuroscience and architecture are often combined to study the impact of environment, physical spaces, colors, shapes, and buildings on brain activity and health. This is an emerging field with distinct areas examining architecture in relation to neuroscience. Among the numerous elements of architecture, imageability seems to be of particular interest. Imageability refers to the quality of a physical space that evokes strong images in people's mind, and influence cognitive functions including visual, memory, and spatial recall. It is hypothesized that environments, spaces, and buildings with poor imageability might negatively affect cognition, behavior, and brain health. Diverse studies have been conducted to test such a hypothesis however, there is a lack of compiled evidence that highlights how imageability and neurosciences are connected. Therefore, we conducted this systematic review to explore the current understanding of imageability from an architectural perspective in the study of neuroscience with focused on its implications for cognitive health and well-being.
Methods: This review conducted a comprehensive search across four electronic databases: EBSCO, OVID, PubMed, and Web of Science. Our search term included "Imageability" as the main key word combined (AND) with architecture, environment, built environment, neuroarchitecture, aphantasia, urban design, memorability, visual recall, mental visualization, architectural features, façade, wayfinding, familiarity, familiarity, architecture, familiarity, environment, and vividness. Eligibility criteria included peer-reviewed articles in English that focused on the relationship between imageability, health, and architecture. The synthesis of results was conducted following PRISMA's four-phase flow diagram.
Results: The initial search showed 5269 articles, which were screened to exclude duplicates (n=1763). Subsequently, we conducted a thorough review of the remaining 3506 articles and we excluded (n=3393) articles that were not related to the research, non-original research (n=24), systematic review (n=5), not enough data (n=3), non-related articles, and for various other reasons (n=13). The selected studies (n=61) highlighted the impact of architecture on cognitive, the role of urban design in mental health, and the application of brain imaging methods to assess the influence of built environments. Imageability involves and contributes to various cognitive processes such as memory, perception, sensation, and language depending on the type of stimuli used. Imagery was shown to activate the visual cortex and showed great activity in the anterior areas of the brain such as the insula, medial frontal cortex, and left dorsal lateral prefrontal cortex.
Discussion: This review highlights the potential of integrating neuroscience into architectural design, fostering a multidisciplinary approach to enhance cognitive and brain health. Spaces with high imageability, characterized by distinctive, memorable features, can significantly influence cognitive processes, such as memory and spatial navigation. We found limited manuscripts about imageability in elderly people and this is a gap that warrants attention because of the alarming rates of neurodegenerative diseases such as Alzheimer's disease-related disorders in elderly populations. The findings suggest that a more profound understanding of imageability within neuroarchitecture can lead to innovative design strategies that support mental and physical well-being.

**Exploring the Role of Mucin 13 in Hepatocellular Carcinoma**
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Abstract
Background: Hepatocellular carcinoma (HCC) has a poor prognosis due to ineffective therapeutic modality and lack of early diagnostic marker. Accumulating studies have shown that elevated expression of mucin 13 as potential oncogene and predictive biomarker for various cancer. However, very little is known about its expression and function for development and progression of HCC.
Objective: To investigate mucin 13 expression in chemically induced hepatocellular carcinoma model.
Methodology: Diethyl nitrosamine (DEN) and 2-Acetylaminofluorene (2-AAF) induced method was employed for the development of hepatocellular carcinoma in Male Wistar rats. Serum and tissues were collected at regular intervals of time and routinely validated for liver cancer stages. Immunohistochemistry and *in situ* hybridization were performed on formalin-fixed, paraffin-embedded tissues. Molecular docking studies were performed to study the interaction of mucin 13 and DEN.
Results: Our results demonstrate hepatocellular adenoma as observed by histopathological analysis. Biochemical analysis showed a progressive increase in the levels of serum ALT, AST, and ALP, suggesting the development and progression of hepatocellular damage. Notably, mucin 13 expression gradually elevated during consecutive stages of hepatocellular carcinoma. Interestingly, an increase in nuclear localization of mucin 13 was observed in the treated group as compared to control group. *In situ* hybridization analysis showed that a decrease in miR-132 and miR-145, which are inversely related with mucin 13 expression. Moreover, DEN efficiently binds mucin 13 with high affinity and thus stabilizes it as demonstrated by molecular docking analysis.

Conclusion: These results suggest that mucin 13 expression is closely associated with hepatocarcinogenesis and could serve as a predictive candidate biomarker for HCC.

**Integrated Cancer Research Core (ICRC) at the University of Texas Rio Grande Valley (UTRGV)**
*Iris Enriquez*

Background: The University of Texas Rio Grande Valley-School of Medicine (UTRGV-SOM) has founded the South Texas Centre of Excellence in Cancer Research to address disproportionate cancer related health issues in the Valley. UTRGV-SOM intends to establish an Integrated Cancer Research Core (ICRC) Facility at the UTRGV campus to provide cutting-edge technologies to support cancer research investigators of the Rio Grande Valley.
Methods: The ICRC will house most recent technologies (Flow Cytometry, Image Stream Flow, Droplet Digital PCR, molecular imaging, microPET/CT imaging, etc) to support flow cytometric analyses in cell suspension and tissues, cell sorting, analysis of tumor immune components, microbiome and probiotics, cancer health disparity, antibody drug conjugates production, antibody nanoparticle therapies, antibody radionuclides research.
Results: This facility will not only enhance chances of research funding but also provide cost-effective access to the modern cancer research facilities for developing basic and clinical translational collaborative research and trained local medical personnel in the Valley. This allows to train students around cancer biology and cancer immunology to produce a future generation of cancer researchers from the Valley.
Conclusion: Overall, the establishment of this core facility creates a highly conducive environment in the Valley for cancer prevention research as no such facility exists within a 250-mile radius from the proposed site. This core also accelerate cancer research training and education in RGV region.

**Interdisciplinary Integrated Primary and Behavioral Healthcare (I2PBH) Initiative**
*Maria Hernandez*

The Interdisciplinary Integrated Primary and Behavioral Healthcare (I2PBH) Initiative will train University of Texas Rio Grande Valley (UTRGV) mental health graduates to deliver high quality, evidence-based Integrated Behavioral Health (IBH) services in the Rio Grande Valley (RGV) – a high-need, high-demand, medically underserved Hispanic region along the US-Mexico border. Specifically, the I2PBH initiative will train 24 UTRGV mental health graduates annually to deliver high-quality IBH clinical services through the evidence based Primary Care Behavioral Health (PCBH) model. With a training emphasis on basic/advanced theory and clinical skills in the PCBH model, students will serve as Behavioral Health Consultants (BHC) to meet practicum/internship requirements while working alongside healthcare professionals in a primary care setting. The I2PBH initiative increases the presence of culturally concordant, primary care competent BHCs on the front lines of four rural clinics to function as primary care providers (PCP) extenders for all behaviorally informed needs of patients, increasing access and delivering whole-person care.

**Social and Structural Determinants of Health Associated with Cervical Cancer Screening Uptake in South Texas**
*Caroline Puckett*
*Authors: Caroline Puckett B.S., Dolores Garcia B.S., Rebecca Jones, Ph.D*

Background: The Mays Cancer Center (MCC) is one of only four National Cancer Institute (NCI)-designated cancer centers in Texas and the only one in South Texas. The MCC serves 38 counties and over 4.9 million residents; 70% of residents are Hispanic, 25 of 38 counties are designated as rural areas; additionally, 25 of the 38 counties have census tracts designated as areas of persistent poverty. Catchment area residents are at disproportionately greater risk of developing liver and intrahepatic bile duct cancer (64%), cervical cancer (46%), gallbladder cancer (8%), gastric cancer (4%), and pediatric leukemia (32%) compared to the nation. Currently the Papanicolaou test (often called a pap test) is considered the most effective screening test for identifying abnormal cells associated with cervical cancer. During a pap-test, cervical cells are collected and examined for pre-cancerous or cancerous changes. When cancer is detected at an earlier stage, before symptoms start to appear, it is often easier to treat. For South Texas women, evidence suggests that barriers to receiving a pap-test include low health literacy, cultural beliefs and norms, and lack of health insurance coverage. Additionally, there is growing evidence that structural and social determinants of health (SDoH) also impact adherence to cancer screenings. The purpose of this study was to examine select SDoH that may be influencing South Texas women's decisions to receive a pap test.
Methods: In the summer and fall of 2020, the MCC Community Outreach and Engagement team fielded the South Texas Survey. This survey captured information in seven specific domains, two of which are social determinants of health and cancer screening practices. This survey was designed using a probabilistic sampling frame for our catchment area with representation from a metropolitan and race and ethnicity perspective. A total of 555 individuals completed the survey of which 382 identified as female (69%). For this study, descriptive statistics and chi-square analysis were used to examine the study population. Generalized logistic regression were used to identify individual, social, and structural factors associated with cervical cancer screening uptake. All data were weighted, and analyses were conducted using the R statistical software.

Results: Overall, 80% of women indicated they had received a pap-test. When examining women who indicated not receiving a pap-test, a greater proportion were Hispanic, single and did not have health care coverage. For a SDoH perspective, regression results determined that at the individual level, single women had 89% lower odds of having received a pap-test compared to married women (OR: 0.11; CI: 0.03-0.42) and women who had a higher discrimination index score had 27% lower odds of having received a pap-test compared to women who had lower index scores (OR: 0.73; CI: 0.53-1.00).
Conclusion: South Texas women who are single, report perceptions of greater discrimination, and/or do not have healthcare coverage are less likely to have a pap test. Results provide insight into the influence of social and structural determinants of health on South Texas women's decisions to receive a pap-test.

**The Fortify Resilience Initiative**
*Maria Hernandez, Yvette Cantu*

The Fortify Resilience Initiative focuses on building and sustaining a culture of wellbeing for Residents and Fellows (R/Fs) at The University of Texas Rio Grande Valley (UTRGV) School of Medicine's (SOM) Graduate Medical Education (GME) residency and fellowship programs. In order to address the multitude of threats to physician wellness and to mitigate the silent, but pernicious effects of burnout on these physician learners serving in the RGV, this Initiative from the Office of GME will strengthen existing wellbeing pathways while expanding additional solutions that will work to sustain wellbeing. Utilizing a combination of prevention, promotion, and intervention strategies targeted at the individual, program, and system levels, this initiative increases resilience by addressing existing gaps that only further propagate the spread of risk and vulnerability to the community.

**Update on the Role of MUC13 in Pancreatic Cancer: A Promising Early Detection Biomarker**
*Anupam Dhasmana, Swati Dhasmana*

Background: With the rise in pancreatic cancer (PanCa) prevalence and mortality rate, by 2030 it will secure second position among leading causes of cancer-related deaths. Due to poor prognosis of PanCa only 11% of PanCa patients have a 5-year survival rate, resulting in an equal mortality rate and incidence rate. 85% of PanCa are Pancreatic ductal adenocarcinoma (PDAC). The main clinical challenge with PanCa is poor treatment outcomes due the late diagnosis. Currently, there are traditional biomarkers panels available for diagnosis, however, these biomarkers do not have optimal sensitivity and specificity for PanCa. Considering this alarming unmet clinic need, our team has identified a novel transmembrane glycoprotein, MUC13, as a potential biomarker of PanCa by using integrative big data mining and transcriptomic approaches.
Methods: The current study used big transcriptomic data analysis. MUC13 structure was elucidated using SPARKS-X and ConSurf server followed by GTEx server to analyze protein expression coverage & tissue specific gene expression. PDAC patient's gene data was downloaded from TCGA dataset for DEG analysis and R packages "DEseq2 package" was used for the count data normalization and visualization. Furthermore, ONCOMINE and GEPIA2 were used for analyzing and predicting CNV, pathological staging, disease-free survival plot, MUC13 isoforms and phosphorylation sites. Lastly, LinkedOmics was employed for exploring the genes that exhibited disparity in association with MUC13 in Pancreatic Cancer.
Result: We have modeled the structure of MUC13 to visualize its various domains, exposed and functional residues, as its crystal structure is unavailable in public domain. Interestingly, we identified approximately 63 highly conserved, exposed and functionally active residues. It was observed via DEGseq2 of TCGA-PAAD data set that MUC13 had a better expression profile ( 3.73-fold) as compared to MUC1 ( 2.52-fold) in PanCa condition which suggests better specificity of MUC13 over MUC1. The higher expression of MUC13 correlated to a lower disease-free survival in PanCa. Isoform analysis suggested that MUC13 has 5 transcripts, among which only 2 transcripts (ENST00000616727.4 & ENST00000478191.1) of MUC13 are coding. Interestingly, ENST00000616727.4 transcript which encodes for long form of MUC13 (L-MUC13 & 512 residues), is tumorigenic (tMUC13). While ENST00000478191.1 transcript encodes for the short form of the MUC13 (s-MUC13 &184 residues) and has shown less expression in tumors. Socio-behavioral & demographic studies on MUC13 show that ethnicity, age, and gender are important factors for higher expression of MUC13 in PanCa. Our analysis suggests that Afro-American and Asian PanCa patients express relatively higher MUC13 as compared to Caucasian. The higher expression of MUC13 leads to modulation of several important pathways like chemical carcinogenesis, maturity onset diabetes of the young, pancreatic-bile secretion and glucose and lipid metabolism. Interestingly, ENST00000616727.4 transcript which encodes for long form of MUC13 (L-MUC13 & 512 residues), is tumorigenic (tMUC13). While ENST00000478191.1 transcript encodes for the short form of the MUC13 (s-MUC13 &184 residues) and has shown less expression in tumors. Socio-behavioral & demographic studies on MUC13 show that ethnicity, age, and gender are important factors for higher expression of MUC13 in PanCa. Our analysis suggests that Afro-American and Asian PanCa patients express relatively higher MUC13 as compared to Caucasian. The higher expression of MUC13 leads to modulation of several important pathways like chemical carcinogenesis, maturity onset diabetes of the young, pancreatic-bile secretion and glucose and lipid metabolism.
Conclusion: This investigation sheds light on MUC13 as a potential early diagnostic biomarker for PanCa, and it also has prospective to upgrade the effectiveness of the current biomarker panel. This kind of methodology will enhance the conception of the role of MUC13 in PanCa. Additionally, the big data analysis methodology is releasing a significant opportunity for the discoveries of specific and significant biomarkers not only for PanCa but also for other malignancies.

# MEDICAL STUDENT CATEGORY

**A Comparative Literature Review on Temporary Deafferentation Techniques**
*Maria Lozano Bonilla, Jared Hensley, Hunter Butler*

Background: Temporary deafferentation (TD) is an approach aimed at improving motor and somatosensory performance by inducing temporary anesthesia, typically focused on the upper extremity. This approach has demonstrated the capacity to stimulate cortical plasticity, allowing reorganization of the primary motor and somatosensory cortices, which has proven useful in rehabilitation. Various techniques have been used to achieve temporary anesthesia, including pneumatic tourniquet cuffs, blood pressure cuffs, injections, and topical anesthetics. Here, we conducted a literature review to provide a comprehensive comparative analysis of the different methods used to perform TD in the field of neurorehabilitation and sought to identify the advantages and disadvantages of each.
Methods: We conducted a literature review of neurorehabilitation studies that used TD. We performed searches in PUBMED, MEDLINE, and Google Scholar with the following terms: temporary deafferentation, ischemic deafferentation, temporary function deafferentation, deafferentation AND tourniquet, lidocaine hydrochloride AND deafferentation. We evaluated studies from December 2023 to 1997. Our search terms provided 533 possible studies for inclusion in our analysis. We removed duplicate studies, studies not written in English and studies where the manuscript could not be retrieved due to archiving limitations.
Results: Our preliminary analysis revealed 30 studies that have been conducted on TD in neurorehabilitation, primarily in the population of stroke. TD methodology varied with topical anesthetics being the most common. Preliminary findings indicate the efficacy of all three methods at improving sensorimotor function in both anesthetized and non-anesthetized arms. However, variations were observed in the extent of improvement, with only some methods demonstrating prolonged retention. Additionally, certain techniques, such as cuffs and injections, were associated with pain, potentially affecting study outcomes if patients were unable to complete the trial.
Conclusion: Temporary deafferentation shows promise for neurorehabilitation as it has proven to improve motor and somatosensory performance. Although various techniques have been employed to achieve TD, evidence suggests that a topical method is preferred as it provides efficient results while minimizing patient discomfort. Further research is required to establish the best approach to achieve optimal deafferentation with the use of a topical anesthetic.

**A True Bloody Emergency: An Unusual Case of Thrombotic Thrombocytopenic Purpura**
*Authors: Ninan J.*
Background
Thrombotic thrombocytopenic purpura (TTP) is a primary thrombotic microangiopathy that is classically characterized by thrombocytopenia and microangiopathic hemolytic anemia (MAHA). Although rare with an annual incidence of 3.7 cases per one million adults, it is considered a true hematological emergency due to its fatality rate of almost 100% if appropriate treatment is not initiated immediately. This makes it vitally important to identify and treat patients with TTP, a task that becomes unusually challenging in the absence of the disorder's other characteristically diagnostic clinical features such as mucosal bleeding, fever, or presence of schistocytes.
Case Presentation
A 30-year-old gentleman with a past medical history of Autoimmune Hemolytic Anemia is admitted for severe thrombocytopenia found on routine laboratory testing. The patient endorsed generalized weakness, fatigue, headache and dark-colored urine onset three days ago. Three months ago, the patient was admitted for exacerbation of warm AHA and started on Prednisone 100 mg post-discharge. The patient reports he had been tapering off the medication and stopped completely three days ago. The patient's is unremarkable except for tachycardia and mild distress, His laboratory findings include platelet count at 16k/µL, hemoglobin 11.1 gm/dL, hematocrit 33.7%, MCV 92.2 fL, lactic acid 3.17 mmol/L and creatinine 1.3 mg/dL. Initial management plan included methylprednisolone 125 mg IV, peripheral blood smear and CBC to monitor platelet count with transfusion precautions if levels drop below 10k/µL. A few hours into admission, the patient developed confusion, hyperbilirubinemia, worsening thrombocytopenia (13k/µL) and 1.5 mg/dL to 1.7 mg/dL increase in creatinine. Although the lack of schistocytes on peripheral blood smear dismissed the possibility of MAHA, the acutely worsening condition of the patient raised concerns for acute TTP and orders for fresh frozen plasma transfusions and plasmapheresis were empirically initiated. The patient's TTP PLASMIC score was 6 and an ADAMTS13 level was ordered. The patient was also started on daily doses of rituximab 375 mg/m2 and methylprednisolone 1000 mg. Nephrology was consulted for concerns of acute kidney injury given up-trending creatinine levels and urine studies showing microscopic hematuria with 3+ blood in urine. CT abdomen/pelvis and renal doppler ultrasound were performed which resulted in findings indicating inflammatory nephritis. Over the course of the hospital stay, the patient developed small petechiae on his abdomen and bilateral upper extremities and reported episodes of dizziness and nausea. Subsequent daily rounds of TPE, transfusions and steroids, however, resulted in the resolution of both neurological and hematological symptoms.

The patient's creatinine level improved to 0.9 mg/dL, lactic acidosis resolved with lactic acid at 1.24 mmol/L, and bilirubin levels returned to normal limits as well. ADAMTS13 level was noted to be 0.03% which confirmed thrombotic thrombocytopenic purpura as the precipitating pathology in this patient's condition. With a significant improvement of the patient's thrombocytopenia (platelet counts 350k/µL), patient was deemed fit for discharge under strict recommendations to continue steroid treatment and regular follow-up appointments with his hematologist/oncologist.
Conclusion
Acute episodes of TTP are extremely life-threatening situations and immediate recognition and intervention is vital for patient mortality, especially in such non-classical presentations.

**Advanced Neuroimaging Findings in Patients with Neurotoxin-Exposure Related Parkinson's Disease**
*Kevin Garcia Valdez*
*Kevin Garcia Valdez, Daniel Salinas, Blake Martin, Jared Hensley, Hunter M. Butler, Russell W. Wiggins, Chloe Harris, Nawaz Hack, Kelsey Baker*

Background: Neurotoxin exposure has been linked to a variety of neurological conditions and diseases. This is likely because many neurotoxins (natural and synthetic) can cross the blood brain barrier and impact the structure and function of neuronal cells and pathways. In particular, neurotoxin exposure has had a strong association with development of Parkinson's Disease (PD), with the Department of Veteran's affairs acknowledging the link in 2010. Here, we seek to understand if patients with PD and a history of neurotoxin-exposure demonstrate a different disease pathophysiology. Specifically, we want to evaluate microstructural changes in the brain in patients with PD and a history of neurotoxin-exposure.
Methods: We enrolled three subjects with neurotoxin-related PD (identified as Px1, Px2, and Px3) and one healthy control. Subject Px1 identified household lead pipes for neurotoxin exposure and was classified a 2 on the Hoen-Yahr (HY) scale by study physician. Subject Px2 identified pesticides for neurotoxin exposure and was classified a 1 on the HY scale. Subject Px3 identified pesticides for neurotoxin exposure and was classified a 2 on the HY scale. Following enrollment, the patients and control underwent magnetic resonance imaging (MRI) of the brain. Our scanning sequence included structural (T1) imaging, and multi-shell diffusion weighted imaging (DWI). Images were preprocessed using FSL and Neurite Orientation Dispersion and Density Imaging (NODDI) post processing was calculated using Accelerated Microstructure Imaging via Convex Optimization (AMICO). For all subjects, the region of interest (ROI) of subthalamic nucleus was drawn and analysis performed using FSL.
Results: The subthalamic nucleus (STN) from the healthy control and three patients with Parkinson's Disease with varying neurotoxin exposure was evaluated for neurite density and volume. The healthy control had values of 2221.556810 and 256.640625 $mm^3$ for neurite density and volume, respectively. Px1 had density and volume of 2184.348319 and 195.117106 $mm^3$. Px2 had density and volume of 2400.858196 and 165.234283. Px3 had density and volume of 2076.662732 and 272.461028.
Conclusions: When neurotoxins enter the nervous system, they alter the structure and function of neuronal pathways. These changes have been linked to neurodegenerative disorders such as Parkinson's Disease. Compared to the control, subject Px1 showed a reduced volume and neurite density of the STN. Subject Px2 displayed a reduced volume and a greater neurite density. Subject Px3 showed a reduced neurite density and an elevated volume. A decrease in volume and neurite density suggests that exposure to neurotoxins such as household lead pipe and pesticides can impact neuronal architecture and therefore predispose individuals to Parkinson's Disease. The paradoxical elevated neurite density and volume seen in subjects Px2 and Px3, respectively, alludes to the pathophysiological variability between patients. It would be relevant to further evaluate the extent of microstructural changes and their relationship with type and duration of neurotoxin exposure, and clinical symptoms such as tremors, cognitive impairment, and behavioral changes.

**Addressing Educational Disparities to Improve Health**
*Ronald Shaju*

Background: The Rio Grande Valley (RGV) has emerged as a region in the United States grappling with profound health challenges, prominently characterized by elevated rates of diabetes and obesity among its diverse residents. In this comprehensive public health research endeavor, our objective is to delve deeply into the intricate relationship between educational disparities and health outcomes of the RGV population. This connection can then be utilized as a focal point for breaking down the health disparities in the RGV.
Methods: Employing a rigorous methodology, we conduct an exhaustive literature review to illuminate the connection between educational disparities and healthcare disparities in the Rio Grande Valley. Our exploration aims to find the extent to which disparities in education contribute to the emergence and perpetuation of healthcare disparities within the region. Drawing on valuable insights garnered from over nine-hundred hours of active participation in the "College 1st" educational outreach program, we underscore the pivotal role that a higher education may present for betterment of health. Surveys are done after each event and 300 of these surveys were collected to be analyzed. Each survey contained questions assessing the effectiveness of the camp in changing the students' cognitions about college, homework, and school.

Results: The findings underscore an urgent and compelling need to address prevailing educational gaps within the RGV, emphasizing that rectifying these gaps is not only crucial but also a fundamental step toward improving the overall health and well-being of its diverse residents. Despite the region's enduring reputation as a focal point for health disparities, acknowledging the connection between educational disparities and health disparities is paramount in designing effective intervention strategies such as College 1st. Survey analysis is still underway, but the preliminary data showcases that the implementation of College 1st program helps to improve the students' cognitions surrounding homework, school, and attending college.
Conclusion: This research contributes to the broader discourse on health disparities by providing an examination of the relationship between educational disparities and health outcomes in the Rio Grande Valley. It is imperative to address educational gaps to find a solution for the healthcare disparities in the Rio Grande Valley. Through these efforts we can pave the way for a healthier and more equitable future for the Rio Grande Valley, which can serve as a model for the world.

**Birth defect trends within Texas Public Health Region 11, 2000-2019: an analysis of Texas Department of State Health Services public data**

*Miguel Lopez, Jonathan Hebert*

South Texas is a predominantly Hispanic region with high rates of chronic illness, poor healthcare access, and a history of birth defect clusters. Between 1986 and 1991, 47 cases of anencephaly in Cameron County were linked to elevated fumonisins in the region's corn-based diet, prompting a series of ongoing public health efforts. This paper aims to identify changes in prevalence for CNS defects, in addition to cardiac, circulatory, gastrointestinal, and genitourinary defects in South Texas within the last two decades. Public data on 20 birth defects from the Texas Department of State Health Services were obtained for decades 2000-2010 and 2010-2019 in Texas Public Health Region 11 and the remaining regions of Texas. We report that Region 11 saw larger birth defect prevalences compared to the remainder of Texas in both decades studied. When looking at single regions between decades, there was an increase in the prevalences of microcephaly, ASD, pulmonary valve atresia or stenosis, PDA, and hypospadias within Region 11 in 2010-2019; the prevalences of these defects also increased in the remaining regions of Texas in 2010-2019, with the addition of 8 more: hydrocephaly, double outlet right ventricle, tetralogy of Fallot, VSD, tricuspid valve atresia or stenosis, coarctation of the aorta, stenosis or atresia of the small intestine, and renal agenesis/dysgenesis. Pyloric stenosis alone saw a significant decrease in prevalence in 2010-2019 for both regions in this study. Furthermore, it was found that the prevalences of anencephaly and spina bifida without anencephaly were unchanged in both regions.

**Characterization of Anti-Cancer properties of Fungal Metabolite Ophiobolin A**

*Asma Syed, Rozena Shirvani*
*Title: Characterization of Anti-Cancer properties of Fungal Metabolite Ophiobolin A*
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*Abstract Type: Research/Clinical Abstract*

Background: Ophiobolin A (Oph A) is a secondary metabolite and a phytotoxin produced by the pathogenic fungi *Cochliobolus heterostrophus* that causes "southern corn leaf blight" disease in maize via modulation of the calcium binding protein calmodulin. Numerous studies have found antiproliferative effects of Ophiobolin A against a variety of cells including bacteria and various cancers including melanoma, glioma and leukemia. Recent studies have shown that OphA induces paraptosis-like cell death in glioblastoma multiforme (GBM) cells via vacuolization of the cytoplasm and enlargement of the mitochondria and endoplasmic reticulum. Notably, unlike apoptosis, paraptosis cell death lacks DNA fragmentation and activation of caspases, creating a possible mechanism for targeted treatment of malignant brain tissue of GBM patients who have GBM cells that are highly resistant to pro-apoptotic treatments. This study aimed to further characterize the effects of this promising anti-cancer agent on glioblastoma (U118 and U87), breast cancer (MCF7 and T47D), neuroblastoma cells (SH-SY5Y) and rat pheochromocytoma cells (PC12). SH-SY5Y and rat PC12 commonly serve as two popular neuron models to test drug toxicity and viability.
Methods: The effects of Ophiobolin A were studied in six cancer cell lines from various tissue types including glioblastoma, breast cancer and rat pheochromocytoma cells: U87, MCF-7, T47D, U118, SH-SY5Y and PC12. Over 4 weeks, cell lines were recovered and cultured until adequate confluence was reached with subculturing and medium refreshing. Once adequate growth and attachment was confirmed with microscopy, cells were seeded in 6-well plates from 100k to 600k and treated with either DMSO or 1 µM of Ophiobolin A. Cell morphology was monitored using inverted microscope at 1 hour, 3 hours, and 6 hours following Ophiobolin A application. Cell survivability was measured using Countess at 6 hours post drug treatment.
Results: Researchers observed morphological changes post treatment with 1 µM Ophiobolin A (Oph A) in breast cancer cells (MCF), pheochromocytoma cells (PC12) and glioblastoma cells (U87) compared to DMSO controls.

The impact of Oph A on cell morphology changes demonstrated a time-dependent manner using computer assisted microscopy. In MCF7 cells, we observed increased vacuolization after drug treatment. In PC12 we observed changes in cell body morphology and increased cell detachment. In U87 we observed elongated neuronal bodies and enlongated cell processes post Oph A treatment. Cell survival rate of each cell line 6 hours post treatment was measured and analyzed using a two sample equal variance T-Test. P values: *MCF= 8.761E-06, PC12= 0.725, U87= 0.125. *denotes p value Conclusions: This study highlights the importance of understanding the mechanisms underlying the OphA-induced cell death as a potential candidate for cancer therapy in cancers highly resistant to current treatment options. In previous studies, OphA was found to induce cell paraptosis and decrease cell numbers in glioblastoma cell lines. In this study, MCF cell lines showed statistically significant reduction in cell survivability post Oph A treatment and this change was not observed in PC12 cells. Citation: Marco Masi, Ramesh Dasari, Antonio Evidente, Veronique Mathieu, Alexander Kornienko, Chemistry and biology of ophiobolin A and its congeners, Bioorganic & Medicinal Chemistry Letters, Volume 29, Issue 7, 2019, Pages 859-869, ISSN 0960-894X, https://doi.org/10.1016/j.bmcl.2019.02.007.

**Contribution of 24-h Blood Pressure Variability to Dementia-Related Disorders in Hispanics**

*Nura Salhadar*

Introduction: As the number of people living with dementia is increasing worldwide, there is an urgent need to understand the physiopathology of dementia syndromes. Among the most important preventable risk factors, treatment of vascular risk factors such as high blood pressure (BP) decreases the risk of Alzheimer's disease and related dementias (ADRD). Recent evidence suggests that examining BP variability provides additional physiopathological and predictive information above the mean BP level, especially excessive BP variability. However, studies examining the relationship between 24-h BP variability and ADRD are limited, and evidence of the association with dementia has not been documented. Therefore, we aimed in this study to assess the association of 24-h ambulatory BP variability with brain imaging and cognitive markers of ADRD.
Methods: A cross-sectional observational study was conducted using a subset of 420 individuals from the Maracaibo Aging Study aged 40 years (women, 73.2%; mean age, 57
years). Study participants underwent brain MRI scanning, and 24-h ambulatory BP measures were collected. Markers of ADRD included 1) cerebral small vessel disease (CSVD, defined as white matter hyperintensities, presence of lacunes, cerebral microbleeds, and enlarged perivascular spaces, and hippocampal volume), 2) cognitive functioning addressed with the mini-mental state exam (EMEMS), and 3) diagnose of dementia. 24-h ambulatory BP variability was studied as the average real variability index. Linear and logistic regression models were used to analyze the association between 24-h BP and ADRD.
Results: The mean age was 57.1±11.8 years old and 73.2% were women (n=303). In adjusted analysis, the each unit increase in the 24-h systolic BP variability was significantly associated with lower hippocampus volume ($\beta$, -0.036; 95% confidence interval [CI], -0.064, -0.008, $P$=0.011), greater white matter hyper intensities volume ($\beta$, 0.026; 95% CI, 0.008, 0.044; $P$=0.006), lower cognitive scores ($\beta$, -0.370; 95% CI, -0.729, -0.011; $P$=0.044), greater presence of lacunes (Odds ratios [OR], 1.38; 95% CI, 1.10, 1.71; $P$=0.004), enlarged perivascular spaces (OR,1.34; 95% CI, 1.08, 1.67; $P$=0.007), and dementia (OR, 1.41; 95% CI, 1.07, 1.85; $P$=0.014). 24-hour diastolic blood pressure variability was only significantly associated with lacunes (OR, 1.42; 95% CI, 1.06, 1.90; $P$=0.017). In exploratory analysis, we found that neither daytime nor nighttime variability in BP significantly relate with ADRD.
Conclusions: Excessive 24-h BP variability associates with ADRD independently of the mean BP level.
Understanding the physiological mechanisms explaining the relationship between excessive 24-h BP variability and ADRD may be clinically relevant in the prevention of ADRDs.

**Correlation Between Lecture Engagement and Academic Performance in the UTRGV School of Medicine**

*Chloe Harris*
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Introduction
The landscape of medical education has witnessed significant changes in recent years, marked by a decline in both in-person lecture attendance and online lecture viewership. This trend is particularly notable in the post-COVID-19 era and raises important questions about the relationship between lecture engagement and academic performance among medical students. Our study seeks to investigate the correlation between lecture viewership and course performance, with a specific focus on the unique context of the UTRGV School of Medicine. Our primary objectives were to (1) evaluate the correlation between lecture viewership and academic performance and (2) examine the relationship between lecture engagement and academic outcomes based on the medical school year (MS1 and MS2). We hypothesized that performance would be related to lecture engagement in a discipline-related manner. Our findings aim to provide insights into the effectiveness of lecture engagement as a learning method and its potential influence on academic success in the unique context of the UTRGV School of Medicine. The results may serve as valuable guidance for administrative decisions regarding the optimization of lecture formats to better meet the needs of medical students.

Methodology
Lecture engagement was evaluated by analyzing viewing data from Panopto, the UTRGV learning management system capture system. The study focused on the class of 2026 in the 2023 Renal and Male Reproduction (RMR) module. De-identified viewing data from the module was systematically categorized by discipline and week. We evaluated relationships with viewing data to corresponding performance overall and by discipline. Statistical analysis was conducted using SPSS.
Results
Preliminary data from the Renal and Male Reproduction module suggests that total minutes of lecture viewing had a negative trend in relation to end of module performance (R=0.0135; slope = -0.2874). Notably, students performing above 90% were identified in both students who watched a substantial number of lectures and those with minimal viewership. However, a positive trend was observed between online lecture engagement and performance on the quizzes, particularly in quiz 4 (R=0.0585, slope=87.575).
Conclusions
In summary, our findings suggest that online lecture engagement correlates to a trend in increased quiz grades but decreased performance on the end-of-module exam. We attribute this pattern to the nature of assessments, with quizzes being professor-written and end-of-module exam questions sourced from an NBME standardized question bank. Also, it is worth noting that the higher scores associated with decreased lecture watching may point to active, in-class participation. Therefore, this negative trend may indicate a potential benefit of in-person lecture attendance and engagement. Moreover, reduced lecture engagement may suggest that those students could be utilizing self-study and alternative means of learning to fulfill course outcomes.

**Development of an Experiential Learning Lab Activity on Skeletal Muscle Physiology in Undergraduate MD pre-Clerkship Curriculum**
*Meyer Maddox*

Background: Experiential learning is an important part of the medical education curriculum. Due to the clinical relevance of skeletal muscle strength in evaluating patients' complaints of muscle weakness or imbalance, skeletal muscle physiology is an important concept with extensive potential for experiential learning opportunities. Our goal was to establish an experiential learning skeletal muscle physiology lab activity that would improve undergraduate MD pre-clerkship students' skill to collect muscle force measurements using a hand-held dynamometer (HHD) and understanding of core physiological concepts.
Methods: As part of the Musculoskeletal and Dermatology (MSKD) Module in the undergraduate MD pre-clerkship curriculum, we developed a lab activity where fifty-five Year 2 students performed two experiments using an HHD: (1) The Elbow Flexion Experiment, and (2) The Fatigue Experiment. The Elbow Flexion Experiment was used to obtain measurements of force produced by the bicep muscles at 6 different angles, and (2) the Fatigue Experiment was used to determine the effect of fatigue on hand muscle strength. Students' understanding of the activity's learning objectives was evaluated using a two-prong approach. First, each student completed a laboratory session write-up assignment explaining how collected results and observations were related to muscle physiological principles. Second, students completed a survey assessing their self-reported understanding of the activity's concepts.
Results: The response rate of participants were 100% and 55.76% in 2022 and 2023, respectively. Demographic data revealed that majority of student participants were Hispanic/Latino (50.46%) followed by White/Caucasian (18.69%) and Asian (11.21%). Furthermore, male and female students were 55.14% and 44.85%, respectively. Post-activity survey data demonstrated that students had the ability to utilize HHD to collect muscle force measurement and understood the muscle length-tension relationship (98.5% agree/strongly agree rate). Our laboratory design was validated as collected data demonstrated a significant decrease in pinch strength post-exercise and significant changes in overall strength following a quadratic curve with arm angle degrees.
Conclusions: The activity enhanced medical students' understanding of skeletal muscle length-tension relationships, evidenced by students' positive survey responses. Given that knowledge of skeletal muscle physiology spans various sectors of clinical practice, this activity may be applicable in the training of other healthcare professionals as well.

**Title: Development of Solitary Keratoacanthoma from a Cutaneous Wart**
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Background
Common cutaneous warts, referred to in medicine as verrucae vulgaris, are proliferative lesions caused by human papillomavirus. These lesions are mostly benign and usually resolve without incident, except in the case of the patient mentioned in this report. Our patient developed a solitary keratoacanthoma, currently accepted as a clinical variant of squamous cell carcinoma, as a result of several risk factors and traumatic exposure. The current literature does not have an established association of HPV with solitary keratoacanthomas. This case report explores the presentation and pathogenesis of solitary keratoacanthomas within the setting of HPV.

Case Presentation
48-year-old Caucasian female presents to the family medicine clinic regarding a “bumpy lesion” on her right dorsal hand onset one month ago. She mentioned that she previously had a 5 mm wart in that location that was unchanged for several years. Two months ago, she “sprayed it with some Biofreeze spray” hoping that the wart would “dry up and fall off”. She reported that the spray caused burning pain and the wart formed a blister but did not fall off. Over the past month, she reported the wart transformed into the presenting lesion and added that she “bumped it a few times”, causing it to bleed and scab over. She admitted spending considerable time outdoors without adequate sunscreen use. Additional history included x-rays of right hand for history of right wrist carpal tunnel syndrome. She denied any pain to her right hand, wrist or fingers, pruritis, swelling, rash, sensory/motor/neurological changes. Physical exam revealed a 1 cm x 1 cm x 0.7 cm circular, crateriform nodular lesion on the dorsal surface of the third proximal metacarpal with a central keratinous filling and pink, indurated, hyperplastic borders. Evidence of solar lentigines noted on surrounding skin. Based on the presentation of the lesion, course of lesion progression, and associated history of the patient, a diagnosis of solitary keratoacanthoma was made. A shave biopsy was performed in the clinic, and the pathology report confirmed the diagnosis. However, the lesion demonstrated regrowth with a with continued pink, hyperplastic growth 1 cm x 1 cm and 0.3 cm above the skin surface. The patient was referred to dermatology for Moh’s procedure and further consultation.
Conclusion
This middle-aged, lightly-pigmented patient presented with a long-term cutaneous wart on a location previously exposed to radiation and chronic sun that underwent transformation into a solitary keratoacanthoma after being subjected to both mechanical and chemical trauma. This case is instanced as another example of keratoacanthomas occurring in the setting of HPV. A dual-insult driven etiology for the pathogenesis of sKA fits well in the context of our patient’s symptom presentation. It must be emphasized that the scope of skilled dermatologic intervention must be expanded to family medicine doctors and general practitioners, especially in underserved areas where specialties such as dermatology can be expensive with long wait times. Such interventions in the field of dermatology and oncology eliminate health disparity and adverse outcomes in our communities and promote overall health equity in the greater RGV.

**Embolic Protection during Radical Nephrectomy with Inferior Vena Thrombectomy Using the Protrieve Sheath**
*Mia Schmolze*

Renal Cell Carcinoma (RCC) is complicated by inferior vena cava (IVC) involvement in 4-10% of cases with intraoperative tumor embolization associated with 60-75% mortality. Currently, there is no high-level evidence demonstrating which surgical techniques provide optimal outcomes.
The Protrieve SheathTM (Inari Medical®) has been used in the vascular setting for complex deep vein thrombosis(DVT) and non-tumor IVC thrombectomy cases. We aim to describe its first use in the setting of RCC with IVC thrombectomy to help prevent intraoperative tumor embolization.
The Protrieve SheathTM successfully provided potentially life-saving protection from clot embolization when performing radical nephrectomy with IVC thrombectomy.

**Establishing the First Student-Run Clinic to Provide Free Health Care to a South Texas Colonia**
*Andrew Callan, John Cauba*

Purpose: The purpose of the University of Texas Rio Grande Valley School of Medicine Student Run Clinic is to bridge the healthcare gap in the local colonias by providing excellent, compassionate primary care to all who enter our doors, free of charge. We also work to connect our patients to low-cost, high-quality services in the area like women’s health visits, appointments with social workers, and dental services. Additionally, we offer medical students’ opportunities from their first year to serve an underserved population and get hands-on experience with patients.
Many patients in colonias face challenges like limited transportation options and a lack of awareness about affordable healthcare services, which can prevent them from accessing primary care. To address these issues, we chose to establish our student run clinic in 2016 at a community center located in Las Penitas, a location that is accessible to the target population.
Description: Patients first get their vitals and blood glucose taken followed by bloodwork. HbA1c is checked every three months and a lipid panel is done yearly. The patient is then assigned to an examination room where a team of student volunteers conduct the patient interview and perform relevant physical exams briefly presenting to the attending physician and identifying a care plan. The team then discusses the diagnosis and treatment with the patient. The goal is to have the patient able to explain back to the care team about their disease management by the time of discharge.
Under the supervision of a nurse practitioner, students are taught decision-making skills for performing lab draws. Regular lab work is crucial for managing chronic conditions like diabetes mellitus and hypertension.
We make it a point to ensure the privacy of all our patients, as many are wary of seeking medical care in fear of being deported because of their undocumented status. Legal residency status is not a question we ask our patients, and we encourage patients to inform their friends and family that we are a space for all community members.

Partners: We are supported our nurse practitioner who allows us to do routine blood work. Physicians associated with the school of medicine regularly volunteer their time to see patients with students and teach us valuable skills and medical knowledge. We work with the community center staff to schedule clinic dates, reach out to patients, manage patient paperwork, and facilitate our clinic days. Finally, our patients, the residents of Penitas and the neighboring areas, are also important stakeholders in our clinic.
Looking Ahead: The clinic has seen amazing patient outcomes, especially seeing patients able to get affordable management of their chronic conditions like diabetes and hypertension. As the clinic expands, we envision going from monthly clinics to every two weeks. We also are currently working on moving to electronic medical records, so patient information can be logged and tracked much more efficiently. We would also like to use the patient data from our clinic to analyze so we can identify trends and potentially improve our methods of care.

**Examining the Matrix: A Case of Anti-Nuclear Matrix Protein 2 (NXP-2) PositiveDermatomyositisAuthors:Victoria Cuello, Ramiro Oquita, Emilia Dulgheru MD *University of Texas Rio Grande Valley School of Medicine***
*Ramiro Oquita*

Background: Dermatomyositis is a disease characterized by proximal muscle weakness, elevated muscle enzymes and cutaneous skin findings including heliotrope rash, periungual erythema, Gottron's papules/sign and shawl-sign. Myositis-specific autoantibodies (MSA) can be used to predict disease manifestations, response to therapy and prognosis. Specifically, patients with dermatomyositis with positive anti-nuclear matrix protein (NXP-2) typically present with classical skin findings, subcutaneous edema, profound muscle weakness, severe dysphagia and hypophonia.
Case Presentation: A 21-year-old Hispanic lady presented to the hospital for severe muscle weakness. She reported soreness in her thighs for three months prior to presentation followed by arm soreness. She also developed a facial and left neck rash along with complaints of dysphagia and hypophonia. She denied joint pain or constitutional symptoms. Exam was notable for marked edema of upper and lower extremities, malar erythema not sparing the nasolabial folds, heliotrope rash, and a crusting lesion on the left breast that was previously reported as necrotic. Muscle strength was diminished in the proximal aspect of extremities; patient was unable to stand from a seated position. Her initial CPK was 14000. In addition, AST and ALT were elevated and ANA was positive with a nuclear speckled pattern. Chest CT was remarkable for a focal opacity in the posterior right upper lobe, most likely focal aspiration pneumonia for which the patient was placed on antibiotics. She was initially managed with vigorous IV fluids for suspected rhabdomyolysis, and then subsequently with oral prednisone with improvement of her CPK values and modest improvement of her muscle weakness. The 11 antibody myositis panel was positive for the NXP-2 antibody. Due to the patient's presentation of profound muscle weakness and suggestive rash, in addition to high titer myositis specific antibodies, she was diagnosed with dermatomyositis and started on pulse IV steroids, intravenous immunoglobins and azathioprine. In the following days, patient was able to ambulate and CPK, aldolase and liver function tests were down trending.
Conclusions: Myositis specific antibodies are a group of antibodies that have been associated with idiopathic inflammatory myopathies (IIM) and can be utilized in the diagnosis, assessment, management and prognosis of IIM. These antibodies are generally mutually exclusive and may help in characterization of a phenotypic presentation. Furthermore, MSA may be valuable in targeted therapies. Our patient had high titer NXP2 antibodies. These antibodies are more common in the juvenile form of dermatomyositis. Anti-NXP2 autoantibodies are found to confer a risk of developing five clinical characteristics: peripheral edema, muscle weakness, myalgia, dysphagia, and a reduced risk of interstitial lung disease.

**Gene by Environment Interaction: The Social Determinants of Health and Depression**
*Sowmya Duddu*

Background: Social Determinants of Health (SDoH) influence health through psychological, social, environmental, and cultural domains according to the psychosocial-cultural model of health. This report provides evidence of the intricate relationship between genetics, depression, and the Social Determinants of Health (SDoH). We applied a joint interaction model to account for G×SDoH interaction in the face of depression to establish if both types of interactions are important and independent of one another in the setting of depression. We estimated the corresponding genetic effect and extracted envophenotypes using Best Linear Unbiased Prediction to remove the influence of genetic variation on expression. Using the resultant envophenotypes, we used a genome-wide scan of RNA sequence data to identify transcripts jointly associated with SDoH, and depression. This research aims to understand the complex interplay of genes, SDoH, and depression in Mexican Americans.
Methods: We employed a cross-sectional family-based design of 525 participants belonging to large Mexican-American families, highlighting the heritability of depression (as measured by the Beck Depression Inventory-II) and SDoH (as measured by the Social Determinants of Health evaluations determined by The Centers for Medicare and Medicaid Services (CMS) Accountable Health Communities Health-Related Social Needs Screening Tool (AHC HRSN). Using statistical inference models for the phenotypic expression of depression, we estimated the corresponding genetic effect and extracted envophenotypes using Best Linear Unbiased Prediction to remove the influence of genetic variation on expression. Using the resultant envophenotypes, we used a genome-wide scan of RNA sequence data to identify transcripts jointly associated with depression and Social Determinants of Health.

Results: We present the observed significant associations between environmentally determined gene expression with Social Determinants of Health and depression. By controlling genetic factors, we identified these expression phenotypes as potentially involved in the gene-environmental axis affecting depression and SdoH. We also established that there are Gene-by SDoH interactions, which are independent.
Conclusions: Our findings highlight the importance of considering gene-environmental interactions in depression and the Social Determinants of Health. The shared genetic associations warrant further investigation as potential targets for therapeutic interventions and predictive models in managing depression, Social Determinants of Health in Mexican Americans. Future longitudinal studies in diverse populations will enhance our understanding of these complex gene- environmental interactions and their implications for precision medicine.

**Honey targets ribosome biogenesis process in human pancreatic cancer cells to inhibit their growth and metastatic phenotypes.**
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ABSTRACT
Background: Pancreatic cancer (PanCa) is the fourth deadliest cancer worldwide and is expected to become the second deadliest cancer by 2030. In the USA, the National Cancer Institute put forth a grim prediction stating that there will be 64,050 new cases in 2023 alone and about 50,000 of these patients will die. Existing therapeutic regimens against PanCa are not that effective and show unacceptable toxicities. Therefore, developing highly effective new agents with less toxicity is urgently required, which could be used as a monotherapy or as an adjuvant to treat PanCa patients. Honey is known for its tremendous health benefits and has been used in various traditional medicines. Several studies have defined honey to be cardioprotective, neuroprotective, ant-inflammatory, and immunomodulatory. In addition to these advantages, honey also possesses anti-cancer properties. However, no study has explored its effect against PanCa. In this study, we evaluated the anti-cancer potential of honey and its molecular mechanisms against PanCa.
Methods: MTT assay and colony formation assays were performed to determine the effect of honey on proliferation of human PanCa cells (AsPc-1 and MiaPaCa-2). Wound healing assay was performed to evaluate the antimigratory potential of honey against PanCa cells. Western blot and qPCR assays were performed to determine the effect of honey on the regulation of ribosome biogenesis, cell proliferation, and apoptosis in PanCa cells.
Results: Ribosome biogenesis is dysregulated in most cancer types, which results in aggressive metastatic phenotypes seen in cancer cells. We observed that the transcription factor UBTF and other ribosome biogenesis components were aberrantly overexpressed in pancreatic tumor tissues as compared to normal pancreatic tissues. These associations are linked to aggressive phenotypes seen in PanCa cells. Interestingly, we observed that honey treatment significantly suppressed ribosome biogenesis in PanCa cells as observed by significant decrease in UBTF and 5'-ETS expression. Honey treatment markedly inhibited the expression of RPA194, a catalytic unit of RNA polymerase I, and nucleolin in both AsPC-1 and MiaPaCa-2 cells. MTT analysis indicated that honey exerted dose-dependent cellular cytotoxicity in AsPc-1 and MiaPaCa-2 cells, with IC50 values 45.2 and 30.3 mg/ml, respectively. In addition to its potential cytotoxicity, honey treatment clearly interfered with the wound healing and clonogenic ability of these PanCa cells as demonstrated by wound healing and colony formation assays. We also observed that honey treatment induced apoptosis in AsPC-1 and MiaPaCa-2 cells, which was confirmed by the the increase of the population of Annexin V positive cells and cleavage of PARP protein. A decrease in expression of Bcl-2 and p53 (truncated in AsPc-1 and mutated in MiaPaCa-2) also indicated that these cancer cells were undergoing apoptosis. Conclusions: Overall, our results demonstrated for the first time that honey has potential to induce apoptosis and prevent pancreatic cancer cell growth through modulation of ribosome biogenesis process. It implies that honey could be used as a natural remedy to prevent human pancreatic cancer and utilized as an adjuvant in ongoing chemo/-immunotherapy regimens. Further detailed studies using appropriate pre-clinical mouse models of PanCa will be warranted to establish its anti-cancer potential for the treatment of PanCa.

**Hypermetabolic lymphadenopathy following the administration of COVID-19 vaccine and immunotherapy in a lung cancer patient: a case report**
*Nathaniel Alvarez*

Abstract
Background: Given the current climate of the pandemic, lung cancer patients are especially vulnerable to complica- tions from severe acute respiratory syndrome coronavirus 2 infection. As a high-risk population group, these patients are strongly advised to receive coronavirus disease 2019 vaccination in accordance with Center for Disease Control and Prevention guidelines to minimize morbidity and mortality. In recent years, immunotherapy has taken a preemi- nent role in the treatment of non-small cell lung cancer with dramatic improvement in overall survival. Reactive lym- phadenopathy following the administration of a coronavirus disease 2019 vaccination can confound the radiographic interpretation of positron emission tomography–computed tomography or computed tomography scans from lung cancer patients receiving immunotherapy.

Case presentation: Here, we present a case of a 61-year-old Caucasian female and former smoker who developed cervical, hilar, supraclavicular, mediastinal, and left retroauricular lymphadenopathy following her coronavirus dis- ease 2019 booster vaccination. At the time, she had been receiving long-term immunotherapy for the treatment of advanced lung adenocarcinoma. Biopsy was pursued owing to concerns of treatment failure and confirmed recurrent malignancy.
Conclusion: This case report highlights the importance of lymph node biopsies in lung cancer patients who present with contralateral lymphadenopathy following coronavirus disease 2019 vaccination to rule out tumor recurrence in this deserving patient population.
Keywords: COVID-19, COVID-19 vaccine, Lung cancer, Lymphadenopathy, Case report

**Inflammatory Breast Carcinoma in the Rio Grande Valley: A Case Report**
*Miguel Lopez, Maria Villegas*

Inflammatory breast carcinoma (IBC) is a rare and aggressive subtype of invasive breast cancer found in a small percentage of patients in the United States.The Rio Grande Valley is a region of Texas with pockets of low socioeconomic status and increased rates of obesity, a large risk factor for IBC. The cutaneous presentation of IBC is a tender, erythematous patch or plaque that surrounds hair follicles (referred to as peau d'orange) and is frequently edematous. Diagnosis requires a high clinical index of suspicion supported by a thorough patient history and pathology consistent with invasive carcinoma. This case study discusses a 59-year-old overweight female patient who presented with a sudden eruption on her left breast. Clinical presentation was suspicious for inflammatory breast carcinoma, and the diagnosis was later confirmed via biopsy. While rare, IBC has a high mortality rate and a higher likelihood of metastasis if not detected and treated in early stages of a patient's presentation.

**Influence of Neurotoxin Load on Parkinson's Disease Pathophysiology**
*Abhishekh Pokhrel, Daniel Salinas, Nawaz Hack, Kelsey Baker*

Background: Parkinson's disease (PD) is a debilitating neurodegenerative disorder impacting movement, mood, and cognition. Among those affected, veterans, due to their occupational exposures, are particularly susceptible, contributing to over 110,000 PD cases in the United States. Studies have largely attributed this increased prevalence among veterans to environmental neurotoxins such as Agent Orange, MPTP, and 6-OHDA. However, it remains unclear how neurotoxin exposure load influences biological mechanisms in PD. This study aims to elucidate the influence of neurotoxin load on PD-associated molecular changes, neuroplasticity, neurodegeneration, and cognitive and motor function within a clinical population of the Rio Grande Valley region in Texas.
Methods: Here we conducted a longitudinal, repeated-measures clinical observational study in PD patients with self-reported exposure to neurotoxins and sex-, age-, and occupation-matched healthy controls. Participants underwent comprehensive evaluations at two-time points—enrollment and 2 months—enabling the exploration of neurotoxin-related influences on disease progression. After enrollment, subjects underwent subjective surveys to assess prior neurotoxin exposure, including pesticides, Agent Orange, heavy metals, and other occupational or environmental toxins, and were evaluated for neurotoxins via blood samples. At all time points, we collected MRI and diffusion-weighted imaging (DWI) data to measure neurodegeneration, neurophysiological assessments using transcranial magnetic stimulation (TMS) to measure corticospinal excitability and neuroplasticity, and comprehensive functional evaluations encompassing motor function via MDS-UPDRS and HY scales, cognitive status via SLUMS, handedness via Edinburgh Handedness Inventory, gait analysis via Zeno™ Walkway Gait Analysis System, and arm/hand functionality via Bionik InMotion2 Arm/Hand robot.
Results and Discussion: To date, we have enrolled 7 participants with self-reported exposure to neurotoxins and 2 healthy controls. Enrolled PD subjects' ages ranged from 66 to 87, with 5/7 reporting prior agricultural exposure to Pesticides (Methylbromide / Organochlorides) and 1 PD subject reporting exposure to engine exhaust from aviation fuel. One enrolled patient was withdrawn from the study due to an inability to obtain an MRI. Thus, we evaluated the 6 PD enrolled subjects. Initial motor UPDRS scoring revealed 4/6 patients with mild, 1/6 patients with moderate, and 1/6 patients with severe motor impairment of daily living. In addition, 5/6 patients exhibited mild and 1/6 patients revealed moderate impairment during motor examinations. Pending blood labs, the degree of influence neurotoxin blood has on motor and cognitive function remains to be seen. Data from prior PET studies have shown lower dopamine transporter uptake in all basal ganglia areas, as well as a higher asymmetry index in PD patients exposed to Agent Orange compared to PD patients with no exposure. In addition, PD symptoms have been shown to emerge more on the dominant hand side. Therefore, we expect increased cognitive and motor burden, especially on the dominant side, with increased neurotoxic load.
Conclusion: This research holds considerable promise for advancing our understanding of PD, particularly in the context of neurotoxin-related etiology. By deciphering the molecular pathways influenced by neurotoxin exposure, the study sets the stage for targeted therapeutic interventions and potentially less invasive treatments, thereby improving the outlook for individuals grappling with neurotoxin-related PD.

**Investigation of the efficacy of local anesthetics for perioperative peripheral nerve blocks in patients with and without diabetes: A Retrospective Chart Review**

*Andrew Kolodziej*

Background: Peripheral nerve neuralgia is a common manifestation of diabetes and can cause patients to have severe episodes of pain. Although pharmacologic treatment with antineuropathic drugs are first line, they often are not effective and can warrant further treatment with peripheral nerve blocks. Further studies support that peripheral nerve blocks should be considered as the first option for anesthesia for lower limb surgery in diabetic patients. The goal of this study is to evaluate local anesthetic agent regimens in the Rio Grande Valley for peripheral nerve blocks in diabetic patients and determine post operative effectiveness and complications.
Methods: We conducted a retrospective chart review within UT Health RGV. We evaluated medical charts had current procedural terminology (CPT) codes for perioperative peripheral nerve blocks. Additionally, we cross referenced the CPT codes with ICD-10 codes for diabetes to compare patients with diabetes and without diabetes. For all referenced medical charts, we evaluated sex, age, ethnicity, past medical history, diagnosed conditions requiring nerve blocks, and post-operative follow up notes on pain and paresthesias. Medical charts were excluded from the analysis that were duplicative, incomplete, or misclassified. Data was analyzed in SPSS to determine if there were significant advantages for certain anesthetic agents or certain complications that arose more frequently with certain anesthetic agents.
Results/Discussion: In our study, we found that most nerve blocks were offered to non-diabetics compared to patients with diabetes. Only 17 of the 100 patients that received a nerve block had diabetes. Additionally, we found that patients with diabetes had significantly worse outcomes at their follow-up visits. Patients with diabetes more often reported pain and paresthesias in the area where they received the nerve block in comparison to patients without diabetes. While diabetic patients have neural damage that non-diabetic patients do not, our preliminary results suggest nerve blocks are not as therapeutic as literature indicates. Our plans for future work include analyzing the outcomes at follow-up visits by comparing anesthetic agents used for blocks, which nerves are blocked, and the differences in outcomes to determine how to best prevent pain in patients with diabetes undergoing procedures.

**Leveraging a Human-Centered Design Framework to Improve Longitudinal Care at a Student Free Clinic : A Quality Improvement Initiative**

*Rosa Martinez, Alexis de Montfort Shepherd, Sahana Prabhu, Krishna Hariprasad, Sanika Mhatre*

Background: The CD Doyle (CDD) Clinic is a student-run free clinic with a mission to provide high-quality equitable care to those in need, regardless of their background. Since 2020, our clinic has served residents at the Esperanza community, a state-sanctioned encampment for people experiencing homelessness in Austin. The majority of the patients that we serve navigate complex medical and psychosocial conditions. Our clinic operates as an ambulatory care center, primarily addressing acute care concerns. Recognizing our limits as a free clinic, we collaborate alongside community partners, such as Travis County's CommUnityCare (CUC) Mobile Health Team (MHT), to better serve our patients with ongoing chronic or preventative care needs. Coordination of these handoffs primarily takes place through email updates to patients' on-site case managers and the MHT, mid-week follow-up calls to patients, and patient-centered "health passports" outlining pertinent information regarding follow-up care. However, insufficient standardized operational procedures and a lack of closed-loop communication about what happens after a patient concludes their CDD visit have resulted in continuing care gaps, recommendations that do not consider patients' ability to access care, and increased patient revisitation rates.
Methods: Our approach is rooted in a variety of human-centered design methodologies. Our goal is to design a stakeholder/systems journey map to illuminate the breadth of healthcare-related interactions our patients navigate starting from the conclusion of their CDD clinic visit. Then, interview key stakeholders (patient's case managers, on-site care professionals) regarding transitions of care from CDD to longitudinal care avenues (including medication access, specialty care, and preventative care). Additional opportunities for residents to share overall perceptions of health were conducted through a 9-item survey. This is further supplemented with a retrospective chart review to characterize the number of CDD visits and referrals by specialty.
Results: In our retrospective analysis of July through October 2023, 32 patients visited CUC within 3 months of their CDD visit. CDD had referred to MHT in 17 of the 32 cases for further follow-up. Stakeholder interviews revealed several breakdowns in our follow up procedures. A survey of Esperanza residents demonstrated an understanding of health-related screening and the value of primary care. Based on our data collection, we created a map to capture the diverse interactions and flows of care among stakeholders along with relevant gaps that our patients may face.
Conclusions: Human-centered design places humans first by embracing co-creation with the community and intentional innovation that acknowledges systems, political contexts, and power dynamics. Understanding patients' entire care journey through this lens is critical to fostering trust and better health outcomes; our CDD patients navigate complex psychosocial conditions and an intricate network of care while also experiencing homelessness or a recent transition to becoming sheltered.

As Esperanza Community has grown, its health needs have stabilized, a theme echoed in provider interviews as well as patient-reported perceptions. Points of improvement for the CDD clinic flow include standardizing weekly operations, enhancing communication with partners, and critically reflecting on policy implications and the broader healthcare landscape when providing care recommendations.

**Modern Neurosurgical Techniques for Surgical Excision of Neoplasms**

*Kevin Garcia Valdez, Sonya Bhatia, Natasha Bell, Karen Murambadoro, Victoria Elizondo, Fernando Cisneros, Kory Punch, Dr. Alan Francis*

Background: Neurosurgical excision of brain tumors is still regarded as the first line treatment for multiple neoplasms, including meningiomas, gliomas, and metastatic tumors. Advances in technology have allowed neurosurgeons to utilize various approaches that significantly reduce mortality and surgical morbidity. However, the high risk of neurological deficit and tumor recurrence remains. Here, we analyze three neurosurgical approaches: craniotomy, microsurgery with tubular retraction, and tumor ablation. While craniotomy remains as the first line treatment for most brain tumors, we hypothesize that microsurgery and ablation will show greater success of tumor removal with fewer complications.
Methods: Published literature was reviewed from PubMed using search terms “neurosurgical techniques,” “brain neoplasm,” “tubular retractor,” “craniotomy,” “stereotactic radiosurgery,” or “ablation” in varying combinations with Boolean commands. Only papers that provided relevant data on extent of resection (EOR) and perioperative or postoperative neurological complications were included. Articles were not screened for identification of brain tumor or patient demographics.
Results: Ten articles that complied with the inclusion criteria were incorporated in the literary review. Craniotomy achieved gross total resection (GTR) in 54% across all studies, with a complication average of 12.1%. The use of ablation showed GTR in 20% of cases and a 20% complication average. Tubular retractors used for the microsurgery approach yielded a 77.1% and 7.5% GTR and morbidity average respectively. The mentioned results are a compilation of data from all types of tumors. Microsurgery showed a greater reduction in morbidities when used for deep-seated tumors, as compared to craniotomy.
Conclusion: Brain neoplasms portray a significant challenge for neurosurgeons because of the brain’s variety of tumor etiologies, location, and extreme sensitivity; these reasons prohibit a singular method to be established. Microsurgery with tubular retraction, when compared to craniotomy and tumor ablation, demonstrated the highest rate of EOR with the least resulting neurological deficits or surgical complications. Craniotomy and microsurgery had comparable EOR, but craniotomy had a higher percentage of morbidities. Our results suggest that although advances in technology allow for greater surgical success with reduced morbidities, craniotomy remains a suitable choice for brain neoplasm resection. It is important to note that multiple strategies for craniotomy are performed and there are several modalities of tumor ablation technology, creating a challenge in accurately comparing each approach. It is also worth mentioning that in all cases, the rate of morbidity and mortality correlated to the tumor’s size, location, and etiology (i.e., primary tumor vs. metastasis), and the patient’s age

**"Neurological Comorbidities in Hispanic Skin Cancer Patients in South Texas: A Five-Year Retrospective Study"**

*Authors: Jared Hensley, Russell Wiggins, Blake Martin, Hunter Butler, Kevin Garcia Valdez, Dr. Kelsey Baker*

Background: Skin cancer is highly prevalent in the South Texas region due to a combination of increased UVB exposure, occupational hazards, and lack of access to preventative screening. Unfortunately, several studies have suggested that skin cancer diagnosis may be linked with an increased risk of neurodegenerative conditions, such as Alzheimer’s Disease. For example, it was recently suggested that amyloid beta plaques may have a role in melanoma metastasis. Here, we seek to evaluate the role of skin cancer incidence and the degree and severity of neurological comorbidities, with a particular focus on the Hispanic patient population. We anticipate that a unique relationship will exist in the Hispanic patient population, where a history of skin cancer may be related to a more severe or early-onset neurodegenerative disease.
Method: We conducted a comprehensive retrospective chart review of UT Health RGV medical records for all common skin cancers dating back five years. We included common skin cancers within ICD-10 codes C44, C43, and C4A for our analysis. This classification includes basal cell carcinoma, squamous cell carcinoma, Merkel cell carcinoma, and malignant melanoma. Inclusion criteria for the study included patients who self-reported as Hispanic or Latino/Spanish. We excluded charts that were duplicative or needed more information. After screening, we reviewed 838 unique patient charts for accompanying neurological comorbidities. We examined neurological comorbidities classified by the following ICD-10 codes: Q85.0, G20, and G30. These classifications include neurofibromatosis type 1, type 2, schwannomatosis, Parkinson’s disease, and Alzheimer’s disease. Statistical analysis consisted of chi-square testing, correlation analysis, and survival analysis.
Results: Our analysis aimed to help elucidate potential relationships between common skin cancers and neurological comorbidities. Specifically, we evaluated the association between having skin cancer and neurological comorbidities, the strength and direction of the relationship between variables, and the time it takes to develop neurological conditions after the development of skin cancer. Our preliminary findings suggest a relationship will exist between the existence of our neurological comorbidities of interest and prior or concurrent skin cancer malignancies.

Conclusion: This project aims to elucidate the prevalence of neurological disorders in patients with current or prior cases of common skin cancers. Due to the geographical and socioeconomic nature of the Rio Grande Valley, our patient population is increasingly susceptible to various skin-related malignancies. With a lack of specialists and medical professionals as a whole, this issue is compounded, leading to adverse outcomes in the long term. If a relationship is shown, the long-term goal would be to promote the education and screening of neurological and skin conditions in the area and provide additional fodder for future research goals.

**New ways to improve dispersibility of nanotubes: Approaching from the formation of Silicon Nanoparticles by High Energy Reactive Ball Milling (HERM) in Polar Solvent**

*Yolanda Gutierrez*

This research aims to synthesize stable silicon nanoparticles using different molar ratios of N-Cyclohexyl-2-pyrrolidone (CHP) and Silicon to demonstrate if there is any significance towards the production of effective nanotubes. To determine this, the synthesized nanoparticles will be characterized by scanning electron microscopy (SEM), UV visible absorption spectroscopy, and photoluminescence spectroscopy (PL). Extensive research in nanomaterials has shown that nanotubes have become one of the best nominees to improve the world of science. Their outstanding mechanical, electrical/thermal properties, high tensile strength, and elasticity make them a good candidate to be implemented in various applications. Some applications may include: energy storage, air/water filtration, thermal conductivity, and biomedical applications. Despite their advantageous properties, nanotubes possess some inconveniences that limit their implementation in possible applications. One of the biggest drawbacks of nanotubes is their hydrophobicity, due to this, they tend to agglomerate when placed in water and organic solvents. An appropriate depression of nanotubes is indispensable for them to be able to retain their exciting properties. A common approach of dispersing nanotubes is by mechanical methods, where nanotubes are subjected to sonication and high shear mixing for large periods of time. The goal of this experiment is to develop a new environmentally friendly method that will successfully disperse nanotubes without the use of a reducing agent. This aim is a top-bottom synthesis of Silicon nanoparticles (SiNPs) that would be able to chemically interact with nanotubes and improve their limitations. High energy ball milling was used in order to modify the surface of Si and CHP, a hydrocarbon used as a ligand.

**Oxidative Stress, a culprit in cardiovascular diseases and Resveratrol as a potential therapeutic agent**

*Justin Heckman, Alyssa Sepulveda*

Background

Cardiovascular diseases (CVDs) are the leading cause of mortality worldwide. Oxidative Stress (OS) is one of the pathophysiological mechanisms of CVDs such as hypertension, atherosclerosis, myocardial infarction (MI), ischemia reperfusion (I/R) injury, and heart failure. Resveratrol, a natural polyphenol compound has cardioprotective effects acting via several molecular mechanisms including reduction of OS in cardiovascular system. Our goal was to provide an updated overview of the pathophysiological role of OS in CVDs. Furthermore, we explored the cardiovascular effect of resveratrol and its mechanism of actions involved, especially focusing on its role as an antioxidant molecule in CVDs.

Methods

We searched through 50-60 original research and review articles using online databases (PubMed, science direct, google scholar). Research papers relevant to our review study published in the last 20 years, mainly using papers published in the last 5 years. Figures were created using Adobe illustrator.

Results

It has been shown that OS cause endothelial dysfunction leading to the CVDs. Reactive Oxygen Species (ROS) may lead to endothelial dysfunction by decreasing Nitrous Oxide (NO) through oxidizing an important cofactor tetrahydrobiopterin to trihydrobiopterin radical. This oxidation reaction leads to endothelial nitric oxide synthase uncoupling and induces free radical and superoxide generation that might lead to a multitude of CVDs. Endothelial dysfunction may cause inflammation, through cell signaling by TNFa, Angiotensin-II, and Endothelin-1. Endothelin-1 causes a positive feedback loop of increasing OS and is a key factor in vascular inflammation which is central for foam cell production, and lipid accumulation on arterial walls known as atherosclerosis. Atherosclerotic plaque rupture and subsequent formation of occlusive thrombi may induce MI. MI can also be induced by endothelial disruption of NO production that leads to platelet aggregation occluding blood vessel. Paradoxically, the reperfusion of ischemic or infarcted myocardium can lead to further injury, called I/R injury, that is characterized by induction of ROS. Excessive ROS activates autophagy, apoptotic and inflammatory pathways through Beclin-1, NLRP3 and MAPKs, respectively. This increases proapoptotic protein Bax while decreasing anti-apoptotic protein Bcl-2 which causes cardiomyocyte death, leading to heart failure. Resveratrol has been found to have potential therapeutic effects in CVDs. The activation of SIRT1/ AMPK pathway helps regulate cardiomyocyte energy and cellular repair which contributes to the mitigation of hypertension by promoting vasodilation, reducing OS, and improving endothelial function. Inhibiting PI3K/Akt/mTOR prevents excessive vascular smooth muscle cell proliferation, a key factor in atherosclerosis development. Resveratrol's action in blocking TGF-β/Smad pathways may limit cardiac fibrosis, crucial in conditions like heart failure due to myocardial ischemia or MI-induced cardiac remodeling.

Additionally, activation of Nrf2 pathway by resveratrol promotes antioxidant enzyme production and inhibits NADPH oxidase, alleviating OS associated with ischemia-reperfusion injury and MI.
Conclusion
OS is shown to be involved in the pathogenesis of several CVDs such as hypertension, atherosclerosis, ischemia reperfusion, MI, and heart failure. A natural antioxidant compound resveratrol has been shown to have potential as a therapeutic agent, to treat these CVDs. The beneficial cardiovascular effects of resveratrol may involve several molecular pathways such as SIRT1/AMPK, PI3K/Akt/mTOR, TGF- β/Smad, and Nrf2.

**Quantifying incidence of early onset neurodegenerative disease post traumatic brain injury in the Rio Grande Valley, a retrospective chart review**
*Russell W. Wiggins, Jared Hensley, Hunter Butler, Blake Martin, Kevin Garcia Valdez, Chloe Harris, Kelsey Baker, PhD*

Background: Researchers have previously established evidence of early onset neurodegenerative disease (NDD) in certain patient populations post traumatic brain injury (TBI). The Rio Grande Valley (RGV) is a region in South Texas which has some of the largest health disparities in the United States. This retrospective chart review aims to quantify the difference in onset of NDD in patients with and without history of TBI within the RGV from that of the broader American population in order to determine the extent at which early onset NDD may be mitigated with appropriate intervention.
Methods: A retrospective chart review was conducted using electronic medical records (EMR) obtained from the UTRGV health system databases composed of multidisciplinary clinics and hospitals. A search query revealed 2686 unique patients that met our inclusion criteria for charts containing ICD-10 codes of S06.X or Z87.820 (TBI); and either G20, G23, G30, or G31-G35 (NDD). After review and curation, 594 charts met exclusion criteria for insufficient data, erroneous data entry, and redundant entry resulting in 2092 charts that met final inclusion criteria. Population coverage was determined by mapping clinic location via zip code and using corresponding census population data to reach a population estimate of 1,167,792. Patients with concomitant diagnoses of TBI and NDD were compared with patients with a history inclusive for only one of the pathologies by way of relative risk (RR) calculation.
Results: Data analysis suggests a RR of 3.42 favoring NDD development in TBI positive patients within the RGV (RR = 3.42, 95% CI 1.10 - 10.6 ). This risk ratio nominally exceeds that found in comparable American populations (older American veterans; RR: 1.57, 95% CI 1.35–1.83). Average patient age of initial encounter for NDD within the RGV was 73 ±1.72 compared to 64.3 ±27.35 for dually diagnosed TBI/NDD patients.
Conclusions: Trends exist in our current data which suggest an earlier onset of NDD in patients with a history of TBI compared to patients without TBI in the RGV. There also seems to be a greater relative risk for development of NDD in TBI positive patients within the RGV when compared to their broader American counterparts. Adoption of screening techniques aimed at identifying patients with history of TBI may lead to a timelier diagnosis and earlier initiation of treatment of NDD, reducing severity and burden of disease in the valley. In order to strengthen and establish significance of these observed trends, more patient EMRs must be identified which meet study criteria for review and continued analysis to clarify, discover, and strengthen the aforementioned relationships. Different methods can be adopted in the future to create a more robust and accurate data pool. We suggest employing TBI oriented survey questions and screening tools for appropriate patients.

**Reactive Oxygen Species in Acute Coronary Syndrome**
*Fernando Cisneros*

Background
Reactive oxygen species (ROS) play a dual role in biomedicine, acting as essential molecules in cellular metabolism and signaling while posing a threat to cellular components through oxidative damage. This poster explores the significance of ROS in various cardiovascular diseases, specifically hypertension, atherosclerosis, and myocardial infarction, all of which are associated with acute coronary syndrome. Correlations have linked ROS culprits, particularly upregulation of NADPH oxidase (NOX) 1 and NOX 2, with accelerated progression of atherosclerosis and acute coronary syndrome. Similar mechanisms of increased oxidative stress have also been associated with vasoconstriction and thrombosis. Furthermore, auto-phagosome function by lysosomes during cardiac arrest and return of spontaneous circulation (CA-ROSC) has proven to be impaired due to ROS dysregulating Beclin-1 and lysosomal-associated membrane protein 2 (LAMP2) following sudden cardiac events. Thus, minimizing levels of ROS can potentially prevent, reduce, and minimize myocardial injury. This understanding of ROS in biomedicine guides innovative therapies to restore redox balance and reduce the detrimental effects of oxidative stress.
Methods
We conducted an extensive search across more than 30 recent and relevant studies concerning ROS in cardiovascular diseases, utilizing various online databases such as PubMed, Google Scholar, and Web of Science. Our aim was to synthesize essential insights gleaned from the latest available research, thereby establishing a comprehensive framework that encapsulates the current knowledge regarding the role of ROS in cardiac disorders. All statistical analyses mentioned in the literature were carried out by the respective study authors.

Results
ROS are crucial in cell signaling, immune response, and gene expression, but excessive levels can lead to oxidative stress and cellular dysfunction. Oxidative stress can disrupt intracellular processes, including programmed cell death, downstream signaling, and cellular proliferation. Consequently, the regular functioning of tissues subject to oxidative stress is altered, enabling the development of diseases. In the pathogenesis of acute coronary syndrome, ROS are significant contributors, influencing hypertension, vascular remodeling, lipid peroxidation, thrombosis, vasoconstriction, and atherogenesis all of which contribute to the likelihood of acute coronary syndrome. ROS production modifies LDL which causes the formation of highly reactive lipid peroxyl radicals which may cause atherosclerosis. Increased ROS production in vascular smooth muscle cells cause vasoconstriction and impaired endothelial function also promoting the development of atherosclerosis. Furthermore, preceding events triggered by NOX isoforms enhance superoxide production, which interacts directly with nitric oxide (NO), leading to a decrease in both NO levels and a reduction in NO formation by inhibiting eNOS.
Conclusions
This research underscores the pivotal role of ROS in various cellular processes, including cell signaling, immune response, and gene expression. However, an excess of ROS can induce oxidative stress and disrupt essential intracellular functions, leading to the development of cardiac diseases and ultimately sudden cardiac arrest. Potential therapeutic interventions such as polyphenols and ROS scavengers offer promising avenues for mitigating oxidative stress by altering these physiologic mechanisms.

**Spontaneous Tumor Lysis Syndrome in a Patient with Recent Diagnosis of Multiple Myeloma — An Unusual Presentation**
*Taha Al Hassan*

Introduction: Tumor lysis syndrome (TLS) is an oncologic emergency that surfaces as a constellation of metabolic imbalances due to the rapid destruction of cancer cells and subsequent dissemination of their contents. However, a markedly rare subtype of TLS, known as spontaneous TLS (STLS), occurs without an evident trigger, such as cytotoxic therapy, and carries a similar mortality risk. It is paramount to recognize high-risk cases early and implement therapeutic measures to prevent complications of STLS.
Case Presentation: We present the case of a 74-year-old gentleman with a recent diagnosis of Kappa-restricted multiple myeloma who presented with worsening weakness, tremors, ataxia, anorexia, constipation, and unintentional weight loss over the past month. Initial workup revealed hypercalcemia, hyperuricemia, hyperphosphatemia, and new onset acute kidney injury, findings consistent with TLS based on the Cairo-Bishop Criteria; however, the patient was diagnosed with STLS as no trigger was identified. After a perusing diagnostic assessment, however, we were able to determine patient's and tumor's characteristics that places the patient at high risk for developing STLS.
Conclusion: Our manuscript deepens clinical knowledge on potential causes that predisposes patients to develop STLS. Additionally, we highlight the diagnostic assessment, management, complications, and relevant learning points for clinicians.

**The Influence of Head Trauma on Serum Calcium Levels: A Retrospective Chart Review**
*Hunter Butler*

Background
With almost 2 million Americans suffering from traumatic brain injury every year, there is extensive research suggesting many complications associated with the injury. Clinical research has suggested that concussion-like events can lead to hypoparathyroidism and decreased bone mass. However, a specific correlation between the loss of available blood calcium after presence of a traumatic brain injury has yet to be identified. In this study, we will explore the clinical correlations between traumatic brain injury and its potential association with serum calcium levels, in the unique and diverse population of the Rio Grande Valley.
Methods
We conducted a Retrospective Chart Review of UTRGV Health. We anticipate evaluating 15 medical charts. The medical charts were evaluated with the following ICD-10 codes in mind: S06.0, S06.0X0, S06.0X1, S06.0XA, S06.0X9, S02, and S07. Additionally, sex, ethnicity/demographics, and age were all analyzed independently to determine the effects of TBI on different demographics within the study population. ICD-10 concussion codes were cross-referenced with calcium serum levels derived by the metabolic blood panel data to determine if there is a correlation between the different criteria. Additional factors considered included: severity of the head trauma, time between the head trauma and calcium results, and relevant clinical pathologies the patients might possess. Within these parameters, 15 individuals were identified and evaluated. Average calcium was determined by taking the average of all available calcium tests within the Athena database (n=20,960).
Results/Discussion
We anticipate evaluating 15 medical charts. The average of these values is 9.227 mg/dL with a standard deviation of 0.375 mg/dL. This shows a notable decrease in the average which was determined to be 9.35 mg/dL. The range was 8.6 - 9.9 mg/dL. The mode was 9.6 mg/dL, and the median was 9. mg/dL. Additionally, calcium values were divided amongst ethnicity, age, and sex. After comparison of serum calcium numbers among qualifying individuals, the data indicates being an ethnic minority, middle aged, and female exacerbates the serum calcium level drop resulting from a traumatic brain injury.

Conclusion
This indicates that traumatic brain injury may be implicated in causing a lowered serum calcium in patients, implying there may be a clinically relevant link between TBI and decreased serum calcium levels. Changes in the serum calcium level vary depending on different demographics and ingroups that have been analyzed in the study, with being over 50, white, or a male reducing the drop in serum calcium the most. This decreased serum calcium may be indicated as a risk factor for a plethora of other bone related illnesses, including osteoporosis and bone fractures. The resultant decrease in serum calcium levels may lead to osteopenia, a condition that is implicated with bone fractures.

**The Link Between Depression and Bone Fractures in the Rio Grande Valley**

*Blake Martin*
Authors: Blake Martin, Hunter Butler, Russell Wiggins, Jared Hensley, Kevin Garcia Valdez, Chloe Harris, Dr. Kelsey Baker.

Abstract:
Background: The prevalence of depression has increased throughout the twenty-first century, with depression experiencing a spike in recent years due to the COVID-19 pandemic. Recent evidence has suggested that depression may be linked with changes in bone health such as fractures which may then worsen the existing depression. In this study, we sought to evaluate the relationship between depression and bone health directly in the patient population in the Rio Grande Valley, an area that is medically underserved. Specifically, we sought to determine how a history of depression was related to bone fracture frequency in our target population. We hypothesized that individuals diagnosed with depression would have an increased risk of bone fractures compared to individuals without depression.
Methods: We conducted a retrospective chart review of electronic medical records within the UTHealth RGV database at the University of Texas Rio Grande Valley (UTRGV) from 2019 through 2022. We analyzed medical records that had: (1) a dual diagnosis of depression and a bone fracture (Group 1; n=32), (2) a diagnosis of only a fractured bone (Group 2; n=117), (3) a diagnosis of only depression (Group 3; n=1,918), and (4) the total number of patients (Group 4; n=50,784). We then calculated the fracture incidence rate for individuals in Group 3 and compared it to the fracture incidence rate in individuals without depression. The depression prevalence and fracture incidence rates for our data were also calculated and compared to the national depression prevalence and fracture incidence rate. Lastly, 95% confidence intervals were calculated to evaluate the uncertainty in our data.
Results: Our findings suggest that the fracture rate for individuals diagnosed with depression was 1.67% in the Rio Grande Valley, while the fracture incidence rate in individuals without depression was observed to be 0.24% (pConclusion: Our results suggest that bone health may be impacted in individuals with depression in the Rio Grande Valley. Overall, we anticipate that our findings may be used to improve bone health in individuals with depression in order to prevent future fractures.

# GRADUATE STUDENT CATEGORY

**Alteration of the Interferon alpha signaling pathway significantly affects Alzheimer's Disease Pathology in APP/PS1 mouse model.**

*Ranjit Kumar Das*

Alzheimer's disease (AD) is a progressive neurodegenerative disorder characterized by cognitive impairment, memory loss, and disturbances in behavior. The exact cause of the disease is unknown but there is evidence of molecular pathways alteration within the brain. Many molecular mechanisms have been studied for the progression of AD. Nonetheless, the contribution of the signal transducer and activator of transcription 1 (STAT1) and interferon-induced protein with tetratricopeptide repeats 3 (IFIT3) genes have not been well established in AD pathology. This study aimed to investigate the contribution of STAT1 and IFIT3 in the BALB/C and APP/PS1 mouse models. We analyzed the STAT1 and IFIT3 gene expression through RT-PCR and protein expression using immunohistochemistry in BALB/C and APP/PS1 mouse models. We found that the expression of STAT1 and IFIT3 genes was significantly higher in APP/PS1 mouse compared to the wild type-mouse, indicating that these two genes were involved in the pathogenesis of AD. Our results suggest that STAT1 and IFIT3 genes may be useful therapeutic targets for the early diagnosis and treatment of AD.

**β-cyclodextrin-enzalutamide self-assembly complexes for prostate cancer therapy**

*Meghana Kolli*

Background: Prostate cancer (PC) remains one of the most commonly diagnosed and a leading cause of cancer-related deaths among men in the United States. PC (approximately 80-90%) is largely dependent on androgens activating through the androgen receptor to give rise to proliferative and invasive cells. Endocrine therapy is often utilized to reduce serum androgens and inhibit AR. Androgen ablation treatment eventually fails and PC progresses to a hormone-refractory or castration-resistant state. Enzalutamide (Xtandi®, AR inhibitor) is a proven oral medication for metastatic castration-resistant prostate cancer (CRPC) that helps to reduce 61% of disease progression and extend overall survival. In this work, we developed and characterized self-assembly complexes β-cyclodextrin-enzalutamide (EZA-β-CD) for improved therapeutic benefit for PC treatment.
Methods: Enz-loaded self-assembly complexes (EZA-β-CD) formulation was developed using solvent evaporation method utilizing β-cyclodextrin (β-CD) as a solubilizer, which has a well-documented safety profile and FDA approval. The inclusion complex formation has been confirmed from dynamic light scattering (DLS), Fourier-transformed infrared spectroscopy (FTIR), scanning electron microscopy (SEM), differential scanning calorimetry (DSC), thermogravimetric analysis (TGA), and nuclear magnetic resonance (NMR) spectral analysis, respectively. The cellular internalization capacity of this formulation was evaluated using flow cytometry and fluorescent microscopy. The therapeutic efficacy of EZA-β-CD self-assembly complexes was evaluated using clinically relevant PC cell lines (C4-2B) through cell proliferation and colony formation assays.
Results: Our DLS, DSC, XRD, FTIR, and NMR studies demonstrated that Enz was entrapped in the inner cavity of β-CD, and the inclusion complex formed in an amorphous state. The particle size of the inclusion complex was found less than 100 nm, while zeta potential and PDI were −7.6 mV and 0.2, respectively. Moreover, inclusion complexes have the characteristic structure of an adduct, in which one compound (EZA) guest molecule, is enclosed to the host molecule i.e. β-CD. Interestingly the guest molecule is situated in the cavity of the host without significantly affecting the host framework structure. The flow cytometry and fluorescence microscopy analysis confirmed a dose-dependent cellular uptake in PC cells. The CCK-8 assay confirms that EZA-β-CD self-assembly complexes exhibited anticancer effects in C42B 1 cells, like bare Enz. A similar clonogenic potential was noticed with the EZA-β-CD self-assembly complexes.
Conclusions: Taken together, our results demonstrate that EZA-β-CD self-assembly complexes exhibit superior anti-cancer potential than free enzalutamide. Thus, this formulation may serve as a novel therapeutic modality for the management of CRPC therapy.

**Evaluating WHO, CCI, and CFS Performance in Predicting Non-Small Cell Lung Cancer Survival in Finland: Insights Beyond CONCORD-3**

*Ville Paappanen*

Background
Set against the backdrop of the 2018 CONCORD-3 study, our research focuses on Finland's lung cancer survival rates. Despite similar tax-funded and public healthcare systems, these rates lag behind those of other Nordic countries, prompting an investigation into the underlying causes. This research aims to unravel these disparities, particularly in the context of Finland's success in other cancer domains, contributing to broader efforts to elevate lung cancer outcomes to the level of other Nordic cancer success stories.

Study objective
Our study focuses on evaluating clinical performance classification systems like WHO Score, Charlson Comorbidity Index (CCI), and Clinical Frailty Scale (CFS) for their predictive accuracy in NSCLC survival and treatment outcomes. We analyze the CCI's weighted scoring of comorbidities and the CFS's applicability in assessing elderly patients' fitness or frailty. This research is integral to our broader aim of identifying key factors influencing lung cancer care in Finland. By integrating these systems into lung cancer treatment evaluations, we endeavor to improve care quality and decision-making in treatment strategies, thereby enhancing patient management effectiveness.
Methods
For this study, we analyzed 2018 data on NSCLC patients from Finnish university and central hospitals. This is part of a larger project that includes all lung cancer histologies. Concurrently, we are compiling a 2021 cohort to assess recent advancements in lung cancer care, with a focus on the effectiveness of immunotherapy and personalized treatment modalities. This work is supported by ethical approval from Findata and includes a partnership with the Norwegian Cancer Registry for comparative analysis. Utilizing R, we conducted Kaplan-Meier and Cox regression analyses on a dataset of 427 variables, ensuring robust survival analysis and significance testing.
Results
We found that the WHO Score remains the gold standard in clinical performance evaluation, demonstrating significant statistical differences between scores (pConclusions: Our study emphasizes the critical role of functional capacity assessments in lung cancer evaluations, highlighting the need for thorough validation. The underperformance of the CCI in our NSCLC cohort underscores the importance of expanding beyond traditional comorbidity indices. Particularly for the oncogeriatric population, our findings stress the value of individualized functional assessments, like WHO and CFS, in crafting effective, personalized treatment plans.

**Investigating Interrater-reliability in Assessing Social Behavior of *Monodelphis domestica***
*Bianca Camacho*

Background
Reliable, consistent, and objective data is a goal all studies aim to achieve, but many struggle to obtain when subjective biases between researchers can occur. Inter-rater reliability (IRR) is a statistical measure used to quantify the degree of agreement between researchers qualitatively scoring the same phenomenon. The primary goal of this study is to enhance the methodology used to achieve optimal IRR. Using an established ethogram, our team of researchers scored the social behavior of the adult gray short-tailed opossum (*Monodelphis domestica*) to propose an effective method for achieving high IRR that can contribute to future research in data accuracy across multiple disciplinary fields, including clinical research and practice.
Methods
A team of two raters used the Behavioral Observation Research Interactive Software (BORIS) to individually score the social behavior of each paired *Monodelphis domestica* subjects based on an ethogram with species-typical behaviors through observational recordings. Raters scored animal subjects' behaviors while pausing throughout each observational video. After the first session of scoring, the team discussed misunderstandings of the ethogram during a review of our respective behavioral scorings for the first-paired animals throughout various timestamps. The team proceeded to individually score the behavior of the remaining four-paired subjects with continued pausing and without further consultations between raters on IRR. To measure IRR for every subject, Cohen's Kappa Coefficient was used due to its strict statistical outcome that accounts for the possibility of false agreement between raters.
Results
Initial scoring sessions for the first-paired subjects yielded low IRR scores (k=0.356, k=0.317). Subsequent scoring sessions for the second-paired subjects demonstrated an increase in IRR (k=0.743, k=0.730). Our final scoring sessions for the third-paired subjects resulted in the highest IRR amongst all scoring sessions (k=0.906, k=0.879). A progressive trend towards increased agreement and inter-rater reliability scores were obtained as familiarity and understanding of the ethogram and BORIS program heightened in each rater.
Conclusions
By using an animal model to train our research team in increasing IRR, our study demonstrated how adjustments of an approach towards reducing subjectivity can contribute to the understanding of improving consistency in future studies. Reliable evaluations are critical for providing excellent healthcare, decreasing misdiagnoses, and optimizing treatment plans for patients. Examining methods towards increasing inter-rater reliability amongst healthcare providers and scientists can strengthen the standardization of clinical research and practice. A limitation in our study was that the consistent pausing while scoring social behaviors led to variation in the time raters spent scoring each subject. These variations in time may contribute to confounding variables, such as potentially inaccurate IRR scores. Our identified limitation emphasizes the importance of delving deeper into researching IRR for a more standardized scoring approach. Therefore, our goal in future investigations is to revise our initial methodology by scoring behavior in real time, decrease time spent on scoring, and assess any disparities in interrater-reliability scores as shown by differences in our methodology.

**LncRNA Impact on Regorafenib Resistance in Colorectal Cancer**

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Cancer metastasis is one of the deadliest aspects of the disease, with about 90% of all cancer-related deaths due to its development at different sites within the body. Colorectal cancer (CRC) is the second leading cause of cancer mortality in the United States, with 40-50% of all patients developing metastasis at some point during their fight with the disease. With the approval of Regorafenib for treating metastatic colorectal cancer, steps have been taken to combat metastasis in colorectal cancer. Regorafenib is an oral small-molecule multiple kinase inhibitor. It is indicated worldwide for patients with metastatic colorectal cancer (mCRC), Regorafenib has already begun to show resistance in CRC. Understanding the mechanisms behind Regorafenib resistance in CRC is vital. Studies have demonstrated the expression of Long-non-coding RNA (LncRNA) to be linked to cancer metastasis and drug resistance. LncRNA UCA1 has been shown in other cancers to lead to resistance to different drugs like cisplatin, gemcitabine, 5-FU, tamoxifen, imatinib, and EGFR-TKIs. In our lab, we have found the LncRNA UCA1 to be overexpressed in CRC patient tissues, with increasing expression across stages I-III, compared to normal tissue. High UCA1 expression has decreased survival among colorectal cancer patients, per the TCGA patient cohort analysis. Furthermore, we found that high UCA1 expression in colorectal cancer cell lines leads to high IC50 values for Regorafenib. Lentiviral transduce stable overexpression (SW480), and knockdown (SW620) cell lines were developed and are being used for UCA1-regorafenib drug resistance mechanistic studies. Increased expression of UCA1 led to increased expression of crucial drug resistance genes (MDR1, ABCB1, and FOXM1) and increased IC50 compared to the control vector. A 3D spheroid model was utilized to assay regorafenib sensitivity to the UCA1 overexpressed cell lines. High UCA1 expression leads to the formation of a higher number of 3D spheroid bodies and size when compared to vector. We treated the spheroid with IC50 concentration, and data will be presented. We also plan to study the lncRNA UCA1 associated Regorafenib resistance in the SW620 knockdown cell lines. In future, we will also analyze the signaling pathways modulated by UCA1 in CRC cell lines, which may be involved in enhancing the regorafenib resistance. This supports the notion that UCA1 is critical in enhancing the resistance to regorafenib in CRC by activating drug resistance pathways. For the first time, this study demonstrates that UCA1 provides drug resistance to regorafenib in CRC, facilitating the progression of CRC metastasis.

**Mechanistic Investigation of C—C Bond Activation of Phosphaalkynes with Pt(0) Complexes**

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Carbon–carbon (C–C) bond activation has gained increased attention as a direct method for the synthesis of pharmaceuticals. Due to the thermodynamic stability and kinetic inaccessibility of the C–C bonds, however, activation of C–C bonds by homogeneous transition-metal catalysts under mild homogeneous conditions is still a challenge. Most of the systems in which the activation occurs either have aromatization or relief of ring strain as the primary driving force. The activation of unstrained C–C bonds of phosphaalkynes does not have this advantage. This study employs Density Functional Theory (DFT) calculations to elucidate Pt(0)-mediated C–CP bond activation mechanisms in phosphaalkynes. Investigating the thermal reductive elimination pathway from the C–CP bond activation product, we present energetics of resulting metal complexes, potential reaction intermediates, and reaction pathways leading to product formation in polar solvent (tetrahydrofuran). Additionally, comparisons between mesityl and phenyl groups, Pt(0) and Ni(0), and ligand-substituent variations reveal nuanced influences on product stability and transition state energies. Parallels with C–CN bond activation in benzonitriles with the Ni(0) fragment provide additional context, advancing understanding not only of C–C bond activation but also offering insights into broader transition-metal-catalyzed reactions. This study, encapsulating the intricacies of C–C bond activation in phosphaalkynes and its implications, contributes to the evolving landscape of pharmaceutical synthesis and catalysis. For example, the application of Pt(0)-mediated C–CP bond activation in the pharmaceutical industry offers innovative routes for drug synthesis, potentially leading to production of novel therapeutic agents with enhanced biological activities.

Keywords: Carbon–carbon (C–C) bond activation, homogeneous transition-metal catalysts, phosphaalkynes, Density Functional Theory, Pt(0)-mediated C–CP bond activation, reductive elimination pathway, energetics, metal complexes, reaction intermediates, reaction pathways, polar solvent (tetrahydrofuran), mesityl and phenyl groups, Pt(0) and Ni(0), transition state energies, C–CN bond activation, benzonitriles, transition-metal-catalyzed reactions, pharmaceutical synthesis, catalysis, pharmaceutical industry, drug synthesis, therapeutic agents

**Oral administration of pH-responsive polymeric nanoparticles based on zein and their therapeutic potential on cancer**
*Bruno Valades-Aguilar*

Zein is a water-insoluble protein extracted from the endosperm of corn seeds, this polymer is an attractive matrix to encapsulate hydrophilic compounds because of its high proportion of hydrophobic amino acids, making it a potential smart delivery material for several treatments in the biopharmaceutical industry. Nanoparticles have been used as drug delivery systems for the improvement of oral bioavailability; however, the strategies of nanoparticle obtention need the addition of stabilizers. In this study, a modified method to obtain zein nanoparticles was developed.
The thermic treatment on zein allowed the obtention of pH-dependant nanoparticles without additional stabilizer, using the antisolvent precipitation method. Additionally, the encapsulation efficiency was improved at 20% in comparison to other syntheses. The release profile on a gastrointestinal *in vitro* model of zein nanoparticles showed their capability as an oral drug delivery system; for this the egg white protein, ovabulmin, was used as a charged model. Finally, the cytotoxic effect of zein nanoparticles and zein solutions against cell lines were evaluated. zein nanoparticles highly decreased the viability on HT29 colon cancer cells in comparison to Huvec endothelial cells; this was evaluated to explore the future therapeutic potential of pH-responsive polymeric nanoparticles.

**P22 viral capsid nanocomposites for enzyme prodrug therapy of breast cancer**
*Astrid Rebeca Luna Rios*

Background: Current treatment strategies against breast cancer have limitations due to lack in selectivity. Most drugs, such as tamoxifen, require metabolic activation by cytochrome P450 (CYP) enzymes to perform greater anticancer effects. However, the concentration of CYP varies and is low in tumor cells, resulting in side-effects. In enzyme prodrug therapy (EPT), enzymes are targeted to the tumor cells for prodrug transformation, involving the sequential delivery of the enzyme followed by prodrug. However, differences in pharmacokinetics and pharmacodynamics are a major hindrance. Thus, co-delivery of prodrug and enzyme is essential to ensure their favorable interaction at target site.
Results: This work reports a new class of therapeutic nanocomposites based on P22 virus like particles (VLPs) confining the CYP activity (P22CYP), surface functionalized with glucose oxidase (GOx) that transforms glucose into D-glucono-δ-lactone producing hydrogen peroxide, the final electron acceptor in the CYP-mediated transformation of tamoxifen, and together conjugated with a tamoxifen derivative as prodrug and targeting ligand using polyethylene glycol as a linker. In glucose-rich tumor microenvironment, these nanocomposites with average size ~70 nm can produce active drug in situ. The physicochemical properties were successfully characterized and nanocomposites represented sequential glucose-mediated catalysis. In vitro studies demonstrated a decrease in cell viability in both ER+ and ER- breast cancer cell lines. However, cellular internalization in the absence of glucose showed improved uptake of targeted VLPs in both cell lines demonstrating improved uptake after pegylation. While the uptake in ER+ cells was significantly higher highlighting the targeting efficiency of functionalized tamoxifen.
Conclusions: The co-delivery of enzymes and prodrug with improved localization of developed VLPs after tamoxifen functionalization, suggests the potential of developed nanocomposites to overcome the existing challenges of EPT and improve the therapeutic outcomes with reduced side effects.

**Relationship Among Cognitive Performance, Physical Activity (PA), Demographic, and Individual Lifestyle Characteristics Among Aging Hispanic Population**
*Ulku Karabulut, Bryan Mixumi, Imtiaz Masfique Dowllah, and Murat Karabulut.*
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PURPOSE: To investigate the association among different intensity and duration of physical activity (PA), cognitive domains (executive function, processing speed, and memory), and demographic and lifestyle characteristics of aging Hispanic population. PA has been identified as a promising non-pharmaceutical preventative intervention for Alzheimer disease and other dementias. This study aims to provide a better understanding of the effects of different intensities of PA on cognitive performances of aging Hispanic population.
METHODS: For this study, 441 Hispanic (age 60) participants' data from NHANES (2011-2014) were analyzed. The protocol was approved by the NCHS Research Ethics Review Board, and all data collection performed by NHANES. Participants self-reported demographic, lifestyle, and health characteristics. The Global Physical Activity Questionnaire (GPAQ) was used for PA pattern, the frequency and duration of moderate intensity PA (MPA) and vigorous intensity PA (VPA) in a typical week, weekly PA was calculated from the product of the number of days by minutes per day and reported as minutes per week. American College of Sports Medicine (ACSM) guidelines were used for both VPA and MPA and for defining inactive, insufficiently active, sufficiently active categories for each intensity level. Participants completed cognitive performance assessment in verbal learning and memory (immediate / delayed), verbal fluency (animal fluency test (AFT)), and processing speed, sustained attention, and working memory (the Digit Symbol Substitution (DSS)) domains.

RESULTS: Out of 441 participants, 225 were female (age= 66.83±5.49 yr.) and 216 were male (age= 66.62±5.81 yr.). Based on reported total minutes of PA in a typical week, 83% and 37% of participants were placed in a physically inactive category for VPA and MPA, respectively. There was no significant difference in immediate recall and DSS assessment between VPA and MPA groups. VPA's sufficiently active subgroup showed a trend to improve delayed recall scores/ delayed memory domain (p*MPA's sufficiently active subgroup showed significantly improved scores in the AFT/ executive function domain (p* Gender and education level were positively, age and depression state were negatively correlated with cognitive performances. There was a positive relationship among HDL and immediate / delayed, AFT, and DSS tests.
CONCLUSIONS: The findings of the study indicated that magnitude of impacts and the degree of association between various PA intensities and different cognitive domain may vary. In addition, many aging Hispanic individuals reported that they are not physically active, and more than two thirds of the participants do not engage in enough vigorous activity. Therefore, caution is advised when interpreting the findings related to the effects of VPA on various cognitive domains. It should be also noted that the positive relationship between HDL levels and immediate / delayed, AFT, and DSS tests may be unique for Hispanic population and demands further attention.

**The carcinogenic effect of heavy metals and its association with epigenetic changes in urothelial bladder cancer patient**
*GANESH KUMAR VERMA*
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Background: According to Global Cancer Observatory (GLOBOCON) 2020, urothelial bladder cancer (UBC) is the 17th most common malignancy in India, and bladder cancer represents a significant health problem due to the potential morbidity and mortality. UBC is commonly associated with industrialized regions and the most accountable risk factors have been tobacco smoke along with occupational chemical exposures including heavy metals. A massive increase in human exposure to heavy metals has been noticed due to industrial activities since last century and an emerging understanding of the carcinogenic effects of certain heavy metals is now being considered as causative agents for UBC. However, the carcinogenic role of certain heavy metals in the etiology of UBC has not yet been studied comprehensively.
Aims and objectives : The present study aims to investigate the carcinogenic role of heavy metals through epigenetic changes in UBC. 1. Assessment of heavy metals in Blood, urine & and tissue samples. 2. Assessment of Global DNA methylation (5-mC%) in tumor and adjacent healthy tissues.
Methodology: The cross-sectional study where a total of 50 patients with urothelial bladder cancer and 50 healthy controls were recruited. Heavy metals were quantified using Inductively coupled plasma-Mass-spectrometry (ICP-MS) in blood, urine, and tissues. The global DNA methylation level was assessed by ELISA in cancer and adjacent healthy tissue obtained from patients.
Results: The mean age of patients with UBC and healthy control were 56.96 ± 13.4; 31.78 ± 7 respectively. The heavy metals chromium (2.72 ± 7.1 vs 2.57 ± 2.9; p 0.02), arsenic (0.27 ± 1.5 vs 0.2 ± 0.7; p 0.007), and cadmium (0.61 ± 3.9 vs 0.16 ± 0.2; p 0.01) in urine were high in patients. Lead in blood (8.15 ± 9.6 vs 5.0 ± 4.5 p 0.02) was elevated in patients.
The 5-mC% is significantly higher in cancer tissue (24.5 ± 17) compared to adjacent healthy tissue (2.3 ± 2.1) p ConclusionHeavy metals may be one of the major risk factor for the development of urothelial bladder cancer by inducing epigenetic modification as DNA methylation.· Name of Presenting Author: Ganesh Kumar Verma· Academic status of Presenting author: PhD Scholar· Discipline: Biochemistry·

**The role of age and biological sex on short-term memory in the Syrian hamster (Mesocricetus auratus).**
*Authors: 1Laura Emma Garcia, 1,2Mario Gil, PhD. 1Department of Psychological Science, 2School of Medicine and Institute of Neuroscience.*

Background: Aging plays an important role in cognitive function, memory, and mental health. As we age, some cognitive functions become weaker. Aging is also a critical risk factor for dementia and Alzheimer's Disease. With recent advances seen in public health, humans are living longer years. This makes it a challenge to make those added years healthy. For this reason, it is important to continue studying its effects by using, the Syrian hamster, as a model. The purpose of the study is to identify other factors that might also have a role in short-term memory, besides aging.
Methods: All behavioral experiments were conducted using a 16-hole board apparatus. Each hole was categorized as a zone to collect data. Zone 6 was used to expose the control/treatment during trial 2. Subjects were exposed to either clean or dirty bedding (corresponding to opposite sex odors), control or treatment during this trial. Animals were placed in the hole board for five 3-minute trials; Preexposure, Treatment/Exposure, and Memory tests 1-3. Memory tests 2 and 3 consisted of 15 and 30-minute inter-trial intervals, respectfully, from time of exposure to treatment/control. Three cohorts of male (n=28) and female (n=36) subjects, including young, middle, and old aged dependent on their date of birth, randomly chosen. First, Preexposure trial was done for 3 minutes to collect data on zone preference and expose hamsters to the apparatus. Second, hamsters were exposed to treatment/control on trial 2 for 3 minutes. Thorough cleaning done in between each trial. Hamsters were then tested right after cleaning for memory trial 1. Memory trial 2 took place 15 minutes from the time of treatment/control exposure. Memory trial 3, 30 minutes from the time of treatment/control exposure.

Results: Data was collected using ANY-maze tracking system and analyzed with SPSS software using repeated measures ANOVA. Males had more overall entries into the stimulus zone (6) compared to females F (1, 41) = 10.507, p=0.002, but there was no significant interaction between sex and trials F (3, 123) =1.648, p=0.182. There was no sex difference in overall entries into a neutral zone (1) F (1, 41) =0.012, p=0.914 and there was no significant interaction between sex and trials F (3, 123) =0.603, p=0.614. There was no significant interaction between age and trials; old cohort - F (1,41) = .260, p=.613; middle cohort - F (1,41) = 1.526, p=.224; young cohort – F (1,41) = .143, p=.7.07.
Conclusion: Our results provide evidence that the Syrian hamster is an excellent animal model for the study of exploring sex differences in learning and memory. Males approached a social stimulus more often than females, and the middle-aged cohort demonstrated a higher main effect during memory trials. In this present study, we will further explore factors like, housing, weight, and age, and their role in short-term memory.

**Trimeric Antp-TBP complexes with TFIIEβ and Exd modulate the transcriptional activity of Antp**
*Norma Carolina Hernández Bautista, Claudia Altamirano-Torres, Ruben Montalvo-Mendez , Gustavo Jimenez-Mejia, Diana Resendez-Perez*

Homeoproteins are transcription factors that bind to DNA through a highly conserved binding domain known as the homeodomain (HD), which recognizes short regions rich in A-T. This DNA binding triggers the modulation of multiple target genes responsible for identity in various structures along the A-P axis of *D. melanogaster*. However, the high similarity of homeodomains and their affinity for widely distributed sites in the genome poses the "Hox paradox," which seeks to explain how homeoproteins acquire functional specificity so precisely. Previous evidence shows that homeoproteins like Antennapedia (Antp) collaborate with cofactors and other transcription factors to modulate their transcriptional activity. For this reason, we have conducted transactivation analyses to determine the effect of trimeric complexes on Antp's transcriptional activity. The experimental strategy involved transfecting HEK-293 cells with different combinations of plasmids producing Antp, TBP, TFIIEβ, and Exd, along with two reporter plasmids: a luciferase reporter with 5 binding sites for Antp and the sequence of a *D. melanogaster* basal promoter, and a β-galactosidase reporter. Our results reveal that the trimeric interaction of Antp-TBP-TFIIEβ induces a highly significant increase of 193% in Antp transcriptional activity. On the other hand, Antp-TBP-Exd trimer shows opposite results by decreasing Antp transcriptional activity until 61%. These findings suggest that the trimeric complexes Antp-TBP/TFIIEβ/Exd play a role in modulating Antp's transcriptional activity, raising new questions about the *in vivo* effects of these complexes on Antp interactome and target genes.

**Ultrabithorax, Abdominal-A and Abdominal-B forms homodimers and heterodimers in living cells by BiFC**
*Rubén de Jesús Montalvo-Méndez, Gustavo Jiménez-Mejía*

Background: Hox genes encode transcriptional factors that regulate the expression of specific target genes along the anterior-posterior axis determining the segment identity during embryonic development. These master genes are expressed in the same order in which are located in the third chromosome of *Drosophila melanogaster*. As it was previously reported the interaction of neighboring Hox proteins as Scr-Antp and Antp-Ubx and one of the mechanisms for Hox protein regulation is protein-protein interaction, we determined the heterodimer formation of Ultrabithorax (Ubx) with Abdominal (Abd-A) and Abdominal-B (Abd-B) as well as the homodimer formation of these proteins in living cells which in turn could shed light about gene regulation mechanisms of their target genes and phenotypic suppression during *Drosophila* development.
Methods: We used Bimolecular Fluorescent Complementation (BiFC) to determine protein interactions between Ubx - Abd-A and Abd-B, Abd-A - Abd-B as well as the homodimer formation of Ubx, Abd-A and Abd-B. We constructed expression vectors carrying the coding sequences of Ubx, Abd-A and Abd-B fused to the N-terminal and C-terminal halves of Venus (VN and VC, respectively). Expression vectors were co-transfected with pCAGmCherry in HEK293 cells and red (Cherry) and green (BiFC) fluorescent cells were quantified using imageJ to determine BiFC percentage.
Results: The results show that Ubx interacts with Abd-A and with Abd-B giving 80% and 86% of interaction, respectively and Abd-B also interacts with Abd-A giving 90% of interaction. Surprisingly, interactions of Ubx – Abd-A and Abd-B – Abd-A are affected by BiFC topology. Our results also demonstrated that Ubx and AbdB forms homodimers giving 82% and 96% of interaction, while Abd-A does not.
Conclusions: We found heterodimer interactions of Ubx with Abd-A and Abd-B as well as Abd-A with Abd-B. Also, we showed homodimers formation of Ubx and Abd-B while Abd-A does not form homodimers. Altogether, these Hox interactions increase the evidence of neighboring interactions between Hox proteins and suggest a similar regulation mechanism that open the possibility to study its effect on the regulation of their target genes as well as phenotypic suppression during *Drosophila* development.

**Unraveling Sorafenib Resistance in Hepatocellular Carcinoma: Exploring Key Facets**

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Abstract

Hepatocellular carcinoma (HCC) stands as the prevalent form of primary liver cancer worldwide, diagnosing over half a million new cases annually. Surgical interventions like hepatectomy and liver transplantation offer a potential cure for early-stage HCC. However, the prognosis for advanced stages remains grim due to drug resistance, particularly with high refractoriness rates. Sorafenib, a tyrosine kinase inhibitor, is an approved treatment for advanced HCC. Despite its use, the overall survival extension for these patients remains limited due to the drug's ineffectiveness, and the mechanism behind advanced HCC's resistance to sorafenib remains elusive. TCGA analysis of HCC patient cohorts reveals elevated YBX1 expression in tumors compared to normal tissues. This heightened expression correlates with disease progression, metastasis, and poor survival rates among HCC patients. YBX1, a transcription factor known for regulating drug resistance in various cancers, particularly in colorectal and breast cancers, piqued our interest in investigating its potential involvement in sorafenib resistance within HCC. Employing MTT assays, we determined the IC50 values of sorafenib at different time points (24 and 48 hrs) on HCC cell lines obtained from ATCC. Overexpression of YBX1 resulted in increased cell survival and raised IC50 values for sorafenib at both time points, supporting the hypothesis that underscores YBX1's pivotal role in promoting sorafenib resistance in advanced HCC. To delve deeper, our lab developed puromycin-stable GFP-expressing cell lines for both YBX1 overexpression and knockdown, facilitating further mechanistic investigations. Additionally, we are progressing in establishing sorafenib-resistant HCC cell lines and conducting ongoing protein and RNA analyses on these resistant cells. Understanding the efficacy of sorafenib in treating advanced HCC and unraveling the signaling pathways associated with YBX1-induced drug resistance holds promise in significantly improving the prognosis for individuals battling this malignancy.

# UNDERGRADUATE/HIGH SCHOOL STUDENT CATEGORY

**An Evaluation of the combination of Metformin and Y15 for the treatment of Platinum-Resistant Ovarian Cancer**
*Manuel Duarte, Kristal Garcia*

Background
Ovarian cancer is the fifth leading cause of cancer mortality among women. This high mortality rate is linked to the development of resistance to first-line chemotherapy with platinum compounds which has been attributed in part to increased activity of focal adhesion kinase (FAK). The anti-diabetic drug Metformin was previously shown to inhibit the proliferation and migration of ovarian cancer cells and thus, the combination of a FAK inhibitor, Y15, and Metformin may be a promising treatment for platinum-resistant ovarian cancer (PROC). The objective of this study was to evaluate the combination of Y15 and Metformin on PROC cell viability and the mechanism of cell death.
Methods
An MTT assay was used to analyze cell viability in PROC OVCAR3 cells after 48 h of treatment by measuring the absorbance at 570 nm with a microplate reader. Western blot was used to determine the protein levels of the apoptosis marker cleaved PARP and caspase 3 and the autophagy marker LC3B-II.
Results
The exposure of OVCAR3 platinum-resistant ovarian cancer cells to Metformin (4.5 mM) + Y15 (5.5 µM) resulted in a significantly enhanced cytotoxicity (32.6 ± 1.8%) compared to single drug treatment with either Metformin (4.5 mM) (65.0 ± 4.2%) or Y15 (5.5 µM) (66.0 ± 4.8%). A combination of Metformin (7.8 mM) + Y15 (5.5 µM) resulted in a significantly enhanced cytotoxicity (22.8 ± 1.5%) compared to single drug treatment with either Metformin (7.8 mM) (50.7 ± 3.7%) or Y15 (5.5 µM) (66.0 ± 4.8%). Cells treated with the combination of Metformin and Y15 for 24, 48, and 72-hours showed an increase in cleaved PARP compared to the control, Metformin alone and Y15 alone. For Metformin alone, there was an increase in cleaved PARP compared to the control at all timepoints and increased from 24 hours to 72 hours. For Y15 alone, the amount of cleaved PARP was also higher compared to the control at all time points, however, decreased over time from 24 to 72 hours. Cells treated with the combination of Metformin and Y15 for 72-hours showed an increase in caspase 3 compared to control, Metformin alone and Y15 alone. Cells treated with Metformin, Y15, and a combination of Metformin and Y15 showed no conversion of LC3B-I to LC3B-II, which occurs during autophagy.
Conclusion
Thus, it is concluded that the mechanism of cell death for both Metformin and Y15 is through apoptosis and not autophagy and apoptosis is enhanced with the combination of the drugs. The delivery of Metformin and Y15 can result in an additive effect on cell viability through apoptosis and can be further explored as a promising approach for the treatment of PROC.

**Carbon Monoxide Suppresses hEAG1 Potassium Channels and Cell Growth in Liver Cancer**
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Abstract:
Background: Heme contains iron and porphyrin. It degrades into metabolites like biliverdin, carbon monoxide (CO), and bilirubin. High levels of these can be detrimental. However, at normal levels they may act as signaling molecules. One example is CO which comes from heme breakdown by heme oxygenase. Dysregulation of CO and heme metabolism has been linked to some cancers. Methods: This study examined how external application of CO affects hEAG1 potassium channels and proliferation of Hepa 1 hepatocellular carcinoma (HCC) cells. CO was introduced using a caged donor molecule called CORM-2, which releases controlled CO at the target site. hEAG1 activity was measured by patch clamp electrophysiology. Proliferation was assessed by holography microscopy. Lysosome numbers were visualized by LAMP1 immunocytochemistry and quantified by confocal microscopy. Results: Our result showed that CORM-2 suppressed hEAG1 currents. It also decreased Hepa-1 HCC cell proliferation. Immunocytochemical analysis showed CORM-2 increased lysosomes. Western blot results also supported this, revealing more LAMP1 protein. These results suggest a lysosome pathway involved in the cytotoxicity of HCC cells. Conclusions: Our findings suggest external CO influences hEAG1 channel activity, HCC cell proliferation, and lysosome levels. This adds to the understanding of how CO metabolites may signal to impact cancer cell behavior. Targeting pathways regulating CO and heme could potentially serve as an HCC treatment

**Development of liposomes using microfluids for delivery of miR-205**
*Victoria Herrera*

Background: The therapeutic application of microRNA(s) in the field of cancer has generated significant attention in research. miR-205 is a tumor suppressor in various cancers. However, the delivery of miR-205 is an unmet clinical need. Thus, the development of liposomal formulation platform to deliver miR-205 is highly sought. The most common applications of liposome formulations are vaccines and anticancer formulations (e.g., mRNA, small molecule drugs). However, large-scale production with precise control of size and size distribution of the lipid-based drug delivery systems (DDSs) is one of the major challenges in the pharmaceutical industry. The objective of this study is to develop liposomal formulation with precise size and optimal for delivery of miR-205.
Methods: Microfluidics chip designed based on commercial microfluidic device platform was employed for preparation of liposomes. The device is set for the synthesis of liposome at total flow rate (FRR) 10 ml min−1 and 1:3 flow rate ratio (TFR). To determine the optimal conditions, the effect of different factors including FRR, TFR, and total lipid concentration (lipid and cholesterol) on particle size and size distribution is investigated. Liposomes are also produced by a bulk method to compare the properties of the liposomes formed through these methods. The obtained formulations were tested to analyses different physiochemical properties (DLS, FTIR, DSC, and TGA), stability studies and optimized liposomal formulation was confirmed by examining the intracellular accumulation.
Results: All formulations displayed an average size less than 200 nm and exhibited acceptable physicochemical behavior. This design demonstrated high productivity and better control of liposome size and polydispersity index (PDI) than conventional liposome preparation methods. The microfluidic devices were used to produce miR-205-loaded liposomes under different processing conditions which were later characterized and studied in vitro to evaluate their efficiency as a drug delivery system.
Conclusions: The obtained results demonstrated that the liposomes can effectively deliver miR-205 into cancer cells. Therefore, the microfluidic devices platform are promising devices for reproducible and scalable manufacturing of liposomal formulation.

**Evaluating Alternate Motor Pathway Changes following a Stroke**
*Rama Shaath, Marylu Cabello, Daniel Salinas, Michael Dobbs, Kelsey Baker*
*UTRGV School of Medicine*

Keywords: Stroke, Rehabilitation, Neuroregeneration, Neurodegeneration, Disability, diffusion weighted imaging (DWI)

Background: Stroke is the fifth cause of death in the United States. Not only is stroke a leading cause of death but it is also a leading cause of long-term disability in the United States. Long-term impairments after stroke include gait instability, upper limb paralysis, sensory deficits, pain, depression, and cognitive impairments. The most common impairment is motor paresis of the upper and lower limb. Rehabilitation remains the gold standard in addressing motor paresis with the goal of enabling subjects to regain independence and daily living skills. Strokes often impact the crossed lateral corticospinal tract, by damaging the tract or the neighboring pathways. The damage within these pathways results in motor deficits. Detailed understanding of changes to the corticospinal tract, major neuronal pathway providing voluntary motor function, after stroke has resulted in the use of targeted therapies to improve rehabilitation outcomes. Alternate motor pathways also give a significant role in stroke recovery. This may be because many of the pathways work independently or work together with the corticospinal tract to trigger motor and sensory function. The overall goal of the project was to evaluate neurodegeneration and neuroregeneration in alternate motor pathways in patients who have suffered an acute ischemic stroke.
Methods: Within this study 30 subjects who have suffered an acute stroke and 10 healthy control patients will be enrolled into the study. We will conduct motor function exams and collect neuroimages at two, twelve, and twenty-four weeks after the initial stroke event in each subject. Ten healthy age-matched controls will also be enrolled for a single MRI collection visit. We collected T1-weighted magnetic resonance images (MRI) and diffusion weighted imaging (DWI). When analyzing the images we used DSI studio to shade in our regions of interest. FSL was utilized to extract integrity of evaluated tracts.
Results: We observed neuroanatomical differences at the level of the cerebral peduncle and posterior limb of internal capsule in both the affected (stroke-side) and unaffected hemispheres of the brain. Our preliminary data suggests that immediately after a stroke event, minimal changes are noted that become more dramatic over time.
Conclusions: Our results suggest that alternate motor pathways undergo dynamic changes post-stroke. Our pilot work has found that while the corticospinal tract remains a critical component in recovery, other pathways may also be impacted post-stroke in a time dependent manner. Future work will evaluate advanced neurite imaging modalities, like NODDI, to evaluate microstructural property changes post-stroke.

**Evaluation of Multi-Session Temporary Anesthesia to Improve Upper Limb Recovery: A Preliminary Analysis in Healthy Subjects**
*Ashley Tijerina, Abdallah Galal, Daniel Salinas, Kelsey Baker*
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Background: Traditional therapy routines utilized in the recovery of patients with spinal cord injury suffer from several limitations, including the long duration needed to achieve benefits and the inability to overcome activation of spared muscles. As a result, motor and sensory function almost never reach their full potential for recovery using traditional methods. Temporary functional deafferentation (TFD) is a relatively new and noninvasive technique that can modulate mechanisms of cortical reorganization in patients with spinal cord injury to improve therapy approaches. TFD involves temporarily numbing adjacent ipsilateral muscles to improve motor and sensory outcomes in the target muscle. The purpose of this study is to investigate the effectiveness of a multiple session approach of temporarily numbing the biceps of healthy subjects in improving the sensory and motor functions of the ipsilateral triceps.
Methods: Five subjects were recruited to the study. Enrolled subjects had age ranges of nineteen to twenty-two, and two were male and three were female. After enrollment, baseline measurements of hand and grip strength, dexterity, pinch strength, and triceps strength were obtained by measuring the electrical activity of muscle groups using an electromyograph. Subjects then underwent a three-day exercise regimen targeting the triceps, following TFD of the biceps. TFD was achieved each session by applying lidocaine numbing cream directly to the biceps and covering the cream with tape corresponding to the size of the subject's arm circumference to ensure the integrity of the cream during the numbing process. The lidocaine cream was left on the muscle for fifty minutes to allow sufficient absorption and numbing of the area.
Results: The study yielded a significant increase in muscle electrical activity in the biceps, triceps, and grip strength between different sessions after the application of lidocaine numbing cream. There were no significant differences between sessions after performing vigorous activity. This could potentially be explained by the timing of the muscle activity recording after the exercise. Electromyograph recordings were taken immediately after exercise, which may impact the readings if the participants' muscles were exhausted after exercising.
Conclusion: This study implicates the effectiveness of using temporary deafferentation in improving motor and sensory functions and suggests a role for this technique in future therapy routines involving patients with spinal cord injury. A follow-up to this study should be conducted in which electrical activity measurements are taken a short time after completion of the training phase, to allow fatigued muscles to recover and regain strength.

**Examining CETP gene associated with AD-related diseases of the Hispanic population in the Rio Grande Valley.**
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Background: There are currently about 6 million people in the United States that suffer from Alzheimer's Disease (AD) and Alzheimer's Disease related dementia (ADRD). It is a progressive disease beginning with mild memory loss and possibly leading to loss of the ability to carry on a conversation and respond to the environment. Over time, these conditions can cause many different health issues that decrease the quality of life. In addition, Hispanic people are twice as likely to develop AD or AD related dementia than non-Hispanic White people. In our study, we are investigating a known gene, CETP, that directly corresponds with AD and dementia in the U.S Hispanic population.
Methodology: A total of 200 Hispanic subjects were collected from the Rio Grande Valley (RGV, N = 200). Questionnaires from demographics, lifestyles, medical history and saliva samples were collected. We genotyped for the CETP gene based on one SNP, with statistical analysis being performed through Chi-squares tests, independent sample t-test, and multivariable logistic regression models using SPSS version.
Results: Current findings have indicated that there was a higher index of Hispanics in the RGV with both the major and minor alleles (A;G), Preliminary findings also showed that there was a higher frequency of individuals with (A;A, risk allele) than any of the other genotypes. There was a weak correlation found with lung problems and AD/ADRD.
Conclusion: The CETP gene has shown increased risks of AD related diseases in the RGV Hispanic population. However, further research is needed to confirm our current findings for this specific population

**Exploring the Anti-Cancer Potential of Anthocyanins via Autophagy Overactivation**
*Sumeet Chauhan*

Anthocyanins, a class of natural pigments found in plants, provide many fruits and vegetables with brilliant colors, known for their robust health-promoting properties, including antioxidant, anti-inflammatory, and anticancer effects. One such anthocyanin called Dracorhodin, derived from the fruit of *Daemonorops draco*, also known as 'dragon's blood has exhibited promising anticancer effects.However, the molecular mechanisms underlying Dracorhodin's antitumor activities remain poorly defined. Here, we explored whether a synthetic derivative of Dracorhodin called Dracorhodin Perchlorate (DP) could induce cytotoxic autophagy, a cellular self-digestion process, in colorectal cancer cells.

We found DP potently suppressed colorectal cancer cell SW480 proliferation by excessively activating autophagy. Further analysis revealed DP triggered autophagy by inhibiting the mammalian target of rapamycin (mTOR) signaling, activating the autophagy regulator transcription factor EB (TFEB), enhancing lysosome expression and function, and mobilizing calcium stores to induce calcium-dependent autophagy. Our findings provide insight into Dracorhodin's anticancer potential by delineating specific mechanisms of cytotoxic autophagy induction via coordinated effects on mTOR-TFEB and calcium signaling.

**Gamma-aminobutyric acid (GABA) Neurons and Perineuronal Nets (PNN) in the *Monodelphis domestica* and Relevance to Neuropsychiatric Disorders**

*Jatziry Luna*

Gamma-aminobutyric acid (GABA) is an amino acid that serves as the central nervous system's (CNS) main inhibitory neurotransmitter. By inhibiting nerve transmission, it works to lower neuronal excitability. Altered GABA levels have been associated with a variety of psychiatric disorders, for example Epilepsies, Parkinson's Disease, and Schizophrenia. Perineuronal nets (PNN) are extracellular molecules that are released by neurons and glial cells that modulate many neuronal and glial functions by encapsulating the inhibitory cells and neurites. They play a key role in creating a physical barrier to control the changes in the formation of new connections between neurons. Altered PNN levels serve as a potential trigger to synaptic imbalance. The purpose of this study is to quantify and analyze the presence, change in number, and area difference of $GAD_{67}$ and Lectin in the brain of the *Monodelphis domestica.*

**Impact of a Costello Syndrome-Causing Mutation on Learning and Myelin-Producing Cells**

*Carlos A. Cisneros, Daniella Hernandez, Saul Lopez, Celeste Martinez, Kaelah Araiza, Ana Gutierrez, Alejandro Lopez-Juarez*

Background: Costello Syndrome (CS) is a rare genetic disorder caused by hyperactivating mutations in the *HRAS* gene, which controls the RAS/MAPK intracellular pathway. Symptoms of CS typically include neurocognitive developmental delays, increased risk of autism spectrum disorder, intellectual disabilities, and other neurological issues. Additionally, most CS patients present with white matter (WM) abnormalities. WM has been proposed to regulate learning due to its roles in increasing/synchronizing action potentials and protecting neuronal axons. Females of a myelin-focused mouse model of CS (*PlpCre;HRasG12V*; *pHRas*) show learning deficits in a myelin-regulated test (the complex running wheel; CW) that resolve with time. To shed light onto the mechanisms of these learning deficits, the goal of our study is to describe changes in oligodendrocyte (OL; myelin-producing cells) lineage cells in *pHRas* mice.
Methods: To correlate the cellular and functional impact of *HRas* mutation on OL lineage (OLL) cells, *HRas* mutation was induced in mature OLs (mOL) using a tamoxifen-inducible system. Four months after recombination, mice were subjected to the voluntary CW test (a wheel with unevenly spaced rungs), and learning curves were analyzed. An acquisition phase of 14 days was followed by a break from CWs of 3 weeks and a second CW phase of 7 days (memory of skills acquired). After the second exposure to the CW, mice were euthanized, and brain sections were collected for staining with DAPI (nucleated cells), GFP (recombinant cells), PDGFRa (oligodendrocyte precursor cells; OPCs), and Sox10 (OLL cells). Then, cell quantification was performed after conducting confocal imaging. Seven regions of the corpus callosum, across 4 coronal sections, were analyzed for regional differences in the numbers of $PDGFRa^+$ OPCs and $Sox10^+$ OLL cells.
Results: Our antecedents show that at 2 weeks and 2 months post-mutation, there were significant decreases in distance, average speed, and max speed ran, as well as activity in mutant mice compared to WTs (defective learning curves). However, at 4 months post-mutation, there were no significant differences in learning curves between mutant and WT mice. We then wondered how OPC populations remained at 4 months post-tamoxifen when differences in behavioral phenotypes were no longer detected. We observed that the number of $PDGFRa^+$ OPCs decreased in the lateral region of the most anterior coronal section of the corpus callosum, suggesting non-cell-autonomous effects of *pHRas* on proliferation and/or differentiation of OPCs.
Conclusion: Taken together, our results shed light on the role of the *HRas* mutation on CS mouse models that show transient learning deficits on the CW after the induction of the mutation. Our working hypothesis is that decreased number of OPCs may be a result of an increase in their differentiation into mOLs (to form myelin that restores learning) but lead to a proliferative exhaustion state. We propose an immediate impact (weeks) of *HRas* mutation on learning that is ameliorated by OPC differentiation.

**$La_{1-x}Sr_xCoO_3$ perovskite nanomaterial: Synthesis, characterization, and its biomedical application**

*Adhira Tippur*

Early cancer detection is paramount for effective treatment and potential cures. This research explores the application of perovskite materials, specifically Sr2+-doped Lanthanum Cobaltite (La1-xSrxCoO3) nanomaterials, in cancer detection, with a focus on rats as an experimental model. The ferroelectric nature of these materials, synthesized through a combination of sol-gel and molten-salt processes, was examined at varying Sr2+ doping levels (1-20 wt%). Rigorous characterization, employing X-ray diffraction and scanning electron microscopy, confirmed the uniform morphology of nano cubes, laying the foundation for subsequent investigations.

The magnetic properties of the perovskite nanoparticles were probed, suggesting their potential as a diagnostic tool for cancer detection. This study extends to the synthesis and characterization of inner transition metal oxide (perovskite-type structure), utilizing X-ray diffraction spectroscopy to assess phase purity. Scanning electron microscopy provides insights into material morphology, while FTIR spectroscopy and UV-Visible spectroscopy contribute additional justification for the synthesized material. The research aims to integrate these findings into the development of a diagnostic tool for cancer, with a particular emphasis on hyperthermia therapy. Future research is needed to realize the full potential of this innovative approach in human cancer detection and treatment. This project bridges the fields of material synthesis, thorough characterization, and biomedical application, offering a promising avenue for advancing cancer diagnostics and treatment modalities.

**Ongoing Study on the Association of APOE Gene Polymorphisms and Education with Cognitive Impairment in the Rio Grande Valley Hispanic Population**

*Jaime Saveron, Daniela Gamez*

Alzheimer's disease (AD), stands as one of the most prevalent forms of dementia, affecting around 6.7 million people over the age of 65 in the United States [1]. This neurodegenerative disorder is marked by a progressive cognitive decline, and its risk factors include behaviors such as smoking or poor diet and preexisting conditions such as diabetes and mild cognitive impairment (MCI) [2]. MCI represents an intermediate stage between typical and atypical cognitive aging, manifesting as forgetfulness and confusion without severely impeding daily tasks [2]. It serves as a transitional phase in the progression of AD, for everyone who eventually develops AD experiences MCI first [1]. While certain demographic factors and genetic variants associated with AD and MCI have been identified in non-Hispanic populations, limited research exists on this subject within the Hispanic population, particularly in the Rio Grande Valley. Therefore, this study aims to investigate the relationship between the APOE gene, specifically its ε4 allele, and cognitive impairment in the Hispanic population of the Rio Grande Valley. A total of 252 Hispanic subjects over the age of 60 were recruited from the Rio Grande Valley. Questionnaires from demographics, lifestyles, medical history, MoCA exam and saliva samples were collected, and genotyping for APOE allele was performed using two SNP's. Statistical analysis, such as Chi-Square analysis, Fisher's Exact Test, and Generalized Linear Models were performed using IBM SPSS Statistics software version 28.0.1.1. Our study sheds light on the potential association between APOE gene polymorphisms, particularly the ε4 allele, as well as education as the two most important factors on cognitive impairment in the Hispanic population of the Rio Grande Valley. Further analysis and recruiting must be done to strengthen and examine other findings, such as various genotype related outcomes such as diabetes, hyperlipidemia, and cardiovascular problems.

**Optimization of Baseline Values for the Bionik InMotion Arm/Hand Rehabilitation Robot**

*Marysol Cabello*, Diego Rojano*, Daniel Salinas, Ramiro Oquita, Victoria Cuello, Kelsey Baker*

Background: Clinical studies utilizing the Bionik InMotion Arm/Hand robot are increasing in recent years. However, there is a lack of standardized values from healthy participants that can be used to compare to patients with stroke or other neurodegenerative disorders. We aim to create this standardized set of values by employing the new Bionik InMotion Arm/Hand robot on healthy participants. Establishing normative values will allow our lab, and other laboratories, to have a baseline that can be used to track rehabilitation progress in patients with neurological disorders, such as stroke. Creating this standardization may also allow for more tailored rehabilitative approaches to be created for individual patients and their needs.
Methods: Our IRB-approved study involved a single session visit that lasted approximately 90 minutes. Healthy participants were enrolled, and handedness determined using the Edinburg Handedness Inventory. For each participant, the right and left arm was evaluated with the Bionik InMotion Arm/Hand robot. On both arms, evaluations were collected for the arm and hand in 45- and 90-degrees forearm supination. Evaluation required participants to draw a circle, reach targets on the computer screen and grip as quickly as possible with their hands.
Results: A MANOVA testing positioning angle resulted in significant main effect on Arm evaluation metrics, Pillai's Trace = .19, $F(11, 139) = 2.94$, partial eta2 = .19, $p$ . Post hoc tests revealed better performance on 45 degrees compared to zero degrees on Point-to-Point Smoothness. Conversely, zero degrees performance was better in Target Accuracy but slower in movement duration. For mean wrist evaluations, A MANCOVA resulted in significant interaction effects of positioning angle and range of motion grasp cm were witnessed, Pillai's Trace = .61, $F(22, 268) = 5.37$, partial eta2 = .31, $p$ . Post hoc tests revealed better hand performance on a zero-degree angle compared to 45 on active range of motion (ROM) hand grasp time, and passive hand ROM grasp force, $p$
Conclusion: Our results indicate that wrist position in the Bionik InMotion Arm/Hand unit was significantly related to outcome metrics. Specifically, a wrist angle of 45 degrees was related to improved arm performance. This result is plausible since typical reaching patterns of the upper limb naturally have the wrist in the 45 degree angle. Overall, our results highlight that optimized baseline values for the robotic arm rehabilitation device are dependent on wrist position, handedness and area measured. We anticipate our values will provide normal comparators as this device is used to treat and diagnosis patients with neurological disorders.

**Optimization of Gait Analysis System for Clinical Applications**

*Gerardo Medellin, Katherine Bolado, Daniel Salinas, Kelsey Baker*
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Background: Adequate gait function is pivotal for many activities of daily living and high quality of life. Following many neurodegenerative diseases, such as Parkinson's Disease, gait abnormalities can manifest and range from reduced stride length, inability to turn, foot drop or shuffling. To track and monitor such changes in gait, gait analysis techniques are gaining clinical popularity and have the ability to gather a range of data in a short duration. Gait analysis techniques go beyond simple visual observation and include instrumental gait analysis and weight distribution of the gait cycle. Here, we sought to optimize the gait analysis system located at the Institute of Neuroscience at UTRGV School of Medicine. Specifically, numerous studies have indicated that factors such as BMI, foot size, foot covering and leg length can influence metrics collected with a gait analysis system. Here, we sought to identify the influence of such variables on clinical assessments with the gait analysis system.
Methods: A single-session study was conducted wherein healthy controls were recruited to undergo gait analysis in three conditions: bare foot, socks and with shoes. We used the ProtoKinetics Zeno Walkway Gait Analysis system for the study. The study received IRB approval and has enrolled 5 participants to date. After enrollment, we collected each subject's height, weight and foot size (Brannock device). Gait belts were worn by all subjects as a safety precaution for fall risk during the study. Subjects were then instructed to walk normally on the gait mat, making several passes back and forth (one ambulation period is across the mat and back, participants performed at least 7 ambulation periods for optimal data receiving). Subjects were asked to complete the gait passes with shoes, socks and barefoot.
Results/Discussion: We observed that ambulation foot velocity varied depending on foot covering. For example, mean foot velocity barefoot was faster (111.40 cm/s) compared to wearing shoes (107.69 cm/s). In addition, we observed changes in participant cadence between conditions. Mean cadence barefoot was slower (106.1 steps/min) compared to with shoes (108.8 steps/min).
Conclusion: Our preliminary findings suggest that foot coverings play a significant role in gait analysis metrics. While subjects velocity was faster moving their feet when they were barefoot, their mean cadence was slower. We believe this result might be instinctual and related to added comfort that is provided when wearing shoes. We anticipate our findings will help research groups establish standard operating procedures for clinical deployment of gait analysis in clinical populations.

**Primary Care Behavioral Health Partnerships Advancing & Transforming Health Sciences (PCBH PATHS)**

*Mayrin Perez, Maria Hernandez*

Primary Care Behavioral Health Partnerships Advancing & Transforming Health Sciences (PCBH PATHS) is a workforce development pipeline project aimed at permanently augmenting UTRGV's institutional capacity to address shortage of an Integrated Behavioral Health (IBH) competent workforce locally, regionally and nationally. Our initiative, aligned with UTRGV strategic priorities and key initiatives, will integrate basic(model specific strategy and operational elements), mid-level (role identity and profession specific behavioral competencies specific to each health profession), and advanced (behavioral medicine clinical skills) applications of the evidence based PCBH model of delivery. A PCBH focused delivery system (clinical and educational), in which primary care providers (PCPs) and behavioral health consultants (BHCs) are trained to provide routine, population-based, biopsychosocial care in the Rio Grande Valley (RGV) can increase parity for mental health access, minimize toxic effects of culturally bound stigma, reduce fragmentation of physical-mental health and stave off the effect of an expanding opioid use disorder (OUD) crisis.

**Stress Hormone and Heart Rate Responses to Various Exercise Training Methods**

*Tomas Gomez, Rebekah Schlatter*

Background: Blood Flow Resistance (BFR) training has garnered attention for its ability to induce positive physiological adaptations with low-load resistance exercise. The present study aimed to examine the responses of catabolic hormones and heart rates (HR) to various BFR training protocols. This investigation seeks to provide insights into the stress levels induced by different protocols and identification behind the most effective protocol for optimal positive exercise-related adaptations.
s protocol more than 3 times per week, as excessive elevations in cortisol levels may have negative consequences.
Methods: Study population involved 10 healthy adult males (height: 175.0±5.0 cm, weight: 96.67±26.6 kg, age: 21.3±2.67 yr.) in a five-session investigation. Informed consent paperwork, pre-testing and anthropometric measurements served as session 1; sessions 2-5 focused on 4 different protocol-specific trainings, using a randomized within-subjects design to assess Cortisol and HR responses. The training protocols included: 20% 1-RM with BFR+rest pause (RP), 20% 1-RM with BFR+drop set (DS), 70-80% 1-RM (HI) without BFR, and 20% 1-RM without BFR with matched RP volumes (CON). Prior to RP and DS sessions, BFR pneumatic cuffs were placed around the upper third of the quadriceps bilaterally and inflated to a pressure that allows for skin to regain its color within 1-2 sec. after pressure has been applied with the thumb. Training sessions encompassed two-circuits (each with leg press and leg extension exercises) that were separated by 5 minutes of passive rest. Each exercise was performed until the participant could no longer perform the action with proper form (volitional fatigue) or until the set number of reps and sets of leg press and leg extension exercises were completed. Salivary samples that were collected pre- and post-training using passive drool saliva collection method were labeled and stored immediately by qualified study personnel at 4-8°C refrigeration.

Results: Statistical analyses revealed significant time main effect changes in CORT concentrations pre to post for RP (pConclusion: It may be speculated that notable elevations in CORT following RP protocol are indicative of greater level of stress and may optimize skeletal muscle adaptations with proper recovery and nutrition. Findings suggest that RP protocol with BFR is cardiovascularly more demanding than the CON protocol, while using similar lifting loads on Leg Extension. This implies that the RP with BRF protocol acutely enhances training intensity and may also result in cardiovascular adaptations. It should be noted that the present study examined acute CORT responses; therefore, future studies should investigate physiologic adaptations to chronic RP training with / without BFR. Since current recommendations advise individuals to limit RP training to one or two sessions per week for gaining positive physiological adaptations, RP with BFR protocol may be an alternative training technique for those who does not have enough time to meet the exercise guidelines that requires several training sessions per week. Caution should be extended to sedentary and active populations from partaking in this protocol more than 3 times per week, as excessive elevations in cortisol levels may have negative consequences.

**Unveiling the Influence of Transcription Factor YB1 in Hepatocellular Carcinoma Cell Lines**

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Background: In the Rio Grande Valley (RGV), Hispanics constitute the predominant ethnic group, accounting for over 94% of the population. Projections for 2023 indicate liver cancer to rank fifth in male and seventh in female mortality. Hepatocellular carcinoma (HCC) manifests as a significant concern within the Hispanic community, influenced by a spectrum of factors—biogenetic, social, and cultural—that intertwine with genetic predispositions and dietary practices. The incidence of HCC is intertwined with various illnesses like hepatitis, liver cirrhosis, druginduced liver injury (DILI), adding to the burden of cancer. Over the years, HCC prevalence in the RGV has surged by over 35%. A critical factor contributing to the high mortality rates is the development of drug resistance in the primary treatments. Notably, Y-Box Binding Protein 1 (YBX1) exhibits overexpression in HCC based on TCGA data, belonging to a protein superfamily governing mRNA translation. Further exploration of this protein might unveil insights into the mechanisms underpinning treatment resistance in HCC. Understanding its interactions with other pathways and genes could offer pivotal revelations for advancing therapeutic approaches.
Methods: SKHEP-1 HCC cells were obtained and cultured following the guidelines provided by ATCC. An overexpression plasmid for YBX1 (Lentiviral), featuring a Puromycin selection marker and GFP, was custom-designed and acquired from abmgood. The control and overexpression plasmids were introduced into the cells using Lipofectamine 2000. Transfected cells expressing GFP were isolated through FACS enrichment and subsequently maintained in puromycin-containing media to establish stable cell lines. The mRNA expression was assessed using RT-PCR, while protein levels were evaluated via western blot analysis. The overexpression cell lines underwent characterization through oncogenic phenotypic assays, including assessments of migration, invasion, proliferation, and colony formation. Additionally, Xcelligence was utilized to measure the impedance and proliferation dynamics of the recombinant cell lines.
Results: Our TCGA analysis highlighted the significant upregulation of YBX1 in HCC patient tumors compared to normal tissue. This elevated YBX1 expression strongly correlates with the progression and metastasis of HCC, indicating its role as an indicator of poor survival among HCC patients. Successfully achieving YBX1 overexpression via transient transfection, we enriched GFP-expressing cells and subsequently developed stable SK-Hep1 cell lines with both puromycin resistance and GFP expression, ensuring sustained YBX1 overexpression. In parallel, control cell lines were developed using an empty vector with GFP and puromycin resistance. The observed overexpression of YBX1 led to notable enhancements in proliferation, migration, invasion, and colony formation capabilities of the cell lines. This increase in YBX1 expression was confirmed at both mRNA and protein levels, establishing its impact on various cellular functions in the context of HCC progression.
Conclusion and Future Directions: We've successfully established stable SK-Hep1 cell lines with YBX1 overexpression and thoroughly characterized them. Our next step involves delving deeper into the associated proteins by conducting comprehensive proteomic studies.