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"Comparing Treatments Among Youth: Exploring Potential Implications for Mental Health Stigma"

BACKGROUND: Mental health stigma—negative perceptions of mental illness—can discourage or delay treatment seeking. Some diagnoses may be more stigmatized than others. Understanding how stigma may influence service use is important for improving engagement in evidence-based practices (EBPs). Functional Family Therapy (FFT) is a family-based EBP that ameliorates youth behavioral concerns by communication whereas Trauma-Focused Cognitive Behavioral Therapy (TF-CBT) helps families manage trauma-related thoughts, emotions, and symptoms. Although both approaches focus on families, they are used with different diagnoses. (FFT with ADHD and TF-CBT with PTSD)

OBJECTIVES: This study aims to determine if the average number of claims differs between FFT and TF-CBT for Louisiana Medicaid youth aged 6–18 by analyzing the association between FFT and TF-CBT average claim counts while accounting for age and gender.

METHOD: The study is a retrospective, cross-sectional examination of de-identified 2024 Medicaid claims data (N=8007). Inclusion criteria included only FFT and TF-CBT claims, aged 6-18, ADHD and PTSD diagnoses, resulting in a final study sample size of 1904. Data was analyzed utilizing Microsoft Excel and SAS version 9.4 using two-sample t-test, multivariable logistic, and linear regression to determine possible associations.

RESULTS: Among 8,007 claims, 7,748 (96.8%) were FFT and 259 (3.2%) were TF-CBT. After adjusting for age group and gender, FFT was associated with an average of 1.14 more claims per recipient than TF-CBT (β=1.145, SE=0.080, $p<.0001$). Youth aged 11–15 had significantly more claims per recipient than youth aged 6–10 (β=0.276, $p=.026$), whereas the number of claims did not differ significantly for youth aged 16–18 compared with those aged 6–10 ($p=.690$) or by gender ($p=.594$). Across demographic groups, average claims per recipient were consistently higher for FFT than TF-CBT.

CONCLUSION: The statistical analyses show that there are differences between FFT and TF-CBT treatment claims. According to the literature, stigma could serve as a potential contributor to this difference. To further examine this, surveys aimed at measuring stigma should be administered. Possible confounding variables: lack of resources, ability to attend, and cost could not be controlled for.

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"Development of PDE10A Inhibitor for the treatment of Triple Negative Breast Cancer"

Triple-Negative Breast Cancer (TNBC) is the most aggressive form of breast cancer, and it accounts for about 15-20% of all breast cancer cases. Patients with TNBC have the highest likelihood of metastasis and relapse. Unlike most breast cancers, TNBC is negative for Estrogen, Progesterone, and HER2 receptors which makes it difficult to treat with targeted therapies. The only effective therapies are chemotherapy and radiation which have debilitating side effects. Due to these limitations, existing therapies either need to be improved or new therapies need to be developed.

PDE10A is an enzyme that regulates intracellular signaling by degrading cyclic adenosine monophosphate (cAMP) and cyclic guanosine monophosphate (cGMP). In many TNBC cases, overexpression of PDE10A leads to the activation of signaling pathways, such as the Ras-MAPK pathway, that are responsible for aggressive cell proliferation and metastasis. These findings make PDE10A a potential therapeutic target for treating TNBC. Considering the current therapeutic role of PDE10A inhibition, Dr. Gary Piazza and his colleagues (Auburn University) have developed and characterized a novel PDE10A inhibitor named compound Y (due to patenting issues, the exact name and chemical formula of the compound is kept unknown and we will use the compound name as Y from hereafter). Using the Kaplan Meier Plot, we found a higher expression of PDE10A in TNBC patients, and we confirmed overexpression of PDE10A in TNBC cell lines. In our current study, we evaluated the effects of compound Y on TNBC cells. We performed MTT assays, colony forming assays, apoptosis assays, and mammosphere formation assays.

Our results indicated that compound Y significantly inhibited TNBC cell proliferation and colony forming ability in dose-dependent manners. Further, compound Y significantly induced apoptosis in TNBC cells. We also analyzed the effects of compound Y on *in vitro* tumor models, mammospheres. The results demonstrated that compound Y significantly inhibited mammosphere growth in dose-dependent manners with an $IC_{50\ of}$ approximately 0.45 μM. Considering our current results, we may conclude that compound Y seems to be a promising candidate for targeting TNBC. However, further studies are needed to evaluate the mechanisms of compound Y and its applicability with other therapies such as chemo- and immunotherapies.

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"A Double Burden: Age- and Sex-Dependent Phenotype of Alzheimer's Disease in 5xFAD Mice"

BACKGROUND: Alzheimer's Disease (AD) is the leading cause of dementia worldwide. It affects approximately 7 million people in the U.S., with nearly two-thirds of cases occurring in women. AD is characterized by progressive memory loss, neuronal degradation, and accumulation of beta-amyloid plaques (Aβ), tau pathology, and neurofibrillary tangles (NFTs). Although these pathological features of AD are well characterized, effective treatments or preventive therapies remain limited. In addition, relatively little research has focused on sex-dependent mechanisms that might contribute to disease progression. Animals are therefore essential for studying these mechanisms, particularly those that closely mimic AD in humans. While several transgenic mouse models have advanced our understanding of the pathology, many do not fully recapitulate the disease in both males and females.

OBJECTIVES: The 5xFAD mouse model reproduces key pathological features, including robust Aβ pathology and neuronal loss, making it well suited for examining sex-dependent differences in the behavioral and cognitive manifestations of AD.

METHODS: In this preliminary study, we used young adult (4 months of age) and one-year-old male and female 5xFAD mice to characterize age- and sex-dependent differences in behavioral phenotypes. Groups were subjected to three different behavioral testing including the Novel Arm Y-maze, Open Field (OF), and Novel Object Recognition (NOR) tests. At the end, mice were single housed in Metabolic Cages for 5 days to assess physiological parameters and locomotor activity.

RESULTS: We observed distinct age- and sex-dependent differences in the AD phenotype. Compared with younger females, older female 5xFAD mice displayed increased anxiety-like behavior in the OF, altered novel arm entry ratios in the Y-Maze, and reduced performance in the NOR test. Additionally, metabolic cage assessments revealed genotype-dependent divergences in locomotor activity and energy balance in both males and females.

CONCLUSIONS: Together, these findings demonstrate that both sex and age significantly affect AD progression and behavior outcomes, supporting the use of the 5xFAD mouse model to investigate sex-dependent mechanisms in AD pathogenesis. Further research is needed to determine the translational relevance of these findings to human AD.

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Stem Cell Density-dependent Growth characteristics

Mesenchymal stem cells (MSCs) are increasingly being used in regenerative medicine because of their ability to self-renew, proliferate, and differentiate into multiple cell lineages. Adipose-derived stem cells (ADSCs) and bone marrow-derived stem cells (BMSCs) are among the commonly studied sources. Proliferative capacity is a critical factor influencing the expansion of stem cells for therapeutic applications, as it affects the feasibility of generating sufficient cell numbers for cell-based therapies. Comparing the proliferative properties of ADSCs and BMSCs based on cell density is therefore essential for identifying the most effective and practical stem cell source. There have been many past studies on the growth rate of ADSCS and BMSCS. However, how cell seeding density affects proliferation remains unclear. Our aim is to study cell density-related effects on growth of stem cells. We hypothesize that ADSCs exhibit a higher proliferation rate than BMSCs when cultured under identical *in vitro* conditions.

ADSCs and BMSCs were cultured in tissue culture flasks for two weeks prior to seeding into 96-well plates. Cells were plated at low (1000 cells/well), medium (3000 cells/well), and high (8000 cells/well) densities. Cell proliferation was assessed after 1, 3, and 7 days using the Cell Counting Kit (CCK-8) assay to compare the proliferative capacity of ADSCs and BMSCs across varying seeding densities and time points. From the results, both cells showed relatively the same growth rates. However, high density ADSCs and medium density BMSCs proliferated faster than the rest of the sample. In conclusion, although ADSCs and BMSCs exhibit comparable proliferation rates overall, cell seeding density influences the growth kinetics of both cell types.

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"Towards Intoxication Detection via Non-invasive Natural Typing"

Cellphone use over the years has risen to near ubiquitous levels, and with it has risen the possibility of and interest in using phones as diagnostic devices. A person's typing can give insight into their motor coordination and if they are possibly inebriated, providing alternatives to diagnostic methods that rely on specialized machines.

Generally, people have a typing style that, while possessing variation, is fairly consistent. Given this, along with the fact that motor impairment would hinder the coordination required to type, it should be possible to infer whether one is impaired based on the regularity of one's typing. This has been supported by research regarding mobile typing and Parkinson's disease by Giancardo et al. However, the limits of this approach's precision in impairment detection remain unclear, leading to the question of if it can be used for more subtle impairments like that caused by inebriation.

To this end, we propose a set of signal-processed features based on covariance, Euclidean distance, and analysis of errors using the backspace key to capture signs of inebriation-related motor impairment. These features were tested with a population of 5 sober individuals and 21 individuals of varying levels of intoxication, achieving sensitivity/specificity of 0.9236 for the best performing model.

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"Assessment of the Menopause Community Resource Landscape for Black Women in Southeast Louisiana"

BACKGROUND: Black women often experience menopause earlier, for longer durations, and with more severe symptoms than White women. Yet, they face significant barriers to culturally responsive menopause care, including provider bias, limited provider training, and medical mistrust. These barriers, coupled with unmet informational and supportive needs, may lead Black women to seek menopause information and symptom support outside traditional healthcare settings, including through informal social networks and community-based resources. However, little is known about the availability and characteristics of menopause-related resources for Black women in Southeast Louisiana. To address this knowledge gap and complement an ongoing qualitative study exploring Black women's lived experiences and perceptions of menopause, we conducted a companion environmental scan of the menopause resource landscape across Southeast Louisiana.

PURPOSE: To identify and characterize publicly available menopause-related resources for Black women in Southeast Louisiana (LDH Regions 1, 2, 3, and 9) and identify gaps in resource availability, cultural relevance, representation, accessibility, and geographic distribution.

METHODS: A systematic environmental scan was conducted across LDH Regions 1, 2, 3, and 9 using a standardized search protocol and predefined eligibility criteria. Resources were independently screened by two reviewers, with discrepancies resolved by team consensus. Included resources were categorized by type, location, target population, accessibility, menopause focus, cultural tailoring, and representation of Black women.

RESULTS: A total of 73 menopause-related resources were identified across Southeast Louisiana; 63% were menopause-specific. Among identified resources, 32% provided medical and clinical services, 40% provided educational resources, 27% offered community and social support, and 27% provided wellness and lifestyle services. Nearly two-thirds (64%) were national in reach, while few were local (7%) or statewide (5%). Although 55% visibly represented Black women, only 11% met criteria for culturally tailored content, revealing a notable gap between representation and cultural responsiveness. Resources were also geographically uneven, with the greatest concentration in LDH Region 2 and the fewest in Region 3, and limited physical resources in rural areas. Overall, the findings demonstrate that resource availability does not necessarily translate to locally accessible, culturally responsive menopause support for Black women.

CONCLUSIONS: While menopause-related resources exist across Southeast Louisiana, important gaps remain in culturally responsive programming, local resource availability, and equitable geographic access. These findings can inform efforts to expand menopause education, strengthen community-based resources, and improve equitable access to culturally responsive support for Black women in the region.

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"Estimation of Impact of Playground Exposures to Soil Lead on Childhood Blood Lead Levels and IQ"

Background: Lead (Pb) is a neurotoxic heavy metal that persists in soil due to long-term environmental dispersal. Primary sources of soil Pb include historic use of leaded gasoline and paint, and industry and waste incineration. In children, Pb exposure is associated with impaired cognitive function, attention deficit disorders, and antisocial behavior. The 2024 U.S. Environmental Protection Agency (EPA) established a regional screening level (RSL) of 100 ppm to protect children in areas with multiple sources of lead. Exposure to playground soil of 80 ppm for four days per week is associated with an increase in childhood blood lead level (BLL) of 1 µg/dL (90^{th} percentile). Increases in 1 µg/dL of BLL is roughly associated with up to a one-point decrease in IQ. The US Centers for Disease Control and Prevention (CDC) reference level for blood lead level (BLL) is 3.5 µg/dL, however, this is only a population screening value— the CDC acknowledges that there is no safe level of Pb exposure. The aim of this study is to evaluate potential Pb exposures at playgrounds on childhood BLLs and their associated neurocognitive impacts (estimated IQ).

Methods: Soil samples (n=858) were collected at 35 parks in the greater New Orleans area using a grid-based system. Samples were analyzed for Pb with a SciApps X-550 XRF analyzer (LOD-5 ppm). Results were compared to the 2024 RSL guideline of 100 ppm. In addition, the Integrated Exposure Uptake Biokinetic model (IEUBK) was used to estimate the impact of soil Pb exposure. The 95^{th} percentile soil lead level (SLL) was derived for each park to estimate BLLs for children aged six months to seven years (95^{th} percentile BLL). A low dose response relationship from CA OEHHA (2007) was used to derive an estimate of IQ loss for this age group.

Results: The 95th percentile of SLLs across sampled parks ranged from 40 to 1,498 ppm, with modeled increases in park soil-specific BLLs for all age groups ranging from 1 to 9 µg/dL. Using the associations between BLLs and cognitive deficiencies in the literature, these predicted exposure levels corresponded to an estimated 1- to 9-point reduction in IQ. Twenty-five of 35 parks (71%) were associated with an estimated decrease of ≥2 IQ points.

Conclusion: These results suggest that children playing in parks with SLLs exceeding 100 ppm are at risk of developing neurocognitive deficiencies. Remediation of elevated SLLs should reduce Pb exposure, which in turn will reduce the risk of IQ deficits. Future studies should assess the efficacy of different remediation approaches in maintaining long-term SLL reductions.

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"Impact of policy change to soil lead remediation guidelines on childhood lead exposure in New Orleans parks"

BACKGROUND: Legacy lead (Pb), from the historical use of leaded gasoline and paint, lead water pipes, and industrial and waste management activities, remains a major public health concern, as it is a non-degradable metal known for its neuro-developmental effects in children. Its ubiquity throughout New Orleans, LA (NOLA) makes NOLA a high-risk area for children, as they can ingest or inhale Pb from soil while playing at parks. Even low soil lead levels (SLLs) of 100 parts per million (ppm) are associated with elevations in child blood lead levels (BLLs) of 1-12 µg/dL. The US Centers for Disease Control and Prevention (CDC) has set a child BLL reference screening value of 3.5 µg/dL, although the CDC acknowledges that no level of Pb exposure is safe. In 2025, the US Environmental Protection Agency (EPA) increased its regional soil screening level (RSL) for Pb from 100 ppm for children in high-risk areas with multiple Pb sources to 200 ppm and increased its regional removal management level (RML) from 200 ppm to 600 ppm. This study evaluates the potential health impacts of the 2025 policy change.

METHODOLOGY: Soil samples (n=858) from parks (n=35) throughout NOLA were collected in a grid-based pattern and quantified for Pb using a SciApps X-550 X-ray fluorescence analyzer (XRF) (LOD-5 ppm). Parks that would not be remediated under the new policy were identified (parks with 95th percentile SLLs ≥100 ppm and <600 ppm). The 95th percentile SLL in each park was used to estimate the 95th percentile BLLs for children ages 0.5-7 years. The US EPA's Integrated Exposure Uptake Biokinetic (IEUBK) model was used to estimate BLLs, assuming recreational exposures, or 3 days per week at playgrounds, and no other Pb source exposures. Estimated BLLs were compared to the CA Office of Environmental Health Hazard Assessment and American Academy of Pediatrics Pb reference value of 1 µg/dL BLL, as no increase in BLL is safe.

RESULTS: Under the EPA's 2024 and 2025 guidelines, respectively, an estimated 28 parks (80% of 35 parks tested) and 9 parks (26%) would be remediated. Thus, 19 parks (54%) would remain unremediated under the current policy. The estimated 95th percentile childhood BLLs associated with the unremediated parks would increase BLLs of children playing at these parks by 1-4 µg/dL for children of this age group, and by 9-53 µg/dL for pica children (children who consume dirt). Of these 19 parks, 16 (84%) would have an increase in the 95th percentile BLL exceeding 1 µg/dL.

CONCLUSION: The change in EPA Pb soil remediation guidelines would increase children's BLLs at over half of the tested parks at levels ranging from 1-4 µg/dL for non-pica children. These levels are expected to have measurable impacts on children's health based on prior studies. These findings suggest that the current 2025 guidelines pose a greater risk to children.

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"Indolelactic acid (ILA) as a marker of vaginal health"

Background: Indolelactic acid (ILA) is a major metabolite of tryptophan and important for maturing and preventing inflammation in the infant gastrointestinal (GI) mucosa via activation of the aryl hydrocarbon receptor (AhR) pathway. ILA is known to be produced by *Bifidobacter*, a dominant bacterium in the infant GI microbiome. *Bifidobacter* synthesize ILA by metabolizing tryptophan, which is found in high concentrations in breastmilk. Importantly, we recently also found significant concentrations of ILA in the vaginal secretions of some women, and this was strongly associated with the abundance of *Lactobacillus crispatus*, the dominant bacterium in an optimal, healthy vaginal microbiome.
Objectives: Since a dysbiotic, non-optimal microbiome, most commonly caused by bacterial vaginosis (BV), is associated with inflammation and negative reproductive health outcomes, my objectives were to determine if vaginal ILA could (1) be used as a biomarker of vaginal health and (2) play a role in modulating vaginal inflammation.
Methods: Using a de-identified clinical dataset, I set out to determine if there was a significant difference in vaginal ILA concentrations (i) in women with clinically diagnosed BV (Amsel score of 3 or 4) *v* no BV (Amsel score 0-2) (ii) with a positive or negative score for each of the individual Amsel criteria (pH >4.5, vaginal discharge, odor, or presence of Clue cells), and (iii) in the presence or absence of a high proinflammatory cytokine index. Using the HT29 epithelial cell line engineered to express the AhR, I am also determining if, *in vitro*, ILA (i) can block the production of inflammatory cytokines and (ii) if this is via the AhR receptor.
Preliminary Results and Conclusions: With the clinical data provided, I ran Mann-Whitney tests and found that (i) ILA concentration was significantly elevated in samples with positive Amsel criteria for pH>4.5 ($p<0.0001$) and odor ($p<0.0002$) but not for discharge ($p=0.3517$) or Clue cells ($p=0.71$). When all the Amsel criteria were taken into account for a clinical BV diagnosis (Amsel 3 or 4) I also found a strong trend for ILA to differentiate between BV positive and BV negative samples ($p=0.0581$). However, there was no significant difference in ILA concentrations in samples with a high or low inflammatory index. In my *in vitro* experiments, I have so far determined that ILA activates the AhR and its genomic signaling pathway at concentrations found in vaginal secretions. I have also established that the proinflammatory cytokine TNFα can activate my cell line to produce IL8. I am now determining if ILA can prevent IL8 secretion and if this can be blocked with a known AhR antagonist. The results of my study suggest that ILA could be used as a marker of vaginal health, may provide an insight into its role in inflammation.

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Automated Behavioral Analysis Using DeepLabCut for Mouse Behavioral Phenotyping

Background: Attention-Deficit Hyperactivity Disorder (ADHD) is a highly heritable neurodevelopmental disorder characterized by persistent symptoms of inattention, hyperactivity, and impulsivity. Preclinical animal models provide a valuable framework for investigating the genetic and neurobiological mechanisms underlying psychiatric disorders, as behavioral phenotypes represent a measurable output of neural circuit function. Progress into improving our understanding of ADHD is hampered by limitations of existing animal models for ADHD where there is no universal behavioral phenotype for ADHD and most often, the behavioral outcome measures are collected at single timepoints, require subjective observation, and are fragile to behavioral variability. To improve the rigor and translational relevance of preclinical ADHD behavioral studies, continuous, robust, and automated analytical pipelines are required. Such approaches may enhance reproducibility, reduce observer-dependent variability, and facilitate standardized behavioral assessments for comparison across ADHD models. DeepLabCut is a deep learning-based motion tracking platform that enables automatic tracking of user-defined body landmarks from video recordings. We hypothesize that implementation of DeepLabCut will aid in establishing a tractable behavioral phenotype within the *Lphn3* mouse model of ADHD by providing high data throughput, unbiased, and ultimately comprehensive analysis of animal behavior.

Methods: To test our hypothesis, we first set out to establish and develop an automated pipeline, DeepLabCut (DLC), for training and analyzing large-scale behavioral datasets. Behavioral videos were analyzed using DLC within a dedicated Anaconda computing environment. A total of 17 datasets from various behavioral tasks such as novel object recognition and open field tests were processed with approximately one hundred frames extracted from each video. Each video was manually labeled, totaling ~1700 labeled frames for training the pipeline. In each frame, six anatomical landmarks were identified: the mouse's nose, left ear, right ear, center, base of tail, and tip of tail. DeepLabCut2Kinematics was installed within the same environment to analyze locomotive activity using an h5 file obtained during the initial analysis. Through DeepLabCut2Kinematics, we obtained data quantifying distance traveled, percentage of time moving, and mean speed. We proceeded to compare eight videos analyzed by DeepLabCut to the same eight videos scored manually. For a deeper analysis, we took six novel object recognition task (NORT) videos and monitored the percentage of interaction time and compared those against manual scoring for accuracy.

Results: We successfully established a DeepLabCut automated pipeline to train a complex neural network. As far as locomotor activity, our DLC analyzed videos produced similar results to those previously analyzed with Bonsai software. Up until now, NORT interactions were manually counted. Upon implementation of the DLC pipeline, we observed inconsistencies between our manual and DLC behavioral measures and believe implementation of DLC analyses not only improves the objectivity and resolution of existing analyses, but can be used to query more complex behavioral measures relevant to ADHD, and improve the field.

Future Directions: We will continue to optimize and scale our DLC pipeline and behavioral analyses. We plan to next apply DLC to our *Lphn3* behavioral dataset on Lphn3 deletion using a floxed Lphn3 mouseline and crosses to target all neurons (*Syn-Cre*), prefrontal cortex neurons (*PFC-Cre*), glutamate neurons (V*glut2-Cre*), and dopamine neurons (*DAT-Cre)*. This previously collected dataset includes >1200 videos across several tasks used to measure locomotor activity, anxiety-like behavior, spatial recognition memory, working memory, and novelty-induced hyperactivity. This tool is a valuable resource that can improve our understanding of our ADHD model.

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"Changes in Youth Tobacco Product Access After Federal Tobacco 21 Legislation: Evidence from the National Youth Tobacco Survey"

Introduction: The United States has a high prevalence of electronic cigarette (e-cigarette) use among adolescents. In 2025, 7.2% of students ages 9-19 reported using tobacco products, with 5.2% using e-cigarettes. Adolescent tobacco use can cause respiratory illness, lead to addiction, and affect cognitive functions. Because of these health concerns, several policies have been introduced to reduce youth access to tobacco products. Federal Tobacco 21 (T21), enacted in December 2019, raised the minimum legal age to purchase tobacco products from 18 to 21 years. Although previous studies have evaluated the short-term effects of T21 on youth tobacco use, less is known about whether the policy changed how adolescents who continue to use e-cigarettes obtain these products. This study examined the changes in e-cigarette acquisition sources before and after implementation of T21.

Methodology: We conducted a repeated cross-sectional study using data from the National Youth Tobacco Survey (NYTS) between 2018 and 2025. We restricted the sample to adolescents who used and had purchased e-cigarettes within the past 30 days and had valid information on acquisition sources (N=11,281). Survey years were grouped into pre-T21 (2018-2019) and post-T21 (2021-2025). The year 2020 served as a transition year, excluded from regression but retained in temporal trend visualizations. The primary dependent variable was the source of e-cigarette acquisition, categorized as commercial, online, social, or other. The primary independent variable was the policy period (pre- and post–T21), and covariates included age, sex, race, and grade level. Survey-weighted descriptive statistics were used to summarize participant characteristics and acquisition sources. Chi-Square tests compared participants' characteristics by policy period, while survey-weighted regressions examined the associations between the policy period and e-cigarette acquisition sources after adjusting for covariates.

Results: Respondents most reported obtaining their e-cigarettes from social sources (54.2%), followed by commercial purchases (37.2%). Across the study period, the proportion of youth obtaining e-cigarettes through social sources declined, whereas commercial purchasing became more common following implementation of T21. Youth surveyed post-T21 implementation had three times higher odds of obtaining e-cigarettes from a commercial retailer, compared to the youth pre-T21 policy (AOR=3.10, 95% CI: 2.69–3.58, $p<0.001$). Also, post-T21 implementation, youth had 36% lower odds of reporting e-cigarette online purchasing rather than commercial sources (AOR=0.64, 95% CI: 0.48–0.87, $p=0.004$) and 80% lower odds of reporting social acquisition compared to commercial purchases (AOR=0.20, 95% CI: 0.17–0.23, $p<0.001$). However, post-T21 youth had 2.85 times higher odds of reporting "other" for method of obtainment rather than commercial sources (AOR=2.85, 95% CI: 2.13–3.80, $p<0.001$).

Conclusion: These findings found significant changes in where adolescents bought their e-cigarettes after implementation of T21. Results highlight the importance of continued monitoring and enforcement of adolescent e-cigarette acquisition. Future research should assess what "other" sources of e-cigarette acquisition include, and the cause of its increase post-T21 implementation.

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“Maresin-1 prevents alpha-synuclein activation of microglia in vitro and in vivo”

Parkinson's Disease (PD) is characterized by the loss of neurons in the Sustantia Nigra pars compacta (SNpc) and the presence of Lewy bodies, an abnormal aggregation of alpha-synuclein (a-Syn) and other proteins. At the time of diagnosis, patients have lost 30-50% of dopaminergic neurons in the SNpc. By this time, some events preceded the dopaminergic loss for over 10 years before the motor symptoms arose. Anosmia/hyposmia, a proposed prodromal symptom, is observed in 50-90% of the PD patients. Recently, it was observed that Maresin-1 (MaR-1), an omega-3 fatty acid derivative, reduces the dopaminergic cell death in 6-hydroxy-dopamine (6HODA) injection model and decreases the activation of microglial cells. We hypothesize that changes in support cells, such as microglia, precede the death of the dopaminergic neurons in SNpc and Maresin-1 prevents these changes.

To determine the symptoms emerging in the early events before diagnosis, we developed a rat model to study the propagation of a-syn aggregates from the nasal epithelium as proposed in Braak et al. theory of the stages of PD. These rats presented with anosmia/hyposmia 24 weeks post-intranasal delivery of a-syn preformed fibrils as of the Buried Food behavioral test. This time was taken as a starting point to screen for changes in microglia phenotypes. Two months after the appearance of the prodromal symptom, an increased content of α-Syn Phosphorylated in Serine 129 was detected along with a reduced number of dopaminergic neurons. Mar-1 administration rescued dopaminergic neurons from death and improved their overall morphology. We found an increase in microglial inflammasome formation 8 weeks after the first indication of olfactory deficiencies arises. Microglial inflammasome formation was determined by immunocytochemistry targeting ASC polymerization speckles in cells depicting IBA1, a marker of microglial cells. Maresin-1 administered intranasally 24 hours and 1 week after a-syn PFF delivery decreased the formation of inflammasome in microglia, suggesting that the bioactive lipid possessed anti-inflammatory properties.

In conclusion, the loss of smell correlated with the formation of protein aggregates in the SNpc in the animal model. Mar-1 reduced olfactory deficits along with the prevention of inflammasome triggered death of microglial cells to protect neurons in the SNpc. This evidence lays the basis to unveil the mechanisms by which the a-syn adducts spread, leading to the phenotype change of supportive cells that drives the SNpc neurodegeneration. In addition, Maresin-1 can be evaluated as a candidate for treatment of PD during prodromal stages.

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"Comparative RNA-Seq Analysis of Long Interspersed Element-1 (L1) Expression in Two- and Three-Dimensional models and Patient-Derived Triple Negative Breast Cancer (TNBC)"

Long Interspersed Element-1 (L1) is the only autonomously active retrotransposon in the human genome and is frequently reactivated in cancers of epithelial origin, including breast cancer, due to epigenetic dysregulation. However, it remains unclear whether commonly used experimental models accurately reflect L1 mRNA expression in cancer patients. This study compared locus specific L1 expression in 2D and 3D breast cancer cell cultures with patient derived triple negative breast cancer (TNBC) samples to determine how accurately experimental models reflect patient L1 expression.

To address this aim, L1 RNA-Seq alignment approach and L1ABA automated pipelines were used to identify expressed L1 loci from publicly available RNA sequencing datasets of breast cancer cell lines and patients with TNBC tumors. As a quality control, we confirmed that file sizes did not differ significantly among datasets and were not correlated with the number of expressed L1s. While each of the three tested 2D cell culture models (MCF7, Luminal A; HCC1806, TNBC; BT474, Luminal B) expressed at least one hundred L1 loci, only eight L1 loci were expressed in all three cell lines. In 3D models (HCC1806 and BT474 at Day 10 and Day 20), ten expressed L1 loci were shared among all the samples. L1 expression did not significantly differ between the 2D and 3D models, although some L1 loci were uniquely expressed in each model. In contrast, when the identities of L1 loci expressed in samples collected from patients with Grade 2 TNBC were compared to the HCC1806 2D and 3D cell culture models, there was a statistically significant difference in the repertoire of expressed L1s. None of the L1 loci expressed in 2D or 3D models were expressed in any of the three TNBC patient samples.

Our findings establish that although 2D and 3D breast cancer models exhibit similar locus-specific L1 expression patterns, neither accurately captures the heterogeneity of L1 expression observed in human tumors.

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The Impact of Obesity on Prostate Cancer

Introduction: Over the past four decades, the global prevalence of obesity (BMI > 30 kg/m2) has risen dramatically, alongside a steady increase in obesity-associated malignancies. Epidemiological data indicate that excess body weight is associated with an elevated risk of at least 13 distinct cancer types. The impact of obesity on prostate cancer pathogenesis is critical, given that prostate cancer remains the most frequently diagnosed non-cutaneous malignancy in men globally. Despite documented clinical correlations, the precise molecular mechanisms linking obesity to prostate cancer development and racial disparities remain highly complex and poorly understood.

Methods: Proteomic analysis quantified differential protein expression between obese and lean prostate cancer tissue samples from Black and White patients in the Louisiana cohorts. Notably, all patients in this study were untreated and had a Gleason score of 7.0. Identified proteins were mapped to distinct biological pathways, including peroxisomal metabolism, fatty acid and lipid metabolism, intracellular vesicular trafficking, post-transcriptional RNA processing, and mitochondrial bioenergetics. To validate these proteomic findings, histopathological alterations were assessed using Hematoxylin and Eosin (H&E) staining. Additionally, immunohistochemistry (IHC) was used to quantify progression biomarkers associated with obesity-induced prostate cancer, focusing on the lipid metabolism enzymes alpha-methylacyl-CoA racemase (AMACR) and glycerol-3-phosphate acyltransferase 4 (GPAT4).

Results: Proteomic analysis revealed that obesity inundates prostate tissue with lipids, prompting tumors to upregulate the alternative fat-burning HACL1 and lipid-building MCAT pathways. Furthermore, lipid overload stresses mitochondrial energy centers, including COQ8B, coinciding with the downregulation of structural anchors within the Keratin family and EPB41. The data demonstrate that these metabolic alterations are significantly more pronounced within this intermediate-risk cohort and correlate with higher AMACR and GPAT4 expression on IHC.

Conclusions: These findings suggest that obesity accelerates prostate cancer progression in part by driving a distinct lipid-mediated metabolic reprogramming of intermediate-grade tumors.

Limitations: The study used only 16 patient samples, and analysis of larger samples is needed to strengthen these conclusions.

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"Sex-specific Metabolic and Cognitive Differences in a Mouse Model of Alzheimer's Disease. Is Ang IV-IRAP signaling involved?"

BACKGROUND: Alzheimer's disease (AD) is an age-related neurodegenerative disorder that frequently exists with obesity and insulin resistance, comorbidities associated with accelerated cognitive decline and disease progression. Insulin-regulated aminopeptidase (IRAP/AT4R), the proposed receptor for angiotensin IV (Ang IV), regulates glucose homeostasis and neuropeptide metabolism. Emerging evidence suggests that dysregulation of IRAP/Ang IV axis is associated with cognitive impairment and may contribute to AD pathogenesis. We investigated early sex-specific metabolic and cognitive alterations in 5xFAD (Five Familial Alzheimer's Disease) mice. We also explored cortical IRAP expression in this strain to understand whether differences in this Ang IV receptor may be involved in the early development of AD.

METHODS: Five-month-old heterozygous 5xFAD male and female mice underwent baseline metabolic phenotyping, which included a glucose tolerance test (GTT) and an insulin tolerance test (ITT), to assess glucose tolerance and insulin sensitivity, respectively. Cognitive function was evaluated using the Novel Object Recognition (NOR) and Y-maze tests to assess short-term working memory and spatial memory, respectively. Brain tissue was analyzed by immunohistochemistry (IHC) and droplet digital PCR (ddPCR).

RESULTS: Female 5xFAD mice exhibited a trend toward impaired glucose tolerance (AUC: 727.0 ± 90.72 vs. 619.5 ± 28.82) and greater insulin resistance (AUC: 323.1 ± 18.88 vs. 284.5 ± 19.07) compared to males. Male 5xFAD mice showed decreased spontaneous alternation in the Y-maze test compared to females (30.43% vs. 38.40%, $p=0.0453$) but similar total arm entries (28 vs. 27.7, $p>0.05$), indicating impaired spatial memory in males despite equivalent locomotor activity. In the NOR test, however, females demonstrated worse object recognition memory as evidenced by reduced investigation time toward the novel object. This was shown by a reduced ratio of time spent investigating the novel object verses time investigating the familiar object (females: 0.48 ± 0.1; males: 1.25 ± 0.21, $p=0.013$). Additionally, ddPCR demonstrated reduced cortical IRAP mRNA expression in male 5xFAD mice compared to wild-type controls (12,378 vs. 19,062 copies, $p=0.008$). Consistent with these findings, IHC showed reduced cortical IRAP protein expression in male 5xFAD mice compared to wild-type controls as well as increased microglial activation, consistent with enhanced brain inflammation.

CONCLUSIONS: Our findings show distinct metabolic and cognitive differences between male and female 5xFAD mice. Reduced cortical IRAP expression and increased microglial activation in 5xFAD mice suggests that early dysregulation of IRAP signaling may be mechanistic in the neuroinflammatory and cognitive alterations observed in this strain. These findings highlight distinct metabolic and neuropathological trajectories between sexes and support IRAP as a potential therapeutic pathway in AD. Future studies will investigate whether differences in sex-specific cortical IRAP expression exist.

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"Developing a CD36 Inhibitor as a Novel Therapeutic Agent for Triple-Negative Breast Cancer"

Triple-negative breast cancer (TNBC) is the most aggressive subtype of breast cancer and is associated with poor prognosis, high rates of recurrence, metastasis, and limited treatment options due to the absence of estrogen receptor, progesterone receptor, and HER2 expression. Although chemotherapy remains the standard treatment for TNBC, therapeutic resistance and relapse remain a major challenge. Evidence suggests that cancer stem cells (CSCs) contribute to tumor progression, metastasis, and therapeutic resistance through enhanced fatty acid metabolism. CD36 is a transmembrane fatty acid transporter that mediates the uptake of long-chain fatty acids and has been implicated in lipid metabolism, tumor progression, metastasis, and CSC maintenance, making it a promising therapeutic target in TNBC.

In this study, we investigated the effects of a newly developed novel CD36 inhibitor, FRCD36-01 using the human TNBC cell lines MDA-MB-231 and MDA-MB-468 as well as the mouse cell line C0321. Kaplan-Meier analysis demonstrated that elevated CD36 expression is associated with poorer relapse free survival in patients with TNBC suggesting its clinical relevance. We also confirmed higher levels of CD36 protein in mammospheres compared to the adherent cells by Western blot analysis. These results suggested the association of CD36 with cancer stemness. We assessed cell viability using MTT assays, cell proliferative capacity through colony formation assays, apoptosis assays to determine the effects of FRCD36-01 on programmed cell death. We also conducted mammosphere formation assays to assess CSC's self-renewal ability, and functional assays, such as fatty acid uptake experiments using the fluorescent fatty acid analog BODIPY FL C16 to confirm inhibition of CD36 mediated fatty acid uptake by TNBC cells. The results demonstrated that FRCD36-01 significantly reduced cell viability, colony formation ability, fatty acid uptake, and mammosphere formation while promoting apoptosis in TNBC cells. The combinational treatment of FRCD36-01 with chemotherapy drug paclitaxel produced greater inhibition compared to either single treatment suggesting that FRCD36-01 enhanced therapeutic efficacy of chemotherapy. These findings demonstrate that inhibition of CD36-mediated fatty acid uptake with our novel FRCD36-01 would be an effective therapeutic approach to treat TNBC. However, further studies are warranted to identify the detail mechanism of this pathway as well as applicability of FRCD36-1 with immunotherapy to treat TNBC.

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Impact of MFAP5-Overexpressing on Breast Cancer Progression.

Estrogen receptor positive (ER+) breast cancer accounts for 70% of breast cancers, making it the most common subtype of breast cancer. 40% of ER+ breast cancers develop resistance to of endocrine therapy and progress to a more aggressive phenotype. While prior studies suggest alterations to ER expression and activation, to date, the cell-extrinsic mediators of tumor progression remain incompletely understood. Microfibrillar-associated glycoprotein 5 (MFAP5) is a matrix-associated protein linked to tumor invasion, metastasis, and poor prognosis in breast cancer. However, the role of MFAP5 in ER+ breast cancer is not yet defined.

To determine the impact of cell-extrinsic mediator MFAP5 on ER+ breast cancer progression and ER signaling, MFAP5 was overexpressed in the ER+ cell line, MCF-7. We validated overexpression of MFAP5 gene and protein (cell lysate and media) expression in MCF-7-MFAP5 OE cell lines compared to MCF-7 vector control. RNA sequencing results demonstrated alterations to mTOR/AKT and TGF-β signaling pathways, with additional changes observed in ER-mediated genes. Further confirmation studies showed a significant decrease in the ER response gene progesterone receptor (PGR) but no change in ER in MFAP5-OE cells compared to vector control cells. Estradiol (E2) treatment increased PGR expression, suggesting ER function remained.

Overall, our results demonstrate that MFAP5 enhances expression of genes associated with growth and EMT-related signaling pathways, while suppressing PGR. Together, this suggests the ECM protein MFAP5 as a cell-extrinsic mediator of the ER signaling axis. Future work will expand on this to determine if MFAP5 alters the endocrine response in ER+ breast cancer.

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"Protein S Modulates the Hypoxic Response in Pancreatic Cancer Cells"

Objective: Pancreatic ductal adenocarcinoma (PDAC) is the third leading cause of cancer-related mortality in the United States and remains one of the deadliest malignancies due to its aggressive biology, late-stage diagnosis, and limited therapeutic options. Tumor hypoxia is a defining feature of PDAC and promotes metabolic reprogramming, tumor progression, immune evasion, and resistance to therapy. Patients with PDAC also have a markedly increased risk of venous thromboembolism (VTE), which is frequently associated with reduced circulating levels of Protein S (PS), a vitamin K-dependent anticoagulant. While Protein S is well recognized for its anticoagulant function, accumulating evidence from our laboratory suggests that hypoxia suppresses PS expression, raising the possibility that PS also modulates hypoxia-driven signaling pathways. In this study, we investigated whether exogenous Protein S attenuates hypoxia-induced signaling in human pancreatic cancer cells.

Method: Human pancreatic cancer cell lines PANC-1 and MIA PaCa-2 were treated with 150 nM recombinant human Protein S and cultured under normoxic or hypoxic conditions for 8, 24, and 48 hours. Protein expression was analyzed by Western blotting to determine the levels of hypoxia-inducible factor-1α (HIF-1α), hypoxia-inducible factor-2α (HIF-2α), phosphorylated AKT (p-AKT), total AKT, and glucose transporter-1 (GLUT1), using glyceraldehyde-3-phosphate dehydrogenase (GAPDH) as the loading control. Quantitative real-time PCR (RT-qPCR) was performed to assess the expression of hypoxia-responsive target genes.

Results: Exposure to hypoxia markedly increased HIF-1α and HIF-2α protein expression in both PANC-1 and MIA PaCa-2 cells, with the most robust and reproducible induction observed after 8 hours of hypoxic exposure. Treatment with Protein S significantly attenuated hypoxia-induced accumulation of both HIF-1α and HIF-2α in each cell line compared with hypoxia alone, suggesting that Protein S suppresses hypoxia-responsive signaling.
Hypoxia also increased AKT phosphorylation, consistent with activation of pro-survival signaling pathways. In contrast, total AKT and GLUT1 protein levels remained largely unchanged under the experimental conditions. Ongoing RT-qPCR analyses of hypoxia-responsive genes will determine whether the reduction in HIF-1α and HIF-2α protein levels is accompanied by corresponding decreases in the transcriptional activation of downstream target genes.

Conclusion: These findings demonstrate that Protein S suppresses hypoxia-induced accumulation of HIF-1α and HIF-2α in human pancreatic cancer cells, supporting a previously unrecognized role for Protein S as a negative regulator of the cellular hypoxic response. Collectively, these preliminary data suggest that Protein S modulates hypoxia-driven signaling in PDAC and provide a strong mechanistic rationale for future studies to define its molecular mechanisms of action and evaluate its therapeutic potential as a novel target for pancreatic cancer.

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"To What Extent Does Social Environmental Ranking Explain Racial Disparities in Breast Cancer Survival in Louisiana"

INTRODUCTION**:** In Louisiana, NHB women are 40.5% more likely to die from breast cancer than NHW women and are more likely to be diagnosed at younger ages and later stages. NHB patients disproportionately experience adverse social determinants of health that shape the quality of cancer care. The Social Environmental Ranking (SER) is a composite measure of neighborhood-level social and environmental conditions, which may contribute to these disparities. Although SER has been associated with cancer outcomes, its role in racial disparities in breast cancer survival remains poorly understood in population-based studies. Thus, this study assesses differences in overall survival (OS) and cause-specific survival (CSS) across levels of neighborhood social-environmental burden and evaluates the extent to which this burden contributes to the NHB-NHW breast cancer survival disparity in Louisiana.

METHOD**:** We linked de-identified Louisiana Tumor Registry patient records for 6,454 NHB and 13,379 NHW women (>=20) diagnosed with first primary breast cancer between 2016 and 2022 to the 2022 CDC Environmental Justice Index SER scores at the census tract level; death certificate-only and autopsy-only cases were excluded. SER percentiles were grouped into quartiles (Q1=lowest burden, Q4=highest burden). Outcomes were OS and CSS; the primary exposures were race (NHB vs. NHW) and SER. Descriptive statistics, Kaplan-Meier analysis with log-rank tests, and multivariable Cox proportional hazards models were used. Covariates were added sequentially (age and diagnosis year; stage and molecular subtype; then treatment and comorbidity) with SER entered last after each adjustment covariate set to assess its contribution to the NHB–NHW disparity.

RESULTS**:** NHB patients were more likely to reside in high-SER neighborhoods and to present with advanced-stage disease and triple-negative breast cancer. Kaplan–Meier analyses showed significantly poorer OS and BCSS among NHB women and among patients residing in higher-SER neighborhoods (both log-rank $p < 0.0001$). After adjustment for age and diagnosis year, NHB women had significantly higher overall mortality (HR = 1.36, 95% CI: 1.28–1.45) and breast cancer-specific mortality (HR = 1.57, 95% CI: 1.44–1.71) than NHW women. Adjusting for SER attenuated the NHB-NHW disparity by 25% (OS) and 24.5% (CSS) beyond age and diagnosis year, and by 32.9% (OS) and 32.2% (CSS) after further adjustment for stage and subtype. SER continued to attenuate the disparity even after adjustment for treatment and comorbidity.

CONCLUSION**:** Higher neighborhood-level social-environmental burden was independently associated with poor OS and CSS. SER may capture dimensions of neighborhood disadvantage beyond traditional clinical prognostic factors. Incorporating SER into cancer surveillance may improve identification of high-risk populations and inform targeted interventions.

KEYWORDS: Breast Cancer Survival, Health Disparities, Social-Environmental Ranking

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“Spatial Analysis of Cortex–Medulla Interactions in the Mouse Adrenal Gland”

The adrenal gland consists of two functionally distinct regions: the cortex, which produces steroid hormones including cortisol, and the medulla, which contains neuroendocrine chromaffin cells that release catecholamines such as norepinephrine. Although these regions produce different hormones, both contribute to the physiological response to stress on different timescales, suggesting that communication between the cortex and medulla may help coordinate adrenal function. However, the structural relationship between these regions throughout the adrenal gland remains poorly understood.

The goal of this study was to examine the spatial organization of the adrenal cortex and medulla in mouse adrenal tissue and determine whether consistent patterns of interaction are present across different regions of the gland. Serial tissue sections spanning from the peripheral tip of the adrenal gland, where the medulla is absent, to the central sections containing the largest medullary area were analyzed to assess how the cortex and medulla are organized relative to one another.

Mouse adrenal tissue was stained immunohistochemically using an antibody against tyrosine hydroxylase (DAB), the fluorescent dye LipidSpot 488, and 4′,6-diamidino-2-phenylindole (DAPI) to distinguish the medulla and cortex while searching for unusual tissue architecture. TH staining was used to label chromaffin cells (mainly located in the medulla), LipidSpot 488 labeled the lipid-rich cortex, and DAPI was used to visualize cell nuclei. Images from serial sections were compared to examine changes in cortical and medullary organization throughout the gland. Initial findings indicate there are regions where the inner cortex and tge medulla interdigitate providing a potential anatomical site for zonal communication.

Characterizing the spatial relationship between the adrenal cortex and medulla provides a foundation for understanding how these functionally distinct regions may interact. Identifying consistent organizational patterns across serial sections may offer insight into mechanisms that coordinate rapid catecholamine release from the medulla with slower steroid hormone signaling from the cortex and may guide future studies investigating functional communication within the adrenal gland.

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"Investigating the Role of Inducible Nitric Oxide Synthase in Gut Dysmotility and Inflammation in Cystic Fibrosis"

Background: Cystic Fibrosis (CF) is an inherited disease caused by mutations in the Cystic Fibrosis Transmembrane Conductance Regulator (CFTR) gene. Intestinal inflammation and obstruction constitute the major manifestations of the disease. The underlying mechanisms remain poorly understood. We hypothesize that nitric oxide (NO), produced by inducible nitric oxide synthase (iNOS, encoded by the NOS2 gene), causes smooth muscle relaxation, which contributes to CF intestinal obstruction.

Methods: To test the hypothesis, CF mice were treated with the selective iNOS inhibitor GW274150 or control, and gastrointestinal transit was measured using a gut transit assay. Based on the pharmacological findings, *Nos2* and *Cftr* double gene-knockout mice were generated by crossbreeding *Nos2*$^{+/-}$ mice with *Cftr*$^{+/-}$ *mice*. Multiple experiments were conducted from these animal models, including polymerase chain reaction (PCR), Griess Reagent Assay (GRA), and intracellular iNOS immunostaining. First, PCR genotyping was performed to identify *Nos2*$^{+/+}$*Cftr*$^{-/-}$ and *Nos2*$^{-/-}$*Cftr*$^{-/-}$ mice. Second, a GRA was performed to determine NO production under lipopolysaccharide (LPS)-stimulation and conditions of no stimuli or treatment of GW274150. Finally, intracellular iNOS immunostaining and flow cytometry were used to determine iNOS expression in macrophages and neutrophils.

Results: Pharmacological inhibition of iNOS with GW274150 significantly shortened gastrointestinal transit time in CF mice compared with vehicle-treated controls, suggesting improved intestinal motility. After crossbreeding, mouse tails were cut for PCR-genotyping using specific primers to *Nos2* and *Cftr. G*el electrophoresis of the PCR amplicons revealed the desired *Nos2* and *Cftr* double knockout (*Nos2*$^{-/-}$*Cftr*$^{-/-}$*)* or the corresponding control (*Nos2*$^{+/+}$*Cftr*$^{-/-}$*)* mice. Leukocytes from both genotypes were isolated, cultured, and stimulated with LPS, and the supernatants were subject to nitrite measurement by GRA. Results show that the *Nos2*$^{+/+}$ *Cftr*$^{-/-}$ cells produced high levels of NO. Selective iNOS inhibitor GW274150 abolished such NO production. Moreover, the *Nos2*$^{-/-}$*Cftr*$^{-/-}$ cells produced little NO. Intracellular immunostaining of iNOS and flow cytometry demonstrate that LPS stimulation augmented iNOS expression in the control cells, but not *Nos2*$^{-/-}$*Cftr*$^{-/-}$ cells. Treatment of the control cells with GW274150 decreased frequencies and lowered iNOS expression in the macrophages/neutrophils.

Conclusion: These findings support a role for iNOS-derived NO in CF-associated gut dysmotility. We established a iNOS gene-knockout in CF mouse model. With this model we can further determine the role of iNOS in intestinal inflammation/obstruction. Specific iNOS inhibition could be beneficial in restoring gut motility and mitigating inflammatory effects in CF patients.

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The Cognitive Vaping Gap: Examining Past-Year Quit Attempts Among Youth Electronic Vapor Users with and Without Cognitive Difficulties

BACKGROUND: In 2021, 18.0% of U.S. youth (9-19+) vaped (i.e., using electronic cigarettes). Nicotine exposure during adolescence may affect physical health and cognitive functioning. Although quitting can reduce these risks, nicotine dependence and withdrawal may make cessation difficult. Limited research has examined whether youth who report serious difficulty concentrating, remembering, or making decisions because of a physical, mental, or emotional condition are more likely to attempt quitting e-cigarettes. This study addresses that gap by comparing the odds of past-year e-cigarette quit attempts among youth with and without these cognitive difficulties.

METHODS: We conducted a quantitative cross-sectional study using 2025 National Youth Tobacco Survey (NYTS) data. The study included adolescents who reported current e-cigarette use in the past 30 days (N=1,259) with valid responses to the quit attempt question and the question: "*Because of a physical, mental, or emotional condition, do you have serious difficulty concentrating, remembering, or making decisions?*" Survey-weighted descriptive statistics summarized participants' sociodemographic characteristics. Chi-square tests compared participant characteristics by number of quit attempts, while survey-weighted ordinal regression models examined the association between serious difficulty and quit attempts, adjusting for sociodemographic and tobacco related covariates. Multicollinearity among independent variables was assessed using correlation coefficients.

RESULTS: Among 1,259 current e-cigarette smokers, 26.5% reported no quit attempts, 43.1% reported 1-5 quit attempts and 31.8% reported ≥ 6 quit attempts. In bivariate analyses, quit attempts differed significantly by age (p=0.005), sex (p=0.007), race/ethnicity (p=0.020), education level (p=0.006) and whether they use combustible tobacco products (p=0.043). In adjusted ordinal regression model, male participants had 43% higher odds of reporting more quit attempts than females (AOR=1.43, 95% CI: 1.06–1.93), whereas Hispanic/Latino had lower odds o reporting more quit attempts compared to white participants AOR=0.63, 95% CI: 0.47–0.85). Concentration difficulties (AOR=1.02, 95% CI: 0.82–1.26) and mental health symptoms (AOR=1.04, 95% CI: 0.75–1.43) were not significantly associated with quit attempts after adjustment. There was no difference in quit attempts by mental health symptoms or concentration difficulties.

CONCLUSION: Among youth who currently use e-cigarettes, serious difficulty concentrating, remembering, or making decisions was not associated with quit attempts after adjusting for sociodemographic and tobacco-related factors. Future research should examine demographic differences in adolescent e-cigarette cessation.

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"Characterization of astrocytes phenotype in models of alpha-synuclein toxicity"

Although Parkinson's disease (PD) is clinically recognized by its motor symptoms, pathological changes begin years before diagnosis with the accumulation of alpha-synuclein (α-syn)-containing Lewy bodies and progressive loss of dopaminergic neurons causing neurodegeneration. Increasing evidence suggests that dysfunction of non-neuronal support cells contributes to these early disease processes. Astrocytes are essential for maintaining neuronal homeostasis, and their loss of protective function has emerged as a potential driver of PD progression.

It has recently been shown that Maresin-1 (Mar1), an omega 3-derived specialized pro-resolving lipid mediator, reduced dopaminergic neuron loss in a 6-hydroxydopamine (6-OHDA) model of PD and preserves the homeostatic phenotype of astrocytes in vitro. We hypothesize that astrocyte senescence precedes dopaminergic neuron degeneration in the SNpc and that Mar1 prevents these pathological changes, thereby preserving astrocyte homeostasis during PD progression.

To investigate early astrocyte dysfunction during PD progression, we employed a rat model based on Braak's hypothesis in which α-syn preformed fibrils (PFFs) were administered intranasally to initiate pathology through the olfactory (OF) pathway. Buried Food behavioral test supported the idea of development of OF deficits prior to substantial dopaminergic neuron loss. Brain sections from the OF bulb, substantia nigra (SN), and prefrontal cortex (PFC) were analyzed using immunohistochemistry and confocal microscopy to evaluate astrocyte phenotype, senescence, and α-syn pathology. Astrocyte senescence was assessed by β-galactosidase activity colocalized with GFAP-positive cells. Because senescent astrocytes lose their homeostatic and phagocytic functions, we evaluated changes in astrocyte morphology and α-syn clearance. Mar1 treatment reduced astrocyte senescence and preserved a more homeostatic astrocyte phenotype.

In conclusion, OF dysfunction correlated with α-syn pathology and astrocyte senescence during the early stages of PD in this animal model. Mar1 reduced OF deficits and preserved a homeostatic astrocyte phenotype, suggesting that astrocyte dysfunction is an early contributor to PD progression. These findings support the growing recognition of astrocytes as key mediators of neurodegeneration rather than passive support cells. Targeting astrocyte senescence may therefore represent a promising therapeutic strategy for slowing PD progression during its prodromal stages.

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"Evaluation of RhoA Activation in RSK2-Mediated Invasion in Pancreatic Cancer."

Pancreatic Ductal Adenocarcinoma (PDAC) is one of the most aggressive and deadliest forms of pancreatic cancer, covering around 90% of all cases. Mutations in the oncogene KRAS are amongst the earliest and common genetic changes in pancreatic cancer and contribute to the overall development and progression of the disease. This is reflected by behaviors such as increased cellular invasion, metastasis, and cell proliferation. This creates the importance of investigating therapeutic targets within the KRAS signaling cascade, such as RSK2. Previous studies have demonstrated that RSK2 promotes aggressive cancer cell behaviors by regulating the RhoA signaling pathway. RhoA functions as a molecular switch to control cytoskeleton remodeling, cell motility, and division. Therefore, investigating RhoA activation and downstream signaling will help elucidate how RSK2 contributes to the invasiveness of PDAC. My experiment utilized the Miapaca2 cell line that had been transfected with anti-RSK2 shRNA and non-targeted shRNA to examine the hypothesis that RSK2 inhibition reduces RhoA activation, potentially contributing to lower rates of cellular invasion. My approach to test this was immunocytofluoresence to quantify overall RhoA-GTP (active RhoA) expression, and western blots to quantify activation of targets that are downstream of RhoA (pMYPT1 and pMCL2). Following confirmation of efficient RSK2 knockdown by Western blot analysis, fluorescence imaging revealed a stronger RhoA-GTP signal in RSK2-knockdown cells. The phosphorylation of downstream RhoA signaling targets was increased following RSK2 depletion. These findings suggest that RSK2 negatively regulates RhoA activity in MiaPaCa-2 cells, and that RSK2 loss enhances RhoA-GTPase activation.

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"tES Induces Frequency Dependent Patterns of Neuronal Activation"

Transcranial electrical stimulation (tES) is an emerging non-invasive technique for modulating neural circuit activity and behavior. We previously demonstrated that 6 Hz tES reduces fear memory retention and enhances fear extinction. To better understand the cellular mechanisms underlying these behavioral effects, we examined frequency-dependent neuronal activation across the cerebellar cortex following stimulation.

Adult male wild-type mice received either sham stimulation or tES at 6 Hz or 40 Hz. Ninety minutes after stimulation, brains were collected, fixed, cryoprotected, sectioned, and processed for immunohistochemistry. Sections containing cerebellar vermis lobules V–VI were stained for c-Fos, calbindin, and neuronal nitric oxide synthase (nNOS), counterstained with DAPI, and imaged using confocal microscopy. Quantitative fluorescence analysis was used to compare neuronal activity across the granule cell layer, Purkinje cell layer, and molecular layer.

Compared with sham controls, 6 Hz stimulation significantly increased c-Fos immunoreactivity throughout the granule cell layer, Purkinje cell layer, and molecular layer. In contrast, 40 Hz stimulation produced a more restricted pattern of activation, with increased c-Fos expression primarily in the outer molecular layer. Direct comparisons between stimulation frequencies showed that 6 Hz elicited significantly greater c-Fos activity in the outer molecular layer and Purkinje cell layer than 40 Hz stimulation, while only a trend toward greater activation was observed in the granule cell layer. Notably, these frequency-dependent activation patterns were not confined to lobules V–VI, suggesting that different stimulation frequencies recruit distinct neuronal populations throughout the cerebellar cortex.

Together, these findings demonstrate that tES frequency shapes the spatial pattern of neuronal activation within the cerebellar cortex. We propose that these differences are driven primarily by the biophysical properties of brain tissue, with lower-frequency stimulation penetrating more effectively into deeper cerebellar layers and higher-frequency stimulation preferentially influencing more superficial regions. Ongoing analyses will identify the specific neuronal populations recruited by each stimulation frequency and further define how these circuit-level effects contribute to the modulation of fear memory.

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"Learning experience shapes neuron structure and gene expression in the song circuit during the critical period"

BACKGROUND: Neurodevelopmental disorders such as Autism Spectrum Disorder affects many individuals across the globe. These individuals often experience impairments in cognition, social interaction, and language. Studying the song development in juvenile zebra finches serves as an excellent model to study language development in humans as zebra finches have a well-developed and similar vocal learning process to humans. In the zebra finch's brain, we study Area X, an avian homolog of the basal ganglia of mammals. To understand the plasticity of the song circuit in response to learning experiences, we examined neuronal structure and the expression of the FOXP2 gene of zebra finches at different ages.

OBJECTIVES: This study aims to identify morphological changes of neurons and FOXP2 gene expression in Area X in response to the learning environment of zebra finches.

METHODS: A previous lab member prepared virus-labeled neurons and used immunohistochemistry to stain the brain sections of juvenile zebra finches at 45, 60, and 100 days (d) of age. A confocal microscope was used to image the samples. I used the filament function of Imaris 9.2.1 to measure dendrite length, spine density, spine length, and spine head volume. OlyVIA was used to open and convert the confocal images into Tagged Image File Format (TIFF). ImageJ was used to quantify the intensity of FOXP2.

RESULTS: Four 60d birds were analyzed for neuronal structure. Group 1, consisting of two birds, had an average dendrite length of 36.1 um while Group 2 was 41.6 um. Group 1 had an average spine density of 1.50 spines per 10 um of dendritic length and Group 2 measured to be 2.39 per 10 um of dendritic length. Group 1's average spine length was 1.09 um and Group 2's measurement was 1.37 um. The average spine head volume of Group 1 was 0.036 um^3 and Group 2 measured to be 0.020 um^3. Next, comparing the FOXP2 gene expression in Area X, the 45d bird had an average mean gray value of 602.2 while the 60d and 100d birds had a value of 950.2 and 411.9, respectively. Outside Area X, the birds had an average mean gray value of 391.1, 387.6, and 296.4 for the 45d, 60d, and 100d birds, respectively.

CONCLUSIONS: Because this is a double blind study and our data is preliminary, we will continue to analyze more dendrites and FOXP2 gene expression to collect more data and have a higher statistical power. Ultimately we aim draw a conclusion on the effect of a song tutor on neuron structure and the regulation of the FOXP2 gene.

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Alcohol-Induced Cardiac Remodeling: An Age-Dependent Vulnerability

Background: Alcohol misuse is a persistent public health concern with increasing prevalence, particularly among older adults, placing this population at growing risk for alcohol-related morbidity. Alcohol-related cardiomyopathy is characterized by progressive myocardial functional deterioration, yet the drivers of this pathomechanism remain poorly understood.

Objectives: This study investigated the morphological and functional impact of chronic alcohol misuse by employing a chronic-*plus*-binge alcohol feeding paradigm in adult and aged mice to better understand the progression of alcohol-related heart disease and potential real-world implications for human cardiac health.

Methods: Male and female adult (12-week) and aged (72-week) mice were subjected to a 30-day Lieber-DeCarli chronic-*plus*-binge alcohol feeding regimen. Alcohol-fed groups received a liquid diet containing 5% ethanol, supplemented by oral ethanol binges (5 g/kg) on days 10 and 30. Control mice received an isocaloric maltose-dextrin control diet in combination with isocaloric liquid gavages. Cardiac function was evaluated via terminal invasive left ventricular pressure-volume catheterization. Cardiac tissue weights were measured and normalized to tibia lengths. Histological analysis quantified myocyte cross-sectional area and perimeter in transverse-oriented cardiac cross-sections.

Results: Hemodynamic assessments indicated that alcohol exposure significantly impaired left ventricular load- and heart rate-independent indices of systolic function, regardless of age. Alcohol-fed aged mice, however, showed significantly increased heart weight normalized to tibial length. Furthermore, histological analysis documented significantly increased myocyte cross-sectional area and perimeter in alcohol-exposed aged mice compared to age-matched controls or young mice, suggesting potential cardiac remodeling and a hypertrophic response following chronic-*plus*-binge alcohol feeding.

Conclusions: These findings demonstrate that chronic-*plus*-binge alcohol exposure may act as a driver of myocardial hypertrophy, particularly in aged mice, which may contribute to impaired cardiac performance. Our data indicates that as the heart ages, it experiences an increased vulnerability to alcohol-induced structural changes, which may affect hemodynamics. Overall, by identifying these vulnerabilities, this study sheds light on the importance of alcohol-consumption-related risks to cardiovascular health throughout aging.

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mir-206 expression in plasma-derived extracellular vesicles in children with cerebral palsy

BACKGROUND: Cerebral palsy (CP) is a non-degenerative neurological disorder that leads to muscular dysfunction over time, causing decreased muscle size and function such as a decrease in contractility, increase in stiffness and fibrosis, and increase in muscle dysfunction. Skeletal muscle differentiation (myogenesis) occurs when satellite cells become activated to proliferate myoblast stem cells that differentiate into myotubes and later fuse forming muscle fibers. Micro-RNA (miRNA) is a small non-coding RNA molecule that regulates gene expression by primarily binding to 3' UTR (untranslated region). Muscle-enriched miRNAs, such as miR-206, act as a "post-transcriptional switch" that can push skeletal muscle cells from a proliferative to a differentiated state, causing an increase in myotube size and fusion by targeting anti-differentiation genes. Delivering exogenous mir-206 using plasma from EVs (extracellular vesicles) can improve differentiation, thus improving muscle health in CP. An initial step is to optimize ddPCR to determine the endogenous miR-206 levels in plasma-derived EVs.

OBJECTIVE: The purpose of this study was to assess miR-206 expression in plasma derived EVs in children with CP and identify predicted targets of miR-206.

METHODS: Blood was collected from 7 participants with CP between ages 6-25 years. EVs were isolated from plasma (500, 250, 175, and 100 µl) using the ultracentrifugation method. RNA was extracted from both isolated EVs and the EV depleted plasma using the miRNeasy Micro Kit, and 10ng of RNA was then reverse transcribed into cDNA. The expression of miR-206 was determined in the plasma EVs using ddPCR (Digital Droplet PCR). Specific target genes of miR-206 were identified using databases TargetScanHuman, miRTARGET, and miRDB. Cut off score for miRTARGET and TargetScanHuman data was <50, and mirDB was n=50. DAVID Bioinformatics was used to identify the top 10 targets, and common target genes from the three databases. Subcategories used were "KEGG PATHWAY", "UP KW CELLULAR COMPONENT", "HPA NORMAL TISSUE", "HPA NORMAL TISSUE CELLTYPE", "GAD DISEASE", and "OMIM DISEASE" to determine common target genes among the 3 databases and the functions of those genes.

RESULTS: From DAVID Bioinformatics in the top 10 most significant genes from the 3 databases, TAGLN2 (transgelin 2) was one of the common genes, and its function was aligned with the cerebral cortex. KCNIP3 (potassium voltage-gated channel interacting protein 3, calsenilin) was an additional common gene which aligned with seminal vesicles. PAX7 (paired box 7) functions in maintaining skeletal cells in proliferative states, preventing myogenic differentiation.

SUMMARY: We have identified target genes of miR-206, and future studies will determine the functional relevance of these genes in the skeletal muscle differentiation of children with CP.

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BACKGROUND: Pancreatic Ductal Adenocarcinoma (PDAC) is the most common and aggressive form of pancreatic cancer, originating from the cells lining the pancreatic ducts. The tumor growth can obstruct adjacent structures, damage the pancreas, and metastasize to other organs. PDAC's deep anatomical location contributes to late diagnosis, resulting in less than 13% of patients surviving beyond 5 years.[1] Understanding the tumor microenvironment (TME) is crucial for characterizing PDAC's complex biology. Matrix-Assisted Laser Desorption and Ionization Mass Spectrometry Imaging (MALDI-MSI) enables spatial visualization of biomolecules such as lipids, N-glycans, and peptides within tissue samples without the need for chemical labeling. Spatial Omics techniques facilitate the identification of molecular distributions within the tissue, allowing researchers to correlate specific biomolecules to their exact locations. This study aims to use MALDI-MSI to spatially map lipids, N-glycans, and peptides in PDAC tissues to identify molecular patterns and potential biomarkers associated with tumor regions, providing insights into the TME.

METHODS: Fresh frozen PDAC tissue from the KPC mouse model was cryosectioned and prepared for sequential MALDI-MSI analysis. A time-of-flight (TOF) mass spectrometer was used to sequentially profile lipids, N-glycans, and peptides on the same tissue section. Appropriate matrix application, enzyme digestion, and matrix removal steps were performed between analyses to preserve sample integrity. Data were acquired at a spatial resolution of 100 µm. Molecular ion images were generated, allowing the mapping of distinct biomolecules to specific tissue regions and enabling comparison of molecular profiles across different tumor areas, all performed on a single slide to maintain spatial context.

RESULTS & DISCUSSION: After mapping lipids, N-glycans, and peptides within the PDAC tumor microenvironment on the same tissue section to preserve spatial information, using the combination of previously studied multi-omic features, tissue slices were segmented into three distinct regions using the k-means algorithm. One of these regions is representative of the interface between the PDAC tumor and healthy tissue, and the other two regions are representative of the heterogeneous PDAC TME. For instance, the intensity of a glycerophospholipids(PC 30:4) is significantly higher in the interface TME region. Conversely, the intensity of PC 34:0 shows up more strongly in the two internal heterogeneous TME regions. More examples of these signature biomolecules used to define the TME regions will be explored in the poster. This exploratory study highlights the feasibility of profiling multiple biomolecular classes from the same tissue slice to gain a better understanding of PDAC while minimizing spatial mismatches between different modules. Future research aims to enhance spatial resolution to 20µm for more detailed molecular analysis, enabling us to investigate the profiles of TME regions by segmenting them into smaller, more precise areas.

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"Optimization of an *in vitro 3D* method for studying modulators of the immunosuppressive tumor microenvironment for enhancing the response to immunotherapy"

Purpose: This study aims to establish physiologically relevant *in vitro* models of immunosuppressive tumor microenvironment (TME) for multiple myeloma (MM) and triple-negative breast cancer (BC) using three-dimensional (3D) multicellular coculture systems incorporating immunosuppressive and inflammatory myeloid cells, including M2-polarized THP-1 macrophages and low-density neutrophils (LDNs) with RPMI-8226 (MM) or MDA-MB-231 cells (BC). These models will provide a platform to investigate the mechanisms by which myeloid cells promote tumor-associated immune suppression and to evaluate strategies that overcome myeloid-driven immunosuppression, thereby enhancing the efficacy of cancer immunotherapies**.**

Methods: Differentiated THP1 into macrophages using phorbol 12-myristate 13-acetate (PMA) were polarized into immunosuppressive-like phenotype (M2) with recombinant human IL4 and IL13 cytokines and cancer cells-derived conditioned media (CC-CM). Primary LDNs from peripheral blood were stimulated with visceral adipose tissue-conditioned media (VAT-CM). Fluorescent labeled myeloid M2-THP1 and LDNs. Activation and M2 markers (CD206, CD163, PD-L1, B7-H4, HLA-DR) were evaluated by flow cytometry on alive cells. Collagen-embedded 3D spheroids from MDA-MB-231 and RPMI-8226 cells were formed in ultra-low-attachment (ULA) 96-round-well-plates and cocultured with different ratios and treatments of myeloid cells. Spheroid size over time and infiltration of myeloid cells was tracked using the Live-Cell Imaging Incucyte SX3.

Findings: Coculture with M2-THP1 macrophages enhanced the growth of 3D spheroids generated from RPMI-8226, with the greatest increase observed when macrophages were exposed to tumor cell-conditioned medium, compared with spheroids cultured alone or with M2-polarized THP1 macrophages in the absence of conditioned medium. Similarly, MDA-MB-231 spheroids exhibited increased size when cocultured with M2-polarized THP1 macrophages, regardless of the presence of conditioned medium, relative to tumor spheroids cultured alone. Notably, coculture with VAT-CM-activated LDNs produced a marked acceleration of MDA-MB-231 spheroid growth over time. This tumor-promoting effect was selective for malignant cells, as VAT-CM-activated LDNs did not alter the growth of spheroids generated from the non-malignant mammary epithelial cell line 184A1.

Conclusion: We were able to replicate an immunosuppressive and inflammatory TME to evaluate tumor growth *in vitro*. Further optimization is fundamental for evaluating the effect of therapeutic modulation of the myeloid phenotype on spheroid growth and response of cellular immunotherapy.

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"Characterization of Ependymal Cells and CSF-Contacting Neurons in the Cerebral Aqueduct"

BACKGROUND: Ependymal cells line Cerebral Spinal Fluid (CSF) filled brain ventricles and the spinal cord central canal forming a barrier between CSF and neural tissue while also facilitating CSF circulation. CSF-contacting neurons (CSF-cNs) are interspersed within and around the ependymal layer and possess an axon extending into the CNS parenchyma, allowing them to detect changes in CSF composition and flow. A novel population of prodynorphin (pDyn)-expressing ependymal cells was recently found to be closely associated with kappa opioid receptor (KOR) expressing CSF-cNs. Dynorphin (Dyn) is the endogenous ligand for KOR. Dyn/KOR signaling in the spinal cord regulates injury-induced ependymal proliferation into astroglia. Dyn/KOR signaling in the brain regulates stress and alcohol withdrawal (WD). We have identified abundant pDyn+ cells surrounding the midbrain cerebral aqueduct suggesting that the cerebral aqueduct represents an unexplored ventricular niche.

OBJECTIVE: To characterize pDyn-expressing ependymal cells and CSF-cNs around the midbrain aqueduct and to determine if the architecture between the two cell populations parallels that in the spinal cord. Additionally, this study will investigate whether alcohol withdrawal alters ependymal cell proliferation and pDyn expression.

METHODS: Male and female *Pdyn-Cre;Ai14* mice, which express tdTomato reporter in pDyn+ cells, or male C57BL/6J mice were used for all experiments. CSF-cNs were identified using immunohistochemistry (IHC) for Pkd2l1, intracerebroventricular (ICV) AAV2-CAG-GFP injection, and cholera toxin B (CTB) injections that retrogradely trace CSF-cNs. RNAscope was performed on fresh frozen brain sections using probes for ependymal markers *Foxj1*, *pDyn*, and *Sntn*. Colocalization between CTB-labeled CSF-cNs and *pDyn* and/or *Oprk1* was determined. To assess cell proliferation after alcohol injections, mice received EdU injections after they received an ethanol/saline injection for 4 days followed by a 24 hr withdrawal. Mice were sacrificed 4hrs later during WD, and brains were processed for EdU detection.

RESULTS: *Foxj1* labeled all ependymal cells surrounding the aqueduct, and most co-expressed Sntn, indicating mature ependymal cells. pDyn+ cells were enriched along the ventral ependymal lining. Pkd2l1+ and CTB-labeled CSF-cNs were closely associated with pDyn+ cells. CTB-labeled neurons also expressed *Oprk1*, consistent with the spinal cord central canal. No EdU+ cells were detected after ethanol withdrawal, although pDyn expression increased in ependymal cells.

CONCLUSION: We identified a novel population of mature pDyn-expressing ependymal cells surrounding the cerebral aqueduct that are near *Oprk1*-expressing CSF-cNs, suggesting Dyn released into the CSF may regulate ventricular stress signaling. This establishes the cerebral aqueduct as a previously unrecognized dynorphin sensitive ventricular niche and lays the groundwork for future studies on Dyn/KOR signaling in ependymal cells in disease states.

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Associations Between Five-year Cancer Survival, Natural Hazard Risk, Community Resilience, and Social Vulnerability in Louisiana

Cancer survival is influenced by complex interactions among many biological, clinical, social, and environmental factors. Five-year cancer survival refers to the percentage of cancer patients who remain alive for at least five years after their diagnosis, which is commonly utilized as a measure of cancer prognosis. Survival rates vary based on many factors, including climate hazards, socioeconomic status, access to healthcare, and geographic location. Extreme climate events (e.g., hurricanes, tornadoes, and extreme heat, etc.) can disrupt healthcare resources, damage public infrastructure, and induce population displacement, contributing to barriers in treatment.

Associations among five-year cancer survival, aggregated annual losses attributed to climate hazards, social vulnerability, and community resilience across Louisiana parishes were studied. Parish level Cancer Survival Rates were obtained from Louisiana Tumor Registry (LTR), Community Resilience (CR) and Expected Annual Loss (EAL) data were sourced from the FEMA National Risk Index, while Social Vulnerability Index (SVI) were acquired from the CDC. The Ordinary Least Squares (OLS) regression model analyzed the associations. Global Moran's I analysis of the model residuals was conducted to determine if spatial autocorrelation was present across Louisiana parishes.

Significant associations (F-test, $p<0.001$) explained 52.3% ($R^2=0.523$) of the variation in cancer survival rates. No significant spatial autocorrelation was observed among the residuals ($I=0.027$, $p=0.580$). Parishes with negative residuals had lower observed cancer survival rates than predicted by the model.

The limitations of this study include researching stratified annual loss to climate hazard types and their association with poorer survival outcomes. Additionally, specific cancer types were not researched, suggesting that the model is not able to account for specific cancer survival rate associations. This highlights the need for further research into specific annual loss attributed to hazard types or pathways with stratified cancer survival. Communities identified by this model with lower-than-expected cancer survival rates may represent priority areas for public health interventions and resource allocation to improve cancer survival rates.

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"Post-mortem identification of Kaposi Sarcoma-Associated Herpesvirus reservoirs despite seronegative status"

Background

Kaposi Sarcoma-Associated Herpesvirus (KSHV) undergoes a biphasic lifecycle in which a latent infection is established, followed by periodic lytic reactivation. KSHV is endemic to areas such as sub-Saharan Africa, where KSHV is primarily transmitted during childhood via saliva. A major risk occurs in those co-infected with HIV, where Kaposi Sarcoma (KS), a vascular endothelial cancer, becomes a major cause of mortality in these individuals. The ability of KSHV to undergo latency allows the virus to establish reservoirs which can reactivate and mediate KS occurrence upon immune dysregulation. Although previous studies in Zambia have identified KSHV reservoirs within the central nervous system (CNS) of HIV positive adults and documented sero-reversion in children, it remains unclear whether adults with undetectable KSHV antibodies from endemic areas, in spite of being seronegative can also harbor the virus, and where are the potential reservoir sites.

Methods

An extensive list of post-mortem tissue samples was collected from autopsy cases and processed into Formalin Fixed and Paraffin Embedded (FFPE) blocks by our collaborator in Zambia. Sociodemographic information, such as the cause of death, age, and sex, were documented at enrollment. HIV-1 serostatus was determined using HIV-1 Rapid Test, and KSHV serostatus was determined by an in-house immunofluorescence assay. The presence of KSHV in the tissues was determined by PCR and southern blot. The positive tissues' viral loads were determined by digital PCR against KSHV LANA and ORF26 genes. Immunohistochemistry against LANA protein was used to localize infected cells in positive tissues that had highest viral copy numbers.

Results

Here we analyzed 62 tissue blocks from a 36-year-old, KSHV and HIV-1 seronegative female who died of Pontine Hemorrhage due to hypertension. We were only able to analyze one case due to the time limits that I was able to work on. We detected KSHV in multiple anatomic sites including the CNS, oral respiratory tract, lymph nodes, ovaries, adrenal glands and the pancreas. The CNS tissues; choroid and meninges, had relatively the highest copy numbers for both LANA and ORF 26.

Conclusion:

Detection of KSHV reservoirs in a KSHV seronegative individual, highlights that antibody testing may underestimate KSHV prevalence as well as presents a risk in transplant patients, where an infected seronegative donor could still transmit KSHV to a recipient, who is at an increased risk of developing KS upon transplant.

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Evaluating the Association Between Alcohol Misuse and Alcohol Treatment Utilization in the New Orleans Alcohol Use in HIV (NOAH) Study

Alcohol misuse is common among people with HIV (PWH) and is associated with poor health outcomes, including decreased adherence to antiretroviral therapy and increased disease progression. Although effective alcohol treatment programs are available, many people with alcohol use disorder do not receive treatment. Our study objective was to determine the prevalence of hazardous alcohol use among people with and without HIV and the utilization of alcohol treatment programs. A cross-sectional analysis of the New Orleans Alcohol Use in HIV (NOAH) Study was conducted (N=460). Alcohol use was measured by the Alcohol Use Disorders Identification Test (AUDIT) score, phosphatidyl ethanol (PEth) levels, and 30-Day Timeline Followback (TLFB) drinks reported during the past 30 days. Alcohol treatment history was defined as self-reported receipt of any alcohol-related treatment, including Alcoholics Anonymous (AA), detoxification, outpatient treatment, residential rehabilitation, psychiatric inpatient treatment, and the number of previous alcohol treatment episodes. Alcohol-related outcomes were compared between participants with and without a history of alcohol treatment by HIV status using Chi-square, Student's T-test, correlations, and logistic regression. 86.7% of participants were PWH and 13.3% of participants were HIV negative. Most participants were African American (81.1%), male (67.4%), and had an annual income of below $20,000 (84.4%). 131 (28.4%) reported a history of alcohol-related treatment. Mean AUDIT scores among PWH and those without HIV were 8.2±8.2 and 7.2±8.5, respectively. PEth scores were different between PWH and those without (178.3±462 and 52.6±123.4, respectively). Among PWH, 25.1% reported prior alcohol treatment compared with 3.3% of HIV-negative participants. People who had been to treatment more times also tended to have higher AUDIT scores ($p < 0.0001$). People who had been to treatment more times also tended to have higher PEth levels ($p = 0.0357$). There was no meaningful relationship between the number of times someone had been in treatment and how many drinks they reported in the last 30 days ($p = 0.3323$). Participants who reported a history of alcohol treatment tended to have higher alcohol use measures. This may be because people with more severe alcohol misuse are more likely to seek treatment. Future research should follow participants over time to better understand how alcohol treatment affects sustained reductions in alcohol use and to identify ways to improve treatment and support for those who need it. Understanding treatment utilization and its relationship with alcohol use may help identify opportunities to improve care.

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"Targeting CISD1 to Overcome Endocrine Resistance in Breast Cancer"

Breast cancer is the second most common cancer type in American women, affecting about 1 in every 8 females, or about 13% of the female population in the country. Estrogen Receptor Positive breast cancer (**EPBC)** is the most common subtype, comprising ~70% of breast cancer cases. These tumors are dependent on the estrogen receptor (**ERα**), and modulating **ERα** activity through endocrine therapy (ET) is a current treatment approach. Targeted treatment with selective estrogen receptor modulators (**SERMs**; e.g., Tamoxifen) and selective estrogen receptor degraders (**SERDs**; e.g., Fulvestrant) has been an effective approach for some cases. However, prolonged therapeutic use often causes resistance via multiple mechanisms, including mutation or loss of **ERα**, metabolic rewiring, or upregulation of alternative pathways (HER2, MAPK, AKT, etc.), often leading to cancer relapse. Of the 70% of ER+ breast cancer patients, about 40% of this population ultimately develop drug resistance. When including the rare ~15% of BC cases that are triple-negative and ~15% that are ER negative, ~70% of all breast cancer patients remain without any effective targeted therapeutic treatment.

CISD1, also known as mitoNEET, is an iron-sulfur cluster protein bound to the outer mitochondrial membrane that is responsible for maintaining iron homeostasis, possesses enzymatic redox functions, and is often overexpressed in cancers, including EPBC. Increasing evidence suggests that aberrant CISD1 overexpression promotes cancer cell survival and enhances energy metabolism to sustain rapid proliferation. The abundance of overexpression in tumor cells, as well as destructive cellular effects upon its suppression, highlights CISD1 as a potential cancer biomarker and therapeutic target.

Based on these findings, we hypothesize that targeted inhibition of CISD1 will effectively inhibit the growth and proliferation of wild-type and tamoxifen-resistant EPBC cells *in vitro* and will synergize with antihormonal therapies to overcome resistance to SERMs.
To test this hypothesis, we employed bioinformatics approaches, cell viability assays (MTT), and immunoblotting. We found that CISD1 mRNA and protein expression are negatively correlated with ESR1/ERα expression in wild-type and Tamoxifen-resistant EPBC cells. We also found that exogenous overexpression of CISD1 significantly decreases sensitivity to both 4-OH Tamoxifen and Fulvestrant, and that CISD1 inhibition enhances Tamoxifen-resistant MCF-7 cells' sensitivity to Tamoxifen.

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Investigating cysteine-mediated interactions between *Chlamydia trachomatis* Chaperones Scc1 and Scc4

Background: *Chlamydia trachomatis* is an obligate intracellular pathogen responsible for widespread human diseases, including the most common bacterial sexually transmitted infection. To establish infection, it utilizes a type III secretion (T3S) system to translocate virulence effectors into the host cell. SccI and Scc4 are two critical T3S chaperones that function together to stabilize and facilitate the secretion of CopN, a crucial effector and "plug" protein. Beyond secretion, Scc1 interacts with bacterial RNA polymerase to regulate transcription.

Objectives: This study investigates whether the two cysteine residues in Scc1 (C28 and C99), which potentially form covalent disulfide linkages, are essential for its interaction with Scc4.

Methods: A combined strategy of site-directed mutagenesis and a transcription activation-based bacterial two-hybrid assay was utilized. Mutated SccI fragments (C28A, C99A, and the C28A/C99A double mutant) were cloned into pBR vectors to express fusions with the N-terminal domain of the *E. coli* RNA polymerase α-subunit (α-NTD) under an IPTG-inducible promoter. These constructs were co-transformed with a plasmid encoding a λCI DNA-binding domain-Scc4 fusion into the FW102 OL2-62 reporter strain. Protein-protein interactions were quantified via β-galactosidase assays measuring *lacZ* expression.

Results and Conclusions: The β-galactosidase assays revealed that the single C28A and C99A mutations caused no significant change in *lacZ* activity compared to the wild-type control. However, the double mutant exhibited a significant decrease in reporter activity under IPTG-induced conditions. Additionally, all mutant variants demonstrated significantly higher *lacZ* activity when induced compared to their uninduced states. These findings suggest that while individual cysteines are dispensable, the presence of at least one functional cysteine residue is critical for the Scc1–Scc4 chaperone interaction.

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"Effects of High Fat Diet on Placental ACE2 Expression and Vascularization in Female Mice"

BACKGROUND: Obesity-induced metabolic dysfunction impairs cardiovascular and reproductive health in women, potentially through an imbalance within the Renin-Angiotensin System (RAS). Angiotensin II (Ang II), acting through angiotensin type I receptors (AT_1R), promotes vasoconstriction and oxidative stress whereas angiotensin-converting enzyme 2 (ACE2) metabolizes Ang II to anti-inflammatory/vasodilatory peptide Ang-(1-7). Previous studies from Dr. Yue's group have shown that female C57BL/6 mice fed a high fat diet (HFD) for 10-12 weeks developed cardiometabolic dysfunctions including reduced glucose and insulin tolerance, elevated blood pressure, and autonomic dysfunction compared to regular diet (RD) fed controls. Importantly, following mating with RD-fed males, offspring from HFD-fed females suffered low birth weight and increased postnatal mortality. Mechanistically, reduced expression of ACE2 was observed in placenta from HFD-fed females, and treatment with Ang-(1-7) improved the cardiometabolic and reproductive function in HFD-females.

OBJECTIVE: The objective of this study was to examine the expression and localization of ACE2 in placenta and its relationship with placental vascularization and tissue morphology in RD and HFD fed females and the potential therapeutic effects of Ang-(1-7) treatment.

METHODS: Formalin-fixed, paraffin-embedded mouse placenta sections (4 μm) were prepared for histological and immunohistochemical (IHC) staining. IHC with ACE2 and Wheat Germ Agglutinin (WGA) antibodies were performed on placenta sections to assess ACE2 expression and vascularization, respectively. Hematoxylin and Eosin (H&E) stained placenta sections were imaged to evaluate placental morphology, and average junctional zone-to-labyrinth ratios were measured using Cell Sense software. IHC images were acquired using a Keyence microscope, and ACE2 and WGA expression levels were quantified from 40X images in the labyrinth zone of the placenta. Lastly, RT-qPCR was performed using placental cDNA to quantify ACE2 mRNA expression.

RESULTS/CONCLUSIONS: In the labyrinth zone (LZ) of the placenta, ACE2 is expressed in syncytiotrophoblasts, the cell type that promotes nutrient exchange and fetal development. HFD leads to decreased ACE2 expression in the placental LZ and reduced labyrinth area compared to RD controls, which was alleviated by Ang-(1-7) treatment. Area measurements of the LZ and junctional zone (JZ) showed a higher average LZ area in the RD and ANG 1-7 treatment groups than in HFD. The average LZ area (pixels) was 5,399,313 ± 57,212 (mean ± standard error) in RD, 4,204,575.2 ± 345,931 in HFD, and 5,249,899.4 ± 154,304 in the Ang-(1-7) treatment group. One-way Anova statistical analysis showed that the LZ area was significantly lower in HFD compared to other treatment groups, but there was no significant difference between the LZ area in RD and Ang-(1-7) treatment group. WGA staining showed a simplification of the maze-like vascular network in the labyrinth in the HFD group compared to RD controls, and Ang-(1-7) treatment resulted in improved vascularization. RT-qPCR data showed a significant decrease in ACE2 mRNA levels in the HFD treatment group, consistent with the IHC results. Taken together, our study suggests that HFD downregulates ACE2 in the placenta, leading to reduced labyrinth growth and vascularization.

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Acute Rate-Decreasing and Antinociceptive Effects of Morphine and Oxycodone in Male and Female Long-Evans Rats

Background: According to the CDC there were over 69,973 opioid-related deaths in the United States during 2025.[1] One major obstacle to reducing physical and psychological dependence on opioids is the aversive withdrawal syndrome that follows termination of opioid use. Therefore, the purpose of this preclinical experiment was to establish a behavioral model of withdrawal using a naltrexone discrimination procedure to aid in the development of interventions to alleviate withdrawal symptoms. Prior to establishing physical dependence in both male and female rats, however, the effective doses of two opioids (morphine and oxycodone) were determined on food-maintained operant responding (lever pressing) and in a warm-water tail-withdrawal assay.

Methods: Initially, twelve Long-Evans rats (6 male, 6 female) were placed in operant behavioral chambers and trained to make a single lever press to receive a food pellet during 60-minute behavioral sessions. Over successive sessions, the number of lever presses gradually increased until the subjects were making 20 lever presses for each food pellet. When responding under this FR-20 schedule stabilized, test sessions were intermingled twice per week with training sessions to assess the effects of morphine (1-18 mg/kg) and oxycodone (0.32-5.6 mg/kg). During these test sessions, there were four 15-minute components of responding under the FR-20 schedule with a 15-minute blackout before each component to allow for the administration of increasing cumulative doses. At the end of these test sessions the subjects were removed from the chambers and tail-withdrawal latencies were measured in a water bath maintained at either 40°C or 50°C. As controls for these drug administrations and the involvement of the mu opioid receptor, saline injections were also administered during separate test sessions and naltrexone (0.1-3.2 mg/kg) was administered both alone and prior to morphine, respectively. Response rate served as the dependent variable for lever pressing, whereas tail-withdrawal latency served as the dependent measure for antinociception.

Results: Morphine and oxycodone dose-dependently decreased response rate, with female rats more potently affected than male rats. Naltrexone administration produced similar rate-decreasing effects in both sexes when administered alone, but the antagonism of morphine by naltrexone was more complete in males than females. Lastly, morphine was equipotent in increasing tail-withdrawal latency in both male and female rats, but oxycodone was more potent in male rats than female rats.

Conclusions: These findings demonstrate the capacity mu opioid receptor agonists to decrease motivated behavioral responding and produce antinociception in a sex-dependent manner. Further, these findings will allow us to assess how these responses change during both chronic opioid administration and withdrawal.

[1] Opioid Overdose Deaths: National Trends and Variation by Demographics and States | KFF

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Associations Between E-Cigarette Use Frequency and Mental Health Symptoms on Adolescent Academic Performance

BACKGROUND: While current electronic cigarette (e-cigarette) use has declined in the United States (US), from 7.7% in 2023 to 5.9% in 2024, they remain popular among adolescents. Mental health symptoms such as depression and anxiety are more prevalent among adolescent e-cigarette users than non-users, and both e-cigarette use and poor mental health have been associated with poor academic performance. However, less is known about whether the frequency of e-cigarette use, independently or jointly with mental health symptoms, influences academic performance. This study examined the association between frequency of current e-cigarette use and mental health symptoms independently and jointly on subsequent academic performance among US adolescents.

METHODS: Using a cross-sectional study design, we analyzed data from the 2025 National Youth Tobacco Survey (NYTS). The study included youth who reported e-cigarette use within the past 30 days and had valid information on academic performance and mental health symptoms (N=950). The primary outcome was self-reported academic performance (grades A to F). The predictors of interest were e-cigarette use frequency and mental health symptoms, and covariates included age, sex, race, and grade level. Survey weighted descriptive statistics summarized participant characteristics. Chi-square tests compared participant characteristics by academic performance, while weighted ordinal regressions examined the independent and joint associations of e-cigarette use frequency and mental health symptoms with academic performance, after adjusting for covariates.

RESULTS: Among 950 current e-cigarette users, most reported earning mostly A's (31.4%) or mostly B's (34.1%), while 12.6% reported mostly D/F grades. In bivariate analysis, academic performance did not differ significantly by e-cigarette use frequency ($p = 0.219$) but did not differ significantly with mental health symptoms ($p = 0.151$). After adjusting for age, sex, race, and grade level, mental health symptoms had 45% higher odds of poorer academic performance (AOR =1.45, 95% CI =1.07-1.96; $p = 0.018$). Additionally, those who used e-cigarettes on 20-30 days/month had significantly higher odds of poorer academics compared to those smoking 1-5 days (AOR=1.45, 95% CI= 1.06-1.99; $p = 0.023$). No significant interactions were observed between e-cigarette use frequency and any mental health symptom, indicating that the association between mental health and academic performance did not differ by e-cigarette use.

CONCLUSION: Among youth who currently use e-cigarettes, mental health symptoms, and more frequent e-cigarette use was not associated with poorer academic performance in unadjusted analyses, when examined with covariates this relationship is better explained. These findings highlight the importance of addressing mental health alongside tobacco prevention and cessation efforts to support academic success among adolescents.

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"Genetic Complementation of the EDE1 Endocytosis Mutant in the Pathogenic fungus *Cryptococcus neoformans*"

Introduction. *Cryptococcus neoformans* is an opportunistic fungal pathogen and a leading cause of life-threatening meningitis, especially in people with weakened or immunocompromised immune systems. One process that helps this fungus grow and survive is endocytosis, in which cells take up materials from their surroundings. Its capacity to cause disease depends on endocytosis and intracellular membrane trafficking, which support growth, morphogenesis, signal transduction, and virulence. The *EDE1* gene encodes a multi-modular endocytic scaffold protein that organizes clathrin-mediated endocytosis and actin remodeling. Previous studies showed that deleting *EDE1* (creating a *Δede1* mutant) reduces uptake of the fluorescent dye FM4-64, suggesting that endocytosis is impaired. In this project, we aim to restore a normal copy of *EDE1* to the *Δede1* mutant strain to determine whether normal endocytosis can be restored.

Hypothesis. We hypothesize that the endocytic defect of the Δede1 mutant results specifically from the loss of EDE1, and that reintroducing a wild-type copy of EDE1 into the mutant will restore normal FM4-64 dye uptake. Therefore, the complemented strain is predicted to recover wild-type endocytic function, while the uncomplemented Δede1 mutant remains impaired.

Methods. The wild-type *EDE1* gene was amplified from *C. neoformans* genomic DNA using PCR with primers designed to amplify the promoter and coding regions. PCR products were analyzed by agarose gel electrophoresis. DNA fragments of the expected size were cloned into plasmid vectors, propagated in *Escherichia coli*, and verified by DNA sequencing. The verified construct will then be introduced into the *Δede1* mutant using biolistic (gene gun) transformation for genetic complementation.

Results. We first compared wildtype and *Δede1* strains using FM4-64 dye uptake and observed delayed uptake in the *Δede1* mutant, consistent with impaired endocytosis. To generate the complementation construct, we designed PCR primers based on publicly available *EDE1* sequences. While we successfully amplified a fragment of approximately 3 kb from the 3′ region of the gene, repeated attempts to amplify the remaining regions were unsuccessful. Comparison of *EDE1* sequences from multiple *C. neoformans* strains suggested sequence variation that likely affected primer binding. Based on this analysis, we redesigned the primers and are currently testing them to obtain the complete *EDE1* gene for complementation.

Discussion. If reintroducing the wild-type *EDE1* gene restores FM4-64 dye uptake, it would confirm that loss of *EDE1* is responsible for the observed endocytosis defect. These findings would improve our understanding of how endocytosis contributes to the biology of *C. neoformans* and may provide insight into cellular processes that support fungal disease.

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Baseline humoral antibody repertoires in HIV-1 infected KS patients against Kaposi sarcoma herpesvirus (KSHV) using high-resolution antibody profiling

Kaposi sarcoma-associated herpesvirus (KSHV, or HHV-8) is the aetiologic agent of Kaposi Sarcoma (KS) and other rare lymphoproliferative diseases. KS is one of the most common cancers among people living with HIV (PLWH). In the era of antiretroviral treatment (ART), KS still occurs in PLWH despite complete suppression of HIV-1 viral load and with reconstituted CD4+ T cell counts. While HIV-1 co-infection increases the risk of KS onset ~600-fold, individuals infected with KSHV often remain asymptomatic, making it difficult to identify surrogate immune markers of protection against KS. Moreover, more than half of KS patients in sub-Saharan Africa (SSA) fail to achieve complete resolution with chemotherapeutic treatment, and 20-40% experience disease relapse within a year. These findings underscore the need to define prognostic indicators, such as the antibodies (Ab) against different KSHV viral antigenic determinants, or repertoire, at baseline upon KS diagnosis, and how they relate to longitudinal clinical factors and disease trajectory. Phage immunoprecipitation sequencing (PhIP-seq) enables high-throughput, peptide-level profiling of patient Ab repertoires against the human virome using the VirScan phage display library. Such profiles support broad and unbiased characterization of prior and ongoing antiviral humoral immune responses. Our preliminary data showed high similarity between patient antiviral Ab repertoires identified at baseline and post-chemotherapy; however, Ab repertoires identified segregated between responders and non-responders. These findings support the hypothesis that the presence of baseline Ab repertoires could serve as biomarkers to predict KS treatment outcomes. In this study, we profiled the Ab repertoires of treatment-naïve (baseline) plasma samples from 28 Zambian PLWH with KS, as a validation set for our main longitudinal study (n=260, Uganda). Viral Ab repertoires were quantified (breadth and magnitude), at the peptide, protein, and organism-level. These baseline serologic data will be integrated with longitudinal laboratory and clinical metadata, including HIV-related parameters, immune status, and clinical assessments. Our study establishes a framework for combining high-resolution Ab profiling with longitudinal clinical metadata to identify candidate immune biomarkers of KS progression and treatment response. Findings from this cohort may inform future studies aimed at improving risk stratification and monitoring KS clinical settings.

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"Effects of Neighborhood Deprivation and Cortisol on Cognition in Patients with Multiple Sclerosis"

Background: Social Drivers of Health (SDOH) are the conditions under which individuals learn, work, socialize, and live. Individuals in environments that are associated with adverse SDOH conditions are left more vulnerable to negative healthcare outcomes (Vrtikapa, 2025). The Area Deprivation Index (ADI) is a validated measure of neighborhood environmental and social factors that can influence health (i.e., income, education access, housing) (Kind & Buckingham, 2018). It uses an index number to compare each neighborhood's conditions to state deciles (SD-ADI) of neighborhoods. One social factor which influences health outcomes is stress, a feeling which can be tangibly measured by hair cortisol concentration (HCC) levels (Moody, 2026). Multiple Sclerosis (MS) is an autoimmune disease of the central nervous system where the body attacks myelin, the fatty sheath which protects the axons of the nerve cells, leading to neurological symptoms such as blurred vision, mobility issues, and importantly cognitive decline such as trouble with memory, concentration, and thinking (*NIH-NINDS*, n.d.).

Objective: We set out to examine whether there is an association between HCC and patients' neighborhood ADI, and if this relationship impacts cognition in patients with MS.

Methods: The Symbol Digit Modalities Test (SDMT) was administered to 88 patients with MS to measure cognition. SDMT raw scores were standardized using age and education (T-Scores or SDMT-t). Using provided participant address information, we determined each individual's ADI number using the ADI Wisconsin Neighborhood Atlas Use Map. A subset (N=58) also provided hair samples collected from the parietal region of the scalp and were assayed for cumulative cortisol. Multiple Sclerosis Severity Score (MSSS) was computed using patient Expanded Disability Severity Scores. Correlations for variables of interest, t-tests, and regression models were run using JASP v 0.96.3.

Results: SD-ADI was negatively correlated with both SDMT-t (r=-0.31, p<0.01) and HCC (r=-0.31, p=0.02). A regression analysis was conducted with SDMT-t as the dependent variable and ADI, cortisol levels, and race entered as independent variables. The overall model was significant, $F(3, 50) = 3.82$, $p = .02$, and accounted for 19% of the variance in SDMT-t (adjusted $R^2 = .14$). Cortisol levels were negatively associated with SDMT-t scores, $t = -2.26$, $p = .03$. SD-ADI and race were not significant predictors.

Conclusion: We hypothesized that people living with more neighborhood stress would have higher levels of cortisol; this was not supported. Living in worse neighborhood conditions and increased HCC were associated with lower SDMT-t-scores. However, in our full model it was stress, not neighborhood or race, that remained significantly associated with cognition.