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2025 Columbia University Postdoctoral Research Symposium

Tuesday, May 13, 2025
CUIMC, Roy and Diana Vagelos Education Center (VEC)

Session I – Poster Session
10:30 am - 12 pm
VEC 401

1 – Alberto Nordmann – Rheumatology

"CAR T-cell Therapy in SLE: A Systematic Review and Pooled Analysis."

Introduction: CAR T-cell therapy is poised to revolutionize the treatment of SLE. Several publications reported on small numbers of SLE patients treated with CAR-T.

Objective: To review and summarize the available clinical evidence published or presented to date on the use of CAR therapy in SLE.

Methods: We conducted a systematic review to evaluate all clinical studies assessing safety and efficacy outcomes of CAR-therapy in SLE. For this review, we followed PRISMA recommendations. MEDLINE (PubMed), Embase, Scopus, and CENTRAL were consulted, as well as the ACR Convergence 2024 Archives.

Results: The search strategy yielded 4,943 results, of which eight were included from the consulted databases and six from the 2024 ACR Archive, for a total of 14 studies and 98 participants. All studies included patients with refractory SLE who had failed at least 2 immunosuppressive therapies. Most studies (n=12 [85%]) used a CD19-targetted CAR, with only 2 groups (15%) assessing bispecific CD19-BCMA CAR T-cells. Two studies (15%) evaluated allogeneic CAR products. All groups achieved B-cell depletion within 1-3 weeks following CAR infusion, with B-cell reconstitution observed after 2-3 months. Pooled analysis of the 63 individual SLEDAI scores available showed a median decrease in SLEDAI of 8 points at 3 and 6 months. The mean baseline SLEDAI of 12.8 (95% CI 11.7-13.8) decreased to 2.3 (95% CI 1.1-3.5) after 6 months. CRS occurred in 56 (57.1%) participants, of which 50 were grade 1, 5 grade 2, and 1 grade 3. ICANS was reported in three patients: 1 grade 1, 1 grade 3, and 1 grade 4.

Conclusion: This systematic review evaluated all the available clinical evidence on the use of CAR therapy in patients with SLE. The findings reinforce the efficacy of this treatment modality and underscore the need for further research to better define its role in SLE management.

2 – Anna-Katharina von Krauland – Climate School

"Characterizing New York State Offshore Wind Energy Curtailment to Support Net Zero Technology Growth."

The speed and scale needed to transition to a clean energy grid requires an integrated approach to address challenges in variability of energy resources and grid load. This research provides novel insights into the optimal use of otherwise-curtailed energy by analyzing the impacts of deploying different levels of renewable capacity on the cost, curtailment, and emissions reductions of future grid scenarios. The focus on New York State is motivated by a combination of its substantial energy demand stemming from large population centers, sizable offshore wind energy pipeline, enormous offshore wind energy potential, and ambitious clean energy targets. The extent to which supply is projected to outpace demand is quantified by employing grid load data in conjunction with high spatial and temporal resolution wind energy datasets with wind speeds at relevant hub heights for modern wind turbines. The resulting model captures a range of possible renewable capacity buildout scenarios mirroring existing state energy policy, and reports the quantity and temporal variation of curtailed energy for each. The study further identifies the conditions under which it would be most efficient to use this excess energy to fulfill requirements for technology such as green hydrogen electrolysis, carbon capture systems, or battery storage. This information can facilitate decision-making for strategic grid integration planning, including investment decisions around infrastructure that will help decarbonize hard-to-abate sectors. This study aims to enhance grid planning that will better serve end users by providing reliable and low-cost clean energy and support the burgeoning net zero carbon economy.

3 – Apoorva Singh – Irving Institute for Cancer Dynamics

"Dissecting mechanisms of marginal zone B cell ontogenesis and homeostasis."

Marginal zone (MZ) B cells, located at the junction of the red and white pulp in the spleen, are essential for our defense against blood-borne pathogens. Owing to their innate-like features, MZ B cells generate rapid, early antibody responses and also help kickstart the T cell-dependent high-affinity responses. Indeed, their dysregulation is linked with an increased risk of sepsis, infections from encapsulated bacteria, and autoimmune pathologies. Despite their significance, quantitative aspects of MZ B cell biology–development, maintenance, longevity, and population structure–are still poorly understood. Conventionally, MZ B cells are considered long-lived, self-renewing populations that develop from late-stage transitional B cells. However, recent studies indicate that their immediate precursors constitute a heterogeneous subset of cells representing the full spectrum of transitional stages, potentially encompassing fully differentiated follicular (Fo) B cells. To resolve this, we studied their numbers, replacement dynamics, and extent of division using a well-established bone marrow chimera system, which tracks the infiltration of new donor-derived cells in intact host lymphocyte compartments. We combined these data with an array of mechanistic mathematical models that explored different pathways of MZ B cell development and quantified the contributions of precursor influx, division, and loss to their maintenance.

Contrary to the prevailing view, our analyses reveal that MZ B cells are a short-lived, homogeneous population that are largely maintained by a continuous flux from both early and late-stage transitional B cells. Further validating our models using data from young (day 10 onwards) animals revealed inefficient generation of MZ B cells in early life. Our findings provide a continuous picture of age-dependent dynamic regulation of MZ B cell development and homeostasis from the neonatal period to adulthood.

4 – Azin Mehrjoo – Engineering

"Real-Time Structural Damage Detection Using Kolmogorov-Arnold Neural Networks and Cepstral Features."

Structural damage detection is essential for ensuring the safety and functionality of engineering systems subjected to dynamic loads. Real-time online detection of damage poses a challenge, as it requires rapid and accurate assessment of the structural states without lengthy in-situ evaluations. This work investigates the use of Kolmogorov-Arnold Networks (KAN) for assessing structural damage, using their ability to model complex nonlinear relationships and provide reliable classification of structural states. Compared to traditional Multi-Layer Perceptrons (MLPs), KANs offer many advantages, including their multi-parametric nonlinear structure, enabling effective capturing of complex patterns in data and enhanced feature representation. This allows for nuanced understanding of high-dimensional inputs while retaining the hierarchical traits of traditional neural networks, resulting in a high-dimensional nonlinear separation of the function space. Additionally, KANs exhibit better generalization capabilities and robustness to noise, making them particularly suitable for damage detection tasks where accuracy and adaptability are critical. As damage sensitive features, the proposed framework uses cepstral coefficients extracted from vibration signals. These coefficients are well-established features for capturing subtle changes indicative of structural damage. Cepstral features are fed into a recursive KAN to classify structural states as damaged or undamaged. By addressing the need for online damage detection in structural systems, this study highlights the potential of Kolmogorov-Arnold Networks as robust tools for advancing vibration-based monitoring and damage detection.

5 – Divya Vimal – Genetics and Development

"Osteocalcin signaling transiently supports Œ≤-cell plasticity and glucose homeostasis following gonadal failure."

Estrogen (E2) depletion, such as that occurring with menopause or in young women undergoing estrogen blockade for breast cancer or endometriosis leads to widespread physiological changes, including altered bone remodeling and increased metabolic stress. Paradoxically given the well-documented detrimental effects of E2 depletion on pancreatic Œ≤-cell survival, postmenopausal women maintain normoglycemia for a significant period of time. In contrast, E2 depletion rapidly increases bone resorption as well as a delayed but sustained decline in bone formation. As bone resorption increases the release of active osteocalcin (Ocn), a bone-derived hormone known to regulate insulin secretion and Œ≤-cell proliferation, we hypothesized that increased Ocn signaling in Œ≤-cells may act as a compensatory mechanism to preserve glucose homeostasis following gonadal failure.

To test this, we ovariectomized (OVX) mice at two human-relevant ages: young (4 months, modeling E2 depletion in premenopausal women receiving E2 blockers) and aged (12 months, modeling menopause) to perform metabolic and histological analyses. In young mice, OVX induced an increase in Œ≤-cell proliferation and enhanced glucose-stimulated insulin secretion (GSIS). These effects did not occur in Ocn-deficient mice demonstrating the existence of a bone mediated adaptive proliferative response to gonadal failure. Over time, however Œ≤-cell proliferation decreased and glucose intolerance developed in wild type mice.

In older mice, Œ≤-cell proliferation and insulin secretion increased at later time points post-OVX, but to a lesser extent than in young mice. These findings suggest that while the osteocalcin-mediated response is conserved with age, it is blunted compared to young mice. We are now defining the molecular mechanisms underlying this osteocalcin-mediated protective effect using transcriptomic analyses (RNA-seq).

From a clinical perspective, these results suggest that initiating anti-resorptive therapy early especially in younger women undergoing estrogen blockade to preserve bone mas may increase their risk of developing type 2 diabetes.

6 – Elanur Yilmaz – Neurology & Taub Institute

"Beyond the Barrier: Unveiling Vascular Risk Pathways in Alzheimer's Disease Among Hispanic Individuals."

Blood-brain barrier (BBB) dysfunction and vascular dysregulation are major contributors to Alzheimer's disease (AD), particularly in the presence of the APOE-Œμ4 allele, and these effects are more pronounced in the Hispanic population compared to other populations. However, most studies and omics datasets have been derived from non-Hispanic white individuals, and the molecular underpinnings of vascular contributions to AD in Hispanics remain largely unknown.

In this study, we generated the largest single-nucleus RNA sequencing (snRNA-seq) dataset of Hispanic brains to date, comprising 446,675 nuclei from the BA9 region of 53 postmortem brain samples. After filtering out low-quality cells and doublets, we analyzed 328,720 high-quality nuclei, which were distributed across 36 clusters assigned to 8 distinct cell types.

To investigate the role of APOE-Œμ4 allele in vascular contributions to AD, we compared carriers with non-carriers. We performed differential gene expression and pathway enrichment analyses on gliovascular cells to identify potential cellular interactions between vascular and glial cells. These results were compared with the ROS/MAP dataset, which predominantly includes data from non-Hispanic whites, to identify distinct vascular dysregulation in AD in Hispanics.

Our findings suggest that in Hispanic brains, astrocytes play a major role in regulating angiogenesis and vascular development. Disruption of these pathways may contribute to increased vulnerability to vascular-associated Alzheimer's disease in Hispanic population. This study provides novel insights into vascular dysregulation mechanisms in AD and represents the most extensive transcriptomic analysis of Hispanic brains to date.

7 – Elena Floris – Thoracic Surgery

"Aspiration of conjugated Bile Acids is associated with increased CD8 T cells in the bronchoalveolar lavage of lung transplant recipients."

Background: Chronic lung allograft dysfunction (CLAD) limits lung transplant recipients (LTRs) survival. Aspiration of bile acids (BA), especially conjugated BA, and increased CD8 T cells in the bronchoalveolar lavage (BAL) are independently CLAD risk factors. We sought to study for the first time the relationship between aspirated BA and T cell-mediated immune response.

Methods: LTRs from 2020-2022 without connective tissue disorders or sarcoidosis were considered (n=32). Concomitant BAL and large airway bronchial washings (LABW) were prospectively collected at the 3 months surveillance bronchoscopy. BAL samples were not considered if: alive T cell count lower than 50; positive for infection; from LTRs with greater than A1B0 acute cellular rejection. BAL cells were stained for CD45, CD3, CD8, CD4 and with a viability dye, then analyzed by flow cytometry. LABW were assayed for 15 conjugated and unconjugated BA species using mass spectrometry, and for 48 cytokines by multiplex.

Results: Total conjugated BA correlated with CD8/total T cells percentage (Spearman r=0.45, p=0.009) versus unconjugated BA (r=0.06 p=0.75). Correlation was found for the conjugated BA:

GCA (r=0.60); GCDCA (r=0.39); TCA (r=0.46); and TCDCA (r=0.44) (p<0.05). Samples with high conjugated BA levels (upper tertile) showed greater CD8% compared to the rest: 56% vs 45% (Mann-Whitney p=0.028). Conjugated BA correlated inversely with the CD4/total T cells percentage (GCA r=-0.59, p=0.0004). CD8 T cells correlated with various cytokines, IL-13 having the strongest correlation (r=0.57, p=0.0006).

Conclusion: Our original findings on the role of BA aspiration on LTRs airway immune cells show that conjugated BA, in particular GCA, correlate with CD8 T cells presence. This suggests that one of the injury mechanisms of conjugated BA is through the CD8 T cells action. Additionally, CD8 T cells correlated with IL-13, a fibrosis mediator. These findings warrant confirmation and further characterization of specific CD8 T cell phenotypes.

8 – Kaiyuan Zheng – Biomedical Engineering

"Focused ultrasound-mediated APOE4 knockdown in mouse brain."

Alzheimer's disease (AD) is the most prevalent neurodegenerative disorder worldwide, characterized by the accumulation of amyloid-beta (AŒ≤) plaques and hyperphosphorylated tau (p-tau) neurofibrillary tangles, accompanied by synapse and neuron loss, neuritic dystrophy, vascular alterations, and inflammatory responses. Apolipoprotein E4 (APOE4) is recognized as the strongest genetic risk factor for late-onset Alzheimer's Disease (LOAD), which is implicated in 55-75% of AD dementia cases. Therefore, therapeutic strategies to reduce apoE4 protein expression in APOE4 carriers present a promising approach to attenuate neuroinflammatory and neurodegenerative processes driving disease progression. In this study, we employed focused ultrasound (FUS) to transiently open the blood-brain barrier (BBB) for efficient knockdown of humanized APOE4 in the mouse brain via gene editing. Specifically, we designed the all-in-one CRISPR-based adeno-associated virus (AAV) vectors to target humanized APOE4 and intravenously administered at a dose of 1.5√ó10^12 vg per mouse for gene editing in the brain. The FUS-induced BBB opening significantly enhanced AAV delivery in mouse brain, resulting in a 12.6% reduction in APOE4 gene expression in the targeted hippocampus with minimal off-target effect. Correspondingly, we observed over 20% decrease in apoE4 protein levels, along with significant reductions in astrocyte and microglia levels. Importantly, the reduction of APOE4 expression correlated with marked decrease in astrogliosis, microgliosis, and AŒ≤ accumulation in aged mice, indicating the potential to mitigate APOE4-driven neuroinflammation and neurodegeneration for AD treatment. Our findings demonstrate a noninvasive, precise, targeted approach for APOE4 knockdown, highlighting FUS-mediated brain-directed genome editing as a promising therapeutic strategy to combat APOE4-driven neurodegeneration, offering valuable insights into the development of advanced therapeutic strategies for AD and other neurodegenerative disorders. Additionally, our results underscore the versatility of FUS-based delivery technologies in facilitating precise and effective BBB penetration for central nervous system therapeutic interventions.

9 – Keitaro Akita – Cardiology

"Comprehensive Proteomics Profiling Identifies Circulating Biomarkers to Distinguish Hypertrophic Cardiomyopathy from Other Cardiomyopathies with Left Ventricular Hypertrophy."

Background: Distinguishing hypertrophic cardiomyopathy (HCM) from other cardiomyopathies with left ventricular hypertrophy (LVH), such as hypertensive LVH, transthyretin amyloid cardiomyopathy (ATTR-CM), and aortic stenosis (AS), is sometimes challenging. Using plasma

proteomics profiling, we aimed to identify circulating biomarkers and dysregulated signaling pathways specific to HCM.

Methods: In this multicenter case-control study, plasma proteomics profiling was performed in cases with HCM and controls with hypertensive LVH, ATTR-CM, and AS. Two-thirds of patients enrolled earlier in each disease group were defined as the training set, and the remaining one-third as the test set. Protein concentrations in HCM were compared with those in hypertensive LVH (comparison 1), ATTR-CM (comparison 2), and AS (comparison 3). Candidate proteins that meet the following 2 criteria were selected: (1) Higher abundance in HCM throughout all 3 comparisons or lower abundance in HCM throughout all 3 comparisons with univariable $P<0.05$ and $|\log2(\text{fold change})| > 0.5$ in both the training and test sets and (2) Independently associated with HCM with multivariable $P<0.05$ after adjusting for clinical parameters significantly different between HCM and controls. Using the selected candidate proteins, a logistic regression model to distinguish HCM from controls was developed in the training set and applied to the test set. Finally, pathway analysis was performed in each comparison using proteins with different abundance.

Results: Overall, 4,979 proteins in 1,415 patients (HCM, n=879; hypertensive LVH, n=331; ATTR-CM, n=169; AS, n=36) were analyzed. Of those, 5 proteins were selected as candidate proteins. The logistic regression model with these 5 proteins had an area under the receiver-operating-characteristic curve of 0.86 (95% CI 0.82-0.89) in the test set. The MAPK and HIF-1 pathways were dysregulated in HCM throughout the 3 comparisons.

Conclusions: This study identified circulating biomarkers that distinguish HCM from other cardiomyopathies with LVH independently from confounders and revealed signaling pathways associated with HCM.

10 – Krystyna Wieszczek – English and Comparative Literature
"Understanding the Social Impact of Literature: Empowerment."
In seeking to expand our understanding of literature's potential to contribute to mitigating social challenges, this Horizon Europe Marie Curie research project investigates whether reading can enhance an individual's sense of empowerment.

Empowerment is understood here as an expansion of freedom of choice and action through increased authority over decisions that affect one's life. Research shows that wellbeing is influenced by various interconnected factors, including those often shaped by our choices–such as education, employment, housing, and health. Our relationships and the media–our augmented environment–significantly influence our identity, beliefs, and attitudes, and thereby our life choices. Since media industries often promote insecurity, social division, consumerism, and addictive behaviors, and since one's environment may embed limiting beliefs and conformism, mitigating such disempowering influences is a critical step.

Empirical studies exploring the effects of literature on health, wellbeing, and psychology suggest that literary reading can stimulate shifts in worldview, mentalising ability (Theory of Mind), and self-understanding–encouraging openness to self-transformation and even personality trait modification. This may point to literature's potential to also foster empowerment.

The project aims to identify five literary works of prose and a short story that may enhance readers' empowerment to counter the effects of disempowering influences in their environment. It seeks

to develop an interdisciplinary methodological tool to measure such effects and test it empirically on diverse groups of disadvantaged young readers and control groups in Italy and the United States.

11 – Laura Quevedo – Institute for Cancer Genetics

"Targeting the tumor microenvironment by duvelisib in peripheral T-cell lymphomas."

Introduction: Peripheral T-cell lymphomas (PTCLs) are aggressive hematologic malignancies with poor responses to traditional chemotherapy, necessitating novel therapies. The tumor microenvironment (TME) plays a key role in PTCL progression. Angioimmunoblastic T-cell lymphoma (AITL), a common PTCL subtype, is associated with RHOAG17V mutations and TET2 loss. Our genetically engineered mouse model mimics AITL's histology and genetics.

Therapeutic Context: PI3K signaling is a crucial oncogenic driver in T-cell lymphomas. Duvelisib, a selective PI3KŒ¥/ŒE≥ inhibitor, has shown promising efficacy in PTCL, with an overall response rate of >50%. Beyond its effect on PI3K signaling, duvelisib modulates the TME in solid tumors and B-cell lymphomas. We hypothesize that duvelisib's therapeutic effects in PTCL result from its dual action on both tumor cells and the TME.

Objective: This study aims to investigate the structural and functional changes in PTCL and its TME in response to duvelisib, focusing on its impact on anti-tumor activity. We will utilize single-cell RNA sequencing (scRNA-seq) to analyze lymphoma and TME alterations in duvelisib-treated murine PTCL models.

Methods: Mice with Tet2,$^{-/-}$ RhoaG17V lymphoma were treated with vehicle or duvelisib. Tumor load was assessed by bioluminescence. scRNA-seq data were analyzed to identify gene expression changes, cell lineage distributions, and cell-cell interactions. Differential expression and gene set enrichment analyses identified pathways altered by duvelisib treatment.

Results: Duvelisib treatment reduced lymphoma cell proliferation and altered the TME, expanding granulocytes. GSEA revealed changes in oxidative phosphorylation, TNFA signaling, and PI3K/Akt/mTOR pathways. Cell-cell interaction analysis showed altered interactions between lymphoma cells and immune cells, including granulocytes and monocytes, mediated by VCAM1 and CD40LG.

Conclusion: These findings suggest that duvelisib modulates the TME and lymphoma cell interactions, offering insights for improving PTCL treatment strategies.

12 – Leila Simanijoni – Cognitive Neuroscience Division / Neurology

"Transcranial Direct Current Stimulation on Language Function in Multiple Sclerosis: Is It Beneficial in Single-Session Administration?"

Language dysfunction is receiving increasing attention as a prevalent and early affected cognitive domain in individuals with multiple sclerosis (MS). This pilot study testing the potential effect of transcranial direct current stimulation (tDCS) on language improvement in 32 MS participants, targeting the left/right dorsolateral prefrontal cortex (dlPFC) and left/right inferior frontal gyrus (IFG) versus sham treatments. Verbal fluency was evaluated pre and post intervention. Anodal stimulation of the left dlPFC significantly enhanced semantic fluency, while other regions showed no significant effects. The left dlPFC group showed higher semantic scores compared to other anodal groups, suggesting targeted dlPFC stimulation as a promising approach for improving language functions in MS patients.

13 – Maithe Rocha Monteiro de Barros – Irving Institute for Cancer Dynamics

"Single-nuclei RNA and ATAC multiome sequencing in the study of gastric stem cells and cell fate."

Gastric cancer (GC) is the fifth leading cause of cancer death worldwide, with a diffuse form associated with inactivation of CDH1 and an intestinal form associated with inactivation of TP53. Furthermore, chronic infection of the stomach with Helicobacter pylori and dietary nitrate ingestion are major risk factors for GC. Nevertheless, cells of origin and meditators of progression in GC remain unknown. Gastric epithelial cells maintain homeostasis through dynamic self-renewal mechanisms involving stem and progenitor cells, however identifying these cells has been challenging. This study aims to identify stem cells on healthy gastric tissue to help define the cells of origin in GC.

A total of 10 gastric samples were collected from healthy mice at 8-12 weeks of age. Isolated nuclei were subjected to simultaneous profiling of gene expression and chromatin accessibility through single-cell multiome sequencing. After quality control a total of 31,538 cells were analyzed using the Seurat and Signac packages and weighted-nearest neighbors analysis allowed for joint clustering of the RNA and ATAC assays. Epithelial cells were subsetted resulting in 27,533 cells, 527,963 regions and 24,596 genes that were used as an input for SCENIC+ to uncover gene regulatory networks. A total of 212 direct and 320 extended eregulons were produced. Direct eregulons had on average 247 target regions and 112 target genes. SCENIC+ recovered a known master regulator that governs the differentiation of stem cells into acid-secreting parietal cells.

Multiome sequencing enables the exploration of transcriptional and regulatory programs underlying cell development. This approach can identify TF combinations specific to each epithelial cell type and uncover the regulatory networks that govern cell identity and function. Analysis across all cell types is ongoing, and the results will serve as a foundation for investigating key TFs through spatial profiling and single-cell analysis in gastric organoids, GC mouse models and human gastric samples.

14 – Marco Tagliafierro – Cardiothoracic Surgery

"When Repair Fails: Predictors of Intra-Operative Failure of Mitral Valve Repair Attempt."

Background: Mitral valve repair (MVr) is the gold standard for treating MV disease, while mitral valve replacement (MVR) is preferred when MVr is unfeasible. Extended surgeries are linked to worse outcomes. When intraoperative saline testing reveals a failed MVr, converting to MVR prolongs operative time and risk.

Objective: To retrospectively identify pre- and intra-operative predictors of MVr failure to optimize patient selection and improve surgical outcomes.

Methods: Data were retrospectively collected from 600 patients who underwent MV surgery at Columbia University Medical Center between July 2020 and July 2023. Of 277 MVr attempts, 249 (89.9%) were successful (S group) and 28 (10.1%) failed (F group). A total of 101 variables were analyzed using descriptive, univariate, and multivariable analysis.

Results: Successful MVr was significantly associated with male sex (OR 0.39, $p=0.02$), elongated chordae (100% success, $p=0.007$), myxomatous leaflets (ML; OR 0.28, $p=0.024$), annuloplasty (OR 0.16, $p<0.001$), and lower residual MV regurgitation (36% vs 69%, $p<0.001$). MVr failure was

associated with chronic lung disease (OR 2.44, p=0.037), NYHA III-IV (OR 3.79, p=0.032), anterior (ALP; OR 3.59, p=0.002) or bileaflet prolapse (OR 3.58, p=0.006), any calcification (OR 12.8, p<0.001), anterior (aLMAC; OR 99.2, p<0.001) or posterior MAC (pLMAC; OR 11.1, p<0.001), rheumatic leaflets (OR 3.5, p=0.016), leaflet clefts (OR 2.96, p=0.035), longer bypass (Œ-70 min, p<0.001) and cross-clamp times (Œ-65 min, p<0.001), concomitant tricuspid repair (OR 4.06, p=0.001), longer ventilation (Œ-10 h, p<0.001), and higher reoperation rates. Multivariable analysis: ML (OR 0.25, p=0.034) was protective, while ALP (OR 5.01, p=0.002), aLMAC (OR 15.6, p=0.019), and pLMAC (OR 6.92, p=0.001) independently predicted MVr failure.

Conclusions: MVr outcomes are significantly influenced by anatomical and clinical factors. Identifying predictors can guide optimal procedure selection and improve outcomes. Future research should validate these predictors.

15 – Natalie Greaves-Peters – Health Studies & Applied Educational Psychology
"Enhancing K-12 School-Based Food and Nutrition Education: Developing and Validating a Socioecological Framework and Practical Evaluation Tool."
School-based food and nutrition education (SBFNE) programs are critical to addressing rising ultra-processed food consumption and declining fruit and vegetable intake among children. This three-paper dissertation identifies the core components of effective SBFNE programs and develops theory-informed, practical tools to assess and improve them.

Article 1: An umbrella review of 44 systematic reviews, covering 1,115 unique primary studies from 1900-2022, identified 20 critical components that contribute to positive dietary behavior change in K-12 settings. These components–such as cultural inclusivity, family engagement, and integration with school meals–were organized into a theoretical framework mapped to the first four levels of the Socioecological Model (SEM).

Article 2: Using the 20 components, a pilot evaluation tool was developed with a standardized 0-3 scoring system and detailed rating guide. Three SBFNE program developers tested the tool through ranking activities, concept mapping, and curriculum review. Findings showed that concept mapping was intuitive and helpful, while ranking was difficult due to component overlap. The pilot informed tool refinements and streamlined the data collection protocol.

Article 3: A formative evaluation with nine program developers further tested and refined the tool. Participants categorized components, created concept maps, reviewed their programs, received targeted recommendations, and selected improvement strategies. A practice-based logic model emerged from the concept maps. Developers reported the tool was useful and actionable for strengthening program effectiveness.

Conclusion: This dissertation bridges research and practice by integrating evidence-based components into a robust SEM framework and creating tools that support real-world SBFNE program improvement. These tools offer practical guidance to enhance dietary behavior outcomes for children in school settings.

16 – Pallavi Gupta – Nursing

"Developing a Novel Approach to Video-Based Fall Risk Assessment in Home Healthcare Using Multimodal Large Language Models: A Pilot Study."

Objective: Falls cause millions of injuries and deaths annually, making fall prevention a critical healthcare priority, especially in home healthcare (HHC) settings. Traditional risk assessments often fail to capture the complex interplay of personal, environmental, and behavioral factors. This study introduces a novel approach utilizing Multimodal Large Language Models (MLLMs) to analyze in-home video data for fall risk assessment. Specifically, it explores the use of the LLaVA-NeXT-Video7B-hf model, a compact and lightweight MLLM with fewer than 10 billion parameters enabling efficient fall risk assessment in resource-constrained environments.

Methods: Video data simulated a patient's in-home settings, and twelve fall risk factors (intrinsic, extrinsic, and behavioral) identified from the literature formed the basis for developing prompts to detect fall risks from video. The video was processed into 24 temporally equally spaced frames, ensuring a representative visual summary while adhering to the model's computational constraints. The MLLM model LLaVA-NeXTVideo7B-hf was iteratively assessed with the prompts, where concise prompts helped with simple inferences, while elaborated prompts showcased the model's ability for complex assessments. To ensure reliability, the model was executed three times per prompt, with consensus-based MLLM results compared to expert evaluations. %In the end, a set of benchmark prompts was developed specifically for fall risk assessment.

Results: When compared to human evaluations, the MLLM achieved 85.71% accuracy with concise prompts for easy inferences on seven simple risk factors and 100% accuracy with elaborated prompts for complex inferences on two risk factors. However, for two risk factors where clinical expertise was required or data was insufficient, the model produced 100% incorrect responses with both concise and elaborated prompts, highlighting its limitations in such scenarios.

Discussion and Conclusion: This study highlights the potential of MLLMs for fall risk assessment in home healthcare, achieving high accuracy with well-structured prompts. However, the model struggled with risk factors requiring clinical expertise or insufficient data.

17 – Paula Rodriguez Villamayor – Zuckerman Institute

"Neural Mechanisms Underlying Visually-Evoked Aggression in Male and Female Siamese Fighting Fish."

Aggression is a fundamental aspect of social behavior across species, influencing the dynamics of human and animal societies. While visual cues prominently trigger aggression in humans and primates, rodent and fly models, commonly used in behavioral studies, primarily rely on olfactory cues. Consequently, the impact of visual stimuli on aggression, and social behavior more broadly, remains relatively underexplored. Siamese fighting fish (Betta splendens), selectively bred for robust aggressive behavior driven by visual cues, offers a promising new model species to investigate the neurobiological underpinnings of visually-evoked aggression. This study aimed to (i) develop behavioral assays that elicit aggressive display in adult male and female and (ii) identify the neural circuits underlying visually-evoked aggression. Leveraging recent advances in machine learning, we employed markerless tracking methods to capture behavioral hallmarks of aggression (i.e. flaring). Additionally, tracing, phosphorylation of ribosomal subunit-S6 labeling, and RNA-sequencing (RNAseq) were utilized to map the neural representation of aggressive behavior. Our findings revealed that males exhibit significantly higher levels of aggression than females when visually exposed to a male conspecific. PS6 immunolabelling revealed an increase of activated

neurons in the dorsomedial-pallium (Dm), and other telencephalic regions during aggressive display to a conspecific, compared to exposure to an empty tank. Furthermore, RNAseq analysis identified several differentially expressed genes (DEGs) in these brain regions between males and females, potentially contributing to sex differences in aggressive behavior. Finally, we identified distinct projections of retinal ganglion cells to lateral thalamic nucleus and optic tectum, but not to Dm, suggesting a complex neural pathway involved in processing visual stimuli triggering aggression. Our study provides valuable insights into the neurobiological mechanisms underlying sex differences in visually-evoked aggression in Betta. Future directions involve genetic manipulation of DEGs between males and females and elucidating the intersection of identified brain regions with the visual system.

18 – Tanaporn Na Narong – Data Science Institute / Applied Physics & Applied Mathematics
"Diffusion Models for Determining Local Atomic Structure from X-Ray Absorption Spectra."

Given a measured signal or spectrum from a material, material scientists often wonder: what is the geometrical arrangement of the atoms? This "inverse" problem is crucial for understanding materials' properties but remains very challenging due to its ill-posed nature. Inspired by recent applications of generative AI in predicting plausible atomic and molecular structures, we train a latent diffusion model to determine the full 3D local geometry around transition metal sites in oxide structures conditioned on X-ray near-edge absorption spectra (XANES), which contain rich information about the local atomic environment around the absorbing atoms but are less commonly used as a structural probe. Parameterized by equivariant graph neural networks, the model outputs cartesian coordinates of the metal's nearest neighbors (oxygen atoms). The model is evaluated by comparing the atomic pair distribution functions (PDFs) of ground truth and generated structures, and we find that conditioning on XANES spectra improved the model performance over the baseline unconditional model. Our results show that diffusion models can effectively extract the local coordination geometry from XANES spectra of transition metal atoms in oxides, highlighting the potential of modern AI techniques in accelerating material characterization by leveraging electronic and structural information contained in XANES.

19 – Tarun Kaniganti – Pediatrics
"Studies of copper transporter CTR1 in model systems."

CTR1 (SLC31A1) is a high-affinity importer of the trace metal copper required for proper infant brain development, and that also enhances the metastatic potential of certain copper-dependent cancers. Our report of twin males homozygous for a novel CTR1 missense variant, R95H, provided the first detailed description of human CTR1 disease (Batzios et al., 2022). These subjects manifested a distinctive clinical phenotype: profound infantile-onset neurodegeneration characterized by seizures, neurodevelopmental delays, hypotonia, and brain atrophy. To further characterize this disorder, we are conducting detailed biochemical and molecular studies, and experimental therapeutics in cerebral organoids derived from iPSCs generated from cultured fibroblasts of affected individuals. We are performing transcriptomic and proteomic profiling of these brain organoids harboring CTR1R95H and other variants (CTR1L79P, CTR1Q125K) from other recently identified subjects. These studies include CRISPR-corrected isogenic controls as well as complete CTR1 knockout brain organoids. The evaluation of the impact of AAV6-SLC31A1 gene transfer on cerebral organoid phenotypes is also in progress, since the AAV6 serotype has high tropism for cerebral organoids. We will also employ a base editing molecular approach to correct the missense mutations as an alternative strategy for altering the disease phenotype. Furthermore, we are evaluating the survival and natural history of our recently developed mouse

model for CTR1R95H in advance of experimental therapeutics. Considering the absence of CTR1 deficiency models, our combined studies provide novel insight concerning 1) the brain-associated functions of CTR1, 2) disease mechanisms related to CTR1 deficiency, and 3) proof of principle for AAV-mediated gene therapy in affected patients.

20 – Vikas Malik – Medicine

"Leveraging Stem Cell Intrinsic Immunity and ADAR1 Editing to Combat HIV-1."

HIV-1 remains a global challenge due to drug resistance and the limitations of antiretroviral therapy, highlighting the need for new strategies to eliminate viral RNA and proteins. While most antiviral research focuses on differentiated cells relying on interferon signaling, human embryonic stem cells (hESCs) exhibit intrinsic resistance to viral infections, offering a unique model to explore novel antiviral mechanisms. Our pilot data suggest that, within just 2 hours of HIV-1 exposure, hESCs initiate a rapid, sustained transcriptional response, contrasting sharply with HIV-1 infection in highly susceptible SupT1 cells. Using genome-wide CRISPR-inhibition and activation (CRISPRi/a) in hESCs, we have identified stem cell-specific anti- and pro-HIV-1 factors, revealing previously unexplored intrinsic immunity mechanisms. This project will further examine how CRISPR-screen candidates enhance HIV-1 elimination in hESCs compared to differentiated cells.

Notably, ADAR1, a pluripotency-inducing factor and the primary enzyme responsible for A-to-I editing of double-stranded RNA (dsRNA) of both host and HIV-1 origins, is significantly induced upon HIV-1 infection in hESCs. A-to-I RNA editing could affect RNA structure, replication, degradation, RNA-silencing, pre-mRNA splicing, and mRNA translation. While ADAR1 has been primarily reported as a pro-HIV-1 factor except for only one study as an anti-HIV-1 factor, our study using ADAR1-knockout (AKO) hESCs demonstrates its antiviral role. Many interferon-stimulated genes regulated in hESCs during HIV-1 infection are ADAR1 editing targets, placing ADAR1 at the intersection of pluripotency and antiviral immunity. This study aims to identify stem cell-specific anti-HIV-1 factors, viral RNA editing and degradation mechanisms, and host-viral proteomic interactions, advancing stem cell-based antiviral strategies and novel HIV therapeutics.

21 – Vikash Kumar Shah – Pulmonary Medicine

"Endothelial Antioxidant Responses in Stable and Variable Sleep."

Background: Variable sleep, a highly prevalent sleep pattern of high day-to-day variability in sleep timing, is associated with increased cardiovascular risk in observational studies. Importantly, sleep variability is a stronger predictor of mortality risk than sleep duration, and it does not correlate with sleep duration, suggesting that variability in sleep timing increases CV risk in a distinct manner. However, the causal evidence is lacking and mechanisms are unknown. We recently identified novel axis SRF/DCUN1D3/Cul3/Nrf2 as a mediator of impaired antioxidant responses in insufficient sleep. Surprisingly, we also noticed that, compared to habitual variable sleep timing, simply adhering to stable bedtimes for 6 weeks, while maintaining habitual 7-9 h sleep duration, reduces endothelial oxidative stress and inflammation in healthy women. This led us to investigate whether reducing bedtime variability, while maintaining habitual sleep duration, improves oxidative stress, inflammation, and upregulates antioxidant responses in endothelial cells (ECs).

Methods: We used direct assessment of the human endothelium (harvested venous ECs) and a randomized parallel design (maintain habitual irregular sleep defined as a 2-wk standard deviation [SD] of sleep onset timing >60 min by actigraphy vs. stabilize bedtime [SD ,â§45 min]) for 12 weeks in healthy participants (n=10). We assessed oxidative stress (CellROX Green), inflammation

(nuclear fluorescence of NF-ÔÅ´B), and antioxidant responses (RNAScope SOD, TXNRD-1, HMOX-1) in harvested ECs.

Results: Preliminary analysis suggests that EC oxidative stress and inflammation tend to be lower while antioxidant responses (SOD) mediated by SRF/DCUN1D3/Cul3/Nrf2 signaling tend to improve after sleep stabilization compared with variable sleep.

Conclusions: Stabilizing sleep schedule may improve endothelial function in individuals with habitual variable sleep, which overtime may reduce cardiovascular risk.

Abbreviation: Serum response factor (SRF); Defective in Cullin Neddylation-1 Domain Containing 3 (DCUN1D3); Cullin3 (Cul3); Nuclear factor erythroid 2, related factor 2 (Nrf2); Thioredoxin Reductase 1 (TXNRD-1); SuperOxide Dismutase (SOD); Heme Oxygenase-1 (HMOX-1)

22 – Yen-Chu Lin – Neuroscience and Behavior / Barnard College

"Greater neural dissimilarity between emotional stimuli in early visual areas is associated with symptoms of psychopathology in adolescents."

Adolescence is a period of heightened risk for psychopathology. Recent studies suggest shared neural correlates across mental disorders, particularly in frontolimbic networks involved in emotional processes. However, the visual network–crucial for extracting and processing sensory and emotional information–has been less of a focus in these studies. Here, using a transdiagnostic approach, we focus on visual pathways and investigate how individuals with psychopathology represent and differentiate between emotional categories.

We utilized imaging and questionnaire data from the Adolescent Brain Cognitive Development Study SM Release 4.0 (11-12 year-old; n=4953 after quality control). Symptoms of psychopathology were based on the Child Behavior Checklist total problem scores. Participants performed an emotional n-back working memory task with three emotional categories (happy, fearful, and neutral faces) with high and low memory loads. Representational similarity analysis was used to examine dissimilarities in neural representations of emotional face categories in the visual network and its subregions. The associations between dissimilarity, levels of psychopathology, and task performance were tested using linear models.

The analysis of neural representations of emotional face categories showed greater dissimilarities in individuals with more symptoms of psychopathology in the visual network. Behaviorally, we also found that greater dissimilarities in emotional categories were associated with diminished working memory performance. Moreover, the primary visual cortex exhibited the most robust patterns of dissimilarities between categories. Together, our results suggest distinct patterns of emotional information processing in early visual areas in adolescents with more symptoms of psychopathology, illustrating the importance of the visual network in psychopathology studies.

23 – Axinia Radeva & Jessica Eaton – Research Computing Services - CUIT

24 – Columbia University Postdoctoral Society

Session III - MBTI Workshop
1:30 - 3:00 PM
VEC 401
Presenters: Rosa Chavarro & Ericka Peterson

In this interactive session, Postdocs and Associate Research Scientists will explore their Myers-Briggs Type Indicator | Global Step I (MBTI®) personality types to build self-awareness, improve communication, and strengthen team dynamics. Participants will engage in guided activities and receive their personalized MBTI® Global Step I report.

Contact Information
Office for Postdoctoral Affairs and Early Career Research
212-305-4073
postdocaffairs@columbia.edu