Intrapersonal Barriers and Facilitators to Cervical Cancer Screening of Women with HIV in Surat, India

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**BACKGROUND**
- Women who have HIV have an increased risk of being infected with HPV, HPV is often the established cause of Cervical Cancer (CC).
- HIV positive women have an increased prevalence rate of HPV and are at a higher risk of being diagnosed with invasive CC.
- It is recommended that HIV positive women be screened for CC every 6 months starting within 1 year of diagnosis of HIV and yearly afterward.
- However, currently, less than 3% of Indian women receive CC screening.

**SPECIFIC AIMS**
- Evaluate the intrapersonal barriers and facilitators in receiving Cervical Cancer prevention, screening, and treatment for HIV positive women living in Surat, India.
- Create an intervention plan to help improve access and adherence to Cervical Cancer prevention, screening, and treatment services.

**METHODS**
- An Intervention Mapping approach, guided by the Social Ecological Model (SEM).
- In-depth interviews with 25 women living with HIV and 15 stakeholders at the New Civil Hospital ART Center in Surat or at a private location specified by the participant.
- Interviews lasted approximately 35 minutes and will follow a semi-structured interview guide.
- Interviews were then audiotaped, transcribed and translated into English.
- Once translated the interviews were imported into NVivo, a qualitative software. When entered into NVivo the data was analyzed for key/emerging themes and for any intrapersonal barriers and facilitators.

**RESULTS**

<table>
<thead>
<tr>
<th>Results from Interviews</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wants to learn more about Pap Smears</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Believe they need test or follow-up care</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Afraid of receiving Pap Smears</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Knowledgeable about Pap Smears &amp; CC</td>
<td></td>
<td></td>
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<tr>
<td>Comfortable with male Doctors</td>
<td></td>
<td></td>
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<tr>
<td>Responsibilities suffer for a Doctors appointment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Can afford travel expenses related to screening</td>
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</tbody>
</table>

**CONCLUSIONS**
- To improve Cervical Cancer prevention, screening and treatment outcomes factors from the intrapersonal level can be addressed.
- Continued research will be conducted in order to develop an intervention program that will utilize these research findings.

**REFERENCES**

For more information and references please contact Asha Abdulahi at: aabulahi@sdstate.edu

Research reported in this poster was supported by the National Cancer Institute of the National Institutes of Health under award numbers U54CA132384 & U54CA132379
The immune system plays an important role in defending the body of the host and protecting against foreign invaders such as cancer. This system is comprised of both innate and adaptive immunity. Antigen presenting cells (APCs) such as dendritic cells (DCs) and macrophages (Macs) are mostly members of the innate immune system. APCs engulf antigens in a process called phagocytosis. Antigens are processed, bound to the major histocompatibility complex/class II (MHC II) within the endosome of APCs, and transported to the cell surface as part of the APC maturation process. Once APCs mature, antigen presentation occurs and cytokine release is observed.

In this experiment, we exposed Macs (RAW cells) to media preconditioned by colon cancer cells (CT26) treated by heat, mechanical disruption, and starvation to release cancer antigens and determine if these treatment strategies induce inflammatory cytokine synthesis on maturing Macs.

The specific aims of this study are:
1. To determine cell death levels of CT26 cells after heat shock, mechanical disruption, and starvation at 6 hours and 24 hours.
2. To measure and analyze cytokine transcript levels from RAW cells treated with CT26 media from aim 1.
3. To establish a baseline cytokine evaluation that will later be used to compare TRl7 conjugated nanoshell treatment to free TRl7 in cancer antigen matured Macs.
4. To detect the effects each treatment has on Macs maturation, the aims of this study are:

- **Cell death**: Heat shock treatment induced highest cell death in CT26 cells (data not shown).
- **IL-6**: All treatments stimulate transcription of IL-6 over by 24 hr.
- **IP-10**: CT26 treated supernatants induce a slight increase of IP-10 transcription in RAW cells at 6 hr but not at 24 hr.
- **MCP-1**: MCP-1 expression is not significantly induced by any treatments at 6 hr or 24 hr.
- **MIP-1α**: MIP-1α expression is not significantly induced by any treatments at 6 hr or 24 hr.
- **TNF-α**: TNF-α expression is not significantly induced by any treatments at 6 hr or 24 hr.

It might be beneficial to test target cytokine mRNA expression after two hours, twelve hours, eighteen hours, and thirty-four hours in order to establish a more detailed time course. More time points would demonstrate the type of effects the CT26 media has on macrophage maturation as measured by cytokine transcript levels.

The broader significance of this work is to contribute to the Parent Project 1. Once we know which treatment causes most cell death and antigen release and leads to highest cytokine transcript levels by the antigen-presenting cells (APCs), in this case macrophages, then we can have a baseline for the parent project.

**REFERENCES**

**ACKNOWLEDGMENTS**
I would like to thank Dr. Crowe, Dr. Bernstein, and Jennifer Sugg for giving me this opportunity. I would also like to thank Dr. Sussman and Dr. Natalie Gude for giving me the opportunity to work in their lab and Dr. Natalie for constantly checking up on my progress. Last but not least, I would like to extend my greatest gratitude to Joi Weeks for being just an amazing mentor and for always challenging me. I would like to show my deepest appreciation for Oscar Echeagaray and Nick Vallez for their tremendous assistance during this project.

**Funding**
Research reported in this poster was supported by the National Cancer Institute of the National Institutes of Health under award numbers: U54CA132384 & U54CA132379.
Assessing Fasting Plasma Glucose Change in Breast Cancer Survivors Participating in a Randomized Control Trial of Physical Activity

Max Fang¹, Sheri J. Hartman³, Dorothy Sears²,³
SDSU¹, UCSD Departments of Medicine² and Family Medicine and Public Health³

BACKGROUND

- Many breast cancer survivors experience problems with cognition that persist for years, especially in those who are physically inactive.
- Physical activity (PA) improves cognition in healthy and cognitively impaired adults, but the effect on cognition in cancer survivors is unknown.
- The Memory & Motion Study (parent study of this summer project) was a randomized controlled trial to examine the effects of a 3-month PA intervention on cognition in breast cancer survivors.
- The exercise intervention significantly improved processing speed, a component of cognition, in participants who had been diagnosed within 2 years of study enrollment.
- Secondary outcomes of Memory & Motion include plasma biomarkers related to PA and cognition, including brain-derived neurotrophic factor, insulin-like growth factor-1, insulin resistance (determined using insulin and glucose), and c-reactive protein.
- Hyperglycemia is associated with carcinogenesis and is a risk factor for invasive breast cancer recurrence and distant metastasis.

SPECIFIC AIM AND HYPOTHESIS

- Assess fasting plasma glucose change in breast cancer survivors participating in a randomized control trial of PA.
- We hypothesized that plasma biomarkers related to PA and cognition, including glucose, would be reduced in the physical activity group but not in the control group. Glucose was the focus of this summer project.

METHODS

- 87 participants were randomized to exercise or waitlist wellness control groups. The intervention period was 12 weeks.
- The PA intervention goal was engaging in ≥150 minutes per week of moderate-to-vigorous PA (MVPA) on at least 5 days per week.
- The YSI 2900 Biochemistry Analyzer, fasting glucose levels were measured in archival baseline and endpoint plasma samples from the Memory & Motion Study.

RESULTS

- Table 1. Baseline Characteristics by Group

<table>
<thead>
<tr>
<th></th>
<th>Exercise Intervention (n=43)</th>
<th>Wellness Control (n=44)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years, mean (SD)</td>
<td>58.2 (11.37)</td>
<td>56.2 (9.30)</td>
</tr>
<tr>
<td>Not Hispanic, n/%</td>
<td>35 / 81%</td>
<td>37 / 84%</td>
</tr>
<tr>
<td>White, n/%</td>
<td>36 /84%</td>
<td>35 / 80%</td>
</tr>
<tr>
<td>At least college degree, n/%</td>
<td>29 / 67%</td>
<td>33 / 75%</td>
</tr>
<tr>
<td>BMI, kg/m², mean (SD)</td>
<td>26.7 (6.20)</td>
<td>27.3 (6.40)</td>
</tr>
</tbody>
</table>

*Data from reference # 1

- Table 2. Breast Cancer Characteristics by Group

<table>
<thead>
<tr>
<th></th>
<th>Exercise Intervention (n=43)</th>
<th>Wellness Control (n=44)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time since dx, months, mean (SD)</td>
<td>30.3 (17.41)</td>
<td>30.0 (16.08)</td>
</tr>
<tr>
<td>Stage 1, n/%</td>
<td>27/63%</td>
<td>26/59%</td>
</tr>
<tr>
<td>Received chemotherapy, n/%</td>
<td>23/54%</td>
<td>23/52%</td>
</tr>
<tr>
<td>Current AI or tamoxifen, n/%</td>
<td>31/72%</td>
<td>30/68%</td>
</tr>
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</table>

*Data from reference # 1

- Figure 1. Change in MVPA

<table>
<thead>
<tr>
<th>Month/Week in MVPA</th>
<th>Exercise</th>
<th>Wellness Control</th>
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<tbody>
<tr>
<td>1 Week</td>
<td>42.7</td>
<td>34.3</td>
</tr>
<tr>
<td>12 Weeks</td>
<td>118.3</td>
<td>30.8</td>
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</table>

*Data from Reference # 1

- Figure 2. Average Fasting Plasma Glucose Levels (Baseline vs. Endpoint)

<table>
<thead>
<tr>
<th></th>
<th>Exercise</th>
<th>Wellness Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>60.0</td>
<td>65.0</td>
</tr>
<tr>
<td>Endpoint</td>
<td>67.5</td>
<td>70.0</td>
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</tbody>
</table>

REFERENCES


FOR MORE INFORMATION:
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ACKNOWLEDGEMENTS

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The Effects of Triptolide on the phenotype of pancreatic cancer cells.

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²Department of Surgery, Division of Surgical Oncology
Moore’s Cancer Center, University of California San Diego

BACKGROUND

Pancreatic ductal adenocarcinoma (PDAC) is one of the most lethal human malignancies. This may be attributed to several factors including late detection, the propensity for metastases, and profound resistance to therapies. Due to these factors, PDAC has a grim median survival of less than one year and an overall 5-year survival rate of less than 10% (Kleef et al. 2016).

Triptolide, a novel therapeutic derived from a Chinese herb, Tripterygium wilfordii, has demonstrated anti-proliferative activity in vitro and demonstrated tumor reduction in vivo displaying promising anti-cancer capabilities (Wang et al. 2014). The specific mechanism of action is still not understood, but it can affect cells in multiple ways such as reduction in NF-κB activity, iNOS and Cox-2 expressions, and decrease HSP70 expression. The objective of these studies was to understand mechanisms of resistance to Triptolide in pancreatic cancer cells using a murine model of pancreatic cancer (KPC) which harbors Kras and p53 mutations.

SPECIFIC AIMS

1. Determine how triptolide treatment modifies the phenotype of KPC cells with particular attention to:
   a. Examining any induction of epithelial-mesenchymal transition (EMT) by characterizing cell morphology as well as specific epithelial and mesenchymal markers.
   b. Evaluating stem cell marker expression in treated versus untreated populations.

METHODS

PDAC cells, derived from a genetically engineered mouse model (KPC), were initially treated with three different concentrations, 250nM, 500nM, and 1000nM over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000nm over a three day period. Resistant cells were then passaged in increasing concentrations, 250nm, 500nm, and 1000n...
Impact of mailed outreach on colorectal cancer screening: a systematic review and meta-analysis

Mark Jager, Balambal Bharti PhD, Karen Heskett, Siddharth Singh MD, Samir Gupta MD MSCS
University of California San Diego Moores Cancer Center

BACKGROUND
- Colorectal cancer (CRC) is the 2nd leading cause of cancer deaths in the US. Screening reduces mortality and morbidity, but it is under-utilized.
- Multiple randomized controlled trials (RCT) suggest that mailed outreach invitations to complete CRC screening with a stool blood test can increase screening rates.
- However, mailed outreach has not been implemented on a widespread basis, perhaps due to lack of systematic synthesis of the consistency and magnitude of mailed outreach as a strategy for increasing screening.
- A systematic review and meta-analysis of the impact of mailed outreach offering stool blood tests for CRC screening might increase awareness of the strategy, inform public health recommendations (such as by the Community Guide to Preventive Services), and lead to more widespread adoption.

METHODS
- **Research question:** In adults overdue for CRC screening, is mailed outreach offering a stool-based screening test superior to usual care for promoting screening completion?
- **Population:** Patients not up to date with screening who participated in a RCT evaluating the impact and efficacy of mailed intervention for non-invasive stool testing such as FOBT, FIT or multi-target stool DNA.
- **Intervention:** Mailed invitation to complete colorectal cancer screening.
- **Comparison:** Usual care screening, defined as office visit-based opportunistic opportunistic offers to complete screening.
- **Outcomes:** The primary outcome is mailed screening completion rate.
- **Inclusion criteria:** Patients 18 and older not up to date with CRC screening who participated in a RCT evaluating a mailed stool CRC test; usual care control group; studies from 1980-present
- **Exclusion criteria:** Absence of usual care control; incomplete data; inclusion of patients at higher risk of CRC (IBD, Crohn’s disease)

RESULTS
- Total articles from literature search: n=1,087
  - Title Review: n=1,087
    - Excluded for being obviously irrelevant-doesn’t utilize mailed outreach, etc. n=291
    - Excluded for not being a RCT. n=1
    - Excluded for patients at higher risk of colon cancer. n=49
    - Excluded for absence of usual care control. n=1
    - Excluded for studies of follow-up screening. n=21
  - Abstract Review: n=724
    - Excluded for being obviously irrelevant-don’t utilize mailed outreach, etc. n=528
    - Excluded for not being a RCT. n=30
    - Excluded for incomplete data. n=3
    - Excluded for patients at higher risk of colon cancer. n=20
    - Excluded for absence of usual care control. n=102
    - Excluded for studies of follow-up screening. n=14
- Manuscript Review: n=27 In progress!

FUTURE STEPS
- Completion of manuscript review and data extraction
- Meta-analysis
- Drafting the final paper
- Publication!

CONCLUSIONS
- Out of 1,087 studies, around 13 will ultimately be included in the final meta-analysis
- Preliminary data shows that mailed access to CRC screening kits significantly improves completion rates
- Implications for future standard of care: will mass mailed CRC screening be achievable? Will it become standard?
- Will insurance providers find it feasible to offer mailed CRC screening to all eligible patients? Will this be profitable?
- Mass uptake of mailed CRC screening may save lives by detecting polyps and cancer in early stages.

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Antisense oligonucleotide inhibition of CTGF in Chronic Myeloid Leukemia

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University of San Diego, California

Chronic Myeloid Leukemia (CML) is a progressive hematopoietic malignancy where the abnormal Bcr-Abl fusion protein skew hematopoietic stem cell differentiation toward the myeloid lineage, creating an aberrantly self-renewing progenitor population that gives rise to leukemia stem cells (LSC). Though Tyrosine Kinase Inhibitors (TKI) have greatly increased life expectancy for Chronic Myeloid Leukemia (CML) patients, they have difficulty targeting leukemia stem cells (LSC) that drive disease progression, relapse and Blast Crisis (BC) CML transformation.

PCR Microarray Results:
A PCR microarray analysis of ECM and adhesion protein expression in different myeloproliferative neoplasms identified a significant upregulation of Connective Tissue Growth Factor (CTGF) in BC CML.

RESULTS

Figure 1: CTGF exists as a cleavable protein with a signal peptide preceding four protein binding domains

Figure 2: To create a more effective drug, an analysis of the LSC’s was performed and it identified a significant increase in BC chronic phase CML expression of CTGF.

Objectives:
The lack of curative results from TKI monotherapy underscores the urgent need to map disease progression of chronic phase (CP) CML to its acute stage, blast crisis. This underlying population of leukemia stem cells are believed to be the cause of the blast crisis progression and relapse.

Hypothesis:
We targeted CTGF in hopes of improving our understanding of the mechanisms responsible for CML growth and progression, and also indicate that CTGF represents a viable therapeutic target for the treatment of CML when used in combination with TKI’s.

CONCLUSIONS

Implications of Results:
Taken together, this data improves our understanding of the mechanisms responsible for CML growth and progression, and also indicate that CTGF represents a viable therapeutic target for the treatment of CML when used in combination with TKI’s.

Next Steps:
Further testing needs to be performed in order to optimize conditions for the ASO treatment, more experiments will need to be designed and run preferably with longer time periods and smaller concentrations.

We would need to determine if the decrease in CTGF has any negative effects on the cell and assess for toxicity.

The ASO’s should also be tested with TKI’s to treat Chronic Phase CML, in order to investigate if this combination stops blast crisis transformation.

METHODS

Cloning and design of CTGF overexpression plasmid:
- We inserted the full-length CTGF gene into a lentiviral plasmid.

Figure 3: Cloning the pCDH-CTGF lentiviral overexpression plasmid. (a) Sequences of forward and reverse primers for amplifying CTGF CDS by PCR. (b) Vector diagram for the pCDH-Empty lentiviral packaging plasmid. (c) Vector diagram for the pCDH-CTGF lentiviral packaging plasmid.

Figure 4: Transfection of K562 cells. After 72 hours, fluorescent images were taken of K562 transfected with (a) pCDH-Empty control and (b) pCDH-CTGF plasmids. (c) Fold change in CTGF mRNA expression by qRT-PCR of RNA extracted from whole cellular lysates of transfected cells versus pCDH-Empty control. This was used to to establish stable cell lines for in vitro characterization of CTGF expression

Antisense Oligonucleotides (ASOs)

Figure 5: In antisense gene therapy, short single-stranded pieces of chemically modified nucleotides, known as oligonucleotides are inserted into cells. These short strands are chemically engineered to be complimentary to specific mRNA in the cell, physically blocking translation or recruiting an enzyme known as RNase H to degrade the mRNA.

Figure 6: The qPCR results of the CTGF expression was normalized against a housekeeping gene HPRT. In this bar graph, the 2^-delta Ct value was taken and used as the y-axis normalized against a housekeeping gene HPRT. In this bar graph, the 2^-delta Ct value was taken and used as the y-axis normalized against a housekeeping gene HPRT.
A COMPARISON STUDY OF TEENAGE SMOKING RATES IN CALIFORNIA AND SAN DIEGO COUNTY

Michael Morrison¹, Chelsea Obrochta², Brady Stanton³, Caroline A. Thompson²,⁴

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³. Department of Sociology, San Diego State University ⁴. Department of Family Medicine and Public Health, UC San Diego

BACKGROUND

- In 2015, 30% of all cancer deaths in the United States were related to tobacco.
- Cancer is the leading cause of death among Hispanics and the second leading cause of death among non-Hispanic Whites.
- In addition to cancer, youth smoking has been linked to retarded lung growth, incapacitated cardiovascular functions, and other impairments to the still-growing body.
- California as a whole has made substantial strides in reducing teen smoking.
- It is unclear whether the trends in San Diego county reflect these statewide achievements.

DEMographic Characteristics of San Diego and California

<table>
<thead>
<tr>
<th>Race/Ethnicity</th>
<th>California (%)</th>
<th>San Diego County (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>NH White</td>
<td>37.7%</td>
<td>46%</td>
</tr>
<tr>
<td>Hispanic</td>
<td>38.9%</td>
<td>33.5%</td>
</tr>
</tbody>
</table>

METHODS

Data Source: California Health Interview Survey (CHIS), 2011-2014; 500 cities data (spatial analysis)

Study Population: Residents of California and San Diego who fall within the age range of 13-19 (teens); Adults who live in San Diego County

Outcome: Teens who have smoked in the past 30 days are classified as current smokers. Teens who have never smoked, or have not smoked in the past 30 days are non-smokers. Respondents were also asked “Have you ever smoked electronic cigarettes, also known as e-cigarettes or vaporizer cigarettes?”

Stratification variables: (1) Race/Ethnicity (2) Gender (3) Geographical location

Statistical Analysis: All analyses were conducted separately for Female and Male, California and San Diego, and Hispanic and non-Hispanic White using Microsoft Excel and Joinpoint Regression Program. Bar graphs, line graphs and ArcGIS software (version 10.8.1) were used to examine if, and where, statistically significant trends occurred within the study population.

RESULTS

CURRENT SMOKING STATUS AMONG MALE TEENS IN CA (2011-2014)

CURRENT SMOKING STATUS AMONG FEMALE TEENS IN CA (2011-2014)

CURRENT SMOKING STATUS AMONG MALE TEENS IN SAN DIEGO (2011-2014)

CURRENT SMOKING STATUS AMONG FEMALE TEENS IN SAN DIEGO (2011-2014)

To describe and compare teen smoking rates among White and Latinos in California and San Diego County.

REFERENCES


CONCLUSIONS

- The data show that male white teens in San Diego have had a recent significant increase in current smoking status from 2012-2014. This increase is a higher percentage of current smokers than majority of the San Diego regions where adult smoking status was measured.
- A higher percentage of Male and Female Latino teens in San Diego have smoked an e-cigarette than Male and Female Latino teens in California.

SPECIFIC AIM

- To describe and compare teen smoking rates among White and Latinos in California and San Diego County.

FOR MORE INFORMATION:

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Caroline Thompson: caroline.Thompson@sdsu.edu

Research reported in this poster was supported by the National Cancer Institute of the National Institutes of Health under award numbers: U54CA132384 & U54CA132379
The hepatocyte growth factor receptor (MET, c-MET) plays a key role in both tumorigenesis and metastasis. Furthermore, increased MET protein expression correlates with poor prognosis in a variety of cancer types. Our preliminary data indicate that MET is a substrate for MARCH1, MARCH4, and MARCH8. We hypothesize that MARCH1/4/8 can limit MET function through modification of membrane proximal lysine residues with ubiquitin, targeting MET for lysosomal degradation. To test this hypothesis, we will reconstitute MET knockout cell lines with either wild-type MET (control) or a mutant of MET where all four putative (membrane proximal) ubiquitination acceptor sites are mutated to Arginine (4KR).

Using these reconstituted cell lines, we will first validate that MET is a substrate for MARCH1/4/8 subtypes and determine if MET is regulated through ubiquitination of these four membrane proximal lysine residues. If so, the biological role of MET ubiquitination at these residues will also be examined. Taken together, these studies will investigate a potential mechanism by which MET ubiquitination affects MET function.

Hypothesis: MARCH-mediated Post-translational regulation of Hepatocyte Growth Factor Receptor at the cell surface.

Figure 1: Pathway for the Hepatocyte Growth Factor Receptor, MET.

MET is a receptor tyrosine kinase with high binding affinity for the ligand HGF. MET binds to HGF to induce proliferation and activate other ligand-dependent post-translational pathways. Adapted from Lilly Oncology Pipeline.

Methodology:

- The Hepatocyte Growth Factor Receptor (MET) is amplified in many cancers and a key therapeutic target to limit tumor cell growth.
- The Membrane Associated RING-CH (MARCH) proteins are E3 ubiquitin ligases that modify transmembrane receptors to regulate their surface expression.

Hypothesis: MARCH1/4/8 can limit MET function by modification of membrane proximal lysine residues, targeting MET for degradation.

- This study will characterize a novel mechanism of regulating MET surface expression and function through MARCH-mediated ubiquitination.
- A better understanding of MARCH-mediated MET expression could impact new therapeutics designed to limit MET function in cancer cells.

Figure 2: Generation of MET 4KR.

The nuclear domain sequence of MET coding for the transmembrane domain (yellow) and flanking membrane proximal residues are depicted with the MET reading frame is shown above. The mutations changing the membrane proximal lysine residues to Arginine are highlighted in green. Note the full ORF for wt MET isoform2 (NM_000245.3) and the 4KR mutant were cloned into pCDNA5-FRT-TO with a C-terminal HA epitope tag (YPYDVPDYA).

Figure 3: Workflow to generate Inducible HeLa Flp-In™ T-REX cells to rescue MET KO.

This system permits generation of stable isogenic HeLa cell lines in which MET expression is Doxycycline inducible. (1) HeLa Flp-In™ T-REX cells were described previously (Kean et al. 2013) (2 and 3) Co-transfection with pOG44 and pCDNAs/FRT-MET-wt or 4KR expression vectors allows for insertion of MET via Flp recombinase-mediated DNA recombination. (4) MET; wt or 4KR is expressed through Doxycycline treatment. Adapted from Thermo Fisher.

REFERENCES


ACKNOWLEDGEMENTS

Sanford Bernstein Ph.D., Jennifer Suggs M.S., Sheila Crowe M.D., Cynthia Park Ph.D.

CONCLUSIONS

- MET is a Substrate of MARCH1, MARCH4, and MARCH8, and the catalytic activity of MARCH is required to down regulate MET from the cell surface.
- MET 4KR is expressed at a higher level at the cell surface. Since expression of both wt and 4KR MET are under control of the same transcriptional system (DOX) and both cell lines are isogenic clones with a single integration, this suggests that MET is regulated post-translationally, via ubiquitination of membrane proximal lysine residues on the cytoplasmic tail.
- MET 4KR is resistant to the effects of MARCH1, MARCH4 and MARCH8 suggesting that MARCH proteins regulate MET surface expression through ubiquitination of membrane proximal lysine residues.

Future Directions

- Biochemically characterize MARCH-mediated Ubiquitination of wt vs. 4KR MET upon over expression of MARCH1, MARCH4 or MARCH8 in HeLa Flp-In T-REX MET rescue cell lines.
- Compare signaling of wt MET vs. 4KR MET upon over expression of MARCH1, MARCH4 or MARCH8 in HeLa Flp-In T-REX MET rescue lines.
- Compare signaling of wt MET with and without MARCH1, MARCH4 or MARCH8 over expression upon HGF stimulation in HeLa Flp-In T-REX MET rescue cell lines.
- Compare signaling of 4KR MET with c-terminal HA tag in HeLa parental cells with or without T-REX T-REX MET rescue.

Figure 4: Generation of MET KO Hela Flp-In™ T-REX™ cells. Hela Flp-In™ T-REX™ cells were transfected with p53Cas9-ZFMini encoding a MET specific siRNA (si49). Cells were transiently selected with Puromycin and cultured for 10 days. After 10 days a sub-population of MET KO cells was purified by magnetic depletion with anti-human MET. Depletion of MET positive cells was verified by flow cytometry with a non-cross competing MET antibody.

Figure 5: Doxycycline Induction of MET wt and MET 4KR. Rescued MET KO Hela Flp-In™ T-REX™ cells were treated with indicated doses of Doxycycline for 40 hours and surface MET expression determined by flow cytometry. The MFI of MET KO cells was subtracted and the ratio of the 4KR: wt MET is shown as a function of [Dox]. The arrow indicates the dose used for subsequent experiments.

Figure 6: MET 4KR is resistant to MARCH-mediated Regulation MET KO HeLa Flp-In T-REX cells rescued with either empty vector (MET-KO), MET-wt or MET 4KR were induced with 62.5ng/ml of Doxycycline for 24 hours and then transfected with the indicated MARCH-GFP expression vectors. 24 hours post transfection, single cells suspensions of cells were surface stained for MET and viability using antibodies to MET expression of viable (PI negative)/GFP+ cells are depicted.

METHODS

CONCLUSIONS

SPECIFIC AIMS

Aim 1: Generate tools to study MARCH Mediated regulation of MET.
- Design and generate sgRNAs targeting human MET (completed).
- Generate MET KO Flp-In™ T-REX™ HeLa cells (completed).
- Clone wt and 4KR MET with c-terminal HA tags into pcDNS5/FRT-MET vectors (completed).
- Rescue MET KO HeLa Flp-In T-REX cells with either:
  - Empty vector
  - MET wt-HA
  - MET 4KR-HA

Aim 2: Characterize MET 4KR mutant
- RT-TO: validation of wt vs. 4KR MET upon transient transfection of MARCH1, MARCH4 or MARCH8.
Cancer Fatalism in Latino/as: Relationship to Sociodemographics

Davana F. Rosas\textsuperscript{ab}, Sandy S. Bohan\textsuperscript{bc}, France Nguyen-Grozavu\textsuperscript{b}, Lawrence Alfred\textsuperscript{b}, Georgia Robins Sadler\textsuperscript{bef}, Vanessa L. Malcarne\textsuperscript{bdf}

\textsuperscript{a}University of California, San Diego; \textsuperscript{b}UC San Diego Moores Cancer Center; \textsuperscript{c}San Diego State University/UC San Diego Joint Doctoral Program in Public Health; \textsuperscript{d}SDSU; \textsuperscript{e}UC San Diego School of Medicine; \textsuperscript{f}SDSU/UC San Diego Joint Doctoral Program in Clinical Psychology

\textbf{Background/Purpose}

- Cancer screening improves cancer outcomes.
- Latino/as have low rates of cancer screening.
- Cancer fatalism is the belief that a diagnosis of cancer leads to death.
- Cancer fatalism may negatively impact cancer screening behavior.
- The purpose of this study is to examine cancer fatalism among Latino/as in relation to sociodemographic factors.

\begin{table}[h]
\centering
\begin{tabular}{|l|c|c|}
\hline
\textbf{Sample Characteristics (N = 1858)} & \textbf{n} & \textbf{\%} \\
\hline
\textbf{Gender} & & \\
Male & 654 & 35.2 \\
Female & 1204 & 64.8 \\
\hline
\textbf{Education Level} & & \\
\textless High school diploma & 648 & 34.9 \\
High school diploma or equivalent & 316 & 17.0 \\
Vocational/trade school or associate degree & 249 & 13.4 \\
Some college, no degree & 332 & 17.9 \\
\geq Bachelor’s degree & 282 & 15.2 \\
Don’t know & 3 & 0.1 \\
missing & 28 & 1.5 \\
\hline
\textbf{Language Preference} & & \\
English & 920 & 49.5 \\
Spanish & 938 & 50.1 \\
\hline
\textbf{Age, years (range: 25 - 89)} & & \\
M & 47.5 & 13.7 \\
SD & & \\
\hline
\textbf{Powe Fatalism Inventory, total} & 4.4 & 3.2 \\
Duke University Religion Index & & \\
Attends church or other religious meetings & 3.7 & 1.6 \\
Spends time in private religious activities & 4.0 & 1.8 \\
\hline
\textbf{Brief Acculturation Scale for Hispanics} & 10.9 & 5.2 \\
\hline
\textbf{Perceived Health Literacy} & & \\
Confident filling out medical forms & 2.0 & 1.0 \\
Needs help reading hospital materials & 2.1 & 1.2 \\
\hline
\textbf{MacArthur Scale of Subjective Social Status} & 5.0 & 2.0 \\
in the United States & & \\
\hline
\end{tabular}
\end{table}

\textbf{Methods}

- Data from 5 San Diego area studies.
- Participants were:
  - Hispanic/Latino
  - United States residents
  - \geq 21 years old
  - proficient in English or Spanish
- Data analysis:
  - descriptive statistics
  - linear regression model evaluated sociodemographic factors as predictors of cancer fatalism

\textbf{Measures}

- \textbf{Powe Fatalism Inventory (PFI)}
  - score range: 0 - 15
  - higher score indicates higher fatalism
- \textbf{Duke University Religion Index}
  - score range: 1 - 6
  - higher score indicates more religious involvement
- \textbf{Brief Acculturation Scale for Hispanics}
  - score range: 4 - 20
  - higher score indicates greater level of acculturation
- \textbf{Perceived Health Literacy}
  - score range: 1 - 5
  - higher score indicates lower health literacy
- \textbf{MacArthur Scale of Subjective Social Status}
  - in the United States
  - score range: 0 - 10
  - higher score indicates higher perceived social status

\textbf{Results}

- Average cancer fatalism was low in the study sample.
- Higher fatalism was found among individuals who:
  - were older, less educated, less health literate
  - preferred English
- There were no other significant predictors of fatalism.

\textbf{References}


This research was supported by grants from the National Cancer Institute of the National Institutes of Health: U54CA132384, U54CA132379, R25CA132699, R25CA130869; & California Breast Cancer Research Programs 13AB-35.3700, 14BB2601. For more information contact Vanessa L. Malcarne vmalcarne@mail.sdsu.edu
**BACKGROUND**

Hispanics/Latinos and Physical Activity
- Hispanics/Latinos are the largest minority in the United States and they are expected to triple by 2050. As the Hispanic/Latino population grows, their prevalence to cancer and other chronic diseases also increases.¹
- Meeting the necessary guidelines of 150 minutes a week of moderate to vigorous physical activity, has been linked to lower cancer risk. In comparison to non-Hispanic whites, Hispanics/Latinos are less likely to engage in leisure physical activity, placing them at great risk for chronic illnesses and cancer.²,³
- Specifically Hispanic/Latino women, engage in less leisure time physical activity compared to Hispanic/Latino men.⁴

Faith-Based Settings and Hispanics/Latinos
- Churches can have great influence on the health practices of the community that they serve. Practice of religion and church involvement has shown to be a great part of the Hispanic community, with 68% of Hispanics identifying as Catholic. Church setting interventions can be a good approach to promoting health, since religion and faith has shown to be an important aspect of many people in the Hispanic community.¹,²
- Fe en Acción was a study conducted in catholic churches of San Diego County, with the purpose of increasing physical activity among churchgoing women. This study used a “promotor” model which involved training individual women from the community to give them the capacity of implementing physical activity classes in churches.¹
- The study increased participants’ physical activity, and reduced their BMI at 12 months following baseline.²

**METHODS**

**Procedures:**
- Descriptive Data Collection
  - Data collection will take approximately 3 months to complete.

**Design:**
- Mixed methodology: Qualitative and quantitative data.
- Individual level research: multiple choice and free response survey for participants, interview with church leaders, focus groups with promotores.

**INSTRUMENT**

Using the framework, sample questions for interview, focus groups and surveys were developed:

- Promoters:
  - What costs have you incurred since the completion of the program? What costs should be considered to help sustain the program?
  - Are you currently leading a physical activity group? To what extent? (e.g., days, type, etc.)
  - What support do you need to continue to implement the program on your own?

- Participants:
  - In what way could Fe en Acción have better impacted your involvement in physical activity? Explain
  - If you no longer partake in physical activity, what were some barriers that prevented you from continuing physical activity?
    - Financial burden
    - Lack of time
    - Transportation constraints
    - Other.
  - Would you still be interested in programs that promote physical activity?
    - a. No.
    - b. Yes.
    - If yes, describe what kind of programs would you like to see?

- Church Leadership:
  - What facilitated the continuation of Fe en Acción? What resources would the church need to sustain the program effectively?
  - What benefits do you see as significant in partnering with an institution? Did involving the university program Fe en Acción, give the church community a sense of ownership?
  - How would you describe the communication amongst the church? i.e., between church leaders and volunteers, staff, public.

**NEXT STEPS**

- **Timeline:**
  - 1 month - Finalize survey, interview questions, and focus group discussion questions, invitation letters and call scripts.
  - 1 month - Mail surveys to participants, schedule interviews with church leaders, schedule focus group dates and contact promotores.
  - 3 months - Collecting data, implementing research: conducting interviews, facilitating focus groups, contacting participants who have not completed the survey.
  - 3 months - Analyzing data collected to formulate an action plan for addressing sustainability issues.

---

**REFERENCES**


**SPECIFIC AIDS**

Evaluate factors that influence program sustainability from the perspective of participants, church leaders, and promotores

- Findings from the current study will inform an implementation study with the goal of sustaining activities following completion of the implementation study.
- The current study is innovative in that there is no published research reporting on program sustainability in faith based settings promoting physical activity.

**ACKNOWLEDGEMENTS**

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**FOR MORE INFORMATION:**

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Elva Arredondo at: earredondo@mail.sdsu.edu
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Cancer Immunotherapy Response Rates Across Ethnic Groups

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¹San Diego State University ²UC San Diego Moores Cancer Center

BACKGROUND

Cancer immunotherapy is a treatment that stimulates your own immune system to fight cancer cells. Today, the analysis of patients under this treatment could help researchers understand how well immunotherapy works and how safe it is, and to explore why some patients may respond better. Focusing on ethnicity across histology could reveal differences in response rates in ethnic groups. Across the multiple factors that could explain response rate, ethnicity is an important filter because it composes a multifactorial concept including, but not limited to genetic background.

Fig. 1 Immunotherapy Patients Across Ethnicity

TABLE 1. Demographics

<table>
<thead>
<tr>
<th>Criteria</th>
<th>Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median age</td>
<td>62</td>
</tr>
<tr>
<td>Age Range</td>
<td>[27-90]</td>
</tr>
<tr>
<td>Female</td>
<td>71 (43%)</td>
</tr>
<tr>
<td>Male</td>
<td>93 (57%)</td>
</tr>
<tr>
<td>Patients on FDA</td>
<td>56%</td>
</tr>
<tr>
<td>Patients on Clinical Trial</td>
<td>44%</td>
</tr>
</tbody>
</table>

Total No. of Patients: 164
No. PSF Patients: 125
Indeterminate: 20
Pending: 19

How Does Immunotherapy Work?

Tumor cells tend to Traits to deactivate them
Immunotherapy drugs can block tumor cells from deactivating Traits

SPECIFIC AIMS

1. Illustrate the demographics of patients under cancer immunotherapy and retrospectively interpret clinical data from patients who obtained immunotherapy treatment.
2. Present how the response rate in Hispanic and Latino patients differs from the rest of the ethnic groups in this study: Asian, Black, White.
3. Display the response rate of histology across all ethnicities and present major differences among them.

METHODS

A retrospective chart review was conducted by collecting data from 164 patients. This information was coded with a study number to avoid disclosing the patient’s name or identifying information. Clinical data included information such as demographics, cancer diagnosis, treatment histories, treatment outcomes, family history, cancer, and toxicity. Progression Free Survival (PFS) is the length of time during and after the immunotherapy treatment of a disease, such as cancer, that a patient lives without the disease but it does not worsen or deteriorate. PFS was calculated in months from the start date to the day they progressed or based on their most recent visit with their oncologist. The following concept map illustrates the criteria of categorizing a patient as responder or non-responder:

RESULTS

Fig. 2 Average PFS Across Ethnicities

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Median PFS (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asian</td>
<td>4.05</td>
</tr>
<tr>
<td>White</td>
<td>4.28</td>
</tr>
<tr>
<td>Black</td>
<td>2.95</td>
</tr>
<tr>
<td>Hispanic</td>
<td>3.18</td>
</tr>
</tbody>
</table>

Fig. 3 Response Rate Across Ethnicity

<table>
<thead>
<tr>
<th>Ethnicity</th>
<th>Response Rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hispanic</td>
<td>20%</td>
</tr>
<tr>
<td>Asian</td>
<td>50%</td>
</tr>
<tr>
<td>White</td>
<td>55%</td>
</tr>
</tbody>
</table>

Fig. 4 Average Progression Free Survival of Hispanic/Latino vs. Whites

<table>
<thead>
<tr>
<th>Response to treatment</th>
<th>Progression of Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hispanic</td>
<td>11.43</td>
</tr>
<tr>
<td>White</td>
<td>12.91</td>
</tr>
<tr>
<td>Head and Neck</td>
<td>7</td>
</tr>
<tr>
<td>Brown</td>
<td>1.96</td>
</tr>
<tr>
<td>Lung</td>
<td>2.43</td>
</tr>
</tbody>
</table>

Fig. 5 Average Progression Free Survival Across Histology

<table>
<thead>
<tr>
<th>Histology</th>
<th>Total Number of Patients</th>
<th>Number of Responders</th>
<th>Number of Non-Responders</th>
<th>% Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head and Neck</td>
<td>38</td>
<td>14</td>
<td>15</td>
<td>36.8%</td>
</tr>
<tr>
<td>Melanoma</td>
<td>36</td>
<td>15</td>
<td>13</td>
<td>41.7%</td>
</tr>
<tr>
<td>Lung</td>
<td>30</td>
<td>16</td>
<td>14</td>
<td>30.0%</td>
</tr>
<tr>
<td>GI</td>
<td>28</td>
<td>20</td>
<td>8</td>
<td>71.4%</td>
</tr>
<tr>
<td>GU</td>
<td>16</td>
<td>4</td>
<td>12</td>
<td>25.0%</td>
</tr>
<tr>
<td>Breast</td>
<td>8</td>
<td>4</td>
<td>4</td>
<td>50.0%</td>
</tr>
<tr>
<td>Hematologic</td>
<td>5</td>
<td>3</td>
<td>2</td>
<td>60.0%</td>
</tr>
</tbody>
</table>

CONCLUSIONS

Figure 1 illustrates the majority of immunotherapy patients under the White ethnic group. According to Figure 2, the Black population had an overall longer PFS average across histology compared to other ethnicities. Across all ethnicities, the Hispanic/Latinos had the lowest response rate to cancer immunotherapy treatment (20%) compared to other ethnicities (Figure 3). There was only enough patient data to make a comparison between two ethnic groups: Hispanic/Latinos and Whites. Figure 4 illustrates their comparison across the three most common cancer histology: melanoma, head and neck, and lung. It is observed that PFS is longer in Whites (except for head and neck cancers). This means that their disease could remain stable or respond to treatment without progressing. Figure 5 displays the PFS average across some of the most common types of cancers, comparing the responder patients vs. the non-responder patients. Lung and breast cancer evidently have the leading PFS across histology for the responders, and simultaneously, the shortest PFS for non-responders.

Hispánics seem to have similar benefit to cancer immunotherapy to other ethnic groups, however the overall response rate in Hispanics is more 20% lower than the rest. However, those patients that did response tended to have prolonged survival, especially if they had lung or head and neck cancer. Research into biomarkers to determine which patients benefit from cancer immunotherapy are crucial in determining which patients benefit from cancer immunotherapy, and research efforts into explaining the lower rate of response to cancer immunotherapy in Hispanic patients should focus on social as well as biologic. Another following step in this research would be to consider a more diverse population or analyze more patients that fall under the minority population: Hispanics, Asian and Blacks. It would be important to consider community outreach efforts to make sure Hispanic cancer patients are aware of cancer immunotherapy as a treatment option. Furthering these studies would help us understand what factors influence response rates across different ethnicities.

REFERENCES

• Treatment with Immunotherapy.
• UCSD Moores Cancer Center Immunoscope Database.

FOR MORE INFORMATION:

Valeria Mejia, SDSU-UCSD Partnership Scholar Email: vmejia@sdstate.edu

Research reported in this poster was supported by the National Cancer Institute of the National Institutes of Health under award numbers: U54CA132334 & U54CA132379.
Is Hispanic ethnicity associated with acceptance of not having more children in adolescent and young adult cancer survivors?

A. Vasilieva¹, S. Stark², H.I. Su³
¹Sd State, ²UCSD Moores Cancer Center

BACKGROUND

- Adolescent Young Adult (AYA) women diagnosed with cancer undergo treatments such as radiation, surgery, or chemotherapy that can impart reproductive consequences (infertility, pregnancy complications, etc.).
- After treatment, AYA survivors experience a number of reproductive health concerns.
- Reproductive behavior of Hispanic women differ from non-Hispanic women. Example: Hispanic women have children earlier in life and larger families than non-Hispanic women.
- There are no data that examine if reproductive health concerns vary by Hispanic ethnicity.
- Reproductive health concerns can be measured by the Reproductive Concerns After Cancer (RCAC) scale, which includes six domains: 1. Acceptance of not having more children 2. Partner disclosure 3. Child’s health 4. Personal Health 5. Fertility potential 6. Becoming Pregnant
- Post Traumatic Growth Inventory (PTGI) has two questions that measure the degree to which participants reported 1) a better health concerns. 2. Through praying or meditating.
- Adolescent Young Adult (AYA) women diagnosed with cancer undergo treatments such as radiation, surgery, or chemotherapy that can impart reproductive consequences (infertility, pregnancy complications, etc.).

SPECIFIC AIMS

**Aim 1**: To test the association between Hispanic ethnicity and acceptance of not having more children after cancer

**Hypothesis 1**: As compared to non-Hispanic participants, Hispanic participants will report being more accepting of not having additional children after cancer.

**Aim 2**: To test the association between religiosity and/or spirituality after cancer and acceptance of not having more children after cancer

**Hypothesis 2**: Participants who report greater religiosity and/or spirituality after cancer will report being more accepting of not having additional children after cancer.

METHODS

Study design: Retrospective cohort

Participants: Female cancer survivors who are 18-40 years old and participating in the Window Study on ovarian function

Exposure: Hispanic ethnicity

Outcome: Acceptance of not having more children after cancer (RCAC Scale, Acceptance subscale, range 1-5).

Subscale questions:
- 1. I will feel content if I do not have (more) children.
- 2. I will be happy with life whether or not I have (more) children someday.
- 3. I can accept it if I’m unable to have (more) children.

Analysis:
1. Descriptive data
2. Dichotomize Acceptance score into ≤3 and ≥3
3. Bivariate analysis to estimate the association between the exposures and outcome (Student’s t-test, Chi-square)

RESULTS

**Key Findings**
1. In our cohort of 747 participants, 25% reported that they cannot accept if they are unable to have more children.
2. Compared with non-Hispanic participants, Hispanic women were not more likely to report acceptance (Figure 1).
3. Spirituality and religiosity were not associated with acceptance (Figure 2).
4. Compared with non-Hispanic participants, Hispanic women reported greater religiosity/spirituality in coping.

**Table 1: Association between demographics and acceptance of not having more children in female AYA cancer survivors (n=747)**

<table>
<thead>
<tr>
<th>Overall</th>
<th>Accept</th>
<th>Don't Accept</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>18-25</td>
<td>61 (8)</td>
<td>38 (7)</td>
<td>23 (12)</td>
</tr>
<tr>
<td>25-30</td>
<td>178 (24)</td>
<td>114 (21)</td>
<td>64 (34)</td>
</tr>
<tr>
<td>31-35</td>
<td>250 (34)</td>
<td>186 (34)</td>
<td>64 (34)</td>
</tr>
<tr>
<td>36-41</td>
<td>247 (34)</td>
<td>212 (39)</td>
<td>35 (19)</td>
</tr>
<tr>
<td>Race</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black or African American</td>
<td>16 (2)</td>
<td>9 (2)</td>
<td>7 (4)</td>
</tr>
<tr>
<td>White</td>
<td>544 (73)</td>
<td>406 (73)</td>
<td>138 (73)</td>
</tr>
<tr>
<td>Asian</td>
<td>45 (6)</td>
<td>35 (6)</td>
<td>10 (5)</td>
</tr>
<tr>
<td>American Indian or Alaskan Native</td>
<td>2 (0.3)</td>
<td>2 (0.4)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Native Hawaiian or other Pacific Islander</td>
<td>5 (7.7)</td>
<td>3 (5)</td>
<td>2 (1)</td>
</tr>
<tr>
<td>Mixed race</td>
<td>58 (8)</td>
<td>43 (8)</td>
<td>15 (8)</td>
</tr>
<tr>
<td>Some other race</td>
<td>58 (8)</td>
<td>46 (8)</td>
<td>12 (6)</td>
</tr>
<tr>
<td>Income</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; $5,000</td>
<td>200 (27)</td>
<td>144 (26)</td>
<td>56 (30)</td>
</tr>
<tr>
<td>$5,000 to $10,000</td>
<td>253 (34)</td>
<td>192 (34)</td>
<td>61 (33)</td>
</tr>
<tr>
<td>$10,000 to $150,000</td>
<td>140 (18.7)</td>
<td>104 (18.6)</td>
<td>36 (19)</td>
</tr>
<tr>
<td>&gt; $150,000</td>
<td>107 (14)</td>
<td>83 (15)</td>
<td>24 (13)</td>
</tr>
<tr>
<td>Marital Status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Married or living together with a heterosexual partner</td>
<td>249 (33)</td>
<td>177 (32)</td>
<td>72 (38)</td>
</tr>
<tr>
<td>Same sex partner, widow, divorced, never married</td>
<td>496 (67)</td>
<td>382 (68)</td>
<td>116 (62)</td>
</tr>
</tbody>
</table>

**Figure 1: Hispanic Ethnicity and Acceptance**

![Image](image1)

**Figure 2: Religious/ Spirituality in Posttraumatic Growth and Acceptance**

![Image](image2)

**Table 2: Association between health and acceptance of not having more children in female AYA cancer survivors (n=747)**

<table>
<thead>
<tr>
<th>Overall</th>
<th>Accept</th>
<th>Don't Accept</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>General health</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excellent</td>
<td>70 (9)</td>
<td>54 (10)</td>
<td>16 (9)</td>
</tr>
<tr>
<td>Very good</td>
<td>288 (39)</td>
<td>215 (39)</td>
<td>73 (39)</td>
</tr>
<tr>
<td>Good</td>
<td>301 (40)</td>
<td>228 (41)</td>
<td>73 (39)</td>
</tr>
<tr>
<td>Fair</td>
<td>81 (11)</td>
<td>57 (10)</td>
<td>24 (13)</td>
</tr>
<tr>
<td>Poor</td>
<td>5 (7)</td>
<td>3 (5)</td>
<td>2 (1)</td>
</tr>
<tr>
<td>I have no medical problems</td>
<td>193 (26)</td>
<td>155 (28)</td>
<td>38 (20)</td>
</tr>
<tr>
<td>Hysterectomy</td>
<td>17 (2)</td>
<td>15 (3)</td>
<td>2 (1)</td>
</tr>
<tr>
<td>Unilateral oophorectomy</td>
<td>49 (7)</td>
<td>40 (7)</td>
<td>9 (6)</td>
</tr>
<tr>
<td>History of Live Birth</td>
<td>202 (39)</td>
<td>257 (40)</td>
<td>35 (19)</td>
</tr>
</tbody>
</table>

**Cancer type**

- Thyroid/skin/melanoma
- Breast
- Cervical/uterine/ovarian
- Bone/blood/lymphoma/soft tissue
- GI
- Surgery to remove tumor
- Radiation
- Chemotherapy

**Table 3: Association between religiosity/spirituality and acceptance of not having more children in female AYA cancer survivors (n=747)**

<table>
<thead>
<tr>
<th>Religious/ Spirituality</th>
<th>Accept</th>
<th>Don't Accept</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Accept</td>
<td>455 (61)</td>
<td>339 (61)</td>
<td>116 (62)</td>
</tr>
<tr>
<td>Don’t Accept</td>
<td>544 (73)</td>
<td>406 (73)</td>
<td>138 (73)</td>
</tr>
</tbody>
</table>

**Figure 3: Religious/ Spirituality in Posttraumatic Growth and Acceptance**

![Image](image3)

**Figure 4: Religious/ Spirituality in Posttraumatic Growth and Coping Differs by Hispanic Ethnicity**

![Image](image4)

REFERENCES


ACKNOWLEDGEMENTS

We thank participants of the Window Study and members of the Window Study research team.

FOR MORE INFORMATION:

http://www.youngcancersurvivor.com/
Role of insulin-like growth factor 2 (IGF2) in hepatocellular carcinoma development in Serine/arginine-rich splicing factor 3 (SRSF3) KO mice

Morgan Knudsen, Deepak Kumar PhD, Nicholas Webster PhD
University of California San Diego

BACKGROUND

Serine/arginine-rich splicing factor 3 (SRSF3), one of the smallest members of the SR protein family, plays a role in RNA splicing. The deletion of the gene SRSF3 has been shown to be related to liver fibrosis and hepatocellular carcinoma.

Studies conducted on the deletion of SRSF3 in hepatocytes of mice have shown a number of different results including impaired hepatocyte metabolism and maturation. Further, in SRSF3 knockout mice there is decreased glycogen storage, lactic acidosis, and an increase in insulin sensitivity. Also in SRSF3 knockout mice, an increase in IGF2 expression was found in mice liver tumors and hepatocytes. An increase in IGF2 can contribute to proliferation of the liver tumors and is related to a poor prognosis in multiple cancers.

METHODS

Using PCR we were able to identify and obtain our desired genotype for our experiment. Using RT-qPCR we were able to quantify the DNA by dissection and perfusion of the liver we isolated hepatocytes for obtaining genomic DNA. We were also able to obtain tissue samples for staining.

For analyzing the histology of the liver samples we looked at:
1. H&E staining- which enabled us to look for lipid droplets and nuclear structure
2. PAS staining- allowed us to look at differences in glycogen storage
3. Trichome Staining- allowed us to analyze differences in collagen formation

RESULTS

Upon dissection of the mice:
1. We did not see a significant difference in body weight or liver and spleen size when comparing the double knockout mice to the WT mice.
2. Part of the small intestine and the cecum was black in the SRSF3/IGF2KO mice.
3. When analyzing the histology of the mice we noticed the following:
   a. A difference in nuclei structure in which the double knockout mice had nuclei structures resembling those of the SRSF3 KO mice.
   b. A difference in the amount of collagen between the two mice livers, especially thickened amounts of collagen outlining the edges of the liver as well as surrounding the arteries.

Finally, from this experiment we have not yet seen tumor growth in the double knockout mice. However, we need more time to come to a more accurate conclusion regarding the role of IGF2 on tumor growth because the mice we have dissected were still young.

REFERENCES


ACKNOWLEDGEMENTS

We thank the VA and the National Cancer Institute for supportive funding for this research project. We thank UCSD Stein Clinical Research Center for letting us carry out our project here. We thank Consuelo Saeza, Emilie Gross, and Manasi Das for supportive help on our project.

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Identification of a Polarized Exocytic Machinery Important for Cancer Invasion
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2. Department of Medicine, University of California, San Diego School of Medicine, La Jolla, CA 92033, USA
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BACKGROUND

Polarized exocytosis is an essential biological function that has been conserved throughout nearly all eukaryotic cells and is necessary for cell growth and directional cell migration. This process involves delivering cargo from within the cell to specific sites on the plasma membrane. Targeting vesicles to specific sites on the plasma membrane requires a great degree of spatial control, which the actin cytoskeleton, microtubule motors, and Rab-family GTPases are important regulators. Rab GTPases direct the budding of exocytic vesicles containing polarized cargo. In this case, Rab GTPases regulate the budding of exocytic vesicles containing polarized cargo, which is critical for cell migration. However, no homologues or orthologues of ERM proteins have been found in this process.

We noted that much like ERM proteins in other systems, GUVs (i.e., giant vesicles) are specialized in forming actin structures. GUVs are an excellent model for studying exocytosis, especially in the context of cancer cell migration. The ability of GUVs to form actin structures allows for the study of exocytosis in a dynamic environment. The formation of actin structures on the cell surface can be visualized using fluorescent microscopy, providing valuable insights into the mechanism of exocytosis.

RESULTS

A. GUVs and BeImP

B. GUVs and BeImP

C. GUVs and BeImP

D. GUVs and BeImP

E. GUVs and BeImP

F. GUVs and BeImP

G. GUVs and BeImP

H. GUVs and BeImP

I. GUVs and BeImP

J. GUVs and BeImP

K. GUVs and BeImP

L. GUVs and BeImP

M. GUVs and BeImP

N. GUVs and BeImP

O. GUVs and BeImP

P. GUVs and BeImP

Q. GUVs and BeImP

R. GUVs and BeImP

S. GUVs and BeImP

T. GUVs and BeImP

U. GUVs and BeImP

V. GUVs and BeImP

W. GUVs and BeImP

X. GUVs and BeImP

Y. GUVs and BeImP

Z. GUVs and BeImP

SPECIFIC AIMS

To test our hypothesis of GUVs binding to Exo70 to regulate polarized exocytosis when stimulated, we aimed to:

- Identify the interaction sites on Exo70 responsible for binding with GUV
- Identify the interaction site on GUV required for binding with Exo70
- Investigate GUV role in polarized exocytosis important for cancer invasion

METHODS

GUV Formation

GUVs were formed by the standard method of extrusion using a polycarbonate filter with a 0.1 μm pore size and a negative pressure of -100 mbar. The final size of the GUVs was measured using a DLS technique. The GUVs were then stained with a fluorescent dye (Rhodamine Dextran, molecular weight 10,000 Da) to detect exocytosis.

Exocytosis Assay

Cells were plated on coverslips and treated with an inhibitor of exocytosis (e.g., 10 μM LY294002). After a specified time, the cells were fixed and stained with an antibody specific to the exocytosis marker. The percentage of cells with exocytosis was quantified using ImageJ software. The results were analyzed using a one-way ANOVA with a Tukey post-hoc test.

RESULTS

A. GUVs and BeImP

B. GUVs and BeImP

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REFERENCES


CONCLUSIONS

- Our results suggest that a GUV may be the mammalian counterpart of BeImP.
- Exo70 translocation to the plasma membrane is regulated by GUV, suggesting that polarized exocytosis is impaired.
- Depletion of GUV resulted in a dramatic reduction of MTI-MMP delivery to specific sites on the plasma membrane, including cancer cells.
- Additionally, GUVs repressed cell migration for less degradation of their surrounding ECM compared to the control cells, suggesting that MMP secretion was nearly inhibited. With such a handicap, cancer cells would be unable to invade neighboring tissues and metastasize.
- Our future direction will include determining binding domains of human Exo70 that the protein interacts with.

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