SUPPLEMENT 6

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9th Hong Kong Neurological Congress cum 38th Annual Scientific Meeting of The Hong Kong Neurological Society

1 – 2 November 2025

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Scientific Programme

Venue: Grand Ballroom, Level 3, JW Marriott Hotel, Admiralty, Hong Kong SAR

1 November 2025, Saturday

08:30 – 09:40	**Dissertation Highlights** Panel Judges: *Dr Herrick LAU, Dr WK CHENG* **Application of local field potential in managing Parkinson's disease patients with deep brain stimulation** *Chung Yuen Tom CHAN* **Clinical profiles, disability, psychiatric comorbidities and predictors of subcutaneous calcitonin gene–related peptide monoclonal antibody response in Hong Kong migraineurs: A single-centre study** *Tsz Fung Warren CHEUNG* **Retrospective study on clinical and genetic analysis of hereditary neuropathies in a single genetic centre in Hong Kong** *Hon Wing CHEUNG* **Safety of recanalisation therapy for acute ischaemic stroke in cancer patients: A local multicentre retrospective cohort study in Hong Kong** *Cheuk Fung HAN* **Intracranial carotid artery calcification as an imaging marker for intracranial angiographic features and ischaemic stroke outcome: A retrospective case-control study** *Pui Hung HO* **Functional outcomes of mechanical thrombectomy in acute stroke patients** *Andrea LEE*
09:40 – 09:50	Break/Air time
09:50 – 10:35	**Education Session** Chairpersons: *Dr Eric CHAN, Dr Shirley CHEUNG* **Parry–Romberg syndrome** *Shi Hon NG, Shirley CHEUNG*
10:35 – 11:20	**TauRx Dementia Symposium** (co-organised with Hong Kong Movement Disorder Society) Chairpersons: *Dr Carlin CHANG, Dr Shirley CHEUNG* **Beneficial clinical, imaging and blood biomarker outcomes in a randomised phase 3 clinical trial of tau aggregation inhibitor hydromethylthionine mesylate in early to moderate Alzheimer's disease** *Claude M WISCHIK*
11:20 – 12:05	**Movement Disorders Symposium** (co-organised with Hong Kong Movement Disorder Society) Chairpersons: *Dr Germaine CHAN, Dr Michael LEE* **Rewiring Parkinson's disease: A successful story through neuromodulation** *Elena MORO*

1 November 2025, Saturday

12:05 – 12:20	**Opening Ceremony** Guest of Honour: *Prof Elena MORO* President, European Academy of Neurology
12:20 – 13:20	**Lunch Symposium on Parkinson's Disease** (sponsored by Eisai) (co-organised with Hong Kong Movement Disorder Society) Chairpersons: *Dr Helen YIP, Dr TL POON* **Beyond dopamine: Leveraging dual mechanism for holistic Parkinson's disease management—evidence from real-world practice and expert consensus** *Fabrizio STOCCHI*
13:20 – 14:05	**Lundbeck Symposium on Neurological Disorders Depression** Chairpersons: *Dr Charing SZETO, Dr June WONG* **Depression management in neurological disorders** *Ming Cheuk Michael WONG*
14:05 – 14:50	**Eisai Dementia Symposium** Chairpersons: *Dr Noble KWAN, Dr Colin LUI* **Alzheimer's disease: A chronic journey requiring early and continuous intervention** *Takahito YOSHIZAKI*
14:50 – 15:10	Break/Air time
15:10 – 15:55	**Eli Lilly Dementia Symposium** Chairpersons: *Dr Noble KWAN, Dr Colin LUI* **The impact of current diagnostic and treatment advancements on the lives of patients with Alzheimer's disease** *Colin J MAHONEY*

2 November 2025, Sunday

08:30 – 09:40	**Free Paper Presentation** Free Paper Panel Judges: *Dr Nelson CHEUNG, Dr Richard LI* **Incidence, treatment metrics and outcomes of intracerebral haemorrhage in Hong Kong: A 10-year territory-wide study** *Bianca CHAN, Bella WONG, Bono LI, Trista HUNG, Thomas LEUNG, Bonaventure IP* **Oral anticoagulation after spontaneous intracerebral haemorrhage in patients with non-valvular atrial fibrillation** *Nicholas FOK* **Effect of mirror therapy on upper limb function in stroke patients: A retrospective study** *Wing Chong LO* **Radiological determinants of tissue fate in plain brain computed tomography during acute large vessel occlusion: A multicentre study** *Billy LAM, Bianca CHAN, Sze Ho MA, Thomas LEUNG, Haipeng LI, Bonaventure IP* **Characterising LRRK2 Asian missense mutations in the context of Parkinson's disease** *Manyu WANG* **Role of stroke team pharmacists in identifying drug-related problems in a local public hospital** *SK YAU* **Exploring the intersection of ageing, obesity, and glucagon-like peptide-1 receptor signalling in Alzheimer's disease** *Xianyi ZHENG, Mengran XIONG, Leo YC YAN, Danny CW CHAN, Lai Shan YUEN, Panwu ZHAO, Ho KO, Junzhe HUANG*
09:40 – 09:50	Break/Air time
09:50 – 10:35	**Medison Pharma Symposium on Amyloid Transthyretin Amyloidosis** Chairpersons: *Dr WT WONG, Dr Germaine CHAN* **Unmasking and managing transthyretin amyloidosis** *Matt SILSBY*
10:35 – 11:20	**Novartis Multiple Sclerosis Symposium** (co-organised with Hong Kong Multiple Sclerosis Society) Chairpersons: *Dr Jessica LI, Dr Stephen CHENG* **Smouldering-associated worsening in multiple sclerosis and the importance of using high efficacy treatment in early multiple sclerosis** *Mar TINTORE*
11:20 – 12:05	**Eisai Multiple Sclerosis Symposium** (co-organised with Hong Kong Multiple Sclerosis Society) Chairpersons: *Dr WK CHENG, Dr KL SHIU* **Personalising multiple sclerosis therapy: Guidelines, treatment selection and risk mitigation** *Kazuo FUJIHARA*

2 November 2025, Sunday

12:05 – 13:05	**Lunch Symposium on Stroke** (sponsored by Boehringer Ingelheim) Chairpersons: *Dr Richard LI, Dr Bonaventure IP* **The changing landscape of thrombolysis in acute ischaemic stroke management** *Keith MUIR*
13:05 – 13:50	**Neuromuscular Disease Symposium** Chairpersons: *Dr June WONG, Dr Noble KWAN* **Expanding aetiologies immunological mechanisms and diagnostic approaches in small fibre neuropathy** *Amanda CHAN*
13:50 – 14:35	**Teva Migraine Symposium** Chairpersons: *Dr Yannie SOO, Dr Carlin CHANG* **Innovating migraine care: Unlocking the full potential of calcitonin gene–related peptide pathway therapies** *Piero BARBANTI*
14:35 – 14:55	Break/Air time
14:55 – 15:40	**AbbVie Migraine Symposium** Chairpersons: *Dr Yannie SOO, Dr Carlin CHANG* **Mastering migraine therapies** *William KINGSTON*
15:40 – 16:25	**GSK Shingles Symposium** Chairpersons: *Dr Gardian FONG, Dr Yannie SOO* **Zoster vaccination: A neurologist's tool for zoster and stroke prevention** *Kay Cheong TEO*
16:25 – 16:35	Closing Ceremony

DH 1

Application of local field potential in managing Parkinson's disease patients with deep brain stimulation

Chung Yuen Tom CHAN
Department of Medicine, Queen Elizabeth Hospital, Hong Kong SAR, China

Background: Deep brain stimulation (DBS) is a well-established treatment for patients with advanced Parkinson's disease (PD). Optimal DBS programming is a crucial factor in achieving effective symptom control. Local field potential (LFP) is an emerging biomarker for both initial DBS programming and subsequent symptom monitoring. Commercially available DBS devices enable the recording of LFP and the delivery of stimulation simultaneously.
Objectives: To evaluate the applicability of LFP as an objective biomarker in the management of patients with advanced PD undergoing DBS.
Methods: This was a single-centre, retrospective observational cohort study conducted at Queen Elizabeth Hospital, Hong Kong. PD patients implanted with the Medtronic Percept PC device were recruited. LFP data were collected during the initial programming and the characteristics of LFP were reviewed. Contact selections for initial programming using LFP-guided, image-guided, and clinical programming techniques were compared. Characteristics of LFP during subsequent follow-up visits were correlated and analysed.
Results: A total of 32 patients who underwent DBS were included in this study. The mean age at DBS was 66.0 years. Thirty (93.8%) patients were treated with bilateral subthalamic nucleus DBS and two patients were treated with bilateral globus pallidus internus DBS. A beta peak was present in 81.7% of the contact pairs, and 92.2% of them demonstrated at least one alpha-beta peak. A positive correlation in contact selection was demonstrated among LFP-guided, image-guided, and clinical programming. Various LFP frequencies identified had their specific characteristics. The beta peak correlated with the OFF state, while the alpha peak was associated with tremor.
Conclusions: LFP is a useful biomarker in the management of PD patients with DBS. It provides an effective and efficient programming compared to conventional programming, meanwhile reflecting the disease control to allow optimal patient management.

DH 2

Clinical profiles, disability, psychiatric comorbidities and predictors of subcutaneous calcitonin gene–related peptide monoclonal antibody response in Hong Kong migraineurs: A single-centre study

Tsz Fung Warren CHEUNG
Department of Medicine and Geriatrics, United Christian Hospital, Hong Kong SAR, China

Background: Migraine ranks as the third leading cause of disability among neurological disorders globally, affecting an estimated 12.5% of Hong Kong's population. While calcitonin gene–related peptide (CGRP) monoclonal antibodies (mAbs) provide targeted migraine prevention, landmark studies indicate an inadequate response in more than one-third of patients. Understanding factors influencing treatment outcomes, particularly psychiatric comorbidities, is crucial for optimising care. In this study, we aim to (1) characterise clinical profiles, disability burden, and psychiatric comorbidities in Hong Kong migraine patients; (2) evaluate effectiveness and tolerability of three subcutaneous CGRP mAbs (erenumab, galcanezumab, fremanezumab); and (3) identify predictors of treatment response.

Methods: This is a retrospective, single-centre study conducted at the Headache and Facial Pain Clinic and Neurology Clinic at United Christian Hospital in Hong Kong from July 2011 to January 2025. Clinical data from migraine patients were retrieved from electronic medical records. A subset of patients completed questionnaires assessing disability (Headache Impact Test-6, Migraine Disability Assessment) and psychiatric symptoms (Hospital Anxiety and Depression Scale, Beck Depression Inventory-II). Treatment response to CGRP mAbs was defined as a ≥50% reduction in monthly migraine headache days. Side effects and outcomes after switching between CGRP mAbs were documented. Statistical analyses were conducted to compare chronic migraine (CM) and episodic migraine (EM) patients across all clinical parameters and quantify the prevalence of psychiatric illness among migraineurs. Multivariate logistic regression was employed to identify predictors of treatment response.

Results: A total of 592 migraine patients were identified. Of these, 163 (27.5%) had CM, who demonstrated significantly higher disability scores and psychiatric symptom severity, compared to EM patients. Nearly one-fifth (19.6%) of patients had documented psychiatric diagnoses, with depressive disorder being the most prevalent. A subset of 222 patients completed questionnaire assessments. Among patients without known psychiatric diagnoses, 34% met the screening criteria for probable depression or anxiety, highlighting a significant treatment gap. For 109 patient-episodes of CGRP mAb use, the overall treatment response rate was 67%, with no significant efficacy differences between the three medications. Constipation was the most common side effect, particularly among erenumab users. All eight patient-episodes that experienced significant side-effects had improved tolerability after switching to another CGRP mAb, while seven out of eleven patient-episodes experienced improved clinical response after switching. Multivariate analysis identified triptan responsiveness (odds ratio=5.14, 95% confidence interval=1.09-24.27) as a positive predictor, while higher baseline monthly headache days (odds ratio=0.87, 95% confidence interval=0.78-0.97) served as a negative predictor of CGRP mAb response. Additionally, Chi squared analysis revealed that the presence of medication overuse headache, underlying psychiatric illness, and a higher number of previously failed preventives were significantly associated with non-responsiveness to CGRP mAbs.

Conclusion: This study provides comprehensive data on Hong Kong Chinese migraine patients, highlighting significant interactions between migraine chronicity, disability, and psychiatric comorbidities. CGRP mAbs demonstrated effective preventive efficacy, with triptan responsiveness serving as a key predictor of treatment success. Switching between CGRP mAbs may improve both clinical response and tolerability. Psychiatric co-morbidity is associated with treatment failure; and notably, underdiagnosis is prevalent in this population. Early diagnosis, and management of psychiatric conditions and medication overuse, combined with multidisciplinary approaches, are essential for optimising outcomes for migraine patients.

DH 3

Retrospective study on clinical and genetic analysis of hereditary neuropathies in a single genetic centre in Hong Kong

Hon Wing CHEUNG
Department of Medicine and Geriatrics, United Christian Hospital, Hong Kong SAR, China

Background and Objectives: This study aims to explore the genetic epidemiology of hereditary neuropathies in Hong Kong and compare them to those in nearby regions, identify novel pathogenic variants within the cohort if any, and analyse the clinical features of prevalent and novel mutations.
Methods: The cohort included 163 patients with confirmed genetic diagnoses of hereditary neuropathy from the Clinical Genetics Service Unit of the Hong Kong Children's Hospital and the now-closed Clinical Genetic Service of the Department of Health, which had transferred its services to Hong Kong Children's Hospital. These 163 patients included 128 primary patients who were referred to the genetic centre due to clinical suspicion of a hereditary neuropathy, and 35 individuals identified through family screening.
Results: The gene most commonly identified with a mutation causing hereditary neuropathies in this cohort was GJB1 (41.1%), and the most prevalent variant was c.-103C>T in the 5'untranslated region, which was identified in 52% of patients with a GJB1 mutation. PMP22 mutations were the second most common (28%), predominantly duplications (87% of all PMP22 mutations). Novel pathogenic variants of SH3TC2, GJB1, and MPZ genes were identified. Nerve conduction study findings indicated that neuropathies caused by PMP22 duplications predominantly exhibited demyelinating features, whereas GJB1-related neuropathies displayed mixed and indeterminate patterns, consistent with the literature.
Conclusion: This study suggests a high prevalence of GJB1 mutations, possibly higher than that of PMP22 mutations, in Hong Kong, which may contrast with nearby regions where PMP22 mutations account for the largest proportion of hereditary neuropathies. In view of the high prevalence of GJB1 pathogenic variants and PMP22 duplications, either GJB1 or PMP22, or both, might be prioritised for testing in suspected hereditary neuropathies, and the decision depends on the nerve conduction study findings and the observed inheritance pattern.

DH 4

Safety of recanalisation therapy for acute ischaemic stroke in cancer patients: A local multicentre retrospective cohort study in Hong Kong

Cheuk Fung HAN
Department of Medicine, Pamela Youde Nethersole Eastern Hospital, Hong Kong SAR, China

Introduction: The association between malignancy and ischaemic stroke has been increasingly recognised. The safety of recanalisation therapy in cancer patients with acute ischaemic stroke (AIS) remains uncertain.
Purpose: This study evaluated the outcomes following recanalisation therapy for AIS in patients with active cancer (AC).
Methods: This was a multicentre retrospective cohort study that reviewed patients presenting with AIS who received intravenous thrombolysis and/or mechanical thrombectomy between 2019 and 2021. Subjects were categorised into AC and non-cancer (NC) groups. AC was defined as a cancer diagnosis or receipt of cancer treatment within 12 months of the index event. Outcomes including intracranial haemorrhage (ICH), non-ICH systemic bleeding, mortality and functional independence at 3 months, were compared.
Results: 1282 patients were analysed, with 1206 (94.1%) in the NC group and 76 (5.9%) in the AC group. There was no statistically significant difference in the incidence of symptomatic ICH (odds ratio [OR]=0.64, 95% confidence interval [95% CI]=0.20-2.07, P=0.618), asymptomatic ICH (OR=1.23, 95% CI=0.63-2.38, P=0.541), or non-ICH systemic bleeding (OR=2.83, 95% CI=0.82-9.78, P=0.112). Three-month all-cause mortality was higher in the AC group (OR=2.18, 95% CI=1.31-3.61, P=0.002), but none were direct consequences of recanalisation therapy. The rate of 3-month functional independence was significantly lower in the AC group (OR=0.60, 95% CI=0.37-0.99, P=0.042) in univariate analysis, but this was not significant after adjusting for covariates.
Conclusion: Recanalisation therapy was safe in cancer patients who suffered from acute ischaemic stroke.

DH 5

Intracranial carotid artery calcification as an imaging marker for intracranial angiographic features and ischaemic stroke outcome: A retrospective case-control study

Pui Hung HO
Department of Medicine, Pamela Youde Nethersole Eastern Hospital, Hong Kong SAR, China

Background: Intracranial carotid artery calcification (ICAC) refers to calcification located in the cavernous or supraclinoid segment of the internal carotid artery, which is identifiable on plain computed tomography of the brain. ICAC has been identified as a risk factor for stroke, but the limited studies on its impact on angiographic features and stroke outcomes reported inconsistent results. This study evaluated the association of ICAC with co-existing intracranial atherosclerotic disease, collateral status, ischaemic stroke outcomes, and parameters in endovascular thrombectomy.
Methods: Anterior circulation ischaemic stroke patients from July 2021 to June 2023 who underwent angiography were retrospectively reviewed. Eligible patients were categorised into ICAC and no-ICAC groups for comparison. Subgroup analyses based on ICAC severity and calcification pattern were also performed.
Results: A total of 193 patients with ICAC (65.6%) and 101 patients without ICAC (34.4%) were identified and analysed. After adjustment for confounding factors, patients with more severe ICAC were significantly associated with co-existing intracranial atherosclerotic disease (odds ratio [OR]=2.71, P=0.035). A trend towards worse collateral status in patients with more severe ICAC was also observed in binomial logistic regression, although did not reach statistical significance (OR=0.52, P=0.146). The presence of ICAC was associated with worse 90-day functional outcomes: functional independence (OR=0.46, P=0.042), excellent neurological outcome (OR=0.36, P=0.007), ordinal shift in modified Rankin Scale (OR=2.22, P=0.003). For ICAC patients who underwent endovascular thrombectomy, the intimal calcification pattern was linked to higher rates of successful recanalisation (P=0.036).
Conclusion: ICAC may serve as an imaging marker for angiographic abnormalities and poor functional outcomes in ischaemic stroke patients.

DH 6

Functional outcomes of mechanical thrombectomy in acute stroke patients

Andrea LEE
Department of Medicine and Geriatrics, Princess Margaret Hospital, Hong Kong SAR, China

Background: Stroke is a leading cause of disability and death worldwide, with ischaemic stroke accounting for 70% of cases. Endovascular thrombectomy (EVT) is an effective treatment for acute ischaemic stroke (AIS) caused by large vessel occlusion. While EVT improves functional outcomes, variability in results highlights the need to identify factors influencing success. This study evaluates clinical, radiographic, and operational predictors of functional independence in real-world settings.
Methods: This retrospective study included 94 AIS patients with anterior circulation large vessel occlusion who underwent EVT in the Kowloon West Cluster from August 2023 to September 2024. Baseline demographics, operational metrics, and stroke characteristics were analysed. The primary outcome was functional independence at 90 days, defined as a Modified Rankin Scale (mRS) score of 0 to 2 or no worsening from the premorbid mRS. Multivariate logistic regression was used to identify predictors of favourable outcomes.
Results: Good functional outcomes were observed in 34% of patients. Key predictors included better premorbid mRS (odds ratio [OR]=0.36, P=0.005), cardioembolic stroke (OR=3.30, P=0.057) and larger mismatch volume (OR=1.01, P=0.039). Poor outcomes were associated with diabetes (OR=0.18, P=0.028) and post-thrombectomy intracerebral haemorrhage (OR=0.21, P=0.036). Operational inefficiencies, such as prolonged workflow times, were also identified.
Conclusions: Patient selection and efficient operational workflows are critical to optimising EVT outcomes. Key predictors of success include better premorbid status, cardioembolic stroke, and timely intervention, while diabetes and postprocedural intracerebral haemorrhage are adverse factors. Systematic improvements in prehospital coordination and imaging protocols are essential to enhance EVT efficacy. Further research in larger cohorts is warranted.

S 1

TauRx Dementia Symposium (co-organised with Hong Kong Movement Disorder Society)

Beneficial clinical, imaging and blood biomarker outcomes in a randomised phase 3 clinical trial of tau aggregation inhibitor hydromethylthionine mesylate in early to moderate Alzheimer's disease

Claude M WISCHIK[1,2]
[1] School of Medicine, Medical Sciences and Nutrition, University of Aberdeen, Scotland, United Kingdom
[2] Executive Chairman, TauRx Pharmaceuticals, Aberdeen, United Kingdom

Hydromethylthionine mesylate (HMTM) is an orally administered tau aggregation inhibitor that also exhibits tau-independent symptomatic activity. In the phase 3 trial TRx-237-039, participants with early-to-mild/moderate Alzheimer's disease (AD) received either HMTM 16 mg/day or a low dose of methylthioninium chloride (MTC, which also delivers hydromethylthionine) administered as 4 mg twice weekly to maintain blinding due to potential urinary discolouration. HMTM produced a statistically significant improvement over baseline in cognitive function which was sustained to 18 months with no significant decline at 24 months in patients with early AD. In contrast, those receiving MTC demonstrated a similar initial improvement but declined significantly at 18 and 24 months, with statistically significant differences relative to MTC at 18 (P=0.0291) and 24 months in early AD (P=0.0308). Statistical parametric mapping of serial magnetic resonance imaging revealed significant treatment differences between MTC and HMTM in grey matter atrophy in the hippocampus, temporal lobe, and cingulate cortex at 12 and 24 months. These effects were consistent with significant treatment differences in plasma biomarkers measuring neurodegeneration (neurofilament light chain) and tau aggregation pathology (phosphorylated tau 217), confirming HMTM treatment effects on underlying tau aggregation pathology and neurodegeneration. Since initial symptomatic activity in the MTC arm confounded assessment of the clinical efficacy of HMTM at the 12-month primary endpoint, a confirmatory study (protocol TRx-237-080) compared outcomes in participants receiving HMTM 16 mg/day over 12, 18 and 24 months with propensity score–matched placebo controls from the Critical Path in AD database with the same inclusion/exclusion criteria. Matched participants from the Alzheimer's Disease Neuroimaging Initiative and meta-analytic placebo data from published trials in comparable populations were also compared. HMTM 16 mg/day significantly reduced cognitive and functional decline at all three timepoints relative to case-matched and meta-analytic external controls. There was also a significant reduction in the progression of brain atrophy relative to Critical Path in AD and Alzheimer's Disease Neuroimaging Initiative controls and to the meta-analysis of placebo arms in trials of recently approved anti-amyloid treatments. HMTM was generally well tolerated: headache (1.5%) and diarrhoea (1.2%) were the most frequent adverse effects and there was no risk of treatment-induced amyloid-related imaging abnormalities. HMTM is an oral and convenient potential new treatment for AD which combines symptomatic and disease-modifying therapeutic activity. This dual pharmacology complicates the demonstration of clinical efficacy within a conventional placebo-controlled design whilst maintaining blinding. Nevertheless, consistent treatment benefit has been demonstrated on biological outcomes and relative to matched external controls.

S 2

Movement Disorders Symposium (co-organised with Hong Kong Movement Disorder Society)

Rewiring Parkinson's disease: A successful story through neuromodulation

Elena MORO
Professor of Neurology, Grenoble Aples University, France

Parkinson's disease (PD) is a progressive disabling neurodegenerative disease. Technological advances have allowed a better understanding of PD pathophysiology and optimised the management of motor and non-motor symptoms. Among neuromodulation techniques, deep brain stimulation (DBS) of the basal ganglia is among the first-line treatments for advanced PD and other hyperkinetic movement disorders not well managed with medication. However, despite 35 years of experience with DBS, there are still some issues that need to be optimised with this technique. These issues span from the selection to the postsurgical management of patients. Recent advances in the DBS field support earlier surgery and have identified predictive factors of DBS benefit. Moreover, new technologies have allowed an easier targeting and programming of the stimulators. Additionally, adaptive DBS might help in tuning stimulation according to symptoms and the real needs of patients. Combined strategies (neuroimaging, wearable devices, machine learning) might improve and expand this still-growing area.

S 3

Lunch Symposium on Parkinson's Disease (sponsored by Eisai, co-organised with Hong Kong Movement Disorder Society)

Beyond dopamine: Leveraging dual mechanism for holistic Parkinson's disease management—evidence from real-world practice and expert consensus

Fabrizio STOCCHI
Professor of Neurology, University and Institute for Research and Medical Care, San Raffaele, Roma, Italy

Symptoms of Parkinson's disease are not due to dopamine dysfunction only. Other systems are involved especially glutamatergic system. Targeting both dopamine and glutamate pathways implicated in the pathophysiology of Parkinson's disease enables a holistic approach to managing motor symptoms and addressing various non-motor benefits, such as sleep, pain, and depression. The early use of a third-generation monoamine oxidase B inhibitor has demonstrated promise as an adjunctive therapy to low-dose levodopa, offering several advantages over other dopaminergic drugs. In line with the European Delphi consensus, early intervention with this dual-targeting monoamine oxidase B inhibitor at higher dosage can reduce the risk of motor complications like dyskinesia, making it a valuable option for diverse patient populations, including older adults. Prof Fabrizio Stocchi will share insights from his real-world experience, providing compelling evidence to support his choice of adjunct to levodopa. This presentation aims to equip healthcare professionals with a deeper understanding of optimising early Parkinson's disease management, ultimately enhancing patient outcomes and quality of life.

S 4

Lundbeck Symposium on Neurological Disorders Depression

Depression management in neurological disorders

Ming Cheuk Michael WONG
Private Practice, Hong Kong SAR, China

This presentation focuses on the complexities of managing major depressive disorder in patients with concurrent neurological conditions, such as Alzheimer's and Parkinson's diseases. It emphasises the clinical heterogeneity of depression and the need for tailored treatment strategies that consider individual patient profiles and comorbidities. The presentation will review the evolution of treatment approaches, highlighting the importance of early diagnosis, continuous assessment, and evidence-based interventions. Additionally, it will address the role of emerging therapies (eg, vortioxetine) and the significance of the dose-response relationship in optimising treatment outcomes. The symposium aims to enhance understanding of the interplay between depression and neurological disorders, emphasising the necessity of a comprehensive management plan to improve functional recovery and quality of life for affected individuals.

S 5

Eisai Dementia Symposium

Alzheimer's disease: A chronic journey requiring early and continuous intervention

Takahito YOSHIZAKI
Director, Kosaiin Hospital, Osaka, Japan

Alzheimer's disease (AD) is a progressive neurodegenerative disorder that requires early and continuous intervention to modify its long-term trajectory. Emerging evidence highlights the importance of addressing both amyloid plaques and protofibrils, which contribute to ongoing neuronal damage even after plaque clearance. The concept of AD continuum emphasises that treatment should begin in the early stages and be sustained to preserve cognitive and functional abilities. Shared decision making plays a critical role in initiating disease-modifying therapies, with efficacy and safety as the primary considerations. Longitudinal data demonstrate that early intervention can stabilise disease progression, particularly in individuals with low amyloid burden and minimal tau pathology. Functional outcomes, including instrumental and basic activities of daily living, are better preserved with continuous treatment over extended periods. Clinical monitoring through cognitive assessments, fluid biomarkers, and imaging techniques is essential to evaluate treatment response and guide ongoing care. Real-world cases have shown that sustained therapy may maintain amyloid clearance and support continued disease stabilisation. This presentation will explore the rationale for early and ongoing intervention, the clinical markers used to guide treatment, and the key factors influencing therapeutic decisions in early-stage AD.

S 6

Eli Lilly Dementia Symposium

The impact of current diagnostic and treatment advancements on the lives of patients with Alzheimer's disease

Colin J MAHONEY
Neurologist and Director, Cognitive Neurology Service, South Western Sydney Local Health District, New South Wales, Australia

Alzheimer's disease (AD) is the commonest cause of dementia globally, with up to 40 million people affected. Economic and societal costs are vast, with direct and indirect expenses estimated to reach US$2 trillion by 2030. Recent advances, such as biofluid and imaging quantification of the key proteins associated with AD, have allowed earlier, even presymptomatic, diagnosis. This creates significant opportunities to modify disease trajectories at an earlier stage, with the potential to improve clinical outcomes for patients and families affected. Anti-amyloid therapies have been a cornerstone of therapeutic trials in AD for over 20 years and have now been licensed for use in several jurisdictions, representing a paradigm shift towards disease modification. Whilst great opportunities exist, there are significant challenges as the number of people living with mild cognitive impairment or presymptomatic AD may represent between 20% and 40% of community dwellers over the age of 50. Here, we will discuss the advances in the diagnosis of AD utilising emerging biofluid and imaging markers, including appropriate patient selection, interpretation, and practical implementation in the clinical environment. Once a diagnosis is confirmed, attention shifts to supporting our patients with a focus on the maintenance of cognitive health. The evidence for pharmacological and non-pharmacological treatments in AD will be reviewed, with a particular focus on the role of anti-amyloid therapies. A case-based approach regarding suitable patient selection and the provision of personalised risk and benefit for anti-amyloid therapy will also be discussed.

S 7

Medison Pharma Symposium on Amyloid Transthyretin Amyloidosis

Unmasking and managing transthyretin amyloidosis

Matt SILSBY
Director, Neurophysiology and Neuromuscular Services, Westmead Hospital, Sydney, Australia

Transthyretin (TTR) amyloidosis is an underrecognised, progressive, and multisystemic disorder that presents a diagnostic and therapeutic challenge. The disease spectrum extends to involve the nerves, heart, autonomic nervous system, and other organs. In hereditary TTR amyloidosis, peripheral neuropathy is frequently the most prominent manifestation, leading to significant morbidity and a high risk of premature mortality if left untreated. In this talk, Dr Silsby will explore the evolving understanding of TTR amyloidosis, highlighting the clinical heterogeneity and diagnostic pitfalls that often delay recognition. Drawing on his experience as a clinician, researcher, and trial investigator, Dr Silsby will discuss practical strategies for early diagnosis, the role of neurophysiology in disease detection, and the importance of a multidisciplinary approach to management. The session will also review current disease-modifying therapies, with a focus on how timely intervention can alter the natural history of the disease and improve patient outcomes. Through real-world case insights, attendees will gain a deeper appreciation of how to unmask TTR amyloidosis and optimise care across its diverse clinical presentations.

S 8

Novartis Multiple Sclerosis Symposium (co-organised with Hong Kong Multiple Sclerosis Society)

Smouldering-associated worsening in multiple sclerosis and the importance of using high efficacy treatment in early multiple sclerosis

Mar TINTORE
Department of Neurology, MS Centre of Catalonia (Cemcat), University Hospital Vall d'Hebron, Barcelona, Spain

Smouldering multiple sclerosis, marked by progression independent of relapse activity, drives cumulative disability. Prof Mar Tintore will outline how smouldering processes affect patients across functional domains (mobility, dexterity, vision, cognition, fatigue) and may remain underdetected without structured, longitudinal assessment. Early recognition requires integrated monitoring: combining neurological assessments, magnetic resonance imaging, patient-reported outcomes, and the use of emerging biomarkers like neurofilament light chain. Prof Tintore will highlight the role of high-efficacy therapies in limiting smouldering activity and long-term decline, balanced against safety, tolerability, and monitoring needs. Practical considerations, including defining "early", assessing the risk/benefit profile, and pregnancy planning, will also be covered. Together, these insights will help provide a potential framework for detecting progression independent of relapse activity early and support the timely use of high-efficacy therapy to improve long-term outcomes.

S 9

Eisai Multiple Sclerosis Symposium (co-organised with Hong Kong Multiple Sclerosis Society)

Personalising multiple sclerosis therapy: Guidelines, treatment selection and risk mitigation

Kazuo FUJIHARA[1,2]
[1] Department of Multiple Sclerosis Therapeutics, Fukushima Medical University School of Medicine, Fukushima, Japan
[2] Director, Multiple Sclerosis and Neuromyelitis Optica Center, Southern TOHOKU Research Institute for Neuroscience (STRINS), Koriyama, Japan

Multiple sclerosis (MS) is an inflammatory demyelinating disease of the central nervous system that develops through tissue injury and compensatory mechanisms in the context of various genetic and environmental factors. The clinical course is influenced by central nervous system tissue reserve and repair and biological ageing. Recent evidence suggests that the clinical course of MS should be considered a continuum rather than categorised into distinct clinical phenotypes such as relapsing-remitting, secondary progressive, and primary progressive. Considering various factors, the treatment of MS should be personalised and patients should be involved in therapeutic choices for better adherence, quality of life, and to help manage caregiver burden. Early and accurate diagnosis and early initiation of treatment are indispensable to improve long-term prognosis. In this regard, the 2024 revisions of the McDonald Criteria are useful to expedite diagnosis while maintaining specificity. Meanwhile, careful monitoring and measures to reduce risks (eg, infections) associated with disease-modifying therapies are important in the management. In this presentation, an overview of personalised treatment for MS will be provided, including the Japanese Clinical Practice Guidelines of Multiple Sclerosis and Neuromyelitis Optica Spectrum Disorder (2023) will be provided.

S 10

Lunch Symposium on Stroke (sponsored by Boehringer Ingelheim)
The changing landscape of thrombolysis in acute ischaemic stroke management

Keith MUIR
School of Cardiovascular and Metabolic Health, University of Glasgow, Scotland, United Kingdom

Stroke affects 0.8% of Hong Kong's population and remains the fourth leading cause of death, causing over 3,000 fatalities annually. Acute ischaemic stroke (AIS) comprises approximately 80% of all stroke cases, and delays in treatment often result in significant long-term disability, severely affecting patients' quality of life. Over the past decade, AIS management has evolved dramatically, with growing evidence and updated international guidelines demonstrating the benefits of thrombolytic therapies in improving patient outcomes. To continue advancing stroke care, it is essential to incorporate novel parameters and state-of-the-art neuroimaging techniques that enhance patient selection, optimise treatment timing, and better assess risk—thereby enabling more tailored and effective interventions. This lecture is timely and critical, given the ongoing challenges in the timely and precise treatment of AIS within Hong Kong's healthcare system. It will highlight recent advances in thrombolytic agents by examining updated guideline recommendations and real-world applications. A major focus will be on how advanced imaging modalities are transforming thrombolysis by expanding eligibility criteria and providing more accurate outcome predictions, with practical insights drawn from international models such as the United Kingdom healthcare system. Additionally, the lecture will explore emerging personalised strategies to optimise AIS care and address key clinical and operational challenges. Healthcare professionals will gain evidence-based knowledge and practical tools to integrate these innovations into daily practice, helping to close existing treatment gaps. By enhancing the precision and effectiveness of thrombolytic therapy, this lecture aims to improve stroke outcomes, reduce the burden on patients and healthcare services, and advance the future of acute stroke management in Hong Kong.

S 11

Neuromuscular Disease Symposium
Expanding aetiologies immunological mechanisms and diagnostic approaches in small fibre neuropathy

Amanda CHAN
Division of Neurology, National University Healthcare System, Singapore

In this talk, Dr Chan will provide a comprehensive update on small fibre neuropathy, emphasising advances in both the aetiological spectrum and diagnostic workup. She will review evolving clinical approaches, including the integration of autonomic testing, skin biopsy, and novel biomarker panels, which together are enhancing diagnostic accuracy in a condition long considered difficult to characterise. Research in small fibre neuropathy is progressing rapidly, with emerging evidence of immune-mediated subtypes and the discovery of novel autoantibodies that are beginning to redefine disease classification. These antibody findings not only offer mechanistic insights into pathogenesis but also hold promise as tools for precision diagnostics and targeted therapies. By bridging fast-moving scientific advances with practical clinical application, her presentation aims to equip neurologists with both the latest knowledge and its implications for patient care.

S 12

Teva Migraine Symposium

Innovating migraine care: Unlocking the full potential of calcitonin gene–related pathway therapies

Piero BARBANTI[1,2]
[1] Professor of Neurology, San Raffaele University, Rome, Italy
[2] Director, Headache and Pain Unit, IRCCS San Raffaele, Rome, Italy

Migraine is a complex neurovascular disorder characterised by heterogeneity in clinical presentation, disease trajectory, and treatment response. The introduction of calcitonin gene–related peptide (CGRP) pathway-targeting therapies represents a paradigm shift in migraine management. These agents offer a mechanism-specific approach that bypasses traditional limitations associated with non-selective therapies. In this symposium, Professor Piero Barbanti will critically examine the evolving role of CGRP-targeted treatments within the broader therapeutic landscape. The session will address key clinical considerations, including the optimal timing of initiation, patient selection criteria, and strategies for managing partial or non-response. Emphasis will be placed on integrating CGRP therapies into individualised treatment plans, informed by disease severity, comorbidities, and prior treatment history. Recent data from randomised controlled trials and real-world studies will be reviewed to assess efficacy, safety, and long-term outcomes. The discussion will also explore emerging concepts such as treatment sustainability and the implications of CGRP blockade on disease modification. This session aims to provide a comprehensive and evidence-based framework for clinicians seeking to optimise migraine care through the rational use of CGRP pathway therapies, while also identifying gaps in current practice and future directions for research.

S 13

AbbVie Migraine Symposium

Mastering migraine therapies

William KINGSTON
Headache Neurologist, Sunnybrook Health Science Centre, Toronto, Canada

Migraine remains a leading cause of disability, demanding continued innovation in preventive and acute management strategies. This symposium delivers a comprehensive, clinically driven update on the fundamentals of antimigraine care, with a focus on calcitonin gene–related peptide (CGRP) pathway modulation. CGRP is central to migraine pathogenesis via nociception and neurogenic inflammation; thus, inhibitors targeting CGRP, including atogepant—a selective oral CGRP receptor antagonist—have transformed therapeutic possibilities for both episodic and chronic migraine. A highlight will be the latest data that offer direct, head-to-head evidence comparing atogepant with topiramate, a traditional oral prophylactic. The session will also discuss emerging clinical experience in Canada. Best practice pearls for patient selection will be shared, emphasising early intervention for those with frequent, disabling migraines who have failed conventional therapies.

S 14

GSK Shingles Symposium
Zoster vaccination: A neurologist's tool for zoster and stroke prevention

Kay Cheong TEO
Department of Medicine, The University of Hong Kong, Hong Kong SAR, China

Herpes zoster (shingles) results from reactivation of latent varicella-zoster virus in dorsal root or cranial nerve ganglia, causing a unilateral, painful vesicular rash in a dermatomal distribution. Approximately one in three people will develop shingles at some point in their lives, and the risk increases for older adults and those who are immunocompromised. While most individuals recover completely, the symptoms can be quite distressing, and postherpetic neuralgia occurs in up to 20% of cases. Other neurological complications include encephalitis, myelitis, cranial neuropathies, and stroke. This session will review the pathophysiology and clinical spectrum of herpes zoster using illustrative cases. The relationship between herpes zoster and stroke will be examined, highlighting the mechanisms of heightened vascular risks following an acute infection. Next, the evidence for the recombinant zoster vaccine will be summarised. Recombinant zoster vaccine offers highly efficacious and long-lasting protection against herpes zoster and postherpetic neuralgia, with emerging real-world data linking zoster vaccination with lower stroke and dementia risk. The session will conclude with practical recommendations for neurologists on how to incorporate zoster vaccination into clinical practice to enhance patient outcomes.

ES 1

Education Session
Parry–Romberg syndrome

Shi Hon NG[1], Shirley CHEUNG[2]
[1] Department of Medicine, Pok Oi Hospital, Hong Kong SAR, China
[2] Department of Medicine, St Teresa's Hospital, Hong Kong SAR, China

Parry–Romberg syndrome (progressive hemifacial atrophy) is a rare, mostly sporadic syndrome of unknown aetiology, characterised by variable progressive self-limited but irreversible atrophy on one side of the face and sometimes the ipsilateral trunk and limbs (skin, soft tissues, cartilage, muscle and bones). In Part One, Dr Ng will give a brief review of the subject including some personal local experience over the past 40 years with a nostalgic history of Queen Mary Hospital. Neurological involvement in the syndrome will be emphasised. In Part Two, Dr Cheung will present a recent local case with a detailed work-up to illustrate this syndrome. The presentation will conclude, as usual, with our "dessert" comprising three quizzes on movement disorders (two of which will be in video format).

FP 1

Incidence, treatment metrics and outcomes of intracerebral haemorrhage in Hong Kong: A 10-year territory-wide study

Bianca CHAN*, Bella WONG*, Bono LI, Trista HUNG, Thomas LEUNG, Bonaventure IP
Division of Neurology, Department of Medicine and Therapeutics, Prince of Wales Hospital, The Chinese University of Hong Kong, Hong Kong SAR, China
* Equal contributions

Background: Characterising temporal trends in spontaneous intracerebral haemorrhage (ICH) care identifies areas for clinical improvement, establishes key baseline performance metrics, and guides healthcare resource allocation. We aimed to determine the temporal trends in the incidence, treatment metrics, and outcomes of ICH in Hong Kong.

Methods: We conducted a retrospective cohort study utilising an electronic healthcare database from the Hong Kong Hospital Authority. ICH patients were identified by the principal diagnosis code of a hospitalisation episode. We retrieved the time of emergency department (ED) arrival, brain computed tomography (CT) and CT angiography (CTA), ward admissions, and systolic blood pressure (SBP) on ward admission. Temporal trends in crude incidence, age-standardised incidence, mortality, case fatality rate, ED-to-CT interval, ED-to-ward admission interval, proportion of patients achieving SBP ≤140 mm Hg, and CTA utilisation from 2014 to 2024 were analysed using Joinpoint regression.

Results: We identified 25 448 ICH patients during the 10-year study period. The crude incidence of ICH did not change significantly (range, 34.2-41.2 per 100 000 person-years, P=0.204). Age-standardised incidence declined by 0.592 per 100 000 person-years (95% confidence interval=0.292-0.892, P=0.002). Age-standardised mortality rate declined by 0.27 deaths per 100 000 per year (95% confidence interval=0.186-0.359, P<0.001). The 30-day case fatality rates were static across age-groups (20-39 years: 11.4-18.1%, 40-59 years: 15.7-17.8%, 60-79 years: 22.4-26.2%, ≥80 years: 37.8-42.1%). The median ED-to-CT time range was 26 to 39 minutes, and the median ED-to-ward admission time range was 68 to 92 minutes. The percentage of ICH patients achieving an SBP of ≤140 mm Hg on ward admission increased from 17.5% to 22.8% (P=0.017). CTA utilisation rate remained low (range: 0.9-3.7%, P=0.055).

Conclusion: Age-standardised incidence and mortality rates of ICH have declined in Hong Kong. Yet, the persistently high case fatality rate highlights inadequacies in ICH care, particularly the low proportion of patients achieving early intensive SBP control. These findings underscore the need for implementation of territory-wide, multidisciplinary ICH care pathways.

FP 2

Oral anticoagulation after spontaneous intracerebral haemorrhage in patients with non-valvular atrial fibrillation

Nicholas FOK
Tuen Mun Hospital, Hong Kong SAR, China

Background: Patients with non-valvular atrial fibrillation (NVAF) who had a spontaneous intracerebral haemorrhage (sICH) are at high risk of both recurrent sICH and ischaemic stroke. Current guidelines do not provide strong recommendations on whether, or when, to use oral anticoagulants in this population. This study aimed to consolidate the evidence on the efficacy and safety of oral anticoagulant use in patients with NVAF and sICH.

Methods: A systematic review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines. PubMed, EMBASE, and the Cochrane Library were searched for randomised trials and observational studies comparing (re)initiation or avoidance of oral anticoagulants in patients with NVAF post-sICH. The primary outcomes were recurrent sICH and ischaemic stroke. The secondary outcome was all-cause mortality. A narrative synthesis was performed to identify the strengths and gaps in the existing literature.

Results: Four randomised trials and 32 observational studies were included. The evidence suggests a consistent potential benefit of anticoagulation in reducing ischaemic stroke in patients with NVAF after sICH. Uncertainty, however, remains regarding its impact on recurrent sICH, all-cause mortality, and long-term functional outcomes. The optimal timing of anticoagulation (re)initiation has not been investigated in randomised trials, and findings from observational studies have been conflicting. Owing to heterogeneous subgroups, variable phenotypic details, and limited sample sizes, applying these results to individualised risk-benefit stratification remains a challenge.

Conclusion: The risk-benefit balance of anticoagulant use in patients with NVAF after sICH remains unclear at the individual level. A blanket generalisation of study conclusions is unlikely to inform clinical decision making adequately. More nuanced and detailed subgroup phenotyping would provide greater insight into practical risk stratification strategies.

FP 3

Effect of mirror therapy on upper limb function in stroke patients: A retrospective study

Wing Chong LO
Caritas Medical Centre, Hong Kong SAR, China

Background: Mirror therapy (MT) has recently been integrated into the daily rehabilitation practices within the Occupational Therapy Department of Caritas Medical Centre. However, there is insufficient local review on the efficacy of MT as an adjunct to conventional rehabilitation.
Methods: This was a retrospective cohort study involving 10 stroke patients at Caritas Medical Centre from July to November 2024. Patients were divided into two groups: those who received conventional rehabilitation alone (control group) and those who received conventional rehabilitation supplemented with MT (study group). The Mann-Whitney *U* test was used to evaluate comparability between the study and control groups. Changes in FMA-UE (Fugl–Meyer Assessment for Upper Extremity), FTHUE-HK (Functional Test for the Hemiplegic Upper Extremity–Hong Kong Version), MMT (Manual Muscle Testing), mRS (modified Rankin Scale), and MBI (Modified Barthel Index) scores were examined for both groups. The Wilcoxon signed-rank test was used to analyse changes between pre- and post-treatment within each group.
Results: Both the study and control groups showed improvement in upper limb function, with the study group demonstrating a more significant enhancement. Regarding FMA-UE total scores, there was a statistically significant difference (P=0.019) in the change of scores between the study and control groups, with mean (± standard deviation) score changes of 15.83 (±12.48) and 3.25 (±2.63), respectively. Patients in both groups also showed improvement in functional outcomes, including reduced disability and increased functional independence, with the improvement in the study group being more marked than in the control group.
Conclusion: This study suggests that MT, as an adjunct to conventional rehabilitation, improves upper limb function and functional outcomes more effectively than conventional rehabilitation alone.

FP 4

Radiological determinants of tissue fate in plain brain computed tomography during acute large vessel occlusion: A multicentre study

Billy LAM, Bianca CHAN, Sze Ho MA, Thomas LEUNG, Haipeng LI, Bonaventure IP
Division of Neurology, Department of Medicine and Therapeutics, Prince of Wales Hospital, The Chinese University of Hong Kong, Hong Kong SAR, China

Introduction: Endovascular thrombectomy (EVT) triage based on the semiquantitative Alberta Stroke Program Early CT Score (ASPECTS) is increasingly adopted. However, the relative contribution of components of baseline ASPECTS (hypoattenuation, loss of grey-white differentiation [LGWD], and sulcal effacement or mass effect [SE/ME]) to tissue fate in patients with large vessel occlusion (LVO) undergoing EVT is uncertain. We aimed to elucidate the association between specific ASPECTS features and final infarct volume.
Methods: We conducted a multicentre retrospective cohort study across four hospitals in Hong Kong and mainland China. Pre-EVT ASPECTS was assessed by neuroradiologists and automated software. Hypoattenuation, LGWD, and SE/ME were graded manually for each ASPECTS region. The primary outcome was the final infarct volume on post-EVT magnetic resonance imaging. Multivariable regression was used to determine the relationship between each ASPECTS component and final infarct volume, with the effect estimate for each feature used to construct a weighted ASPECTS.
Results: Among 375 sets of plain computed tomography brain scans in patients with LVO undergoing EVT between January 2020 and July 2024, hypoattenuation and LGWD showed significant associations with final infarct volume across all ASPECTS regions, whereas SE/ME was correlated with final infarct volume in M1-6 regions. Hypoattenuation was the strongest predictor of final infarct volume. The weighted ASPECTS, integrating the weights of all three radiological features (β-coefficient= –0.77 [-0.89 to -0.65], P<0.001), demonstrated a stronger association with final infarct volume than neuroradiologist-graded (β-coefficient=0.54 [-0.63 to -0.45], P<0.001) or artificial intelligence–graded ASPECTS (β-coefficient = –0.58 [-0.70 to -0.46], P<0.001).
Discussion: Components of ASPECTS differ in their predictive strength for final infarct volume. EVT triage based solely on ASPECTS may overestimate the risk of irreversible infarct core in patients with "low ASPECTS" driven by features with weaker correlations. A weighted feature-informed ASPECTS may improve patient selection for EVT.

FP 5

Characterising LRRK2 Asian missense mutations in the context of Parkinson's disease

Manyu WANG
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Background: Missense mutations in the gene encoding leucine-rich repeat kinase 2 (LRRK2) represent a major genetic cause of Parkinson's disease (PD). Interestingly, the mutation spectrum of LRRK2 exhibits significant racial variation. In the Asian population, specific mutations such as p.R1628P, p.S1647T, and p.G2385R, are frequently observed and are associated with an early age at onset of PD. However, the mechanisms driving this phenotype and its potential correction by LRRK2 kinase inhibitors, a promising therapeutic class in advanced clinical development, remain unknown. In this study, we aimed to address these questions through deep phenotyping in both animal and cell models.
Methods: We generated two transgenic mouse models in which the endogenous murine *Lrrk2* gene was replaced with the human wild-type or mutant LRRK2 coding sequence carrying the three Asian mutations (3× knock-in). Parkinsonism-like phenotypes were assessed longitudinally using behavioural tests. Brain tissues were collected for histological examinations and transcriptomic analysis. Cell models were used to characterise mutant LRRK2 kinase activity, its interactome, and to validate findings from the animal studies. Finally, we tested the effects of LRRK2 kinase inhibitors in these models.
Results: We found no developmental deficits in mice carrying the human LRRK2 transgenes. The 3× knock-in mice developed age-dependent motor deficits, including gait abnormalities (prolonged stance or swing time and shortened stride length). Transcriptomic profiling of midbrain and striatal tissues revealed significant dysregulation of pathways related to mitochondrial function, synaptic homeostasis, and immune-inflammatory responses. Cell-based studies suggested that 3× mutant LRRK2 exhibits a lower phosphorylation level at serine 935, which has been reported to be required for its interaction with 14-3-3 protein.
Conclusion: We successfully recapitulated key PD features in mice harbouring the three LRRK2 Asian mutations. Preliminary data implicate pathological alterations in mitochondria, synaptic function, and immune-inflammation in the striatum and midbrain. Molecularly, these mutations may alter the interaction of LRRK2 with its downstream effectors. These findings warrant further comprehensive investigation.

FP 6

Role of stroke team pharmacists in identifying drug-related problems in a local public hospital

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Background: Stroke patients often face polypharmacy, significantly increasing the risk of drug-related problems (DRPs). At Caritas Medical Centre, a designated stroke team pharmacist plays a crucial role in managing medications and enhancing patient safety within the Acute Stroke Unit. By participating in daily rounds and weekly grand rounds, the pharmacist collaborates closely with neurologists, stroke nurses, and other healthcare professionals to ensure comprehensive care. This integrated approach allows for real-time identification and resolution of medication-related issues, fostering a multidisciplinary team environment essential for optimising patient outcomes in stroke care.
Methods: A retrospective review of patient profiles was conducted over 12 months, from 1 September 2024 to 31 August 2025. The National Coordinating Council for Medication Error Reporting and Prevention classification system was used to identify and categorise DRPs systematically. Data on the number of patients reviewed and types of DRPs, including unnecessary therapy, adverse drug reactions, and non-adherence, were collected and analysed using descriptive statistics.
Results: A total of 764 patients were reviewed. In all, 246 DRPs in 146 patients were identified by pharmacists, with relevant suggestions being accepted by physicians (approximately 20% of total cases). Most DRPs were corrected during hospitalisation (78.8%), with additional corrections at admission (13.4%) and discharge (7.8%). The primary DRP categories were issues related to treatment effectiveness (44.3%), treatment safety (27.6%), and discrepancies during transitions of care (19.9%). Notably, 6.1% of DRPs were associated with patient non-adherence, for which pharmacists provided interventions and education to enhance compliance. The most affected medication classes included lipid-regulating drugs (17.9%) and antiplatelet agents and antidiabetic drugs including insulin (both 8.5%).
Conclusion: The involvement of pharmacists in stroke care significantly improves the identification of DRPs. Their collaborative approach is essential for enhancing patient safety and optimising medication management. This study highlights the importance of pharmacist participation in multidisciplinary teams to ensure effective medication management for stroke patients.

FP 7

Exploring the intersection of ageing, obesity, and glucagon-like peptide-1 receptor signalling in Alzheimer's disease

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* Co-first authors

Background: Ageing and obesity are major risk factors for Alzheimer's disease (AD). The glucagon-like peptide-1 receptor (GLP-1R), primarily expressed in the hypothalamus and pancreas, is essential for metabolic homeostasis and may mechanistically link ageing, metabolic dysfunction, and neurodegeneration. Several GLP-1R agonists (GLP-1RAs) have demonstrated neuroprotective effects and reduced AD risk in preclinical models and clinical trials for obesity. This study investigates the systemic and neurological effects, as well as the underlying mechanisms of GLP-1RAs in preclinical models of AD pathology involving ageing and obesity, aiming to identify benefits that are independent of food intake and body weight, and to guide their optimal use in AD treatment.

Methods: To capture the effects of both ageing and obesity, C57BL/6 mice are fed either a normal chow or a high-fat diet (HFD; 60 kcal% from fat) at different ages. After obesity is established, mice are treated with semaglutide (a GLP-1RA) or calorie restriction to reduce body weight. Calorie restriction is implemented via paired feeding, providing mice the same amount of food consumed by semaglutide-treated animals to isolate pharmacological effects beyond appetite suppression. To mimic real-world weight cycling, treatments are withdrawn once the effects plateau, and the obesity-treatment cycle is repeated twice. At the end of the study, tissues from the cortex, hippocampus, and hypothalamus will be collected for RNA sequencing to profile transcriptomic changes.

Results: Age-associated phenotypic differences were observed in obesity and semaglutide treatment response. Young HFD-fed mice gained body weight faster than middle-aged counterparts, whereas middle-aged obese mice exhibited greater fluctuations in blood glucose. Additionally, body weight reduction was less effective in middle-aged mice under paired feeding. Notably, hypothalamic GLP-1 levels were significantly reduced in aged and adult AD model mice, supporting the hypothesis that altered GLP-1R signalling mechanistically links ageing, metabolic dysfunction, and neurodegeneration.

Conclusion: We are now profiling the complex brain transcriptomic alterations induced by the convergence of systemic metabolic dysregulation, advanced age, and the impact of agonising GLP-1R signalling in this real-world mimicking setting.

AUTHOR INDEX

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†The ADVANCE pivotal prevention study evaluated AQUIPTA® for the prophylaxis of episodic migraine in patients who met the International Classification of Headache Disorders (ICHD) criteria for a diagnosis of migraine with or without aura.[1,4]
‡Results based on the off-treatment hypothetical estimand (OTHE) population.[1,4]
§The PROGRESS pivotal prevention study evaluated AQUIPTA® for the prophylaxis of chronic migraine in patients who met the ICHD criteria for chronic migraine.[1,3]

CGRP, calcitonin gene-related peptide; **CrCl**, creatinine clearance; **CYP3A4**, cytochrome P450 family 3 subfamily A member 4; **OATP**, organic anion transporting polypeptide.

References: 1. AQUIPTA® (atogepant) Hong Kong Prescribing Information, April 2024. **2.** Ailani J, et al. *N Engl J Med* 2021; 384:695-706. **3.** Schwedt TJ, et al. *Cephalalgia* 2022;42(13):1193-1204. **4.** Goadsby PJ, et al. *Lancet* 2023; 402(10404):761-773.

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Experts now believe that the pathophysiological changes of Alzheimer's disease, such as the abnormal build-up of amyloid and subsequent accumulation of tau, may begin up to 20 years before symptoms emerge.[1,3] Once symptoms present, time is of the essence. Early detection and diagnosis may make a difference for your patients.[1,3,4]

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