

# SANS CONFERENCE 2026

Advancing Brain Health - From Molecules to Mind

MARRIOTT HOTEL, CRYSTAL TOWERS, CAPE TOWN, SOUTH AFRICA



*Southern African  
Neuroscience Society*



## Abstract Booklet



## *Welcome!*

On behalf of the Local Organising Committee, it is my privilege to invite you once again to an exciting and inspiring gathering of the SADC neuroscience community. We look forward to welcoming researchers, clinicians, educators, students, industry partners, policymakers, and innovators from across Southern Africa and around the world as we come together to advance neuroscience research, education, and collaboration.

The theme of the SANS Conference 2026, "Advancing Brain Health: From Molecules to Mind," reflects the breadth and diversity of modern neuroscience. From fundamental discoveries at the molecular and cellular level to clinical translation, population neuroscience, and innovations in brain health, the conference aims to showcase the latest advances that are transforming our understanding of the nervous system and improving outcomes for neurological and psychiatric disorders. We hope to foster an environment that encourages scientific excellence, interdisciplinary collaboration, and the exchange of innovative ideas that will shape the future of neuroscience in Africa and beyond.

The scientific programme has been designed to reflect the rapidly evolving landscape of neuroscience and will feature internationally recognised speakers, symposia, workshops, oral and poster presentations, panel discussions, and networking opportunities. In addition to the scientific programme, the conference will provide dedicated opportunities for students and early-career researchers through professional development workshops, mentoring sessions, travel awards, career discussions, and networking events. These activities reflect the Society's ongoing commitment to nurturing the next generation of African neuroscientists and strengthening neuroscience capacity across the region.

This year's conference is particularly significant as it serves as a prelude to the IBRO World Congress 2027, which SANS has the distinct honour of hosting in Cape Town for the first time in Africa. The IBRO World Congress is the flagship international meeting of the global neuroscience community, bringing together thousands of neuroscientists from across the world. As we prepare to welcome the international neuroscience community to South Africa in 2027, the SANS Conference 2026 provides a unique opportunity to showcase the excellence, innovation, and growing impact of neuroscience research across Southern Africa. It also offers a platform to strengthen regional collaborations, engage with emerging scientific leaders, and build momentum towards a landmark event that will leave a lasting legacy for neuroscience on the African continent.

We warmly invite researchers, clinicians, educators, students, healthcare professionals, industry partners, funding agencies, and organisations committed to brain research and brain health to be part of this exciting event. Your participation and contributions will help create a vibrant scientific meeting that celebrates excellence, promotes innovation, fosters meaningful collaborations, and strengthens the neuroscience community throughout Southern Africa.

We look forward to welcoming you to Cape Town for the SANS 2026, where together we will explore new discoveries, build lasting collaborations, and advance our shared vision of improving brain health through neuroscience.

Yours sincerely,

Associate Professor Rachael Dangarembizi  
Chair of the Local Organising Committee  
Southern African Neuroscience Society (SANS) Conference 2026



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## PROGRAMME

**DAY 1: THURSDAY 17 SEPTEMBER 2026**

|               |                                                                                                                                                        |  |
|---------------|--------------------------------------------------------------------------------------------------------------------------------------------------------|--|
| 08:30 – 13:00 | Pre-Symposium Workshop – Mental Health Capacity Building for Primary Health Care<br>Pre-Symposium Workshop – Neuromics and Bioinformatics              |  |
| 14:00 – 15:00 | <b>Arrival and Registration</b>                                                                                                                        |  |
| 18:00 – 18:30 | <b>Opening ceremony and welcome – Chair A/Prof Rachael Dangarembizi</b>                                                                                |  |
| 18:30 – 19:30 | <b>Opening Keynote</b><br>Professor Graham Fieggen<br><i>The Neuroscience Institute at UCT: A decade of discovery, and our strategy for the future</i> |  |
| 19:30 – 21:00 | <b>Cocktail Dinner and Networking</b> ( <i>light meal provided</i> )                                                                                   |  |

**DAY 2: FRIDAY 18 SEPTEMBER 2026**

|               |                                                                                                                                                                                                                                                |                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                   |
|---------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| 08:00 – 08:30 | Registration                                                                                                                                                                                                                                   |                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                   |
|               | <b>Plenary Session - Chair Dr Dorit Hockman</b>                                                                                                                                                                                                |                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                   |
|               | <i>Matthijs Verhoog</i>                                                                                                                                                                                                                        | <i>Human neurons undergo protracted functional maturation into adulthood</i>                                                                                                                                                                                     |                                                                                                                                                                                                                                   |
| 08:30 – 10:00 | <i>Caroline Ross</i>                                                                                                                                                                                                                           | <i>Inducible formation of fusion transcripts upregulates haploin sufficient CHD2 gene expression</i>                                                                                                                                                             |                                                                                                                                                                                                                                   |
|               | <i>Chris Dulla</i>                                                                                                                                                                                                                             | <i>When your Ferrari breaks down: Cell type-specific metabolism in PV+ interneurons powers 'high performance' inhibition</i>                                                                                                                                     |                                                                                                                                                                                                                                   |
| 10:00 – 10:30 | <b>Tea Break</b>                                                                                                                                                                                                                               |                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                   |
|               | <b>Plenary Session - Chair A/Prof Joseph Raimondo</b>                                                                                                                                                                                          |                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                   |
|               | <i>Monray Williams</i>                                                                                                                                                                                                                         | <i>HIV-1 viral diversity and mechanisms relevant to HIV neuropathogenesis: integrating preclinical and clinical evidence</i>                                                                                                                                     |                                                                                                                                                                                                                                   |
| 10:30 – 12:00 | <i>Blessing Mangale</i>                                                                                                                                                                                                                        | <i>Direct mycobacterial interaction with immature neural cells: A proteomic investigation into CNS tuberculosis neuropathogenesis.</i>                                                                                                                           |                                                                                                                                                                                                                                   |
|               | <i>Rachael Dangarembizi</i>                                                                                                                                                                                                                    | <i>Mechanisms underlying brain injury in fungal meningitis (A BIOCUM Africa - sponsored lecture)</i>                                                                                                                                                             |                                                                                                                                                                                                                                   |
| 12:00 – 13:00 | <b>Panel discussion - Beyond Discovery: Translating Brain Research into Societal Impact</b><br>Chair – A/Prof Sylvester Omoruyi<br>Panellists – Bonginkosi Chiliza; Sian Hemmings; Basil Enicker; Tjerignimin Adissa Silue and Brian Harvey    |                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                   |
| 13:00 – 14:00 | <b>Lunch</b>                                                                                                                                                                                                                                   |                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                   |
| 14:00 – 15:30 | <b>Concurrent Oral Session</b><br><b>Crystal Room 1</b><br><br><b>Chair – Prof Pradeep Kumar</b><br><br><i>Tabo Mwila</i><br><i>Amy van der Hoven</i><br><i>Tosin A. Olasehinde</i><br><i>Zethembiso B. Ngcobo</i><br><i>Victoria Williams</i> | <b>Concurrent Oral Session</b><br><b>Crystal Room 3</b><br><br><b>Chair – A/Prof Bonginkosi Chiliza</b><br><br><i>Blessing Gumbu</i><br><i>Oladiran Olateju</i><br><i>Simran Atulkumar Patel</i><br><i>Taskeen Fathima Docrat</i><br><i>Tiaan Anthonie Steyn</i> | <b>Concurrent Oral Session</b><br><b>Boardroom 6</b><br><br><b>Chair – Dr Zama Msibi</b><br><br><i>Rinae P. Gadisi</i><br><i>Sancia Govender</i><br><i>Kamohelo L. Mokhudo</i><br><i>Amanda Miya</i><br><i>Shameemah Abrahams</i> |
| 15:30 – 16:30 | <b>Break/ Poster Session</b>                                                                                                                                                                                                                   |                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                   |
| 16:30 – 18:00 | <b>SANS Annual General Meeting 2026</b>                                                                                                                                                                                                        |                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                                   |

## PROGRAMME

**DAY 3: SATURDAY 19 SEPTEMBER 2026**

|               |                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                         |                                                                                                                                                                                                                          |
|---------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| 08:00 - 08:30 | <b>Registration</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                     |                                                                                                                                                                                                                          |
| 08:30 - 10:00 | <b>Plenary Session – Chair A/Prof Monray Williams</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   |                                                                                                                                                                                                                          |
|               | <i>Peter Kamerman</i>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                   | <i>Pain in keloid scars</i>                                                                                                                                                                                              |
|               | <i>Victoria Madden</i>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  | <i>Studying pain in the whole person: spinal signalling, psychology, and immune signalling</i>                                                                                                                           |
|               | <i>Ronald Kipkirui</i>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  | <i>Altered executive functioning, maladaptive metacognitions, and psychopathology in gambling disorder</i>                                                                                                               |
| 10:00 - 10:30 | <b>Break</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            |                                                                                                                                                                                                                          |
| 10:30 - 12:00 | <b>Plenary Session - Chair A/Prof Oladiran Olateju</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  |                                                                                                                                                                                                                          |
|               | <i>Jenna Annandale</i>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                  | <i>Antenatal exposure to maternal depressive symptoms and early-life longitudinal immune markers and behaviour in South African children</i>                                                                             |
|               | <i>Pradeep Kumar</i>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                    | <i>Functionalized Neuromaterials for Neurodegenerative Interventions and Effective Neuroregeneration</i>                                                                                                                 |
|               | <i>Tjerignimin A. Silue</i>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             | <i>Building Sustainable Brain Health Innovation Ecosystems in Africa: From Research Discovery to Patient Access and Economic Development</i>                                                                             |
| 12:00 - 13:00 | <b>Special Session: Challenging false dichotomies of caregiving in science</b><br><i>Chairs: Dr Sahba Besharati, Dr Dorit Hockman</i>                                                                                                                                                                                                                                                                                                                                                                                                                   |                                                                                                                                                                                                                          |
| 13:00 - 14:00 | <b>Lunch</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                            |                                                                                                                                                                                                                          |
| 14:00 - 15:30 | <b>SANS Student Oral Competition</b><br><b>Chairs: A/Prof Ajonijebu C. Duyilemi and Dr Amalia Awala</b><br><br><i>Aminu Musa - Nelson Mandela University</i><br><i>Jessica Kanyemba - University of Cape Town</i><br><i>Mickey Banda - University of the Witwatersrand</i><br><i>Zolani Jacobs - University of the Western Cape</i><br><i>Livhuwani Mabasha - North-West University</i><br><i>Tusekile S Kangwa - University of Cape Town</i><br><i>Hendrina M Marole - Sefako Makgatho University</i><br><i>Yanga B Pato - University of Cape Town</i> |                                                                                                                                                                                                                          |
| 15:30 - 16:30 | <b>Break/Poster Session</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                             |                                                                                                                                                                                                                          |
| 16:30 - 18:00 | <b>Concurrent Oral Session<br/>Crystal Room 1</b><br><br><b>Chair – Dr Basil Enicker</b><br><br><i>Joel Defo</i><br><i>Alexander Sittmann</i><br><i>Justine van Greenen</i><br><i>Taskeen F. Docrat o.b.o. Elias Chipofya</i><br><i>Amalia Awala</i>                                                                                                                                                                                                                                                                                                    | <b>Concurrent Oral Session<br/>Crystal Room 3</b><br><br><b>Chair – Dr Tanya Calvey</b><br><br><i>Naadiya Seedat</i><br><i>Seipati Mokhosi</i><br><i>Ngoni Magate</i><br><i>Yomelela Golimpi</i><br><i>Demi Govender</i> |
| 18:00 - 19:00 | <b>Free Time</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                        |                                                                                                                                                                                                                          |
| 19:00 - 20:00 | <b>Closing Keynote – Chair Dr Zama Msibi</b><br><i>Dr Sahba Besharati</i><br><i>The social origins of bodily self-consciousness</i>                                                                                                                                                                                                                                                                                                                                                                                                                     |                                                                                                                                                                                                                          |
| 20:00 - 22:00 | <b>Conference Dinner + Prize Giving</b>                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 |                                                                                                                                                                                                                          |

## OPENING KEYNOTE SPEAKER



### PROF. GRAHAM FIEGGEN

*Director of Neuroscience Institute,  
University of Cape Town South Africa*

### ***The Neuroscience Institute at UCT: A decade of discovery, and our strategy for the future***

The Neuroscience Institute (NI) was founded to strengthen brain research at the University of Cape Town around a simple conviction: that understanding the brain in an African context is among the most urgent and exciting challenges in science, and one that matters everywhere.

Based in a purpose-built facility on the Groote Schuur Hospital campus and integrated with its clinical platform, including the Red Cross War Memorial Children's Hospital, the NI brings together a wide range of disciplines including clinicians, basic scientists, imaging scientists, social scientists and creative thinkers around a shared interest in brain health across the lifespan.

This talk reflects on a decade of discovery. Over ten years, work by NI members has appeared in more than 2,700 peer-reviewed publications, contributing in areas as diverse as tuberculous and cryptococcal meningitis, the impact of HIV and other factors on the developing brain, paediatric brain injury, the genomics of African populations, cellular mechanisms of epilepsy and cortical maturation, and the classification of mental illness. What links these contributions is a common model: access to clinically distinctive African populations, deep phenotyping, the integration of imaging, genomics and cellular neuroscience, and a commitment to translating discovery into diagnostics, treatment and policy, while helping train the next generation of African neuroscientists.

Looking ahead, our 2026 - 2030 strategy sets out how we intend to convert that momentum into enduring impact: African-led discovery on neglected brain conditions, brain health across the whole lifespan, equitable genomics and precision medicine, work spanning the single cell to the whole patient, responsible use of artificial intelligence, and a shortened arc from discovery to clinic, policy and public trust. Our vision is an Africa where people achieve their full potential through brain health and we seek to build partnerships with all who share this purpose.

## CLOSING KEYNOTE SPEAKER



**DR. SAHBA NOMVULA BESHARATI**

*Co-director of Wits NeuRL,  
University of the Witwatersrand*

***The social origins of bodily self-consciousness***

What makes you self-aware?

In recent decades, the research traditions of (first-person) embodied cognition and of (third-person) social cognition have approached the study of self-awareness with relative independence. However, recent experimental and neuroanatomical evidence has converged to suggest that self-awareness is grounded in the body and is socially mediated.

In this talk, I will first draw on research conducted using neurological disorders of self-awareness that offer a unifying perspective to investigate the contribution of embodiment and social cognition to self-awareness. Next, a series of cross-cultural studies using neurophysiological methods and immersive virtual reality will be presented to argue that sociocultural factors are embedded in the development of bodily self-awareness. Lastly, results from longitudinal cohort studies in community-based setting in South Africa will be used to highlight the role of social-emotional processes in early childhood and adolescence as critical developmental periods for the development of self-awareness.

In doing so, I will demonstrate that expanding representation in cognitive neuroscience is not only a matter of equity - but that social diversity provides a unique avenue to study how human social behaviour can shape the neural and embodied basis of self-awareness.

## PLENARY SPEAKERS

**MS. JENNA ANNANDALE***Neuroscience Institute, University of Cape Town****Antenatal exposure to maternal depressive symptoms and early-life longitudinal immune markers and behaviour in South African children***

Antenatal depressive symptoms are prevalent in low- and middle-income countries (LMICs) and associated with child behaviour. Increasing evidence indicates that immune dysregulation, may be involved in the association of antenatal depressive symptoms with behaviour in children. Immune investigation in children in these associations is lacking, especially longitudinally and in LMICs. This study aims to bridge these gaps and investigate the associations of antenatal depressive symptoms with a broad range of immune markers antenatally and in children over time and with child behaviour.

A sub-sample of mother-child pairs (n=319) from the Drakenstein Child Health Study were included. Pregnant women were enrolled in their second trimester and followed longitudinally. Antenatal depressive symptoms were measured at enrolment using the Beck Depression Inventory 2nd edition. Eighteen serum immune markers were measured in pregnant mothers and children at 8 weeks, 2-, 3- and 5-years. Child behaviour was assessed at 5 years with the Child Behaviour Checklist. Antenatal depressive scores were positively associated with immune activation in mothers, and with child behaviour problems. Higher antenatal depressive scores were associated with persistently elevated neutrophil gelatinase-associated lipocalin (NGAL) levels in children. Elevated maternal antenatal IL-10 levels were associated with externalising behaviours, while lower antenatal chitinase-3-like protein-1 levels were associated with internalising behaviours. Higher longitudinal child IL-4, IL-5, IL-6, IL-10, IL-13, and MMP-9 levels were associated with child behaviour outcomes.

Antenatal depressive symptoms are associated with altered immune markers in mothers during their second trimester and in their children during early childhood as well as their behaviour. Alterations in maternal antenatal immune markers and children were associated with child behaviour. Consistently elevated NGAL levels in children suggest long-lasting influence of antenatal depressive symptoms. Elevated antenatal IL-10 levels are potentially mechanistically involved in the association between antenatal depressive symptoms and child externalising behaviour.

## PLENARY SPEAKERS

**ASSOC. PROF. RACHAEL DANGAREMBIZI**

*Neuroscience Institute,  
University of Cape Town*

***Mechanisms underlying brain injury in fungal meningitis***

Cryptococcal meningitis is a life-threatening fungal infection of the brain and a leading cause of brain injury and mortality among people living with HIV/AIDS. Despite its devastating clinical consequences, the biological mechanisms driving neuropathology during cryptococcal infection are not fully understood.

Here we combined multi-omics analyses, advanced bioimaging approaches, and cell-type-specific molecular profiling across complementary models of neurocryptococcosis to define the mechanisms underlying brain injury.

Our findings demonstrate that neuropathology in cryptococcosis arises through multiple interconnected processes that include an early state of neuroimmune quiescence that permits fungal expansion, progressive inflammatory dysregulation, neurometabolic dysfunction, and disruption of tissue homeostasis. We further identify mechanisms underlying the disruption of glymphatic fluid transport thus providing mechanistic insights into the development of elevated intracranial pressure and hydrocephalus.

Collectively, these findings advance our understanding of neuropathogenesis of cryptococcal infection, demonstrating that disease progression is driven by a complex interplay of neuroimmune dysfunction, neurometabolic perturbation, and impaired neurofluid homeostasis.



## PLENARY SPEAKERS

**PROF. CHRIS DULLA***School of Medicine, Tufts University, USA****When your Ferrari breaks down: Cell type-specific metabolism in PV+ interneurons powers 'high performance' inhibition***

Parvalbumin-positive interneurons (PV-INs) regulate neuronal and circuit activity. Their loss and dysfunction are observed across neurological conditions, including traumatic brain injury (TBI), epilepsy, Alzheimer's disease, and schizophrenia, but the mechanisms underlying their dysfunction are not clear. PV-IN metabolic demands are thought to be unique due to their high level of electrical activity, but whether they are metabolically specialized and if this specialization make them vulnerable to loss and dysfunction is largely unknown. Their dysfunction is often associated with a loss of parvalbumin (PV) protein, making it challenging to discriminate between cell loss and PV protein loss, and creating a barrier to study PV-INs subtype-specific metabolism.

Here, we use single-nucleus RNA-sequencing (snRNAseq), coupled with in situ spatial metabolomics, to demonstrate that PV-INs are metabolically specialized compared to other neuronal subtypes and experience unique metabolic stress after TBI in a mouse model. We generate a novel PV-IN transcriptional identity module comprised primarily of genes encoding specialized ion channels, metabolic enzymes, and synaptic machinery, that identifies heterogenous subsets of injury-associated PV-INs with loss of PV-IN transcriptional identity.

We show that changes in metabolic specialization are coupled to changes in transcriptional identity in PV-INs and validate our findings using cell type-resolved in situ MALDI-TAG metabolomics. Mechanistically, we implicate the PV-IN-preferential transcriptional co-activator, Ppargc1a, as a potential key mediator of PV-IN transcriptional metabolic dysfunction. We also identify a family of long non-coding RNAs enriched in this subset of transcriptionally dysfunctional PV-INs that negatively correlates with PV-IN metabolic specialization. Lastly, we utilize these tools to interrogate published human TBI snRNA-seq data and identify analogous changes in PV-IN transcriptional and metabolic expression, underscoring the relevance of metabolic stress in PV-IN dysfunction in the pathology of TBI.

## PLENARY SPEAKERS



### PROF. PETER KAMERMAN

*Department of Physiology, University of the Witwatersrand*

#### ***Pain in keloid scars***

Keloids are scars that appear as hard, fibrous nodules that grow outside the margins of the original skin lesion. Keloids often present with debilitating pain that may be neuropathic in nature. Our research aims to characterise the pain in people living with keloids.

I will report on our most recent data obtained using questionnaires to characterise the frequency, characteristics, and impact of pain in keloid scars.

I will also report on a deeply phenotyped subset of study participants who were assessed for changes in sensory function (using quantitative sensory testing) and epidermal innervation in the scar tissue.

## PLENARY SPEAKERS

**PROF. PRADEEP KUMAR**

*Department of Pharmacy, University of the Witwatersrand*

***Functionalized Neuromaterials for Neurodegenerative Interventions and Effective Neuroregeneration***

Neuromaterial strategies, platforms, and interventions provide a unique and efficient way to overcome the various brain delivery related challenges, e.g., the low permeability through the blood brain barrier or absence of a neuro-conductive microenvironment which can be overcome by a rational choice of intelligent biomaterials and surface functionalization of such platforms with suitable physical, chemical, and molecular cues. The most effective neurotherapeutic approaches can be designed by incorporating the very complex neuroactive entities into nanostructures with directional cues and robust material characteristics for predetermined and site-specific release of the drugs.

This plenary talk will present several such strategies developed over the last decade including pH-sensitive platforms for the delivery of interferon- $\beta$  (multiple sclerosis), polymer coated cerium oxide nanoparticles as superantioxidant agents (Parkinson's disease), amantadine-loaded polylactide)-polymethacrylate nanospheres (amyotrophic lateral sclerosis), implantable nanoenabled multipolymeric scaffold (AIDS dementia complex), nanoliposomes functionalized with chelating ligands for neurotoxicity modulation of  $\beta$ -Amyloid aggregates (Alzheimer's disease), apo-lactoferrin-Galantamine proteo-alkaloid conjugate (Alzheimer's disease), nicotine-gelospheres compressed into polymeric matrix (Parkinson's disease), interpolymeric blend/nanoenabled levodopa delivery systems, and highly functional neuromaterials platforms for neurotrauma intervention covering spinal cord, peripheral nerve, and brain injuries.

The advanced neuromaterial platforms tested in these research initiatives demonstrated immense potential of nano and material interventions in improving the neurological, neurochemical, and behavioral outcome inherent to effective and efficient neuro-protective, -regenerative, -reparative, and -restorative therapies.

## PLENARY SPEAKERS



### ASSOC. PROF. VICTORIA MADDEN

*Neuroscience Institute, University of Cape Town*

***Studying pain in the whole person: spinal signalling, psychology, and immune signalling***

Approximately 1 in 5 adults lives with persistent pain, and painful conditions are a leading cause of disability.

Pain is recognised to be a complex, biopsychosocial experience that is potentially influenced by central nervous signalling, yet our insight to the interactions between psychological processes and central pain-related signalling remains limited.

This talk will present research that used a human surrogate model of spinal long-term-potentialiation to investigate interactions between clinical pain, central pain-related signalling, psychological variables, and blood cytokine expression.

It will discuss our findings using this model in laboratory and para-clinical contexts, and explore the next steps to better understand the biopsychosocial experience that is pain.

## PLENARY SPEAKERS

**MR. BLESSING MANGALE**

*Institute of Infectious Diseases and Molecular Medicine,  
University of Cape Town*

***Direct Mycobacterial Interaction with Immature Neural Cells: A Proteomic Investigation into CNS Tuberculosis Neuropathogenesis.***

Central nervous system tuberculosis (CNS-TB) neuropathogenesis has been characterized almost exclusively through meningeal inflammation, vasculitis, and raised intracranial pressure. Postmortem detection of *Mycobacterium tuberculosis* (Mtb) within brain parenchyma, beyond the meningeal compartment, raises the possibility that direct Mtb-neural cell interaction contributes to neuropathology independently of the inflammatory cascade. Emerging models of Mtb infection in human neural progenitor cells and organoids have shown direct bacterial internalization, but the proteomic response of immature neural cells to this interaction remains largely uncharacterized. The study aims to characterize the proteomic response of undifferentiated SH-SY5Y cells, as a neural progenitor-like model, to direct exposure to *Mycobacterium bovis* BCG, identifying pathways perturbed independently of meningeal inflammation. Undifferentiated SH-SY5Y cells were exposed to BCG at a multiplicity of infection of 10, selected by widefield fluorescence microscopy confirming bacterial association with SH-SY5Y cells. Samples were profiled by DIA-MS. Data were log-transformed, filtered for 70% valid values, and analyzed by LIMMA (adjusted  $p \leq 0.05$ ). Significant proteins underwent hierarchical clustering and enrichment analysis, focusing on the 6-hour response. Of 7,646 identified proteins, 3.5% were mycobacterial. At 6 hours, 106 proteins were significantly differentially expressed (54 upregulated, 52 downregulated). Upregulated proteins were enriched for RNA processing, spliceosome activity, mitochondrial matrix function, and metabolic adaptation, consistent with a stress-associated response. Downregulated proteins showed a neuronal signature, enriched for nervous system development, axon guidance signaling (ROBO/RAC1/CDC42/RAP1), cell adhesion/cadherin binding, and vesicle-mediated trafficking (RAB5A/7A/9A/10/18), consistent with dysregulated growth cone dynamics and impaired neurite/synaptic connectivity. Direct BCG exposure of immature neuron-like cells triggers proteomic remodeling, a stress-associated response alongside dysregulation of neurodevelopmental and connectivity pathways, offering a plausible mechanism linking mycobacteria-neural progenitor interaction to persistent CNS-TB deficits, independent of meningeal inflammation. These hypothesis-generating findings warrant validation via microscopy-based neurite/synapse quantification and comparison with differentiated neuronal models.

## PLENARY SPEAKERS

**DR. RONALD KIPKIRUI NGETICH**

*Institute for Human Development, Aga Khan University, Kenya*

***Altered executive functioning, maladaptive metacognitions, and psychopathology in gambling disorder***

Gambling disorder (GD) is linked to disrupted cognitive functioning and maladaptive neural alterations, particularly in the prefrontal cortex. However, findings on executive function are mixed, partly because of the diverse task paradigms used. This study aimed to assess executive functioning in GD using both simpler and more cognitively demanding tasks, while analysing vulnerabilities such as psychopathology and maladaptive metacognition side by side.

A cross-sectional online study was conducted in a UK community sample recruited via Prolific (N = 219). Participants were classified into healthy control (HCG), low-risk (LRG), high-risk (HRG), and GD groups (GDG) based on PGSI scores. Self-report measures assessed impulsivity (BIS-R-21-SF), psychopathology (DASS-21), compulsivity (DSM-5 OCPD criteria-based items), and gambling-related metacognitions (MGQ). Executive functions were evaluated using domain-specific tasks: working memory (2-back, 3-back, and digit span), cognitive control (Go/No-Go), and cognitive flexibility (Wisconsin Card Sorting Task).

Higher gambling severity was associated with poorer cognitive flexibility and impaired high-load working memory performance, with healthy controls outperforming the high-risk and GD groups. Compulsivity and psychopathology increased with gambling severity. Positive and negative metacognitions were higher in the high-risk and GD groups than in the healthy control and low-risk groups and were positively correlated with PGSI scores. However, no between-group differences were observed in cognitive control or impulsivity.

Gambling disorder reflects a convergence of vulnerabilities: disrupted high-load WM, decreased cognitive flexibility, elevated compulsivity, and maladaptive metacognitions.

## PLENARY SPEAKERS

**DR. CAROLINE ROSS**

*Molecular and Cell Biology Department, Neuroscience Institute, University of Cape Town*

***Inducible formation of fusion transcripts upregulates haploinsufficient CHD2 gene expression***

Precise dosage of chromatin regulators is essential for normal brain development. CHD2 exemplifies this requirement: CHD2 haploinsufficiency causes developmental and epileptic encephalopathy (DEE), intellectual disability, and developmental delay, whereas excessive CHD2 expression is also deleterious. Therapeutic strategies therefore require controlled restoration of CHD2 levels without triggering overdose. Modes of action of long noncoding RNAs (lncRNAs) remain poorly understood, yet many regulate nearby genes in cis. We explored this principle in the CHASERR-CHD2 axis, where CHASERR is a broadly expressed lncRNA transcribed immediately upstream of CHD2 that acts as a cis-repressor of CHD2 and is essential for mouse viability. Complete loss of Chaserr causes lethal CHD2 overexpression, highlighting the need for dosage-sensitive modulation. We designed antisense oligonucleotides (ASOs) targeting conserved motifs in the terminal exon of CHASERR to achieve controlled derepression of CHD2. In parallel, we generated a mouse model with deletion of the same conserved motifs to genetically validate mechanism and dosing effects. Molecular, transcriptomic, chromatin, and behavioral analyses were performed across multiple cell lines and in Chd2+/- mice following perinatal ASO administration. ASO treatment induced transcriptional readthrough from CHASERR into CHD2, generating a fusion transcript joining CHASERR to CHD2. This transcript was exported to the cytoplasm and translated into full-length CHD2 protein. Deletion of the same motifs in mice phenocopied the ASO effect, increasing CHD2 protein without the deleterious consequences associated with complete CHASERR loss. Fusion transcripts were also expressed endogenously and induced in activated neurons. Their constitutive induction altered neuronal gene expression and chromatin accessibility. In Chd2+/- mice, perinatal ASO treatment upregulated CHD2 toward physiological levels and alleviated behavioral phenotypes caused by CHD2 haploinsufficiency. Fusion-inducing ASOs represent a completely novel RNA therapeutic strategy that reprograms transcriptional termination in the brain to finely tune endogenous gene dosage. This approach enables therapeutic CHD2 restoration while avoiding overdose and may provide a broadly relevant platform for haploinsufficient neurodevelopmental disorders.

## PLENARY SPEAKERS

**DR. TJERIGNIMIN ADISSA SILUE***National Institute of Neurological Disorders and Stroke, USA****Building Sustainable Brain Health Innovation Ecosystems in Africa: From Research Discovery to Patient Access and Economic Development***

Neurological disorders represent a growing health, social, and economic challenge across Africa, with consequences extending beyond individual patients to families, health systems, workforce productivity, and human capital development. At the same time, rapid advances in biomarkers, precision medicine, gene and cell therapies, digital technologies, and translational neuroscience are transforming the prevention, diagnosis, and treatment of neurological disorders. Ensuring that African populations benefit equitably from these advances will require more than scientific discovery; it will require sustainable ecosystems capable of translating innovation into accessible and scalable health solutions. This presentation will examine four interconnected pillars necessary to strengthen brain health innovation across Africa. First, it will explore brain health as a development priority, highlighting the relationship between neurological disease burden, disability, workforce participation, caregiver burden, productivity, and economic development. Second, it will examine the pathway from discovery to delivery, considering the infrastructure, regulatory capacity, manufacturing, financing, and implementation challenges associated with translating emerging neurological technologies into patient care. Third, drawing on lessons from large-scale U.S. National Institutes of Health translational and clinical research programs and networks, the presentation will discuss opportunities to strengthen clinical research capacity through sustainable research infrastructure, workforce development, multisite networks, data systems, regulatory frameworks, and collaborative governance. Finally, it will propose a framework for building sustainable African brain health innovation ecosystems through partnerships among governments, academic institutions, communities, industry, funders, and international organizations. Africa has an opportunity not only to participate in the next generation of neuroscience innovation, but to shape it. Building locally responsive research and clinical ecosystems can strengthen scientific capacity while improving access to innovation and advancing broader health and economic development. Transforming discovery into impact will ultimately depend on creating systems that ensure scientific advances reach the patients and communities that need them most.

## PLENARY SPEAKERS

**DR. MATTHIJS VERHOOG***Neuroscience Institute, University of Cape Town****Human neurons undergo protracted functional maturation into adulthood***

Human cognitive development is uniquely prolonged, reflecting the extended postnatal maturation of the cerebral cortex where cell-type differentiation, synaptogenesis, myelination and transcriptional regulation all follow protracted developmental timelines. However, when human cortical neurons reach functional electrophysiological maturity and how their developmental trajectory compares to other species remains unknown.

Here we show through patch-clamp recordings of human temporal cortex from infancy to adulthood that supragranular pyramidal neurons exhibit pronounced neoteny of their functional properties, with physiological maturation continuing well into adulthood.

Comparing human and mouse developmental trajectories reveals human neurons are on a much slower developmental timeline, maturing physiologically hundreds of times slower than mouse and 2-6 times slower than would be predicted from anatomical brain growth differences between species. This extreme protraction of neurophysiological development provides a cellular basis for prolonged human cognitive maturation.

## PLENARY SPEAKERS

**ASSOC. PROF. MONRAY EDWARD WILLIAMS**

*Biomedical and Molecular Metabolism Research, North West University*

***HIV-1 viral diversity and mechanisms relevant to HIV neuropathogenesis: integrating preclinical and clinical evidence***

Despite effective antiretroviral therapy, HIV-associated brain injury (HABI) remains prevalent, particularly in regions where HIV-1 subtype C predominates. The contribution of HIV-1 viral diversity to the mechanisms underlying HABI remains poorly understood. This study integrated preclinical and clinical investigations to evaluate whether HIV-1 viral diversity influences pathways relevant to HABI. Preclinical investigations evaluated macrophage polarisation following acute stimulation with recombinant Tat and gp120 subtype B and C proteins, chronic infection with HIV-1 subtype A and B isolates, astrocyte inflammatory responses to Tat and gp120 subtype variants, and neuronal toxicity induced by Tat subtype C and CRF01\_AE. Clinical investigations in South African people living with HIV-1 subtype C examined Tat and Vpr sequence variation in relation to peripheral inflammatory markers, tryptophan-kynurenine (Trp-Kyn) metabolites, and exploratory cognitive domain performance. Across complementary preclinical models, HIV-1 proteins and viral strains induced innate immune activation and neuronal injury, with evidence of subtype- and strain-specific effects. Acute stimulation with Tat and gp120 increased macrophage polarisation without significant differences between subtype B and subtype C proteins, whereas chronic infection with the HIV-1 subtype A induced greater macrophage polarisation than subtype B. Astrocytes exposed to Tat and gp120 exhibited broadly comparable cytokine profiles between subtype pairs, although Tat subtype C elicited greater expression of selected inflammatory mediators. In neuronal models, Tat CRF01\_AE induced greater neurotoxicity than subtype C. Clinically, Tat sequence variation was associated with peripheral inflammatory markers, Trp-Kyn metabolites and cognitive domain performance, whereas Vpr variation demonstrated fewer and less consistent associations. Collectively, these findings provide preliminary evidence that HIV-1 viral diversity may influence inflammatory, immunometabolic and neurotoxic pathways relevant to HABI. The convergence of observations across experimental and clinical investigations supports further investigation into subtype diversity as a potential determinant of HIV neuropathogenesis, particularly in populations where non-subtype B HIV-1 predominates.

ORAL  
PRESENTATIONS



## ORAL PRESENTATIONS

**Amalia Awala**

**University of Cape Town**

### Background:

Cryptococcal meningitis (CM) remains a major cause of HIV-associated mortality in Sub-Saharan Africa and a significant contributor to neurological morbidity among affected patients. Brain injury in CM is often linked to dysregulated inflammation, yet the contribution of resident brain immune cells remains poorly understood. In particular, it is unclear how microglia, the innate immune sentinels of the brain, respond to invasion by *Cryptococcus neoformans*, the causative pathogen. We therefore used complementary rodent and human brain-based experimental models to investigate microglial-fungal interactions and characterise neuroimmune activation during cryptococcal infection.

### Methods:

Human and rodent organotypic brain slices (OBSs) were infected with a GFP/mCherry-expressing H99 strain of *C. neoformans*, and host-pathogen interactions were examined using immunofluorescent labelling of microglia (IBA1). Microglial activation was measured using nuclear localisation of the transcription factor, nuclear factor for interleukin-6 (NF-IL6): a robust marker for inflammatory signalling. Immune responses in *C. neoformans*-treated slices were compared to untreated controls and to slices treated with the canonical immunogen- lipopolysaccharide (LPS). Inflammatory responses were further validated using single-nucleus RNA sequencing and multiplex cytokine profiling.

### Results:

*C. neoformans* was readily internalised by microglia in OBSs, yet fungal uptake was not accompanied by inflammatory activation. Unlike LPS-treated slices, cryptococcal infection induced minimal microglial NF-IL6 nuclear translocation and negligible pro-inflammatory cytokine release, comparable to untreated controls. Single-nucleus RNA sequencing similarly revealed a muted inflammatory microglial response.

### Conclusions:

Our findings suggest that ineffective resident brain immune activation may be a key contributor to fungal persistence and neurological injury in cryptococcal meningitis.



## ORAL PRESENTATIONS

**Shameemah Abrahams**

**University of Cape Town**

### Background/Aim:

Traumatic brain injury (TBI) can present heterogeneously across patients. Biomarkers can be key to assist diagnostic and death determination in living and deceased individuals, respectively. Literature was investigated to determine the relevance of previous TBI biomarkers in both clinical and postmortem settings.

### Methods:

Peer-reviewed articles (2018-2026) investigating interleukin (IL)-6, IL-10, S100 calcium-binding protein B (S100b) or glial fibrillary acidic protein (GFAP) biomarkers in TBI clinical and postmortem cases were included. Per PRISMA, the Quality Assessment for Diverse Studies (QuADS) tool and the Grading of Recommendations, Assessment, Development, and Evaluations (GRADE) approach were used for quality appraisal and certainty of evidence, respectively. A subset of studies underwent a meta-analysis.

### Results:

From 2,043 titles screened, 109 articles were included, of which 18 underwent the meta-analysis. GFAP (66% of 109 articles) and S100b (52%) showed the strongest correlations with predicting TBI prognosis. However, studies on systemic markers IL-6 (22%) and IL-10 (15%) were limited and inconclusive. The meta-analysis for all biomarkers reported a non-significant pooled effect size with high heterogeneity (97.2%). The overall certainty of evidence for all the studies was rated as very low.

### Conclusion:

GFAP was the most consistent candidate for TBI prediction. However, inconsistencies across all studies limit applicability. Although TBI biomarkers are used as adjuncts to routine diagnostic testing, its relevance to the forensic setting is currently limited. Future research should investigate multiple biomarkers, standardise postmortem intervals and confirm with validated methods.



## ORAL PRESENTATIONS

**Mickey Banda**

**University of the Witwatersrand**

Student Competition

### Background:

HIV-associated neurocognitive disorders (HAND) arise from persistent neuroinflammation and neurodegeneration, including direct toxicity from HIV-1 proteins. While subtype B predominates in North America and Europe and subtype C is most prevalent globally, especially in sub-Saharan Africa, differences in their neurotoxic effects remain poorly characterised.

**Objective:** The primary objective of this study was to quantitatively assess and compare the neurotoxic effects of HIV-1 subtype B and C viral proteins in in vitro CNS models

### Methods:

A PRISMA-compliant systematic review identified 59 studies; 6 provided sufficient quantitative data for meta-analysis ( $k = 6$  independent effect sizes). We searched across PubMed, Scopus, and Web of Science from January 2006 to April 2026. Two independent reviewers screened studies and extracted data from in vitro CNS models exposed to HIV-1 proteins (Tat, Vpr, gp120, Nef) using Rayyan. Standardised mean differences (Hedges  $g$ ) were pooled using a random-effects model. Risk of bias was evaluated using the ToxRTool by two independent reviewers. Data were analysed using R 4.5.3.

### Results:

The pooled effect size (DerSimonian-Laird) indicated a large neurotoxic effect (Hedges  $g = 2.41$ , 95% CI 1.75–3.06;  $I^2 = 82.5\%$ ). Subgroup analysis suggested larger effect sizes for subtype B Tat ( $g = 2.8$ ,  $k = 3$ ) than for subtype C ( $g = 1.2$ ,  $k = 1$ ), although small sample sizes limit this comparison. Publication bias was explored cautiously due to the small number of studies ( $k = 6$ ). Egger's test did not indicate strong asymmetry ( $p = 0.08$ ). Most studies were classified as low risk of bias (ToxRTool Categories 1–2).

### Conclusion:

Tat was the most consistently studied protein and may exert comparatively stronger neurotoxic effects than other HIV-1 proteins. A notable absence of studies originating from Africa, despite the high prevalence of subtype C, highlights a critical geographic and subtype-specific research gap.

## ORAL PRESENTATIONS

**Elias Chipofya**

**Cape Peninsula University of Technology**

### Background:

Amyloid-beta ( $A\beta$ ) toxicity drives neurodegeneration via GSK-3 $\beta$  dysregulation and proteostatic collapse. While cells attempt to mitigate this by transcriptionally upregulating the Insulin-Degrading Enzyme (IDE), functional protein recovery is attenuated by the kinetic saturation of the proteostasis network, where epigenetic repressors maintain the IDE locus in a transcriptionally silenced state. This study investigates the ability of aqueous (RRA) and ethanolic (RRE) extracts, both prepared from fermented Rooibos, to bypass this barrier and restore the kinase-miRNA-IDE signalling network.

### Methods:

SH-SY5Y cells were pretreated (2 h) with RRA and RRE (60-150 mg/mL) before  $A\beta$  (40  $\mu$ M, 24 h) exposure. Gene expression (GSK-3 $\beta$ , MAPT, APOE4, miRNAs) was quantified via qPCR, while protein expression (P-GSK-3 $\beta$ /Total ratio, IDE, 68 kDa Tau) was assessed via Western blot. Statistical significance ( $P \leq 0.05$ ) was calculated using one-way ANOVA with Tukey's post hoc tests.

### Results:

$A\beta$  exposure induced significant GSK-3 $\beta$  transcriptional downregulation (Mean Diff. = 0.34; 95% CI [0.09 to 0.60];  $P=0.006$ ) and kinase overactivation (P-GSK-3 $\beta$ /Total ratio; Mean Diff. = -0.68;  $P<0.0001$ ). The miR-7110-5p was identified as an epigenetic gatekeeper of IDE that is pathologically upregulated by  $A\beta$  ( $P<0.0001$ ). Rooibos extracts successfully restored proteostatic flux: RRA150 attenuated GSK-3 $\beta$  (Mean Diff. = -0.45;  $P<0.0001$ ), while RRE60 significantly reversed MAPT suppression (Mean Diff. = 0.80;  $P \leq 0.05$ ) and decreased pathological 68 kDa Tau ( $P<0.0001$ ) in vitro. Furthermore, treatments restored miR-23-3p/miR-219-5p homeostasis ( $P<0.0001$ ) and modulated APOE4 (RRA60; Mean Diff. = 1.08;  $P<0.0001$ ).

### Conclusion:

Rooibos extracts act as metabolic facilitators that retune the GSK-3 $\beta$  regulatory circuit. By reprogramming the miR-7110-5p gatekeeper and stabilising the miRNA-IDE-Tau axis, Rooibos extracts provide a multi-target strategy to rescue proteostasis in Alzheimer's disease.

## ORAL PRESENTATIONS

**Thabisile Cindi**

**University of the Witwatersrand**

### Background:

Emergency room (ER) doctors work rigorous rotating shift patterns that negatively affect sleep and wellbeing. Furthermore, ER doctors in the Western Cape are exposed to high rates of violent trauma. Notwithstanding this, the effects of these conditions sleep and wellbeing remains under-researched, especially in low-to-middle income countries, like South Africa.

### Methods:

ER doctors across 4 public hospitals in the Western Cape were recruited. Demographics and validated questionnaires investigated: sleep quality (Pittsburgh Sleep Quality Index, PSQI), depression symptom risk (Patient Health Questionnaire-2, PHQ-2), sleep complaints (PHQ-3), shift work disorder risk (SWD) (Shift Work Disorder Questionnaire, SWDQ), and risk for post-traumatic stress disorder (PTSD) (PSQI-Addendum for PTSD (PSQI-A)).

### Results:

77 ER doctors (67.5% female,  $31.1 \pm 3.9$  years) participated, with a median of 5 [IQR: 3-8] years' work experience. The mean PHQ-2 score was 1.4 ( $\pm 1.4$ ), with 26% ( $n=20$ ) at risk for depressive symptoms ( $\text{PHQ-2} \geq 3$ ). Sleep complaints were present in 36% ( $n=27$ ) participants (PHQ-3). The global PSQI score was 8.2 ( $\pm 3.7$ ), with 71% ( $n=55$ ) classified as poor sleepers ( $\text{PSQI} > 5$ ). PTSD risk was present in 34.3% ( $n=24$ ) of participants (PSQI-A). Poor sleep was associated with higher risk for depressive symptoms and PTSD ( $p < 0.05$ ). A high risk for SWD was found for 63.6% ( $n=49$ ) participants and was associated with poor sleep quality, and higher risk of depressive and PTSD symptoms ( $p < 0.05$ ).

### Conclusion:

ER doctors report poor sleep and are at significant risk for depressive symptoms, PTSD and SWD. These factors may increase fatigue-related medical errors, adverse medical events, and workplace injury, while negatively impacting ER doctors' physical and mental health. Shift schedule interventions and sleep health education are suggested in order to preserve the health and wellbeing of ER doctors, and to ensure the safety of their patients.



## ORAL PRESENTATIONS

**Joel Defo**

**University of Cape Town**

Suicidality is a major global public health concern, yet its neurobiological basis remains incompletely understood. While neuroimaging studies implicate subcortical brain structures and genetic studies suggest heritability, their shared genetic architecture is unclear.

We leveraged genome-wide association study (GWAS) integrating data from the UK Biobank (suicide attempts, self-harm, and thoughts of life not worth living), FinnGen (suicide), and ENIGMA2 (subcortical brain volumes and intracranial volume). Genomic structural equation modelling identified latent genetic factors, followed by cross-phenotype meta-analyses at variant, gene, and network levels, with pathway and Gene Ontology analyses to characterize underlying biology.

A common genetic factor linking suicidality traits and subcortical volumes was identified; divided into two groups of phenotypes: suicidal traits from the UK Biobank as the first group; Suicide from FinnGen and subcortical brain volumes and intracranial volume being the second group. A positive genetic correlation was observed between suicide and intracranial volume ( $r_g = 0.21$ ,  $p = 0.004$ ).

Meta-analyses revealed 34 novel genome-wide significant variants within genes, including DCC, STIM2, MEAF6, and RSPO1. 50 hub genes were enriched in brain regions such as the anterior cingulate cortex, hippocampus, putamen, and substantia nigra. Additionally, 64 SNPs showed pleiotropic effects, and 22 hub genes were enriched in peripheral tissues, including the heart, pancreas, and basal ganglia. Pathway analyses highlighted processes related to blood-brain barrier permeability and inflammatory signalling.

These findings support a shared genetic architecture between suicidality and brain structure, implicating neuroinflammatory pathways as a key mechanism and highlighting brain-immune interactions as potential targets for intervention.

## ORAL PRESENTATIONS

**Taskeen Fathima Docrat**

**Cape Peninsula University of Technology**

### Background:

Neuroinflammation and dysregulated amyloid beta ( $A\beta$ ) processing are central to the pathogenesis of Alzheimer's disease (AD). There is a critical need for novel, safe interventions that target these molecular mechanisms. Rooibos (*Aspalathus linearis*), a South African herbal tea, is rich in polyphenols and has shown promise as a neuroprotective agent, but its effects on neuroinflammatory and amyloidogenic pathways remain underexplored.

### Methods:

Using a differentiated SH-SY5Y neuronal cell line as a translational in vitro model of AD, we investigated the effects of fermented Rooibos extracts (60 and 150  $\mu$ M, 2h) on  $A\beta$ -induced neuroinflammation (40  $\mu$ M, 24h). The anti-inflammatory agent BAY-11-7082 (2  $\mu$ M, 2h) was used as a positive control. We assessed cytokine profiles using an ELISA array. Inflammasome gene and protein expression (qPCR, Western blot), and regulatory microRNA-146a in neurodegeneration.

### Results:

Rooibos extracts reversed  $A\beta$ -induced suppression of key cytokines (IL-1 $\alpha$ , IL-6, TNF- $\alpha$ ), suggesting restoration of immune signalling. Rooibos pre-treatment downregulated inflammasome-related genes (NLRX1, NLRC4, CIITA, TIRAP, TRAF6, IRF1, MAPK13) and reduced IKK- $\beta$  protein levels, thereby restoring I $\kappa$ B- $\alpha$  levels and inhibiting NF- $\kappa$ B activation. Notably, Rooibos modulated microRNA-146a, a negative feedback regulator of the NF- $\kappa$ B pathway, thereby supporting its anti-inflammatory and neuroprotective effects. Ethanolic extracts demonstrated superior efficacy, likely due to enhanced bioavailability of active compounds.

### Conclusion:

Rooibos herbal tea extracts offer a novel approach to AD by attenuating neuroinflammation at the molecular level. These findings support the translational potential of Rooibos as a safe, accessible intervention for advancing brain health. Further in vivo and clinical studies are warranted to validate these promising results.



# ORAL PRESENTATIONS

**Rinae Prisca Gadisi**

**University of KwaZulu-Natal**

## Introduction:

Sodium iodide symporter (NIS), an intrinsic trans-membrane glycoprotein, has been identified as an antigen in auto-immune thyroid disease (AITD). Whilst primarily found in thyroid gland, widespread extra-thyroidal localization of NIS-mediated iodide transport has been documented in several mammalian tissues; however, NIS expression in human brain tissue has not been identified. Recently, our group was the first to demonstrate thyroid-specific proteins, thyroglobulin and thyroid stimulating hormone-receptor, in limbic vasculature and neurons of adult human brain. In the current study, we extend this investigation to immunolocalise NIS protein in adult human limbic regions.

## Methodology:

Normal human limbic tissues (amygdala, thalamus, frontal cortex, hippocampus, hypothalamus and cingulate gyrus) were acquired via postmortem with none of the patients' deaths associated with head trauma nor any diagnosed psychological abnormalities. Immunohistochemistry was used to immuno-detect NIS, and image analysis generated quantitative assessments of NIS protein within limbic structures.

## Results:

We demonstrated immuno-reactive NIS in neuronal cell bodies and axons of all limbic areas examined with no evidence of neuro-vasculature localisation. The amygdala group exclusively displayed immuno-reactive NIS in its neuronal nuclei. Quantitative inter-regional comparisons demonstrated strong NIS expression levels for the cingulate gyri and least expression levels for amygdalae.

## Conclusion:

Our novel identification of immuno-stained NIS in limbic structures expands on the mounting evidence of widespread expression of thyroid components, particularly in the paleo-mammalian cortex; further, potentially offering innovative therapeutic approaches during AITD and BD co-occurrence. On-going and future projects have been designed to compare NIS protein in human bipolar brain.



## ORAL PRESENTATIONS

**Yomelela Golimpi**

**Stellenbosch University**

Psychiatric genomics research is an expanding field in Africa, that seeks to understand the genetic contributions to psychiatric disorders such as schizophrenia and bipolar disorder. Although psychiatric genomics research has advanced globally, African populations remain underrepresented in genomics research. And the ethical, legal, social, cultural and policy implications of this research remain underexplored.

**Aims:** This study aims to (1) explore how community engagement is understood and practiced by researchers involved in psychiatric genomics research in Africa, (2) explore how researchers understand and address the intersecting influences of stigma, mistrust and cultural beliefs through their community engagement approaches, and (3) identify the unique ethical challenges and opportunities that researchers identify in psychiatric genomics research in Africa

**Methods:** This study will adopt a qualitative, exploratory design guided by the interpretivist paradigm. Semi-Structured interviews will be conducted with approximately 12-16 researchers recruited from the PGC Africa network in African countries through purposive sampling. Participants involved in psychiatric genomics research across African contexts will be eligible for inclusion. Data collection will be hybrid data collection approach. Data will be analysed using thematic analysis following Braun and Clarke's six-phase approach. The Community-Based Participatory Research (CBPR) framework will guide the interpretation of the findings.

**Ethics:** Ethical clearance will be obtained from the Stellenbosch University Health Research Ethics Committee (SU HREC) prior to data collection.

**Expected Outcomes:** This study will provide empirical evidence on how researchers understand and practice CE, navigate stigma, mistrust and cultural beliefs and identify the unique ethical challenges and opportunities, offering guidance for more inclusive psychiatric genomics research across Africa.



## ORAL PRESENTATIONS

**Demi Govender**

**University of Cape Town**

### Introduction:

Demyelinating disorders such as multiple sclerosis (MS), traumatic brain injury or opioid use disorder can cause lifelong morbidity. There are limited treatment options for these disorders with very few offering the hope for remyelination. Our recent preclinical findings showed that ibogaine increases myelin related proteins, myelin basic protein (MBP) and CNPase, following chronic morphine administration and a recent clinical case study showed ibogaine is able to decrease MS lesion volume. To date, ibogaine has not been tested against a demyelination model which is the aim of our current research.

### Methods:

The ethical approval for this study was issued by the Animal Research Ethics Committee at the University of the Cape Town (021\_021). A pilot study was conducted using 10 Long-Evans rats split into 3 groups: a control group, (ground rat chow for 2 weeks), a cuprizone group (0.6% cuprizone coated ground rat chow) and an oxycodone group (30 days of 15 mg/kg oxycodone in condensed milk). Rats were then euthanised, their brains were fixed in 4% paraformaldehyde then stained for MBP and counterstained with DAPI. The immunofluorescent intensity was measured in the corpus callosum.

### Results:

Compared to the control group, there was a 67% and 32% reduction in MBP in the oxycodone and cuprizone groups, respectively. These pilot results indicate we have established a demyelination model.

### Conclusions:

These findings reveal that both oxycodone and cuprizone are robust demyelination models. Both models will be used to test ibogaine and the remyelination mechanism will be compared and contrasted in both models.

# ORAL PRESENTATIONS

**Sancia Govender**

**University of KwaZulu-Natal**

## Background/Aim

The cerebellum, traditionally linked to motor coordination, is increasingly recognized as a key regulator of cognition, affect, and behaviour. Immune-mediated disruption of cerebellar circuits may contribute to the mood disturbances, cognitive dysfunction, and coordination deficits reported in subsets of patients with Autoimmune thyroid diseases. Emerging evidence of extra-thyroidal thyroid-stimulating hormone receptor (TSH-R) expression in the central nervous system suggests a potential role in thyroid-brain communication. Mapping its cerebellar distribution may therefore reveal structural substrates linking thyroid autoimmunity to cognitive and affective dysfunction.

## Method

Normal adult human cerebellar tissue collected postmortem ( $n = 5$ ) was examined using immunohistochemical staining to assess TSH-R protein expression across the vermis, floccular nodular, and dentate nucleus. Quantitative image analysis was performed to evaluate regional staining intensity and cellular localization, followed by statistical comparison of receptor expression across cerebellar subregions.

## Results

Distinct region-specific patterns of TSH-R localization were identified, demonstrating heterogeneous receptor expression across cerebellar subregions and within specific neuronal populations. Neuronal TSH-R expression was greatest in the vermis and moderate in the dentate nucleus and floccular nodular regions, with significant regional differences observed ( $p < 0.001$ ). Additional immunoreactivity was observed in glial cells, suggesting broader cellular distribution within cerebellar tissue.

## Conclusion

These findings provide the first anatomical mapping of TSH-R expression in the adult human cerebellum, offering novel insight into thyroid-brain interactions and a structural framework for investigating neuropsychiatric manifestations associated with thyroid autoimmunity.



## ORAL PRESENTATIONS

**Blessing Gumbu**

**University of Cape Town**

### Background

Cryptococcal meningitis (CM) is a life-threatening fungal brain infection, accounting for 15-20% of AIDS-related deaths in Sub-Saharan Africa. CM causes severe inflammatory neurological damage, yet the underlying neuroimmune mechanisms remain unknown. Previous studies have focused on microglia, but recent evidence indicates they are immunologically quiescent fungal reservoirs during infection. Astrocytes are immunocompetent cells that contribute to neuroimmune responses, yet their role in cryptococcosis pathogenesis remains unexplored. This study investigated astrocytic responses to cryptococcal infection.

### Methods

Hippocampal organotypic slices (HOCs) were treated with a GFP/mCherry-expressing *Cryptococcus neoformans* reporter strain and compared with untreated or lipopolysaccharide (LPS) controls. Immunofluorescence staining was used to track nuclear factor interleukin-6 (NF-IL6), a biomarker of neuroinflammation, alongside S100B, an astrocyte-specific marker. Transcriptional changes were assessed using single-nucleus RNA sequencing (snRNA-seq), validated by immunofluorescence-coupled in situ hybridization chain reaction (HCR-immuno).

### Results

We found minimal NF-IL6 immunoreactivity in astrocytes in *C. neoformans*-treated slices, indicating no inflammatory capacity, while LPS-treated slices confirmed astrocyte inflammatory capacity. However, snRNA-seq revealed a functional shift, with upregulation of tumor necrosis factor alpha (TNF)/nuclear kappa-light-chain-enhancer of activated B cells (NF- $\kappa$ B) and IL-6/JAK/STAT3 genes, and downregulation of glutamate receptor signaling and mitochondrial oxidative phosphorylation genes.

### Conclusion

*C. neoformans* did not induce classical NF-IL6-mediated inflammatory activation in astrocytes; however, snRNA-seq revealed upregulation of both inflammatory and immunometabolic genes. These results suggest that astrocytes are central participants in cryptococcal neuroimmunity.



## ORAL PRESENTATIONS

**Zolani Jacobs**

**University of the Western Cape**

Student Competition

### Background:

The blood-brain barrier (BBB) is a highly selective semipermeable border that controls molecules and ions that pass through the brain and blood. This control protects the brain from toxins and pathogens and enables neurons to function properly without interruption. Substances and drugs such as nicotine and antiretroviral agents (FTC, TDF & TAF) can disrupt this barrier by changing BBB permeability and homeostasis. Therefore, this study aimed to investigate the combined effects of FTC, TDF, TAF, and nicotine on endothelial cells of the blood-brain barrier.

### Methods:

The bEnd.5 cell line was used to evaluate the separate and combination effects of FTC (0.3  $\mu$ M), TDF (79 ng/ml), TAF (5 ng/ml), and nicotine (100 ng/ml and 500ng/ml) on the endothelial cells of the blood-brain barrier. The Trypan blue assay was used to assess cell proliferation, cell death, and cell suppression at different periods (24 to 96 hours). Additionally, the XTT assay was also used to evaluate mitochondrial function after exposure.

### Results:

Increasing concentrations of nicotine over time suppressed cell numbers, and this effect was exacerbated when combined with antiretroviral drugs (FTC, TDF & TAF), with a similar trend observed in mitochondrial activity.

### Conclusion:

The human blood plasma concentration significantly suppressed cellular division in BBB endothelial cells for both nicotine and antiretroviral drugs, with a synergistic effect when combined. This may lead to a change in brain endothelial cell function for people who are on pre-exposure prophylaxis and at the same time smoking.



# ORAL PRESENTATIONS

**Jessica Kanyemba**

**University of Cape Town**

Student Competition

## Background/Aim

Neurocysticercosis (NCC), a parasitic infection of the brain caused by the pork tapeworm *Taenia solium* (*T. solium*), is the leading cause of adult-acquired epilepsy. The parasite can reside in the brain for extended periods without causing any symptoms. However, little is known about the host inflammatory gene expression and molecular dynamics during this asymptomatic phase. This study investigated the expression profiles of the inflammatory genes *Timp1*, *Lcn2* and *Ccl5* in astrocytes and microglia following exposure to different treatment groups. We further aimed to identify the active molecules within the homogenate responsible for this immune suppression.

## Methods

Mouse hippocampal organotypic brain slice cultures (OBSCs) were treated with either lipopolysaccharide (LPS), *Taenia crassiceps* (*T. crassiceps*) homogenate, a cestode closely related to *T. solium*, or a combination of both. Gene expression was assessed using in situ hybridization chain reaction and confocal imaging. Additional cultures were treated with *T. crassiceps* homogenate in which protein and glycan components were disrupted, and media was collected for Enzyme-Linked Immunosorbent Assays (ELISAs) to identify immunosuppressive components.

## Results

LPS significantly unregulated *Timp1*, *Lcn2* and *Ccl5* with astrocytes predominantly expressing *Timp1* and *Lcn2*, and microglia expressing *Ccl5*. The combination of LPS and *T. crassiceps* homogenate significantly suppressed LPS-induced expression. However, the suppressive effect of *T. crassiceps* was lost when its glycan and protein components were inactivated.

## Conclusion

*T. crassiceps* homogenate suppressed LPS induced expression of inflammatory genes, likely by outcompeting for binding on immune cells. The active molecule responsible for immunosuppression is likely a proteoglycan.



## ORAL PRESENTATIONS

**Tusekile Sarah Kangwa**

**University of Cape Town**

Student Competition

### Background:

Maternal HIV and altered cytokine profiles during pregnancy may affect foetal immune and neurodevelopment. We evaluated (i) cytokine levels in 2nd trimester pregnant women with and without HIV, and longitudinal cytokine patterns in their HIV-exposed uninfected (HEU) and HIV-unexposed uninfected (HUU) children, (ii) the effect of maternal antiretroviral therapy (ART) timing on cytokine levels, and (iii) longitudinal associations between child cytokine levels and expressive language at ages 3.5 and 5.

### Methods:

This sub-study included mother-child pairs from the Drakenstein Child Health Study, assessed during pregnancy and at 7 weeks, 2-, 3-, and 5 years. Eighteen immune markers were measured. Group differences were analysed with regression and mixed-effects models, and principal component analysis identified cytokine patterns. Expressive language outcomes were assessed with the Kaufman Assessment Battery for Children at 3.5 and 5 years.

### Results:

Pregnant women with HIV presented with a disrupted Th1/Th2 balance. HEU children had lower GM-CSF, IL-10, IL-12p70, IL-1 $\beta$ , IL-2, and IL-4 levels and higher sCD14 levels over time than HUU children, revealing distinct immunoregulatory profiles. ART initiation during compared to before pregnancy, was associated with higher maternal immune marker levels. Time-specific associations, particularly in HUU children (notably IL-12p70, IL-2, IL-4, IL-13), were negatively associated with language at 3.5 years. Longitudinally, IL-12p70 and IL-2 were positively associated with language at 3.5 years in HUU, with no significant associations at 5 years or in HEU children.

### Conclusion:

Altered maternal immune responses persist longitudinally in HEU children; however, there was minimal evidence of immune markers impacting language.

## ORAL PRESENTATIONS

**Livhuwani Mabasha**

**North-West University**

Student Competition

### Background:

Postpartum depression (PPD) is a significant health concern, with limited approved treatment options available. The selective serotonin reuptake inhibitor (SSRI), fluoxetine, is, however, commonly prescribed. Importantly, fluoxetine is excreted in breast milk; concerns about its effects on early neuroimmune development exist. Given the role of serotonin in immune regulation, this study aimed to investigate the effects of passively administered fluoxetine exposure on brain interleukin-1 $\beta$  (IL-1 $\beta$ ) and matrix metalloproteinase-9 (MMP-9) levels in pre-pubertal Flinders line rat offspring.

### Methods:

Flinders sensitive (FSL) and resistant line (FRL) dams either received 10 mg/kg/day fluoxetine or a placebo, from postnatal day 4 (PND04) to 18, to passively expose their offspring. Pups were euthanized on PND22, whereafter whole-brain tissue was collected, homogenized, and analysed using ELISA kits. Data were analysed using ordinary two-way ANOVAs to assess the effects of strain and treatment. The study received ethical approval (NWU-00031-25-A5).

### Results:

No statistically significant drug\*strain interactions for either IL-1 $\beta$  ( $p = 0.869$ ) or MMP-9 ( $p = 0.225$ ), nor any significant main effects were identified. Despite the established depressive-like bio-behavioural profile of the FSL rat, these findings indicate comparable baseline strain values of inflammatory markers at this early developmental stage. Irrespective of strain, passively administered fluoxetine did not influence these neuroinflammatory markers in offspring.

### Conclusion:

Comparable strain whole-brain inflammatory marker profiles suggest that the behavioural differences of this model may not be driven by an inflammatory construct. Importantly, the lack of treatment effects supports the absence of detectable changes in these neuroinflammatory markers under the conditions tested.



## ORAL PRESENTATIONS

**Ngoni Magate**

**Stellenbosch University**

Psychiatric disorders exhibit complex, heterogeneous biology involving immune, inflammatory, and transcriptional dysregulation. While RNA-seq and cytokine data hold promise for biomarker discovery, small, noisy datasets challenge conventional machine learning (ML), often yielding unreliable case-control discrimination. Transformer-based foundation models like the Tabular Prior-data Fitted Network (TabPFN), optimized for low-sample, high-dimensional tabular data, offer a potential solution, yet remain untested in psychiatric risk classification. This study benchmarks TabPFN against Elastic Net, LightGBM, RF, SVM and other traditional ML models to classify PTSD cases versus trauma-exposed controls using secondary RNA-seq and cytokine data from the Shared Roots study. Predictive accuracy, feature stability, and biological interpretability will be evaluated through nested cross-validation, addressing a critical gap in small-cohort omics analysis.



## ORAL PRESENTATIONS

**Hendrina Mosima Marole**

**Sefako Makgatho Health Sciences University**

Student Competition

### Background:

Mild Traumatic Brain Injury (mTBI) among adults is a significant public health concern, with over 90% of TBI patients in Western hospitals classified as having mTBI. In South Africa, the burden of mTBI is heightened by interpersonal violence, motor vehicle accidents, inadequate reporting and limited resources. Adults with mTBI experience cognitive deficits, emotional dysregulation, behavioural changes and neuropsychological symptoms impacting daily functioning and quality of life. Adult mTBI survivors experience significant neuropsychological rehabilitation barriers, exacerbated by a lack of clinical practice guidelines (CPGs) for resource-constrained areas in South Africa. This study aims to contextualise CPGs for neuropsychological rehabilitation of adults with mTBI developed in high-income countries (HICs), for resource-constrained areas in South Africa.

### Methods:

An exploratory sequential mixed methods design will be implemented across three phases. Phase 1 employs a PRISMA-ScR guided scoping review across MEDLINE, CINAHL, PubMed, Web of Science, and Scopus, including grey literature. Phase 2 entails semi-structured interviews with adult mTBI patients and focus groups with multidisciplinary team members and family members exploring care pathways. Phase 3 employs a Delphi technique with expert neuropsychologists to validate evidence-based statements and recommendations.

### Results:

Findings will map and appraise existing CPGs, identify gaps in neuropsychological rehabilitation care pathways for adults with mTBI in resource-constrained settings, and present validated evidence-based recommendations contextualised for the South African healthcare system.

### Conclusion:

This study is expected to generate evidence-based contextually relevant statements, addressing the unique socioeconomic, cultural and resource-constrained context in South Africa, to improve functional outcomes and quality of life for adults with mTBI.



## ORAL PRESENTATIONS

**Amanda Miya**

**University of KwaZulu-Natal**

### Background:

Ghrelin is a metabolic hormone that regulates appetite and plays a role in central nervous system signalling. Dysregulation of ghrelin signalling has been implicated in the pathophysiology of neuropsychiatric disorders. Despite this, brain-derived ghrelin signalling, particularly via the ghrelin receptor (GHSR1a), remains poorly characterised. To address this gap, the current study aimed to quantify baseline expression and distribution of GHSR1a and ghrelin in human brain regions involved in metabolic regulation and mood-related processes.

### Methods:

Formalin-fixed, paraffin-embedded postmortem human brain tissues (n=5) from the hypothalamus, pituitary and medulla were analysed. Immunohistochemistry was performed using antibodies against GHSR1a and ghrelin. Qualitative assessment of localisation and distribution was complemented by quantitative image analysis, with regional comparisons conducted using a linear mixed model.

### Results:

The GHSR1a expression showed regional variation with consistent region-specific patterns observed across groups. The medulla exhibited intense GHSR1a immunostaining in the NTS with evident ghrelin co-localisation. In the hypothalamus, neurons in the arcuate nucleus showed positive GHSR1a staining with minimal ghrelin presence, while the anterior pituitary displayed strong receptor expression with sparse ghrelin staining. Quantitative analysis confirmed higher GHSR1a expression in the medulla ( $p < 0.001$ ) and lower expression in the pituitary relative to the hypothalamus ( $p < 0.001$ ).

### Conclusion:

These findings demonstrate region-specific GHSR1a expression and ghrelin signalling in the human brain. This pilot data highlights distinct regional patterns that may influence feeding regulation, endocrine function and neurological disease susceptibility. The data provides a foundation for future studies investigating dysregulation of ghrelin signalling in metabolic and neuropsychiatric co-pathologies.



## ORAL PRESENTATIONS

**Seipati Mokhosi**

**University of KwaZulu Natal**

Brain metastasis (BM) remains a major clinical challenge in lung and aggressive breast cancers (HER2-positive and triple-negative), largely due to the blood-brain barrier (BBB) and limited targeted delivery systems. This study developed biocompatible cobalt-derived ferrite nanoparticles (CoFeNPs) as gene delivery nanocarriers, incorporating Angiopep (ANG) to enable BBB targeting via LRP-1 receptors. Cobalt ferrite ( $\text{CoFe}_2\text{O}_4$ ) and substituted  $\text{Mg}_{0.5}\text{Co}_{0.5}\text{Fe}_2\text{O}_4$  were synthesised using a glycol-thermal method and functionalised with chitosan (CS) and polyethylene glycol (PEG). ANG was conjugated using SPDP and Mal-PEG-NHS crosslinkers, yielding ANG-CSFeNPs and ANG-PEG-CSFeNPs to enhance stability, biocompatibility, and DNA binding. Characterisation confirmed spinel ferrite structures (7.25–12.98 nm core size) with high magnetisation (up to 73.9 emu/g). CS coating shifted surface charge to +20.7 mV, promoting nucleic acid interaction, while hydrodynamic sizes remained <110 nm. ANG and PEG conjugation increased size (up to 284 nm) and improved stability while maintaining favourable  $\zeta$ -potentials. Biological evaluation showed high biocompatibility (>80% viability) in HEK293 and SVG-p12 cells. Cancer cell lines (MDA-MB-231, A549, Caco-2) exhibited improved tolerance with Mg substitution, polymer coating, and ANG conjugation. Minimal oxidative stress and apoptosis were observed. Gene delivery efficiency, assessed via luciferase assays, depended strongly on surface chemistry: PEG enhanced transfection of ANG-CSCoFeNPs in MDA-MB-231, but reduced efficiency in Mg-substituted derivatives. Internalisation and docking studies confirmed superior uptake and LRP-1 binding of ANG-PEG-CSCoFeNPs. These nanoparticles show promise for safe, BBB-targeted gene delivery, with potential applications in imaging, drug delivery, and hyperthermia, pending further optimisation.

## ORAL PRESENTATIONS

**Kamohelo Mokhudu**

**University of KwaZulu-Natal**

### Background:

Thyroglobulin (TG) is essential protein for thyroid hormone synthesis and is primarily localized in the thyroid gland; however, extra-thyroidal expression has been reported in mammalian epidermis, kidney, thymus, and hair follicles. Recently, our group demonstrated TG expression in the normal adult human brain, particularly within the smooth muscle cells of the tunica media/adventitia in cerebro-vasculature, and in neurons. This study extends these findings to the adult human cerebellum. Given the cerebellum's role in emotional and cognitive processing and its connectivity with limbic regions, it is plausible that autoimmune anti-thyroid antibodies may bind and antagonise limbic- and cerebellar-derived thyroid-related factors thereby contributing to dysregulation of emotional processing. Such association, potentially contributing to mood dysregulation in autoimmune thyroid disease (AITD). This study therefore examines TG expression in the normal adult human cerebellum as a potential autoimmune target in AITD.

### Methods:

Immunohistochemistry was used to detect TG expression postmortem in cerebellar regions, including the cerebrocerebellar hemisphere, vermis, flocculonodular lobe, and dentate nucleus. Quantitative image analysis and inter-regional comparisons were performed using a linear mixed-effects model.

### Results:

TG expression was identified in vascular smooth muscle of the tunica media/adventitia across all regions. Significant regional variation was observed ( $p < 0.001$ ), with higher expression in the dentate nucleus and flocculonodular lobe. Furthermore, TG neuronal staining was observed within the dentate nucleus.

### Conclusion:

Cerebellar TG expression suggests a potential autoantigen in AITD, with implications for cerebellar dysfunction and neuropsychiatric disturbances. Immune-mediated interactions may disrupt neuronal function, contributing to mood dysregulation, observed in bipolar disorders, and highlight potential therapeutic targets.



## ORAL PRESENTATIONS

**Tabo Mwila**

**University of the Witwatersrand**

Adolescent alcohol use is a significant public health concern in South Africa, posing long-term risks due to ongoing brain development during this critical period. The hippocampus, which plays a central role in learning and memory, is particularly vulnerable to alcohol-induced oxidative stress. Obestatin, a gut-derived peptide, has demonstrated neuroprotective antioxidant properties; however, its role in mitigating alcohol-related hippocampal damage remains poorly understood.

Adolescent male and female C57BL/6J mice underwent 28-day voluntary two-bottle choice alcohol consumption (10% ethanol) or saccharin-sweetened water (control). Mice received daily subcutaneous injections of saline or obestatin (0.1 or 0.5  $\mu\text{g/mL}$ ). Hippocampal TSOD activity was measured, and gene expression of SOX2, Nestin, DCX, BDNF, CREB1, GFAP, and AIFM1 was analysed using qRT-PCR.

This model achieved blood alcohol concentrations (BACs) from 10.34 to 100.03 mg/dL, confirming systemic alcohol exposure with substantial variability. Hippocampal TSOD activity was significantly reduced in males receiving alcohol and 0.1  $\mu\text{g/mL}$  obestatin compared with controls ( $p = 0.0267$ ), whereas no differences were observed in females ( $p = 0.712$ ). GSH-Px activity was not significantly altered in either males ( $p = 0.86$ ) or females ( $p = 0.14$ ). qRT-PCR analyses revealed no significant changes in the expression of SOX2, Nestin, DCX, BDNF, CREB1, GFAP, or AIFM1 across treatment groups.

Collectively, these findings suggest that moderate voluntary alcohol exposure during adolescence did not produce detectable hippocampal molecular stress in this model. While obestatin showed no consistent modulatory effects, the male-specific reduction in TSOD activity at the lower dose suggests subtle, sex-dependent interactions that may occur beyond transcriptional regulation.

## ORAL PRESENTATIONS

**Aminu Musa**

**Nelson Mandela University**

Student Competition

### Background

Cannabis, like other psychotropic drugs, triggers heightened incentive salience within the mesocorticolimbic system and overlapping brain regions involved in memory and cognition. The legalization of cannabis over the past decade has made it more accessible and widely used. This places individuals with a genetic susceptibility at risk of developing cannabis use disorder (CUD). The mechanisms that drive neuroplastic changes in CUD are not well understood.

### Methods

Twenty-four male C57BL/6 mice were randomly divided into four groups (n=6 per group) and received a Cremophor/saline solution (control), delta-9-tetrahydrocannabinol (THC) (5 mg/kg), cannabidiol (CBD) (20 mg/kg), or both within the conditioned place preference (CPP) apparatus to evaluate cue-induced drug-seeking behaviour. Anxiety-like behaviour was assessed using the open field test (OFT) and elevated plus maze (EPM), while cognitive performance was evaluated using the novel object recognition test. Thereafter, drug-induced transcriptional changes and miRNA profiling were assessed in the prefrontal cortex and hippocampus using RT-qPCR.

### Results

7 days of THC exposure significantly increased the time spent in the least preferred compartment compared to control ( $p < 0.05$ ), whereas CBD produced an opposite effect. THC also upregulated miRNA Let7D in both PFC and hippocampus ( $p < 0.05$ ), while CBD and THC co-exposure significantly increased PFC  $\Delta$ FosB expression ( $p < 0.05$ ). Phytocannabinoid exposure did not alter exploratory or anxiety-like behavior ( $p > 0.05$ ), but THC-treated mice showed impaired discrimination memory.

### Conclusion

In conclusion, we demonstrated that exposure to THC induces place preference, while CBD induced place aversion, and coadministration of CBD and THC produced an overall THC-like effect rather than a CBD-like effect.

## ORAL PRESENTATIONS

**Zethembiso B. Ngcobo**

**University of Cape Town**

### Background:

HIV-exposed uninfected (HEU) children are at increased risk of neurodevelopmental delays compared to HIV-unexposed uninfected (HUU) peers. The underlying biological mechanisms remain unclear; however, emerging evidence suggests a role for immune dysregulation. This study aimed to (i) compare immune marker levels between HEU and HUU children, (ii) assess whether immune markers predicted neurodevelopmental outcomes at 2 years, and (iii) explore potential mediation pathways.

### Methods:

This sub-study included 280 mother-child dyads from the Khula birth cohort in Gugulethu, Cape Town. Sixteen immune markers were measured in child peripheral blood at 2-5, 6-11 and 12-19 months. Neurodevelopment at 2 years was assessed using the Bayley Scales of Infant and Toddler Development, Fourth Edition. Linear regression and mediation analyses were performed and adjusted for covariates.

### Results:

The study included 98 HEU children (50.0% male) and 182 HUU children (50.5% male). At 12-19 months, HEU children had significantly lower interferon-gamma (IFN- $\gamma$ ), interleukin-10 (IL-10), IL-12p70, IL-13, IL-1 $\beta$ , and IL-7 concentrations compared to HUU children. In HEU children, higher levels of IFN- $\gamma$ , granulocyte-macrophage colony-stimulating factor (GM-CSF), tumor necrosis factor alpha (TNF- $\alpha$ ), IL-10, IL-12p70, IL-5, and IL-7 at 12-19 months were associated with poorer language performance at 2 years (all p-values <0.04). Among these, IL-7 significantly mediated the association between HIV exposure and language outcomes.

### Conclusion:

Prenatal HIV exposure is associated with immune dysregulation in early life, particularly at 12-19 months, and with poorer language outcomes at 2 years in HEU children. IL-7 may contribute to biological pathways linking HIV exposure to early language development.



## ORAL PRESENTATIONS

**Tosin Abiola Olasehinde**

**University of KwaZulu-Natal**

Type 2 diabetes is associated with insulin resistance and pancreatic  $\beta$ -cell dysfunction, and its progression is linked to glucotoxicity, lipotoxicity, altered neurochemistry, and neurodegeneration. This study investigated the effects of sulphated polysaccharides (SPs) isolated from the South African red alga *Gelidium pristoides* on glucose and lipid metabolism, brain ultrastructure, oxidative stress markers, and pro-inflammatory cytokines in diabetic rats. Sprague-Dawley rats (180–200 g) were rendered diabetic via oral fructose (10%) administration and intraperitoneal streptozotocin injection (40 mg/kg body weight). Rats with blood glucose levels exceeding 200 mg/dL were randomized into groups and treated daily with metformin (200 mg/kg) or SPs (150 and 300 mg/kg) for 28 days. SP treatment significantly improved glucose metabolism, evidenced by reduced activities of glycogen phosphorylase, fructose-1,6-bisphosphatase, ATPase, and modulation of GLUT-4 levels. Lipid metabolism was also improved, with normalization of saturated and unsaturated fatty acids and fatty acid esters. Transmission electron microscopy revealed that diabetic rats exhibited shrinkage, reduced synaptic vesicles, and mitochondrial swelling; these alterations were markedly attenuated by SP treatment. Furthermore, SPs significantly decreased brain levels of pro-inflammatory cytokines (IL-6, IL-1 $\beta$ , and TNF- $\alpha$ ) compared to diabetic controls. Oxidative stress was mitigated, as indicated by reduced malondialdehyde levels and modulation of antioxidant enzymes, including catalase, superoxide dismutase, and glutathione peroxidase. These findings highlight the therapeutic potential of sulphated polysaccharides from *Gelidium pristoides* in ameliorating hyperglycemia-induced neurochemical alterations through improved metabolic regulation, protection of brain ultrastructure, and attenuation of neuroinflammation.

## ORAL PRESENTATIONS

**Simran Atulkumar Patel**

**University of Cape Town**

### Background

Cryptococcal meningitis (CM) is an invasive fungal infection of the brain associated with severe neurological complications including hydrocephalus and elevated intracranial pressure (ICP). Dilated perivascular spaces have long been recognised as a radiological hallmark of CM, but the mechanisms underlying this pathology remain poorly understood. Here, we hypothesise that neuropathology in CM may arise from glymphatic disruption and investigate glymphatic function in murine models of CM.

### Methods

Male C57BL/6 mice (8-12 weeks old) were intravenously injected with either  $5 \times 10^4$  colony forming units (CFU) of *Cryptococcus neoformans* or saline. To resolve the spatial organisation of fungal cells within the perivascular compartment, optically cleared whole-brains were imaged using light-sheet fluorescence microscopy, followed by three-dimensional segmentation to quantify fungal aggregation. Glymphatic function was assessed by intracisternal infusion of a fluorescent ovalbumin tracer, with brain-wide tracer penetration and lymphatic drainage quantified.

### Results

We present the first three-dimensional map of cryptococcal distribution in the mouse brain. Our results show a multi-compartmental dissemination of cryptococcal cells across the vascular, perivascular, and parenchymal compartments, with preferential aggregation within the perivascular compartment. Fungal dissemination was accompanied with impaired glymphatic function, evidenced by reduced tracer penetration and lymphatic drainage in infected animals compared to healthy controls.

### Conclusion

Our findings provide the first experimental evidence of glymphatic dysfunction during CM, potentially arising from occlusion of the perivascular compartment by aggregated cryptococcal cells. This mechanism offers a plausible biological framework linking fungal neuroinvasion to the hydrocephalus and elevated ICP that characterises clinical disease.



## ORAL PRESENTATIONS

**Yanga Bridgette Pato**

**University of Cape Town**

Student Competition

### Background/Aim

Cryptococcal meningitis (CM) is a fungal infection of the brain that is associated with severe neurological injury. The pathological mechanisms of CM include the formation of cryptococcomas, these are densely massed fungal lesions within a polysaccharide-rich capsule that heavily restricts the diffusion of metabolites and immune mediators. CM patients also present with elevated lactate and decreased brain glucose; both associated with poor survival. Despite evidence linking metabolism to pathology, mechanisms underlying metabolic changes in CM remain unknown. We therefore investigated metabolic homeostasis disruption as a driver of bioenergetic failure and neurological damage in CM.

### Methods

Bulk metabolomics was performed on male C57BL/6 mice (n=8, 8-12 weeks) intravenously injected with  $5 \times 10^4$  CFU *C. neoformans* to assess metabolic reprogramming. Single-nucleus RNA sequencing was performed on organotypic hippocampal brain slices infected with *C. neoformans* to characterize metabolic transcriptional changes in neurons and glia. Lactate and glucose assays quantified waste accumulation and glucose utilization in infected mice as measures of metabolic dysfunction.

### Results and conclusion

Results showed significant reprogramming of nitrogen-associated amino acids and neurotransmitters, with downregulation of glutamate (12-fold) and GABA (12.4-fold), indicating disrupted metabolic homeostasis. Astrocytic populations showed the highest response, followed by excitatory neurons, with coordinated upregulation of glycolytic and hypoxia-responsive genes combined with suppressed cellular respiration, consistent with functional oxygen limitation and restricted glucose availability. Lactate and glucose assays indicated disrupted glucose and elevated lactate, often linked to infection-associated metabolic dysfunction. These findings identify metabolic dysregulation as a central driver of cryptococcal brain injury.

## ORAL PRESENTATIONS

**Naadiya Seedat**

**University of Cape Town**

### Background:

Nyaope, a heroin-cannabis mixture endemic to disadvantaged South African communities, is associated with significant cortical atrophy in frontotemporal regions. Whether this atrophy is predisposed or acquired remains unclear. Intracranial volume (ICV) serves as a premorbid brain size marker, yet cortical scaling relative to ICV is unexamined in substance use disorders. White matter correlates in this cohort also remain unexplored.

### Methods:

Male nyaope users ( $n = 28$ ) and matched controls ( $n = 30$  for structural;  $n = 26$  for diffusion analyses) underwent 3T MRI. ICV, cortical thickness and volume were extracted using FreeSurfer v7.4.1. Allometric scaling was assessed via log-transformed regression. Fractional anisotropy (FA) maps from diffusion-weighted imaging were analysed using tract-based spatial statistics in FSL.

### Results:

Cortical volume scaled positively with ICV in both groups ( $p < 0.001$ ); cortical thickness did not. Age as well as duration of nyaope use reduced cortical volume in users only ( $p < 0.05$ ). Users showed reduced FA in the left anterior thalamic radiation, inferior and superior longitudinal fasciculi, inferior fronto-occipital fasciculus, and cingulum ( $p < 0.05$ ) however significance was lost when testing for multiple comparisons. Overlap analyses identified convergent vulnerability in the left anterior thalamic radiation.

### Conclusions:

Cortical volume but not thickness scales to ICV, validating ICV as a premorbid marker of brain size. Nyaope users display greater age-associated cortical volume reductions, suggesting acquired rather than pre-existing atrophy. Contrasting cortical changes with white matter microstructure suggests that white matter pathology may be secondary to cortical atrophy which supports previous studies in SUDs.



## ORAL PRESENTATIONS

**Tiaan Antonie Steyn**

**North West University**

### Background:

HIV-associated neurocognitive disorders (HAND) persist despite effective antiretroviral therapy and are driven in part by chronic neuroinflammation and direct viral neurotoxicity. The HIV-1 trans-activator of transcription (Tat) protein contributes to neuronal injury by disrupting cellular homeostasis and promoting neurotoxic signalling. Sequence variation in the Tat protein of genetically distinct HIV-1 subtypes and circulating recombinant forms (CRFs) may influence its neurotoxic potential. While subtype B Tat has been extensively studied, less is known about subtype C and CRF01\_AE. Given the global predominance of subtype C and the increasing prevalence of CRFs, investigating subtype-specific differences in Tat neurotoxicity is important for improving our understanding of HIV neuropathogenesis.

### Methods:

Differentiated human neuroblastoma (SH-SY5Y) cells were used as an in vitro neuronal model. Cells were differentiated with 10  $\mu\text{M}$  all-trans retinoic acid for five days and subsequently exposed to recombinant Tat subtype C or Tat CRF01\_AE at concentrations of 100, 500, and 1000 ng/mL for 24 hours. Neuronal damage was assessed through qualitative morphological analysis and cell viability using the MTT assay.

### Results:

Compared to negative control, Tat CRF01\_AE induced a significant and concentration-dependent reduction in cell viability ( $p < 0.05$ ), while Tat C showed minimal effects, reaching significance only at 1000 ng/mL ( $p < 0.05$ ).

### Conclusion:

These findings demonstrate subtype-specific differences in HIV-1 Tat neurotoxicity, with CRF01\_AE exhibiting greater neuronal damage than Tat subtype C in a differentiated human neuroblastoma model. Further studies are needed to investigate the molecular basis of these differences and their implications for HAND pathogenesis.



## ORAL PRESENTATIONS

**Amy van der Hoven**

**University of Cape Town**

### Background/aim

Central nervous system (CNS) development begins with neural tube formation and subsequent neurogenesis, disruptions of which result in neural tube defects and neurodevelopmental disorders, respectively. Although the morphogens retinoic acid (RA) and BMP4 are understood to play a significant role in early neural development, their precise roles remain unclear. Neural tube organoids (NTOs), 3D aggregates of pluripotent stem cells, provide a platform to model these processes in vitro. This project aims to generate an in vitro organoid model of early neural development by investigating the morphogenic roles of RA and BMP4.

### Methods

Day 6 NTOs were produced by aggregation of 500 PSCs and treated with pulses of 250nM RA from days 2-3. For more mature NTOs, organoids were cultured for 10 days and treated with a 1ng.mL- pulse of BMP4 from days 5-6 and continuous treatment of 250nM RA from days 5-10. Organoids were assessed for temporal and spatial neural marker expression using ICC and RT-qPCR and were analysed morphological changes.

### Results

RT-qPCR results from day 6 organoids demonstrated significant upregulation of midbrain neural markers compared to controls. ICC results further revealed the formation of neural rosettes and complex tube-like structures. Results from day 10 organoids similarly showed upregulation of neural markers but ICC confirmed the presence of post-mitotic neurons.

### Conclusions

Our findings show the successful generation of in vitro models capable of recapitulating aspects of early neural tube morphology and neurogenesis, a valuable tool to investigate early neural development and defects.



## ORAL PRESENTATIONS

**Victoria Williams**

**University of the Witwatersrand**

### Background/ Aim

Clinical empathy is a key component of patient-centered care. In social neuroscience, it is understood as part of broader socio-affective processes, including emotion recognition, affective responsiveness, perspective-taking, interpersonal sensitivity, and emotional regulation. Among medical students, these capacities are not only shaped by training, but also by personality traits and earlier life experiences, however, these relationships remain largely underexplored. This study examined the relationships between empathy, dark personality traits, and adverse childhood experiences (ACEs) in South African medical students.

### Methods

An online survey was completed by 234 medical students (years: 1–6), University of the Witwatersrand, Johannesburg, South Africa. This included a demographic questionnaire and validated self-report measures of empathy (Toronto Empathy Questionnaire, TEQ), dark personality traits (70-item D-score questionnaire, D70), and Adverse Childhood Experiences (ACE) questionnaire.

### Results

A total of 234 students responded (mean age  $21.65 \pm 2.46$  years; 69.7% female). Internal consistency was excellent for the D70 ( $\alpha = 0.946$ ) and TEQ ( $\alpha = 0.903$ ). Females scored higher on empathy than males ( $50.78 \pm 8.60$  vs  $46.65 \pm 11.06$ ;  $p = 0.038$ ), whereas males scored higher on dark traits ( $2.37 \pm 0.51$  vs  $2.09 \pm 0.47$ ;  $p < 0.001$ ). Empathy was inversely associated with dark score ( $\rho = -0.660$ ,  $p < 0.001$ ); strongest for callousness ( $\rho = -0.716$ ,  $p < 0.001$ ). Mean ACE- score was  $2.84 \pm 2.14$ ; showing no significant association with empathy or dark traits.

### Conclusion

Empathy was more strongly related to dark personality traits, particularly callousness, than to ACEs. From a social neuroscience perspective, this suggests that higher callousness may be associated with reduced responsiveness to patients' distress; highlighting the importance of socio-affective training to strengthen clinical empathy in future doctors.



POSTER  
PRESENTATIONS



# POSTER PRESENTATIONS

**Levanco Asia****North-West University****SANS26-P47**

## Background

Antiretroviral therapy (ART) effectively suppresses viral load but does not resolve HIV-associated neurocognitive disorders (HAND), with milder forms persisting in people living with HIV (PLHIV). HIV subtypes exhibit significant amino acid variation in viral proteins, which may influence neuropathogenesis. The transactivator of transcription (Tat) protein enhances HIV replication, and specific amino acid substitutions, including T24K, C31S, and R57S, are associated with differential neurovirulence in HIV-1 subtype C. Although they are well characterized in cART-experienced subtype B cohorts, it remains poorly characterized in subtype C cohorts.

## Aim

This study aimed to determine the frequency of amino acid variations at Tat positions linked to differential neuropathogenesis (T24K, C31S, and R57S) in HIV-1 subtype C.

## Methods

Plasma from 278 cART-treated PLHIV were analysed. Viral RNA was extracted, reverse transcribed, and amplified by PCR targeting the Tat exon 1/Vpr region. Amplicons were Sanger sequenced. Jalview and ExPASy translate were used to identify key amino acids in Tat.

## Results

Due to low plasma viral load, the full Tat sequences were obtained for 46 participants (16.5%). At position 24, amino acids observed were N (41%), K (33%), R (9%), Q (7%), T (4%), S (4%), and P (2%). At position 31, S (83%) and C (17%) were observed. At position 57, S (76%), R (18%), K (4%), and C (2%) were observed.

## Conclusion:

The predominant amino acids at key Tat positions have been associated with reduced neurovirulence. These findings provide insight into subtype C Tat variability in a South African cohort and may contribute to understanding subtype-specific differences in HAND.



# POSTER PRESENTATIONS

**Banele Banda**

**Stellenbosch University**

**SANS26-P18**

## Background

Social adversities may exacerbate underlying biological vulnerabilities in individuals at risk for psychosis. People with schizophrenia show heightened reactivity to social and environmental stressors, partly due to increased childhood trauma (CT) exposure. There is a need for reliable, ecologically valid biomarkers of stress reactivity in schizophrenia.

## Methods

This virtual reality (VR)-based pilot study examined the clinical and psychosocial correlates of VR-elicited stress reactivity in patients with schizophrenia ( $n = 44$ ) compared to healthy controls (HC) ( $n = 43$ ) assessed using a validated fear-of-heights (FOH) paradigm. Validated scales were used to assess CT (MACE), self-esteem (SERF-SF), resilience (CD-RISC), and social adversity (PCSAQ). Psychopathology was assessed in patients using the PANSS. VR-related side-effects as well as self-reported stress and objective stress reactivity (CDA) measures were assessed at baseline, peak stress, and recovery periods.

## Results

Patients reported more severe childhood abuse, poorer resilience, and more pronounced social exclusion during adulthood than HC. Both groups showed similar increases in VR-related nausea and dizziness during the FOH task. Patients reported higher subjective stress overall, partly attributable to more severe childhood abuse. In contrast, patients demonstrated a lesser change in CDA compared to HC, partly due to more severe adulthood social exclusion. Psychopathology further moderated VR-elicited stress reactivity changes in patients.

## Conclusion

Schizophrenia is characterized by dissociated response patterns of subjective vs. physiological stress reactivity. CT was linked to heightened subjective stress, while adult adversity correlated with a blunted physiological response to FOH. These findings suggest stage-dependent effects of social adversity on stress reactivity.



# POSTER PRESENTATIONS

**Pumesha Nishka Bhunjoo**  
**Stellenbosch University**

**SANS26-P38**

## Background

Cannabis use is a risk factor that precipitate schizophrenia in genetically susceptible individuals. Four major genes modulate this risk, including BDNF, CNR1, COMT, and AKT1. Among them, COMT gene with its polymorphism directly implicated in influencing interaction between cannabis exposure and schizophrenia by regulating dopamine degradation in the prefrontal cortex.

## Aim

The study aims to evaluate evidence on genetic susceptibility to cannabis-induced schizophrenia focusing on COMT functional polymorphism involving Met to Val substitution at codon 158. This produces Val and Met alleles with differing enzyme activity to link between adolescent cannabis use and increased risk for schizophrenia in adulthood.

## Methodology

A systematic PubMed (MEDLINE)search using MESH headings and keywords identified clinical trials and cohort studies of cannabis-exposed individuals developing schizophrenia. Articles were independently screened by all the reviewers and risk of bias was assessed.

## Results

There were 10 cohort studies included; no matched clinical trials identified 803 patients which were followed from ages 3 to 26yrs, 13% of adolescent cannabis users with Val/Val genotype developed schizophrenia at age 26 versus 1.4% of non-users with same Val/Val genotype. Odds ratios for adolescent cannabis use and subsequent disorder were 10.9 for (Val/Val), 2.5 for (Val/Met), and 1.1 for (Met/Met). Participants aged >18 years genotype was not predictive of schizophrenia outcomes.

## Conclusion

This study shows that cannabis-related schizophrenia is dependent on genetic susceptibility. Val/Val carriers show highest risk following adolescent cannabis exposure, followed by Val/Met whereas Met/Met appears protective. COMT polymorphism-environment interaction in adolescence is essential for predicting adult schizophrenia emergence however, single-gene variation does not fully account for differential THC sensitivity or psychosis risk.

# POSTER PRESENTATIONS

**Daniel Johannes Botes**

**University of Cape Town**

**SANS26-P30**

## Background/Aim

Autism spectrum disorder (ASD) is characterized by atypical neurological development arising from complex genetic, epigenetic, and environmental interactions. Among environmental factors, short-chain fatty acids such as propionic acid (PPA), a microbial metabolic product, have been implicated in altered neurodevelopmental trajectories. This study aimed to establish a multipotent neural stem cell (NSC) model to investigate how PPA influences NSC fate in vitro.

## Methods

ReNcell CX cultures were differentiated for 7, 14, and 21 days, with parallel groups treated with or without PPA. Changes to NSC fate, morphology, and reactivity were assessed using immunocytochemistry to measure glial fibrillary acidic protein (GFAP) and Nestin. Thiazolyl Blue Tetrazolium Bromide (MTT) was done to validate cell viability for those treated for PPA ranging 3-500mM PPA over 72 hours. qPCR of the mitochondrial NADH dehydrogenase gene ND1 and nuclear reference gene  $\beta$ 2M was used to measure mtDNA copy number. Quantitative analysis of microscopy images was conducted with Fiji/Image J and statistical analyses were performed in GraphPad Prism.

## Results

Microscopy revealed distinct alterations in marker expression between treated and untreated groups. PPA exposure was associated with increased GFAP immunoreactivity, suggesting enhanced astrocytic differentiation. At the same time, PPA significantly increased cell volume and total Nestin expression, indicating glial hypertrophy and reactivity.

## Conclusion

These findings highlight that PPA significantly alters NSC developmental trajectories, shifting the balance between NSC renewal and glial differentiation. This supports the hypothesis that microbial metabolites can influence neurodevelopmental processes, potentially contributing to gliosis and altered neural circuitry observed in ASD.



# POSTER PRESENTATIONS

**Erin Buchanan**

**SANS26-P34**

**University of Cape Town**

## Background/Aim

Mitochondrial (MT) function is essential during neuronal development and function. Consequently, MT dysfunction has been implicated in neurological disorders. Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder with complex aetiology with mechanisms underpinning ASD remaining poorly understood. MT dysfunction has been implicated in ASD pathophysiology. Propionic acid (PPA) is a metabolic stress used to model ASD in vitro and has been associated with MT differential methylation found in a South African ASD cohort. This study aimed to investigate how MT dysfunction affects MT dynamics and structure in neurons using PPA to recapitulate ASD-associated MT dysfunction.

## Methods

Neuronal SH-SY5Y cells were differentiated over nine days into mature neurons in the presence or absence of PPA. Confocal microscopy was used to image mitochondrial content, morphology and networks. Changes in these MT parameters were quantified using Fiji/ImageJ workflows. Rt-qPCR was used to measure gene expression of MT dynamics genes.

## Results

PPA stress during differentiation resulted in increased MT volume and network fragmentation compared to control neurons. PPA induced subtle disruptions to autophagic flux impairing degradation by decreasing autophagosome lysosome fusion. Changes in MT dynamics gene expression supported these morphological results.

## Conclusion

This work investigated the impact of PPA stress on neurons and its impact on neuronal differentiation. These data provide insight into the relationship between MT dynamics and function, generating new knowledge of the association between MT and neuronal function in the context of neurodevelopmental disorders. Ultimately, this work adds to the body of knowledge investigating ASD's aetiology and pathophysiology.



## POSTER PRESENTATIONS

**Lilitha Cengani**

**University of Cape Town**

**SANS26-P63**

Cryptococcal meningitis (CM), caused by two etiologic agents namely *Cryptococcus neoformans* (Cn) and *Cryptococcus gattii* (Cg), is a lethal fungal infection of the central nervous system (CNS) that disproportionately affects low-middle income countries. Disease progression is determined, in part, by the pathogen's ability to modulate immune responses, however how this occurs in the CNS, a main site of infection with exceedingly diverse cell populations, remains poorly elucidated. Moreover, conventional approaches to assess these neuroimmune responses are limited in their ability resolve and assess the functional state of many individual cell types simultaneously.

Therefore, in this study we established a flow cytometry-based pipeline for multiparametric phenotypic and functional characterization of different cell types in the CNS using a multi-colour antibody panel. Hippocampal organotypic brain slice cultures were made from post-natal day 7 C56BL/6 mice and maintained for seven days in vitro. Slices were subjected to treatment with lipopolysaccharide (LPS), Cn, Cg or left untreated (Controls), thereafter dissociated and stained for flow cytometry.

Using a multi-colour antibody panel, we were able to identify and quantify major CNS cell types present in organotypic slice cultures, including microglia, astrocytes, and neurons. Further analysis revealed a shift in transcription factor NFIL-6 (C/EBP  $\beta$ ) expression in multiple cell types across treatment groups.

With this pipeline, we achieved detailed resolution of CNS heterogeneity using a current CM model, moreover preliminary data suggesting its utility for analysing host-pathogen interactions that drive disease progression rendering flow cytometry a powerful technique that can support the identification of potential therapeutic targets.



## POSTER PRESENTATIONS

**Yididya Tilahun Dansa**

**Nelson Mandela University**

**SANS26-P20**

Cannabis and cannabis-based product use has increased following expanded recreational and medicinal access, particularly among adolescents and young adults with developing brains. The principal phytocannabinoids,  $\Delta^9$ -tetrahydrocannabinol (THC) and cannabidiol (CBD), may exert contrasting neurobiological effects. However, their individual and combined effects on hypothalamic inflammation remain insufficiently characterised. While stress is known to promote inflammatory processes, its interaction with THC, including effects on cytokine signalling, epigenetic regulation, gene expression, and sex-specific responses remains poorly understood. This study investigates sex-linked differences in hypothalamic inflammatory responses following exposure to cannabinoids and/or repeated mild stress (RMS), associated alterations in DNA methylation, gene expression and cytokine profiles.

Mice were assigned to two experimental phases. In the first phase, animals were randomized into four treatment groups: negative control, THC (5 mg/kg), CBD (20 mg/kg), and THC + CBD. Phase II examined THC and RMS-induced inflammatory changes using four groups: negative control, THC (5 mg/kg), and RMS + THC. Control animals received Cremophor RH40 dissolved in saline, while RMS groups were subjected to an unpredictable 7-day RMS paradigm.

Following treatment, hypothalamic tissues were collected and analysed for cytokine levels (TNF- $\alpha$  and IL-13) utilising sandwich ELISA. Preliminary data showed no statistically significant differences among treatment groups, although combined exposure resulted in observable trends indicative of modulatory effect. Gene expression and DNA methylation analyses are ongoing.

The research seeks to advance understanding of cannabinoid/stress related neuroinflammatory mechanism, inform future cannabinoid regulatory policies and contribute to therapeutic strategies.



# POSTER PRESENTATIONS

**Veronique de Jager**

**University of Cape Town**

**SANS26-P19**

## Background

Central nervous system (CNS) tuberculosis (TB) causes significant neuroinflammatory disease and frequently results in long-term neurological disability and cognitive impairment. While imaging studies have demonstrated widespread white matter disruption in intracranial TB, the cellular mechanisms underlying this white matter injury are poorly understood.

## Methods

We used single-nucleus RNA sequencing of brain tissue from a *Mycobacterium tuberculosis* (M.tb)-infected CNS-TB mouse model to characterise neuroglial cell-type responses at single-cell resolution. We administered an intracortical dose of H37Rv M.tb to C57BL/6 mice, while uninfected controls received sterile saline intracortically.

## Results

We found a global reduction of oligodendrocytes (OLs) and precursor cells (OPCs) in M.tb-infected tissue relative to uninfected controls. Differential gene expression and pathway enrichment analysis revealed an upregulation of innate immune response genes and interferon-mediated signalling pathways in both OLs and OPCs. In OLs, pathways enriched among downregulated genes include OL differentiation, myelination, and ribosomal subunit biogenesis. OL downregulated genes in infected tissue include Sox10, a master transcription factor for OL lineage cells, and the myelin protein-encoding genes Ppl1 and Opalin. Similarly, OPCs showed downregulation of genes encoding ribosomal proteins, in addition to the transcriptional coactivator Arglu1, and the iron storage protein Fth1. Preliminary data indicates a proportional increase in newly formed oligodendrocytes with a decrease in mature oligodendrocytes when comparing M.tb-infected samples to uninfected controls. This may indicate a stage-specific developmental impairment at the early myelin-generating transition of OL lineage cells.

## Conclusion

CNS-TB induces interferon-mediated immune signalling in OL lineage cells while suppressing cellular protein translation and myelination programs.



# POSTER PRESENTATIONS

**Michael Patrick Edwards**

**University of Cape Town**

**SANS26-P44**

## Background

Long non-coding RNAs (lncRNAs) are essential for human brain function and development. Cis-acting lncRNAs that regulate protein-coding genes linked to neurodevelopmental and neurological disorders are potential targets for therapeutic design particularly when the disorder results from haploinsufficiency. This is exemplified by the regulation of CHD2 by the lncRNA Chaserr with homozygous loss of the latter resulting in CHD2 overdose.

## Methods

Protein-coding genes of specific interest to neurodevelopmental and/or neurological disorders, for which haploinsufficiency was available, were retrieved from the Clinical Genome Resource (ClinGEN). Uniformly processed human central nervous system RNA-seq datasets were accessed from the ARCHS4 database. Correlation network analysis identified chromosome specific patterns of lncRNA and protein-coding genes of interest co-expression. Cis-acting regulatory lncRNAs were matched to potential targets based on proximity on the same DNA strand as well as long-distance interactions on the same chromosome enabled by DNA looping (validated with available Hi-C data). Additional validation of the identified lncRNA and target gene pairs employed assessing co-expression across single cell RNA datasets with specific preference for relevant disease datasets.

## Results

A list of 417 protein-coding genes of interest linked to neurodevelopmental and/or neurological disorders for which strong evidence of haploinsufficiency was produced. Meta-analysis of RNA-seq samples from 1,328 experimental series, representing 11 regions of the brain, provided preliminary results and indicated distinct patterns of co-expression. Chromosome specific pairs of lncRNAs and potential regulatory targets, including Chaserr-CHD2, were identified for downstream validation.

## Conclusion

These positive preliminary results indicate the necessity for further downstream analysis.



## POSTER PRESENTATIONS

**Alice Efundayo**

**SANS26-P59**

**University of Limpopo; University of the Witwatersrand**

Alcohol is a commonly used and abused drug by adolescents and causes neurodevelopmental, neurobehavioral and neurocognitive disorders due to the negative effects of chronic alcohol use on the structure and function of neurons. Simvastatin, an FDA-approved drug for lowering blood cholesterol levels, seems promising in preventing neurodegeneration. This study evaluated the effect of Simvastatin on the sizes of the PMBSF barrels of the somatosensory cortex and morphometry of the sciatic nerve of adolescent mice exposed to alcohol. Four-week old C57BL/6J male and female mice were administered 20% alcohol (i.p.), 5 or 10 mg/kg Simvastatin orally followed by 20% alcohol (i.p.) or the controls (i.e. 5 mg/kg Simvastatin only or non-treated) for 28 consecutive days. The size of the 27-PMSBF barrels and the morphometry of the sciatic nerve were assessed. Alcohol did not significantly reduce the mean areas of the PMBSF barrels, the enclosure, or the septal portion in both sexes. However, barrel-to-barrel comparison revealed alcohol toxicity on specific barrels in specific rows and arcs of the PMBSF barrels. Both Simvastatin concentrations were effective against alcohol-induced damage on those specific barrels. For the sciatic nerves, axonal density and myelination were significantly affected by alcohol in both sexes and Simvastatin was effective against neuropathy. Simvastatin seems to have the ability to protect against the damaging effect of alcohol and may be beneficial in reducing the prevalence of alcohol-related diseases or disabilities, especially in adolescents that are prone to abusing alcohol.

# POSTER PRESENTATIONS

**Yohannes Ayana Ejigu**

**Bahir Dar University**

**SANS26-P15**

We present a clinically oriented deep learning approach for automated detection of epileptic seizures from scalp electroencephalography (EEG) recordings. The proposed method integrates domain-specific features, including spectral band-power and signal complexity measures such as Shannon entropy and Hjorth parameters, into a compact one-dimensional convolutional neural network (CNN). We evaluate the model on the Children's Hospital Boston and Massachusetts Institute of Technology (CHB-MIT) pediatric EEG dataset. While the model achieves high discrimination, with an area under the ROC curve of about 0.98 and accuracy of about 99% on held-out balanced data, the default decision threshold of 0.5 yields poor seizure recall. By lowering the threshold to 0.10 based on validation analysis, seizure sensitivity improves to about 91%, with a manageable false-alarm rate of about two to three per hour. Beyond accuracy, we report cross-validation with confidence intervals, event-based detection metrics, and ablation studies comparing spectral, complexity, and raw EEG inputs. Because the features are physiologically interpretable, model outputs can be linked to meaningful EEG characteristics. We also discuss limitations, including generalization beyond pediatric single-center data, and propose directions for future validation and real-time deployment.



# POSTER PRESENTATIONS

**Snjin Fanner**

**University of Cape Town**

**SANS26-P78**

Haploinsufficiency of the chromatin remodeller, chromodomain helicase DNA-binding protein 2 (CHD2), results in early-onset epileptic encephalopathy and is implicated in timing neurodevelopment. However, the window during which CHD-associated transcription programmes become embedded during human neurodevelopment remains unclear. This project aims to modify an African ancestry human induced pluripotent stem cell (hiPSC) line to carry an inducible degron-tagged CHD2 allele to enable rapid and reversible protein depletion. The goal is to define the temporal requirements of CHD2 during differentiation toward inhibitory interneuron lineages.

## Methods

Degron tags and necessary protease machinery are introduced at the CHD2 and AAVS1 locus in hiPSCs using CRISPR/Cas9-mediated homology-directed repair. Correctly edited clones are identified by PCR screening and sequencing, followed by validation of protein expression and inducible degradation using immunoblotting. Edited lines are subjected to established differentiation protocols toward medial ganglionic eminence-like progenitors and downstream interneuron fates. CHD2 degradation is induced at defined developmental stages, and effects on lineage progression and histone markers are assessed using immunocytochemistry and CUT&RUN targeting bivalent promoters.

## Results

We anticipate establishing a validated CHD2 degron hiPSC line demonstrating efficient and tunable protein degradation. Differentiation is expected to yield interneuron progenitors and mature neuronal populations, providing a platform to assess stage-specific consequences of CHD2 loss on cell fate specification and gene expression programs.

## Conclusions

This work will deliver a controllable system to dissect CHD2 function with temporal precision in human neurodevelopment. The approach is expected to clarify how CHD2 contributes to interneuron-specific transcriptional programmes and may provide insight into CHD2-associated neurodevelopmental disorders.



# POSTER PRESENTATIONS

**Saba Gebrezeilassie**

**University of Cape Town**

**SANS26-P52**

## Background/Aim

Cryptococcal meningitis (CM) is an opportunistic fungal infection of the central nervous system caused by *Cryptococcus* spp.. It disproportionately affects Sub-Saharan Africa. While *C. neoformans* infects hosts via the pulmonary system, it preferentially disseminates to the brain. Once there, fungal cells organise themselves into aggregates that lead to tissue lesioning in the parenchyma, known as cryptococcomas. Preliminary data from our laboratory suggest that cryptococcomas are poorly vascularised and may represent a hypoxic micro-niche for fungal persistence. As *Cryptococcus* utilises alternate metabolic pathways in low-oxygen environments, such niches may promote fungal survival. This study investigated the role of cryptococcomas in pathogenesis by detecting and analysing the presence of hypoxia.

## Methods

Brain sections were obtained from mice injected intravenously with a GFP/mCherry expressing strain of *C. neoformans*. Tissue hypoxia was measured using the Hypoxyprobe™ pimonidazole, a reducing agent that binds to proteins under hypoxic conditions. Immunofluorescent images were obtained using confocal microscopy. We created a customised analysis pipeline in Fiji/ImageJ to quantify the levels of hypoxia within the brain.

## Results

Using our custom analysis macro, we successfully quantified fungal cells and hypoxia levels within lesions considering the three-dimensional context. Most cryptococcal lesions exhibited moderate to high levels of hypoxia, suggesting these microenvironments are conducive to fungal proliferation.

## Conclusion

Cryptococcomas may serve as fungal niches that aid persistence due to the presence of hypoxia.



# POSTER PRESENTATIONS

**Chloë Goldman**

**University of Cape Town**

**SANS26-P35**

## Background/Aim

Mitochondria are central regulators of neural stem cell (NSC) fate, modulating neurogenesis and gliogenesis during neurodevelopment. Although mitochondrial dysfunction is associated with several neurodevelopmental disorders, how metabolic stress shapes early NSC lineage commitment remains poorly understood. Propionic acid (PPA) has been used to model metabolic stress during neurogenesis but is unexplored in the multipotent ReNcell CX NSC system. This study aimed to establish an in vitro NSC differentiation model and assess how PPA-induced metabolic stress influences early gliogenesis.

## Methods

The cytotoxicity of PPA treatment was characterised in the ReNcells. NSCs were differentiated over seven days via growth factor withdrawal. The effects of PPA on NSC morphology, fate and mitochondrial remodelling during early development was examined using immunocytochemistry, widefield and confocal microscopy.

## Results

Effective NSC differentiation was illustrated by an increase in the glial-specific protein, Glial fibrillary acidic protein (GFAP), relative to the stem cell marker, Nestin. However, exposure to PPA induced morphological changes, including hypertrophy and enlarged astroglial processes. PPA increased both total Nestin and GFAP expression, consistent with enhanced gliogenesis, increased glial reactivity, and a gliosis-like phenotype. Moreover, PPA disrupted mitochondrial remodelling during NSC differentiation, marked by an increase in mitochondrial content, density and network complexity.

## Conclusion

This data demonstrates a link between metabolic stress and glial reactivity using a multipotent in vitro system and establishes a workflow for investigating the relationship between metabolic state and NSC maturation. Further characterisation of this model may yield novel insight into how early metabolic perturbations influence mitochondrial dynamics in neurological disease.



## POSTER PRESENTATIONS

**Roxanne Megan Hattingh**

**University of Cape Town**

**SANS26-P54**

Pyramidal neurons are the primary excitatory neurons of the neocortex and possess unique properties which may be necessary for complex cognitive functions. Animal studies have shown these cells undergo considerable maturation after birth. In recent years, evidence that human paediatric neurons differ from adults has come to light, in terms of morphology, dendritic spine density, and electrical properties. However, the manner and timing of their maturation has never been characterised fully in humans, and little is known about the physiological effects of these changes. Making use of ante-mortem brain tissue resected during epilepsy surgery from patients aged 10 months to 50 years, we investigate the postnatal maturation of pyramidal neurons in layer 2/3 of the temporal cortex. Using patch-clamp electrophysiology, synaptic inputs were characterised, revealing a developmental shift in synaptic input profiles. Analysis of 3D whole-cell morphological reconstructions showed that while dendritic diameter increases alongside limited dendritic growth far into adulthood, total dendritic size and complexity are largely established in infancy. Super-resolution confocal microscopy provided detailed characterisation of dendritic spine density and morphology, confirming a decline in spine density and distinct shifts in spine morphology from childhood into adulthood. While spine density decreases into adulthood, spine head diameter increases significantly, indicating a progressive stabilization of neural circuits. This conjunction of functional and morphological data allows us to decouple the effects of structural and molecular changes, suggesting that functional maturation is not driven by major increases in cell size, but by the refinement of synaptic architecture through changes in spine morphology and function.



# POSTER PRESENTATIONS

**Abakar Ali Abdoulaye Haggar**  
**Sidi Mohamed Ben Abdellah**

**SANS26-P05**

Parkinson's disease (PD) is a major complication in older adults and is characterised by the degeneration of dopaminergic neurons. Voxel-based morphometry (VBM) has been used to detect differences in cortical and subcortical structures and to map grey matter volume (GMV) between groups. Studies published on VBM variations in GMV in PD have revealed conflicting results. Our study aims to analyse the structures affected in previous studies in order to identify variations in grey matter volume in Parkinson's disease.

**Methods:** We identified neuroimaging studies in patients with PD and voxel-based morphometry by conducting a literature search in the PubMed and SCOPUS databases. We reviewed articles published between September 2015 and November 2025.

**Results:** We obtained a total of 10 studies, including 416 patients with Parkinson's disease and 358 controls. Significant loss in grey matter volume (GMV) were shown in certain cortical and subcortical regions in patients with Parkinson's disease. They are characterised by significant brain alterations in grey matter in the bilateral hippocampus, right thalamus, amygdala, left subthalamic nucleus and putamen. These results did not show any particular changes during the synthesis of our analysis.

**Conclusion:** Patients with Parkinson's disease showed significant reductions in grey matter volume, particularly in multiple brain regions involved in cognitive functions. These findings provide morphological evidence that contributes to our understanding of the pathophysiology of Parkinson's disease.



## POSTER PRESENTATIONS

**Rajamohamed Haitharali**

**SANS26-P17**

**JSS College of Pharmacy, JSS Academy of Higher Education & Research, Ooty, Nilgiris, Tamil Nadu, India.**

### Background

The gut-brain axis is a bidirectional communication system that links the central nervous system with the enteric nervous system and the gut microbiota, playing a vital role in maintaining neurophysiological homeostasis and cognitive function. Recent advances suggest that gut microbiota-derived metabolites, especially short-chain fatty acids (SCFAs) such as acetate, propionate, and butyrate, may play a crucial role in modulating brain function, behavior, and cognition. Objective: To quantify serum SCFA levels and evaluate their association with Intelligence Quotient (IQ) among healthy Indian adults using LC-MS analysis and validated cognitive testing.

### Methodology

Thirty participants of age from 18-30 years were enrolled, blood serum level were quantified for SCFA using liquid chromatography and mass spectrometry (LC-MS), cognitive performance were assessed using Ravens scale, verified in the use of Indian population. The relation between SCFA and IQ were estimated using spearman correlation coefficient.

### Results

Mean plasma SCFA concentrations ( $\mu\text{mol/L}$ ) were acetate:  $65.4 \pm 12.3$ , propionate:  $21.6 \pm 6.7$ , and butyrate:  $18.9 \pm 5.2$ , with a mean IQ of  $107.3 \pm 11.5$ . A significant positive correlation was observed between total SCFA levels and IQ ( $\rho = 0.62$ ,  $p < 0.01$ ). Among individual metabolites, butyrate demonstrated the strongest association ( $\rho = 0.71$ ,  $p < 0.001$ ), followed by propionate ( $\rho = 0.58$ ,  $p < 0.01$ ), while acetate showed a weaker trend ( $\rho = 0.32$ ,  $p = 0.08$ ).

### Conclusion

These preliminary findings suggest that gut microbial metabolites, particularly butyrate, may play a meaningful role in human cognitive functioning. The strong SCFA-IQ associations highlight the need for larger, mechanistic studies to elucidate how microbial metabolism influences brain health and to explore dietary strategies that enhance beneficial SCFA production.

Keywords: Gut-brain axis, SCFA, cognition, IQ, Indian population.

# POSTER PRESENTATIONS

**August Alessandro Herbert**

**University of Cape Town**

**SANS26-P37**

## Background:

The development of the human brain from the neuroectodermal layer to a highly structured organ is profoundly complex. While bulk RNA-sequencing has traditionally been used to study this process at a molecular level, advances in transcriptomics, including single-nucleus RNA-sequencing (snRNA-seq) and high-resolution spatial transcriptomics, have enabled more comprehensive characterisation. Additionally, emerging approaches such as Smart-seq3 snRNA-seq and multiplexed error-robust fluorescent in situ hybridisation (MERFISH) have provided increasingly detailed insights.

## Aims:

This project, building upon recent work by Steyn et al. (2024) in the postnatal human cortex, will explore Smart-seq3 data to validate existing 10x Genomics Chromium snRNA-seq findings and uncover novel insights into neural gene expression, alongside MERFISH-based validation of previously-described cell-subtype-specific minimal markers.

## Methods:

These aims are primarily being accomplished through in silico analysis using tools such as Seurat and CellPose. Furthermore, experimental techniques including fluorescent in situ hybridisation and chick-embryo in vivo studies will be used to investigate key findings, including novel age-dependent differentially-expressed genes (DEGs) and the tissue-specific activity of gene-regulatory networks.

## Results:

At this stage, the main results have been the development of pipelines for processing Smart-seq3 data and for quantifying nuclear transcript numbers in MERFISH images. Preliminary results also include the identification of novel oligodendrocyte DEGs and associated enriched pathways.

## Conclusion:

Ultimately, this research is ongoing, and the eventual breadth of its outputs will depend on the integration of additional data that is currently being generated. However, the work completed to date is promising and shows a strong trajectory for the project's eventual outcome.



## POSTER PRESENTATIONS

**Nai-Jen Hsu**

**University of Cape Town**

**SANS26-P21**

*Mycobacterium tuberculosis* (Mtb) is the causative pathogen of the tuberculosis diseases. Individuals with compromised immunity are at risk of developing extra-pulmonary tuberculosis, particularly central nervous system tuberculosis (CNS-TB) which is associated with high mortality and morbidity. Although microglia are preferentially infected by Mtb within the CNS, neurons have been identified as host cells for the pathogen and demonstrated their potential to participate in a tightly regulated neuro-immune network.

Previously, we showed that neurons can internalize Mtb bacilli both in cultures and in animals and elicit an immune response by secreting IL-1 $\beta$ , IL-6, and IL-10. Here, we investigated the immune regulatory abilities of neurons in CNS-TB using transcriptomic analysis in primary murine hippocampal neurons infected with H37Rv. After 24 hours of infection, RNA was extracted for microarray analysis, allowing us to assess gene expression differences between infected and non-infected neurons. Transcriptional module enrichment analysis showed the significantly induced genes of biological processes were prominently involved in inflammatory pathways. We observed an upregulation of cytokine and chemokine transcripts (CCL2, CXCL1, CXCL2, CXCL10 and IL6) and detected a significant increase of these protein levels by Luminex. To functionally evaluate the effect of neuronal immune regulation, we cultured mouse splenocytes with neuronal conditioned media, and flowcytometry analysis demonstrated the increased IL-1 $\beta$  expression in CD45+ leukocytes stimulated by infected neurons but not uninfected neurons.

The findings from this study show that neurons are not passive bystanders during CNS infections but capable of producing an immune response and modulating immune functions in response to infection.



## POSTER PRESENTATIONS

**Jay Vivek Jagdale**

**Government Medical College, Nagpur**

**SANS26-P26**

### Introduction:

Stroke is a leading cause of disability, often resulting in hemiplegia and impaired performance in activities of daily living (ADLs). Occupational Therapy (OT) plays a crucial role in improving functional independence through task-oriented and patient-centered interventions. This study aims to evaluate the effect of basic occupational therapy interventions on functional recovery in patients with hemiplegia.

### Methods :

A small group of patients with post-stroke hemiplegia attending an outpatient department were observed over a period of 12 weeks. Baseline and follow-up assessments were conducted using standardized outcome measures including the Barthel Index for ADL performance, the Fugl-Meyer Assessment for motor function, and the Modified Ashworth Scale for muscle tone.

OT interventions included therapeutic positioning, passive and active-assisted range of motion exercises, task-oriented training, and functional activity practice focusing on ADLs such as feeding, grooming, and dressing. Hand function activities and bilateral integration tasks were also incorporated. Patients and caregivers were educated regarding home exercise programs to ensure continuity of care.

### Results :

Patients demonstrated improvement in functional independence, as reflected by increased scores on the Barthel Index. Motor recovery showed positive trends, along with reduction in spasticity as per the Modified Ashworth Scale. Clinically, patients required reduced assistance in ADLs and showed improved coordination and functional use of the affected upper limb.

### Conclusion :

OT interventions, even at a basic level, contribute significantly to functional recovery in patients with post-stroke hemiplegia. Structured rehabilitation focusing on ADLs, along with consistent practice and caregiver involvement, plays a key role in enhancing independence and quality of life.



# POSTER PRESENTATIONS

**Khadija Jamal**

**University of Cape Town**

**SANS26-P64**

## Background/Aim

Tuberculosis (TB) remains a major global health burden, with paediatric central nervous system TB (CNS-TB) associated with high mortality and long-term neurological impairment. Progress in understanding its immunopathogenesis is limited by scarce brain tissue, high clinical variability, and the inherent heterogeneity of human samples. These constraints highlight the need for experimental models that can provide clear mechanistic insight. This project primarily investigates CNS-TB using the C3HeB/FeJ (Kramnik) mouse model, which develops necrotic lesions resembling severe human CNS-TB disease.

## Methods

We use the C3HeB/FeJ (Kramnik) mouse model to investigate CNS-TB. Single-nucleus RNA sequencing (snRNA-seq) is applied to profile cell states (neuronal and non-neuronal), map inflammatory trajectories by comparing day-17 and day-28 samples to identify key pathways during infection at innate and adaptive immune response states. In parallel, snRNA-seq is also being performed on paediatric human brain samples from tuberculoma (contained) and tuberculous meningitis (uncontained) cases. Cross-species integration is used to compare datasets.

## Results

The mouse model enables high-resolution characterisation of immune and neural cell states in a controlled setting, revealing reproducible inflammatory patterns and disease-associated pathways. Human samples capture complex, heterogeneous immune infiltration and lesion diversity. Comparative analysis identifies both conserved and divergent immune signatures between mouse and human CNS-TB.

## Conclusion

This integrative approach combines mechanistic insight from the Kramnik mouse model with clinical relevance from human samples. It provides a framework to evaluate model fidelity and identify conserved pathways driving CNS-TB, supporting future translational research.



## POSTER PRESENTATIONS

**Sahar Bathsheba Jamal**

**University of Cape Town**

**SANS26-P70**

The human brain is a highly dynamic organ that undergoes continuous change throughout the lifespan. Cell differentiation, specificity, function and fate are guided by gene regulatory networks (GRN's). GRN's orchestrate gene expression through conserved and dynamic coordination of molecular components; signalling molecules (such as hormones and metabolites) influence transcription factor (TF) activity and in turn TF motifs bind to cis-regulatory elements (CRE's) such as promoter, enhancer and silencer regions to modulate gene expression alongside non-coding RNAs (ncRNA). Dynamic chromatin accessibility, influenced by chromatin remodelling factors, plays a major role in GRN activity. Across the course of brain maturation, cell-type specific changes in enhancer accessibility allow for both stable and activity-dependent gene expression.

Antemortem temporal lobe access tissue from age groups spanning paediatric to adult have been biobanked, sourced via collaboration with Cape Town Neurosurgeons, obtained through elective epileptic resections in local hospitals. The identification of putative enhancers using single nucleus assay for transposase accessible chromatin sequencing (snATAC-seq) across a selection of these samples allows for the bridging of a major gap in neuro-epigenetic research, as the majority of studies have focused on postmortem samples, with very little representation of the paediatric population.

The optimisation of bioinformatics pipelines for the identification of putative enhancers that are dynamically accessible across the course of brain maturation allows for downstream evaluation of biochemical pathways that not only provide insight into the molecular processes informing cellular function, communication and organisation but also illuminates potential therapeutic targets for the alleviation of neurodevelopmental, neuropsychiatric and neurodegenerative pathologies.



# POSTER PRESENTATIONS

**Lerise Johnson**

**Nelson Mandela University**

**SANS26-P43**

## Background

Despite the efficacy of antiretroviral drugs (ARVs) at suppressing human immunodeficiency virus-1 (HIV-1) replication, many patients still develop neurological complications related to the virus's pathogenicity. In addition to the neurotoxic effects of viral products such as HIV-1 Tat protein, ARVs have also been identified as potential contributors to the development of neuroinflammation. Therefore, the current study aims to investigate the potential synergistic effects of HIV-1 Tat protein and ARVs on proinflammatory markers in cultured human hippocampal astrocytes.

## Methods

We used primary hippocampal astrocytes to evaluate the combined effect of antiretroviral drugs individually and in combination with Tat protein. Cultured astrocytes were exposed to clinically relevant concentrations of dolutegravir (DTG) (0.064 µg/ml), lamivudine (3TC) (1.51 µg/ml), and tenofovir disoproxil fumarate (TDF) (0.3 µg/ml). Following 24 hours of exposure, antiretroviral cytotoxicity was assessed using a colorimetric 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) assay. Additionally, experiments will be conducted to measure interleukin-1β (IL-1β) and tumour necrosis factor-α (TNF-α) concentrations in conditioned media. Expression of NF-κβ and GFAP will be measured using immunocytochemistry.

## Results

Data analysis of MTT results shows that DTG, 3TC, and TDF have no cytotoxic effect on astrocytes compared with the control group.

## Conclusion

Our current results suggest that antiretroviral drugs do not cause toxicity in astrocytes. However, further biochemical analysis of the combined effects of antiretroviral drugs and Tat on proinflammatory markers are still to be investigated in the current experiment.

## POSTER PRESENTATIONS

**Maahir Kauchali**

**SANS26-P74**

**University of Cape Town**

As global temperatures rise and climatic patterns shift, an increasing number of emerging fungal pathogens have been documented worldwide, driving new and expanding epidemics. One example is *Cryptococcus gattii*, an encapsulated yeast that is capable of invading the central nervous system to cause cryptococcal meningitis, a neuroinfectious diseases associated with death and disability HIV patients. Unlike *Cryptococcus neoformans*, which predominantly affects immunocompromised individuals, *C. gattii* frequently infects immunocompetent hosts, often leading to pronounced neurological sequelae.

Patients with CM commonly develop debilitating brain injury that can result in death or persistent neurological impairment; however, the mechanisms underlying this damage remain poorly understood. The aim of our study was to characterise the neuropathogenesis of *C. gattii* and to determine how it differs from that of *C. neoformans*.

We infected male, and female C57BL/6 mice with  $5 \times 10^4$  colony forming units (CFU) of either *C. gattii*, or *C. neoformans*. We carried out a survival analyses for 30 days post infection (dpi) and measured body mass daily. Brains were collected at specified time points for determination of fungal burden, cytokines and for histopathological analyses.

We observed a strain specific, sex-dependent neuropathological response of mice to cryptococcal infection with males are twice as likely to succumb to infection compared to females. Despite no significant differences in fungal burdens between the sexes, distinct differences were observed in lesion numbers and sizes, and cytokine levels in the brain and serum. These findings highlight distinct temporal and pathological features of *C. neoformans* and *C. gattii* infection across genders.



## POSTER PRESENTATIONS

**Sumaiya Khuwaja**

**SANS26-P68**

**Liaquat National Hospital and Medical College Karachi**

### Background:

Cerebral creatine deficiency syndromes (CCDS) are inborn errors of creatine synthesis and transport, leading to reduced cerebral creatine levels and neurological impairment. Creatine transporter defects, such as CCDS type 2, are characterized by intellectual disability, developmental delay, seizures, and abnormal brain MR spectroscopy findings. This report describes a genetically confirmed case of CCDS type 2 from Pakistan, emphasizing diagnostic challenges and the importance of timely genetic evaluation.

### Methods:

A 13-year-old boy with global developmental delay, limited speech, intellectual disability, and epilepsy presented with irritability, sleep disturbance, and a recent generalized tonic-clonic seizure. EEG demonstrated an encephalopathic pattern, while brain MRI revealed bilateral hyperintense signals in the basal ganglia. Whole genome sequencing was performed for genetic evaluation.

### Results:

Serum ammonia levels were elevated due to valproic acid-topiramate interaction, while baseline laboratory tests were normal except for low serum creatine. Whole genome sequencing identified a pathogenic variant in the creatine transporter gene (SLC6A8), confirming CCDS type 2. The patient was managed with adjustment of antiepileptic medications, creatine supplementation, and dietary modifications. Following management, irritability and sleep improved.

### Conclusion:

This genetically confirmed CCDS type 2 case highlights the diagnostic value of genomic testing and the need for clinical awareness of creatine deficiency syndromes to improve outcomes in resource limited settings.



## POSTER PRESENTATIONS

**Sheri-Michelle Koopowitz**

**University of Cape Town**

**SANS26-P22**

### Background

Early life adversity (ELA) is associated with internalizing and externalizing behaviour in children in high-income cohorts; however, it is unclear whether these associations generalize to low- and middle-income countries (LMICs). We investigated the association between ELA and internalizing and externalizing behaviours in children in three LMIC cohorts.

### Methods

Harmonised measurements from three cohorts, the Brazilian High-Risk Cohort (BHRC;  $n = 1111$ ), the Indian Sustainable Programme Incorporating Nutrition and Games (SPRING;  $n = 601$ ), and the South African Drakenstein Child Health Study (DCHS;  $n = 708$ ) were pooled. Children (7-10 years) were assessed using the SDQ. ELA was measured using a cumulative index, including socioeconomic, relational, maternal stress, and child adversity in the first 2 years of life. Linear regression models estimated associations between ELA and behavioural outcomes, adjusting for age, sex, and cohort.

Results 2420 children (53.9% boys; mean age: 8.3 years) were included. Early life adversity was associated with higher SDQ total scores ( $B = 1.97$ , 95% CI: 1.16, 2.78,  $p < .001$ ), and greater internalizing ( $B = 0.84$ , 95% CI: 0.41, 1.27,  $p < .001$ ) and externalizing behaviours ( $B = 1.13$ , 95% CI: 0.62, 1.64,  $p < .001$ ). Across all outcomes, cohort-specific associations were strongest in BHRC and attenuated in SPRING. Associations were stronger at older ages, while sex differences were inconsistent across cohorts and outcomes.

Conclusions: Heterogeneous associations of early life adversity with child behaviour across LMIC settings were observed, highlighting that associations with adversity are context-dependent. Context-specific interventions aimed at local adversities may reduce child behaviour problems associated with adversity.

## POSTER PRESENTATIONS

**Haley Josephine Lewendal**  
**Stellenbosch University**

**SANS26-P71**

### Background:

Neuroinflammation is a key contributor to neurodegenerative disease progression and is linked to glucotoxicity from untreated diabetes. Glucotoxicity drives mitochondrial dysfunction, oxidative stress, and the priming and activation of inflammatory pathways in neuronal cells. These processes converge on NF- $\kappa$ B signalling and NLRP3 inflammasome priming and activation, promoting neuronal injury and impairing neuroprotection. Limited research has examined plant-derived compounds targeting glucotoxicity-induced neuroinflammation. Honeybush (*Cyclopia intermedia*), an indigenous South African plant rich in polyphenols, has shown antioxidant and anti-inflammatory properties. This study is the first to investigate its potential to modulate neuroinflammatory signalling and support neuroprotection under glucotoxic conditions.

### Methods:

Honeybush aqueous extracts were prepared, and the phytochemical fingerprint analysed using HPLC. Neuronal cells were exposed to glucotoxic conditions with or without honeybush extract. Neuroinflammatory, oxidative stress and neuroprotective responses were assessed using microscopy, ELISA and flow cytometry. Mechanistic evaluation included gene and protein expression analysis of key regulators of inflammasome priming and activation.

### Results:

HPLC identified hesperidin and mangiferin as major polyphenols. Under glucotoxic conditions, IL-1 $\beta$  and NLRP3 gene expression were upregulated, indicating inflammasome priming; however, no mature IL-1 $\beta$  was detected, suggesting a lack of inflammasome activation. Honeybush treatment reduced mitochondrial superoxide production, indicating decreased oxidative stress and, therefore, limited inflammasome activation. It also increased BDNF expression and upregulated antioxidant pathways, suggesting enhanced neuroprotection.

### Conclusion:

This study shows that honeybush may prevent progression from inflammatory priming to full inflammasome activation while promoting neuroprotection under glucotoxic conditions.



## ORAL PRESENTATIONS

**Asiphaphola Ludidi**

**University of KwaZulu-Natal**

**SANS26-P13**

### Background:

Parkinson's disease (PD) is the second most prevalent neurodegenerative disease worldwide. Pathology of PD is the degeneration of the dopaminergic neurons and accumulation of alpha-synuclein ( $\alpha$ -syn) in Lewy Bodies. An elevated circulation of pro-inflammatory cytokines rendering neurons vulnerable to toxic insults accompanies chronic stress. Chronic stress therefore exacerbates the progression of PD. Currently, no cure for PD exist, however increased caffeine consumption has been shown to decrease the risk of developing PD. However, caffeine's involvement in alpha-synuclein ( $\alpha$ -syn) accumulation is unknown.

### Aim:

The aim of the study is to investigate the therapeutic effects of caffeine on  $\alpha$ -syn accumulation in a maternally separated rat model.

### Methods:

Sprague Dawley rats were maternally separated to induce stress. Right striatum was stereotaxically lesioned with 6-hydroxydopamine (6-OHDA) and caffeine was used as an intervention. Behavior was assessed using the cylinder and step test. Alpha-synuclein concentration in the striatum was measured.

### Results:

Unilateral infusion with 6-OHDA resulted in contralateral forelimb (left) impairment exacerbated by exposure to maternal separation. Additionally,  $\alpha$ -syn protein was upregulated in the striatum of maternally separated rats. Caffeine ameliorated resultant motor deficits.

### Conclusion:

Caffeine in the striatum of maternally separated rats showed compelling evidence suggesting caffeine as neuroprotective particularly in advanced stages of PD. This finding may potentially bring us closer to a cost-effective and non-invasive treatment for PD. In addition, findings from this study will also help us to identify a novel neuroprotective mechanism of caffeine in patients suffering from PD.



## POSTER PRESENTATIONS

**Asiphaphola Ludidi**

**University of KwaZulu-Natal**

**SANS26-P06**

### Background

Prediabetes in gestation was associated with placental malfunction and increased risk of preeclampsia. Aim: We further investigated if the pathological mechanisms in the erythrocyte-spleen axis increased the risk of preeclampsia in High-fat, high-carb diet-induced prediabetic Sprague-Dawley rats.

### Methodology

21-days old female Sprague-Dawley rats consumed an HFHC diet with fructose for 36 weeks to induce prediabetes. Body weight, blood glucose concentrations, mean arterial pressure (MAP) and albuminuria were monitored on gestational day (GND) 0, 9 and 18. All animals were decapitated on GND 18. Terminally, blood and spleens were collected to assess HbA1c, erythrocyte profile, inflammatory and antiangiogenic biomarkers.

### Results

Prediabetes showed poor glycaemic control, suggesting insulin resistance which was associated with a significant decline in RBC count, increased HbA1c, MCH, and RDW compared to non-prediabetic (NPD) rats, elucidating to exacerbated eryptosis and anaemia. Prediabetic rat spleens showed a proinflammatory status with elevated IFN- $\gamma$  and IL- $1\beta$ , associated with poor spleen vascular homeostasis as observed by reduced PLGF and VEGFR which may decline erythrocyte filtration and spleen function. Prediabetes showed high albuminuria and MAP during gestation, suggesting renal malfunction, thus increasing the risk of preeclampsia. Moreover, prediabetes showed heavier spleens compared to NPD and possible splenomegaly which worsens erythrocyte dysfunction that eventually increases the risk of cardiovascular injury in the prediabetic state during pregnancy.

### Conclusion

Prediabetes may have promoted inflammation and eryptosis, thus, declining erythrocyte and spleen function. Current observations, therefore, warrant the need to screen and manage prediabetes during pregnancy to improve maternal and foetal outcome.



## POSTER PRESENTATIONS

**Philanathi Mabena**

**SANS26-P75**

**Nelson Mandela University**

### Background/Aim:

Diabetic retinopathy (DR) is a progressive complication of diabetes mellitus leading to retinal degeneration and vision loss. Fortunately, cocoa mitigates retinal damage owing to its antioxidant properties. However, its impact on diabetes-induced retinal damage when administered in high, repeated doses remains unclear. We, therefore, hypothesized that repeated high-dose cocoa administration will reverse type 2 diabetes mellitus-induced degeneration of the retinal integrity in adult male Sprague Dawley rats.

### Methods:

A 6-week study was conducted using sixteen (n=16) adult male Sprague Dawley rats (10-11 weeks old, 200-300 g), divided equally into four groups: control, diabetes mellitus (DM), cocoa-treated, and DM+Cocoa. Following study termination, retinal tissues were stained with haematoxylin and eosin for histological evaluation. Data were analysed using the Shapiro-Wilk test for normality, followed by one-way ANOVA and Bonferroni post hoc test, or Kruskal-Wallis and Dunn's post hoc test for non-parametric data.

### Results:

Cocoa-treated groups showed a significant increase in total retinal thickness and in the thickness of most retinal layers (NFL, INL, OPL, ONL, PL, RPEL, and CCL), with the exception of the GCL and IPL. Retinal neuronal cell counts (GCs, INL cells, and PCs) increased in the cocoa group. SVP endothelial cells decreased, while RPE cell counts increased in the DM and DM+Cocoa groups. Additionally, choroidal endothelial cells were reduced in the cocoa-treated rats compared to other groups.

### Conclusion:

Repeated administration of high-dose cocoa enhances total and individual retinal layer thickness in rats, confirming a protective role against diabetes-induced retinal damage and blood-retinal barrier breakdown in both diabetic and non-diabetic conditions.



## POSTER PRESENTATIONS

**Caitlyn Mahoney**

**University of Cape Town**

**SANS26-P02**

Mitochondrial mechanisms are increasingly implicated in complex neurological conditions, including Autism Spectrum Disorder (ASD). Preclinical models of ASD have employed Propionic Acid (PPA) to study the relationship between neuronal metabolism, development and function. PPA has been shown to alter mitochondrial biogenesis, dynamics and turnover in neuronal-like SH-SY5Y cells. However, the relationship between metabolic remodelling and neuronal differentiation remains unexplored in this context. This study examined how PPA-induced mitochondrial dysfunction influenced neuronal metabolism and differentiation in the SH-SY5Y system. PPA significantly perturbed molecular regulators of mitochondrial biogenesis and dynamics in a time- and dose-dependent manner, culminating in disruptions to mitochondrial content, connectivity and turnover. End-point changes to metabolic state were marked by deficits in both oxidative and glycolytic energy production, driven by a shift in TCA cycle flux associated with PPA catabolism. Concurrently, PPA substantially altered gross neuronal morphology during in vitro neurogenesis, decreasing the length and complexity of neurite processes and changing the localisation and expression of cytoskeletal markers. This was accompanied by a remodelling of mitochondrial networks and global changes to the neurogenic transcriptome, marked by signatures associated with developmental delay, neuronal injury and inflammation. Collectively, this data reveals PPA-induced disruptions to mitochondrial homeostasis with functional implications for neuronal metabolism, morphology and maturation. This work expands current characterisations of the PPA model, providing novel insights into the mechanisms and manifestations of PPA-induced neurotoxicity to inform future research on the mitochondrial component of neurological disease.

# POSTER PRESENTATIONS

**Mosa Mafalo**

**SANS26-P28**

**University of the Witwatersrand**

## Aim

Limited access to neuropsychological assessment in low- and middle-income countries constrains early detection and intervention, especially in peri-rural African settings. Digital neurocognitive screening offers potential for scalable access, but its feasibility, validity, and interpretability in such contexts remain underexplored.

## Methods

This study examined NeuroScreen, a tablet-based battery of 11 subtests across six cognitive domains, within the Asenze cohort in peri-rural KwaZulu-Natal. Neurocognitive performance of 978 adult caregivers, mean age 43.8 years, was assessed. Feasibility was examined via completion rates and qualitative data from 135 non-completers; construct validity through associations with age, education, socioeconomic status, and HIV status.

## Results

NeuroScreen achieved 84.9% completion. Literacy-related barriers were the primary cause of non-completion, disproportionately excluding older, lower-education, lower-SES, and people living with HIV individuals. Age was the strongest predictor across domains ( $\eta^2$  up to 0.435). Education and SES showed significant associations with processing speed and executive function. Participants living with HIV displayed selective slowing in processing speed and attention, with comparable performance elsewhere.

## Conclusion

These findings show that non-specialist fieldworkers can reliably administer digital neurocognitive screening at scale, providing infrastructure for population-level surveillance and continuous norming. Critically, observed accuracy-speed dissociations challenge the assumption that speed equates to cognitive competence, suggesting that current Western paradigms may systematically mischaracterise performance in low-resource, culturally distinct settings.



## POSTER PRESENTATIONS

**Khayelihle Brian Makhathini**

**SANS26-P46**

**University of the Western Cape**

### Background and Aim

The Human Immunodeficiency Virus (HIV) has adverse outcomes on the central nervous system (CNS). The CNS vasculature consists of the blood-brain barrier (BBB), which is primarily comprised of microvascular endothelial cells that protect against blood-borne pathogens and harmful substances. However, since alcohol abuse is prevalent in the HIV-infected community, the additive effects of alcohol and HIV may compromise the BBB integrity. This study investigates the combined effects of HL2/3 cell culture-derived HIV paracrine factors and alcohol on brain endothelial cells (BECs) using cell culture and confluent monolayers to mimic an in vitro BBB model.

### Methods

HL2/3 cells are genetically modified HeLa cells that secrete HIV proteins into the culture media. The HL2/3 cell culture media was harvested and employed to mimic brain endothelial cells (bEnd5), which were exposed to HIV proteins, forming the basis of the in vitro HIV-BBB model. The harvested cell culture supernatant was further analysed for the presence of HIV proteins. Additionally, the in vitro HIV-BBB model was treated with alcohol, where cell proliferation and BEC monolayer permeability were observed.

### Results

HIV proteins were detected in HL2/3 cell-conditioned media (CM), making it a viable in vitro HIV model. The HL2/3 cell CM and alcohol decreased cell proliferation, indicating suppression. HL2/3 cell CM increased permeability in a dose-dependent manner, whereas combined treatment with alcohol exacerbated the effects of HL2/3 cell CM.

### Conclusion

HIV-proteins and alcohol suppressed cell proliferation with no combinatory effects. The combined treatment exacerbated the effects on BEC monolayer permeability, indicative of the amplified danger of HIV-positive persons consuming alcohol on the homeostatic regulation of the BBB.



## POSTER PRESENTATIONS

**Hayley Marais**

**SANS26-P62**

**University of Cape Town**

### Aim

Epilepsy affects over 50 million people worldwide, yet the cellular mechanisms underlying epileptogenesis remain poorly understood. The ontogenetic recapitulation hypothesis proposes that epileptic neurons undergo transcriptional reversion to an immature state, shifting the NKCC1/KCC2 balance to produce excitatory rather than inhibitory GABAergic signalling. This study aims to build a single-nucleus RNAseq reference atlas for healthy human cortical ageing from neurosurgical tissue spanning infancy to adulthood, and compare epileptic tissue transcriptomic profiles to identify pathological deviations indicative of immature reversion.

### Methods

Human brain tissue is obtained from local hospitals under ethical approval. Both healthy and epileptic nuclei are profiled using PIPseq, a multiplexed, particle-templated snRNAseq platform. Pseudotime trajectory analysis is used to resolve developmental and pathological cell-state transitions. Expression of immature neuronal and astroglial markers and NKCC1/KCC2 ratios are specifically assessed. Additionally, human developmental trajectories are compared to existing mouse organotypic slice snRNAseq data to evaluate cross-species translational validity.

### Results

Preliminary PIPseq profiling of human temporal cortex tissue confirms successful detection of all major brain cell types with consistent expression of established marker genes, validating our pipeline. Pseudotime analyses from existing mouse snRNAseq data and HCR-RNA-FISH applications have additionally identified distinct developmental trajectories across multiple cell types between acute and organotypic (cultured) slices, therefore allowing for cross-species analysis.

### Conclusion

This work will establish a lifespan snRNAseq reference for the human temporal cortex and provide single-cell resolution transcriptomic evidence testing the ontogenetic recapitulation hypothesis in epilepsy, with broad implications for understanding neurodegeneration disease mechanisms.



# POSTER PRESENTATIONS

**Seke Nzau Mafuika**

**University of KwaZulu-Natal**

**SANS26-P16**

Diagnosis and detection of low levels of serum GFAP and S100B biomarkers in traumatic brain injury (TBI) using highly sensitive blood and fluid assays, such as fluorescence-based techniques, enzyme-linked immunosorbent assays (ELISA), and electrochemiluminescence (ECL), remain major challenges despite improved predictive accuracy.

This prospective study utilizes a multiplex immunoassay (ProcartaPlex, Thermo Fisher Scientific, 2024) to detect human GFAP and S100B proteins in serum from 75 patients with moderate to severe TBI. Statistical analysis was achieved using Statistical Package for the Social Sciences version 30.0.0.0 (SPSS; IBM Corporation). Kruskal-Wallis, Dunn's multiple comparisons, and Pearson's chi-squared test were used to assess the association between variables. The results obtained were expressed as median and interquartile range at  $P < 0.05$ .

Serum concentrations of GFAP and S100B were significantly elevated in the moderate and severe TBI groups compared with the control group ( $P < 0.001$ ). No significant differences between the moderate and severe groups were detected for GFAP or S100B ( $P=0.2623$ ,  $P>0.999$ ).

These findings suggest that elevated serum concentrations of GFAP and S100B are indicative of increasing severity and progression of TBI, supporting the utility of multiplex immunoassay as a predictive tool. These findings suggest that the combined use of GFAP and S100B biomarkers enhances the accuracy of distinguishing between moderate and severe TBI as predictor test indicators.



## POSTER PRESENTATIONS

**Siphesihle Mkhize**

**SANS26-P42**

**University of KwaZulu-Natal**

### Background

Hyperglycaemia is one of the leading risk factors for the development of cerebral atherosclerosis in diabetic individuals. Cerebral atherosclerosis is a chronic inflammatory disorder that is the underlying cause of ischemic stroke. Due to the nature of the disorder and the associated diagnostic procedures, there is limited data on the progression of diabetes-associated changes in the brain.

### Aim

To investigate whether dioxidovanadium can alleviate hyperglycaemia induced cerebral atherosclerosis in STZ-induced diabetic rats.

### Methods

Upon confirmation of diabetes, dioxidovanadium was administered twice every third day (40mg/kg), insulin was administered twice every single day (0,175 mg/kg-1 s c), and blood glucose concentration as well as blood pressure measurements were observed weekly for the experimental period of 5 weeks. Locomotor abilities were assessed on the 5th week of the experimental period using the Morris Water Maze test. Rats were then sacrificed after which the blood, and cerebrum were harvested for biochemical assessment of epigenetic markers of atherosclerosis, from which TEM analysis of structural changes in pericytes, and lumen of cerebral blood vessels and other biochemical tests were performed.

### Results

The administration of the metal-based drug (dioxidovanadium) significantly decreased blood glucose concentration throughout the experimental period compared to the diabetic control group. The dioxidovanadium compound treated group presented with significantly reduced MDA and inflammatory markers compared to the untreated diabetic control group. The MWM test results, which measured locomotor abilities for the vanadium compound treated group were significantly reduced compared to the diabetic group. In addition, the dioxidovanadium compound reduced the gene expression of markers that are responsible for plaque formation and instability, thereby decreasing the likelihood of ischemic stroke.

# POSTER PRESENTATIONS

**Manish Mohapatra**  
**Christ University**

**SANS26-P69**

This research proposal outlines a three-month behavioural experimental study examining the influence of social proof direction (positive, negative, and neutral) on sustainable consumer decision-making. Grounded in established frameworks of social impact and identity-based motivation, the study seeks to understand how normative information shapes consumer behaviour in sustainability-related choices. A controlled within-subjects experimental design will be implemented using PsychoPy, allowing precise manipulation of social proof cues and accurate measurement of behavioural responses. Participants (N = 60) will complete a series of product choice trials in which they select between sustainable and conventional consumer products, each accompanied by experimentally manipulated social proof messages indicating high, low, or neutral popularity. The primary dependent variables include product choice, purchase intention, identity alignment, and reaction time, enabling a multidimensional assessment of both decision outcomes and decision processes. By examining not only what consumers choose but also how quickly and how self-congruently they make these choices, the study aims to shed light on the psychological mechanisms underlying the well-documented attitude-behaviour gap in sustainable consumption. The findings are expected to advance behavioural research on sustainability by clarifying the role of social norms and identity alignment in shaping consumer decisions, while also offering practically relevant insights for designing more effective normative and identity-based messaging in sustainability campaigns and consumer interventions.



## POSTER PRESENTATIONS

**Mahlatse Prudence Mokgotho**

**SANS26-P49**

**University of Cape Town**

### Background

Traumatic brain injury (TBI) is a major contributor to mortality following road traffic collisions (RTCs), yet its burden in South Africa remains poorly documented, and the impact of the COVID-19 lockdown measures remains unclear. This study investigated RTC-related deaths with associated TBI before and during the lockdown.

### Methods

A retrospective review of autopsy reports for RTC-related deaths was conducted at Salt River Mortuary/Observatory Forensic Pathology Institute (SRM/OFPI) between 1 January 2019 and 31 December 2020. TBI cases were compared between COVID-19 pre-lockdown and lockdown periods with an emphasis on demographic and injury characteristics.

### Results

RTC-related deaths accounted for 8.0% (618/7700) of all autopsies conducted during the period, with 81% of fatal RTCs presenting with TBI. Males had a three times higher likelihood of TBI compared to females. Decedents aged 30–39 years were at greatest risk, and those with TBI were significantly younger than those without (median age 34 vs. 39 years,  $p < 0.001$ ). Although alcohol was frequently detected, no significant association was observed between positive alcohol results and TBI presence. Subarachnoid haemorrhage was the most frequently observed TBI subtype (68%), and the majority of cases were classified as severe (96%) TBI. No significant differences in TBI patterns were observed before and during the COVID-19 lockdown period.

### Conclusion

This study highlights the substantial contribution of TBI to RTC-related mortality in our setting, particularly among young males, underscoring the need for targeted prevention strategies for road traffic safety and urban mobility infrastructure.



# POSTER PRESENTATIONS

**Mluleki Luvuno**

**University of KwaZulu-Natal**

**SANS26-P55**

## Introduction

The advancement of technology and civilisation coincides with reduced social isolation and disturbed sleep patterns. This can impact the optimum day-to-day operations. The combined effects of social isolation and sleep disturbance remain unclear. Hence, we investigated the effect of social isolation and sleep disturbance on anxiety-like behaviours in animals.

## Methods

To achieve this, animals were randomly assigned to the following groups (n=7/group) and acclimatised for 7 days: control, group-housed sleep-disturbed (GH-SD), individually housed non-sleep-disturbed (IH-NSD), and individually housed sleep-deprived (IH-SD). On day 18, the animals were subjected to the plus-maze discriminative avoidance task (PM-DAT) to assess anxiety-like behaviour. Thereafter, animals were sacrificed to collect blood and amygdala to measure ACTH, corticosterone and arginine vasopressin.  $P < 0.05$  was considered significant.

## Results

The combination of isolation and sleep disturbance worsened anxiety, as evidenced by the least time spent in the open arms and the most time spent in the closed arms of the PM-DAT. This was also associated with reduced exploration, evidenced by the lowest open arms ratio. This was accompanied by heightened HPA activity due to higher ACTH and vasopressin observed in these animals.

## Discussion

Altogether, the combination of isolation and sleep disturbance exacerbated anxiety and hampered exploratory activity, possibly through activation of the HPA-axis and effects on the systems associated with motivation and exploration. The lifestyle of isolation and altered sleep patterns may harm the day-to-day operations and productivity of humans, particularly the youth.



## POSTER PRESENTATIONS

**Bongekile Mngoma**

**University of KwaZulu-Natal**

**SANS26-P57**

### Introduction:

Adolescent often prioritises phones and social media over sleep, leading to social isolation (SI) and sleep deprivation (SD), and heightened anxiety as virtual interaction replace in person interaction. These factors are well studied individually, their combined effect on adolescent anxiety are underexplored. This study examines effects of SI and SD using behavioural measurements (%time spent in open vs closed arms, open arm ratio in PM-DAT).

### Methods:

The animals were assigned to the following groups (n=7/group) and acclimatised for 7 days: control, group-housed sleep-disturbed (Gh-SD), individual housed non-sleep-disturbed (IH-NSD) and individual housed sleep-disturbed (IH-SD). on day 18, animals were subjected to plus-maze discriminative avoidance tasks (PM-DAT) to assess anxiety like behaviour. Thereafter, the animals were sacrifices to collect blood and amygdala to measure ACTH, corticosterone and Arginine vasopressin.  $P < 0,05$  was considered significant.

### Results:

The combination of isolation and sleep disturbance worsened anxiety, as evidenced by the least time spent in open arms and the most time spent in closed arm. This was also associated with reduced exploration, evidenced by lowest open arms ratio. This is accompanied by heightened HPA activity due to higher ACTH and vasopressin observed in animals.

### Discussion:

Altogether, the combination of isolation and sleep disturbance exacerbated anxiety and hampered exploratory activity, possibly through activation of the HPA axis and effects on the systems associated with motivation and exploration. The lifestyle of isolation and altered sleep patterns may harm day to day operation and productivity of humans, particularly the youth.

## POSTER PRESENTATIONS

**Nombulelo Agata Mthembu**  
**University of KwaZulu-Natal**

**SANS26-P08**

Cognitive impairment is a recognized complication of prediabetes, associated with inflammation, oxidative stress, and impaired synaptic plasticity in the hippocampus. Biguanides such as metformin are widely used in its management; however, their efficacy is often limited by poor adherence to lifestyle modifications. This underscores the need for alternative therapeutic strategies that remain effective independent of lifestyle changes.

Vanadium compounds have demonstrated potential in improving cognitive dysfunction in streptozotocin induced diabetes model diabetic models, but their effects in diet-induced prediabetes remain unclear. This study investigated the effects of a diamagnetic vanadium 2-phenolate-1H-benzimidazole complex on cognition and learning in a high-fat high-carbohydrate (HFHC) diet-induced prediabetic rat model.

Following confirmation of prediabetes, animals were treated with vanadium complex (40 mg/kg, p.o.) or metformin (200 mg/kg, p.o.) once every third day for five weeks. Blood glucose and body weight were monitored weekly. In the final week, behavioural assessments (novel object recognition and Morris water maze) were conducted. Post-sacrifice, hippocampal tissue was analysed for malondialdehyde levels and gene expression, while the cerebral cortex was assessed for structural integrity.

The vanadium complex significantly reduced blood glucose and body weight, improved learning and spatial memory, and attenuated oxidative stress, neuroinflammation, and synaptic dysfunction. Although cerebral damage was not fully reversed, further degeneration was mitigated, with evidence of autophagic activity.

These findings highlight the therapeutic potential of benzimidazole-chelated vanadium complexes in improving cognitive and metabolic outcomes in prediabetes.



## POSTER PRESENTATIONS

**Vhuthuhawe Mugwena**  
**North-West University**

**SANS26-P32**

### Background:

HIV-associated neurocognitive disorders (HAND) persist despite effective antiretroviral therapy and are largely driven by the neurotoxic effects of the viral protein Trans-Activator of Transcription (Tat). Subtype-specific differences exist, with Tat B (subtype B) generally considered more neurotoxic than Tat C (subtype C). Cannabidiol (CBD) has demonstrated neuroprotective properties; however, the role of CBD and its metabolites in mediating subtype-specific Tat-induced neuropathogenesis remains poorly understood.

### Aim

To evaluate the differential neuroprotective effects of CBD and its metabolites against HIV-1 Tat B- and C-induced neuronal cell damage.

### Methods

Primary human neurons were exposed to Tat subtypes B and C (50ng/mL) in the presence or absence of CBD (3  $\mu$ M and 5  $\mu$ M) and its metabolites (3  $\mu$ M), 6-hydroxy-cannabidiol (6-OH-CBD) and 7-hydroxy-cannabidiol (7-OH-CBD) for 24 hours. Cell viability was assessed using the 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) assay. Statistical analysis was conducted using one-way ANOVA and unpaired t-tests.

### Results

Tat B (61%) and Tat C (65%) both significantly decreased neuronal viability compared to controls ( $p < 0.01$ ), with no significant difference between subtypes. CBD significantly attenuated this decrease, with 3  $\mu$ M offering greater neuroprotection than 5  $\mu$ M ( $p < 0.05$ ). Among the metabolites, 6-OH-CBD was most effective, restoring viability to -82% (Tat B) and -87% (Tat C), and was significantly more protective than 7-OH-CBD ( $p < 0.001$ ).

### Conclusion

CBD demonstrated a dose-dependent trend toward neuroprotection against HIV-1 Tat-induced toxicity, with lower concentrations showing greater efficacy. Notably, 6-OH-CBD exhibited enhanced neuroprotective effects compared to CBD and 7-OH-CBD, suggesting a potential role for CBD metabolism in modulating neuronal survival.

## POSTER PRESENTATIONS

**Mdlalose Noxolo Nokulunga**

**University of KwaZulu-Natal**

**SANS26-P50**

### Background/Aim

Neuroinflammation and oxidative stress are key drivers of neuronal dysfunction and cognitive decline, often arising from systemic immune activation and extending to the brain via the gut-brain axis. Short-chain fatty acids (SCFAs), mainly acetate, propionate, and butyrate, are gut-derived metabolites that regulate immune and redox pathways and support brain function. Reduced SCFA availability is linked to heightened inflammation and oxidative stress; however, their ability to counteract acute inflammation-induced neurobiological dysfunction remains unclear. This study aimed to determine whether SCFAs attenuate neuroinflammation, oxidative stress, and cognitive impairment following LPS-induced acute inflammation.

### Methods

Animals were randomly assigned to four groups (n=6/group): control, SCFAs-only, LPS-only, and LPS+SCFAs. A single dose of LPS (10 mg/kg) was administered, while SCFAs were orally given for two weeks. Cognitive function was assessed using the novel object recognition test (NORT). Plasma and hippocampal tissues were collected to quantify pro-inflammatory cytokines, oxidative stress markers and glutamate levels. Data were analysed using ANOVA ( $p < 0.05$ ).

### Results

LPS impaired cognitive performance, while co-administration with SCFAs significantly improved discrimination index compared to LPS-only. Although plasma TNF- $\alpha$  and IL-6 remained elevated, hippocampal cytokine levels were restored following SCFA treatment. SCFAs also attenuated oxidative stress, evidenced by reduced hippocampal 4-HNE. Furthermore, glutamate levels were markedly increased in the LPS+SCFAs group, suggesting enhanced neurotransmission.

### Conclusion

Altogether, SCFAs improved discrimination index, restored inflammation, prevented oxidative stress, and boosted glutamate production following acute inflammation. These findings highlight the therapeutic potential of targeting the gut-brain axis in neuroinflammatory conditions.



# POSTER PRESENTATIONS

**Ampuriire Nyakubaho**

**Ernest Cook University**

**SANS26-P09**

## Background/Aim

Pituitary carcinomas constitute 0.1-0.2% of pituitary neoplasms, defined by metastatic spread and typically diagnosed in older adults. Presentation in adolescence is exceptionally rare, often delaying suspicion of malignancy. Cerebrospinal fluid (CSF) seeding before histopathological confirmation represents an advanced, poor-prognosis state. This report highlights critical neuroimaging features that should prompt early suspicion of pituitary carcinoma in young patients.

## Methods

A 15-year-old female with hyperprolactinemia and progressive visual blurring underwent contrast-enhanced brain magnetic resonance imaging (MRI) on a 1.5 Tesla system. Sequences included T1-weighted pre/post-gadolinium, T2-weighted, and fluid-attenuated inversion recovery (FLAIR). Imaging findings were correlated with clinical presentation and literature on aggressive pituitary tumors.

## Results

MRI demonstrated a large hour-glass-shaped sellar mass with suprasellar extension and bilateral internal carotid artery encasement. Multiple enhancing extra-axial nodular deposits were identified within the ambient cisterns, partially contiguous with the primary mass, consistent with CSF dissemination. The lesion exhibited poor contrast enhancement and no restricted diffusion.

## Conclusion

Hour-glass morphology, arterial encasement, and enhancing leptomeningeal nodules constitute critical neuroimaging red flags for pituitary carcinoma, even in adolescents. Recognition of these aggressive features on MRI facilitates expedited histopathological diagnosis and multidisciplinary management, including temozolomide-based therapy. This case underscores the imperative to consider malignant etiology when CSF seeding is radiologically evident, regardless of patient age.



## POSTER PRESENTATIONS

**Thuveshani Naick**

**Stellenbosch University**

**SANS26-P27**

Consumer sleep technologies are increasingly used for home-based sleep monitoring, yet many devices lack independent validation in clinical populations with suspected obstructive sleep apnea (OSA). Polysomnography (PSG) remains the gold standard for sleep assessment, but its high cost and limited accessibility restrict widespread use, particularly in resource-constrained settings such as South Africa. The Sleepon Go2Sleep 3 ring is a finger-worn wearable device that provides automated sleep staging and oxygen saturation metrics; however, its performance against PSG has not yet been independently validated.

This observational validation study aims to evaluate the performance of the Go2Sleep 3 ring against overnight PSG in adults referred for diagnostic assessment of suspected OSA. Participants undergoing routine PSG at the Durbanville Sleep Lab will simultaneously wear the Go2Sleep 3 ring. Sleep stage classification (Wake, N1, N2, N3, REM) and respiratory metrics, including blood oxygen saturation ( $\text{SpO}_2$ ) and oxygen desaturation index (ODI), will be compared between the two systems.

Data analysis will follow the standardized validation framework proposed by Menghini et al., incorporating epoch-by-epoch agreement analysis, confusion matrices, sensitivity and specificity measures, Cohen's kappa statistics, Bland-Altman plots, and regression-based bias analysis. A minimum of 30 complete datasets will be collected. Data collection is currently underway, with completion anticipated prior to conference presentation.

This study will provide independent evidence regarding the accuracy and clinical utility of the Go2Sleep 3 ring in a real-world OSA population and may support the development of accessible sleep monitoring tools in low-resource settings



# POSTER PRESENTATIONS

**Nadia Olivier**

**North-West University**

**SANS26-P48**

## Background/Aim

Major depressive disorder is a multifactorial condition involving neurochemical changes and inflammation. The Flinders Sensitive Line (FSL) rat displays robust depressive-like behaviours, including despair, and has been reported to display elevated peripheral inflammatory markers compared to the Flinders Resistant Line (FRL) control. This study aimed to characterise the relationship between butyrylcholinesterase (BChE) activity, inflammation, and behaviour in FSL rats, and to evaluate responses to escitalopram and a novel selective BChE inhibitor (BChEI).

## Methods

Male FSL rats received vehicle, escitalopram (20mg/kg), or a selective BChEI (10 or 30mg/kg) via oral gavage. Behavioural despair was assessed using the forced swim test (FST). Serum interleukin-6 (IL-6), IL-10, and C-reactive protein (CRP) levels, as well as BChE activity, were quantified. Statistical analyses included group comparisons and correlation analyses.

## Results

Compared to FRL controls, FSL control rats exhibited increased despair-like behaviour in the FST ( $p<0.0001$ ), accompanied by elevated BChE activity ( $p<0.0001$ ), and increased IL-6 ( $p=0.03$ ) and CRP levels ( $p<0.0001$ ), with no change in IL-10 levels. Both escitalopram and BChEI reversed behavioural despair. Escitalopram reduced IL-6 ( $p=0.004$ ) and CRP ( $p<0.0001$ ) without affecting BChE activity. BChEI 30mg/kg reduced BChE activity ( $p=0.001$ ) without altering serum IL-6 or CRP levels. IL-6 levels were positively associated with despair-like behaviour in FSL rats ( $p=0.004$ ).

## Conclusion

These findings support the validity of the FSL model as a multifactorial model of depression, integrating behavioural, inflammatory, and cholinergic alterations. The dissociation between anti-inflammatory and cholinergic effects, despite convergent behavioural improvement, indicates that antidepressant responses may arise through multiple, distinct biological pathways beyond monoaminergic and inflammatory mechanisms.

# POSTER PRESENTATIONS

**Olawale Tunji Osborne Oderinde**  
**Hult International Business School**

**SANS26-P61**

## Background/Aim

Values-based leadership shapes organisational culture, behaviour, and performance. Behavioural neuroscience has advanced our understanding of the neural substrates of values, identity, and ethical decision-making; however, these literatures rarely intersect. However, the translation of transformational leadership theory into small and medium enterprises (SME), particularly in Sub-Saharan Africa, remains underdeveloped. This review synthesises neuroscience evidence on values-based leadership and identifies mechanisms and translational opportunities for SMEs.

## Methods

A structured narrative review was performed. PubMed, PsycINFO, Scopus, and Web of Science were searched (2010-2025) for peer-reviewed studies on the neural correlates of values processing, identity, or ethical decision-making relevant to leadership. Thematic synthesis was performed.

## Results

Three domains emerged: (i) neural substrates of values-based decision-making, centred on the ventromedial prefrontal cortex, anterior cingulate cortex, and default mode network; (ii) social-cognitive mechanisms of values transmission via observational learning and theory-of-mind systems; and (iii) neurobiological correlates of behavioural integrity, with values-action alignment linked to reduced cognitive conflict signalling. Evidence from Africa and SME is sparse.

## Conclusion

Behavioural neuroscience offers a credible foundation for values-based leadership, but its applied translation in SMEs, especially in Sub-Saharan Africa, is nascent. A research-practice agenda is proposed for neuroscience-informed values-alignment diagnostics in SME leadership.



# POSTER PRESENTATIONS

**Beatrice Atieno Oloo**

**University of Cape Town**

**SANS26-P04**

## Background/Aim

Reading acquisition relies on coordinated activity across brain regions responsible for language, visual processing, and phonological decoding. Advances in cognitive neuroscience have improved understanding of the neural systems that support reading development. Dyslexia, a common neurodevelopmental disorder, is associated with differences in brain circuits involved in phonological processing and word recognition. These differences can affect reading fluency and comprehension if not identified early. This narrative review examines current evidence on the neural circuits underlying reading acquisition and dyslexia and explores implications for early identification and educational intervention, particularly in contexts where specialised learning assessments remain limited.

## Methods

A narrative review of peer-reviewed literature was conducted using databases including PubMed, Scopus, and Google Scholar. Studies examining neuroimaging findings, cognitive models of reading, and neural differences between typical readers and individuals with dyslexia were reviewed.

## Results

Evidence from neuroimaging research indicates that reading relies primarily on a left-lateralised network involving the temporo-parietal cortex, inferior frontal gyrus, and occipito-temporal regions. Individuals with dyslexia often show reduced activation in these areas during reading tasks. Structural imaging studies also highlight differences in white matter pathways such as the arcuate fasciculus.

## Conclusion

Understanding the neural circuitry underlying reading development may support earlier identification of dyslexia and inform more effective educational interventions.



# POSTER PRESENTATIONS

**Tejini Padalkar**

**University of Cape Town**

**SANS26-P11**

## Background

Brain age, derived from machine learning models, is an emerging biomarker of brain health. However, determinants of brain age such as biological, social, and psychological factors, remain underexplored in low- and middle-income countries (LMICs). This study examined risk and protective predictors of brain age in a South African birth cohort.

## Methods

Women (n = 202, mean age = 29.46 years) from the Drakenstein Child Health Study underwent structural MRI. Brain age was estimated using the Pymnet model, and brain age gap (BAG; predicted minus chronological age) was calculated. Clinical and psychosocial data were collected and associations with BAG were examined using simple and hierarchical linear regression models.

## Results

In unadjusted models, alcohol and cannabis use was associated with greater BAG ( $\beta = 2.29$ , 95% CI 0.13, 4.45,  $p = .04$ ), while higher household income was associated with lower BAG ( $\beta = -3.28$ , 95% CI -6.17, -0.39,  $p = .03$ ). Social support showed a trend towards significance ( $p = .06$ ). In adjusted models, older age ( $\beta = -0.24$ , 95% CI: -0.36, -0.11,  $p < .001$ ) and higher household income ( $\beta = -2.84$ , 95% CI: -5.67, 0.04,  $p = .05$ ) remained associated with lower BAG, while alcohol and cannabis use remained associated with higher BAG ( $\beta = 2.37$ , 95% CI: 0.02, 4.73,  $p = .05$ ). Social support retained a protective but non-significant effect.

## Conclusions

Modifiable risk and protective factors are associated with BAG in this LMIC cohort. These findings support the relevance of context-specific interventions to promote brain health in LMICs.

## POSTER PRESENTATIONS

**Taelo Ruth Phukoby**

**University of Johannesburg**

**SANS26-P60**

### Background

Brain health problems has become exceedingly prevalent. The cause is thought to be due to oxidative stress which leads to brain function impairment and cognitive decline. Combretum erythrophyllum been reported to have substantial quantities of phenolic compounds with high antioxidant activity and lacks evidence-based safety data for chronic brain applications. This study aimed to define its neuroprotective and neurotoxic threshold using zebrafish.

### Methods

Acute toxicity was conducted as per OECD guidelines. Eight concentrations of methanolic leaf extract of Combretum erythrophyllum in a geometric series. The median lethal concentration (LC50) was determined using probit analysis. For the subacute toxicity, adult zebrafish were exposed to Combretum erythrophyllum methanolic leaf extract at 2.5 -80mg/ml respectively every 24 hours orally for 28 days. The level of superoxide dismutase (SOD), catalase (CAT), malondialdehyde (MDA), and glutathione dismutase (GSH) were determined. Neural morphology was observed.

### Results

The results demonstrated a concentration-dependent increase in mortality, with an LC 50 value of 90.47, indicating moderate toxicity. Combretum erythrophyllum methanolic leaf extract induced a significant, dose-dependent increase in catalase response, peaking at 40 µmol followed by inhibition at 80 µmol, suggesting a threshold for oxidative stress response. MDA levels remained stable, indicating no lipid peroxidation. GSH trended upward to 40-80 µmol. SOD was unchanged, reflecting selective antioxidant modulation.

In conclusion, Combretum erythrophyllum exhibits a hormetic neuroprotective profile. These data provide the first evidence-based safety window for traditional use, advancing brain health from molecular mechanisms to community applications.

## POSTER PRESENTATIONS

**Taelo Ruth Phukoby**

**University of Johannesburg**

**SANS26-P73**

*Combretum erythrophyllum* is widely used as traditional medicinal plant in curing several diseases such as vaginal diseases and cough. However, their potential neurobehavioural effects remain insufficiently explored. This study was carried out to evaluate the neurobehavioural effects of *Combretum erythrophyllum* methanolic leaf extract in adult zebrafish (*Danio rerio*) using locomotor behavioural analysis.

### Material and methods:

A total of 42 zebra fish were divided into seven groups of six zebrafish. Group I (control) was immersed with salt treated water, Group II to VII were immersed with a single dose of methanolic extract of *Combretum erythrophyllum* (2.5- 80mg/ml of extracts respectively every 24 hours orally for 28 days. For every day the concentrations were replaced throughout the subacute toxicity experiment. Behavioural activity was monitored using EthoVision XT tracking software, assessing parameters including distance travelled, velocity, acceleration, mobility, and rotational movement.

### Results:

Exposure to lower concentrations (2.5 and 5) resulted in increased swimming activity and velocity relative to the control group, suggesting possible anxiogenic or stimulatory effects. In contrast, exposure to intermediate and higher concentrations (10 and 20) produced reduced locomotor activity, decreased mobility, and impaired swimming performance, indicative of sedative or neurotoxic effects. The behavioural responses demonstrated a concentration-dependent pattern characterized by initial hyperactivity followed by locomotor suppression at higher concentrations.

### Conclusion:

These findings suggest that *Combretum erythrophyllum* extract may influence neurological function in zebrafish and may exhibit biphasic neurobehavioural activity depending on concentration. The study further highlights the utility of zebrafish behavioural assays as sensitive models for evaluating the neurotoxicological effects of medicinal plants and natural compounds.

# POSTER PRESENTATIONS

**Tavia Quarmby**

**University of Cape Town**

**SANS26-P12**

## Background

Ibogaine has emerged as a candidate therapeutic for white matter pathology, modulating oligodendrocyte function and myelin protein expression, but the molecular mechanisms remain unclear. One key regulator of oligodendrocyte differentiation is oligodendrocyte transcription factor 1 (Olig1), which governs the transcription of myelin genes such as myelin basic protein. While chronic morphine exposure is known to induce demyelination, whether ibogaine can restore Olig1 expression has not been previously investigated.

## Methods

Fifty Sprague-Dawley rats were divided into control, ibogaine-only, morphine-only, morphine-ibogaine (24 h), and morphine-ibogaine (72 h) groups. Animals received escalating morphine doses for 10 days, acute intraperitoneal ibogaine (50 mg/kg), or both. Prefrontal cortex and ventral pons tissue were collected, RNA extracted, and cDNA synthesized for quantitative PCR analysis of Olig1 mRNA expression.

## Results

Morphine and ibogaine treatment significantly downregulated Olig1 mRNA in the prefrontal cortex compared to the control. However, combination morphine and ibogaine treatment demonstrated Olig1 expression similar to the control group, suggesting some recovery. Conversely, there were no statistically significant differences between any treatment groups in the ventral pons, indicating region-specific vulnerability.

## Conclusion

These findings reveal distinct regional responses to morphine and ibogaine in white-matter transcriptional regulation. The prefrontal cortex, characterized by higher oligodendrocyte turnover, exhibited dynamic Olig1 modulation, whereas the ventral pons remained stable. Notably, ibogaine treatment coincided with partial recovery of Olig1 after morphine exposure. Further time-controlled studies are necessary to determine whether this reflects true pharmacological restoration or spontaneous homeostatic repair.



## POSTER PRESENTATIONS

**Ana Paula Rodrigues de Paulo**

**SANS26-P24**

**Institute of Physiology of Czech Academy of Science**

The cholinergic system critically regulates neuronal excitability and network dynamics, supporting attention, learning, and behavioral flexibility. Nicotinic acetylcholine receptors (nAChRs) are key mediators of fast cholinergic signaling, and their dysfunction has been implicated in autism spectrum disorder (ASD), which is associated with social deficits and excitatory/inhibitory (E/I) imbalance.

This study investigates the expression and functional role of nAChRs in the prefrontal cortex (PFC) of the BTBR mouse model, and evaluates whether targeted genetic manipulation of  $\alpha 7$  and  $\beta 2$  subunits, via CRISPRa or shRNA, can alter circuit function and associated behavioral outcomes.

To characterize nAChR expression in VIP interneurons and PV+ chandelier cells (ChCs), we combined RNAscope and viral labeling (BiPVe4-dTomato) followed by immunostaining, respectively. In the PFC, VIP interneurons comprise approximately 2% of the neuronal population, of which 35% express  $\alpha 7$ , 41.8% express  $\beta 2$ , and 20% co-express both subunits. In addition, 77% of cells transduced by the ChC-targeting virus are PV+ interneurons, representing 1.45% of total PFC cells.

Initial behavioral screening of BTBR mice compared to C57BL/6 controls revealed significant differences. The mice behavioral phenotype was characterized by hyperactivity, altered anxiety-like responses, and impaired cognitive flexibility. Additionally to the traditional behavioral assays, their social spontaneous behavior is being assed throught automated analysis with Live Mouse Tracker (LMT). Together, these findings aim to define how nicotinic receptor modulation in specific interneuron populations shapes prefrontal circuit dynamics and contributes to behavioral phenotypes relevant to ASD.

# POSTER PRESENTATIONS

**Amogelang Ramatsetse**  
**Stellenbosch University**

**SANS26-P66**

**Background/Aim**

Extreme weather events (EWEs) are associated with adverse mental health outcomes and strain on already overburdened health systems. Evidence from African settings examining mental health emergency department (ED) presentations during EWEs remains limited. This study investigated the association between the Western Cape floods of September 2023 and wildfires of January 2024 and mental health ED presentations, compared to matched non-event periods in the preceding year.

**Methodology**

A retrospective matched-period analysis using electronic intake data from EDs at six hospitals, obtained from the Provincial Health Data Centre. EWE exposure and aftermath months were compared to matched control months from the previous year. The sample comprised 2,161 presentations: 714 during EWE periods and 1,447 during matched controls.

**Results**

Presentations fell significantly during EWE event months: -77.8% (September 2023,  $p<0.001$ ) and -72.9% (January 2024,  $p<0.001$ ). Aftermath volumes rebounded but remained below control levels. Flood-period presentations were more likely to result in admission than wildfire-period presentations (OR=1.21, 95% CI 1.02-1.44,  $p=0.025$ ). Length of stay was longer in the flood aftermath (median 13.0 vs 8.5 days,  $p=0.020$ ), with adults aged 60 and older showing twice the expected length of stay (RR=2.02, 95% CI 1.14-3.56,  $p=0.016$ ). Women were over-represented during flood-related EWE periods (38.2% vs 30.8%,  $p=0.020$ ).

**Conclusion**

EWEs were associated with acute suppression of mental health service utilisation, potentially reflecting access barriers. Higher admission rates, longer inpatient stays, and differential effects among older adults suggest those who presented had more complex needs. Differential findings by event type, sex, and age highlight that vulnerability to EWE-related service disruption is not uniform across the population.



# POSTER PRESENTATIONS

**Pia Scharein**

**University of Cape Town**

**SANS26-P65**

## Background

Pyramidal neurons are the principal excitatory cells of the human cortex and underpin cognitive function. Functional connectivity between these neurons is lowest in the paediatric brain and increases into adulthood. Paradoxically, their synaptic density peaks in late childhood before tapering into adulthood. This disparity may be explained by silent synapses, anatomically present but functionally inactive connections at resting membrane potentials - owing to absent AMPA receptors (AMPA-R). My research aims to identify and characterise silent synapses across the human postnatal lifespan.

## Methods

Human cortical tissue is obtained from patients undergoing surgical resection for epilepsy. Patch-clamp recording with minimal stimulation is used to probe synaptic connections between pyramidal neurons in living brain slices using failure rate estimation.

## Results

Preliminary results show synaptic failure rates trending downwards with age, reflecting increasing reliability of neurotransmission. In alignment, silent synapse proportions appear highest in paediatric cortical neurons, and notably persist into adulthood. The AMPA-R/NMDA-R ratio, reflecting the balance of receptors dominating synaptic transmission, shows an increasing trend across development.

## Conclusion

A downward trend in silent synapse populations across development is consistent with experience-dependent synapse maturation. The persistence of silent synapses into adulthood suggests a substrate for lifelong plasticity, which will be further characterised mechanistically through plasticity pairing recording protocols. The increasing AMPA-R/NMDA-R ratio is consistent with activity-dependent AMPA-R insertion at previously silent synapses. Together, these results suggest a developmental shift in pyramidal neuron microcircuit connectivity from a highly plastic state supporting rapid learning in the paediatric brain, to reliable information processing in the adult brain.



# POSTER PRESENTATIONS

**Alexander Sittmann**

**University of Cape Town**

**SANS26-P77**

## Background

Long non-coding RNAs (lncRNAs) are key regulators of chromatin dynamics and transcription in the central nervous system, yet their interactions with chromatin-remodelling genes (CRGs) remain poorly characterised. This study aimed to systematically map tandem lncRNA-CRG pairs and assess whether these loci form context-dependent cis-regulatory modules across neuronal development, injury, and disease.

## Methods

A curated set of 277 lncRNA-CRG pairs was generated using Gene Ontology expansion and genomic proximity filtering ( $\leq 5$  kb). Public bulk RNA-seq datasets spanning neuronal differentiation, activation, injury, and neurodevelopmental disease were processed through a standardised RSEM/DESeq2 pipeline. Co-expression was assessed using correlation analyses, while differential expression analysis identified context-specific regulatory responses. Additional pairs were subsequently examined in an in-vitro RA/BDNF-differentiated SH-SY5Y neuroblastoma model.

## Results

41 lncRNA-chromatin remodeller pairs demonstrated consistent positive co-expression across datasets, with many additional pairs exhibiting strong but context-specific correlations. However, co-differential expression was relatively rare and highly condition-dependent. Notably, ENSG00000240801/IGF2 exhibited regulation across SARS-CoV-2 neuronal infection, a 22q11.2 deletion organoid model, and SH-SY5Y differentiation, implicating a conserved stress- and development-responsive axis. Additional examples further illustrate disease-selective engagement. Experimental validation in SH-SY5Y cells supported several predicted regulatory relationships.

## Conclusion

Tandem lncRNA-CRG loci exhibit widespread but spatiotemporal co-regulation, suggesting that genomic proximity confers latent regulatory potential activated under specific biological conditions across development. This study provides a scalable framework for identifying cis-acting lncRNA regulators and highlights candidates for future investigation.



# POSTER PRESENTATIONS

**Tshiamo Sebigi****North-West University****SANS26-P31**

## Background

More than 50% of people living with HIV (PLWH) develop HIV-associated neurocognitive disorders (HAND). Despite antiretroviral therapy (ART), milder forms of HAND persist. This may be due to dysregulated inflammation and metabolism that persist despite ART. The Tryptophan-Kynurenine (Trp-Kyn) pathway has been implicated in HAND. Additionally, inflammation is known to stimulate Trp-Kyn through the IDO activity. C-reactive protein (CRP) and IL(interleukin)-6 are key markers that link systemic inflammation with neuroinflammation and neurodegeneration.

## Aim

To assess Trp-Kyn pathway alterations and their association with inflammatory markers (CRP and IL-6) in PLWH.

## Methods

Tryptophan (Trp), kynurenine (Kyn), and kynurenic acid (KA) were quantified in serum of ART-treated PLWH (n=278) and healthy controls (HC's) (n=104) using liquid chromatography-tandem mass spectrometry (LC-MS/MS). The Kyn/Trp ratio was calculated as a surrogate marker of IDO-1 activity. IL-6 and CRP were measured using electrochemiluminescence immunoassay and particle-enhanced turbidimetric immunoassay. Associations between metabolites and inflammatory markers were assessed using correlation and regression analyses.

## Results

PLWH demonstrated reduced Trp levels and elevated Kyn, KA, and Kyn/Trp ratios compared to HCs ( $p < 0.05$ ). In PLWH, IL-6 was positively associated with Kyn ( $\beta = 0.146$ ,  $p = 0.039$ ) and the Kyn/Trp ratio ( $\beta = 0.142$ ,  $p = 0.042$ ).

## Conclusion

These findings indicate activation of the Trp-Kyn pathway in PLWH and its association with systemic inflammation. This interaction may represent a mechanistic link to neuroinflammatory processes relevant to HAND.



## POSTER PRESENTATIONS

**Antipa K. Sigilai**

**SANS26-P01**

**Institute for Human Development, Aga Khan University**

### Background

This study examined caregiver assessment of executive functioning (EF) in perinatally HIV-infected (PHIV) and perinatally HIV-exposed but uninfected (PHEU) Kenyan children and the extent to which various biopsychosocial factors influence EF outcomes.

### Methods

Children aged 3-5 years that were PHIV (n=43), PHEU (n=52), and HIV-unexposed uninfected (HUU, n=58) and their caregivers were enrolled. EF was measured using the Childhood Executive Functioning Inventory. Shona Symptoms Questionnaire (SSQ) and parenting behaviour scale were used to evaluate caregivers' common mental disorders (CMDs) and parenting behaviour respectively. We used analyses of variance to assess groups' differences in EF scores, and a hierarchical linear regression model to explore covariates associated with EF outcomes.

### Results

Overall, we observed significant negative effects of HIV exposure on EF scores  $F(2, 149) = 8.591, p < 0.001$ . Compared to HUU children, PHIV children had poor performance in working memory (mean difference (MD), 2.89 [95%CI: 0.65-5.14]  $p = 0.008$ ), inhibitory control (MD, 2.47 [95%CI: 0.55-4.40],  $p = 0.008$ ), and composite EF (MD, 5.37 [95%CI: 1.97-8.76],  $p = 0.001$ ) while PHEU children had poor performance on working memory (MD, 3.24 [95%CI: 1.11-5.37],  $p = 0.001$ ) and composite EF scores (MD, 4.97 [95%CI: 1.75-8.19],  $p = 0.001$ ). The observed EF impairment was strongly associated with caregivers' CMDs and advanced HIV disease in children.

### Conclusion

Our study suggests that caregivers can observe overt executive dysfunction in children who are perinatally exposed to HIV. These findings underscore the importance of ART adherence in PHIV group and psychosocial support to caregivers of HIV-exposed children to help improve EF outcome.

## POSTER PRESENTATIONS

**Sifikile Songo**

**SANS26-P14**

**Manicaland State University of Applied Science**

Neurodivergent individuals experience unique mental health challenges which need to be addressed differently. While mental health outcomes are gaining traction in research, the intersection of neurodivergence and mental health of neurodivergent learners in the African context remains unexplored. This study seeks to fill the gap by examining the interface of neurodivergence and mental health. Understanding these interactions is crucial in developing inclusive mental healthcare practices that address mental health outcomes among neurodivergent learners. An exploratory study was conducted. Participants were purposively selected based on the willingness to take part in the study. Data was collected in November and December 2018 from 15 special needs education teachers in disability support facilities using one-on-one in-depth interviews. Thematic analysis was used to analyze data. The findings revealed that neuro-divergent learners face negative mental health outcomes which emanate from internal factors, psychosocial factors and failure to cope with failure to cope with the learning activities. However, the strategies for addressing mental health challenges which encompass classroom, teacher, school and home set up strategies were revealed. The implications of this study are that there is a need to effectively implement universal design for learning in disability support facilities. Future research studies may need to investigate the efficacy of universal design for learning in these facilities.



# POSTER PRESENTATIONS

**Kaitlyn Sparks**

**University of Oxford**

**SANS26-P58**

## Background and Aims

It is thought that a combination of various factors, including genetic predisposition and environmental stressors, all contribute to the development of schizophrenia. These currently not-well-understood factors are thought to cause signalling cascades that lead to the hallmark symptoms of this condition. Current untargeted treatments do not alleviate all symptoms and can unintentionally disrupt multiple biological pathways, leading to a myriad of side effects. This directly contributes to low adherence to medication and the high societal burden of schizophrenia. Thus, further exploring the molecular nature of this condition in order to pinpoint specific drug targets is of high priority.

## Methods

Using novel computational techniques, we have combined long-read transcriptomics and subcellular proteomics data from the same subjects to explore protein subcellular localisation and isoform expression within the (post-mortem) dorsolateral prefrontal cortex of schizophrenia patients and controls.

## Results and Conclusions

Although no data has been published yet and pipeline optimisation is still ongoing, several avenues of future exploration have already been revealed. We are hopeful that further analysis will eventually lead to the development of better and safer treatments for patients suffering from schizophrenia.



# POSTER PRESENTATIONS

**Anicia Thirion****North-West University****SANS26-P07**

## Background

HIV-associated neurocognitive impairment persists despite widespread antiretroviral therapy. Viral proteins such as transactivator of transcription (Tat) and viral protein R (Vpr) contribute to neurotoxicity and immune activation, yet the impact of subtype-specific sequence variation remains unclear, particularly in regions where HIV-1 subtype C predominates. In parallel, inflammation-driven dysregulation of the kynurenine pathway has been linked to neurocognitive dysfunction. This exploratory study examined associations between Tat and Vpr sequence variation, inflammatory markers, kynurenine metabolites, and neurocognitive outcomes in a South African cohort.

## Methods

Participants with chronic HIV infection were followed longitudinally. Tat and Vpr sequences from baseline plasma (2010) were analyzed for amino acid variation at functionally relevant sites, using subtype C residues as reference. Inflammatory markers and kynurenine metabolites were quantified via mass spectrometry. Neurocognitive performance across domains was assessed at 5-year follow-up. Exploratory analyses included Spearman correlations, t-tests, and unadjusted linear regression ( $p < 0.1$ ; no correction for multiple testing).

## Results

Tat variation at position 68 showed a direct association with attentional performance. However, most mutation effects were attenuated after inclusion of inflammatory markers and kynurenine metabolites. Kynurenine metabolites demonstrated more consistent associations with neurocognitive and psychiatric outcomes.

## Conclusion

These findings suggest that viral sequence variation may influence outcomes indirectly via immune-metabolic pathways. Results are hypothesis-generating and require confirmation in larger, adjusted analyses.



# POSTER PRESENTATIONS

**Babalwa Tshoba**

**University of KwaZulu-Natal**

**SANS26-P41**

## Background

Post-traumatic hydrocephalus (PTH) is a severe neurological condition linked to significant morbidity and mortality. This study explores outcomes of patients treated for PTH at a high-volume neurotrauma centre in KwaZulu-Natal (KZN), South Africa.

## Methods

A retrospective review of patients managed for PTH at Inkosi Albert Luthuli Central Hospital between 2006 and 2024 was conducted. Data collected included demographics, injury mechanisms, clinical and imaging findings, interventions, and Glasgow Outcome Scale (GOS) scores at discharge. The study assessed factors associated with favourable (GOS 4-5) and unfavourable (GOS 1-3) outcomes.

## Results

Fifty-eight patients, predominantly male (88%) with a median age of 26.5 years, were included. The primary causes of injury were assault (39.7%) and motor vehicle collisions (29.3%). Admission Glasgow Coma Scale (GCS) scores were severe (3-8) in 31%, moderate (9-12) in 24%, and mild (13-15) in 45%. Focal neurological deficits (31%) and seizures (20.7%) were common. Surgical interventions included external ventricular drains (25.8%) and decompressive craniectomy (15.5%), with all patients eventually receiving ventriculoperitoneal shunts. Shunt complications occurred in 20.7%. Unfavourable outcomes were observed in 50% of patients, with a 10.3% mortality rate. Factors linked to poorer outcomes included focal deficits ( $p=0.001$ ), hemiparesis ( $p=0.04$ ), infections ( $p=0.007$ ), and elevated INR ( $p=0.04$ ). Prolonged hospital stays were more frequent in this group ( $p=0.004$ ).

## Conclusions

PTH is associated with high rates of neurological deficits, complications, and mortality. Preventing traumatic brain injuries, along with early diagnosis, timely surgical management, and rehabilitation, are critical to improving patient outcomes.



# POSTER PRESENTATIONS

**Godswill Uzoechina**

**SANS26-P67**

**University of Nigeria Teaching Hospital**

## Background/Aim

FAIR brain data and NeuroAI are expanding globally, yet African evidence on governance, infrastructure, and ethics remains fragmented. This review synthesised empirical evidence on brain-data governance, infrastructure capacity, and ethical safeguards in sub-Saharan Africa.

## Methods:

A PRISMA-guided systematic review of peer-reviewed primary studies (2016–2025) was conducted, including MRI infrastructure surveys, neuroimaging datasets, neuroethics, and implementation studies. Data extracted covered FAIR practices, governance structures, consent models, infrastructure capacity, and implementation outcomes.

## Results

Nine studies from Nigeria, South Africa, Uganda, and multi-country SSA settings were included. Evidence included MRI infrastructure surveys (Ogbole et al., 2018; Anazodo et al., 2023), neuroethics studies (Ochang et al., 2023; 2024), implementation research (Nassanga et al., 2025; Hendrikse et al., 2022), and FAIR datasets (Wogu et al., 2025). Findings show severe infrastructure gaps, including <1 MRI scanner per million population, limited computing capacity, and weak data storage systems. FAIR adoption was inconsistent, though the Nigerian MRI dataset demonstrated BIDS conversion, pseudonymisation, and controlled-access sharing. Ethical challenges included variable consent models, weak data sovereignty, stigma risks, incidental findings management, and inequitable HIC-LMIC data flows. Community engagement and local governance were inconsistently reported.

## Conclusion

FAIR NeuroAI and brain-data governance in Africa remain constrained by infrastructure deficits, fragmented ethics, and limited data sovereignty. Strengthening local data governance, standardising FAIR implementation, and promoting equitable collaboration are essential for sustainable NeuroAI development in Africa.



## POSTER PRESENTATIONS

**Justine van Greenen**

**University of Cape Town**

**SANS26-P76**

### Introduction:

During brain maturation, spatiotemporal gene expression is tightly regulated by complex gene regulatory networks (GRNs) controlling cell development and function. Identifying putative enhancers and their targets is essential for building a comprehensive GRN. This study aims to identify enhancers that are differentially regulated between paediatric and adult brain samples to contribute to our understanding of the GRN that guides human brain maturation.

### Methods:

Enhancers, found in non-coding, chromatin regions were identified using ATAC-seq. Bulk ATAC-seq libraries from ten paediatric and four adult temporal lobe samples spanning 10 months to 40 years old, were sequenced and processed with two published bioinformatics pipelines for differential accessibility analysis.

### Results:

A consensus peak set of 64,058 accessible regions, including putative enhancer and promoter regions, was generated. Differential accessibility analysis between adult and paediatric samples over these consensus peaks revealed 181 candidate enhancers with 155 more accessible in paediatric and 26 more accessible in adult. The developing brain undergoes rapid neuronal differentiation, synaptogenesis, and myelination — processes requiring dynamic transcriptional programmes supported by accessible chromatin at regulatory regions. Several of the identified candidate regions are near neurodevelopmental genes such as NFIA, ROBO2 and CHRNA5.

### Discussion:

Identifying differentially accessible peaks overlapping from both pipelines provides high-confidence enhancer candidates guiding brain maturation. This data contributes to the world-wide effort of developing a human cell atlas, which currently lacks global diversity, to gain a deeper understanding of the cellular mechanics behind neuropsychiatric disorders.



## POSTER PRESENTATIONS

**Daneille Sasha-Leigh van Rensburg**  
**University of the Western Cape**

**SANS26-P36**

### Background

The Blood-Brain Barrier (BBB) is a highly selective, semipermeable specialized barrier regulating the passage between blood and the parenchyma. However, the integrity of the BBB can be compromised in conditions such as HIV/AIDS, HIV-associated neurocognitive disorders (HAND), affecting drug penetration into the brain. Tenofovir disoproxil fumarate (TDF) and the newer tenofovir alafenamide (TAF), combined with emtricitabine (FTC), are widely used nucleoside reverse transcriptase inhibitors used in HIV treatment. TAF offers improved intracellular delivery and lower plasma tenofovir concentrations compared to TDF, potentially reducing renal and bone toxicities. However, their comparative effects on the blood-brain barrier, a critical interface regulating CNS drug exposure, still remain underexplored.

### Methodology

b.End5 cells were exposed to clinically relevant concentrations (1.25ng-75ng/ml) of TAF/FTC or TDF/FTC for 24-96 hours. Cell viability, proliferation, toxicity, and suppression were assessed using the trypan blue exclusion assay, while morphological changes were evaluated through haematoxylin and eosin (H&E) staining.

### Results

TDF/FTC significantly reduced cell viability compared to the controls, with prominent morphological changes and cytoplasmic swelling observed with H&E staining. TAF/FTC showed milder viability reductions after 72 hours and preserved cell morphology for a longer period of time.

### Conclusion

TAF/FTC demonstrates greater endothelial safety compared to TDF/FTC, preserving cell viability and morphology.



# POSTER PRESENTATIONS

**Chontrelle Willemse**

**University of the Western Cape**

**SANS26-P39**

## Background

The brain endothelial cells (BECs) are the blood-brain barrier's (BBB) guardian, protecting the brain parenchyma from systemic hazards. One such hazard of drug abuse is methamphetamine (MA), a widely used psychostimulant known for its neurotoxic properties and its ability to compromise the BBB, interfering with the brain's cognitive domains. Aim: This study investigated the in vitro effects of acute and chronic MA exposure on the BECs cellular proliferation, toxicity, permeability, and morphology.

## Methods

bEnd.5 cells were exposed to 0.1 - 100 $\mu$ M MA acutely for 24h only and chronically over a 96h period. The cellular analyses were done using the Trypan Blue Exclusion Assay. The permeability was determined by the transendothelial electrical resistance, and morphological changes were imaged by hematoxylin and eosin stain.

## Results

Cellular suppression was observed for acute and chronic exposure between a range of 5-31%. This inference was drawn from the significant decrease in cellular proliferation across the acute and chronic exposure concentrations. For both the acute and chronic exposure, a recovery period of 48h was observed. For TEER, a significant dose-dependent decrease throughout the 96h was observed at both exposures, and the morphological analyses revealed nuclear abnormalities with consistent mitotic disruption.

## Conclusion

The data show that acute MA exposure can recover cellular proliferation; however, this is not possible for chronic exposure. More importantly, both exposures weaken the physiological integrity of the monolayer permeability, with chronic MA showing a more damaging effect. In safeguarding cognitive health, early intervention should be prioritized at the earliest stage of MA use.



## POSTER PRESENTATIONS

**Kimberley Clare Williams**  
**University of Cape Town**

**SANS26-P56**

Electroencephalography (EEG) provides high-temporal-resolution measurement of brain electrical activity, with spectral power in canonical frequency bands serving as a key biomarker in cognitive and neurological research. Most processing pipelines require familiarity with specialised toolboxes such as EEGLAB, FieldTrip, or MNE-Python, limiting access for researchers without advanced programming experience. Here we present the EEG Processing GUI, a self-contained MATLAB graphical user interface for batch spectral analysis of multi-channel EEG recordings stored in .cnt (ANT Neuro) and European Data Format (.edf) files.

The GUI integrates a complete preprocessing and spectral estimation pipeline with no dependency on the MATLAB Signal Processing Toolbox. Bandpass filtering (default: 0.1–30 Hz) and power-line notch filtering (50 or 60 Hz) are implemented via Fast Fourier Transform (FFT)-domain multiplication. Amplitude-based artifact rejection identifies samples exceeding a user-configurable threshold (default:  $\pm 100$   $\mu\text{V}$ ), replacing them with NaN prior to spectral estimation. Power spectral density is estimated using a custom Welch's method. Relative power in the delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), and beta (13–30 Hz) bands is computed by trapezoidal integration and normalised to total power.

The GUI supports flexible channel selection including standard 10-20 montage presets, and exports per-channel results to Microsoft Excel or CSV. Real-time status feedback and a results preview panel facilitate rapid quality assessment. Requiring only core MATLAB functions, the tool is immediately deployable on standard workstations, addressing a critical gap in accessible, reproducible EEG spectral analysis for clinical and cognitive neuroscience

# POSTER PRESENTATIONS

**Onethemba Xoma**

**University of Cape Town**

**SANS26-P51**

## Background/Aim

Prenatal socioeconomic adversity and antenatal maternal depression (AMD) often co-occur. Both are associated with altered early brain development and neurodevelopmental vulnerability, suggesting that their effects on cognition may emerge early in childhood. Working memory (WM), an executive function critical for academic outcomes, may be particularly sensitive to these early exposures. This study examined associations between prenatal socioeconomic status (SES), AMD, and WM at age 5.

## Methods

Data from 583 mother-child dyads, from the Drakenstein Child Health Study (DCHS), a South African birth cohort, were analysed. Composite SES derived from maternal education level, household income, assets, and employment, was dichotomized (high vs low). AMD was defined as scoring above validated thresholds on either the Beck's Depression Inventory-II ( $\geq 20$ ) or the Edinburgh Postnatal Depression Scale ( $\geq 13$ ) at 28-32 weeks of gestation. WM was assessed at 5 years using the WPPSI-IV: Picture Memory task. Linear regression models examined associations between SES, AMD, and WM, adjusting for child sex and age.

## Results

Among 583 children (mean age 5.08 years; 52% male), 34% were exposed to AMD and 51% were classified as lower SES. Perinatal characteristics were broadly comparable across SES and AMD groups. Lower prenatal SES was associated with poorer WM ( $B = -0.67$ ,  $p < 0.001$ ). AMD was not associated with WM in minimally adjusted models or SES-adjusted models ( $B = -0.10$ ,  $p = 0.60$ ;  $B = 0.016$ ,  $p = 0.93$ ).

## Conclusion

Prenatal socioeconomic adversity may have lasting effects on WM at 5 years. Future analyses will examine potential mediators, including brain structure.

# POSTER PRESENTATIONS

**Ouattara Yacouba**

**SANS26-P72**

**Universite Felix Houphouët-Boigny**

## Background/Aim

Digital learning tools are increasingly used in primary education, yet their neurocognitive impact remains insufficiently characterized. Standard interfaces apply fixed difficulty levels and may induce cognitive overload or disengagement. Adaptive systems, by contrast, dynamically adjust task demands and may optimize cognitive resource allocation. This study aimed to determine whether an adaptive visuospatial learning interface modulates neural markers of cognitive load and attentional engagement in children, compared to a static digital interface.

## Methods

38 pupils performed a visuospatial working memory task (Corsi Block Test) under two counterbalanced conditions: a standard fixed difficulty version (4 blocks) and an adaptive version adjusting difficulty trial by trial based on performance. EEG activity was recorded using a wireless 14 channel EPOC X headset. Spectral analyses focused on frontal theta power (index of mental effort) and parieto occipital alpha power (marker of attentional filtering). Behavioral performance and difficulty progression were also quantified.

## Results

The adaptive condition elicited a marked reduction in frontal theta power, indicating lower cognitive load relative to the standard interface. Conversely, alpha power significantly increased ( $p = 0.012$ ), reflecting enhanced attentional engagement and improved distractor suppression. Behaviorally, a strong positive correlation ( $r = 0.55$ ;  $p = 0.0004$ ) between success rate in the standard condition and difficulty reached in the adaptive mode confirmed that the system effectively targeted each child's proximal zone of development.

## Conclusion

Adaptive learning acts as a dynamic regulator of neural resources, reducing overload while sustaining engagement. These findings highlight the potential of neurotechnology informed educational systems to support cognitive efficiency and contribute to advancing brain health in developing learning environments.



# POSTER PRESENTATIONS

**Simo Zulu**

**Nelson Mandela University**

**SANS26-P45**

## Background

The penetration of antiretroviral drugs into the central nervous system (CNS) is limited by blood-brain barrier (BBB) transporters, particularly efflux proteins such as P-glycoprotein (P-gp) and multidrug resistance-associated proteins (MRPs). HIV-1 Tat, a viral protein released from infected cells even during antiretroviral therapy, has been implicated in regulating these transporters; however, the extent of evidence on this remains unclear. This scoping review aimed to synthesize available data on Tat-mediated regulation of BBB drug transporters and its potential impact on CNS drug penetration.

## Methods

A systematic search following PRISMA-ScR guidelines was conducted in PubMed, Scopus, and Web of Science by two independent reviewers. Eligible studies were assessed for quality, and data relating to Tat-mediated regulation of transporter expression and function were extracted. Of 104 identified studies, 6 met the inclusion criteria.

## Results

Most studies reported upregulation of P-gp in brain endothelial cells following Tat exposure. Findings for MRP-1 were inconsistent, with one study showing upregulation and another reporting no effect. Tat was also found to downregulate ABCA1 expression in astrocytes. Transporter expression was primarily assessed using PCR and Western blot, with some studies evaluating functional activity. Mechanistically, Tat-induced P-gp upregulation was linked to activation of the Rho signalling pathway and NF- $\kappa$ B, while MRP-1 upregulation was associated with MAP-kinase pathway activation.

## Conclusion

Current evidence suggests that Tat may enhance the expression of select BBB efflux transporters, potentially limiting CNS antiretroviral drug penetration. However, the small number of studies and inconsistent findings highlight the need for further research.



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