IIMS INSTITUTE FOR INTEGRATION OF MEDICINE AND SCIENCE
16TH ANNUAL FRONTIERS OF TRANSLATIONAL SCIENCE RESEARCH DAY

Posterboard #: 1

Presenter: Gustavo Almeida, PT, PhD
Institution: UT Health San Antonio
Category: Faculty

Feasibility of Blood-Flow Restriction Training Prehabilitation in Older Adults Awaiting Total Knee Replacement

Gustavo J. Almeida, Mikaela Gilbert, Zachary Tittle, Diana Gasaway, Leah McBrayer, Jasmine Gongora, Jacob Lewis, Ehab Khalaf, Frank Buttacavoli, Boris Zelle*

PURPOSE: Knee osteoarthritis (KOA) often leads to progressive cartilage loss, pain, and reduced exercise tolerance in older adults. Blood Flow Restriction Training (BFRT) enables significant muscle strengthening with low loads, minimizing joint stress. This study examined the feasibility of BFRT as a prehabilitation intervention for older adults awaiting total knee replacement (TKR) and its effects on physical function and quadriceps strength. METHODS: A single-group pre-posttest design was used. Ten participants (5 male, mean age 70.6±7.6 years) completed 6 weeks of BFRT prehabilitation consisting of bilateral leg extensions, leg curls, and sit-to-stand exercises (4 sets: 30, 15, 15, 15 repetitions) with blood flow restricted to 50-80% of arterial occlusion pressure. Assessments occurred at baseline (BA), after 6-week prehabilitation (FU1), and 8 weeks post-TKR (FU2). The primary outcome was feasibility, evaluated by safety and compliance. Secondary outcomes included performance-based and self-reported physical function, quadriceps strength, and health-related quality of life. Changes were analyzed using Wilcoxon Signed-Rank Tests and Hedges' g effect sizes. RESULTS: All 10 participants completed the 12 BFRT sessions without adverse events, demonstrating excellent feasibility and compliance. Among secondary outcomes, only the 30-second Chair Stand Test showed a trend toward improvement. At FU1, 8 of 10 participants increased their chair stands (median difference=1.5 [95%CI: 0.0, 2.5]; p=.065), with 5 exceeding the minimal detectable change. At FU2, 6 participants maintained gains (median difference=1.0 [95%CI: 1.0, 1.0]; p=.253), and 4 exceeded the minimal detectable change. Effect sizes were moderate preoperatively (Hedges' g=0.66, 95%CI: 0.22-1.1, p=.043) and negligible postoperatively (g=0.07, 95%CI: -0.52-0.50, p=.882). CONCLUSIONS: BFRT is a feasible, safe, and well-tolerated prehabilitation strategy for older adults awaiting TKR. While most secondary outcomes did not reach statistical significance, BFRT showed promising potential for improving sit-to-stand performance both preoperatively and postoperatively. These preliminary findings suggest BFRT can help enhance preoperative strength and function with minimal joint stress, which may support faster recovery and better mobility after surgery. Clinicians should consider incorporating BFRT as a prehabilitation option, particularly for patients with reduced exercise tolerance due to KOA, as it offers a practical, low-load approach to optimize function and quality of life around TKR.

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Posterboard #: 2

Presenter: Curtis Bone, MD MHS
Institution: UT Health San Antonio
Category: Faculty

Exploring provider attitudes and behaviors towards opioid induced androgen deficiency (OPIAD) through the lens of the theory of planned behavior

Curtis Bone MD MHS, Chukwuemeka Okafor PhD MPH, Jimmy Arnold MBA, Jennifer S. Potter PhD MPH, Meredith Zozus PhD, Jan Bruder MD, Caroline V. M. Rebicki MD MClSc, Ashlyn Huang MD, Erin P. Finley PhD MPH

Objective: Individuals with opioid use disorder (OUD) have reduced life expectancy and inferior outcomes associated with depression, diabetes, and fractures. Opioid induced androgen deficiency (OPIAD) may contribute to these outcomes, yet few individuals prescribed opioids are evaluated for OPIAD. This study describes provider attitudes and practices related to identifying and managing OPIAD in patients on long-term opioids.

Method: We utilized the Theory of Planned Behavior (TPB) and a previously published survey of international pain specialists to develop a survey for U.S. health care providers. The REDCap-based survey was disseminated via professional networks across Texas. Eligible providers cared for patients using opioids and were able to order lab tests and treatments for OPIAD. Analyses included descriptive statistics, univariate and multivariate logistic regression grounded in TPB, conducted in Stata 16.

Results: Of 116 respondents, 105 met inclusion criteria. Most were unfamiliar with OPIAD (60.1%) or its prevalence (97.1%) and reported never screening (67.6%). Commonly cited barriers included lack of training (59.0%), absence of patient-reported concerns (47.6%), and non-conducive practice settings (33.3%). In univariate and multivariable analysis, low self-efficacy was the primary TPB factor associated with never screening ($p<0.05$).

Conclusions: Prior research demonstrates 63% of patients on chronic opioids develop OPIAD, still routine evaluation remains uncommon. These data indicate lack of training and low provider self-efficacy contribute to this care gap. Targeted education on OPIAD evaluation and management may reduce morbidity and mortality associated with chronic opioid use and OUD.

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Posterboard #: 3

Presenter: Byeongyeob Choi, PhD
Institution: UT Health San Antonio
Category: Faculty

Area deprivation index and breast cancer stage at diagnosis in Texas: a propensity score-weighted analysis

Byeongyeob Choi, Samuel V. David, Anirudh S. Babu, Yong-Fang Kuo

Background: Many studies have used the Area Deprivation Index (ADI) to examine associations between neighborhood disadvantage with advanced-stage breast cancer (ABC) at diagnosis. However, most rely on conventional regression approaches that may depend heavily on extrapolation when the distributions of individual-level characteristics differ substantially across ADI levels. In contrast, propensity score (PS) weighting methods provide a principled framework to address limited covariate overlap and to explicitly define the target populations.

Methods: We conducted a cross-sectional, population-based study using Surveillance, Epidemiology, and End Results (SEER) data linked to Medicare claims for women aged ≥ 66 years who were diagnosed with primary breast cancer between 2011 and 2017 and resided in Texas. Neighborhood disadvantage was measured using the ADI and dichotomized as > 85 (more deprived) versus ≤ 85 (less deprived). We estimated PSs for residence in more deprived neighborhoods based on observed covariates. Augmented PS-weighted estimators, which combine PS weighting with outcome regression, were used to obtain doubly robust estimates.

Results: The unadjusted risk difference (RD) was 7.07%, indicating a higher risk of ABC among women residing in neighborhoods with ADI > 85 compared with those in neighborhoods with ADI ≤ 85. After inverse probability weighting, the RD decreased to 4.15%, reflecting adjustment for observed covariates differences. Using overlap weighting, the adjusted RD was further attenuated to 3.36%, indicating reduced influence from comparisons involving individuals with extreme PSs.

Conclusions: Residence in more deprived neighborhoods was associated with a higher risk of ABC diagnosis among older women after PS adjustment. Attenuation of estimates under overlap weighting suggests that part of the observed disparity reflects comparisons involving extreme counterfactuals. Impact: By explicitly defining target populations and emphasizing individuals in equipoise, overlap weighting yields policy-relevant estimates under limited PS overlap.

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Posterboard #: 4

Presenter: Falguni Das, PhD
Institution: UT Health San Antonio
Category: Faculty

Activation of cGAS-STING Signaling and Suppression of eNOS Mediate High Glucose and Adenine-Induced Endothelial Dysfunction.

Falguni Das , Ian M Tamayo, Hak Joo Lee, Soumya Maity ,Francisca Maria Acosta and Kumar Sharma .

Hyperglycemia is a major driver of endothelial dysfunction, an early event in the development of diabetic complications. However, the underlying molecular mechanisms remain incompletely understood. Elucidating these pathways may reveal novel therapeutic targets, particularly for diabetic kidney disease (DKD). In vitro studies were performed using human umbilical vein endothelial cells (HUVECs) and murine microvascular fragments, while in vivo studies STZ-induced diabetic rats and OVE26 mice. Experimental approaches included immunoblotting, immunoprecipitation, siRNA and plasmid transfections, nitrite assays, and hypertrophy analysis. Exposure of HUVECs to high glucose (25 mM) or adenine (20 μM) induced key features of endothelial dysfunction, including reduced eNOS expression and nitric oxide (NO) production. High glucose triggered time-dependent activation of mTORC1 and mTORC2, as indicated by increased phosphorylation of S6K and Akt (Ser473). Pharmacological inhibition of PI3K (LY294002) and Akt (MK-2206) attenuated these effects and reduced hypertrophy and extracellular matrix accumulation. Similar responses were observed with adenine treatment. Both stimuli also upregulated cGAS and STING, leading to increased type I interferon and pro-inflammatory cytokine expression, linking metabolic stress to innate immune activation. Modulation of mTORC1 using rapamycin or constitutively active constructs confirmed mTOR-dependent regulation of cGAS-STING signaling. Importantly, cGAS knockdown and rapamycin treatment restored eNOS expression and NO levels. In addition, inhibition of methylthioadenosine phosphorylase (MTAP) with MTDIA reversed high glucose- and adenine-induced eNOS suppression and improved aberrant microvascular sprouting. In vivo, STZ rats and OVE26 mice exhibited decreased eNOS expression along with increased pS6K, pAkt, cGAS, STING, fibronectin, and collagen I levels in the renal cortex.

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Posterboard #: 5

Presenter: Maryam Garza, PhD, MPH, MMCi
Institution: UT Health San Antonio
Category: Faculty

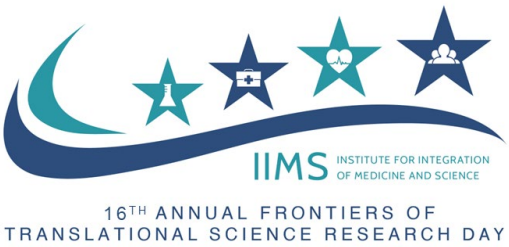
Pilot to Evaluate EHR-Based Research Functionality and Strategies to Enhance and Optimize Clinical Trial Recruitment

Maryam Y. Garza, Monica Carrizal, Muayad Hamidi, Meredith N. Zozus

Recruitment is a fundamental component of research common across clinical translational research (CTR) studies and remains a major bottleneck for researchers. As electronic health records (EHRs) have become increasingly central to healthcare delivery and have begun to offer new research functionality, a unique opportunity exists to streamline participant recruitment methods. However, the use and impact of EHR-based research functionality on study recruitment remains underexplored and little has been published regarding uptake, methods, and outcomes of EHR-based study recruitment functionality and best practices have not been established. Thus, a comprehensive understanding of existing EHR-based strategies, their effectiveness, and their limitations is critical to advancing these methods.

Characterizing current practice and remaining gaps is the critical next step in conducting the clinical translational science necessary to develop and evaluate EHR-based recruitment aids that will benefit CTR. Additionally, feasibility pilots to demonstrate that experiments evaluating EHR-based research functionality can be operationalized are a needed precursor to proposing experiments testing effectiveness of EHR-aided recruitment. We address both through the following aims: (1) Establish the current state of EHR-aided recruitment in CTR, (2) Advance EHR-aided recruitment in CTR, and (3) Demonstrate feasibility of experiments evaluating EHR-aided recruitment at our institution through a pilot study evaluating EHR-based eConsent in clinical studies. This work will characterizing current approaches, standardize outcomes and methods for assessing EHR-based recruitment aids, and provide actionable insights for improving EHR-based recruitment strategies across different healthcare settings and CTR studies. Any gains through EHR-aided recruitment will directly impact the efficiency of clinical studies. Gains in evaluation methods will help trialists optimize recruitment faster.

The outcomes of this initiative have the potential to reshape recruitment practices, making them more efficient and inclusive. Moreover, this work will address a major rate-limiting factor in clinical translational science (participant recruitment) and identify potential EHR-based solutions that benefit all investigators across the clinical research spectrum. We anticipate the results of this study will provide preliminary data needed to support future grant submissions targeted at optimizing recruitment efforts across clinical translational research.



Posterboard #: 6

Presenter: Muayad Hamidi, MBChB
Institution: UT Health San Antonio
Category: Faculty

Validation of Multi-Source Mortality Data: NDI Benchmarking of Two Health Systems and SSADMF Death Information Concordance

Muayad Hamidi, MBChB, Zhan Wang, Ph.D, Mahanaz Syed, Ph.D, Eric L. Eisenstein, DBA, Shweta Bansal, MD, Meredith N. Zozus, Ph.D

Multiple data sources document mortality in the United States, yet obtaining high-quality, comprehensive death information remains a significant challenge for clinical research and healthcare operations. Our previous work revealed substantial discordance between the Electronic Health Records (EHRs) of two health systems and the Social Security Administration Death Master File (SSADMF). This follow-up study extends that work by incorporating the National Death Index (NDI). To evaluate the concordance of death information from two health systems serving the same geographic region and SSADMF, we compared them against the NDI using a stratified sampling design across patient likelihood of being deceased categories. Using a stratified sampling approach to request NDI data, we identified four population groups based on likelihood of being deceased: (1) very likely deceased (indicated as dead in EHR); (2) somewhat likely deceased (age >75 with no visit for 12 months); (3) less likely deceased (random sample of younger ages stratified by 15-year increments with no visits for 12 months); and (4) very likely alive (recent active encounters within 12 months). We compared the data of two health systems and SSADMF with NDI data to get the concordance of these data sources. We also applied classification rules to identify and adjudicate discrepancies. Among the 85,531 patient records evaluated, System A demonstrated higher sensitivity consistent with NDI (44.61%, 95% CI: 43.95%-45.27%) compared to SSA-DMF (6.22%, 95% CI: 5.90%-6.55%) and System B (9.20%, 95% CI: 8.82%-9.59%). Chart review of discrepant records identified patterns consistent with NDI linkage errors (42 of 101 cases with potential post-death encounters), anomalous data activities or data errors (16 cases), and valid health systems records missed by NDI (48 cases). NDI is the most confident data source for mortality ascertainment compared to EHR or SSADMF data. Neither EHR nor SSADMF could be used as a confident data source alone. Integration of multiple data sources combined with systematic data quality evaluation rules is essential for identifying and resolving death record discordances. Healthcare institutions should implement processes to obtain and validate external mortality data sources to ensure accuracy and completeness of mortality information for clinical research and quality improvement initiatives.

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Posterboard #: 7

Presenter: Rebecca Jones, PhD
Institution: UT Health San Antonio
Category: Faculty

Advancing Community-Engaged Research Through Small Grants: A Content Analysis of Community Engagement Small Project Grants (2015-2024)

Elisabeth M. de la Rosa and Rebecca Jones

Background: Since 2015, the Institute for Integration of Medicine & Science (IIMS), in collaboration with partners including UTSA College for Health Community and Policy (HCAP), has awarded 54 Community Engagement Small Project Grants (CESPGs) to support community-academic partnerships addressing locally identified health priorities across South Texas. These grants-up to $5,000 each-support new and established partnerships and aim to advance the translation of research into public health benefit across research, training, and dissemination activities.

Methods: We conducted a three-phase content analysis of CESPG-funded projects from 2015-2024 to examine project characteristics, partnership processes, and contributions to translational science. Data were abstracted from proposals, final reports, and dissemination products. An iterative coding framework was developed and applied to identify key themes.

Results: Of 106 applications submitted, 54 were funded, representing a total investment of $245,000. At the time of analysis, 48 completed projects had sufficient documentation for review. Several cross-cutting themes emerged. Partnerships were strengthened through trust-building, cultural humility, and shared leadership. Projects demonstrated adaptability to evolving community needs and context-specific challenges. Sustainability planning was evident, with many partnerships continuing beyond the funding period through ongoing activities and leveraged resources. CESPGs also supported co-learning and mutual capacity-building among academic and community partners. Additionally, projects generated practical tools-including curricula, educational materials, mobile applications, and survey instruments-for continued community use.

Conclusions: Small-scale funding mechanisms such as CESPGs play a critical role in advancing community-engaged and translational research by fostering equitable partnerships and supporting community-driven solutions.

Implications for Community Engaged Research: Investing in community-academic partnerships through flexible, small-scale funding can strengthen local capacity, enhance relevance of research, and support sustainable, community-informed public health impact.

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Posterboard #: 8

Presenter: Soumya Maity, PhD
Institution: UT Health San Antonio
Category: Faculty

ATP-Citrate Lyase Is a Potential Therapeutic Target for Diabetic Kidney Disease

ATP-Citrate Lyase Is a Potential Therapeutic Target for Diabetic Kidney Disease

Diabetic kidney disease (DKD) is the leading cause of end-stage kidney disease and kidney failure worldwide, and mitochondrial dysfunction is closely associated with its progression. However, metabolic mechanisms linking mitochondrial dysfunction to pathogenic signaling remain incompletely understood. Citrate, a central intermediate of the tricarboxylic acid (TCA) cycle, links mitochondrial metabolism with cytosolic anabolic pathways. Citrate is exported from mitochondria to the cytosol via SLC25A1, where ATP-citrate lyase (ACLY) converts it to acetyl-CoA for de novo lipogenesis. Previous clinical studies have shown reduced urinary citrate excretion in patients with DKD compared with individuals with type 2 diabetes without kidney disease, suggesting decreased citrate levels are associated with kidney injury rather than hyperglycemia. Here, we investigated the role of intracellular citrate homeostasis in DKD pathology.

Spatial metabolomics using MALDI-MSI of human kidney biopsies from the Kidney Precision Medicine Project revealed significantly reduced citrate abundance in DKD kidneys compared with healthy controls, with lower levels in tubular regions relative to other areas. Multimodal spatial imaging further demonstrated that tubular regions with higher phosphorylated ribosomal protein S6 exhibit lower citrate abundance, linking reduced citrate to increased mTORC1 activity in DKD. Single-cell transcriptomic analysis showed proximal tubule-specific downregulation of most TCA cycle genes and both apical and basolateral citrate transporters with preserved expression of citrate synthase and SLC25A1, indicating dysregulation of intracellular tubular citrate homeostasis with partial TCA cycle maintenance. In addition, ACLY expression was significantly increased in proximal tubules, suggesting reduced tubular citrate levels are driven by enhanced conversion of cytosolic citrate to acetyl-CoA. Mechanistic studies in human proximal tubular epithelial cells demonstrated that suppressing mitochondrial citrate export using siRNA against SLC25A1 reduced cytosolic citrate availability and activated mTORC1, whereas citrate supplementation attenuated both SLC25A1 knockdown-mediated and high glucose (HG)-induced mTORC1 activation. Furthermore, increasing cytosolic citrate through ACLY inhibition suppressed HG-induced mTORC1 activation and fibronectin expression.

Together, these findings identify intracellular citrate deficiency as a metabolic driver of mTORC1 activation in DKD and suggest that targeting ACLY to restore citrate homeostasis may represent a novel metabolic strategy to attenuate kidney injury and slow disease progression in type 2 diabetes.

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Posterboard #: 9

Presenter: Aurora Merovci, MD, MPH
Institution: UT Health San Antonio
Category: Faculty

Effects of Empagliflozin on Hepatic Glucose Production, Gluconeogenesis, and Lipolysis in Type 2 Diabetes

Aurora Merovci

Background:
We previously showed that SGLT2 inhibitors stimulate endogenous glucose production, offsetting their glucosuria-induced glucose lowering action, and that this effect is not explained by changes in plasma glucose, insulin, or glucagon concentration using a pancreatic clamp.

Objective:
To investigate the impact of Empagliflozin (an SGLT2 inhibitor) on fasting hepatic glucose production (HGP), gluconeogenic flux, and lipolytic activity in type 2 diabetes individuals.

Methods:
15 individuals with type 2 diabetes (Age 57 ± 2 yrs, HbA1c = 9.0 ± 1.0%; BMI 31 ± 1.5 kg/m²) underwent a tracer-based metabolic study with 6, 6-^{2}H2-Glucose (to measure HGP) and $^{2}H_5$-Glycerol (to measure lipolysis). Tracers were infused continuously from -180 min to 300 min (total 480 min). D2O (to measure gluconeogenesis) was ingested on the night prior to study. Empagliflozin 25 mg was ingested at time 0 min. This design allowed quantitation of baseline (following overnight fast) and hepatic and adipocyte responses to SGLT2 inhibition over a 5-hour treatment period.

Results:
Baseline (following overnight fast) HGP and gluconeogenesis were 2.33 and 1.57 mg/kg/min, respectively. Following Empagliflozin gluconeogenesis (GNG) increased to 1.79 mg/kg/min ($p<0.01$), while HGP increased to 2.45 mg/kg.min ($p<0.01$). Baseline glycerol Ra was 3.57 µmol/kg/min and following empagliflozin increased to 4.02 µmol/kg/min ($p<0.01$), in association with increases in plasma glycerol (134 to 157 µmol/L, $p<0.01$) and FFA (0.51 to 0.71 µmol/L, $p<0.01$) concentrations.

Conclusions:
SGLT2 inhibiton with empagliflozin: (i) stimulates gluconeogenesis, preventing the normal fasting - induced increase in HGP and (ii) augments lipolysis, providing glycerol as a substrate to drive gluconeogenesis. This explains why SGLT2i do not cause hypoglycemia.

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Posterboard #: 10

Presenter: Rocio Norman, PhD
Institution: UT Health San Antonio
Category: Faculty

Cross Domain Stability in Cognitive Communication and Processing Outcomes Among Women with Mild Traumatic Brain Injury

Dr. Rocio Norman, Gabriela McNelly

Women with mild traumatic brain injury (mTBI) remain critically underrepresented in the TBI literature, despite growing evidence that they experience distinct symptom trajectories and may be disproportionately affected by persistent symptoms. In moderate-severe TBI, chronicity is a well established predictor of recovery, with characteristic early gains, plateau phases, and later emerging deficits across cognitive and communication domains. Whether similar time dependent patterns occur in women with mTBI is not well understood. This study examined whether chronicity predicts differences in subjective communication complaints, objective processing speed, and performance on a speeded language comprehension task in a sample of adult women with mTBI. Twenty women were assigned to two chronicity groups: <1095 days post injury (n = 8) and >1095 days (n = 12). Participants completed the La Trobe Communication Questionnaire (LCQ), the WAIS IV Processing Speed Index (PSI), and a speeded language comprehension task measuring accuracy, reaction time (RT), and RT variability. Mann-Whitney U tests and multivariate visualizations were used to evaluate group differences. Across all domains, chronicity did not predict performance. LCQ scores were comparable between groups ($U = 46.5$, $p = .90$), indicating stable self reported communication difficulties. PSI scores also did not differ ($U = 47.5$, $p = .51$), suggesting consistent processing efficiency across time. Similarly, accuracy ($U = 45.0$, $p = .93$), RT ($U = 44.0$, $p = .88$), and RT variability ($U = 43.0$, $p = .84$) showed no chronicity related differences. Multivariate profiles revealed stable cross domain patterns and greater variability in the chronic group without systematic improvement or decline. These findings contrast with moderate-severe TBI trajectories and contribute to the emerging understanding that women with mTBI may experience persistent yet stable cognitive communication profiles. Prior research suggests that hormonal influences, sex specific neuroinflammatory responses, and heightened awareness may shape symptom expression in women, potentially explaining the dissociation observed here between subjective complaints and objective performance. Clinically, these results underscore the need for sex responsive, symptom based monitoring, rather than assumptions of recovery based on time post injury. Women with mTBI may benefit from ongoing access to services regardless of chronicity, reflecting the unique and enduring nature of

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Posterboard #: 11

Presenter: Anjali Sivaramakrishnan, PT, PhD
Institution: UT Health San Antonio
Category: Faculty

Exercise training combined with virtual reality-based games can improve balance in Parkinson's disease

Anjali Sivaramakrishnan, Alyssa Main, Jonathan Gelfond, Chun-Liang Chen, Okeanis Vaou and Daniel Corcos

Introduction: Postural instability is a hallmark motor symptom in Parkinson's disease (PD) and a major contributor to falls. Non-motor symptoms such as cognitive impairment are associated with postural instability, and increased risk of fall. Aerobic exercise training is an intervention that can increase cardiovascular fitness and improve motor symptoms and cognition in individuals with PD. When aerobic exercise is combined with targeted cognitive-motor rehabilitation such as virtual reality (VR) based training, it could produce additive effects and potentially reduce the risk of fall. This pilot study investigated the effects of exercise training followed by VR games on balance and cognition in PD. Methods: Individuals with PD were randomized to high intensity interval training (HIIT) and VR games (HIIT-VR, n=8) or stretching and VR games (St-VR, n=7). The HIIT-VR group performed 30 minutes of exercise (intervals at >80% heart rate maximum) on a recumbent stepper before 30 minutes of VR games thrice per week for 8 weeks. The dose-matched St-VR group performed whole body stretches prior to VR games. Outcomes were obtained at baseline, post-intervention, and 6 week follow up. The primary outcome was the mini-Balance Evaluation Systems Test (miniBESTest). Secondary outcomes included 6-minute walk distance (6MWD), postural sway, PD-cognition rating scale, spatiotemporal measures of gait during single and dual task conditions and quality of life. Results: Fifteen individuals [mean ± SD age: 68.1 ± 10.5 years, Hoehn and Yahr stages 2-3] participated in this study. Significant group by time interactions were observed for the miniBESTest, [$F(1.3, 16.9) = 5.36$, $p = 0.02$] and 6MWD, [$F(2, 26) = 5.7$, $p = 0.009$]. The HIIT-VR group showed greater change in the miniBESTest (6.3 points, 95% CI 0.6, 12) and 6MWD (141.3 m, 95% CI 7.5, 275) compared to the St-VR group at post assessment. There were no differences between groups in measures of postural sway, cognition, gait and quality of life. Conclusion: Our findings show that combining HIIT with VR-based training appears to elicit meaningful gains in balance and endurance, suggesting that it may facilitate motor learning and improve rehabilitative outcomes in PD. It is plausible that increased cardiorespiratory fitness in the HIIT-VR group

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Posterboard #: 12

Presenter: Mahanaz Syed, PhD
Institution: UT Health San Antonio
Category: Faculty

AI-Based Eligibility Reasoning over EHR Data for Clinical Trial Recruitment

Mahanaz Syed, Muayad Hamidi, Manju Bikkanuri, Meredith Zozus, Antonio Teixeira

Background: Clinical trial recruitment remains a major bottleneck in translational research, requiring manual review of complex eligibility criteria against heterogeneous electronic health record (EHR) data. While structured queries can identify broad cohorts, key eligibility details are often embedded in unstructured clinical notes, limiting efficiency and scalability.

Objective: To develop and evaluate an artificial intelligence (AI)-based framework that automates trial eligibility assessment by combining trial criteria retrieval with clinical note interpretation.

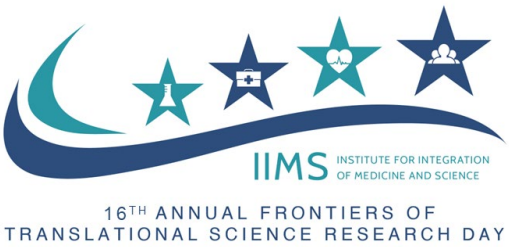
Methods: We implemented an end-to-end pipeline that retrieves study eligibility criteria from ClinicalTrials.gov via API and applies a locally deployed large language model to evaluate patient eligibility using unstructured EHR notes. The system extends the TrialGPT framework through a patient-encounter-note representation of longitudinal data and performs criterion-level reasoning to generate patient-level eligibility predictions. Performance was evaluated against an expert-adjudicated reference standard (n=149) and compared with manual screening (n=55).

Results: The system achieved high agreement with expert review (sensitivity 81.8%, specificity 97.8%) and identified more than twice as many eligible patients compared to manual screening (81.8% vs 36.4%) while maintaining comparable specificity. Automation reduced manual chart review burden and enabled scalable pre-screening across patient populations.

Innovation and Impact: This framework integrates automated trial criteria retrieval with AI-based eligibility reasoning over clinical narratives, enabling interpretation of complex inclusion and exclusion criteria directly from EHR data. Unlike traditional cohort discovery tools, it operates on both trial definitions and patient records, bridging the gap between protocol design and real-world data.

The system operates within institutional infrastructure using locally hosted models, ensuring privacy, reproducibility, and operational feasibility. The results presented are derived from a validated real-world clinical trial implementation (JAMIA, 2026). Ongoing work aims to incorporate structured EHR data and assess generalizability across multiple trials and settings.

Conclusion: AI-based eligibility reasoning over EHR data provides a scalable, institution-ready approach to improve clinical trial recruitment by transforming trial criteria and clinical documentation into actionable screening intelligence.



Posterboard #: 13

Presenter: Tamara Alhasanat, BS
Institution: UT Health San Antonio
Category: Other: Research Coordinator

Bridging Oral and Nutritional Health: A Pilot Food Insecurity Initiative in Dental Clinics

Tamara Alhasanat, Dr. Alexander Testa, Dr. Rahma Mungia, Caitlin Sangdahl, Manju Bikkanuri

Background: Food insecurity (FI), defined as limited or uncertain access to adequate and nutritious food, is a key social determinant linked to poor overall and oral health, yet dental settings remain underutilized for screening and referral. This study evaluates the feasibility of integrating food insecurity screening and referral into routine dental care.

Methods: This pilot study evaluates the feasibility and acceptability of integrating the Hunger Vital Sign™ (HVS) FI screener into routine dental care and establishing a direct referral pathway to the San Antonio Food Bank (SAFB). Adult patients presenting for routine care at three South Texas Oral Health Network (STOHN) clinics are screened using the two-item HVS via REDCap. Patients who respond "often true" or "sometimes true" to either item are classified as food insecure and invited to participate. Baseline assessments include the SPAN dietary screener, Oral Health Impact Profile (OHIP-5), self-reported oral health measures, and clinical indicators (gingival index [GI], plaque index [PI], and bleeding on probing [BOP]).

Results: To date, over 100 patients have been screened, with 16 identified as food insecure and referred to SAFB. Implementation across clinics has been successful, with consistent use of screening tools and referral workflows. Preliminary findings indicate that integrating FI screening into dental visits is feasible and acceptable to both patients and providers.

Conclusion: Integrating FI screening and referral within dental settings is feasible and shows promise as an approach to address nutrition-related health disparities. Expanding the role of dental providers in identifying and addressing food insecurity may improve both oral and overall health outcomes, particularly in underserved communities.

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Posterboard #: 14

Presenter: Sarah Dean, BS
Institution: UT Health San Antonio
Category: Other: Research Assistant

ADRD Biomarker Stability in HEMAcollect PROTEIN Blood Collection Tubes

Sarah Dean, Jake Pap, Avery Zuniga, Haritha Katragadda, Rose Ann Barajas, Claudia Satizabal, Tiffany Kautz

Blood plasma biomarkers are now used to support diagnosis, staging, and monitoring for Alzheimer's disease and related dementias (ADRDs). Traditional ADRD blood biomarker collection uses EDTA-treated tubes, which require same-day processing and ultracold storage, limiting their utility in under-resourced and rural settings. However, HEMAcollect PROTEIN (HCP) blood collection tubes (BCT) preserve proteins at room temperature (RT) for up to seven days. We hypothesized that ADRD biomarkers would remain stable in HCP BCTs stored at room temperature, whereas EDTA-derived biomarkers would degrade.

Blood was collected into four EDTA and four HCP BCTs. One EDTA and HCP BCT were centrifuged immediately after collection (day 0, D0). Plasma aliquots were stored at -80°C. Other BCTs were stored at RT until processing at day 4 (D4), D7, and D10. ADRD biomarkers were measured in duplicate using the Alamar NULISAseq CNS Disease Panel 120 and only used for analysis if they passed QC (i.e. sample biomarker detectability >80%, individual biomarker detectability >75%). For each biomarker, measurements at D4, D7, and D10 are expressed as the percent change relative to D0.

EDTA showed low CVs (mean CV=0.71%), outperforming the HCP BCTs (mean CV=2.57%). Overall detectability was also worse in HCP BCTs (90.03%) compared to EDTA (94.86%). Several biomarkers (APOE4, IL17A, PTN, SNCB, UCHL1) failed QC for both, while additional failures occurred in HCP BCTs (BD-pTau-217, BD-pTau-231, VGF, pTDP43-409, and pTau-217). By D7, EDTA Aβ38 decreased 22.95% compared to HCP BCTs which decreased 15.64%. EDTA Aβ40 decreased by 25.77%. In contrast, HCP BCTs also had decreases in CD40LG (33.14%), IL10 (18.17%), NPY 22.89%), Oligo-SNCA (0.04%), SQSTM1 (19.30%), and TARDBP (30.54%). Several biomarkers increased in EDTA tubes, including ANXA5 (65.57%), ARSA (47.58%), CXCL8 (58.77%), IL1B (73.69%), PGK1 (75.04%), PSEN1 (52.37%), S100A12 (62.91%), and HBA1 (77.37%). HBA1 also increased in HCP BCTs by 210.18%.

In conclusion, contrary to our hypothesis, HCP BCTs had lower detectability rates and more biomarker degradation over time. However, EDTA BCTs were more likely to experience biomarker increases, likely due to hemolysis. In the future, we will replicate this experiment using a second purification step recommended by the HCP BCT manufacturer.

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Posterboard #: 15

Presenter: Samantha Gates, BA
Institution: UT Health San Antonio
Category: Other: Research Assistant

Sleep, Cognition, and Depression: Sex-specific Analyses from the Framingham Heart Study

Samantha N. Gates, Vanessa M. Young, Rebecca Bernal, Andree-Ann Baril, Arash Salardini, Crystal Wiedner, Carlos Gaona, Matthew P. Pase, Alexa Beiser, Antonio Lucio Teixeira, Sudha Seshadri, Jayandra Jung Himali

Sleep patterns exhibit marked sex differences, with females disproportionately reporting sleep disturbances and extremes in duration. Similarly, depression is more prevalent in females, frequently co-occurs with sleep disturbances, and may modify sleep-cognition relationships. Despite this evidence, sex-specific interactions among sleep duration, depression severity, and treatment remain underexplored. This analysis extends prior work (Young et al., 2025) by examining sex-specific associations between sleep duration and cognition, and the moderating role of depression.

The sample included middle-aged adults (mean age ≈ 50 years) in the Third-Generation, Omni 2, and New Offspring Framingham Heart Study cohorts. Participants were a priori sex-stratified and categorized into three sleep groups: short (≤6h), average (>6 to <9h; reference), and long (≥9h). Depression status was defined using CES-D scores (≥16) and antidepressant use, yielding four groups: control (no depression/no medication), medication only (CES-D <16/antidepressant use), depression only (CES-D ≥16/no medication), and both conditions (CES-D ≥16/antidepressant use). We conducted multivariable linear regressions adjusted for age, education, comorbidities, and lifestyle factors. Interactions between sleep duration and depression status were tested.

In males, long sleep duration was significantly associated with reduced performance in visual reproduction (β±SE= -1.64±0.75, p = 0.030) and logical memory (-2.05±1.04, p = 0.049). Notably, untreated depressive symptoms in males sleeping ≤6 hours were linked to better similarities test performance (2.53±1.03, p = 0.018), suggesting a complex interplay between sleep, cognition, and depression.
In females, long sleep duration was associated with worse global cognition (-0.27±0.08, p = 0.001), slower trail B (-0.13±0.04, p = 0.002), and poorer visual reproduction (-1.80±0.51, p = <0.001). In stratification analyses, females with long sleep duration, antidepressant use, and depressive symptoms exhibited deficits in global cognition (-1.05±0.29, p = 0.001) and visual reproduction (-5.78 ±1.96, p = 0.006).

Long sleep duration is a sex-differentiated marker of cognitive risk, with females exhibiting heightened vulnerability, particularly when depression and antidepressant use coincide. These findings emphasize the need for sex-specific research and targeted clinical interventions to optimize sleep duration and mitigate cognitive decline.

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Posterboard #: 16

Presenter: Diamond Hercules, BS
Institution: UT Health San Antonio
Category: Other: Community Health Worker

Community Perceptions of Long COVID, Medical Mistrust, and Mental Health

Diamond Hercules, Salomé Adelia Wilfred, Karen Morado, Raquel Romero, Sarah Lill, Ludivina Hernandez, Ariel Gomez, Taylur Loera, & Lisa Smith Kilpela

Marginalized communities often report higher levels of medical mistrust due to historical neglect and discrimination by the healthcare system. This pattern of vulnerability was evident through the evolution of the COVID-19 pandemic. Long COVID is a chronic illness in which an individual infected by the COVID-19 virus experiences a wide range of health issues, such as fatigue, brain fog, and/or hair loss, for at least three months afterwards. It is important to include community voices in research efforts to reduce the knowledge gap between healthcare professionals and marginalized communities. The following study investigates the relationship between community awareness of Long COVID, medical mistrust, and mental health. Participants were recruited by word-of-mouth and flyers placed in community spaces. To ensure accessibility, focus groups were held at local community centers and conducted in both English and Spanish. Participants were aware that conversations would focus on Long COVID, but knowledge on this condition was not necessary to participate. Prior to each session, participants completed a self-report questionnaire on knowledge and past experiences with Long COVID, measures of depression (PHQ-9), anxiety (GAD-7), general mental health (PROMIS-10), and medical mistrust (GBMMS). Data collection is ongoing with 16 focus groups conducted thus far. Participants (N = 140) aged 22-83, primarily identified as Hispanic/Latina (85.7%), female (71.4%), and married or living with a partner (53.6%). Roughly half of the sample reported high levels of medical mistrust (M=29.892, SD = 8.610). Similarly, 75% of participants reported average to below mental well-being (M = 46.158, SD = 8.706). Roughly half of the sample reported mild to moderate depression (M = 5.971, SD = 5.721) and 44% reported mild to severe anxiety (M = 5.648, SD = 5.385). Most participants (86%) reported little to no knowledge on Long COVID resources and 82% reported a general unawareness and lack of concern for this illness in their community. Findings highlight the need to assess community awareness of Long COVID and to provide resources to combat the unease surrounding this illness. This is especially important for historically overlooked and underserved populations, to alleviate the mental and physical health disparities that these communities face.

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Posterboard #: 17

Presenter: Ysabel Rose Lew
Institution: UT Health San Antonio
Category: Other: Research Assistant

Addressing Knowledge Gaps in Gastrointestinal Cancer Risk Among Hispanic/Latino Survivors in South Texas

Ysabel Rose Lew, BA, Derek Rodriguez, PhD, Patricia Chalela, DrPH, Natalie Rodriguez, MBA, Juan Nevarez Ramos, BA, Jacqueline Cardenas, BS, Carla Carmona, BA, Gabriella Santillan, BS, Edgar Muñoz, MS, Amelie G. Ramirez, DrPH, Frank Penedo, PhD

Background: Cancer is one of the leading causes of mortality among Hispanic/Latino populations in Texas, with gastrointestinal (GI) cancers demonstrating particularly low survival rates. With higher risk of delayed diagnosis and care, Hispanic/Latino colorectal and stomach cancer survivors face recurrence rates of approximately 30% and 39%, respectively. Limited awareness of cancer risk factors may contribute to disparities in survivorship outcomes. This study evaluates knowledge gaps in colorectal and stomach cancer risk factors among Hispanic/Latino survivors to inform culturally tailored survivorship plans.

Methods: A cross-sectional analysis was conducted using baseline data from Avanzado Caminos/Leading Pathways, an ongoing NCI-funded prospective cohort study on Hispanic/Latino cancer survivors who completed primary treatment within the past 10 years. Colorectal and stomach cancer survivors (N=78) completed self-report questionnaires, including items adapted from the NIH Health Information National Trends Survey to assess cancer risk factor knowledge. Linear regression models assessed associations between sociodemographic factors, cancer type, and beliefs about colorectal and stomach cancer risk factors, adjusting for relevant covariates.

Results: Results show that female survivors were less likely to believe that lack of fruit and vegetable consumption is strongly associated with cancer risk (β=-0.396, p=0.046). Those with a high school/GED education or less were less likely to believe that lack of exercise is strongly associated with cancer risk (β=-0.409, p=0.050). Stomach survivors were less likely to believe that radon exposure is strongly associated with cancer risk (β=-1.070, p=0.022). These findings highlight disparities in risk factor awareness across demographic and clinical subgroups.

Conclusion: Targeted, culturally responsive survivorship education is needed to address knowledge gaps in GI cancer risk factors among Hispanic/Latino survivors. Integrating tailored health education into survivorship care may improve health behaviors and outcomes, reduce recurrence risk in survivors, and curb the cost of cancer care for families and across healthcare systems.

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Posterboard #: 18

Presenter: Sarai Llamas, MBA
Institution: UT Health San Antonio
Category: Other: Research Area Specialist

Therapeutic Yoga Improves Quality of Life and Reduces Perceived Stress in Women with Breast Cancer: A 6-Month Intervention in South Texas Communities

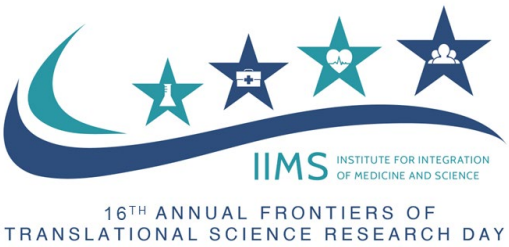
Sarai E. Llamas1, Gustavo J. Almeida1 , Natalie A. Rodriguez1, Daniel C. Hughes2 , Celeste Sanchez2 , Nydia Darby3 , Tim Calderon1 , Tony (Dongxiao) Zhang4 , Julie Bazan5 , Mitzi Deselles-Apter6 , Mary Ayon7, Bryanna Scheuler8 , Joseph Houpt9 , Amelie G

BACKGROUND: Therapeutic yoga has shown potential benefits for psychosocial and health-related quality of life outcomes among women with breast cancer, yet evidence in diverse community settings remains limited. This study evaluated changes in quality of life, perceived stress, and self-efficacy-related barriers among women with breast cancer in San Antonio (SA) and Laredo (LRD), Texas, participating in a 6-month therapeutic yoga intervention.

METHODS: Women with breast cancer from SA and LRD participated in a 6-month therapeutic yoga program conducted three times per week. Outcomes were assessed at baseline (BA) and follow-up (FU). Health-related quality of life was measured using the SF-36 questionnaire, perceived stress using the Perceived Stress Scale (PSS), and barriers to self-efficacy using the Barriers of Self-Efficacy scale (BSE). Paired t-tests evaluated BA-FU changes, and Welch t-tests compared SA and LRD groups at BA, FU, and for change scores.

RESULTS: Forty-two women were included (67% Latinas, 61±9 years old). Out of the 42, 4 did not complete their assessments, and other 9 did not complete their FU assessment. Thus, we analyzed data from 29 who completed all assessments (19 SA, 10 LRD). Significant improvements BA-FU were observed across multiple SF-36 domains, including physical functioning, vitality, mental health, social functioning, bodily pain, and general health (all $p \leq .020$). Perceived stress significantly decreased (mean change −5.3; 95%CI:−7.9,−2.7; $p < .001$). Barriers of self-efficacy increased over time (mean change +5.9; 95%CI: 1.8,9.9; $p = .006$). No statistically significant differences between SA and LRD groups were found for SF-36, PSS, or BSE total scores, although greater improvements in SF-36 domains of social functioning and bodily pain were observed in SA compared with LRD.

CONCLUSION: A 6-month therapeutic yoga intervention was associated with meaningful improvements in quality of life and reduced perceived stress among women with breast cancer in South Texas, with comparable benefits across urban and border-community settings.



Posterboard #: 19

Presenter: Yelitza Ramirez, MS
Institution: UT Health San Antonio
Category: Other: Research Coordinator

Longitudinal Variability of Plasma Biomarkers in the South Texas Alzheimer's Disease Research Center's Cohort

Yelitza Ramirez, Jennifer Del Bosque, Juan Garcia, Ashley LaRoche, A.Campbell Sullivan, Silvia Mejia-Arango, Joanne Curran, Gladys Maestra, Sudha Seshadri, Tiffany Kautz

Plasma biomarkers for Alzheimer's disease (AD) are increasingly used for patient care due to their minimal invasiveness, cost-effectiveness, and scalability compared to cerebrospinal fluid and positron emission tomography gold-standard measures. Of these, plasma ptau217 is the most accurate for distinguishing AD from other neurodegenerative conditions and cognitively normal individuals. Despite well-established population-level trends, intraindividual longitudinal changes in plasma biomarkers remain insufficiently understood, raising concerns about clinical reliability.

We examined longitudinal fluctuations in plasma pTau217, glial fibrillary acidic protein (GFAP), and neurofilament light chain (NfL) using data from the South Texas Alzheimer's Disease Research Center (STAC). Participants (n=323, mean age 71 (SD=10; 58% female; 46% Hispanic), underwent annual biomarker assessments (54% cognitively unimpaired, 28% mild cognitive impairment, and 17% dementia. Within-person variability was quantified using absolute and percentage changes between annual visits. Clinical impact was evaluated using a predefined ptau217 cut-off of >0.47pg/mL to assess diagnostic classification stability.

Substantial intra-individual variability was observed across all biomarkers. At visit 1 (V1), the average GFAP concentration was 129.73pg/mL (SD=103.19; range 8.42-928.2), NfL was 15.0pg/mL (SD=12.49; range 2.37-113.72), and pTau217 was 0.59pg/mL (SD=0.54; range 0.1-4.64). From V1 to V2, GFAP increased in 56% (n=182; n=118 with >20% increase) and decreased in 43% (n=139; n=79 with >20% decrease). NfL increased in 64% (n=206; n=146 with >20% increase) and decreased in 36% (n=116; n=59 with >20% decrease). pTau217 increased in 65% (n=190; n=127 with >20% increase) and decreased in 35% (n=101; 49 with >20% decrease).

At V1, 41% (n=122) of participants were above the ptau217 cutoff. Of these, 10% (n=12) dropped below the cutoff at V2. The single participant with V3 data remained below. Of those below, the ptau217 cutoff at V1, 14% (n=23) surpassed the cutoff at V2. However, two of the five participants with V3 data decreased below the cutoff.

These findings demonstrate that plasma biomarkers exhibit meaningful longitudinal variability, including bidirectional shifts across clinically relevant diagnostic thresholds. This instability has important implications for their use in longitudinal monitoring and clinical decision-making. Further investigation is required to identify any factors driving this variability and to develop strategies for improving measurement reliability.

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Posterboard #: 20

Presenter: Derek Rodriguez, PhD
Institution: UT Health San Antonio
Category: Other: Research Scientist

Integrating non-medical drivers of health screening among Hispanic/Latina women cancer survivors in South Texas

Derek Rodriguez, PhD Byeong Yeob Choi, PhD Ysabel Rose Lew, BA Natalie Rodriguez, Juan C. Nevarez Ramos, BA Jacqueline Cardenas, BS Carla Carmona, BA Gabriella Santillan, BS, Edgar Munoz, MS1 Patricia Chalela, DrPH1 Frank J. Penedo, PhD, Amelie G. Ramirez

Background: Hispanic/Latina (H/L) women in the United States-particularly those living in medically underrepresented regions such as South Texas (STX)-experience persistent disparities in cancer survivorship, including poorer quality of life (QOL) and limited access to supportive care. Few studies have quantitatively examined how multiple non-medical drivers of health (NMDoH) jointly influence QOL among Latina breast and cervical cancer survivors living in border regions.

Objective: To identify non-medical drivers of health associated with quality of life among Latina breast and cervical cancer survivors residing in South Texas.

Methods: We conducted a cross-sectional analysis using baseline data from Avanzando Caminos, an ongoing NCI-funded prospective cohort study of Hispanic/Latino cancer survivors. The analytic sample included H/L women (n = 170) who completed treatment for breast or cervical cancer within the past 10 years. NMDoH were assessed by aligning items from the Avanzando Salud NMDoH Screener with cohort variables, including financial strain, transportation needs, physical activity, healthcare access, and social support. QOL was measured using site-specific Functional Assessment of Cancer Therapy instruments (FACT-B for breast cancer; FACT-Cx for cervical cancer). Multivariable linear regression models estimated associations between NMDoH and QOL, adjusting for sociodemographic and clinical covariates.

Results: Across both cancer groups, greater financial strain was significantly associated with lower QOL (breast: $\beta = -3.41$; cervical: $\beta = -8.65$; $p < 0.05$). Among cervical cancer survivors, a higher number of comorbidities was associated with lower QOL ($\beta = -4.49$; $p < 0.01$). Among breast cancer survivors, exercise engagement was associated with higher QOL ($\beta = 4.85$; $p < 0.05$). Similar trends were observed among cervical cancer survivors; however, these associations did not reach statistical significance, likely due to limited sample size.

Conclusion: Lower QOL among Latina cancer survivors was associated with unmet NMDoH needs, particularly financial hardship and comorbid conditions. Associations were stronger and more consistent among breast cancer survivors than cervical cancer survivors. These findings underscore the importance of integrating systematic NMDoH screening into survivorship care and implementing culturally tailored, community-based interventions-such as patient navigation and community health worker models-to improve long-term outcomes for Hispanic/Latina cancer survivors in underserved regions.

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Posterboard #: 21

Presenter: Marco Sciortino, BS
Institution: UT Health San Antonio
Category: Other: Research Assistant

Investigating the Effects of Small Molecule Inhibitors on α-Synuclein Accumulation in a Drosophila melanogaster Model

Marco Sciortino, Swati Banerjee

α-synuclein (α-syn) is a neuronal protein that aggregates under pathological conditions to form inclusions called Lewy bodies. These aggregates define a group of neurodegenerative disorders collectively known as synucleinopathies, including Parkinson's disease (PD). In PD, accumulation of α-syn contributes to both motor and non-motor symptoms such as resting tremors, gait disturbances, cognitive decline, depression, and reduced quality of life. Several small molecule inhibitors have been identified in previous studies regarding their ability to inhibit α-syn aggregation revealing the potential for therapeutic ability. In this study, we used a newly developed ELISA assay to determine the effects of these small molecule inhibitors on an elevated α-syn model of Drosophila melanogaster. Results from the ELISA assay conducted showed a decrease in α-syn amongst Congo Red (CR), Rosmarinic Acid (RA), Levadopa (L-DOPA), and Dopamine hydrochloride (DHCL) when compared to the respective untreated control. However, for immunoblotting analysis, both CR and RA revealed lower levels for α-syn compared to L-DOPA and DHCL. Future goals of this study will be to determine the amount of aggregated α-syn in these samples and make a comparison between total α-syn and aggregated α-syn levels. Together, our studies have translational relevance by enabling expansion of an ELISA-based assay to screen drug libraries in an in vivo Drosophila drug discovery platform.

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Posterboard #: 22

Presenter: Emi Varfaj
Institution: UT Health San Antonio
Category: Other: Research Assistant

Validation of Dried Plasma Spot Sampling for Alzheimer's Disease Biomarkers in Diverse and Resource-Limited Settings

Emi Varfaj, Sarah Dean, Layla Garcia, Deshawna Escobedo, Vanessa M. Young, Ashley LaRoche, Roslyn Valdespino, Melissa Zamora, Jennifer G. Del Bosque, Jacob Stallman, Claudia Satizabal, Valentina Garbarino, A. Campbell Sullivan, Jeremy Tanner, Tiffany Kaut

Alzheimer's disease (AD) is the leading cause of dementia, accounting for 50-70% of all cases. While plasma biomarkers use is increasing in AD research and diagnosis, existing data largely reflects high income populations in the Global North with limited diversity and medical comorbidities, thus presenting a critical knowledge gap in global applicability. Present plasma biomarker testing requires access to trained phlebotomists and specialized lab infrastructure such as centrifuges and ultra-low temperature (-80°C) freezers, limiting feasibility in low-resource and rural settings. Dried plasma spot (DPS) technology offers a scalable alternative by eliminating the need for cold chains and advanced lab equipment.

The goal of this study is to evaluate the validity of novel DPS methods for collecting and measuring AD plasma biomarkers in diverse populations, using protocol development in San Antonio to inform implementation in the sub-Saharan Africa (sSA), specifically Uganda.

In a pilot cohort of 10 San Antonio participants with known amyloid status (5 positive, 5 negative), we are comparing the performance of venous dried plasma spot (V-DPS) and finger stick capillary dried plasma spot (C-DPS) to conventionally collected plasma and multimodal Alzheimer's disease biomarkers, including gold-standard amyloid measures from cerebrospinal fluid (CSF) and amyloid positron emission tomography (PET). DPS-derived biomarker values will be assessed for correlation with plasma measures and classification accuracy for AD.

To simulate real-word conditions, multiple DPS samples are collected per participant (6 C-DPS, 9 V-DPS) and stored under three conditions: (1) room temperature (RT) until assay, (2) RT for 2 weeks followed by storage at 4°C until assay, and (3) RT for 2 weeks followed by storage at -80°C until assay. To evaluate biomarker correlations under the different collection and storage conditions, we will use the Alamar NULISA CNS-120 panel to examine >120 biomarkers related to AD and related dementias.

To date, 7 San Antonio participants have been enrolled, with completion expected by the end of April 2026. Findings will guide DPS implementation in Uganda and support broader use in resource-limited settings, such as those found in rural areas of the United States.

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Posterboard #: 23

Presenter: Avery Zuniga, BS
Institution: UT Health San Antonio
Category: Other: Research Assistant

Long-term protein biomarker stability in archived San Antonio Heart Study plasma samples

Avery Zuniga, Jake Pap, Rebecca Bernal, Sarah Dean, Yelitza Ramirez, Roman Fernandez, Chen-Pin Wang, Helen P. Hazuda, Claudia L. Satizabal, Tiffany Kautz

The long-term stability of protein biomarkers related to Alzheimer's disease (AD) and cognitive impairment is poorly understood. We aimed to determine whether these biomarkers could be reliably quantified in frozen plasma samples collected three decades ago from participants enrolled in The San Antonio Heart Study (SAHS).

A total of 43 samples were randomly selected from a library of 2,025 plasma samples collected from 1992-1996. Participants had a mean age of 47±11 years at the time of collection, 56% were women, and 63% were Mexican American. To ensure that a comprehensive number of biomarkers could be examined for detectability, samples were measured in duplicate using the Alamar NULISA CNS 120 panel. We calculated precision and reliability metrics to evaluate biomarker detectability.

51% of plasma samples had at least one replicate below the expected biomarker target detectability of 90%. However, of the 131 target biomarkers, 116 were detected in >75% of the measured plasma samples. This suggests that most of the biomarkers remained measurable despite the long storage time. Across all biomarkers, the mean participant coefficient of variation (CV) was 3.1% between duplicate samples (range 0.466-25.28%). Additionally, only 3 biomarkers had a target CV >10%, indicating strong analytical concordance.

We found that long-term plasma storage (>30 years) does not compromise integrity for 89% of the measured biomarkers. We are currently evaluating the impact of participant covariates on protein biomarker detectability, with particular focus on those implicated in cardiometabolic and neurodegenerative diseases, which may affect detectability in an age-dependent fashion. We plan to compare these biomarker levels, assessed in midlife, with those obtained in late life from the ongoing San Antonio Heart and Mind Study, which is enrolling SAHS participants. This knowledge will provide a better understanding of AD biomarker trajectories throughout the life course.

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Posterboard #: 24

Presenter: Azam Alamdari, MD
Institution: UT Health San Antonio
Category: Postdoctoral Fellow

Semaglutide Improves Postprandial Glucose Metabolism after Spinal Cord Injury: A First Mechanistic Study

Azam Alamdari, Sven Hoekkstra, Amalia Gastaldelli, Michelle B Trbovich, Marzieh salehi

Introduction and Objective
Glucose dysregulation after spinal cord injury (SCI) is characterized by exaggerated postprandial hyperglycemia despite relatively normal fasting glucose, reflecting impaired insulin action, β-cell dysfunction, and disrupted gut-pancreas signaling due to autonomic injury. The metabolic effects of incretin-based therapy in this population are unknown. We examined the effects of the GLP-1 receptor agonist semaglutide on prandial glucose metabolism in individuals with SCI and type 2 diabetes (T2D).

Methods
Five men with T2D and SCI (duration 11±4 years; age 53±3 years; BMI 35±3 kg/m²; HbA1c 7±0.5%) underwent a liquid mixed-meal tolerance test (50 g protein + 50 g glucose) at baseline and after 6 months of semaglutide (2 mg weekly). Plasma glucose, insulin, C-peptide, and glucagon were measured to assess glycemia, insulin sensitivity, insulin clearance, and β-cell function.

Results
Semaglutide induced significant weight loss (10±1 kg; 16±3% of baseline BW, $p<0.05$) and reduced HbA1c (−1±0.4%, $p=0.06$). Total area under the glucose curve decreased by ~30% ($p<0.05$). Improvements were accompanied by enhanced fasting and prandial insulin sensitivity (HOMA-IR, OGIS) and increased insulin clearance ($p<0.05$). β-cell function showed a trend toward improvement, while glucagon responses were unchanged. Treatment was well tolerated with mild gastrointestinal symptoms and stable heart rate and blood pressure.

Conclusion
In this first mechanistic evaluation of GLP-1 receptor agonist therapy in patients with SCI and T2D, semaglutide markedly improved postprandial glucose tolerance primarily through enhanced insulin action and clearance. These findings support incretin-based therapy as a promising strategy to address metabolic dysregulation after SCI and warrant confirmation in larger studies.

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Posterboard #: 25

Presenter: Savannah Heath, PhD
Institution: UT Health San Antonio
Category: Postdoctoral Fellow

Effect of Advanced Glycation End Products on Glycosaminoglycans in Cortical Bone

Savannah Heath, Lidan Zhang, Rui Hua, Jean Jiang, Xiaodu Wang

Aging increases bone fragility partially through the deterioration of bone tissue quality and the loss of glycosaminoglycans (GAGs). These negatively charged molecules are essential for retaining water within the bone matrix, yet they decline significantly as tissue ages, particularly during the transition from young, secondary osteons to older, interstitial tissue. This study investigated whether the accumulation of Advanced Glycation End Products (AGEs), which concurrently build up over time, drives this GAG depletion through biological or material pathways. Colocalization was assessed via alcian blue staining of GAGs concurrently with AGE autofluorescence in cortical bone. Using cadaveric human femurs (ages 17-90), we identified a strong inverse correlation between local AGEs and GAG content ($R^2=0.768$, $p<0.001$) To identify whether AGEs played a causal role of loss of GAGs, we evaluated whether AGEs could influence bone cell activity as well as if whether inducing AGEs in cadaveric tissue physically resulted in loss of GAGs. Experimental results showed that AGEs act primarily as a biological signal: treating bone cells (MLO-A5 and MLO-Y4) with AGEs triggered the fragmentation of proteoglycans and the loss of extracellular GAGs measured via western blot and dimethyl methylene blue assay. This process was regulated by matrix metalloproteinases (MMPs), as broad-spectrum inhibitors were found to halt the degradation. While high levels of ribose or sugar glycation could also cause GAGs to leach out of bone powders due to physical mineral and organic alterations, this material effect required supraphysiological levels of ribose and was deemed a secondary mechanism. Ultimately, the study concludes that AGE accumulation resulted in MMP-mediated GAG loss and thus may be a key mechanistic driver of age-related loss of GAGs and compromised fracture toughness.

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Posterboard #: 26

Presenter: Zoe Hoffpauir, PhD
Institution: UT San Antonio
Category: Postdoctoral Fellow

Characterization of lumazine synthase of Mycobacterium tuberculosis to inform drug design

Zoe Hoffpuair and Audrey Lamb

Riboflavin (vitamin B2) is essential for all life, but only plants and microorganisms are able to synthesize riboflavin. Animals lack riboflavin biosynthetic machinery and many pathogenic bacteria rely on endogenously synthesized riboflavin making the specialized enzymes involved in riboflavin biosynthesis attractive target for novel antibiotics. Riboflavin is synthesized by five unique enzymes which catalyze unusual and complex chemical reactions that are notably slow in vitro. In order to support all life on Earth, in vivo biosynthesis must be much faster. The penultimate enzyme of the pathway, lumazine synthase, forms a capsid, and we propose that all of the enzymes form a protein-based organelle for efficient riboflavin formation. In addition, there are considerable gaps-in-knowledge for lumazine synthase. The hypothetical chemical mechanism lacks evidence. Lumazine synthase is post-translationally acetylated, but the effect of acetylation on structure and kinetics is not determined. To unravel the discrepancies between in vitro and in vivo kinetic parameters and address the gaps-in-knowledge, we are providing direct evidence for the catalytic intermediates for lumazine synthase, determining the effect the post-translational modification acetylation on catalysis and complex formation among the members of the pathway, and defining the protein-protein interactions required for efficient riboflavin biosynthesis. We employ a wide array of methodologies, including 13C-NMR, x-ray crystallography, cryogenic electron microscopy and transient kinetics, we will provide an understanding of riboflavin biosynthetic machinery that will inform rational drug design of novel antibiotics.

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Posterboard #: 27

Presenter: Matteo Maria Lalovich, MD
Institution: UT Health San Antonio
Category: Postdoctoral Fellow

Short-Term Glucagon Adaptation to an Isocaloric Ketogenic Diet in Obese Individuals with Type 2 Diabetes

Matteo Maria Lalovich

Background:
We previously (BMJ Open Diab: e004199, 2024) showed that a 10-day weight-maintaining ketogenic diet in individuals with type 2 diabetes did not change OGTT responses or insulin sensitivity (two-step euglycemic clamp) but increased insulin secretion.

Objective:
To examine the effect of the 10-day weight-maintaining ketogenic or standard diet on fasting plasma glucagon and OGTT glucagon responses.

Methods:
Ten individuals with type 2 diabetes (A1c = 8.0 ± 0.2%; BMI 33 ± 1.0 kg/m²) consumed a weight-maintaining ketogenic diet (80% fat, 15% protein, 5% carbohydrate) and eight individuals with type 2 diabetes (A1c = 8.0 ± 0.1%; BMI 34 ± 1.5 kg/m²) consumed a weight-maintaining standard diet (35% protein, 45% carbohydrate, 20% fat) for 10 days. Plasma glucagon was measured after an overnight fast and during a 2-hour 75g OGTT. Plasma Insulin, C-peptide, FFA, and beta-hydroxy-butyrate were measured.

Results:
Fasting plasma glucagon increased after the ketogenic diet (56 ± 8 to 89 ± 13 pg/mL, $p < 0.05$). Mean OGTT plasma glucagon increased from 60 ± 7 to 82 ± 12 pg/mL ($p = 0.12$). Mean OGTT plasma insulin and C-peptide responses increased from 35 ± 4 to 56 ± 9 µU/mL and from 7.8 ± 0.6 to 11.5 ± 0.9 ng/mL, respectively (both $p < 0.05$). In the control group, fasting and OGTT plasma glucagon concentrations were unchanged (both $p > 0.05$). Efficacy of the ketogenic diet was confirmed by elevated fasting BOHB and increased lipid oxidation/decreased carbohydrate oxidation (indirect calorimetry).

Conclusions:
A short-term ketogenic diet in obese type 2 diabetic subjects increases fasting plasma glucagon and glucagon and insulin responses during the OGTT, whereas no changes occurred with the standard diet. These findings suggest a dual-hormone regulatory pattern in which elevated glucagon and insulin coexist with enhanced ketone production.

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Posterboard #: 28

Presenter: Mohsin Mansoor, MD
Institution: UT Health San Antonio
Category: Postdoctoral Fellow

Bone Mineral Density Screening and Risk Among Individuals Treated With Buprenorphine and Methadone: A Retrospective Cohort Study Using a National Database

Mohsin Mansoor, Jan Bruder, Anthony Wise, Curtis Bone

Objectives:
To compare bone mineral density (BMD) screening practices and risk of abnormal BMD among individuals prescribed buprenorphine and methadone.

Methods:
We conducted a retrospective cohort study using the TriNetX federated research network. Adults with opioid use disorder (OUD) prescribed buprenorphine or methadone were identified using diagnostic and medication codes, with the index date defined as the first recorded prescription. Outcomes included receipt of dual-energy X-ray absorptiometry (DXA) and abnormal BMD, defined by diagnostic codes for disorders of bone density. Propensity score matching (1:1) was performed to balance demographic and clinical characteristics. Outcomes were assessed from 1 day after index date through follow-up, and risk ratios (RRs) with 95% confidence intervals (CIs) were calculated.

Results:
After matching, 39,777 individuals were included in each cohort. DXA screening rates were low and similar between groups (buprenorphine 0.98% vs methadone 0.95%; RR 0.97, 95% CI 0.84-1.12; p=0.69). The risk of abnormal BMD was higher for people on methadone compared to buprenorphine (RR 1.114 CI 1.05,1.183 p<0.01).

Conclusions:
BMD screening rates were low among individuals receiving medications for OUD and did not differ between buprenorphine and methadone. The risk of abnormal BMD was higher among people receiving methadone, highlighting potential gaps in preventive bone health care in this high-risk population.

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Posterboard #: 29

Presenter: Salomé Wilfred, PhD
Institution: UT Health San Antonio
Category: Postdoctoral Fellow

Prospective Health Impacts of Chronic Binge Eating Disorder in Hispanic Older Women Living with Food Insecurity (PROSPERA)

Salomé Adelia Wilfred, Diamond Hercules, Karen Morado, Judith Fuentes, Melissa Narvaez, Taylur Loera, Nadia Lopez, Tayelor Casey, Carolyn Black Becker, & Lisa Smith Kilpela

Introduction: Binge Eating Disorder (BED) is associated with poor cardiometabolic and mental health, controlling for BMI. Yet, BED is understudied among populations deviating from the eating disorders stereotype (young, thin, white, affluent); specifically, women of older age, minority ethnicity, and living with food insecurity (FI). Early cross-sectional data suggest poor mental health outcomes for BED in this population; however, a comprehensive understanding of the prospective multimorbidity of BED in this understudied population remains unknown. This study investigates the prospective whole health impacts of chronic BED for older women with FI, versus those with FI without BED, and explores resource pathways as guided by women with lived experience via qualitative interviews.

Methods: Participants are San Antonio Food Bank (SAFB) neighbors (formerly, "clients") recruited at the SAFB. Participants complete a series of whole health assessments, including a diagnostic interview to confirm BED, and measurements of psychosocial health, nonmedical drivers of health, cardiometabolic health indicators, and strength/balance. Assessments are completed at annual intervals, with interim psychosocial assessments every 3 months.

Results: Data collection is ongoing; N=116 participants have completed baseline assessments. Preliminary rapid thematic analyses identified shame and cultural considerations as barriers to engaging in resources, while strength of older women was highlighted as a facilitator of resource uptake. Updated data will be presented in the poster.

Conclusions: These results will delineate the prospective whole health impacts of chronic BED among older women with FI and inform interventions for BED in this population.

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Posterboard #: 30

Presenter: Hussein A Zaitoon, MD
Institution: UT Health San Antonio
Category: Postdoctoral Fellow

Empagliflozin Enhances Total-Body Norepinephrine Turnover in Parallel with Stimulation of Hepatic Gluconeogenesis and Ketone Production in Type 2 Diabetes

Hussein A Zaitoon, Aurora Merovci, Amalia Gastaldelli, Matteo M. Lalovich, Samantha Pezzica, John Adams, Alberto Chavez, Hansis-Diarte Andrea, Muhammad Abdul-Ghani, Ralph A. DeFronzo

Background
SGLT2 inhibitors lower plasma glucose yet paradoxically increases hepatic glucose production (HGP)/gluconeogenesis and ketone production. Whether activation of the sympathetic nervous system (SNS), assessed by norepinephrine turnover, mediates these effects in humans remains unclear.

Objective
To measure the effect of empagliflozin on HGP/gluconeogenesis, plasma ketone and FFA concentrations, and total-body NE turnover in T2DM diabetes.

Methods
15 patients with type 2 diabetes (Age = 57 ± 2 yrs, HbA1c = 9.0 ± 1.0%, BMI = 31 ± 1.5 kg/m²) participated in the study. HGP, gluconeogenesis and total-body NE turnover were measured with 6,6-^{2}H2-Glucose, D2O and 3H-NE, respectively, before and 5 hours after empagliflozin (25 mg) ingestion. Plasma glucose, insulin, C-peptide, free fatty acid (FFA), and ketone concentrations, NE specific activity, and deuterium enrichment in glucose were measured over 5 hours.

Results
After empagliflozin administration, gluconeogenesis increased from 1.57 to 1.79 mg/kg.min ($p<0.01$) and HGP increased from 2.33 to 2.45 mg/kg.min ($p<0.01$). NE turnover increased by 82% from 689 ± 69 to 1238 ± 183 ng/min ($p<0.01$), in association with a 40% increase in NE clearance rate (1596±224 to 2218±339 ml/min, $p<0.05$). Plasma norepinephrine concentration increased from 297 to 335 pg/mL $p = 0.03$). Plasma FFA (0.512 to 0.705 mmol/L, $p < 0.001$) and plasma ketone (0.358 to 0.538 mmol/L, $p = 0.006$) concentrations increased markedly. Insulin declined from 17 to 14 µU/mL ($p = 0.04$) in response to the glucosuria - induced decline plasma glucose concentration.

Conclusions
Acute empagliflozin administration causes a rapid (within 5 hours) increase norepinephrine turnover, indicating SNS activation. The increase in sympathetic activity explains the increases in plasma FFA and ketone concentrations and augmented the rate of gluconeogenesis observed following empagliflozin administration.

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Posterboard #: 31

Presenter: Dongmei Zuo, PhD PhD MS
Institution: UT Health San Antonio
Category: Postdoctoral Fellow

Development and Pilot Evaluation of an NLP-Augmented Digital Support Program for Prostate Cancer Patients and Caregivers: Implications for Digital Health Policy

Dongmei Zuo PhD PhD MS1; Furkan Dursun MD 2; Jia Liu, PhD MSN 1, Xiaomeng Wang MS3, Fei Yu, PhD 4; Hung-Jui Tan MD MSHPM 5; Lixin Song, PhD, RN, FAAN 1

Purpose: Patients with localized prostate cancer and their caregivers face complex treatment decisions with limited access to ongoing support beyond the clinical encounter. The Interactive Prostate Cancer Information, Communication, and Support (iPICS) program, an NLP-augmented digital platform was developed and tested to address this gap.

Methods: In this 2-phase mixed-method study, Phrase I used a 4-step iterative, user-centered process guided by Responsible AI principles: (1) needs assessment, (2) formative prototype evaluation, (3) summative prototype evaluation, and (4) field deployment readiness evaluation. Semi-structured interviews with prostate cancer patients and caregivers were analyzed using thematic analysis. In Phase II, we conducted a multisite pilot to access feasibility, acceptability, usability, and adoption using descriptive measures and qualitative interviews.

Results: In Phase I, 18 patients and 7 family members participated across two interviews and 17 focus groups, identifying five major themes: functional requirements, user interface design, content needs, delivery preferences, and privacy concerns. These guided development of three iPICS components: Inform, a multimedia health information hub; Dialog, an NLP-augmented consultation tool, and Snap, a nurse-moderated peer forum. In Phase II, 19 patients and 9 caregivers were enrolled; enrollment was 22% (28/130) and retention was 61% (17/28). Findings demonstrated feasibility, high satisfaction, and strong recommendation intent.

Conclusions: iPICS shows promise as a scalable, ethical, and inclusive supportive care tool. Ongoing trial is testing feasibility and efficacy of EMR integration in a larger sample. Policy Implications: Findings highlight needs to integrate patient-centered digital supportive care tools into healthcare systems through interoperability and responsible AI governance.

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Posterboard #: 32

Presenter: Shahad Abdulsahib
Institution: UT Health San Antonio
Category: Student

THOC7 Supports Ribosome Biogenesis in Myc-Driven Medulloblastoma and Reveals a Therapeutic Vulnerability

*Shahad Abdulsahib 1,2, Ji Jae Hoon Ph.D. *1,2, Prabhakar Pitta Venkata Ph.D. *1,2, Panneerdoss Subbarayalu Ph.D. 1,2, Santosh Timilsina Ph.D. 1,2, Daisy Medina Ph.D. 1,2 , Saif Nirzhor Ph.D. 1,2, Nicolas Ryujin1,, Deepika Singh Ph.D. 1,2, Manjeet Rao Ph.D*

Ribosomal biogenesis is essential for protein synthesis and cellular proliferation, yet its lineage-specific role in brain tumors remains incompletely defined. Here, we identify ribosomal biogenesis as a critical survival dependency in medulloblastoma and establish THOC7 as a previously uncharacterized regulator of this process. Analysis of Cancer Dependency Map data demonstrates a near-universal dependency on THOC7 in medulloblastoma, with elevated expression in aggressive subtypes, particularly MYC-driven Group 3 tumors, where it correlates with poor clinical outcomes. Functional studies reveal that THOC7 is required for medulloblastoma cell viability, proliferation, stemness, migration, tumor growth, and resistance to radiation in both in vitro and in vivo models. Mechanistically, THOC7 sustains ribosome biogenesis, as its depletion disrupts ribosome assembly and protein synthesis. Loss of THOC7 further induces replication stress, R-loop accumulation, S/G2 cell-cycle arrest, and defects in homologous recombination, linking impaired ribosomal output to genome instability. These findings suggest that medulloblastoma cells are highly dependent on elevated ribosomal activity, creating a selective vulnerability centered on RNA polymerase I-driven rRNA transcription. Consistent with this, pharmacologic inhibition of Pol I using CX-5461 phenocopies THOC7 loss and significantly suppresses tumor growth in orthotopic and patient-derived xenograft models. Together, these results define ribosomal biogenesis as a central and therapeutically actionable dependency in high-risk medulloblastoma and position THOC7 as a key regulator of tumor maintenance.

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Posterboard #: 33

Presenter: Emily Aller, BA
Institution: UT Health San Antonio
Category: Student

Novel MDK targeted therapy for the treatment of Endometrial Cancer

Emily J. Aller, Ricardo Martinez, Aditi Subramanya, Baskaran Subramani, Megharani Mahajan, Xue Yang, Paulina Ramirez, Panneerdoss Subbarayalu, Edward R. Kost, Hareesh B. Nair, Ratna K. Vadlamudi, and Suryavathi Viswanadhapalli

Endometrial cancer (ECa) is the fourth most common malignancy in women, with an increasing incidence among those under 40 years of age. Current treatment options for recurrent ECa, including hormonal therapy, chemotherapy, immunotherapy, and targeted agents such as bevacizumab, achieve only limited response rates and confer poor progression-free survival in advanced stages. Therefore, there is an urgent need for novel, more effective targeted therapies. Midkine (MDK), a heparin-binding growth factor, plays a central role in regulating multiple oncogenic signaling pathways and is frequently overexpressed across various cancer types, including ECa. The objective of this study was to develop and evaluate a novel MDK inhibitor for the treatment of ECa.

Based on a Dienogest-derived pharmacophore, we designed HBS-101, a small molecule that directly binds and inhibits MDK. Binding was confirmed using microscale thermophoresis and in silico docking analyses. In vitro activity was evaluated using MTT, clonogenic, and apoptosis assays, while mechanistic studies were conducted using Western blotting, RT-qPCR, reporter assays, RNA sequencing, and transmission electron microscopy (TEM). MDK expression in ECa was examined using the TNMplot database. Pharmacokinetic properties and in vivo efficacy were assessed in mouse models. The efficacy of HBS-101 in chemotherapy-resistant ECa was further validated both in vitro and in vivo using paclitaxel-resistant primary ECa cells generated in-house. MDK was highly expressed in ECa tumors and cell lines compared with normal tissues, and its overexpression markedly promoted in vivo tumor growth. Conversely, MDK knockdown reduced cell proliferation and colony formation. HBS-101 bound MDK with nanomolar affinity and reduced cell viability, colony formation, and survival. Mechanistic investigations revealed that HBS-101 induced apoptosis, endoplasmic reticulum stress, and ferroptosis, while inhibiting key downstream effectors of MDK, including STAT3 and NFκB. Notably, paclitaxel-resistant cells exhibited enhanced sensitivity to HBS-101. Furthermore, HBS-101 demonstrated favorable pharmacokinetic properties, excellent in vivo stability, and high oral bioavailability, with no observable toxicity at doses up to 10 mg/kg. It significantly suppressed tumor growth in both chemotherapy-sensitive and -resistant patient-derived xenograft models.

Overall, HBS-101 is a promising MDK-targeted therapeutic that shows favorable pharmacokinetics and induces apoptosis and ferroptosis in ECa cells by blocking MDK signaling.

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Posterboard #: 34

Presenter: Adriana Baker
Institution: UT Health San Antonio
Category: Student

Translating ER Stress Induction into HCC Therapy: Preclinical Evaluation of ERX-315

Adriana Baker, Nizalia Siddiqui, Xue Yang, Uday P. Pratap, Ricardo A. Martinez, Baskaran Subramani, Gaurav Sharma, Chia-Yuan Chen, Scott Elmore, Sukeshi Patel Arora, LuZhe Sun, Suryavathi Viswanadhapalli, Ganesh V. Raj, Jung-Mo Ahn, Ratna K. Vadlamudi.

Hepatocellular carcinoma (HCC) accounts for over 90% of liver-cancer cases and is the 5th most common malignancy in the United States1. Most patients present with advanced disease, facing a 5-year survival rate below 5% with current systemic therapies3. We discovered a synthetic oligo-benzamide targeting lysosomal acid lipase A (LIPA), ERX-315, that induces ER stress and cancer-cell death without harming normal cells. This study evaluates ERX-315's therapeutic potential in HCC via ER-stress induction.

LIPA levels were assessed using the TCGA database and tissue microarrays (TMAs). Effects of ERX-315 on HCC cell lines and patient-derived xenograft (PDX) cells were measured by MTT and colony-formation. Normal human hepatocytes were evaluated by caspase-activity and Annexin V assays. CRISPR-mediated LIPA knockout and site-directed mutagenesis were performed to confirm target specificity. ER-stress markers, pathway activation, and ER homeostasis disruption were examined by Western blotting, RT-qPCR, RNA-seq, transmission electron microscopy (TEM), and XBP1-splicing assays. Huh7 organoids generated from xenografts were utilized to evaluate the effects of ERX-315 ex vivo. Huh7 xenografts and PDX models were dosed with ERX-315, and maximum tolerated dose (MTD) studies evaluated organ-specific toxicities.

TCGA and TMA analyses confirmed significantly higher LIPA expression in HCC versus normal liver. ERX-315 inhibited HCC colony formation; reduced HCC cell viability (IC_{50} 30-150 nM); while not inducing significant apoptosis in normal hepatocytes. LIPA knockout or mutation of ERX-315-binding residues markedly attenuated its effects on colony formation and viability. Western blotting, RT-qPCR, RNA-seq, TEM and splicing assay demonstrated dose dependent ER stress activation and ER homeostasis disruption. ERX-315 significantly decreased viability of Huh7 organoids and reduced tumor volumes in both Huh7 xenograft and PDX models. MTD studies showed no significant organ toxicities at doses up to six-fold higher than those used for efficacy.

These findings support ERX-315 as a potent and selective therapeutic for HCC.

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Posterboard #: 35

Presenter: Ashika Bakre, BS
Institution: UT Health San Antonio
Category: Student

Toward Artificial Intelligence-Enabled Decision Support in Neuropsychology: A Pilot Study of Clinical Inference Agreement

Ashika S. Bakre, Elliot T. Morgan, Chloe K. Chui, Anthony M. Hachem, Anthony M. Rios, Summer N. Rolin & Jeremy J. Davis

Artificial intelligence large language models (LLMs) are deployed in clinical settings in information summarization and clinical documentation roles. LLMs may have utility in decision support, but their ability to generate meaningful clinical inferences is unclear. This pilot study examined agreement between LLM-generated inferences from clinical interviews and clinical diagnosis (DX). Participants were 27 older adults (mean age=73.9 years; 59% female) referred for neuropsychological evaluation of memory complaints. DX included normal (n=6; 22.2%), mild cognitive impairment (MCI; n=19; 70.4%), and dementia (n=2; 7.4%). De-identified, unannotated interview transcripts, age, and gender were entered into a secure, HIPAA-compliant LLM chat interface with a standardized prompt to provide hypotheses about the likely cognitive status of participants. LLM inferences based on interview (LLM-INT) and an LLM-generated summary of the report impression section (LLM-IMP) were compared to DX. Agreement was evaluated using Cohen's kappa. LLM-INT showed poor agreement with DX (33.3%; $\kappa=-0.11$). In contrast, LLM-IMP and DX showed perfect agreement (100%; $\kappa=1.00$), consistent with hallucination-free extraction of report content. These preliminary findings support ongoing use of LLMs for summarization and motivate further work to improve inference. Additional research should focus on prompt refinement, enhanced output constraints, and feasibility testing in larger, diagnostically diverse samples.

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Posterboard #: 36

Presenter: Arpita Biswal
Institution: UT Health San Antonio
Category: Student

Investigating the Role of Mitochondrial Complex I Protein, NDUFV2, in Aging and Behavior Using RNA Interference in Drosophila melanogaster

Arpita Biswal, Dr. Swati Banerjee, Raquel Velazquez

Mitochondrial dysfunction, specifically within Complex I of the electron transport chain (ETC), is a hallmark of several neurological disorders, including schizophrenia, bipolar disorder, and Parkinson's disease (PD). A critical subunit of Complex I, NDUFV2, is highly conserved and essential for efficient energy production; however, its specific contribution to neurological decline remains incompletely understood. This study investigates how reduced NDUFV2 activity impacts neuronal function and organismal behavior using Drosophila melanogaster as a model. By utilizing RNA interference (RNAi), we selectively knocked down the NDUFV2 ortholog, ND-24, and compared experimental flies to genetic controls. We assessed neurological and physiological decline through negative geotaxis climbing assays and longevity (survival) assays. Additionally, Western blot and immunohistochemistry analyses were employed to confirm ND-24 knockdown and evaluate subsequent changes in mitochondrial protein expression. We hypothesize that ND-24 knockdown will result in impaired locomotor performance, reduced lifespan, and diminished mitochondrial protein levels, consistent with compromised Complex I function. By linking the molecular disruption of a conserved mitochondrial subunit to behavioral changes, this research advances our understanding of the mitochondrial mechanisms underlying neurodegeneration and development of disease states, being a crucial step in identifying potential therapeutic targets for diseases, especially in the treatment of PD.

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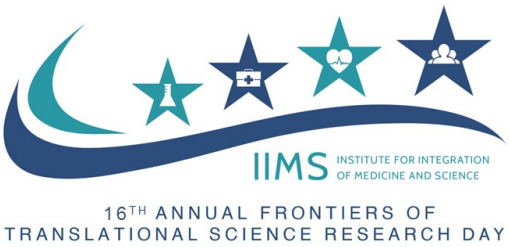
Posterboard #: 37

Presenter: Savannah Boyen
Institution: UT Health San Antonio
Category: Student

Characterizing Mitochondrial Complex I Dysfunction in Parkinson's Disease Through a Multi-System Approach

Savannah K. Boyen and Swati Banerjee

Parkinson's disease (PD) is a progressive neurodegenerative movement disorder characterized by the loss of dopaminergic neurons in the substantia nigra and the accumulation of alpha-synuclein-containing Lewy Bodies. In addition to alpha-synuclein, tubulin polymerization-promoting protein (TPPP) is enriched in Lewy bodies and implicated in PD and other synucleinopathies, yet the cellular and molecular mechanisms linking TPPP to PD pathology are not well defined. In a Drosophila loss-of-function model of Ringer (the human TPPP homolog), transmission electron microscopy revealed significant disruptions in mitochondrial cristae morphology when compared to control wild-type mitochondria. A mass spectrometry-based proteomic screen further revealed key mitochondrial Ringer-interacting proteins, with Complex I subunits of the electron transport chain (ETC) emerging as the top interactors. NDUFV2, a core matrix arm subunit of Complex I, emerged as the top interactor. This project aims to characterize the interaction between Ringer/TPPP with NDUFV2 and its role in mitochondrial Complex I biology using a multi-system approach. Loss of Ringer showed significant reduction of mitochondrial NDUFV2 protein levels suggesting that Ringer may play a role in the stability of the ETC. Mammalian cell culture of rat dopaminergic neurons demonstrates co-localization of TPPP with NDUFV2 in mitochondria. In human PD postmortem brain tissue, TPPP and NDUFV2 also show co-sedimentation in overlapping fractions using Optiprep density gradient centrifugation suggesting that these proteins are present within the same biochemical complex. To further investigate the role of this Complex I subunit, RNAi knockdown and over-expression fly lines for NDUFV2 were generated, and ongoing studies aim to investigate the resulting mitochondrial and neuronal phenotypes. Taken together, these findings suggest a novel link between TPPP and mitochondrial Complex I dysfunction in PD, providing new insight into mitochondrial-based mechanisms underlying neuronal vulnerability in neurodegenerative diseases.



Posterboard #: 38

Presenter: Elena Camargo, MS
Institution: UT San Antonio, UT Health San Antonio
Category: Student

Physical Activity and Mortality Risk Among U.S. Adults with a History of Cancer

Elena Camargo, Jeffrey T. Howard, Kelly Cheever

Introduction: Physical activity (PA) is associated with improved survival in cancer survivors; nationally representative analyses directly comparing adults with and without a history of cancer are limited. We examined associations between moderate-to-vigorous PA (MVPA) and cancer mortality using NHANES data from 1999-2018.

Methods: We analyzed NHANES data collected between 1999-2018 and linked to National Death Index mortality records with mortality follow-up through 2019. Adults aged ≥18 years with available data on cancer history, PA, and mortality were included. Participants were categorized into four groups based on cancer history and PA status. Survey-weighted competing risk regression models estimated hazard ratios (HRs) and 95% confidence intervals (CIs) for cancer mortality, adjusting for age, sex, race/ethnicity, education, marital status, and body mass index.

Results: In this nationally representative sample, engagement in MVPA was associated with lower cancer mortality risk. Adults with cancer history reporting MVPA had a lower risk of cancer mortality compared with those not reporting MVPA. Differences in mortality were observed across PA and cancer status groups after adjustment.

Conclusion: Engagement in MVPA was associated with lower cancer mortality risk among U.S. adults, including those with a history of cancer. These findings support recommendations promoting regular physical activity in adult populations.

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Posterboard #: 39

Presenter: Chloe Chui
Institution: UT Health San Antonio
Category: Student

AI-Assisted Clinical Data Extraction: Evaluating AI Limits and Utility in Clinical Decision Support

Chloe K. Chui, BS; Anthony M. Hachem, BS; Ashika S. Bakre, BS; Elliot T. Morgan, BS; & Jeremy J. Davis, PsyD, ABPP-CN

Clinical datasets possess immense potential for enhancing patient care and informing value metrics. However, traditional manual data entry is a significant bottleneck. Processing 1,000 cases can require 200-250 hours of intensive labor and is prone to human error. This study investigates the feasibility of using Large Language Models (LLMs) to transmute complex, multi-domain assessment data into structured, reproducible inferences, potentially reducing the processing time for 1,000 cases to just 20 minutes. In order to do so, the researchers focused on an AI-assisted extraction development cycle consisting of five stages: Define, Prompt, Generate, Test, and Refine. To ensure the protection of Personal Health Information (PHI), the study utilized an "air-gap" architectural design. The LLM was used exclusively in the development phase to generate VBA scripts based on report structures and synthetic data, while the actual clinical data remained in a local environment where the scripts were executed offline. System-wide validation was performed by comparing AI output against a ground truth of 10-20 manually processed reports. After conducting 3 separate pilot studies focused on evaluating AI's abilities in cognitive inference, Activities of Daily Living (ADL) assessments, and psychiatric diagnosis, we found that the utility of the LLM varied significantly based on the complexity of the task. In administrative summarization, the AI achieved 100% accuracy when extracting information from structured report impressions. However, we did identify that the AI demonstrated poor agreement when generating diagnostic hypotheses from unstructured interviews, alluding to a risk of over-diagnosing ambiguous cases compared to human benchmarks. In conclusion, AI is limited in its ability to perform complex clinical reasoning. Future development should be focused on prompt refinement and feasibility testing with larger, more diverse samples to help bridge the gap between data extraction and diagnostic application.

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Posterboard #: 40

Presenter: Matthew Click
Institution: UT Health San Antonio
Category: Student

Mechanobiological Memory: How Do FSS-Induced Phenotypes Persist in Prostate Cancer Cells?

Matthew Click, Yusheng Qian, Meizhen Chen, Chia-Nung Hung, Nameer Kirma, Michael Liss, Tim H. Huang, Maria Gaczynska, Pawel A. Osmulski

Circulating Tumor Cells (CTCs) are responsible for seeding new metastatic sites. As biomarkers of Prostate Cancer (PCa), their dissemination remains a leading cause of mortality among American men of various racial and ethnic backgrounds. A primary obstacle faced by CTCs is the mechanical forces or Fluid Shear Stress (FSS) they're exposed to in circulation. FSS kills the vast majority of these cells, forcing survivors to adapt and develop distinct phenotypes that enhance their mechanical fitness and overall survival in the bloodstream. We study the mechanical properties of patient-derived CTCs isolated by microfiltration. We use model CTCs generated from cultured cell lines exposed to FSS in microfluidic systems to emulate the strategies observed in patients' CTCs. We employ Peak Force Quantitative Nanomechanics Atomic Force Microscopy (AFM) to quantify the mechanical properties (e.g., adhesion, stiffness, and deformability) that define these phenotypic strategies and characterize their microenvironment (e.g., cluster composition) at the nanoscale. We observed that CTCs, as robust biomarkers of disease progression and treatment response, gain adhesion and clustering capabilities post-FSS exposure, thereby enhancing their survival and invasiveness. However, the extent of adaptation varies markedly across African American, Hispanic, and non-Hispanic White men. These aggressive phenotypic responses persisted in model CTCs. Biophysical phenotyping and microenvironment characterization of CTCs support proper differentiation of metastatic potential, providing strong prognostic potential for PCa patients. By understanding the role and persistence of CTCs, our unique phenotyping and biomarking approaches open avenues for precision medicine that are sensitive to racial and ethnic backgrounds, while studies of model CTCs improve our understanding of the physiological mechanisms underlying cancer metastasis.

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Posterboard #: 41

Presenter: Jamie Edwards
Institution: UT Health San Antonio
Category: Student

Investigating the role of Tubulin Polymerization Promoting Proteins in Alzheimer's disease and related dementias

Jamie L. Edwards, Savannah K. Boyen, Swati Banerjee

Alzheimer's disease (AD) and Alzheimer's disease related dementias (ADRDs) are a family of neurodegenerative disorders that cause significant loss of cognition, independence, and household stability. Aggregates of the microtubule-associated protein (MAP) Tau are a key histological finding in post-mortem AD/ADRD patients. However, it is not well understood how other proteins that interact with Tau might contribute to disease pathogenesis. Tubulin Polymerization Promoting Proteins (TPPP) are a group of MAPs we are interested in as potential players in Tau-driven AD/ADRD pathology. Besides being a MAP, TPPP, like Tau, is an intrinsically disordered protein, and there is evidence that TPPP promotes aggregation of alpha-synuclein, a key protein implicated in synucleinopathies, in vitro. These properties of TPPP led us to hypothesize that TPPP associates with Tau and contributes to disease pathogenesis in AD and ADRDs. To address this hypothesis, we used immunohistochemistry and western blotting to determine whether Tau and TPPP are interdependent in their localizations and levels in both human AD patients and Drosophila AD/ADRD models. We found that Tau and TPPP aggregates overlap in human AD frontal cortex in the soma and in a Drosophila disease model, with overexpression of fly TPPP, known as Ringer, promoting aggregation of expressed human wild-type Tau (TauWT). We also found that Tau and TPPP co-sediment in similar Optiprep density fractions on western blot in both AD and non-AD human cortex, suggesting that these proteins may complex together. Finally, on the sub-cellular level in flies, we found that while TauWT localization is predominantly cytoplasmic, pathological human Tau (TauP301L) localization is largely mitochondrial, suggesting a possible misdistribution of pathological Tau in AD/ADRD. This finding is promising given the extensive mitochondrial dysfunction seen in AD/ADRD and presence of TPPP/Ringer in both mitochondrial and cytoplasmic fractions. Together, these data indicate that in both humans and Drosophila, TPPP and Tau may interact to contribute to AD/ADRD pathology, providing a potential novel target for future AD/ADRD clinical interventions and validating the use of Drosophila as an experimental model to uncover the mechanistic underpinnings of AD/ADRD. Future studies will verify Tau-TPPP interaction using co-immunoprecipitation and elicit a mechanism for Tau-TPPP interaction.

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Posterboard #: 42

Presenter: Anamarie Figueroa
Institution: UT Health San Antonio
Category: Student

Violence Exposure and Dental Care Use in Emerging Adulthood

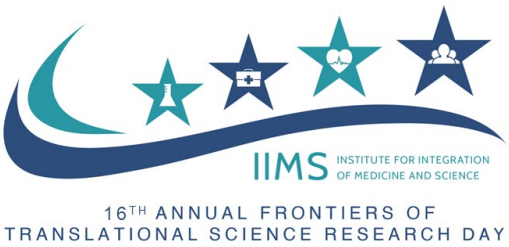
Anamarie Figueroa, Rahma Mungia, BDS, MSc, DDPHRCS[1]; Lindsey Webb, PhD[2]; Dylan B. Jackson, PhD[3]; Nandita Phadke, PhD[4]; Daniel C. Semenza, PhD[5,6]; Richard Stansfield, PhD[5]; Ian Silver, PhD[7]; Alexander Testa, PhD[4]

Background
Regular dental check-ups are a key component of maintaining both oral and overall health, yet a substantial proportion of young adults in the United States do not visit a dental provider annually. Prior research using nationally representative samples of middle-aged adults has identified an association between exposure to community violence and reduced dental care utilization. However, less is known about whether this relationship exists during emerging adulthood, a critical developmental period characterized by increased independence and evolving health behaviors. This study examines the relationship between community violence exposure and dental care utilization at age 22 among a cohort of urban-born youth in the United States.

Methods
Data were obtained from the Future of Families and Child Wellbeing Study (FFCWS), a longitudinal birth cohort of children born in large U.S. cities (N = 2,990; Year 22 wave). Multiple logistic regression models were used to assess the association between past-year community violence exposure (either witnessed or directly experienced) and having had a dental visit within the past year. Sequential models evaluated the impact of sociodemographic characteristics, maternal closeness, and prior dental utilization. Interaction terms were included to assess whether associations differed by sex and race or ethnicity.

Results
In unadjusted analyses, community violence exposure was associated with significantly lower odds of a past-year dental visit (OR = 0.47, $p < .01$). This association remained after adjusting for sociodemographic variables but was attenuated and no longer statistically significant after accounting for maternal closeness and prior dental visits. Interaction analyses revealed that violence exposure was associated with reduced odds of dental care utilization among females, while no significant association was observed among males.

Conclusion
After adjusting for relevant covariates, community violence exposure was not significantly associated with dental care utilization in emerging adulthood. However, subgroup findings suggest that exposure to violence may differentially impact dental care use among females. These results highlight the importance of considering social and contextual factors in understanding disparities in preventive health behaviors among young adults.



Posterboard #: 43

Presenter: Lavanya Gupta
Institution: Health Careers High School
Category: Student

Using AI to Model and Target Unusual RNA Structures for Cancer Therapy

Lavanya Gupta

Majority of drugs that are used for cancer therapy are represented by small molecules that specifically target a specific proteins or chemotherapy agents that target DNA to abolish DNA replication in rapidly dividing cancer cells. Antibody-based drugs that target specific receptors on cancer cells have also become successful in some cancers. Despite these successful strides, we are still far from curing many cancers that are aggressive and metastatic in nature. Proteins that support tumor growth have been subjected for targeted inhibition by small molecules in cancer therapy for decades. Interestingly, >95% of human genome remains non-coding but play important role in gene expression and regulation. To this end, targeting RNA and developing RNA-based therapies are emerging areas of cancer drug discovery. My research focuses on studying unusual RNA structures within the non-coding regions of the genes whose encoded proteins cause cancers. By identifying the folded regions of RNA and targeting them by small molecules, my research aim is to target the genes at their RNA level itself rather their encoded proteins downstream. I specifically focus on genes that have unusually long RNA transcript compared to proteins they encode. I identified a specific region in RNA of a gene (BCL-2) which can fold into an unusual structure called G-quadruplexes (rG4). BCL-2 promotes the growth of several blood cancers/leukemias (CML, AML). Next, I used artificial intelligence (AI)-based computational modeling tools to generate 3D models of this RNA sequence and screened thousands of compounds virtually. Next, I computationally docked the top scoring compounds into different potential pockets of BCL2 rG4 and identified compounds with drug-like properties. I developed a cost and resource-effective screening approach with broad utility for identification of novel RNA structure and developing novel small molecules to target unusual RNA structures implicated in cancer and viral infections.

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Posterboard #: 44

Presenter: Jian Huang, BS
Institution: UT Health San Antonio
Category: Student

The role of FOXM1 in regulating immune memory in triple negative breast cancer and targeting FOXM1 to develop a therapeutic cancer vaccine

Jian Yu Huang, Santosh Timilsina, Nourhan Abdelfattah, Daisy Medina, Deepika Singh, Shahad Abdulsahib, Panneerdoss Subbarayalu, Trong Phat Do, Prabhakar Pitta Venkata1, Saif Nirzhor, Mukund Bhandari, Siyuan Zheng, Yidong Chen, Gang Huang, Neelam Mukherje

Background: Triple-negative breast cancer (TNBC) is an aggressive malignancy with limited therapeutic options and poor long-term outcomes. Although immune checkpoint blockade has improved survival for a subset of patients, durable responses remain uncommon, underscoring the need for strategies that enhance tumor immunogenicity and immune memory. We recently identified a novel function of the transcription factor FOXM1 in promoting an immune-suppressive tumor microenvironment (TME) in TNBC by regulating stress ligands and the STING pathway. This study investigates the role of FOXM1 in shaping antitumor immune memory and leverages FOXM1 targeting to identify tumor antigens for therapeutic peptide vaccine development.

Methods: FOXM1 was knocked out in TNBC cell lines using CRISPR/Cas9, and syngeneic mouse models were utilized to evaluate tumor-immune interactions. Single-cell RNA sequencing (scRNA-seq) evaluated immune landscape remodeling following FOXM1 loss. Immunopeptidomics was performed to identify tumor-associated antigens enriched following FOXM1 loss. Candidate peptides were functionally validated using dendritic cell-based antigen presentation assays and patient-derived peripheral blood mononuclear cells (PBMCs). Clinical relevance was evaluated using TNBC patient datasets and immunotherapy response cohorts.

Results: scRNA-seq of syngeneic tumors revealed that FOXM1 deletion reshaped the immune microenvironment, with increased $CD8^+$ T cell and NK cell infiltration and expansion of memory T cell populations. Tumor-intrinsic FOXM1 suppressed antitumor immunity by downregulating MHC-I and the stress ligand ULBP1, impairing signaling pathways required for NK and $CD8^+$ T cell-mediated cytotoxicity. FOXM1-deficient tumors conferred durable protection upon tumor rechallenge, consistent with long-term immune memory. Analysis of patient datasets demonstrated that elevated FOXM1 and DNMT1 expression, coupled with reduced STING and ULBP1 levels, correlated with poorer survival and reduced responsiveness to immunotherapy. Leveraging enhanced antigen presentation in FOXM1-depleted cells, immunopeptidomics identified 275 differentially presented tumor antigens. Dendritic cell pulsing of the top 15 enriched peptides induced robust T cell responses in patient-derived PBMCs, supporting their immunogenicity and its potential to serve as bona fide candidates for a therapeutic vaccine tailored for TNBC patients.

Conclusions: These findings establish FOXM1 as a clinically relevant regulator of immune evasion and immune memory in TNBC. Importantly, the identification of immunogenic peptides provides a strong rationale for the development of a peptide-based therapeutic vaccine, either as *[Reached Word Limit]*

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Posterboard #: 45

Presenter: Nahid Iftkihar, BDS,MDS,MS
Institution: UT Health San Antonio
Category: Student

Pharmacologic Activation of Estrogen Receptor β Suppresses Tumor Growth and Enhances Chemosensitivity in Triple-Negative Breast Cancer

Nahid Iftikhar, Megharani Mahajan, Suryavathi Viswanadhapalli, Michael Tidwell, Stanton McHardy, Andrew Brenner, Ratna K. Vadlamudi, Uday P. Pratap

Background: Breast cancer is the most frequently diagnosed malignancy in women and a leading cause of cancer-related mortality in the United States. Triple-negative breast cancer (TNBC), representing approximately 15-24% of cases, is an aggressive subtype with poor prognosis and limited therapeutic options due to the absence of estrogen receptor alpha (ERα), progesterone receptor (PR), and HER2 expression. TNBC disproportionately contributes to breast cancer-related deaths and lacks clinically effective targeted therapies. Notably, TNBC tumors frequently express estrogen receptor beta (ERβ), which functions as a tumor suppressor; however, its clinical exploitation has been hindered by the lack of highly selective ERβ agonists with favorable pharmacologic properties. Here, we evaluated the therapeutic efficacy of CIDD-0149897, a novel and highly selective ERβ agonist, alone and in combination with chemotherapy in TNBC models.

Methods: CIDD-0149897 was evaluated in a panel of seven TNBC cell lines (SUM159, BT549, MDA-MB-468, HCC1806, HCC70, 4T1, and E0771). ERβ expression was assessed by RT-qPCR and Western blot analysis. Functional assays included cell proliferation, clonogenic survival, invasion, and apoptosis following treatment with CIDD-0149897 alone or in combination with standard chemotherapeutic agents. Mechanistic studies utilized RT-qPCR, ERβ reporter assays, and immunohistochemistry. In vivo antitumor efficacy was examined using TNBC xenograft and E0771 syngeneic mouse models.

Results: ERβ expression was detected at variable levels across all TNBC cell lines analyzed. Treatment with CIDD-0149897 produced a dose-dependent reduction in cell viability and significantly induced apoptosis. Reporter assays confirmed robust activation of ERβ transcriptional activity following treatment. ERβ overexpression enhanced sensitivity to CIDD-0149897, while ERβ knockout markedly attenuated therapeutic response, demonstrating target specificity. Gene expression profiling revealed modulation of ERβ-regulated pathways associated with tumor suppression. Combination treatment with CIDD-0149897 and chemotherapy demonstrated synergistic activity, significantly reducing proliferation, clonogenic survival, and invasive potential compared with monotherapy. In vivo, CIDD-0149897 significantly suppressed tumor growth in both xenograft and E0771 syngeneic models and reduced tumor cell proliferation, as evidenced by decreased Ki-67 staining.

Conclusion: Collectively, these findings establish ERβ as a therapeutically actionable tumor suppressor in TNBC and position CIDD 0149897 as a first in class ERβ agonist with immediate translational potential to enhance standard chemotherapy and expand targeted treatment options

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Posterboard #: 46

Presenter: Liah Jogi, BS
Institution: UT Health San Antonio
Category: Student

Who Brushes When Memory Fades?

Liah Jogi, Dr. Alexander Testa, Dr. Jiaming Liang, Dr. Rahma Mungia

Background: Alzheimer's disease and related dementias (ADRD) are progressive neurodegenerative conditions that significantly affect patients' ability to maintain oral health and access dental care. As cognitive decline advances, impairments in memory, executive function, and motor skills contribute to poor oral hygiene, increased risk of periodontal disease, and greater unmet dental needs. Caregivers play a critical role in supporting daily oral hygiene and facilitating dental visits; however, the extent of caregiver involvement and associated challenges in dental settings remain incompletely understood.

Methods: Data were obtained from the National Dental Practice-Based Research Network ADRD Quick Poll (n = 259). Dental providers reported on the proportion of patients with ADRD in their practice, methods of identifying cognitive impairment, frequency of caregiver interaction, interprofessional collaboration, perceived barriers to care, and interest in participating in future intervention studies. Descriptive statistics were used to summarize responses.

Results: Nearly all providers reported caring for patients with ADRD, though typically at low frequency; 74% estimated that fewer than 5% of their patients have ADRD, while 4% reported none. Identification of ADRD relied primarily on informal methods, including patient or caregiver report (89%) and documentation in records (63%), with 52% recognizing cognitive impairment through clinical observation; no respondents reported using formal screening tools. Caregiver engagement was high, with 87% of providers communicating with caregivers about oral health and home care. However, only 30% reported interprofessional collaboration and 10% reported making referrals for additional support. Key barriers included limited awareness of oral health importance (33%), communication challenges (19%), lack of standardized care integration systems (19%), and time constraints (19%). Overall, 44% of respondents expressed moderate to high interest in participating in studies to improve caregiver-supported oral hygiene and care coordination.

Conclusion: Dental providers frequently encounter patients with ADRD and actively engage caregivers; however, reliance on informal identification methods and limited interdisciplinary collaboration highlight gaps in care delivery. The findings support the need for structured caregiver education, improved care coordination, and development of targeted interventions to enhance oral health management for patients with ADRD in dental practice settings.

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Posterboard #: 47

Presenter: Thomas Kalantzakos
Institution: UT Health San Antonio
Category: Student

Murine models for triple-negative breast cancer with differential responsiveness to immunotherapy

Thomas J Kalantzakos, Yufan Zhou, Xingyu Liu, Joshua Proehl, Cameron Durfee, Ian Tamayo, Nuri Alpay Temiz, Benjamin Troness, Anusha Soni, Harshita B Gupta, Reuben S Harris

Breast cancer is the most common cancer diagnosis in women. Clinical studies with triple-negative breast cancer (TNBC) are encouraging for immunotherapy combined with chemotherapy (anti-PD-1 with paclitaxel and/or carboplatin). However, additional clinical advances may be pursued more rapidly with assistance from preclinical TNBC models including syngeneic mammary tumor cell lines. Here, we report two mammary tumor cell lines that exhibit differential responsiveness to immunotherapy in vivo. Spontaneous mammary tumors from C57BL/6J MMTV-Cre Trp53fl/+ animals were passaged serially in cell culture and in vivo in the mammary fat pad of fully wildtype animals. The resulting lines, MM001i and MM008i, lost Trp53 and formed 1000 mm3 tumors in the mammary fat pad within 21-28 days. Despite originating from the same genetic background, these lines exhibit differential responses to immunotherapy. For anti-PD-1 therapy, MM001i is poorly responsive and MM008i is strongly responsive with near-complete tumor regression. In comparison, both MM001i and MM008i respond rapidly to anti-CTLA-4 therapy. Both models express unique tumor antigens as evidenced by immunity to subsequent engraftments. Primary MM008i tumors exhibit greater T cell infiltration, and CD8-positive T lymphocytes are required for anti-PD-1 responses. These TNBC models are promising for further mechanistic studies and testing future single and combinatorial therapies.

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Posterboard #: 48

Presenter: Bhuwaneswor Kandel, MS
Institution: UT Health San Antonio
Category: Student

Vitamin A boosts IgA CSR through potentiation of TGF-β pathway and promotes plasma cell differentiation for maturation of IgA antibody response

Bhuwaneswor P. Kandel, Zhimin Yang, Shili Li, Kaiyue Zhang, Zhenming Xu, Carlos E. Rivera and Paolo Casali

Vitamin A boosts IgA CSR through potentiation of TGF-β pathway and promotes plasma cell differentiation for maturation of IgA antibody response Vitamin A is an essential factor in immunity. Indeed, vitamin A deficiency impairs vaccine-induced specific antibody responses and compromises host defense against bacterial and viral infections, largely by undermining production of IgA antibodies. Yet, B cell intrinsic activity of retinoic acid (RA), the active metabolite of vitamin A, and the mechanisms by which RA contributes to IgA production remain poorly understood. Here we show that RA boosts T-dependent and T-independent IgA antibody responses as induced by conjugated haptens NP-ovalbumin (OVA) and NP-Escherichia coli lipopolysaccharide (LPS) as well as Salmonella Typhimurium flagellin in C57BL/6 and T cell-deficient Tcrβ-/-Tcrδ-/- mice at systemic and mucosal levels. Our findings extend to the human antibody response to SARS-CoV-2 receptor binding domain (RBD) as induced by Pfizer COVID-19 mRNA vaccine in our humanized THX mice1. RA does not induce class-switch recombination (CSR) to IgA, as evidenced by failure of RA to induce CSR to IgA in immunized AicdaCreTgfbr2fl/fl and NBSGW/B mice (immunodeficient NBSGW mice lacking all immune cells/elements and grafted with purified C57BL/6 B cells), or in in vitro stimulated AicdaCreTgfbr2fl/fl B cells and TGF-β-starved C57BL/6 B cells. Instead, RA synergistically enhances TGF-β-induced CSR to IgA by potentiating transcription of germline Iα-Cα and TGF-β-responsive Tgfb1, Tgfb3, Tgfbr1 and Smad3 genes, while additionally upregulating Runx2 expression by up to 25-folds. RA also promotes differentiation of BCR-crosslinked naïve IgM^+IgD^+ B cells and class-switched IgA^+ B cells into IgM^+ or IgA^+ plasmablasts/plasma cells by promoting Prdm1 (BLIMP-1) expression. RA promotes transcription of these genes by facilitating dissociation of nuclear co-repressor (NCoR), while concomitantly inducing recruitment of nuclear co-activators (NCoAs) to RXRα/RARα heterodimers bound to RA response elements (RAREs) within Tgfb1, Tgfb3, Tgfbr1 and Smad3 promoter regions - RA-mediated enhancement of gene expression is abrogated upon treatment with the pan-RAR inverse agonist BMS493. Thus, RA is a critical vitamin A metabolite central to supporting antibody production, particularly IgA.

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Posterboard #: 49

Presenter: Victoria Mai
Institution: UT Health San Antonio
Category: Student

Targeting RNA demethylase ALKBH5 with mefloquine enhances antitumor immunity and reduces Osteosarcoma progression

Victoria Mai, Daisy Medina, Santosh Timilsina, Panneerdoss Subbarayalu, Deepika Singh, Phat Do, Shuo Zhou, Yogesh Gupta, Daohong Zhou, Prabhakar Pitta Venkata, Yidong Chen, Manjeet Rao The University of Texas Health Science Center at San Antonio

Osteosarcoma (OS) is the most common primary bone malignancy in children and adolescents, with a second incidence peak in adults over 50 years of age. Standard treatment consists of intensive chemotherapy and surgery; however, outcomes for patients with metastatic or relapsed disease remain poor. Approximately 20% of patients present with metastases at diagnosis, and five-year survival is below 30%, underscoring the urgent need for new therapeutic strategies. We recently identified the RNA demethylase ALKBH5 as a critical promoter of OS growth and metastasis. Genetic depletion of ALKBH5 using shRNA and CRISPR-Cas9 significantly suppressed OS cell proliferation and tumor burden in vitro and in vivo. Through fluorescence-based high-throughput screening of FDA-approved and LOPAC compound libraries, we identified mefloquine as a small-molecule inhibitor of ALKBH5. RNA sequencing revealed that mefloquine treatment upregulated immune-related gene pathways. In vitro studies using human and murine OS models demonstrated potent growth inhibition by mefloquine. Surface plasmon resonance and m6A dot blot assays confirmed direct interaction between mefloquine and ALKBH5 and inhibition of ALKBH5 demethylase activity. In vivo efficacy was evaluated using subcutaneous tail vein mouse models of OS. Mefloquine treatment significantly reduced primary tumor growth and metastatic burden and enhanced responses to immune checkpoint blockade. Ongoing studies aim to further define the molecular mechanisms underlying mefloquine-mediated suppression of OS progression. Together, these findings establish ALKBH5 as a therapeutic vulnerability in osteosarcoma and identify mefloquine as a promising inhibitor that suppresses tumor growth and metastasis while enhancing antitumor immune responses. As an FDA-approved antimalarial agent, mefloquine represents a potential repurposable drug for the treatment of osteosarcoma.

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Posterboard #: 50

Presenter: Ali Mansoor
Institution: UT Health San Antonio
Category: Student

Unlocking Legacy Clinical Data Using TRACER: A Framework for Transforming Scanned Records into Research-Ready Data

Ali Mansoor, Syed Mahanaz, Dr. Jonathan Gelfond, Brittany Antopia, Dr. Jannine Cody

Background
Decades of clinically rich information remain locked in scanned medical records, limiting their use for research, cohort identification, and real-world evidence generation. This challenge is particularly pronounced in rare disease research, where longitudinal data are often collected from multiple sources and stored as unstructured or scanned documents. Existing OCR-based methods have largely been evaluated in controlled datasets and may not generalize to heterogeneous, real-world clinical records.

Objective
To develop and evaluate a framework for transforming scanned clinical documents into structured, research-ready data while preserving clinically meaningful information.

Methods
We developed TRACER (Traceable Review and AI-assisted Clinical Extraction for Research), a pipeline that converts scanned medical records into structured data. Using documents from the Chromosome 18 Clinical Research Center, we constructed a gold reference corpus from 10 documents through dual independent annotation, achieving high inter-annotator agreement (Cohen's κ = 0.86). OCR performance was evaluated at the entity level using recall-based metrics to assess preservation of clinically relevant information, including exact recall, strict recall (allowing near-exact matches ≥90% similarity), and miss rate.

Results
Across 10 documents and 329 gold-standard entities, OCR achieved 80% exact recall and 94% strict recall, with a miss rate of 4%. For clinical findings (N = 249), strict recall reached 100%, indicating near-complete preservation of clinically meaningful information. Performance was lower in variable-format fields (N = 80), with 75% strict recall and 15% miss rate, reflecting challenges associated with dense and structurally complex clinical documents. Conclusion This study demonstrates that clinically relevant information can be reliably preserved from heterogeneous scanned medical records using a structured OCR-based pipeline. By prioritizing information preservation and traceability, this approach enables transformation of legacy clinical data into research-ready formats. Impact This work addresses a critical gap in clinical research informatics by unlocking inaccessible historical data. Ongoing work will expand the corpus (n = 100), validate clinical fact extraction, and evaluate cohort-level impact to support scalable, multi-site translational research and future NIH funding.

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Posterboard #: 51

Presenter: Ricardo Martinez, MS
Institution: UT San Antonio
Category: Student

Midkine as a Therapeutic Vulnerability in Hepatocellular Carcinoma: Preclinical Evaluation of HBS-101

Ricardo A. Martinez, Aditi Subramanya, Sridharan Jayamohan, Adriana Baker, Khaled M. Nassar, Baskaran Subramani, Franchesca C. Tinacba, Megharani Mahajan, Hareesh B. Nair , Gangadhara R. Sareddy, Suryavathi Viswanadhapalli, Ratna K. Vadlamudi

Hepatocellular carcinoma (HCC) is the most common primary liver malignancy and is characterized by its poor prognosis (5 year survival ~5-18%) and poor response rate to current therapies (~20%). By 2026, this disease is anticipated to burden more than one million lives. Texas in general and the Mays Cancer Center catchment area, specifically demonstrates the highest incidence of HCC nationwide, highlighting an urgent need for targeted research. These poor outcomes represent the urgent need to identify new, mechanism-based therapeutic strategies for HCC.

HCC cells, although heterogenous, nearly ubiquitously over-express the small heparin binding growth factor, Midkine (MDK) both in patient sera and in vitro. Further, MDK over-expression in HCC is correlated with poor prognosis, and transcriptomic associations between MDK and numerous pro-oncogenic pathways have been identified. Additionally, while MDK is markedly overexpressed in many cancers, including HCC, it is typically minimally expressed in normal adult tissues. The absence of a selective direct-acting small-molecule MDK inhibitor represents a critical gap in translational therapeutic development against HCC and other MDK-overexpressing cancers. My mentor's laboratory has recently developed a first in class MDK inhibitor, HBS 101.

The objective of this study is to define the molecular mechanisms by which HBS-101 inhibits MDK signaling in HCC and evaluate its preclinical therapeutic potential. My central hypothesis is that HBS 101 suppresses HCC progression by directly inhibiting MDK and its downstream oncogenic signaling pathways, resulting in strong preclinical efficacy against HCC.

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Posterboard #: 52

Presenter: Khaled Nassar, MS
Institution: UT Health San Antonio
Category: Student

Inhibiting PELP1 signaling enhances the therapeutic effectiveness of topoisomerase inhibitors through DNA damage and replication stress in triple-negative breast cancer

Khaled Mohamed Nassar, John R. Sanchez, Durga Meenakshi Panneerdoss, Behnam Ebrahimi, Xue Yang, Uday P. Pratap, Megharani Mahajan, Salvador Cardenas Alejo, Panneerdoss Subbarayalu, Daohong Zhou, Rajeshwar R. Tekmal, Gangadhara Reddy Sareddy, Manjeet K. Ra

Background: Triple-negative breast cancer (TNBC) is an aggressive malignancy with metastatic potential and limited targeted therapeutic options, accounting for 15-24% of breast cancer-related deaths. Proline-, glutamic acid-, and leucine-rich protein 1 (PELP1) is an oncogenic scaffolding protein deregulated in TNBC and associated with poor survival. However, its mechanistic role in DNA repair and replication stress is incompletely defined. We hypothesized that PELP1 signaling in DNA repair and replication stress represents a therapeutic vulnerability that can be exploited using the PELP1 inhibitor SMIP34 in combination with topoisomerase inhibitors (TIs) in TNBC.

Methods: IPTG-inducible PELP1 knockdown (PELP1-iKD) TNBC models (MDA-MB-231, BT-549, HCC-1806, SUM-149) were generated via lentiviral transduction. PELP1 signaling was suppressed genetically (IPTG) and pharmacologically using SMIP34. A 140 FDA-approved drug MTT-based screen identified combinatorial vulnerabilities. Functional and mechanistic analyses included colony formation, Annexin V/PI apoptosis, RT-qPCR, Western blotting, comet assays, homologous recombination (HR) and non-homologous end joining (NHEJ) reporter assays, DNA fiber assays, cell-cycle profiling, confocal microscopy, proximity ligation, and immunoprecipitation, with vehicle and non-targeting controls. Therapeutic efficacy of SMIP34 combined with TIs was validated in cell line-derived xenografts (CDXs), patient-derived organoids (PDOs), and patient-derived xenografts (PDXs).

Results: IPTG induction produced dose-dependent PELP1 depletion across TNBC models, significantly reducing proliferation and clonogenic survival while enhancing apoptosis. PELP1 knockdown increased S-phase accumulation, indicating replication stress vulnerability. PELP1 loss disrupted DNA damage signaling, evidenced by sustained γ-H2AX accumulation and attenuated activation of phospho-ATM, ATR, and DNA-PKcs. Mechanistically, PELP1 depletion impaired RAD51 recruitment, suppressed HR activity, altered NHEJ repair dynamics, and exacerbated replication stress, sensitizing TNBC cells to TIs. SMIP34 phenocopied genetic depletion, confirming on-target effects. Drug screening identified TIs as synergistic partners. Both SMIP34+TIs and PELP1-iKD+TIs combinations markedly increased DNA damage burden and comet tail moments compared to monotherapies. In advanced models, SMIP34 combined with mitoxantrone significantly suppressed PDO proliferation and reduced CDX and PDX tumor growth compared to single agents.

Conclusion: To our knowledge, this is the first study to mechanistically link PELP1 to HR and NHEJ regulation, replication stress, and S-phase vulnerability in TNBC, establishing a strong mechanistic and translational foundation for PELP1-targeted combination strategies.

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Posterboard #: 53

Presenter: Durga Meenakshi Panneerdoss
Institution: UT San Antonio
Category: Student

LIPA Inhibition Enhances the Therapeutic Efficacy of DNA-Damaging Agents in Ovarian Cancer Through Induction of ER Stress and Enhancing DNA Damage

Durga Meenakshi Panneerdoss, Tae-Kyung Lee, Khaled Mohamed Nassar, Gaurav Sharma, Scott Elmore, Henry Neal, William Cole Arnold, Edward R. Kost, Suryavathi Viswanadhapalli, Jung-Mo Ahn, Ganesh V. Raj, and Ratna K. Vadlamudi

Background: Ovarian cancer (OCa) is the most lethal gynecologic malignancy in the United States, largely due to the lack of effective early detection strategies and the development of chemoresistance following initial treatment. These challenges emphasize the urgent need for improved therapeutic approaches. Recently, our team identified ERX-208, a potent tris-benzamide molecule with strong activity against OCa cells. ERX-208 targets lysosomal acid lipase A (LIPA), inducing endoplasmic reticulum (ER) stress, disrupting protein synthesis, and promoting apoptosis. The objective of this study is to evaluate the potential of ERX-208 in enhancing the efficacy of FDA-approved chemotherapeutics.

Methods: We screened 147 FDA-approved chemotherapy drugs in combination with ERX-208 to evaluate their impact on OCa cell viability. Synergy was assessed using SynergyFinder Plus and validated through in vitro assays examining proliferation, colony formation, cell cycle, DNA damage, apoptosis, and invasion. The combination's efficacy was further tested in patient-derived organoid (PDO) and xenograft (PDX) models.

Results: Combination therapy screening of 147 FDA-approved chemotherapeutic agents with the LIPA inhibitor ERX-208 identified multiple synergistic drug pairs in OCa models. SynergyFinder analysis revealed strong combination sensitivity and synergy scores for several DNA-damaging agents. ERX-208 monotherapy inhibited OCa cell growth in vitro and in vivo, consistent with its mechanism of inducing LIPA-dependent ER stress. Combination treatments with paclitaxel or cisplatin further amplified these effects, significantly reducing cell viability across OCa cell lines compared to monotherapy. Mechanistic studies demonstrated enhanced gamma-H2AX accumulation, increased DNA damage, robust induction of ER stress markers, and reduced invasion following combination therapy. In PDO and PDX models, ERX-208 combined with DNA-damaging agents produced marked tumor growth suppression beyond monotherapies, confirming the translational relevance of the synergy. Furthermore, ERX-208 effectively reduced the viability of therapy-resistant OCa models in vitro and inhibited xenograft growth in vivo. These findings support ERX-208 as a potent ER stress-inducing agent that enhances the therapeutic efficacy of standard chemotherapies in OCa models.

Conclusions: Our findings demonstrate that combining ERX-208 with DNA-damaging agents significantly enhances therapeutic efficacy, highlighting the potential of ERX-208-based combination therapy for treating OCa.

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Posterboard #: 54

Presenter: Esteban Perez, DO
Institution: UT Health San Antonio
Category: Student

Dietary Supplement Use Is Common but Poorly Captured in Electronic Health Records: A Nationally Weighted Analysis and Health-System EHR Comparison

Esteban Perez, Jonathan Gelfond, Anish Saikumar

Herbal and dietary supplement (HDS) use is widespread in the United States and represents an increasingly recognized cause of drug-induced liver injury (DILI). Accurate identification of hepatotoxic exposures relies on reliable documentation, yet discrepancies between actual supplement use and electronic health record (EHR) capture remain poorly defined.

This mixed-methods analysis compared national HDS use patterns from the National Health and Nutrition Examination Survey (NHANES) to EHR data from a large academic safety-net health system. The goal was to quantify discrepancies between population-level exposure and clinical documentation, informing quality improvement strategies for early detection of supplement-associated DILI.

National prevalence estimates were derived from NHANES 2017-2018 among adults aged ≥20 years using the dietary supplement questionnaire (DSD010). Weighted analyses estimated overall and demographic-specific prevalence. Local EHR patterns were assessed via Epic SlicerDicer, querying structured medication fields for nutritional and herbal supplements. Filters were applied to include adult, living patients and exclude non-relevant entries. Findings were contextualized by a case of biopsy-confirmed supplement-associated liver injury.

Among 15,560 NHANES participants, the weighted national prevalence of supplement use was 52.5%, rising with age (20-39 years: 44.5%; 40-59 years: 58.8%; ≥60 years: 75.1%) and higher among females (65.0%) than males (51.5%). Prevalence varied by race/ethnicity, with lower use among Mexican American adults (45.1%) compared to non-Hispanic White adults (63.4%). In contrast, institutional EHR queries of approximately 3.1 million patients identified only about 4,000 individuals with any supplement-related entry; after exclusions, roughly 120 adult patients remained with documented supplement use.

These findings reveal significant under-capture of dietary supplement exposure in structured EHR fields relative to nationally representative data. Documentation gaps may delay or obscure recognition of supplement-associated DILI, as reflected in an example of hepatotoxic supplement exposure from San Antonio, TX. Improving outpatient screening and structured EHR documentation can strengthen early DILI detection, patient counseling, and surveillance of supplement-related hepatotoxicity.

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Posterboard #: 55

Presenter: Jack Prochnau
Institution: UT Health San Antonio
Category: Student

Exploiting Scaffold/Matrix Attachment Region-Dependent Genomic Instability for Treating Pancreatic Ductal Adenocarcinoma

Jack Prochnau, Subapriya Rajamanickam, Daisy Medina, Xena Huang, Panneerdoss Subbarayalu, Deepika Singh, Stanton McHardy, Manjeet Rao

Pancreatic ductal adenocarcinoma (PDAC) remains a highly lethal malignancy with a five-year survival near 13%, driven by late diagnosis, intrinsic therapeutic resistance, and heavy reliance on stress-adaptation pathways, including the DNA damage response (DDR). Although current regimens exploit PDAC's dependence on DNA repair, durable benefit is limited by adaptive resistance and dose-limiting systemic toxicity. In parallel, PDAC cells rely on dysregulated RNA processing and proteostasis to maintain growth under chronic stress, suggesting an opportunity for therapies that target multiple nonredundant vulnerabilities. Scaffold/matrix attachment regions (S/MARs) are A/T-rich regulatory DNA elements that anchor higher-order chromatin organization and influence transcription, replication, and DNA repair. These regions can be co-opted in cancer to support oncogenic gene programs and treatment resistance. We identified Carbazole Blue (CB) as a small molecule that preferentially associates with A/T-rich S/MAR sequences. CB exposure induces DNA damage and suppresses transcriptional programs linked to replication and repair. CB also disrupts HMGA1, a chromatinassociated architectural factor implicated in PDAC aggressiveness and resistance, consistent with impaired tumor plasticity under genotoxic stress. Transcriptome profiling further revealed a pronounced depletion of spliceosomal U1 snRNAs following CB treatment, accompanied by widespread splicing defects. These RNA processing disruptions coincide with proteotoxic stress responses and apoptotic cell death, indicating that CB couples chromatin-directed damage with collapse of RNA splicing and proteostasis. Together, these findings support a model in which CB exerts tumor-selective cytotoxicity by simultaneously targeting S/MAR-dependent chromatin regulation, DDR-linked survival programs, and spliceosome integrity. Our work therein is focused on mechanistically mapping CB-chromatin engagement, defining the causal link between U1 depletion and proteostasis failure, and evaluating antitumor activity and combinatorial potential with standard-of-care agents in translational PDAC models.

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16TH ANNUAL FRONTIERS OF TRANSLATIONAL SCIENCE RESEARCH DAY

Posterboard #: 56

Presenter: Athena Santi
Institution: UT San Antonio
Category: Student

Chemomagnetic Capsaicin Delivery for TRPV1 Modulation in Chronic Pain and Neural Repair

Athena Santi, Nicolas Muzzio, and Gabriela Romero Uribe

Neuropathic pain is a severe and often debilitating condition affecting approximately 50 million people in the United States. Despite its prevalence, current treatments rely on opioids, which pose significant risks of addiction and systemic side effects. Capsaicin, a chemical derived from chili peppers, has emerged as a promising non-opioid analgesic. Its effects are mediated through binding to the transient receptor potential vanilloid 1 (TRPV1) channel located on sensory neurons. With continuous and prolonged exposure, TRPV1 becomes desensitized, producing sustained analgesia. However, clinical use of capsaicin is limited by poor solubility, rapid degradation, and low tissue retention. To overcome these limitations, we developed a magnetic field-controlled delivery system for capsaicin that enables precise, repeatable, on-demand modulation of TRPV1 activity.

Iron oxide magnetic nanoparticles (MNPs) were surface-functionalized to grow thermoresponsive poly (oligo (ethylene glycol) methyl ether methacrylate) (POEGMA) brushes, forming a nanoscale capsaicin reservoir. Upon exposure to an external alternating magnetic field (AMF), MNPs undergo hysteresis power loss, generating localized nanoscale heat. This heat triggers a reversible thermodynamic phase transition in the POEGMA coating, resulting in a microdose release of capsaicin. Primary hippocampal neurons isolated from newborn Sprague-Dawley rats were used to assess TRPV1 activation and desensitization via calcium imaging. Cell differentiation experiments were performed using ND 7/23 cells (DRG hybrid cell line) in culture media to evaluate the impact of the local release of capsaicin on nerve differentiation and regeneration.

A single dose of AMF-triggered release activates TRPV1 in >75% of neurons, while 3+ AMF cycles cause TRPV1 desensitization in >90% of neurons, achieving analgesic effects consistent with capsaicin's therapeutic mechanism. Importantly, when the AMF is turned off, any remaining capsaicin is re-encapsulated for subsequent release. Minimal cytotoxicity was observed in primary rat neuronal cultures, indicating biocompatibility. Additionally, in ND7/23 cells, capsaicin delivery promoted morphological signs of neuronal differentiation, suggesting potential regenerative benefits.

Collectively, our findings establish POEGMA-functionalized MNPs as a viable platform for remote-controlled, repeatable, and localized neuromodulation with preserved neuronal viability. By enabling magnetically triggered, localized delivery of hydrophobic drugs like capsaicin, this technology has the potential to transform chronic pain treatment by advancing precise, nonopioid therapeutic strategies.

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Posterboard #: 57

Presenter: Dillon Shadowen
Institution: Texas A&M University
Category: Student

Assessing Chronic Microglial Activation Following Insulin-Like Growth Factor 1 Supplementation After Traumatic Brain Injury

Dillon Shadowen, Bharat Salvady, Brock Braden, Reagan Dominy, Gabriel Arisi, Jaclyn Iannucci, Lavanya Venkatasamy, Lee A. Shapiro

Traumatic Brain Injury (TBI) is a leading cause of death and disability worldwide and is associated with aberrant signaling within the hypothalamic-pituitary axis, contributing to decreased levels of insulin-like growth factor 1 (IGF-1). IGF-1 supplementation following TBI has demonstrated beneficial effects in preclinical and clinical trials. However, the extent of systemic IGF-1 on neuroinflammation has yet to be ascertained. Microglia are the immune cells of the brain and are involved in regulation of neuroinflammation. Morphological alterations to microglia following TBI contribute to chronic neuroinflammation and increased risk of neurodegenerative diseases. However, the effects of IGF-1 on the microglial response following TBI have not been fully investigated. We hypothesized that IGF-1 supplementation following TBI would alleviate alterations in microglia. Ten-week-old male Sprague Dawley rats received a mild-to-moderate lateral fluid percussion injury (FPI) or sham surgery followed by IGF-1 or vehicle via intraperitoneal injection at 4- and 24-hours post-injury. Brains were collected 35 days post-injury, processed, then stained with anti-Iba1 to visualize microglia. Utilizing unbiased stereology, Iba1+ cells were quantified in the subregions of the dentate gyrus. Quantitative analysis revealed non-significant increases in Iba+ cells in the FPI group that were not reduced by IGF-1 treatment. These findings suggest that at no significant differences were observed for the number of microglial cells in the dentate gyrus. However, morphological or genetic analysis may reveal that despite a lack of quantitative difference, the activation state of microglial cells may be altered by IGF-1 treatment after TBI. Future studies are needed to assess these possibilities.

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Posterboard #: 58

Presenter: Annie Smelter, BS
Institution: UT Health San Antonio
Category: Student

Hepatic TBX3 Overexpression Disrupts Ammonia Detoxification in Mice

Annie Smelter, Iriscilla Ayala, Skanda Hebbale, Edgar Hinostroza, Marcel Fourcaudot, and Luke Norton

Background: The liver plays an essential metabolic role by regulating glucose, lipid, and amino acid metabolism, as well as detoxifying harmful substances in the body. Hepatocytes can be divided into different zones, significant as hepatocytes in different zones perform different metabolic functions. The transcriptional regulation of these zones is not well understood. In our recent studies, it appears that T-box transcription factor 3 (TBX3) cooperates with transcription factor 7-like 2 (TCF7L2) to regulate zone-specific gene expression. However, the metabolic function of TBX3 in the liver is poorly understood.

Methods: To determine the impact of TBX3 overexpression in wild-type mouse liver, we employed a liver specific adeno-associated virus 8 (AAV8) delivery of either Gfp or Tbx3 mRNA under a hepatocyte-specific promoter. Mice were treated with a single retroorbital injection of either AAV8-GFP or AAV8-TBX3 and observed for 14 days. Body weight, food intake, and plasma ammonia levels were tracked throughout the experiment.

Results/Conclusion: Mice treated with AAV8-TBX3 demonstrated robust mRNA and protein overexpression, as well as rapid and significant body weight reduction in the absence of a change in food intake. Zonal gene expression was minimally altered, with significant changes in the gene expression of key ammonia detoxification genes that correlated with a significant elevation of plasma ammonia levels. These changes, along with the significant elevation in plasma ammonia, suggest that the rapid weight loss and lethality observed in the final days of the experiment may have been due to ammonia toxicity.

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Posterboard #: 59

Presenter: Haven Tillmon
Institution: UT Health San Antonio
Category: Student

TPPP Dysfunction Drives Complex I Deficits in Drosophila and Parkinson's Disease

Haven Tillmon, Savannah Boyen, Marco Sciortino, Swati Banerjee

Tubulin polymerization-promoting proteins (TPPPs) constitute a highly conserved family of microtubule-associated proteins that play essential roles in tubulin stabilization, bundling, and cytoskeletal organization. Despite their evolutionary conservation, the role of mammalian TPPP in disease is largely unknown, creating a gap in our understanding of TPPP dysfunction in synucleinopathies like Parkinson's disease (PD). Previously, our lab identified the only Drosophila homolog of human TPPP called Ringmaker (Ringer). We have shown that ringer mutants recapitulate some of the prominent features of PD, including reduced lifespan, locomotor deficits, neurodegeneration, and mitochondrial structural damage and bioenergetic dysfunction. Although TPPPs were initially thought to be predominantly cytoskeletal, our prior studies indicate that Drosophila with a genetic deletion of ringer display mitochondrial structural and bioenergetic deficits. The objective of this project is to define how Ringer regulates electron transport chain (ETC) complex I integrity and bioenergetic function while also identifying pharmacological interventions that can improve complex I function. These studies will test the overarching hypothesis that mitochondrial dysfunction in Ringer-deficient flies arises from impaired complex I assembly and activity. Building on our previous findings, our recent preliminary data reveal a striking and specific disruption of complex I in Ringer-deficient flies. We observed decreased protein levels of core complex I subunits NDUFS2 and NDUFS3, as well as reduced expression of complex I assembly proteins ECSIT and NDUFAF1 in ringer mutants. These molecular deficits coincide with diminished complex I enzymatic activity in ringer mutants. Furthermore, subcellular fractionation immunoblotting demonstrates a pronounced shift in NDUFS3 protein distribution by density, with milder but detectable shifts in ECSIT and NDUFAF1. Consistent with these findings, human PD tissue from Brodmann area 40 of the parietal cortex shows decreased complex I activity and reduced ECSIT and NDUFS3 levels in males, along with decreased NDUFAF1 levels in females. Together, these data indicate that TPPP loss disrupts early steps of oxidative phosphorylation by impairing complex I function both in a fly model of PD and in human PD. These studies will establish a mechanistic framework for understanding TPPP contribution to mitochondrial function and highlight opportunities for therapeutic intervention aimed at preserving complex I activity.

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Posterboard #: 60

Presenter: Gabriela Torres, BS
Institution: UT Health San Antonio
Category: Student

Investigating the Effects of Vortioxetine on Cognitive Impairment After Androgen Deprivation Therapy

Gabriela Torres, Sarah E. Bulin, Destiny Arenas, Bryce W. Latimer, David A. Morilak

Prostate cancer is the most common type of cancer in American men after basal cell and squamous cell carcinoma, and it's the leading cause of cancer death after lung cancer. Androgen deprivation therapy (ADT) is an effective treatment for prostate cancer and as an adjuvant to treat localized tumors. Unfortunately, more than half of patients treated with ADT experience a decline in cognitive domains, such as executive function and memory, negatively impacting their quality of life. Vortioxetine (VTX) is a multimodal antidepressant that has been shown to improve cognition independently when compared to other antidepressants. We have previously shown that ADT induces deficits in executive function, mediated by the medial prefrontal cortex (mPFC), and in visuospatial memory, mediated by the hippocampus, in healthy cancer-free rats, and that chronic administration of VTX rescued these deficits. However, the effects of VTX on cognition after ADT in the context of prostate cancer pathophysiology remain unexplored. Using a rat model of prostate cancer and ADT, rats were ectopically implanted with syngeneic prostate tumors, treated with GnRH antagonist Degarelix for ADT treatment, and administered VTX (40mg/kg/day) via diet. Behavioral tests including the novel object location (NOL) test and attentional set-shifting test (AST) were conducted starting 21 days after ADT onset. Results from the hippocampal-mediated NOL indicate that animals with either tumor alone or tumors treated with DGX displayed impaired visuospatial memory. Treatment with VTX in the diet reversed this impairment in the tumor group treated with DGX, but not in the tumor alone group. Similarly for the AST, mediated by the mPFC, rats with the tumor alone and the tumor undergoing ADT had significantly impaired cognitive flexibility, a core executive function, and these deficits were reversed by VTX only in the tumor-DGX group. In conclusion, ADT and prostate tumors both produce impairments in cognition mediated by the hippocampus and mPFC. Impairment induced by ADT can be reversed by chronic administration of VTX. This evidence suggests that ADT and cancer induce cognitive deficits through different mechanisms. Ongoing experiments are focused on evaluating the effects of acute VTX treatment on ADT-induced cognitive deficits.

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Posterboard #: 61

Presenter: Adya Tripathi
Institution: Jose M Lopez Middle School
Category: Student

Stabilizing Soil to Reduce Structural Foundation Movement by Using Novel, Non-toxic, and Sustainable Additives

Adya Tripathi

Each year, unstable soils drive infrastructure failure, resulting in billions of dollars in damage and significant public health risks through structural collapse, environmental disruption, and exposure to hazardous materials. Expansive clay soils, characterized by high shrink-swell capacity due to moisture fluctuations, are a primary cause of foundation instability worldwide, accounting for over $15 billion in annual damage.

Conventional soil stabilization relies on chemical additives such as cement and lime, whose production is energy-intensive and emits over 4 billion tons of CO_2 annually. These emissions exacerbate climate change and are associated with adverse health outcomes, including respiratory illness, heat-related mortality, degraded air quality, and increased transmission of infectious diseases. Additionally, production and application release particulate matter (PM) and toxic byproducts that may leach into soil and groundwater, elevating risks of chronic respiratory conditions, neurological disorders, and long-term environmental contamination.

This study investigates waste-derived additives as sustainable, cost-effective alternatives for stabilizing expansive clay soils while mitigating environmental and health impacts. Materials tested include a chicken feather-eggshell (CF-ES) composite, red slate (RS), and wheat husk (WH), all of which are commonly discarded and contribute to landfill accumulation. Repurposing these materials reduces solid waste and limits reliance on high-emission industrial stabilizers.

Soil stabilization effectiveness was evaluated through reductions in plasticity index (PI, soil's moisture-based movement) and soil swell percentage with a concentration of 5% within the soil. Untreated clay served as the control, with a PI of 56 and swell of 5.02%. Laboratory testing included Atterberg Limits (LL, PL, PI), one-dimensional swell testing, Proctor compaction, and No. 200 sieve analysis to characterize physical and mechanical behavior.

All additives improved soil stability relative to the control. The CF-ES composite demonstrated the highest performance, reducing swell to −0.22%. RS reduced PI to 30 with moderate consolidation, while WH showed limited PI reduction but contributed to swell mitigation. Results indicate that waste-based additives exceed industrial performance thresholds, supporting their implementation as low-emission, health-conscious alternatives that reduce infrastructure failure risk and minimize environmental and human toxic exposure, reducing billions of tons of C02 emissions and 500 hundred million tons of waste.

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Posterboard #: 62

Presenter: Raquel Velazquez
Institution: UT Health San Antonio
Category: Student

Functional Characterization of Human Alpha-Synuclein In Transgenic Fly Models

Raquel Velazquez, Paloma Diaz, and Swati Banerjee

The human alpha-synuclein (a−Syn) protein encoded by the SNCA gene is a causative gene that has been linked to familial Parkinson's disease (PD). The importance of a−Syn has been connected to a critical role in synaptic regulation, including synaptic activity, plasticity, and neurotransmitter release. Several pathogenic point mutations in the gene have been identified, however, their role in the development and progression of PD is not well understood. Transgenic Drosophila melanogaster (fly) lines have been generated to express pathogenic human a−Syn mutations such as A30P, A53T, E46K, G51D, and H50Q to investigate the consequences of pathogenic human a−Syn in flies. We used fly genetics, lifespan analysis, climbing assays, immunohistochemistry, confocal imaging, and western blotting to test whether expression of pathogenic human a−Syn mutations in flies recapitulates key features of PD. Our preliminary data shows the recapitulation of key features associated with PD such as reduced lifespan, progressive locomotor deficits, and protein aggregation. Future studies aim to determine the extent of protein aggregation in the various a−Syn mutations, neurodegeneration of dopaminergic neurons, and the presence of mitochondrial dysfunctions. These findings highlight the utility of Drosophila as an experimental model for the functional characterization of human a−Syn and for investigating its mechanistic underpinnings.

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Posterboard #: 63

Presenter: Sandra Villegas Ibarra
Institution: UT San Antonio
Category: Student

Association Between Traumatic Brain Injury and Post-Traumatic Stress Disorder: Evidence from a Large Commercially Insured Population

Villegas Ibarra, S. & Howard, J.

Background: Traumatic brain injury (TBI) represents a significant public health burden, affecting millions of individuals annually. Beyond the immediate physical consequences, TBI has been increasingly recognized as a risk factor for psychiatric disorders, particularly post-traumatic stress disorder (PTSD). While prior research suggests a link between TBI and PTSD, longitudinal evidence quantifying this relationship in large, diverse populations remains limited. Understanding the magnitude and persistence of this association is critical for informing clinical screening protocols and mental health intervention strategies for individuals with TBI history.

Objective: To examine the association between TBI and PTSD incidence over a 10-year follow-up period in a large commercially insured population. Methods: We conducted a retrospective cohort study using data from the Merative MarketScan Commercial Claims and Clinical Encounters Database. The analytic sample included 844,509 individuals, of whom 3,796 had a TBI hospital admission in 2015 and 840,713 (5% random sample of database) did not. PTSD incidence was identified using ICD diagnosis codes during follow-up. We employed Kaplan-Meier survival analysis to estimate PTSD-free survival probabilities and Cox proportional hazards regression to calculate hazard ratios (HR) and 95% confidence intervals (CI), adjusting for age group, sex, geographic region, and insurance plan type.

Results: Over 10 years of follow-up, individuals with TBI experienced significantly higher risk of developing PTSD compared to those without TBI (adjusted HR 4.87, 95% CI: 4.07-5.84, $p<0.001$). Kaplan-Meier curves demonstrated sustained divergence between groups throughout the observation period (log-rank $p<0.001$). Additional factors independently associated with increased PTSD risk included female sex (HR 2.10, 95% CI: 2.00-2.20, $p<0.001$), age groups 35-54 years, residence in the North Central region, and comprehensive insurance plans.

Conclusions: TBI is strongly associated with nearly five-fold increased risk of PTSD among commercially insured individuals, with this elevated risk persisting throughout long-term follow-up. These findings underscore the critical need for systematic PTSD screening, longitudinal monitoring, and early mental health intervention among individuals with TBI history.

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Posterboard #: 64

Presenter: Rachel Westerbeck, BS
Institution: UT Health San Antonio
Category: Student

Title: α-Synuclein Overexpression Drives Brain Renin-Angiotensin System Upregulation and Micturition Circuitry Dysregulation in a Parkinson's Disease Mouse Model

Rachel Rebekah Westerbeck, Ada C. Felix-Ortiz, K.C. Biju, and Robert A. Clark

Urinary dysfunction, including overactive bladder (OAB) and incontinence, is a common and debilitating non-motor symptom of Parkinson's disease (PD) that substantially impairs quality of life, disrupts sleep, and increases fall risk nearly six-fold. Despite its high prevalence (27-85%), the underlying neural mechanisms remain poorly understood, largely because of the lack of animal models that faithfully recapitulate these symptoms.

We discovered that transgenic mice overexpressing human wild-type α-synuclein (Line 61; α-Syn mice) exhibit urinary dysfunction closely mirroring clinical PD. Using highly absorbent TEK-Fresh bedding in home-cage assays, α-Syn mice displayed disorganized voiding patterns with absent urine clumps, unlike control littermates that consistently urinated in designated areas. Conscious cystometry in freely moving mice confirmed an OAB phenotype at 3 months of age-shortened intercontraction intervals, increased voiding frequency, and reduced voided volumes-without changes in maximum voiding pressure or post-void residual volume. By 8-11 months, this progressed to elevated post-void residual volumes, decreased voiding efficiency (~70% to 20%), dribbling incontinence, and bladder contractions of varying amplitude, recapitulating the evolution of urinary dysfunction in PD patients.

Molecular analyses revealed upregulation of central renin-angiotensin system (RAS) components in the prefrontal cortex, including angiotensin-converting enzyme (ACE) transcripts, angiotensin II type 1 (AT1) receptor mRNA and protein, and downstream NADPH oxidase (NOX1 and NOX4) isoforms. These findings support our central hypothesis: α-synuclein overexpression downregulates dopamine release, triggering compensatory RAS overactivation through a counter-regulatory mechanism. This leads to AT1 receptor upregulation, which inhibits GABAergic tone via the Gq/PLC/PKC/NOX/reactive oxygen species pathway, thereby disinhibiting the micturition reflex and producing detrusor overactivity.

Our current goal is to elucidate the precise dysfunctional nodes within the micturition circuitry, map AT1 receptor expression patterns across disease stages, identify the anatomical substrates of α-synuclein pathology responsible for OAB, define central-to-peripheral propagation to the bladder and peripheral ganglia, and uncover molecular signatures via single-nucleus RNA sequencing (snRNA-seq) and spatial transcriptomics. These mechanistic insights will fill a critical knowledge gap and identify novel therapeutic targets for PD-related urinary dysfunction.

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Posterboard #: 65

Presenter: Olivia Yang, BS
Institution: UT Health San Antonio
Category: Student

Neural Circuit Mechanisms and Therapeutic Targets in PTSD with Comorbid Psychosis

Olivia Yang BS, Hannah Elam PhD, Angela Boley PhD, Stephanie Perez PhD, Daniel Lodge PhD

Exposure to trauma is extremely common, yet its psychiatric consequences vary widely across individuals. While many people recover, approximately 20% develop post-traumatic stress disorder (PTSD), and a substantial subset of these patients also experience psychotic symptoms, including hallucinations and delusions. The presence of psychosis in PTSD is associated with more severe functional impairment and diminished treatment responsiveness. Despite the clinical implications, the neurobiological substrates that link trauma exposure to the emergence of psychosis remain insufficiently characterized, limiting the development of therapeutics. Dysregulation of the dopamine system is a central feature of psychosis and is highly sensitive to stress. Specifically, stress increases the number of spontaneously active dopamine neurons in the ventral tegmental area (VTA), a change that amplifies dopamine system responsivity, promoting pathological salience processing. Understanding how stress recruits specific neural circuits to produce persistent dopamine dysfunction is critical for identifying novel intervention strategies. Our work has identified a circuit involving the insular cortex (IC) and orbitofrontal cortex (OFC) as a key regulator of stress-induced dopamine system activation. Building on this circuit-level framework, we investigated the therapeutic potential of targeting orexin signaling, a neuromodulatory system strongly engaged during stress and arousal. We previously found that systemic administration of the dual orexin receptor antagonist suvorexant restores normal dopamine neuron activity following stress. In the present study, we demonstrate that this effect is mediated locally within the OFC, as direct intra-OFC administration of suvorexant reverses stress-induced increases in dopamine neuron population activity. Furthermore, electrophysiological recordings and fiber photometry reveal that suvorexant suppresses insular cortex-driven excitatory signaling in the OFC, identifying a specific synaptic mechanism through which orexin antagonism stabilizes dopamine system function. These findings establish a mechanistic link between stress, cortical circuit dysfunction, and dopamine system dysregulation and identify orexin receptors within the IC-OFC circuit as a promising therapeutic target. By defining both the circuit and molecular substrates underlying stress-induced dopamine abnormalities, this work provides a translational foundation for developing more precise, circuit-informed treatments for PTSD with comorbid psychosis.

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Posterboard #: 66

Presenter: Gheed Zaki, BS
Institution: UT Health San Antonio
Category: Student

Barriers to representation of Arab Americans in dementia research: A mixed methods study

Zaki G., Charaf A., Sangdahl C., Werfelli H., Sowan A., Husain S., Mavrakis N., Samreen S., Ismail I., Khattab I., Masoud S.S.

Background: Arab Americans have unique health experiences related to their distinct cultural, historical, and political backgrounds. Despite global evidence demonstrating elevated risk for dementia, scientific evidence demonstrating the impact of dementia among Arab Americans is limited. A participatory research study (2024-2025) aimed to understand Arab American attitudes toward research and identify barriers to their involvement in dementia studies.

Methods: An explanatory sequential mixed-methods design was used. In the quantitative phase of the study, a survey was co-developed by a Community Council and the study team. The survey examined experiences and perceptions of dementia and clinical dementia research among Arab Americans. The survey included a modified version of the Research Attitudes Questionnaire (RAQ-7), which measures attitudes toward medical research on a 1-5 scale (1 = strongly disagree, 5 = strongly agree). Upon initial analysis of survey data, focus groups were conducted to deepen interpretation of the survey findings. All data was then integrated together by the study team and community partners for final analysis.

Results: N = 52 participants responded to the survey. Most were current or former family caregivers for a person living with dementia (n = 32, 61.5%) and 9 participants (17.3%) had self-reported dementia or memory loss. Survey respondents reported highly favorable attitudes toward clinical dementia research overall (M = 4.12, SD = 0.55), though only 9.6% (n = 5) had participated in any research prior to this study. Focus groups (N = 12) revealed that while participants expressed genuine valuation of research, substantial structural and contextual barriers prevented participation, such as limited perceived relevance of research to community needs; the effects of ongoing geopolitical displacement and war; lack of culturally and linguistically appropriate research opportunities; and logistical barriers (e.g., access and transportation).

Conclusion: Arab Americans have positive attitudes toward dementia research but do not participate in dementia-related studies. Findings demonstrate that barriers to representation among this population are best explained by structural and contextual barriers such as linguistic, cultural, and sociopolitical factors. Translational science frameworks emphasize the need for relationship-centered approaches to meaningfully overcome these challenges and make dementia research more accessible to highly impacted communities.