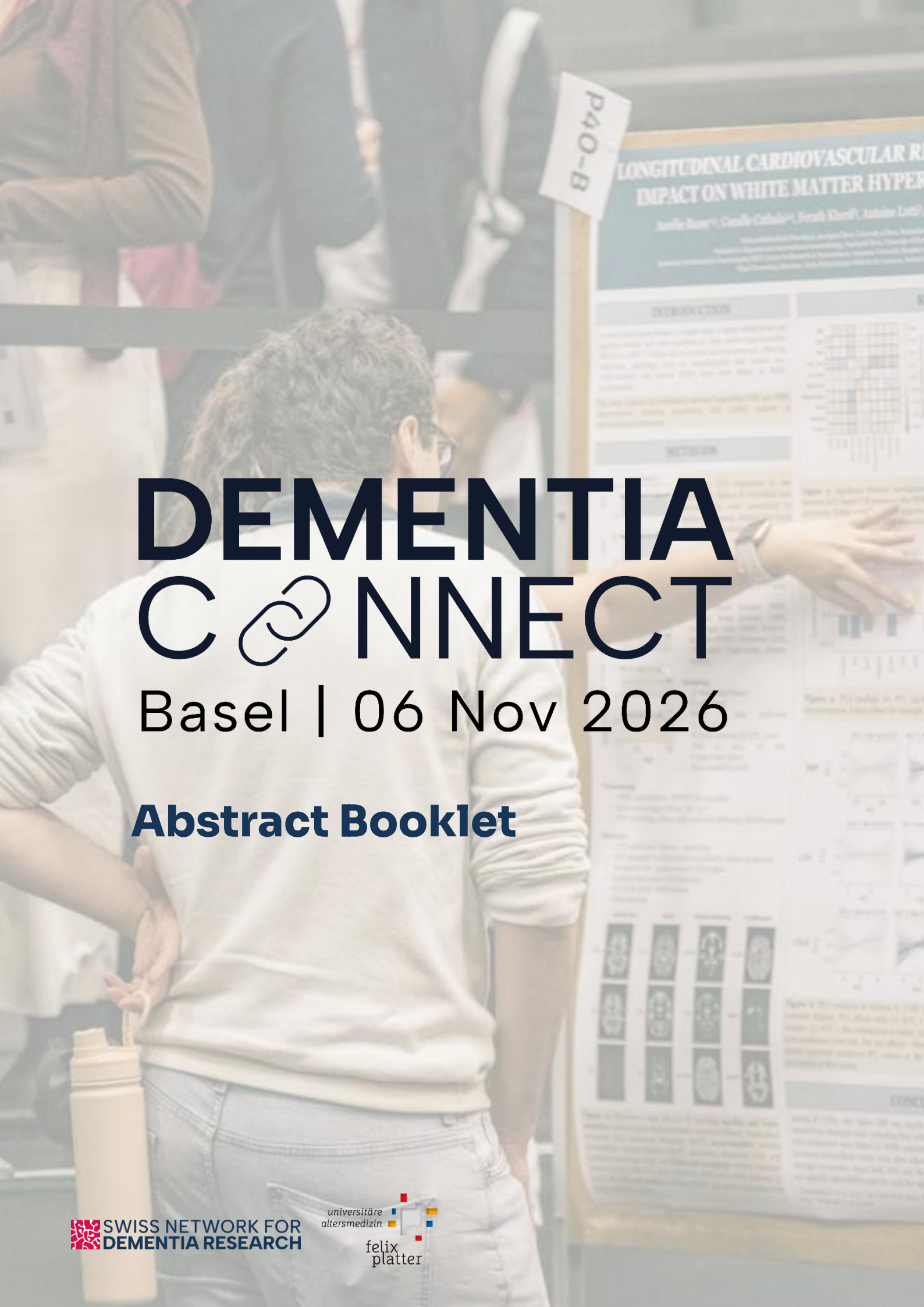


DEMENTIA CONNECT

Basel | 06 Nov 2026

Abstract Booklet

SWISS NETWORK FOR DEMENTIA RESEARCH

universitäre altersmedizin felix platter

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Program Overview

Chair: Prof. Marc Aurel Busche, Universitäre Altersmedizin Felix Platter Basel

09:00 – 09:30 | SITE VISIT REGISTRATION

09:30 – 11:00 | CLINICAL SITE VISIT

10:45 – 11:15 | DEMENTIA CONNECT REGISTRATION

11:15 – 12:00 | KEYNOTE LECTURE

Biofluid biomarkers in Alzheimer's disease
Prof. Henrik Zetterberg, University of Gothenburg, University College London

12:00 – 13:00 | NETWORKING LUNCH

13:00 – 15:00 | SCIENTIFIC EXCHANGE SESSION

Dendritic inhibitory origins of inaccessible, intrusive, and unstable memories
Everton J Agnes, University of Basel

ProMental – A Pragmatic Multidomain Program for Dementia Prevention
PD Dr. Anton Gietl, University Psychiatric Hospital of Zurich - Sponsored by Schwabe Pharma

Early impairment of fast-spiking interneurons in Alzheimer's Disease
Jana Zünkler, University College London, University of Basel, Universitäre Altersmedizin Felix Platter Basel

Early Alzheimer's detection via fluid biomarkers to improve patient care
Dr. Martin Guess - Sponsored by Roche

Nanoscale mapping of presynaptic molecular architecture during ageing
Carola I. Radulescu, Imperial College London

Beyond pTau-217: Expanding Alzheimer's insights with multiplex protein detection
Muamer Hodzic - sponsored by Alamar Biosciences

Recharging brain energy through mitochondrial transplantation: A promising therapeutic avenue for neurodegenerative diseases
Dr. Amandine Grimm, University Psychiatry Clinics Basel

Topline results from CELIA: A phase 2 study to evaluate the tau-targeting ASO diranersen (BIIB080) in patients with early Alzheimer's disease
Dr. Yuval Zabar – Sponsored by Biogen

15:00 – 16:00 | POSTER SESSION

Informal exchange over coffee, featuring poster presentations from researchers and healthcare professionals.

Keynote lecture

Biofluid biomarkers in Alzheimer's disease

Prof. Henrik Zetterberg, University of Gothenburg, University College London



Alzheimer’s disease (AD) is a progressive neurodegenerative disease, and the single commonest cause of dementia. Many other diseases can, however, cause dementia and differential diagnosis can be challenging especially in early disease stages.

For most neurodegenerative dementias, accumulation of brain pathologies starts many years before clinical onset; the ability to detect these pathologies paves the way for targeted disease-modifying prevention trials. AD is associated with amyloid β and tau pathologies which can be quantified using cerebrospinal fluid and imaging biomarkers and, more recently, using highly sensitive blood tests. While for the most part specific biomarkers of non-AD neurodegenerative dementias are lacking, non-specific biomarkers of neurodegeneration are available. This talk summarizes recent advances in the neurodegenerative dementia blood biomarker research, and discusses the next steps required for clinical implementation and research in a global perspective.

Scientific Exchange Session

Dendritic inhibitory origins of inaccessible, intrusive, and unstable memories

Everton J Agnes, Biozentrum, University of Basel

Failure in excitatory-inhibitory (EI) balance is a defining feature of many memory-related diseases, such as Alzheimer's disease (AD). A key unresolved question is whether this EI disruption erases stored memories or renders them inaccessible. Optogenetic reactivation of engram cells in AD models suggests the latter, a state we recently reproduced in a biophysical spiking network model (Berger & Agnes, bioRxiv 2026). In our model, local dendritic EI balance is sufficient to implement high-capacity attractor-based associative memory. Multiple dendrites could thus store a high number of memory sets, one per dendritic branch. We showed that branch-specific alpha5-GABAA inhibition can gate their accessibility for recall. Dendritic inhibition is therefore two physically distinct systems: (i) recurrent, which stores memories and is responsible for local EI balance, and (ii) gating, which controls the communication between dendrites and soma. Gating fails in two directions: too much renders memories inaccessible (amnesia) and too little permits unrelated memories to intrude (like post-traumatic stress disorder). The dendrite-targeting interneurons that feed both systems are affected in many memory-related diseases, yet the consequences remain poorly understood. Here, I will show simulations in which reducing recurrent inhibition leaves recall unaffected over a broad plateau and then collapses it, and raising the gating restores recall, as lowering it does when recurrent inhibition is increased. This would explain why both positive and negative allosteric modulation of alpha5-GABAA receptors show benefits in different contexts. For memory formation, our co-dependent plasticity framework (Agnes & Vogels, Nat Neurosci 2024), in which inhibition gates excitatory plasticity, implies that when inhibition is chronically weakened, excitatory weights are no longer constrained, destabilising existing memory traces and corrupting new ones. Together, the two frameworks provide the first mechanistic account linking dendritic inhibitory dysfunction to both memory inaccessibility and impaired memory formation, laying groundwork for precise targeting of inhibitory circuits.

Early impairment of fast-spiking interneurons in Alzheimer’s Disease

Jana Zünkler, UK Dementia Research Institute at University College London, UK, Department of Biomedicine, University of Basel, University Hospital of Geriatric Medicine FELIX PLATTER

Objectives: Alzheimer's disease (AD) is characterized by a prolonged presymptomatic phase during which amyloid-β accumulates silently across the brain, yet increasing evidence suggests that distinct neuronal subtypes are particularly vulnerable to this early pathology. However, the mechanistic underpinnings of this differential vulnerability and its impact on network-level dysfunction remain to be fully elucidated. We hypothesized that amyloid-β-driven selective dysfunction of cortical fast-spiking interneurons disrupts network oscillations and circuit dynamics crucial for memory and cognition. Testing this hypothesis necessitates an examination of intact neuronal circuits with single cell/unit resolution at early stages of amyloid-β accumulation.

Methods: We performed Neuropixels electrophysiological recordings, assessing single-neuron as well as network activity in a variety of brain regions in the APPNL-G-F mouse models of AD, at two to four months of age, during the awake resting and locomotion states. In parallel, we quantified the development of pathology across brain regions and investigated the mechanistic underpinnings using high-resolution confocal imaging.

Results: We provide evidence for an early destabilization of the spatiotemporal dynamics of neuronal firing and oscillatory activity across the recorded brain regions. Ongoing analyses further suggest that altered contributions of cortical fast-spiking interneurons may underlie these changes in network

oscillations. Mechanistically, we present evidence for changes in perineuronal nets surrounding parvalbumin-interneurons during early stages of pathology accumulation.

Conclusions: Our preliminary results point towards a causal relationship between the accumulation of amyloid-β pathology and neuronal dysfunction within neural circuits that are critically involved in memory and cognition. Functional alterations driven by fast-spiking interneuron dysfunction appear to be an initial event in the course of the disease, and could thus provide an early pathophysiological signature of AD. Importantly, the changes identified in perineuronal nets highlight a promising therapeutic target for early intervention.

Nanoscale mapping of presynaptic molecular architecture during ageing

Carola I. Radulescu, UK Dementia Research Institute, Department of Brain Sciences, Imperial College London

Synapses have a highly complex and organised nanoscale molecular architecture. Although synaptic dysfunction is an early feature of cognitive decline, how the molecular composition and organisation of individual synapses changes during physiological ageing remains poorly understood. Here, we investigate age-related changes in presynaptic proteins essential for vesicle trafficking and neurotransmitter release. Using expansion microscopy (~3.5-fold expansion), coupled with cyclic immunofluorescence (ExM-CycIF) on mouse motor cortex across three age groups, we multiplexed presynaptic markers (αSyn, VAMP2, SV2A, Syt1) alongside excitatory postsynaptic targets (GluN1, PSD95) to map molecular diversity within discrete trans-synaptic nanodomains. Ageing was associated with a redistribution towards low-complexity synapses, marked by an increased proportion of αSyn-only terminals. We also identified a reduction in high-complexity, multi-marker nanoclusters, accompanied by reduced co-localisation of αSyn with key regulators of synaptic vesicle exocytosis, SV2A, Syt1 and VAMP2, in aged animals. Together, these findings suggest an age-related reorganisation of presynaptic molecular composition that may impair neurotransmitter release, and set the stage for interventions to preserve synaptic function in ageing.

Recharging brain energy through mitochondrial transplantation: A promising therapeutic avenue for neurodegenerative diseases

Dr. Amandine Grimm, Cell Biology and Energy Metabolism, Research Cluster Molecular & Cognitive Neuroscience, University Psychiatry Clinics Basel, Basel, Switzerland

Tauopathies, including Alzheimer's disease (AD) and frontotemporal dementia (FTD), are characterized by pathological tau accumulation, mitochondrial dysfunction, and severe neuronal bioenergetic deficits. Recently, intercellular mitochondrial transfer (IMT), particularly from astrocytes to neurons, has emerged as an endogenous neuroprotective mechanism. We investigated how pathological tau modulates astrocyte-to-neuron IMT and evaluated whether mitochondrial transplantation can rescue tau-induced neuronal dysfunction.

We used in vitro tauopathy models with SH-SY5Y neuronal cells expressing the FTD-associated P301L mutant tau alongside A172 astrocytes, and key findings were validated in patient-derived iPSCs differentiated into neurons and astrocytes.

We showed that astrocytic mitochondrial transfer to neurons was significantly increased in the presence of mutant P301L tau compared to controls. This surge was predominantly mediated by contact-dependent pathways, evidenced by an increased density of intercellular tunneling nanotubes (TNTs). Donated astrocytic mitochondria remained functionally intact and successfully restored cellular bioenergetics in recipient neurons.

Furthermore, the artificial transplantation of healthy astrocytic mitochondria effectively restored neuronal bioenergetics and promoted neurite outgrowth in both control and P301L-expressing neurons.

This study provides the first evidence that disease-associated tau actively regulates endogenous IMT, likely reflecting an adaptive compensatory mechanism to counteract metabolic failure. Crucially, direct mitochondrial transplantation successfully reduced tau-mediated bioenergetic deficits and structural damage. These findings establish mitochondrial transplantation as a viable strategy to mitigate neuronal defects, supporting the development of cell-based mitochondrial therapies for neurodegenerative diseases.

Swiss Network for Dementia Research

About us

The Swiss Network for Dementia Research is an association established in October 2023 by Dementia Research Switzerland – Synapsis Foundation. The network is grounded in the belief that pooling resources and promoting interdisciplinary knowledge exchange will drive innovation in dementia research and care. Our mission is to foster a collaborative culture, enabling the discovery of new insights and accelerating their application in clinical practice.

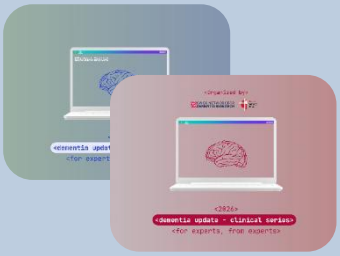
We aim to connect dementia experts across disciplines and institutions throughout Switzerland, creating a national network that bridges gaps in dementia research and care. To facilitate collaboration, we offer services such as a community platform, networking events, and communication channels. Our bottom-up approach ensures that our services are tailored to the needs of the community.

Community platform

The goal of our community platform is to serve as a centralized hub for dementia research and care in Switzerland, connecting all key stakeholders and fostering interdisciplinary collaboration. The platform provides an overview of experts in the field, ongoing projects, current initiatives, and available job and funding opportunities, as well as upcoming events. It also serves as a resource hub, bringing together tools—such as biosamples, animal models or diagnostic tools—to create synergies that support both research and care efforts.

Join us and create your expert profile today!

Dementia Update Series



The **Dementia Update Series** comprises two virtual formats designed to foster collaboration and knowledge exchange in dementia research and care.

Each session combines an expert presentation with an interactive discussion, with educational credits available for participants. The Dementia Update – Clinical Series was developed in collaboration with the Swiss Memory Clinics

Swiss Dementia Forum



A biennial, 1.5-day conference featuring keynote presentations, panel discussions, poster sessions, and workshops, bringing together experts from research, clinical practice, and healthcare delivery. The Swiss Dementia Forum aims to foster interdisciplinary knowledge exchange, initiate new collaborations, and increase the visibility of early-career researchers.

The next Swiss Dementia Forum will take place on 11–12 November 2027.

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Posters

Overview

P1	Ginkgo biloba extract modulates mitophagy and autophagy in a human Alzheimer's disease tau cell line
P2	Exploring the proteomes of vulnerable synapse types in Alzheimer’s Disease progression
P3	Digital approaches to cognitive health
P4	Exosomes in Familial Alzheimer’s disease: drivers of pathogenic cargo spread
P5	Smell, brain health and ageing: A Consensus study to inform dementia prevention
P6	ADAR2, an Alzheimer’s disease-linked RNA-editing enzyme, as a putative regulator of synaptic mRNAs
P7	Language screening in Alzheimer’s Disease: Preliminary findings from the German adaptation of the DTLA
P8	Cognitive spatial maps are less stable across days in an Alzheimer’s Disease mouse model
P9	Investigating the interplay between cell-specific autophagy pathway and AQP4-dependent glymphatic clearance in an Alzheimer's disease mouse model
P10	Beyond Diagnosis: The Caregiver Brain as a Clinical Variable in Dementia Care - A Proposal for The Caregiver Protocol
P11	Bridging the gap in acute geriatric psychiatry: An APN-led implementation plan for the non-pharmacological management of BPSD in a specialised care area (Zone 5)
P12	Turning Microglia Neuroprotective to Combat Alzheimer’s Disease: An FDA-Approved Compound Upregulates the Neurogenic Factor IGF-1
P13	Is brief daily self-report a valid way to track symptoms and lifestyle in MCI and early dementia? A pilot study protocol
P14	Cognitive-Metabolic Decoupling as a Quantitative Trait of Resilience in Alzheimer’s Disease Progression
P15	APN-Led Consultation Services for Delirium and BPSD: Translating Evidence into Clinical Practice
P16	Tracking Within-Person Cognitive Decline Through Automated Speech Analysis: Retrospective Validation and a Planned Clinical Feasibility Study
P17	Nanoscale mapping of presynaptic molecular architecture during ageing
P18	A human, seed-free cell model reproducing the ordered Tau pathology cascade: a translational tool for the dementia field

Poster abstracts

P1: Ginkgo biloba extract modulates mitophagy and autophagy in a human Alzheimer's disease tau cell line

Imane Lejri, Ina Vukalovic, Fairley Lauren, Amandine Grimm, Anne Eckert

University Psychiatry Clinics UPK Basel, Neurobiology Lab for Brain Aging and Mental Health, Basel, Switzerland
University of Basel, Department of Biomedicine, Basel, Switzerland

Aims
Mitochondrial impairment, including impaired mitophagy, has been identified as an early feature of tauopathies, leading to the accumulation of damaged or dysfunctional mitochondria. This study aimed to explore standardized Ginkgo biloba extract (GBE) potential as a therapeutic agent of tauopathies by investigating its e""ects on mitophagy, autophagy and associated protein expression in cells expressing the P301L mutant tau.

Methods
Human neuroblastoma cells SH-SY5Y, overexpressing the P301L mutant tau, were used. Cells were transiently transfected with DsRed-LC3 and then treated with either GBE (LI1370; 100µg/ml) or DMSO vehicle for a 24-hour period. Autophagic flux was assessed using bafilomycin A1 or DMSO, while mitophagy was assessed with FCCP. Mitochondria were visualized with a mitotracker. Autophagy and mitophagy were quantified by measuring fluorescence intensity and assessing colocalization.
To analyze gene expression, total RNA was extracted from cells using the RNeasy Mini kit. RT-qPCR was employed for amplifying and quantifying mRNA expression levels.

Results
Under baseline condition, no impact of a GBE treatment on P301L mutant tau cells was observed. Following stimulation with FCCP, P301L mutant tau cells displayed reduced mitophagy compared to control cells. Baseline levels of LC3 were increased in P301L cells due to impaired autophagosome degradation. Treatment with GBE reduced LC3 levels in P301L cells, indicating improved autophagosome degradation, while it increased in control cells, suggesting enhanced autophagosome formation. Further, treatment with GBE led to changes in the gene expression of PARK2, PINK1 and P62.

Conclusions
Treatment with GBE was able to alleviate mitophagy deficits in P301L mutant tau expressing cells, improve autophagic flux and increase the expression of genes related to mitophagy and autophagy pathways.

P2: Exploring the proteomes of vulnerable synapse types in Alzheimer's Disease progression

Jennifer Baltazar, Simon Waldner, Peter Scheiffele, Marc van Oostrum

Biozentrum, Univeristy of Basel

Alzheimer's disease (AD) is characterized by early synaptic alterations, which precedes pathological features and are critical drivers of cognitive decline. Specific synapse types, particularly in the hippocampus and cortex, show varying vulnerabilities to AD. This project aims to elucidate proteomic alterations at a panel of differentially vulnerable or resilient synapse types including cholinergic, broadly excitatory and inhibitory subtypes, cortical layer-specific, hippocampal input-associated, and interneuron subtypespecific synapse populations during AD progression in amyloid animal models. We are employing our recently developed synapse type-specific proteome analysis, integrating cell-selective presynaptic reporter expression, Fluorescence-activated-Synaptosome sorting (FASS), and mass spectrometry to study type-specific synaptic proteomes in the APPNL-G-F mouse model. We will use computational systems biology approaches to identify molecular changes associated with synapse loss, age, and disease state. This integrated approach will provide insights into the molecular alterations associated with synaptic dysfunction in AD, identify potential avenues for therapeutic intervention and offer a framework to study synaptic molecular changes with type-specificity in other neurodegenerative diseases.

P3: Digital approaches to cognitive health

Arzu Çöltekin

FHNW

The talk samples selected projects at the intersection of computer and data sciences and cognitive health, especially in the context of aging. After summarizing the findings from a visuospatial memory study in a virtual environment including eye movement analyses, we overview the digital biomarkers of cognitive aging and show further results from an augmented reality and artificial intelligence study on predicting, preventing and rehabilitation of post-operative cognitive dysfunction (POCD) in cardiac patients. We finish the talk sharing early work towards using virtual reality for reminiscence therapy which is becoming possible with the use of latest computer vision and deep learning technologies, and a commentary on the promising digital biomarkers in cognitive health.

P4: Exosomes in Familial Alzheimer's disease: drivers of pathogenic cargo spread

Gaiser Carine [1], Motamed Zahra [1,2], Heutschi Daniel [1] and Suter-Dick Laura [1,3]

[1] University of Applied Sciences and Arts Northwestern Switzerland (FHNW), School of Life Sciences, Muttenz, Switzerland
[2] University of Basel, Basel, Switzerland
[3] Swiss Centre for Applied Human Toxicology (SCAHT), Basel, Switzerland

The spread of pathological proteins, notably tau and amyloid-beta (Aβ), is considered a major driver of Alzheimer's disease (AD) progression. Nevertheless, human-specific in vitro models to model disease propagation are still scarce.

We developed a conditioned medium (CM)-based model using human neural progenitor ReN cells carrying Familial AD (FAD) mutations in amyloid precursor protein (APP with Swedish/London variants) and presenilin 1 (PSEN1ΔE9). Healthy ReN-derived neurons and astrocytes exposed to CM from FAD cells for nine weeks exhibited hallmark AD pathologies, including cellular toxicity, neuronal loss, astrocytic hypertrophy, and mitochondrial dysfunction. Interestingly, exosomes released by FAD cells were actively internalized, primarily by astrocytes, via an actin-dependent mechanism. Similarly to FAD cells, healthy ReN-cells exposed to CM from FAD cells released Aβ42. Elevated

Aβ42/40 ratio, characteristic of AD, was detectable in both cell supernatants and exosomes. Additional experiments showed that an acute Aβ42 exposure (20 uM) induces neuro-glial and mitochondrial damage in ReN cells. These findings indicate that released factors contribute to AD pathology dissemination, with exosomes emerging among the key drivers.

Proteomic profiling of FAD-derived exosomes identified proteins involved in inflammation, extracellular matrix remodelling, synaptic dysfunction, and Aβ processing. Notably, we identified two best exploratory candidates, meeting our study-defined FDR cutoff of 0.1, calmodulin-like protein 3 and methyl malonyl-CoA mutase, that may facilitate the intercellular transfer of pathogenic cargo. Independent protein validation will be required in future in vitro studies.

Collectively, these results highlight the potential of exosomes as contributors of the spread of pathological Aβ in AD. Their role in intercellular communication and disease propagation suggests that exosome-associated signatures could support early diagnosis and monitoring of disease progression. Future investigations could focus on validating exosome-associated cargo across different stages of AD using patient-derived biofluids and integrating them into more advanced brain-on-a-chip systems.

P5: Smell, brain health and ageing: A Consensus study to inform dementia prevention

D'Andrea F. [1], Tischler V.[2], Dening T.[3], Boak D.[4], Loughnane M.[2], Middleton P.[5]

[1]University of West London, London, United Kingdom;
[2]Surrey University, United Kingdom;
[3]University of Nottingham, United Kingdom;
[4] SmellTaste charity, United Kingdom;
[5]Person living with dementia

Background: As the world's population is getting older, the proportion of people with cognitive impairment and dementia is increasing. Olfactory dysfunction (reduced/loss of smell) may be a novel modifiable risk factor for cognitive decline and dementia. It affects half of people aged 65 and over and four in five aged 80 and above. People with olfactory dysfunction are at greater risk of cognitive decline, social isolation, malnutrition, depressive symptoms and lower quality of life. Those affected face a higher mortality risk, with people with olfactory dysfunction being over three times more likely to die than those with normal olfactory function. However, due to the regenerative capacity of the olfactory pathway, interventions such as olfactory (smell) training may help mitigate age-related olfactory decline and support healthy ageing. Yet, olfactory dysfunction in older people is under-researched, under-tested and untreated.

Aims: This study aims to advance dementia risk reduction and brain health by: i) establishing an interdisciplinary network of experts in olfaction, ageing and dementia; ii) identifying and reaching consensus on uncertainties, gaps and priorities to drive this field forward; iii) formulating objectives for future collaborative projects.

Methods: A consensus study will be conducted through five iterative online group discussions and a survey with lived-experience and professional experts (n=10–15), informed by the virtual Nominal Group Technique and co-designed with a steering committee.

Results: By establishing an interdisciplinary international research network of people living with dementia, unpaid carers, individuals with olfactory dysfunction, community members, healthcare professionals, industry and charity partners, the project will co-produce priorities to advance knowledge on the role of olfaction in ageing, and cognitive decline.

Conclusion: The identified priorities from this project will shape future research, and policies, lay the groundwork for innovative interventions, and support the integration of olfactory health into dementia prevention efforts.

P6: ADAR2, an Alzheimer's disease-linked RNA-editing enzyme, as a putative regulator of synaptic mRNAs

Marcel Juengling[1,2,3], Susanne tom Dieck[1], Georgi Tushev[1], Janus Mosbacher[1], Elena Ciirdaeva[1], Erin M. Schuman[1]

[1] MPI for Brain Research, Frankfurt, Germany.
[2] Department of Neurodegenerative Diseases, University Hospital of Geriatric Medicine FELIX PLATTER and University of Basel, Switzerland.
[3] Department of Biomedicine, University of Basel, Switzerland

Abstract: Precise regulation of the synaptic proteome is essential for synaptic function and represents a key vulnerability in Alzheimer's disease (AD). Synaptic protein composition is shaped by protein and mRNA transport, local protein synthesis, and post-transcriptional and post-translational regulation. RNA-binding proteins (RBPs) are key post-transcriptional regulators that influence mRNA localisation, stability, and translation. Yet, the RBPs controlling synaptic mRNAs, thereby shaping the synaptic proteome, remain incompletely understood. Using cell-type-specific synaptosome RNA-sequencing, we previously characterised mouse cortical excitatory synapses and identified ADAR2 as a putative regulator of synaptic mRNAs.

ADAR2 catalyses adenosine-to-inosine RNA editing, which can alter RNA function and the amino acid sequence of encoded proteins. A prominent target is Gria2, in which editing at the Q/R and R/G sites regulates AMPA receptor Ca^{2+}-permeability and channel kinetics, respectively. Interestingly, reduced ADAR2 expression and impaired Gria2 editing have been reported in AD brains and proposed to contribute to altered neuronal excitability and circuit dysfunction.

We therefore investigated whether ADAR2 has a broader role in regulating synaptic mRNAs and hypothesised that RNA editing may influence their localisation, as previously shown for Gria2. We cultured neurons on porous membrane inserts, enabling enrichment of somatic and neuritic fractions followed by RNA sequencing. Our analysis recapitulated compartment-specific editing of Gria2 and identified editing in a broader set of synaptic transcripts, including mRNAs encoding the RBP ELAVL3 and the mitochondrial protein MFN1. Using ADAR2 immunofluorescence and fluorescence in situ hybridisation, we detected ADAR2 protein and mRNA within neuronal dendrites, suggesting that ADAR2-mediated RNA editing may also occur locally.

Together, our findings identify ADAR2 as a putative post-transcriptional regulator of synaptic mRNAs and reveal spatial differences in RNA-editing patterns across neuronal compartments. These may arise through compartment-specific editing, selective localisation of edited transcripts, or both, representing an additional layer of synaptic regulation potentially relevant to neuronal circuit dysfunction.

P7: Language screening in Alzheimer's Disease: Preliminary findings from the German adaptation of the DTLA

Fahrni Y.[1, 2], Reinecke R.[3], Macoir J.[4], Lüscher R.[5], Bosch M.[6], Hagmann-von Arx P.[2], Koenig-Bruhin M.[6], Fossard M.[1]

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[4] Université de Laval, Québec
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[6] Luzerner Kantonsspital

INTRODUCTION
Language difficulties can emerge early in Alzheimer's disease (AD) and other neurocognitive disorders, affecting domains like lexical retrieval, linguistic complexity, and comprehension [1], [2]. To assess these language domains, the Detection Test for Language Impairments in Adults and the

Aged (DTLA) is a brief, valid nine-task screening tool, originally developed, validated, and normed in French for adults over 50 years of age [3]. As no German-language equivalent exists, we developed a German adaptation (DTLA-DE).

AIMS
This study aimed (1) to pilot a the DTLA-DE and perform a preliminary item selection in healthy German-speaking adults, and (2) explore its clinical feasibility in a first patient with AD.

METHOD
Subtest structure and item-selection criteria were adapted from the French version. An extended pilot version, containing four times the final number of items, was applied to 74 healthy German-speaking adults aged 50 – 80. The final version was established through systematic item reduction. As a preliminary clinical application, the DTLA-DE was administered to a 74-year-old patient with AD to examine whether it could capture clinically relevant language difficulties.

RESULTS
Adaptation and item selection: Item means and standard deviations were calculated, and a structured expert consensus selected items with high success rates among healthy participants (> 79% correct). The resulting DTLA-DE (37 items across nine subtests) parallels the French DTLA. Clinical case: The patient showed difficulties in naming, lexical fluency, and sentence comprehension, supporting the DTLA-DE's ability to capture language difficulties relevant in AD.

DISCUSSION
The final DTLA-DE comprises a compact item set per subtest, selected for performance variability in healthy participants. A first clinical application in a patient with AD illustrates its practical applicability in a clinical context and its potential to capture relevant language difficulties. Psychometric validation in larger samples and normative data collection are currently underway.

P8: Cognitive spatial maps are less stable across days in an Alzheimer's Disease mouse model

Josef Bitzenhofer[1]*, James M Rowland[1]*, Jonathon Harris[1], Daniel Goodwin[1], Samuel S Harris[1] and Marc Aurel Busche[1,2,3]

*These authors contributed equally.
[1] UK Dementia Research Institute at University College London, London, United Kingdom
[2] Dementia Research Centre, University Hospital of Geriatric Medicine Felix Platter, Basel, Switzerland
[3] Department of Biomedicine, University of Basel, Basel, Switzerland

Amyloid-β pathology is associated with early neural circuit dysfunction in Alzheimer's disease (AD). However, how this pathology affects the stability of cortical representations that are critical for learning and memory remains poorly understood.

To address this question, we trained mice to perform a head-fixed behavioural task in a virtual environment, requiring visuospatial learning and memory, while simultaneously performing two-photon calcium imaging in cortical layers 2/3 of the retrosplenial cortex, a region that serves as an integrator for visuospatial learning and memory and is vulnerable to early amyloid-β accumulation. We recorded from 4-5 month old App NL-G-F mice, as well as age-matched wild-type controls. Both genotypes established cognitive spatial maps, but in App NL-G-F mice, these maps were less stable across days, despite largely preserved within-session coding and no detectable deficit in task acquisition. This dissociation suggests a potential circuit substrate for accelerated long-term forgetting, a phenomenon reported in presymptomatic AD in which initial learning and short-delay recall remain preserved, but memory retention is impaired over longer intervals.

P9: Investigating the interplay between cell-specific autophagy pathway and AQP4-dependent glymphatic clearance in an Alzheimer's disease mouse model

Serena Savoldelli, Ana Isabel Álvarez López, Iben Lundgaard

Glia-Immune interactions, Department of Experimental Medical Science, Faculty of Medicine, Lund University, Lund, Sweden

Alzheimer's disease (AD) is characterised by the aberrant build-up of misfolded amyloid-beta (Aβ) and hyperphosphorylated tau (p-tau) in the form of insoluble extracellular plaques and intracellular neurofibrillary tangles, respectively.
As the accumulation of aggregate-prone proteins has been shown to contribute to neuronal dysfunction and subsequent neuronal loss, preserving the efficiency of cellular mechanisms responsible for protein homeostasis is essential. In this context, macroautophagy, hereafter referred to as autophagy, represents a promising target and a potential mechanism for modulating the cellular burden of potentially toxic protein species, particularly Aβ and p-tau.
Two key autophagy-related proteins, LC3-I/II and p62/SQSTM1, involved in autophagosome formation and selective cargo recognition, respectively, have been shown to undergo adaptive changes in response to amyloid pathology and tauopathy. Astrocytes, which have been shown to exhibit such adaptive changes, are key regulators of the glymphatic system, thus contributing to the extracellular waste clearance through perivascular endfeet enriched in aquaporin-4 (AQP4).
Using both AppNL-G-F knock-in mouse models, which develop progressive amyloid pathology, and AQP4-knockout mice, our research aims to determine whether amyloid pathology alters autophagy in a cell-specific manner and whether these changes are associated with AQP4-dependent glymphatic function. Preliminary quantitative analyses of immunofluorescence staining have revealed cell- and region-specific alterations in p62/SQSTM1, particularly in hippocampal GFAP+- and CD206+-cells, the latter residing along key anatomical routes of the glymphatic system.
Extending this analysis to AQP4-knockout mice will determine whether these amyloid-associated alterations in autophagy-related markers are linked to AQP4-dependent glymphatic modulation.

P10: Beyond Diagnosis: The Caregiver Brain as a Clinical Variable in Dementia Care - A Proposal for The Caregiver Protocol

Andra Bria

University of Bern; "Iuliu Haţieganu" University of Medicine and Pharmacy Romania

Background. Dementia care carries a structural blind spot: at diagnosis, the clinical system hands the day-to-day care environment to an informal caregiver who is neither assessed, monitored, nor recognised as a clinical actor. Yet informal caregivers deliver the majority of dementia care, and the quality of that environment shapes patient course.
Evidence. Peer-reviewed research consistently associates caregiver depression and chronic burden with poorer patient outcomes - including earlier institutionalisation, reduced quality of life, and, in several cohorts, elevated mortality risk. Neuroimaging studies in dementia caregivers document stress-associated structural and functional differences in prefrontal, posterior cingulate, and limbic regions, with intervention studies suggesting these changes are at least partially reversible. The mechanism is dyadic: the brain co-regulates with the nervous systems around it, so a chronically dysregulated caregiver produces a dysregulated care environment. The dyad, not the patient alone, is the functional unit of care.
Proposal. This position paper proposes The Caregiver Protocol - a standardised framework integrating caregiver neurological health as a measured variable across the dementia trajectory: a baseline assessment at diagnosis, structured reviews aligned to clinical follow-up and disease-stage transitions, and longitudinal tracking correlating caregiver wellbeing with patient course. It specifies tiered, risk-proportionate intervention and a distributed "village" care model treating caregiving as systemic rather than individual.
Significance. Romania - high caregiving burden, minimal formal support, a measurable policy gap - serves as a case study, drawing on 70+ structured interviews with family caregivers. The Protocol reframes caregiver neurological health not as a welfare concern but as a primary clinical variable. A

parallel research agenda (observational imaging; dyadic outcome modelling) is proposed to formalise it as a clinical standard.

P11: Bridging the gap in acute geriatric psychiatry: An APN-led implementation plan for the non-pharmacological management of BPSD in a specialised care area (Zone 5).

Célina Heitzmann

Background: Behavioural and psychological symptoms of dementia (BPSD) represent a key challenge in acute geriatric psychiatry. The highly stimulating environment of an acute ward often exacerbates symptoms such as agitation, vocalisation and restlessness. Without structured, non-pharmacological interventions (NPIs), patients face a vicious cycle of pharmacological sedation, coercive measures and functional decline. To address this gap in care, a concept for a milieu-therapeutic care area ('Zone 5') was developed at the University Department for Geriatric Medicine FELIX PLATTER led by an Advanced Practice Nurse (APN).
Methods & Conceptual Framework: The concept establishes an optimized skill-degree mix by integrating a Care Specialist for dedicated daily activation, guided and trained under APN clinical leadership. Key elements include:
1. The DICE Approach: Operationalised as a structured clinical algorithm (Describe, Investigate, Create, Evaluate) to shift staff focus from reactive management to systematic root-cause analysis.
2. Biographical Tooling: Adaptation of the Get-to-Know-Me Board to capture individual preferences, triggers, and identity anchors across three data sources (patient, family, clinical observation).
3. Structured NPIs: Aligning the 6 Dimensions of Care (Paul Schiller Foundation) with sensory and psychosocial interventions (e.g., aromatherapy, tactile activation).
Evaluation Design (Pre-Post Pilot Study): Implementation starts with a 6-month pilot phase following the onboarding of the Care Specialist. The quantitative evaluation design utilises a baseline versus post-implementation analysis:
- Primary Indicators: Analysis of pro re nata (PRN) psychotropic medication management (specifically neuroleptics).
- Secondary Indicators: Audit of frequency and duration of physical restraints.
- Process Quality: Monitoring of de-escalation measures via the DICE approach.

Conclusion: Through the targeted use of a combination of skills, Zone 5 is creating a structured, non-pharmacological 'safe harbour' within acute geriatric psychiatry. The pre-post evaluation will provide valuable insights into how targeted NPIs can be systematically implemented to achieve a reduction in PRN psychotropic medication and coercive measures.

P12: Turning Microglia Neuroprotective to Combat Alzheimer's Disease: An FDA-Approved Compound Upregulates the Neurogenic Factor IGF-1

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Alzheimer's disease (AD), the leading cause of dementia, is expected to affect nearly 140 million people by 2050, creating major economic and healthcare challenges worldwide. Pathologically, it is characterized by amyloid plaques, neurofibrillary tangles, and severe neurodegeneration. This pathology appears quite early in the hippocampus, a region where the generation of new neurons, known as adult hippocampal neurogenesis (AHN), takes place in adulthood. AHN declines sharply in patients during the early stages of AD, preceding cognitive decline. Among the numerous factors influencing AHN, insulin-like growth factor 1 (IGF-1) plays a critical role across multiple stages, including neural stem/progenitor cell (NSPC) proliferation, differentiation, neuronal maturation, and synapse formation. AHN is also modulated by microglia, the resident immune cells of the central

nervous system (CNS). They exhibit diverse phenotypes, one of which is both neuroprotective and neurogenic. Moreover, an enriched environment has been shown to promote AHN in animal models by activating such pro-neurogenic microglia that secrete IGF-1. Therefore, we screened 192 blood-brain-barrier-permeable pharmacological agents approved by the U.S. Food and Drug Administration from a Prestwick library to identify compounds that enhance IGF-1 secretion by microglia, mimicking this pro-neurogenic state. We used BV2 microglia and found that CNS167 consistently increased IGF-1 secretion. Consequently, CNS167-treated microglia show potential to enhance neurogenesis in NSPCs, highlighting it as a promising therapeutic candidate for combating AD and enhancing cognitive function by promoting AHN

P13: Is brief daily self-report a valid way to track symptoms and lifestyle in MCI and early dementia? A pilot study protocol

Tommaso Albizzati

Dr. Nexus

In mild cognitive impairment (MCI) and early dementia, cognition, mood and everyday functioning fluctuate within individuals from day to day, whereas clinical assessment relies on single visits separated by weeks or months. Modifiable lifestyle factors, including physical activity, sleep, and social engagement, are associated with cognitive decline, but their day-to-day relationship with symptoms at the individual level remains poorly characterised. Self-monitoring may be burdensome, particularly in this target population. Daily smartphone assessment in MCI has mainly addressed feasibility, and the validity of daily self-reported symptoms against established clinical instruments remains largely untested. The aim of this pilot is to assess the concurrent validity of brief daily self-reported symptom items against validated clinical instruments. We propose an observational study without intervention (n ≈ 30; duration ≈ 60 days). Participants will complete a 2–3 minute daily smartphone questionnaire on symptoms and lifestyle and will wear a consumer wearable recording steps, active time and sleep. Clinically validated, self-administered tools will be completed at longer intervals. The primary analysis will assess the agreement between the mean of the daily items preceding each assessment and the validated score. Exploratory analyses will examine within-person associations between lifestyle, wearable-derived measures and daily symptoms. Because reduced self-awareness may affect the accuracy of self-report, we will also test whether agreement with the validated scores differs between MCI and mild dementia and according to clinician-rated awareness of cognitive difficulties. The protocol is in preparation for ethics review; no data have been collected. The study will provide initial evidence on the feasibility and concurrent validity of daily self-monitoring in MCI and early dementia, a prerequisite for personalised lifestyle–symptom analyses between clinic visits. It will represent the first step in the validation of a platform intended for commercial development.

P14: Cognitive-Metabolic Decoupling as a Quantitative Trait of Resilience in Alzheimer's Disease Progression

Dany Mukesha

Introduction:
Alzheimer's disease (AD) progression is typically described using categorical clinical stages, despite substantial inter-individual variability in the relationship between biological pathology and cognitive decline. What remains insufficiently understood is why some individuals maintain stable cognition despite high biological burden (resilience), whereas others deteriorate rapidly with comparatively modest pathology. Here, we introduce Cognitive–Metabolic Decoupling (CMD) as a continuous, quantitative trait capturing this cognitive–biological asynchrony and evaluate its prognostic relevance for dementia progression.

Methods:

Data from 798 participants in the Alzheimer's Disease Neuroimaging Initiative (192 cognitively normal, 454 mild cognitive impairment [MCI], 152 AD dementia) were analyzed. A Metabolic Dysregulation Score (MDS) was derived using elastic net regression ($\alpha = 0.5$) integrating FDG-PET and core cerebrospinal fluid biomarkers (Aβ42, total tau, p-tau181) to predict a standardized cognitive composite (MMSE and inverted ADAS-Cog13). CMD was defined as the residual cognitive variance after adjustment for MDS, hippocampal volume normalized by intracranial volume, age, and sex. Cox proportional hazards models assessed associations between CMD and time to dementia conversion, with added prognostic value evaluated using likelihood ratio tests, concordance indices, and time-dependent ROC analysis.

Results:
MDS explained 47% of cognitive variability and performed comparably to joint multimarker models with fewer parameters. CMD captured 52% of residual cognitive variance and was mathematically independent of hippocampal atrophy ($r \approx 0$). Higher CMD was strongly associated with a reduced risk of dementia conversion (hazard ratio per SD=0.48; 95% CI 0.41–0.57; $p<10^{-18}$), achieving a 2-year AUC of 0.77. Adding CMD to models containing FDG-PET, amyloid, tau, age, and sex significantly improved prognostic performance ($\Delta\chi^2=80.5$; concordance index 0.73→0.82). Among individuals with MCI, CMD stratified participants into clinically distinct trajectories, identifying a subgroup with delayed progression despite comparable biological burden. No significant association was observed in cognitively normal individuals, consistent with limited event rates.

Conclusions:
CMD provides a robust, interpretable measure of cognitive resilience and vulnerability that substantially augments classical AD biomarkers. This framework reframes disease progression as a continuous divergence between biological and cognitive aging, enabling improved risk stratification and trial enrichment. Future work will validate CMD in independent cohorts and extend the approach to blood-based biomarkers and longitudinal CMD trajectories to support precision medicine in AD. Although demonstrated in Alzheimer's disease, the CMD computation framework is disease-agnostic and applicable to any setting involving partial decoupling between molecular measurements (multimodal biological data) and phenotypic outcomes.

P15: APN-Led Consultation Services for Delirium and BPSD: Translating Evidence into Clinical Practice

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Background

Delirium and behavioural and psychological symptoms of dementia (BPSD) are common and challenging clinical presentations in older adults. Multicomponent non-pharmacological interventions (NPIs) play a key role to evidence-based delirium care, while person-centred NPIs are recommended as first-line approaches for BPSD. Differentiating delirium from dementia-related symptoms can be challenging, particularly in people with pre-existing dementia. APN-led consultation services provide an opportunity to translate evidence into clinical practice.

Aim

To describe the implementation and evaluation of two APN-led consultation services for delirium and BPSD, focusing on APN collaboration, staff empowerment, and evidence-based non-pharmacological care.

Methods

As part of a pre-implementation needs assessment, staff working on geriatric inpatient wards were surveyed to assess their existing knowledge and identify their needs regarding delirium and BPSD. These findings were incorporated into the development and direction of the consultation services.

Two APNs with complementary expertise in delirium and dementia/BPSD provide case-based assessment, consultation, coaching, and bedside teaching. Collaboration is particularly important when symptoms overlap, or differentiation is uncertain.

On open wards, APNs support staff in identifying causes and triggers of behavioural changes and selecting tailored NPIs. Case-based coaching and bedside teaching aim to empower staff to manage complex situations and promote person-centred, least-restrictive care.

Evaluation and Future Perspectives

Over one year, the services will be evaluated through informal feedback and brief structured staff surveys following consultations and bedside teaching. Feedback will guide iterative adaptation of content and format to staff and clinical needs and inform future evaluation, including potential patient-related outcomes.

Conclusion

APN-led consultation services combine specialist expertise, collaboration, and bedside coaching to translate evidence into practice. A needs-based, iterative approach may empower staff to manage complex presentations and strengthen person-centred, non-pharmacological care for older patients with delirium and BPSD.

P16: Tracking Within-Person Cognitive Decline Through Automated Speech Analysis: Retrospective Validation and a Planned Clinical Feasibility Study

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ANDSpeak

In routine clinical care, cognitive status is evaluated intermittently using brief batteries such as the MoCA or MMSE, which are susceptible to practice effects and day-to-day variability. While speech analysis offers a non-invasive digital biomarker, most existing algorithms rely on cross-sectional datasets that conflate disease progression with baseline individual differences. Clinical monitoring requires markers sensitive to longitudinal within-person change.

To evaluate whether automated speech features can indicate cognitive decline over time rather than simply reveal variability in recordings, we analyzed retrospective speech corpora from public figures who subsequently disclosed an Alzheimer's Disease (AD) diagnosis. In a multi-subject benchmark of 80 age- and sex-matched individuals (40 AD, 40 controls) spanning up to 10 years before diagnosis, multimodal fusion of acoustic and linguistic features achieved classification accuracies of 0.72 in the 6–10 year pre-diagnosis window and 0.80 in the 1–5 year pre-diagnosis window. Early pre-diagnosis windows were driven primarily by linguistic shifts, whereas acoustic markers increasingly contributed closer to diagnosis. Single-subject multi-decade tracking (e.g., a 30-year longitudinal profile of Ronald Reagan, ages 53–80) further confirmed that composite biomarker scores cross clinical impairment thresholds before formal diagnosis.

While these retrospective findings demonstrate that multimodal speech modeling can capture prodromal decline years before clinical presentation, translating these algorithms into routine care requires prospective evaluation in real-world settings. To this end, we have developed an integrated platform centered on Story of My Life, an application that elicits autobiographical narratives through adaptive, synchronized audio-visual prompts. We present the system architecture and the protocol for a six-month clinical feasibility study in 30–40 patients with mild cognitive impairment or early dementia, designed to establish adherence, test–retest reliability, and concordance with standard neuropsychological assessments.

P17: Nanoscale mapping of presynaptic molecular architecture during ageing

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Synapses have a highly complex and organised nanoscale molecular architecture. Although synaptic dysfunction is an early feature of cognitive decline, how the molecular composition and organisation of individual synapses changes during physiological ageing remains poorly understood. Here, we investigate age-related changes in presynaptic proteins essential for vesicle trafficking and neurotransmitter release. Using expansion microscopy (~3.5-fold expansion), coupled with cyclic immunofluorescence (ExM-CycIF) on mouse motor cortex across three age groups, we multiplexed presynaptic markers (αSyn, VAMP2, SV2A, Syt1) alongside excitatory postsynaptic targets (GluN1, PSD95) to map molecular diversity within discrete trans-synaptic nanodomains. Ageing was associated with a redistribution towards low-complexity synapses, marked by an increased proportion of αSyn-only terminals. We also identified a reduction in high-complexity, multi-marker nanoclusters, accompanied by reduced co-localisation of αSyn with key regulators of synaptic vesicle exocytosis, SV2A, Syt1 and VAMP2, in aged animals. Together, these findings suggest an age-related reorganisation of presynaptic molecular composition that may impair neurotransmitter release, and set the stage for interventions to preserve synaptic function in ageing.

P18: A human, seed-free cell model reproducing the ordered Tau pathology cascade: a translational tool for the dementia field

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Background. Tau pathology tracks closely with cognitive decline in Alzheimer's disease (AD) and defines primary tauopathies. Yet the field lacks human models that reproduce how Tau pathology develops from phosphorylation, misfolding and oligomerisation to fibril formation, without artificial triggers such as truncated fragments, seeds, or tags. This limits mechanistic understanding of disease staging and the preclinical testing of therapeutics.

Model. We generated a stable, doxycycline-inducible human H4 neuroglioma cell line expressing untagged 1N4R Tau carrying the pro-aggregant P301L/S320F double mutation. To map the temporal progression of tauopathy and assess aggregate clearance, cells were subjected to a 4-day induction phase followed by transgene silencing. The pathological cascade was tracked using untargeted phosphoproteomics, immunocytochemistry, biochemical fractionation, and cellular viability assays.

Results. Upon induction, the double mutant drove spontaneous, seed-free aggregation in a strictly defined kinetic order. Phosphoproteomics identified 29 Tau phosphosites, 90% overlapping those reported in the human AD brain. Disease-associated modifications followed a staggered temporal hierarchy: Phospho-Tau217 (pT217)—an emerging blood biomarker for AD—rose within 24 hours alongside pathological conformational changes and soluble oligomers. This early phase coincided with metabolic dysfunction and apoptosis. Subsequently, the classical aggregation marker AT8 accumulated, culminating by day four in the appearance of the late-stage aggregate marker pS422 and mature β-sheet-rich, paired-helical-filament-like fibrils in 10–15% of cells. This sequence supports the view that soluble oligomers, rather than mature fibrils, drive early toxicity. Upon transgene silencing, established pathology was fully reversible. In proof-of-concept experiments, a Tau aggregation inhibitor (BSc3094) and an autophagy inducer (SMER28) each successfully prevented aggregation and preserved viability.

Conclusion. This inducible human model reproduces the ordered Tau cascade with high molecular fidelity to human disease in a seed-free, tag-free, reversible system. It is a research tool that offers a human-relevant reproducible platform for screening therapeutics.

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