U54 CARE Partnership

2025 PSC POSTER SESSION PROGRAM

March 3, 2025

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March 3, 2025
11:40 AM - 2:30 PM

POSTER 1

Presenters: Nancy Sanchez (CoH)

Food Deserts and Insulin Resistance: Clinical Trial Recruitment Challenges During COVID-19

Nancy Sanchez 1, Jerneja Tomsic 1, Christina Vidal 1, Cristal Resto 1, Angelica Sanchez 1, Marley Jacobo 1, Veronica Jones 1, Joanne Mortimer 1, Lisa Yee 1, Lesley Taylor 1, James Waisman 1, Victoria Seewald 1

A food desert is a geographical area that lacks grocery stores that sell healthy and affordable food. Young women-of-color (African American, Latina/Hispanic American) are more likely to live in food-deserts than European and Asian-American. Consequently, living in or in close proximity to a food desert limits the amount of local healthy food choices available, which in turn sets these individuals at an increased risk for type-2 diabetes and obesity. . Obesity is a significant risk factor for TNBC and insulin promotes many signaling pathways that define the aggressive biology of TNBC. In this study we aim to test if insulin-resistance promotes epigenetic damage and increases TNBC risk in young women of color living in Los Angeles. This study faced some recruitment challenges during the COVID pandemic period. Here we aim to highlight some of the recruitment strategies that allowed us to continue this study while following the COVID guidelines and keeping our patients safe.

POSTER 2

Presenters: Giulia Alboreggia (UCR)

Targeted Degradation of Pin1 by Protein Destabilizing Compounds

Giulia Alboreggia, PhD

The concept of targeted protein degradation (TPD) is at the forefront of modern drug discovery, which aims to eliminate disease-causing proteins using specific molecules. In this paper, we explored the idea to design protein degraders based on the section of ligands that cause protein destabilization, hence that facilitate the cellular breakdown of the target. Our studies present covalent agents targeting Pin1, a cis-trans prolyl isomerase that plays a crucial role in tumorigenesis. Our design strategy entailed iterative optimizations of agents for potency and Pin1 destabilization in vitro. Biophysical and cellular studies suggest that the agents act like molecular crowbars, displacing protein stabilizing interactions that open the structure for recognition by the ubiquitin-proteasome degradation machinery. This approach resulted in a series of potent and effective Pin1 degraders with potential applications in target validation and in therapeutic development. We propose that our design strategy can identify molecular degraders without engineering bifunctional agents that artificially create interactions between a disease-causing protein and a ubiquitin ligase.

Increasing Latino Participation in Research: Feasibility of the Promotora Model

Mayra Serrano, DrPH, MPH, CHES

Background: Recruiting ethnic minorities, especially Latinos, into clinical and cancer trials is challenging. Latinos often do not achieve proportional representation, comprising only 1–5% of clinical trial participants and under 3% in cancer trials. Culturally tailored programs for encouraging Latino participation are rare. The promotora model has been effective in cancer prevention among Latinos but hasn't been tested for recruiting them into therapeutic cancer trials.
Aim: This study examines the feasibility of using the promotora model to enhance Latino participation in clinical trials at a comprehensive cancer center and explores the facilitators and barriers to this participation.
Methods: A sequential explanatory mixed-methods design combined quantitative and qualitative approaches to evaluate the promotora model's effectiveness in recruiting Latinos. Promotoras engaged Latino patients in two cancer trials. Semi-structured key informant interviews (KII) assessed the model's feasibility and acceptability.
Results: Sixteen patients were approached; 10 (63%) agreed, two (12%) refused, and four (25%) passively refused. Fifteen KII were conducted across four groups: 1) participants (n=4), 2) promotoras (n=3), 3) clinicians/researchers (n=3), and 4) study staff (n=5). Barriers to participation in trials included language, mistrust, lack of awareness, and fear. Facilitators included altruism and language/racial/cultural concordance. Strengths of the process noted were well-trained promotoras, their credibility, a streamlined process, and reduced staff workload. Limitations involved communication gaps, delays between recruitment and enrollment, and CITI certification challenges.
Conclusion: Findings suggest the promotora model is a feasible, acceptable, and effective method for recruiting Latino patients into therapeutic clinical trials at a cancer center. It efficiently addresses barriers and facilitators affecting Latino participation in cancer trials. These findings can guide future efforts to enhance Latino involvement in clinical trials in a culturally appropriate and impactful manner.

Immune, Tumor, and Stromal Interactions in Colorectal Cancer: A Spatial Proteomics Perspective

Maria Ninova, Ph.D. (UCR), Enrique Velazquez-Villarreal, M.D., Ph.D., M.P.H. (COH)

Introduction: Colorectal cancer (CRC) is the second leading cause of cancer-related deaths worldwide, claiming over 900,000 lives annually. In the United States, CRC disproportionately affects Hispanic/Latino populations, who experience higher incidence rates, inequitable access to healthcare, and worse clinical outcomes compared to other groups. The tumor microenvironment (TME) plays a critical role in CRC progression, therapeutic response, and overall patient prognosis. However, the complex spatial interactions within the TME that determine effective versus ineffective tumor control remain poorly understood. Addressing these gaps is crucial for enhancing our understanding of spatial heterogeneity and advancing immunotherapy, particularly in underserved populations such as Hispanics/Latinos. Methodology: To explore these dynamics, we employed a multimodal approach using co-detection by indexing (CODEX) technology on publicly available data from 35 late-stage CRC patients. Our systematic workflow classified cells, analyzed protein expression, and spatially visualized the data. Cells were categorized into three primary classes—stroma, tumor, and immune—based on structural features and biological functions. Protein expression analysis was conducted by integrating individual surface protein expression data, enabling direct comparisons across classes. Through this framework, we profiled 56 protein markers to identify conserved cellular neighborhoods (CNs) that define the spatial and functional organization of the CRC TME. Results: Protein expression analysis revealed distinct patterns across stroma, tumor, and immune classes for the biomarkers assessed. These markers exhibited differential expression profiles, highlighting their unique roles within the TME. For example, immune markers such as CD3, CD8, CD68, and PD-1 demonstrated significant variation between the tumor and immune classes, emphasizing their spatial organization and functional importance. Similarly, functional markers such as MMP9 and Ki-67 showed unique expression dynamics, reflecting their roles in tumor progression and immune responses. Auxiliary markers like cytokeratins underscored structural differences specific to the stromal class. These findings provided a comprehensive understanding of the spatial and functional interplay between immune, tumor, and stromal components in the CRC TME. Conclusion: This study provides a deeper understanding of protein expression within the colorectal cancer tumor microenvironment (CRC TME), enhancing the classification and spatial characterization of tumor ecosystems. By uncovering distinct protein expression profiles, these findings highlight the spatial heterogeneity of the TME, offering valuable insights for future drug discovery and therapeutic development. Furthermore, this research holds significant promise for advancing precision oncology. Notably, future studies in spatial biology that focus on the unique challenges and health disparities faced by Hispanic/Latino populations can pave the way for more equitable and effective cancer care, particularly in underserved communities.

POSTER 5

Presenters: Jeffrey Pino, PhD (UCR), Yifan Zhao (UCR)

MYC acetylated lysine residues drive oncogenic cell transformation and regulate select genetic programs for cell adhesion-independent growth and survival

Jeffrey Pino, PhD, Yifan Zhao

The MYC oncogene is often amplified in aggressive therapy-resistant breast cancers and overexpression of the MYC oncoprotein is associated with worst patient outcomes. MYC gene amplification and MYC pathway activation are more prevalent in women of African ancestry and contributes to the increased tumor aggressiveness and breast cancer mortality in African American women. Here we study MYC signaling pathways with the goal to uncover possible molecular targets for treatment of therapy-resistant breast cancers. We investigate a novel MYC acetylation pathway and its importance for the oncogenic activities of MYC during transformation of human mammary epithelial cells (HMECs, MCF10A) and in breast cancer cells. The MYC oncoprotein is a transcription factor that is acetylated at specific lysine residues (K149, K158, K323) by histone acetyltransferases (P300, GCN5), which regulate MYC stability and turnover through the ubiquitin-proteasome system. However, the role of MYC acetylation in activation of gene transcription and cell transformation, remains unclear. Additionally, it is uncertain whether deregulated MYC acetylation is implicated in cancer progression. In this study, we provide evidence suggesting that MYC acetylation enhances transcription by promoting MYC binding to the promoters of specific cancer-associated target genes (e.g., SNAI1, TERT) which are dependent on all three acetylated lysine sites. The MYC acetyl-lysine (AcK) cofactors and molecular mechanism involved are still being investigated. Our findings indicate that endogenous MYC is acetylated at these three sites in non-malignant cells, while oncogenic MYC overexpression enhances acetylation and alters the regulation of acetylation by proteasome and deacetylase inhibitors in triple-negative breast cancer (TNBC) cells. Our studies will establish the key role of MYC acetylation in breast cancer initiation and progression. The identification of MYC AcK-dependent target genes and coactivators may offer new avenues for selective therapeutic targeting of MYC oncogenic activities in most aggressive breast cancers for which no current targeted therapies exist.

POSTER 6

Presenters: Erik Morales (UCR)

Enhancing Equitable Implementation of Oncology Patient Navigation at an NCI Comprehensive Cancer Center

Erik Morales1, Ana Tergas2, Lauren Cai2, Yongzhe Wang2, Marianne Razavi2, Annette Mercurio2, William Dale2, and Narissa Nonzee2

Background: Patient navigation is an evidence-based intervention, but implementation varies across healthcare systems. As one of 20 hospital systems nationwide supported by the American Cancer Society Patient Navigation Capacity-Building Initiative, our program sought to improve equitable delivery of oncology navigation services. To inform implementation strategy enhancements, we assessed baseline patient navigator reach and process outcomes, including barriers identified and navigation duration.

Methods: We conducted a retrospective analysis of electronic health record data between January 2020 to May 2023 at City of Hope, an NCI comprehensive cancer center. Our sample included adult patients with any cancer who received oncology navigation services. We assessed demographic characteristics and navigation process outcomes, including multilevel barriers (defined as any patient reported or navigator perceived barrier) and navigation duration (defined as cumulative time spent on hospital orientation, barrier assessment, and referrals/intervention).

Findings: Our sample (N=5008) was ethnically diverse (28% Hispanic, 14% Asian, 5% Black, 51% White); 16% spoke a primary language other than English. Approximately 50% were female, 41% age 65+, 52% Medicare, 10% Medicaid. Our navigation program reached patients with hematologic malignancies (44%), followed by cancers of the GI/colon (15%), breast (12%), and prostate (6%). Total navigation duration was on average 26 minutes (SD=18) per patient. Overall, the most frequently identified barriers included fear and anxiety (18%), communication with providers and staff (17%), support service needs (social worker, chaplain) (16%), and language (10%), among other diverse needs such as systems/scheduling and financial barriers. In patients who reported at least one barrier (N=3007), the average count was 1.65 barriers per patient (SD=1.10; range 1-11). Subgroup analyses revealed variation by language. Primarily Korean-speaking individuals showed the highest rates of fear and anxiety (56% vs. 18% English) and language barriers (81% vs. 45-53% among Spanish, Chinese, Vietnamese, and Armenian). The highest rates of provider/staff communication barriers were observed among primarily Chinese-speakers (41% vs. 13% English).

Implications for D&I Research: Findings help to inform navigation implementation strategy enhancements, including infrastructure and protocols to expand reach; account for time spent on navigation; and adequately address potential unmet needs of patients with limited English proficiency and other vulnerable populations.

POSTER 7

Presenters: Xiaoxu Zhang (COH)

Pharmacological inhibition of eIF4A1 suppresses leukemogenesis via specifically rewiring amino acid biosynthesis

Xiaoxu Zhang1,2, Honghai Zhang1,2, Lili Ren1,2, Lei Dong1, Xueer Wang1,2, Hongjie Bi1,2, Alexandra Huang3,4, Seán O'Leary 3,4 , Rui Su1,2

Acute myeloid leukemia (AML) is an aggressive hematological malignancy with a dismal five-year survival rate below 30%. Dysregulation of mRNA translation is a hallmark and a driver of tumorigenesis, including leukemogenesis. However, the precise contributions of translation factors to AML pathogenesis and their potential as therapeutic targets remain poorly understood.

Through comprehensive multi-omics analysis, we identified eukaryotic translation initiation factor 4A1 (eIF4A1) as one of the most highly expressed translation factors in AML. Notably, eIF4A1 expression was significantly elevated in AML cells and patient samples compared to healthy controls. As a DEAD-box ATPase-dependent helicase within eIF4F complex, eIF4A1 facilitates the unwinding of secondary structure of mRNA 5'UTR, thereby promoting translation initiation.

CRISPR/Cas9-mediated knockout (KO) of endogenous eIF4A1 in AML cell lines and patient-derived xenograft (PDX) cells revealed that eIF4A1 KO dramatically inhibited AML cell proliferation, suppressed mitochondrial respiration, and reduced global translation intensity. Integrative RNA-seq and proteomics analyses, coupled with experimental validation showed phosphoglycerate dehydrogenase (PHGDH) as a functionally essential target of eIF4A1, highlighting its crucial role in reprogramming AML metabolism. While eIF4A1 is traditionally studied within the eIF4F complex, our other findings uncover a novel eIF4F complex-independent mechanism. Specifically, BioID assays demonstrated a physiologically relevant and robust interaction between free eIF4A1 and Y-box binding protein 1 (YBX1). YBX1 is known to stabilize mRNAs via recognizing 5-Methylcytosine (m5C) modification. The eIF4A1/YBX1 interaction may not only enhance target mRNA stability but also contribute to defining the specificity of eIF4A1 downstream targets.

Importantly, Zotatifin, an FDA-approved eIF4A1 inhibitor, significantly repressed AML cell growth in vitro, with IC50 values in the low nanomolar range, and substantially prolonged the survival of AML mouse models in vivo.

Overall, our findings establish eIF4A1 as a key regulator of AML pathogenesis and metabolism. Targeting eIF4A1, particularly with Zotatifin, represents a promising therapeutic strategy for AML by specifically disrupting amino acid biosynthesis.

POSTER 8

Presenters: Christina Vidal, PhD (COH)

Insulin Resistance in Women Correlates with Chromatin Histone Lysine Acetylation, Inflammatory Signaling, and Accelerated Aging

Christina M. Vidal, Alva-Ornelas JA, Chen NZ, Senapati P, Tomsic J, Robles VM, Resto C, Sanchez N, Sanchez A, Hyslop T, Emwas N, Aljaber D, Bachelder N, Martinez E, Ann D, Jones V, Winn RA, Miele L, Ochoa AC, Dietze EC, Natarajan R, Schones D, Seewaldt V

Epigenetic changes link medical, social, and environmental factors with chronic medical diseases such as cardiovascular disease, diabetic kidney disease, and more recently with cancer. The links between these factors, particularly the link between metabolic health and epigenetic changes, are only starting to be investigated. In our in vitro and in vivo studies, we performed broad analysis of the link between hyperinsulinemia and chromatin acetylation; our top "hit" was chromatin opening at H3K9ac. Building on our published pre-clinical studies, here we performed detailed analysis of the link between insulin-resistance, chromatin acetylation, and inflammation in an initial test set of 28 women and validation sets of 245, 22, and 53 women. ChIP-seq identified chromatin acetylation and opening at the genes coding for TNFa and IL6 in insulin-resistant women. Pathway analysis identified inflammatory response genes, NFkB/TNFa-signaling, reactome cytokine signaling, innate immunity, and senescence. Consistent with this finding, flow cytometry identified increased senescent circulating peripheral T-cells. DNA methylation analysis identified evidence of accelerated aging in insulin-resistant vs. metabolically healthy women. Conclusions: This study shows that insulin-resistant women have increased chromatin acetylation/opening at inflammatory genes, inflammation, and, perhaps, accelerated aging. Given the role that inflammation plays in cancer initiation and progression, these studies provide a potential mechanistic link between insulinresistance and cancer.

Presenters: Kendrick Davis, Margarita Monge & Angela Reyes (UCR)

Clinical Trials Readiness: Map, Measure, & Match Model

Kendrick Davis1, PhD., Margarita Monge1, BA., Angela Reyes, BS1, Victoria Seewaldt, MD

Clinical trials represent research that can improve the quality of health through innovative treatments and interventions. However, substantive research indicates that clinical trials are often inefficient and lack the inclusion of subpopulations. The need for subpopulations to be included in clinical trials underscores the fact that the benefits of advances in treatments, drug developments, and various interventions rarely, if ever, reach communities rich with subpopulations. We propose a mapping, measuring, and matching model as a solution in addressing the problems stemming from lack of efficiency and representation in clinical trials.
A framework for assessing clinical site readiness for participation in clinical trials has been effectively advanced by Buse et al. (2022) and consists of six domains to assess readiness; research team, infrastructure, study management, data collection and management, quality oversight, and ethics and safety. This framework is well suited for addressing issues stemming from inefficiencies in clinical trials. However, this framework also leaves open considerations of greater efficiency in triangulating site readiness with site interest in phase and type of clinical trial. In addition, patient interest and readiness for participating in clinical trials, and availability of various phases and types of clinical trials for sites and populations are also coordinated.
The coordination of people, services and resources within communities is central to many issues surrounding quality improvement needs in healthcare and the benefits clinical trials can bring to underserved communities. The initial stage of our model consists in mapping clinics in targeted regions rich with subpopulations.
We then measure site and patient readiness for participation in the various phases and types of clinical trials available through our research centers. Our readiness instrument consists in developing and administering items gleaned from six domains of clinical trials readiness advanced by Buse et al. (2022). We measure readiness for all phases of clinical trials for treatments and interventions non-therapeutic and therapeutic.
To ensure a more representative sample of patients are included in our trials two strategies are employed. We use GIS to prioritize and target working with clinics in regions rich in representative samples. We also use emoji as our primary measurement tool with patient populations.
Once we have mapped clinical sites that are both interested and ready to participate in clinical trials and identified which phases and types of clinical trials they are interested in and ready for, we then begin the matching process. The phases and types of clinical trials research (CTR) are matched with clinics and clinical teams that are interested and ready to participate in aligned CTRs from our participating City of Hope CTR center.
Our team provides ongoing monitoring of progress and completion of trials, any attrition, any study deviations or incompletions. We also continue processing and analyzing data around reasons why clinics that are interested and ready to participate in clinical trials do not participate in clinical trials.

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POSTER 10

Presenters: Kendrick Davis, Margarita Monge & Angela Reyes (UCR)

EMOJI: MEASURES OF EMOTIONS & MOODS

Kendrick Davis1, PhD., Margarita Monge1, BA., Angela Reyes, BS1

Background / Context:
The History of Emoji
1999 - The first set of emoji was released - created by their Japanese originator Shigetaka Kurita (). Emoji is a transliteration of the Japanese word (e=picture) 文 (mo=write) 字 (ji=character)[8].
2010 - Emoji were added to Unicode
2011 - Frequent use in communication (Added emoji keyboard to iOS devices and android followed)
1998 - Introduced in research measurement and instrumentation

Problem statement: While emojis are increasingly utilized in research across various industries to measure emotions and moods, existing tools fail to effectively differentiate between these two distinct psychological states. This limitation hinders the accuracy and depth of emotional analysis, which is crucial for developing more precise and contextually relevant insights. Addressing this gap is essential for improving the utility of emojis as valid and reliable indicators of emotions and moods.
Response: The Emoji Psychometrics Lab is addressing the challenge of differentiating between emotions and mood states in emoji-based measurement & instrumentation research. Cancer research is lacking in the measurement of emotions and moods and their role in treatment and adherence behaviors of participants. Our studies aim to identify key distinctions between these psychological states and determine the most appropriate contexts and methodologies for capturing each one. By refining the use of emojis as measurement tools, we seek to enhance the accuracy and consistency of emotional data.

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POSTER 11

Presenters: Narissa Nonzee (COH)

Implementation of Medicaid expansion for patients with cancer: Community perspectives on the California Cancer Care Equity Act

Narissa J. Nonzee*, Brenda Gascon, Genesis Sandoval, Sophia Yeung, Lauren Cai, Kimlin Tam Ashing

Background: Underinsured cancer patients are at risk for late-stage diagnosis and poor survival. In January 2023, the California Cancer Care Equity Act (SB987; hereon CCEA-SB987), an evidence-based cancer control Medicaid expansion, was implemented to increase access to specialized cancer centers for Medicaid patients with complex cancers. We present preliminary findings on community health system leader perspectives on CCEA-SB987 awareness and multilevel determinants of implementation. Methods: Between March and July 2024, we conducted semi-structured interviews with organizational leaders at community health centers and hospitals serving large Medicaid-insured populations in LA County. Guided by the Consolidated Framework for Implementation Research (CFIR), interviews explored inner and outer setting determinants of cancer care access and policy awareness, implementation, and dissemination, and themes were summarized within CFIR. Data collection is ongoing with additional interviews being conducted. Findings: Participants (n=12) comprised 5 clinician leaders (e.g., medical directors) and 7 organizational leaders (e.g., QI, case management); average years of experience=6 (sd 5); 9 female; 9 ethnic minority (7 Latino, 2 Asian). Overall, participants expressed low CCEA-SB987 awareness. Key barriers to cancer care access emerged within the outer setting, including financing and provider network partnerships/connections (e.g., insurance authorization delays; complexity navigating managed care plans). Within the inner setting, participants frequently discussed limited access to knowledge about CCEA-SB9897 and importance of relational connections to reduce fragmented communication between primary care and specialty care providers. At the individual level, transportation and language barriers were commonly cited. Regarding processes, the majority described a top-down approach to successful implementation of coverage expansion policies, starting with senior leadership, then clinicians and staff. Almost all perceived that CCEA-SB987 patient eligibility would be best determined by clinicians, in coordination with referrals and benefits enrollment. Suggestions for CCEA-SB987 policy dissemination included communication from health system leadership, provider and IPA meetings, brief bulletins, and in-language and low-literacy patient education. Implications for D&I Research: One year after the law began, CCEA-SB987 awareness remained low and challenges to cancer care for Medicaid managed care beneficiaries persisted. Policy dissemination should involve multi-stakeholder engagement and leverage community health system leadership to strengthen processes around CCEA-SB987 eligibility determination and communication to ensure equitable cancer care access.

Presenters: Angel Perez-Hunt (COH)

Genetic Insights into Health Disparities; Understanding Ancestral Contributions to Racial Health Disparities in LUAD

Angel Perez-Hunt

Lung cancer is the second most diagnosed cancer in the U.S. and the leading cause of cancer-related deaths in both men and women. Throughout their lifetime, 1 in 17 men and 1 in 18 women will be diagnosed. Lung adenocarcinoma (LUAD), the most common subtype of non-small cell lung cancer (NSCLC), exhibits significant histological, mutational, and epigenomic heterogeneity. However, racial and ethnic health disparities in LUAD incidence and clinical outcomes remain understudied. Characterizing these patterns in US minority demographics is essential for informing targeted public health and clinical strategies in high-risk populations. LUAD occurs at a rate of 68.3 per 100,000 in Black men and 61.5 per 100,000 in White men, reflecting an 11% higher incidence in Black men. Moreover, Black men and women face disproportionately higher mortality rates and poorer prognoses compared to their White counterparts, underscoring significant racial health disparities in LUAD outcomes. Understanding the genetic and molecular factors driving racial health disparities is essential for developing more precise therapies and targeted interventions.
Oncogenic transformation can be influenced by single nucleotide polymorphisms (SNPs), contributing to distinct molecular landscapes in tumors and the surrounding tumor microenvironment (TME). Ancestry may also shape susceptibility to mitochondrial dysfunction, influencing cancer risk and progression. To investigate the disparities in LUAD, we employed germline VCFs for ancestry analysis of 36 LUAD patients (18 Black and 18 White) from the ORIEN data set, while genotype data from the 1000 Genomes Project phase 3, grouped by super population served as a reference. Oncoplots were then generated using a combination of patients' somatic VCF and metadata from ORIEN. Regression analysis adjusting for age, staging, and gender was performed to determine association between ancestry and gene mutations.

Presenters: Benecia Jackson (COH)

Kinesin Family Member C1 (KIFC1/HSET) Underlies Aggressive Disease in Quadruple Negative Breast Cancers

Benecia Jackson 1, Yate-Ching Yuan 2, Padmashree Rida 3 Nikita Jinna1

Quadruple-negative breast cancer (QNBC) lacks traditional actionable targets, including androgen receptor (AR). QNBC disproportionately afflicts and impacts patients of African genetic ancestry. Kinesin family member C1 (KIFC1/HSET), a centrosome clustering protein that prevents cancer cells from undergoing centrosome-amplification-induced apoptosis, has been reported to be upregulated in TNBCs and African-American (AA) TNBCs. Herein, we analyzed KIFC1 RNA levels and their associations with clinical features and outcomes among AR-low and AR-high TNBC tumors in three distinct publicly available gene expression datasets and in the breast cancer gene expression database (bc-GenExMiner). We also knocked down AR in TNBC cells. KIFC1 levels were significantly higher in AR-low and basal-like TNBCs than in AR-high and non-basal-like TNBCs, irrespective of the stage, grade, tumor size, and lymph node status. KIFC1 levels were also upregulated in AR-low tumors relative to AR-high tumors among Black and premenopausal women with TNBC. High KIFC1 levels conferred significantly shorter overall survival, disease-free survival, and distant metastasis-free survival among AR-low and basal-like TNBC patients in Kaplan-Meier analyses. Knockdown of AR in TNBC cells upregulated KIFC1 as well as beta-catenin/TCF4-mediated signaling. In conclusion, KIFC1 levels may be upregulated in AR-low tumors and, specifically, in those of African descent, wherein it may promote poor outcomes. This upregulation may be caused by increased TCF4-mediated transcription of KIFC1. Thus, KIFC1 may be an actionable cancer-cell-specific target for the QNBC subpopulation and could aid in alleviating racial disparities in breast cancer outcomes.

POSTER 14

Presenters: Marley Jacobo (COH)

The Coachella Valley: Metformin (Dimethylbiguanide) and Insulin Resistance in Insulin-Resistant Women

Marley Jacobo1, Mayra Serrano2, Cristal Resto1, Victoria L. Seewaldt1, Kendrick Davis3, Rebecca Nelson4, Christina Vidal1

Background: Obesity and Type 2 diabetes remain two of the topmost preventable diseases in the United States. Standard of care for Type 2 diabetes includes metformin, a drug that improves metabolic health and lowers blood sugar levels. Since 2012, the primary treatment approach for prediabetes/insulin resistance promoted by the ADA has been lifestyle changes like diet and exercise, despite insulin resistance leading to an increased risk of developing Type 2 diabetes. Underserved communities are disproportionately affected by obesity and Type 2 diabetes, and barriers to access and resources prevent healthy living, increasing risk for type-2 diabetes. A 2013 community health needs assessment identified the Coachella Valley as an underserved community with increased risk for poor health and limited access to healthcare, accessible healthy grocery stores, and transportation. Community Based Intervention: Mi Salud, Mi Poder is a community-based nutrition and physical activity intervention modified from the Eat Move Live study optimized to fit the Coachella Valley by addressing the barriers of accessibility due to geological disparities, limited access to care, and culture. Study: We believe Mi Salud, Mi Poder will develop healthier lifestyles, enhance participants' knowledge about health and nutrition, and reduce the risk of obesity. We believe metformin combined with the Mi Salud, Mi Poder intervention, will have a synergistic effect in restoring metabolic health. This study aims to determine the impact of Mi Salud, Mi Poder along with metformin on the reduction of insulin resistance and the reversal of epigenetic damage and inflammation caused by insulin resistance.

A Nurse Led Model for Transition of Patients After Completion of Phase 1 Trials

Betty R. Ferrell1, PhD, RN, CHPN, FAAN, FPCN, Tami Borneman1, MSN, RN, CNS, FPCN, Finly Zachariah1, MD, FAAFP, FAAHPM, FAMIA, Virgina Sun1, PhD, RN, Nathaniel Co2, BS, Vincent Chung1, MD, FACP

Palliative care involves an interdisciplinary approach emphasizing quality of life (QOL) and supportive care to patients with significant morbidity and symptom burden. This has particular benefit and is often provided jointly with cancer therapy.

ASCO released updated guidelines in May 2024 on “Palliative Care for Patients with Cancer”1. As per a review of evidence and series of expert panel deliberations, the guidelines presented six recommendations. This included four recommendations reinforcing basic principles of referring oncology patients to interdisciplinary palliative care services and two recommendations related to specific oncology populations in need of enhanced attention for palliative care needs. These specific populations were those with hematologic malignancies and patients in early phase clinical trials. Underutilization of hospice and palliative care is something that should urgently be addressed in patients completing clinical trials, who are reported to have a median survival between 6-11 months1.

One of the challenges of the inpatient setting is a deficiency in advanced care planning, and in turn limited accessibility to palliative care consulting or goals of care discussions. A previous study notes this population faces high symptom burden, repeat urgent care visits and hospitalizations, limited use of palliative care or other support services, and limited and late referrals to hospice2.

Food Deserts and Insulin Resistance

Lindsey Ondieki1, Christina Vidal, PhD2, Victoria Seewaldt, M.D. 2

~Food Deserts: Areas with limited access to affordable, healthy food.
~Health impact: Increased risk of a poor-quality diet in food deserts negatively affects metabolic health.
~City of Hope: Serves LA county and surrounding diverse areas, each with variability in access to resources.
~Hyperinsulinemia: Increases mitochondrial activity and histone acetylation.
~Leads to tissue-specific inflammation via mTOR and NF-kB signaling, causing chronic inflammation.
~Metabolic Dysfunction: Linked to aging at organismal and molecular levels.
~Chronic insulin exposure (T2DM) causes cell senescence in liver and pancreas.

Study Aim: Explore the link between food deserts and insulin resistance, targeting early interventions to prevent complications like

Impact of Aromatase Inhibitors on Joint Arthropathy in Postmenopausal Breast Cancer Patients

Yash A. Mehta1, B.S., Armine Kasabyan2, B.S., Sreyanth Parupalli3, Kevin Herrera4, B.S., Jennifer Lopez3, M.S., Keilani Luna3, M.S., Shiuan Chen3, Ph.D., Lisa D. Yee3, M.D.

Aromatase inhibitor (AI) therapy has significantly improved breast cancer survival rates but up to 50% of patients experience severe treatment-related arthralgia, impacting quality of life and adherence to therapy1-3. Despite studies evaluating interventions like duloxetine, acupuncture, exercise, and vitamin D, no clear treatment for AI-induced arthropathy (AIIA) has been identified4. The exact mechanisms underlying AIIA remain unclear, though it appears related to hormonal shifts rather than estrogen deprivation alone5,6. The novel AroER Tri-screen assay can assess bioavailable estrogen and androgen levels, potentially identifying women at risk for AIIA and aiding in the development of targeted symptom interventions

Impact of great circle distance on breast cancer survival in NCDB

Yongzhe Wang1 , Christine Quinones1 , Elizabeth Gonzalez 2 , Preeti Farmah 1 , Hans Schoellhammer1 , Lorena Gonzalez1 , Nikita Shah1 , Katharine Schulz-Costello1 , Jennifer Tseng1 , Veronica Jones1

While incidence rates of breast cancer continue to increase, breast cancer mortality rates have declined significantly. From 1989 to 2020, the overall death rate has dropped by 43%. 1 There remains, however, large racial and ethnic disparities in breast cancer mortality.

- Black women have a 41% higher death rate from breast cancer compared to white women, despite a 4% lower incidence rate.1
- Minority women—including NHB, Hispanic, and indigenous women—often present with a more advanced breast cancer at time of diagnosis when compared to NHW women, leading to higher mortality. 2

Investigating the factors that influence the timeliness of primary breast treatment can potentially help reduce these disparities. Timeliness of of breast cancer care is defined as less than 60 days between date of diagnosis to date of first treatment. Time beyond 60 days constitutes a treatment delay.6 Minority women are at risk for delays: one study found Black women and Hispanic women were associated with an additional 6-day delay in between diagnosis and treatment when compared to White patients and Non-Hispanic patients, respectively.9

Currently, scant research exists on how distance from patient residence to cancer treatment facility affects timeliness of breast cancer care (BC). Distance from the treatment facility may pose a significant delay to initiation of treatment and should be evaluated in relation to BC survival. We hypothesized that larger great circle distance (GCD), or distance from patient residence to treatment facility, leads to delayed timeliness of BC care and negatively impacts survival.