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From Bench to Bedside:
Bridging Today's Discoveries with Tomorrow's
Therapeutics for Societal Impact

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KEYNOTE LECTURES

[KL-1.0]

**SHAPING RESEARCH FOR SOCIETY:
STRATEGIC BLUEPRINTS FOR A HEALTHIER PLANET**

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A healthier planet demands research that is purpose-driven, mission-oriented, collaborative, and directly responsive to societal needs. Shaping Research for Society: Strategic Blueprints for a Healthier Planet calls for reimagining research as a driver of transformative change – bridging environmental sustainability, public health, and socio-economic resilience. This vision rests on four pillars: fostering transdisciplinary partnerships, integrating local and Indigenous knowledge, harnessing data and digital innovations, and ensuring equitable access to research benefits. By aligning priorities across universities, policymakers, industry, and communities, research can move from fragmented efforts to coherent strategies that anticipate risks and address complex challenges. Grounded in the principles of Planetary Health and One Health, this paper outlines practical pathways for translating knowledge into action, accelerating impact, and building an adaptive, inclusive research ecosystem capable of guiding humanity towards a more sustainable future.

Keywords: Planetary Health, Environmental sustainability, Transdisciplinary Partnerships, Equitable Access and Impact.

KEYNOTE LECTURES

[KL-2.0]

**FROM NEURAL PATHWAYS TO PATIENT CARE:
TRANSLATING NEUROSCIENCE DISCOVERIES INTO HEALTHCARE
SOLUTIONS – DE AGEING – FACT OR FICTION?**

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Introduction: In movies, digital improvisations are made to revert an old person to his or her younger version. Is this reversal possible physiologically? The relationship between ageing and alterations in the gut microbiota composition has been established. A Ministry of Higher Education's (MOHE) research programme AGELESS has investigated the interplay among gut microbiota, short-chain fatty acids (SCFA), cognitive, physical, and psychosocial performance of Malaysian older adults.

Methods: Physical (eg, gait), cognitive, and psychosocial performance of 231 subjects (average age: 71 years old) were noted. Stool samples were later analysed for 16S v3-v4 rRNA marker genes using the NovaSeq 6000 platform. Faecal SCFAs were quantified using GC-MS.

Results: The present study found significant ($p < 0.05$) associations between the phylum with age and gait speed. Age was negatively associated with Actinobacteriota and Firmicutes (SCFA producers) but positively associated with Proteobacteria (mainly pathobionts). Age was also found to be negatively ($p < 0.05$) associated with faecal acetate, propionate, and butyrate. Participants older than 80 years old presented with significantly lower ($p < 0.05$) faecal acetate than those between 60-69 years old. SCFA was significantly ($p < 0.05$) lower in participants with mild cognitive impairment (MCI). Spearman correlation analysis revealed a significant negative correlation between butyrate and DASS ($r = -0.35$, $p < 0.05$), but a significant positive correlation between acetate and MoCA ($r = 0.27$, $p < 0.05$).

Conclusions: This study provides further evidence that microbiota impacts physical, cognitive, and psychosocial performances of older adults. Programmed interventions that could restore gut microbiota that promote physical and mental well-being would be expected to retard, or even probably, induce physiological de-ageing.

Keywords: Ageing, Cognitive Frailty, Microbiota, Short-Chain Fatty Acids (SCFA).

PLENARY LECTURES

[PL-1.0]

**PHARMACOEPIDEMOLOGY AND PUBLIC HEALTH INTERVENTIONS:
SHAPING GLOBAL STRATEGIES FOR VULNERABLE POPULATIONS**

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Pharmacoepidemiology, or the study of drug use and effects in large populations, plays a crucial role in shaping public health interventions, particularly for vulnerable communities. This presentation explores how pharmacoepidemiological research informs global health strategies to ensure equitable access to safe and effective medications. Importantly, for vulnerable populations – such as the elderly, children, pregnant women, and those in low-income or conflict-affected regions – they often face disparities in healthcare access, medication safety, and treatment outcomes. Pharmacoepidemiology provides critical insights into drug safety, effectiveness, and patterns of use in these populations, guiding policymakers in optimising therapeutic interventions. We will discuss how real-world data, post-marketing surveillance, and pharmacovigilance contribute to identifying adverse drug reactions, ensuring appropriate medication use, and minimising health risks. Case studies will illustrate successful public health interventions, such as vaccination programmes, antimicrobial stewardship, and policies addressing medication shortages in underserved areas. The presentation will highlight the role of collaborations, regulatory agencies, and digital health technologies to improve global health equity. Ultimately, this discussion will underscore the importance of a data-driven, multidisciplinary approach in shaping sustainable global health strategies to protect and empower vulnerable communities.

Keywords: Pharmacoepidemiology, Public Health, Vulnerable Populations, Drug Safety, Health Equity.

PLENARY LECTURES

[PL-2.0]

**MAPPING THE JOURNEY:
FROM MOLECULAR TARGETS TO ALZHEIMER'S THERAPEUTIC PIPELINE**

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Alzheimer's disease (AD) remains a significant global health challenge, affecting millions of individuals aged 65 and older, with projections indicating a growing burden in 2025. This plenary presentation explores the intricate journey from molecular targets to the current therapeutic pipeline for AD, highlighting key pathological mechanisms and innovative treatment strategies. The presentation delves into the role of amyloid-beta (A β) peptides, particularly the A β 42 amino peptide, and tau protein pathology, which contribute to reduced brain volume in critical regions such as the entorhinal cortex and hippocampus, driving the clinical trajectory of AD. Focusing on the 2025 Alzheimer's drug development pipeline, the talk outlines the mechanisms of action of promising agents in Phase 2 and Phase 3 clinical trials, classified using the Common Alzheimer's Disease Research Ontology (CADRO) framework. Notable therapies include monoclonal antibodies targeting A β plaques and oligomers, such as Donanemab, Lecanemab, alongside Gantenerumab. Anti-tau monoclonal antibodies, including E2814 and BMS-986446, aim to block tau propagation, while vaccines like ABvac40 and UB-311 target specific A β isoforms to induce safe, immunogenic responses. Additional approaches address neuroinflammation, synaptic plasticity, and proteostasis. Despite advancements, AD clinical trials face significant barriers, including limited patient awareness, diagnostic challenges, and infrequent trial referrals, which hinder trial completion and therapy approval. This presentation underscores the need for a holistic, collaborative approach among stakeholders to overcome these challenges, enhance patient access to trials, and accelerate the development of precision therapeutics. By mapping the molecular underpinnings to the evolving therapeutic landscape, this talk aims to provide a comprehensive overview of the current state and future directions of AD treatment in 2025.

Keywords: Alzheimer's Disease, Molecular Targets, Therapeutics, Pharmacology.

PLENARY LECTURES

[PL-3.0]

OBESITY, CARDIOVASCULAR DISEASE, DIABETES, AND LEPTIN

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Almost 30% of the world's population is either overweight or obese. Obesity contributes to more than five million deaths annually from cardiovascular diseases (CVD), diabetes mellitus (DM), neurological disorders, respiratory diseases, cancers and digestive disorders. Obesity is also a risk factor in reproductive, perinatal and pelvic disorders. While the precise mechanism linking obesity with obesity-related diseases remains uncertain, the ubiquitous presence of leptin receptors throughout the body, the hyperleptinaemia in the obese, and the pro-inflammatory properties of leptin, make leptin the most likely link between obesity and CVD, DM, and other obesity-related diseases. The hypertensive effects of leptin on blood pressure involve (i) endothelial dysfunction and altered ET-1 and NO levels, (ii) "ACE-AngII-AT1R" and "ACE2-Ang-(1-7)-MasR" axes imbalance, (iii) activation of the leptin-aldosterone-nephrilysin axis, (iv) increased secretion of cardiotoxic steroids, and (v) increased sympathetic activity. In addition, leptin's action on cardiac fibroblasts, cardiomyocytes, endothelial, and immune cells are implicated in obesity-associated cardiac fibrosis and dysfunction. Obesity is a primary factor in hepatic, skeletal muscle and adipose tissue insulin resistance. Hyperleptinaemia, hyperinsulinaemia, and insulin resistance in the obese present an interpretive conundrum, as leptin has also been shown to increase insulin sensitivity. However, plasma leptin levels correlate directly with insulin resistance. Rise in serum leptin levels precedes insulin resistance in the obese. Rats pre-treated with leptin for six weeks when challenged with a glucose load, were noted to have higher insulin levels and a lower rate of glucose clearance, implying the role for leptin in insulin resistance. T2DM itself is a risk factor for coronary artery disease, heart failure and peripheral arterial and microvascular disease. Whether there is a common factor responsible for the cardiovascular and metabolic consequences of obesity remains to be established, there is, nevertheless, enough evidence implicating adipocyte dysfunction, particularly the increased release of leptin by the adipocytes in obesity-related CVD and DM.

Keywords: Obesity, Cardiovascular Disease, Diabetes, Leptin.

SYMPOSIUM LECTURES

[SYMPOSIUM 1 – NEUROPLASTICITY, NEUROMODULATION, AND NEUROPROTECTION]

[SL-1.1]

INNOVATIVE APPROACHES TO RETINAL PROTECTION: EXPLORING PHARMACOLOGICAL AGENTS FOR ISCHEMIC RETINOPATHIES

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Background: The study of possible ways of effective pharmacological correction of retinal ischemia-reperfusion (I/R) injury, which accompanies a number of eye diseases, is relevant today.

Aim: To study the retinoprotective effect of 3-hydroxypyridine derivatives in a rat model of retinal I/R.

Materials and Methods: A pathology model with an increase in intraocular pressure (IOP) to 110 mmHg was used. The retinoprotective effects of 2-ethyl-3-hydroxy-6-methylpyridine nicotinate (EHMP-N), 2-ethyl-3-hydroxy-6-methylpyridine-N-acetyltaurinate (EHMP-NAT), and L-isomer of 2-ethyl-3-hydroxy-6-methylpyridine malate (LEHMP-M) in comparison with emoxipine in equimolar doses were estimated by the changes in the ophthalmoscopic pattern, retinal microcirculation level, electroretinograms (the b/a coefficient). The most effective agent was checked for the changes in the retinal caspase-3, NF- κ B p65, p53 gene expressions in Wistar rats.

Results: The use of EHMP-NAT led to the most pronounced increase in the retinal microcirculation level to 756.5 (median) perfusion units in comparison with emoxipine ($p = 0.045$). The b/a coefficient increased significantly in comparison with emoxipine ($p = 0.0099$), the ophthalmoscopic pattern improved markedly. In the group with EHMP-NAT, the caspase-3 gene expression decreased reliably in comparison with emoxipine ($p = 0.0002$); the NF κ B p65 gene expression decreased in comparison with emoxipine ($p = 0.0009$); the p53 gene expression decreased in comparison with emoxipine ($p = 0.0022$).

Conclusions: EHMP-NAT is the most promising candidate for the further development of a retinoprotector from the studied 3-hydroxypyridine derivatives, the effectiveness of which is superior to emoxipine in a retinal I/R model.

Keywords: Retinal Ischemia, 3-hydroxypyridine Derivatives, Rats, Retinal Microcirculation, Electroretinography.

SYMPOSIUM LECTURES

[SYMPOSIUM 1– NEUROPLASTICITY, NEUROMODULATION, AND NEUROPROTECTION]

[SL-1.2]

**ADVANCING EPILEPSY THERAPEUTICS: BRIDGING
PHARMACOGENOMICS, PERSONALISED MEDICINE, AND
COMPREHENSIVE CARE**

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About 40% of patients with epilepsy are refractory to antiseizure drugs (ASMs). Many recent machine learning models attempted to predict seizure response to ASMs. Similarly, some patients developed specific adverse events to certain antiseizure medications, such as severe cutaneous adverse reactions (SCARs) to carbamazepine. Pharmacogenomics helps to predict the occurrence of these reactions. This lecture will focus on personalised medicine in epilepsy, explicitly focusing on ASM selection to achieve the best seizure outcome with the least adverse events. The role of pharmacogenomics in personalised medicine will be discussed using the role of HLA-B*1502 screening in preventing carbamazepine-related SCARs as an example. An implementation roadmap on personalised medicine in epilepsy for Malaysia to achieve comprehensive epilepsy care will be proposed.

Keywords: Epilepsy, Pharmacogenomics, Personalised Medicine, Antiseizure Drugs, Carbamazepine.

SYMPOSIUM LECTURES

[SYMPOSIUM 1– NEUROPLASTICITY, NEUROMODULATION, AND NEUROPROTECTION]

[SL-1.3]

**NEURODEGENERATION AND BRAIN REWARD CIRCUIT:
BRIDGING THE GAPS IN COGNITIVE NEUROSCIENCE**

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Growing interest in the connection between neurodegenerative diseases and the brain reward circuitry has gained significant momentum, fueled by expanding insights into the distinct components of the reward system. However, major gaps remain in our understanding of how these components interact, and how they are affected in the setting of the complex pathomechanisms of neurodegenerative diseases. From among the disruptions in specific brain regions and networks, these conditions often involve those linked to reward processing, leading to cognitive and behavioral impairments. This talk seeks to highlight the key gaps in our current understanding of the intricate relationship between neurodegenerative diseases and this reward circuit. Bridging these gaps is crucial for advancing research aimed at developing effective treatments and interventions, with particular emphasis on addressing the cognitive and behavioral manifestations of such nervous system disorders. A multidisciplinary approach that integrates advancing methodologies is vital to ultimately improve outcomes for individuals living with these debilitating neurological conditions.

Keywords: Reward, Neurodegenerative Disease, Dementia, Cognition.

SYMPOSIUM LECTURES

[SYMPOSIUM 2– INTEGRATING TRADITIONAL MEDICINE INTO MODERN HEALTHCARE]

[SL-2.1]

**IMMUNOPHARMACOLOGY OF NATURAL PRODUCTS:
EXPLORING ANTI-INFLAMMATORY AND IMMUNOMODULATORY
MECHANISMS FOR THERAPEUTIC INNOVATION – INHIBITORY EFFECTS
OF NATURAL α,β -UNSATURATED CARBONYL-BASED COMPOUNDS,
THEIR ANALOGS AND DERIVATIVES,
ON THE CELLULAR AND HUMORAL IMMUNE RESPONSES**

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In a healthy organism, the immune system and the adequate redox balance of immune cells maintain homeostasis within the body. The failure to maintain the balance may lead to impaired immune response and hence, an abnormal immune system, either overactivity or abnormally low activity of the immune cells resulting in autoimmune or immune deficiency diseases. The α,β -unsaturated carbonyl-based compounds are considered to be the reactive substructures of natural products or synthetic molecules, hence, owing miscellaneous pharmacological activities, ie, potent antioxidant, anti-inflammatory, antiviral, antibacterial, antifungal, antitubercular, and immunomodulatory properties. In fact, one-sixth of the currently known natural products contain these potentially reactive substructures. The most important and widely studied α,β -unsaturated carbonyl-based compounds include the natural products, curcumin, chalcones, zerumbone, and their analogs and derivatives. Numerous studies have revealed the mainly immunosuppressive and anti-inflammatory activities of the aforesaid compounds. This paper highlights the specific immunosuppressive and anti-inflammatory effects of some natural α,β -unsaturated carbonyl-based compounds, and their analogs and derivatives on different types of immune cells of the innate and adaptive immune systems. The effects of the samples on lipopolysaccharide (LPS)-induced nuclear factor-kappa B (NF- κ B), mitogen-activated protein kinase (MAPK), and protein kinase B (Akt) pathways activation in U937 human macrophages were also investigated. The findings indicate that the α,β -unsaturated carbonyl-based compounds were effective immunosuppressive agents. It is of paramount importance to continue generating experimental data on the mechanisms of action of α,β -unsaturated carbonyl-based compounds on immune cells to provide useful information for ensuing research to discover new immunomodulating agents.

Keywords: Immunopharmacology, α,β -unsaturated Carbonyl Compounds, Immunosuppression, Anti-inflammatory, Natural Products.

SYMPOSIUM LECTURES

[SYMPOSIUM 2– INTEGRATING TRADITIONAL MEDICINE INTO MODERN HEALTHCARE]

[SL-2.2]

UNLOCKING THE POTENTIAL OF TOCOTRIENOLS IN OCULAR DISEASES

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Tocotrienols, the lesser-known analogues of vitamin E, are differentiated by unsaturated isoprenoid side chains from tocopherol, the widely commercialised form of vitamin E. This structural difference contributes to their enhanced biological activities compared to tocopherols. Tocotrienols exhibit potent antioxidant, anti-inflammatory, and anti-angiogenic properties among other biological properties, making them a potential therapeutic candidate for ocular diseases. In various *in vivo* experimental models, tocotrienols have been shown to attenuate oxidative-nitrosative stress, pro-apoptotic, pro-inflammatory, and pro-angiogenic markers in ocular tissues, including the lens and retina, thereby preserving ocular function. Clinical studies further support these findings, with supplementation with tocotrienols resulting in a significant reduction in diabetic macular edema among patients with diabetic retinopathy, as well as enhancement of functional visual acuity among healthy adults. In terms of underlying mechanisms, tocotrienols modulate nuclear factor erythroid 2–related factor 2 (Nrf2) to exert antioxidant effects, while their anti-inflammatory actions are mediated through NFκB, AMPK, MAPK, PPARγ, and STAT3 signaling pathways. Additionally, tocotrienols also inhibit pro-angiogenic factors such as vascular endothelial growth factor (VEGF), which is relevant in the pathogenesis of diabetic retinopathy and age-related macular degeneration. The inhibition of the growth factors is contributed by tocotrienol's action on PI3K/Akt and Ang-1/Tie-2 signaling pathways. Collectively, these data highlight the significant therapeutic potential of tocotrienols in ocular diseases in comparison to the inconsistent outcomes reported with conventional vitamin E supplementation in human studies.

Keywords: Tocotrienols, Ocular Diseases, Antioxidant, Anti-inflammatory, Anti-angiogenic.

SYMPOSIUM LECTURES

[SYMPOSIUM 2 – INTEGRATING TRADITIONAL MEDICINE INTO MODERN HEALTHCARE]

[SL-2.3]

**ETHNOPHARMACOLOGY IN THE ASIA-PACIFIC:
A JOURNEY THROUGH TRADITIONAL REMEDIES AND MODERN
APPLICATIONS – MEDICINAL PLANTS OF SABAH (NORTH BORNEO):
LEST WE FORGET**

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The discovery of plants and bioactive compounds with the potential to become botanical or pharmaceutical drugs remains a cornerstone of drug innovation. Many of these valuable molecules originate from traditional botanical pharmacopoeias, repositories of centuries-old knowledge that are often underappreciated in modern research. This review highlights the medicinal plants identified in Sabah from 1922 to 2024, analysing their taxonomical distribution, uses, utilisation among ethnic groups, and their potential for clinical uses. The data for this review were gathered from Google Scholar, PubMed, ScienceDirect, Web of Science, and library searches, with all other sources excluded. A keyword combination of “Medicinal” and “Plants” and “Sabah” yielded 21,700 results. Each result was examined, and articles that did not contain information relevant to the topic or coming from non-peer-reviewed journals were excluded. Each of the remaining 87 selected articles was critically reviewed to extract pertinent information. A review of the available data indicates the use of 713 species of plants in Sabah, of which 412 were angiosperms. These plants are primarily utilised to treat diseases or symptoms related to infections, digestive issues, injuries, and pains. Out of these, 144 species employed by the Dusun, Kadazan, Murut, Rungus, and Lundayeh have not yet been subjected to phytochemical or pharmacological studies. Notably, 28 of these species stand out as particularly worthy of further investigation. Sabah’s medicinal plants offer tremendous potential for discovering natural products of therapeutic value.

Keywords: Austronesians, Drug Discovery, Ethnopharmacology, Medicinal Plants, North Borneo, Sabah.

SYMPOSIUM LECTURES

**[SYMPOSIUM 3: ANTIMICROBIAL BREAKTHROUGHS AND THE MICROBIOME:
TRANSFORMING LABORATORY DISCOVERIES INTO CLINICAL SOLUTIONS]**

[SL-3.1]

**THE MICROBIOME–GUT–BRAIN CONNECTION IN PARKINSON’S DISEASE:
BRIDGING RESEARCH AND CLINICAL PRACTICE**

Ai Huey Tan

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Evidence for a close bidirectional link between the brain and the gut has led to a paradigm shift in neurology, especially in the case of Parkinson disease (PD), in which gastrointestinal (GI) dysfunction is a prominent feature. The GI tract harbours a multitude of microorganisms with diverse functions that live in balance with the host. Gut pathogens and gut dysbiosis (ie, alterations in the composition and function of the gut microbiome) have been postulated to cause peripheral inflammatory states or downstream metabolic effects that can trigger or exacerbate the neurodegenerative process in PD, and/or interfere with the absorption of medications used to treat PD symptoms. Understanding the significance of gut dysbiosis in PD is important, as it represents opportunities for the development of novel biomarkers and therapeutic targets, both being major unmet needs for complex neurological disorders.

Excitingly, various microbial-directed therapies such as diet, antibiotics, supplementation of prebiotics, probiotics, and biologics, as well as faecal microbiota transplantation hold promise as potential symptomatic treatment or disease-modifying strategies for PD. Due to the heterogeneity of PD and inter-individual variations in the gut microbiome, it is envisaged that personalisation of these modalities (for example, according to host genetics, microbiome, enteric, and/or immune profiles) will be required to optimise treatment success. Critically, well-designed randomised controlled trials remain the cornerstone to translate microbiome knowledge into evidence-based clinical practice. This lecture will synthesise the emerging evidence on the role of gut microbiome in PD, as well as discuss the future directions in this fast-growing field.

Keywords: Microbiome, gut-brain axis, Parkinson’s disease, clinical practice.

SYMPOSIUM LECTURES

**[SYMPOSIUM 3: ANTIMICROBIAL BREAKTHROUGHS AND THE MICROBIOME:
TRANSFORMING LABORATORY DISCOVERIES INTO CLINICAL SOLUTIONS]**

[SL-3.2]

**UNRAVELING ANTIMICROBIAL RESISTANCE:
MECHANISMS AND CHALLENGES IN NOSOCOMIAL PATHOGENS**

Hui-min Neoh

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Antimicrobial resistance (AMR) is a critical global health issue, particularly in hospital settings where nosocomial infections are prevalent. AMR occurs through mechanisms such as genetic mutations, horizontal gene transfer, biofilm formation, and efflux pumps. Difficulties in maintaining strict infection control practices, diagnostic challenges, and limited sustained efforts in surveillance add complexity to AMR management. While new antibiotics that target gram-positive pathogens have been approved for clinical use consistently since the 1980s, this has not been the case for gram-negative pathogens. Hospital-associated AMR gram-negative pathogens of concern include carbapenem-resistant *Acinetobacter baumannii* and carbapenem-resistant Enterobacterales (CRE); infections of these pathogens are now in critical need of antibiotic innovation and cause significant health burden. Nonetheless, recent advancements in antibiotic discovery for gram-negative infections have produced antibiotics such as cefiderocol, ceftazidime/avibactam, and meropenem/vaborbactam. These antibiotics are effective against multidrug-resistant pathogens such as CRE and *Pseudomonas aeruginosa*, offering new treatment options for challenging infections. Future directions in managing AMR include exploring innovative treatments like bacteriophage therapy and antimicrobial peptides. Global collaboration in surveillance, research, and policy-making is necessary to address AMR effectively. Public awareness campaigns are also crucial for promoting the responsible use of antibiotics, where pharmacists and public health specialists play a vital role in combating AMR through antimicrobial stewardship, promoting the appropriate use of antimicrobials, and continuous education on AMR and stewardship practices. Collaboration in research and development is essential for discovering new antibiotics and alternative therapies.

Keywords: Antimicrobial Resistance, Nosocomial Infections, Gram-negative Pathogens, Antibiotic Stewardship, Bacteriophage Therapy.

SYMPOSIUM LECTURES

**[SYMPOSIUM 3: ANTIMICROBIAL BREAKTHROUGHS AND THE MICROBIOME:
TRANSFORMING LABORATORY DISCOVERIES INTO CLINICAL SOLUTIONS]**

[SL-3.3]

**GUT MICROBIOTA AND PRECISION MEDICINE: BRIDGING MICROBIAL
RESEARCH WITH CLINICAL GASTROENTEROLOGY**

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The past decade has seen an exponential rise in gut microbiota research and interest. Gut microbiota is no longer a science lab project but has significant bedside potential in clinical gastroenterology and precision medicine. Recent research has shown an intricate relationship between luminal microbiota and the gut-brain axis, which is critical in maintaining gut and brain health. The concept of disruption of a stable microbiota abundance and diversity (dysbiosis) is especially pertinent in the management of gut-related diseases, including functional gastrointestinal (GI) disorders, inflammatory bowel disorders, but also a number of neurological (eg, Parkinson's and Alzheimer's) and psychological (depression and stress) disorders. Causes of dysbiosis may be due to meta-exposome, including dietary change, antibiotics, and environmental insults/ climate change, for example, major floods and pollution. Understanding the causes of dysbiosis is key to its management. Probiotics and faecal microbiota transplantation may be useful through modulation of dysbiosis and the gut-brain axis.

Keywords: Gut microbiota, Precision Medicine, Dysbiosis, Gut-brain Axis, Clinical Gastroenterology.

SYMPOSIUM LECTURES

[SYMPOSIUM 4– DRUG REPURPOSING: EMERGING APPROACH TO IDENTIFY POTENTIAL THERAPEUTICS]

[SL-4.1]

REPURPOSING STATINS FOR NEURODEGENERATIVE DISEASE

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Drug repurposing offers a cost- and time-efficient alternative to de novo drug development by identifying new indications for approved therapeutics. Statins, traditionally used for hypercholesterolemia, exhibit pleiotropic effects such as anti-inflammatory, antioxidant and anti-apoptotic. Hence, statins, beyond their well-established lipid-lowering effects, have emerged as promising candidates for drug repurposing in neurodegenerative diseases.

Multiple preclinical and epidemiological studies suggest a neuroprotective role for statins across several CNS disorders. In Alzheimer's disease models, simvastatin and atorvastatin were shown to reduce β -amyloid accumulation and neuroinflammation, improving cognitive performance. In stroke models, statins provide protective effects through enhanced endothelial function and reduced infarct size.

In terms of potential molecular mechanisms, statins modulate cholesterol and sphingolipid rich lipid rafts involved in synaptic signalling, with downstream effects on NMDA receptor-mediated excitotoxicity, a common mechanism in neurodegeneration. Recent studies have also highlighted statins' ability to modulate peroxisome proliferator-activated receptors (PPAR)- α and γ , which regulate neuroinflammatory and apoptotic pathways. Furthermore, statins influence sphingolipid metabolism by reducing ceramide and enhancing sphingosine-1-phosphate (S1P), thus shifting the balance from cell death to survival.

Building on this, our recent work investigated atorvastatin in rats with NMDA-induced retinal injury, a model for glaucoma that is characterised by retinal ganglion cell (RGC) degeneration and progressive optic neuropathy. In silico modelling demonstrated that atorvastatin exhibits high affinity for both PPAR- α and PPAR- γ receptors, with 2.8-fold and 2.6-fold higher interaction energy, respectively, compared to other statins. In the rat model, atorvastatin pre-treatment significantly reduced retinal ceramide ($p < 0.01$) and increased S1P levels ($p < 0.001$), shifting the ceramide/S1P axis toward cell survival. NMDA-induced altered lipid raft integrity was preserved by atorvastatin as evidenced by recovery of retinal flotillin-1 ($p < 0.01$) and caveolin-1 gene and protein expression ($p < 0.05$). As a consequence, atorvastatin lowered the expression of calcium-regulated proteins ($p < 0.001$) indicating prevention of apoptosis activation. Morphological analysis supported these molecular findings. H&E-stained retinal sections showed preserved ganglion cell layer thickness and cellularity in atorvastatin-treated animals compared to the NMDA-only group. Brn3A immunostaining confirmed a significantly higher RGC count in the atorvastatin compared to NMDA group ($p < 0.001$), indicating structural neuroprotection.

In conclusion, atorvastatin mitigates NMDA-induced retinal injury by modulating lipid signalling, preserving membrane microdomains, and preventing RGC apoptosis. These findings support a target-centric approach to repurposing statins for glaucoma and suggest a broader potential for treating neurodegenerative and inflammatory diseases.

Keywords: Statins, neurodegenerative disease, drug repurposing, pharmacology.

SYMPOSIUM LECTURES

[SYMPOSIUM 4– DRUG REPURPOSING: EMERGING APPROACH TO IDENTIFY POTENTIAL THERAPEUTICS]

[SL-4.2]

**UNCOVERING DRUG REPURPOSING CANDIDATES FOR
HEAD AND NECK CANCERS: INSIGHTS FROM SYSTEMATIC
PHARMACOGENOMICS DATA ANALYSIS**

Annie Wai Yeeng Chai

Cancer Research Malaysia, Malaysia

Effective treatment options for head and neck squamous cell carcinoma (HNSCC) are currently lacking. We exploited the drug response and genomic data of the 28 HNSCC cell lines, screened with 4,518 compounds, from the PRISM repurposing dataset to uncover repurposing drug candidates for HNSCC. A total of 886 active compounds, comprising 418 targeted cancer, 404 non-oncology, and 64 chemotherapy compounds, were identified for HNSCC. Top classes of mechanism of action amongst targeted cancer compounds included PI3K/AKT/MTOR, EGFR, and HDAC inhibitors. We have shortlisted 36 compounds with enriched killing activities for repurposing in HNSCC. The integrative analysis confirmed that the average expression of EGFR ligands (AREG, EREG, HBEGF, TGFA, and EPGN) is associated with osimertinib sensitivity. Novel putative biomarkers of response, including those involved in immune signalling and cell cycle regulation, were found to be associated with sensitivity and resistance to MEK inhibitors, respectively. Additionally, pathway enrichment analyses revealed key molecular dependencies that may influence drug responses. We have also developed an RShiny webpage facilitating interactive visualisation to fuel further hypothesis generation for drug repurposing in HNSCC. Our study provides a rich reference database of HNSCC drug sensitivity profiles, affording an opportunity to explore potential biomarkers of response in prioritised drug candidates. By leveraging systematic pharmacogenomic approaches, our work provides insights that could facilitate drug repurposing strategies in HNSCC.

Keywords: Head and Neck Cancer, Drug Repurposing, Pharmacogenomics, HNSCC, Biomarkers.

YOUNG INVESTIGATOR AWARD

[YI-2.0]

**WIRELESS TECHNOLOGY: THE INVISIBLE THREAT?
ASSESSING THE REPRODUCTIVE TOXICITY OF
5G WIRELESS TECHNOLOGIES IN A RODENT MODEL**

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The launch of Malaysia's JENDELA programme by the Malaysian Communications and Multimedia Commission (MCMC) in 2020 marked a strategic national shift toward enhanced digital infrastructure, involving the decommissioning of the 3G network, upgrading of 4G services, and nationwide deployment of 5G technology. Unlike its predecessors, 5G operates at significantly higher frequencies ranging from 3.5 to 28GHz compared to 4G, which typically uses 2.45GHz and 5GHz, offering faster connectivity and broader bandwidth. Despite the suggestion that the millimetre-wave frequencies of 5G may be safer than 4G's microwaves due to limited penetration, this assumption lacks biological validation, particularly on male reproductive health. The testis is particularly vulnerable to radiofrequency radiation (RFR) due to its minimal skin barrier, which allows deeper tissue penetration. Building upon earlier investigations into 4G-related RFR effects, this study evaluated the impact of high-frequency 5G exposure on male reproductive health. Eighteen male rats (N = 18) were randomly assigned into three groups (n = 6 per group). The first group served as the Control (sham-exposed), while the second and third groups were exposed to 3.5GHz and 24GHz frequencies, respectively. The exposure was conducted for seven hours daily over 60 days. All animals were housed in the same room, but at different time frames to accommodate this experimental setting. The antenna or device used for the exposure was placed 20 cm above the animal cages, arranged in a circular configuration. All animals could move freely inside the cage without any movement restrictions. Histological examination revealed frequency-specific testicular damage. The 3.5GHz group exhibited vacuolation and interstitial edema, while the 24GHz group showed detachment of seminiferous tubules from the basement membrane, indicating more severe structural disruption. Additionally, a differential heat shock protein (HSP) response was observed. While 3.5GHz exposure resulted in increased HSP90 expression, consistent with a classical stress response, the 24GHz group showed significant upregulation of HSP27, an anti-apoptotic factor. However, the absence of HSP70 upregulation, which usually functions to counteract apoptosis, in both exposure groups implies the involvement of other stress compensation mechanisms. This was further supported by the sustained caspase-3 upregulation in the 3.5GHz group, indicating ongoing apoptosis. At the same time, no changes were observed in p53 and Bax expression, suggesting that apoptosis occurred independently of the traditional p53 or Bax pathways, possibly via oxidative stress-related mechanisms. Significantly lower caspase-3 levels in the 24GHz group may indicate reduced testicular apoptosis, potentially due to the upregulation of HSP27 as it suppresses pro-apoptotic pathway activation. These molecular disruptions were reflected in functional reproductive outcomes. Both exposed groups demonstrated significant reductions in sperm concentration and motility. When mated with non-exposed females, both groups showed reduced live birth rates, with the 24GHz group also producing pups with significantly lower birth weights. These findings are critical for informing precautionary measures in response to rising trends in male infertility amid widespread 5G deployment. Ultimately, findings on 5G RFR exposure, particularly its impact on male reproductive function, can contribute to the formulation of evidence-based public health policies.

Keywords: 5G Technology, Radiofrequency Radiation, Male Reproductive Health, Testicular Damage, Apoptosis.

FREE ORAL COMMUNICATIONS

[SESSION 1: NATURAL COMPOUNDS AND MICROBIOTA IN MODULATING DISEASE PATHOPHYSIOLOGY]

[OC-1.1]

LACTIPLANTIBACILLUS PLANTARUM LAB12-INDUCED PROTECTION AGAINST LPS-INDUCED OXIDATIVE STRESS AND INFLAMMATION

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Background: Lipopolysaccharide (LPS)-induced inflammation and oxidative stress contribute to various pathological conditions, including systemic inflammatory disorders. Lactiplantibacillus plantarum LAB12-derived postbiotics, rich in short-chain fatty acids (SCFAs), have shown potential in modulating inflammatory and oxidative responses. This study investigates the protective effects of LAB12 postbiotics against LPS-induced oxidative stress and inflammation in a cellular model.

Methods: Lactiplantibacillus plantarum LAB12 was cultured in sterile soymilk and heat-inactivated to produce postbiotics. GC-MS profiling identified SCFAs (acetate, butyrate, propionate, and valerate) in the postbiotic supernatant. Human peripheral blood mononuclear cells (PBMCs) were pretreated with LAB12 postbiotics (1×10^9 CFU/mL) for 24 hours, followed by LPS stimulation (1 μ g/mL) for six hours. Oxidative stress markers (eg, malondialdehyde, superoxide dismutase) and inflammatory cytokines (eg, TNF- α , IL-6) were measured using ELISA and RT-PCR. Cell viability was assessed via MTT assay.

Results: GC-MS profiling confirmed the presence of SCFAs, including acetate, butyrate, propionate, and valerate, in LAB12 postbiotics. LPS stimulation significantly increased oxidative stress markers (malondialdehyde, $p < 0.05$) and pro-inflammatory cytokines (TNF- α , IL-6, $p < 0.05$) in PBMCs. Pre-treatment with LAB12 postbiotics significantly reduced malondialdehyde levels ($p < 0.05$) and enhanced superoxide dismutase activity ($p < 0.05$). Additionally, LAB12 postbiotics suppressed TNF- α and IL-6 expression ($p < 0.05$), indicating anti-inflammatory effects. Cell viability remained unaffected, suggesting no cytotoxicity.

Conclusions: Lactiplantibacillus plantarum LAB12-derived postbiotics mitigate LPS-induced oxidative stress and inflammation in PBMCs, likely due to the presence of SCFAs. These findings highlight the potential of LAB12 postbiotics as a therapeutic agent for inflammatory conditions, warranting further in vivo studies.

Keywords: Lactiplantibacillus Plantarum LAB12, Postbiotics, LPS, Oxidative Stress, Inflammation, Short-Chain Fatty Acids.

FREE ORAL COMMUNICATIONS

[SESSION 1: NATURAL COMPOUNDS AND MICROBIOTA IN MODULATING DISEASE PATHOPHYSIOLOGY]

[OC-1.3]

CASPASE-DEPENDENT AND -INDEPENDENT MECHANISMS OF CURCUMIN AND ITS DIARYLPENTANOID ANALOGUE IN INDUCING CHRONIC MYELOID LEUKEMIC K562 CELL APOPTOSIS

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Background: Chronic myeloid leukemia (CML) is one of the most common leukemia types among the elderly. Curcumin is a natural bioactive compound isolated from the spice turmeric with promising antioxidative, anti-inflammatory, anti-diabetic, antimicrobial, and anticancer properties. However, the clinical use of curcumin is limited by its poor bioavailability. Chemical modification of the curcumin keto-enol bridge has been shown to improve its bioavailability without compromising its safety profile. We synthesised MS13, a curcumin diarylpentanoid analogue with a modified keto-enol bridge, which has demonstrated promising anticancer activities against several cancer cell lines, including CML K562 cells. In this study, we aimed to determine the underlying apoptotic pathways activated in curcumin and MS13-treated K562 cells.

Methods: Apoptosis induction in K562 cells with 24h treatment of curcumin and MS13 at their respective IC50 values was assessed using the apoptosis assay. The involvement of executioner caspases activation was evaluated via the caspase-3/7 GLO luminescent assay. Lastly, the general caspase inhibitor (z-VAD-fmk) was used to confirm the role of caspase-3/7 activation upon treatment.

Results: Curcumin and MS13 induced apoptosis in K562 cells at their respective IC50 values. Both compounds activated executioner caspases, with MS13 showing a greater potency and inducing faster and higher levels of caspase activity. Parallely, z-VAD-fmk did not significantly suppress curcumin-induced cell death, suggesting the involvement of a caspase-independent mechanism.

Conclusions: Keto-enol bridge modification enhances the apoptotic potency and alters the mechanism of action of curcumin. Further studies are needed to elucidate the upstream molecular mechanisms and targets in MS13-induced apoptosis.

Keywords: Apoptosis, Caspase, Cell Death, Curcumin, Diarylpentanoid, MS13.

FREE ORAL COMMUNICATIONS

[SESSION 1: NATURAL COMPOUNDS AND MICROBIOTA IN MODULATING DISEASE PATHOPHYSIOLOGY]

[OC-1.4]

**PROTECTIVE EFFECTS OF KELULUT HONEY ON METABOLIC
SYNDROME-ASSOCIATED BONE LOSS:
MODULATION OF BIOMECHANICS AND MARROW ADIPOSITY**

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Background/Objective: Metabolic syndrome (MetS), characterised by abdominal obesity, dyslipidemia, hyperglycemia, and hypertension, is a significant risk factor for cardiovascular disease and type 2 diabetes. Emerging evidence links MetS to bone loss, potentially mediated by oxidative stress and bone marrow adiposity. This study investigated the effects of Kelulut honey (KH), a polyphenol-rich functional food, on bone health in a high-carbohydrate high-fat (HCHF) diet-induced MetS rat model.

Methods: Eighteen male Wistar rats were divided into three groups: normal control, HCHF diet, and HCHF diet supplemented with KH (1 g/kg/day) for eight weeks. Bone densitometry, biomechanical strength, microstructural histomorphometry, redox status, and gene expression were assessed.

Results: The HCHF diet reduced bone flexibility (decreased displacement and strain, increased stiffness) and elevated bone marrow adiposity, without altering bone mineral density or microstructure. KH supplementation reversed these biomechanical deficits and reduced bone marrow adipocyte accumulation. While antioxidant enzyme levels remained unchanged, KH decreased malondialdehyde, indicating reduced oxidative stress. No significant changes were observed in osteogenic gene expression (Opg, Ocn, Rankl, Runx2 and Ctsk), but Pparg was downregulated in HCHF-fed rats, unaffected by KH.

Conclusions: These findings suggest that KH mitigates HCHF-induced bone deterioration by improving biomechanical properties and reducing marrow adiposity, potentially via antioxidant mechanisms. Further research is warranted to elucidate KH's molecular pathways and optimize dosing for skeletal benefits.

Keywords: Bone marrow adipocyte, bone strength, high carbohydrate high fat diet, stingless bee honey, oxidative stress.

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[SESSION 1: NATURAL COMPOUNDS AND MICROBIOTA IN MODULATING DISEASE PATHOPHYSIOLOGY]

[OC-1.5]

CHARACTERISATION OF GUT MICROBIOTA METABOLIC ACTIVITIES IN ALZHEIMER'S DISEASE USING PHENOTYPIC MICROARRAY: A PRELIMINARY STUDY

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Background: Alzheimer's disease (AD) is increasingly associated with disruptions in the gut microbiota and its metabolic functions, which may influence disease onset and progression via the gut-brain axis. Gut microbiota dysbiosis can alter the production of key metabolites, reducing beneficial compounds or increasing harmful ones that affect brain health. Metabolic dysfunctions, including those in energy production, cholesterol metabolism, and neurotransmitter synthesis, are also implicated in AD pathology. However, the metabolic activity of gut microbiota in AD remains poorly understood, limiting our ability to interpret the functional impact of these microbial changes. This study aimed to characterise alterations in gut microbiota metabolic activity in AD patients using phenotype microarray (PM) profiling.

Methods: Stool samples were collected from eight participants (four AD patients and four healthy controls). AD diagnosis was confirmed using the Mini-Mental State Examination (MMSE; score < 20), alongside the DSM-IV and NINCDS-ADRDA criteria. Pooled stool samples from each group were analysed using Biolog Phenotype MicroArray plates PM1 and PM2A, which assess the utilisation of 190 carbon sources over a 72-hour incubation period. The results revealed marked differences in metabolic activity between the AD and control groups.

Results: A total of 148 substrates showed altered utilisation patterns, particularly involving carbohydrates and amino acids, indicating a shift in microbial functional capacity.

Conclusions: In conclusion, this study demonstrates that gut microbiota in Alzheimer's disease exhibits distinct metabolic alterations, potentially contributing to disease progression through impaired microbial-derived metabolite production.

Keywords: Alzheimer's Disease, Gut Microbiota, Phenotype Microarray, Metabolic Activities, Gut-Brain-Axis.

FREE ORAL COMMUNICATIONS

[SESSION 2: NEUROPHARMACOLOGY & RETINAL PROTECTION]

[OC-2.1]

ATORVASTATIN RESTORES SPHINGOLIPID BALANCE AND REDUCES LIPID ACCUMULATION IN A RAT MODEL OF RETINAL EXCITOTOXICITY

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Introduction: Glaucoma is a progressive optic neuropathy marked by retinal ganglion cell (RGC) loss. NMDA-mediated excitotoxicity is the underlying pathophysiology. It is exacerbated by elevated retinal ceramide and reduced sphingosine-1-phosphate (S1P) levels, indicating dysregulated sphingolipid rheostat. Accumulation of triglycerides and cholesterol further enhances RGC vulnerability. Atorvastatin (ATV) may offer RGC protection by restoring lipid homeostasis and sphingolipid rheostat. This study evaluated the effects of ATV on sphingolipid regulation and lipid profiles in a rat model of NMDA-induced retinal excitotoxicity.

Methods: Two groups of rats (n=5) received either DMSO (vehicle) or ATV (dose determined by a pilot study), followed by NMDA (80 mM/ μ L), bilaterally and intravitreally. Another group of animals remained untreated. Retinal samples were collected at six hours, day 1, and day 7 post-treatment for quantifying S1P, sphingosine, ceramide, sphingosine kinases (SphK1, SphK2), triglycerides, LDL-cholesterol, and total cholesterol using ELISA. Retinal morphology was assessed by haematoxylin and eosin (H&E) staining.

Results: ATV-treated group showed greater retinal S1P and SphK1/2 expression and lower sphingosine and ceramide levels at all time-points compared to the NMDA-treated group ($p \leq 0.0001$). Additionally, retinal triglycerides, LDL-cholesterol, and total cholesterol were lower on day 7 in the ATV-treated compared to the NMDA-treated group ($p \leq 0.05$ to $p \leq 0.0001$). Histological analysis confirmed that ATV preserved retinal architecture against NMDA-induced injury.

Conclusions: ATV protects against NMDA-induced retinal injury by preventing NMDA-induced imbalance of sphingolipid rheostat and lipid accumulation in rat retinas. Further studies may reveal the potential of this as antiglaucoma agents.

Keywords: Atorvastatin, excitotoxicity, sphingolipid rheostat, neuroprotection, retinal ganglion cells (RGCS).

FREE ORAL COMMUNICATIONS

[SESSION 2: NEUROPHARMACOLOGY & RETINAL PROTECTION]

[OC-2.2]

EXCITOTOXICITY-MEDIATED MODULATION OF THE RETINAL RENIN-ANGIOTENSIN SYSTEM: DISSECTING THE INTERPLAY IN A RAT MODEL

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Background: The renin–angiotensin system (RAS) is locally expressed in neuronal tissues, including the retina, where it contributes to neurodegenerative processes. This study investigated how N-methyl-D-aspartate (NMDA)-induced excitotoxicity affects the expression of RAS components in rat retina and the associated structural changes.

Methods: Sprague-Dawley rats received intravitreal injections of either phosphate-buffered saline (PBS) or NMDA (160 nmol). On day 7, retinas were analysed for mRNA and protein expression of RAS components using real-time PCR and ELISA. Retinal morphology was assessed via hematoxylin and eosin staining and morphometry.

Results: NMDA exposure significantly increased the expression of classical RAS components: angiotensin-converting enzyme (ACE) (mRNA 1.86-fold, $p < 0.01$; protein 2.03-fold, $p < 0.001$), angiotensinogen (mRNA 1.99-fold, $p < 0.05$; protein 2.35-fold, $p < 0.05$), AT1R (mRNA 2.28-fold, $p < 0.0001$; protein 1.75-fold, $p < 0.0001$), and angiotensin II (protein 2.13-fold, $p < 0.001$). In contrast, alternate RAS components were significantly reduced: ACE2 (mRNA decreased 3.09-fold, $p < 0.01$; protein 1.51-fold, $p < 0.05$), angiotensin-(1–7) and -(1–9) (2.41-fold and 2.37-fold decrease; $p < 0.001$), and MAS receptor (mRNA decreased 1.95-fold, $p < 0.01$).

Conclusions: Morphometric analysis showed thinning of the ganglion cell layer and reduced retinal cell density ($p < 0.05$), indicating NMDA-induced retinal damage. These results suggest that excitotoxicity disrupts the balance of RAS towards its classical pro-apoptotic axis, contributing to neurodegeneration.

Keywords: Retinal Excitotoxicity, Renin–Angiotensin System, NMDA, Angiotensin II, Ganglion Cell Layer, Neuroprotection, Normotensive Glaucoma.

FREE ORAL COMMUNICATIONS

[SESSION 2: NEUROPHARMACOLOGY & RETINAL PROTECTION]

[OC-2.3]

**MECHANISMS OF THE PROTECTIVE EFFECTS OF ATORVASTATIN
AGAINST NMDA-INDUCED RETINAL INJURY IN RATS:
FOCUS ON PPAR- γ /CREB PATHWAY**

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Introduction: Atorvastatin (ATV) is known to exert neuroprotective effects; however, the underlying mechanisms remain unclear. Since NMDA-induced excitotoxicity underlies several neurodegenerative conditions including glaucoma, a leading cause of irreversible blindness, we investigated the involvement of PPAR- α /CREB pathway in the neuroprotective effect of ATV against NMDA-induced retinal injury in rats.

Methods: Phase-1 of the study determined ATV's optimum dose for anti-apoptotic effect and included six groups of rats that received either one of the five doses of ATV(0.1 μ M, 0.5 μ M, 1.0 μ M, 20 μ M, 100 μ M) or DMSO (vehicle) followed by NMDA(160 mM), and another group that remained untreated. Retinal morphology among seven groups was compared, and BAX/Bcl-2 was quantified. For Phase-2, rats were divided into five groups: untreated, NMDA-treated, ATV pre-treatment before NMDA exposure, PPAR-alpha antagonist and ATV pre-treatment before NMDA and PPAR-gamma antagonist and ATV pre-treatment before NMDA. CREB, BDNF, NT3, PPAR-alpha, and PPAR-gamma expressions were measured in retinal samples using ELISA.

Results: Among five doses, the 20 μ M dose showed the most prominent protective effect of ATV against NMDA-induced changes in retinal morphology, with the lowest BAX/Bcl2 ratio compared to other ATV-treated groups. NMDA group showed significantly lower NT3 levels compared to the control ($p < 0.0001$). ATV-treated group showed greater CREB, BDNF ($p < 0.0108$) and NT3 ($p < 0.0001$) levels compared to the NMDA group. Expression of PPAR-alpha was lower in the alpha antagonist and ATV groups than in the NMDA group ($p < 0.0006$).

Conclusions: ATV seems to exert a protective effect against NMDA-induced retinal injury by activating PPAR- α /CREB/BDNF-NT3 pathway. Further studies are being conducted to substantiate the results.

Keywords: Atorvastatin, excitotoxicity, retina, glaucoma, PPAR- α /CREB pathway, neuroprotection, retinal ganglion cells (RGCs).

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[SESSION 2: NEUROPHARMACOLOGY & RETINAL PROTECTION]

[OC-2.4]

ATORVASTATIN ATTENUATES OXIDATIVE STRESS BY STABILISING LIPID RAFTS IN NMDA-INDUCED RETINAL GANGLION CELL LOSS

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Background: Progressive vision loss in glaucoma is driven by retinal ganglion cell (RGC) apoptosis caused by calcium overload from excessive glutamate receptor activation, which may be modulated via lipid rafts in the cell membrane. This study investigates atorvastatin's potential to mitigate excitotoxic damage by stabilising lipid raft integrity.

Methods: Sixteen male Sprague-Dawley rats were randomly divided into three groups (n=22 total): control, vehicle treatment, and atorvastatin (15 µM) treatment. The vehicle and atorvastatin groups received NMDA (80 nM, 2 µL, intravitreally and bilaterally) 20 hours post-treatment. Treatments were administered, and seven days later, eyes were enucleated for histopathological analysis using H&E staining and Brn3a immunolabeling. Gene and protein expression of lipid raft markers caveolin-1 and flotillin-1 were assessed via RT-PCR and Western blot. NOX1 and NOX2 levels were evaluated by ELISA and RT-PCR.

Results: NMDA exposure downregulated caveolin-1 and flotillin-1 expression (p=0.05) and upregulated NOX2 oxidase at gene and protein levels (p=0.001). Atorvastatin treatment restored caveolin-1 and flotillin-1 expression (p<0.05) and reduced NOX2 levels (p<0.01). Histopathological analysis revealed improved RGC survival in the atorvastatin-treated group, with increased Brn3a-positive cells compared to the vehicle-treated group (p<0.05).

Conclusions: These findings suggest that atorvastatin mitigates NMDA-induced RGC loss by stabilising lipid rafts and reducing oxidative stress via modulation of NOX2 expression, indicating its potential as a neuroprotective agent in glaucoma.

Keywords: Atorvastatin, NMDA, Retinal Ganglion Cells, Oxidative Stress, Lipid Rafts, Neuroprotection.

FREE ORAL COMMUNICATIONS

[SESSION 2: NEUROPHARMACOLOGY & RETINAL PROTECTION]

[OC-2.5]

TOPICAL RU615 IMPROVES TRABECULAR MESHWORK AND RETINAL HISTOLOGY IN STEROID-INDUCED OCULAR HYPERTENSIVE RAT MODEL

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Introduction: Prolonged corticosteroid use can lead to ocular hypertension (OHT) and structural damage to ocular tissues, including the trabecular meshwork (TM) and retina. RU-615, a derivative of imidazo[1,2-a]benzimidazole, has been shown to counteract the steroid-induced increase in IOP. This study aimed to evaluate the effects of topical RU615 on TM and retinal histology in a steroid-induced OHT rat model.

Methods: Eighteen Sprague-Dawley rats were randomly divided into three groups (n=6/group): (1) normal control, (2) prednisolone-treated (1% topical, twice daily), and (3) co-treatment with prednisolone and RU615 (0.1% topical, twice daily). Treatments were administered for 21 days. At endpoint, IOP was measured, followed by euthanasia and enucleation for histological analysis of the TM and retina.

Results: Topical RU615 treatment resulted in reduction of IOP compared to the prednisolone-only group, although the difference was not statistically significant. Histological analysis showed that TM thickness and cellularity were significantly reduced in the RU615-treated group compared to the prednisolone group. Additionally, retinal morphometry revealed improved ganglion cell layer (GCL) thickness and increased retinal cell count in RU615-treated eyes, suggesting a neuroprotective effect on retinal ganglion cells (RGCs).

Conclusions: Topical RU615 demonstrates beneficial effects on TM structure and retinal morphology in a steroid-induced OHT model, with potential neuroprotective properties. Although IOP reduction was not statistically significant, the histological improvements support further investigation of RU615 as a therapeutic agent for steroid-induced ocular hypertension.

Keywords: Ocular Hypertension, Glaucoma, Intraocular Pressure, Trabecular Meshwork, Retina.

FREE ORAL COMMUNICATIONS

[SESSION 2: NEUROPHARMACOLOGY & RETINAL PROTECTION]

[OC-2.6]

**EFFECTS OF LACTOPLANTIBACILLUS PLANTARUM (LAB12)-DERIVED
POSTBIOTICS ON COGNITIVE FRAILTY AND GUT HEALTH OF
D-GALACTOSE-INDUCED AGEING AND NATURALLY AGED
SPRAGUE DAWLEY RATS**

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Background: Cognitive frailty (CF), characterised by the coexistence of physical frailty and cognitive impairment without being diagnosed as dementia, significantly increases the risk of adverse health outcomes especially in the older adults. The continuous growth of global ageing population calls for alternative approaches that could potentially reverse CF. Recent findings highlighted the roles of gut microbiota in modulating cognitive health and muscle function. In this regard, postbiotics may potentially reverse CF by restoring gut microbiota balance. This study aimed at examining the effects of Lactiplantibacillus plantarum LAB12-derived postbiotics against the CF parameters in D-galactose-induced ageing (animal model 1) and naturally aged Sprague-Dawley rats (animal model 2).

Methods: LAB12 were cultured in sterile soymilk overnight. The fermented culture were heat-inactivated at 80°C for ten minutes. The models, D-galactose-induced ageing SD rats (n=6/group; 27 weeks old) and naturally aged SD rats, (n=3/group; 83 weeks old), were supplemented with 1×10⁹ CFU/mL L. plantarum LAB12-derived postbiotics for 9 and 12 weeks, respectively, using syringe feeding. A series of behavioural (Morris water maze (MWM) test) and physical tests (rotarod and grip strength tests) were conducted.

Results: Both animal models which were supplemented with LAB12-derived postbiotics exhibited significantly shorter escape latency (p<0.005), indicating improved spatial learning and memory recall. The postbiotic-supplemented rats were presented with significant increased muscle strength (p<0.05).

Conclusions: LAB12-derived postbiotics demonstrated the potential as a gut-based strategy for prevention against CF. This warrants further study into the relationship between CF and gut microbiota.

Keywords: Cognitive Impairments, Physical Frailty, D-galactose, Postbiotics, Ageing.

FREE ORAL COMMUNICATIONS

[SESSION 2: NEUROPHARMACOLOGY & RETINAL PROTECTION]

[OC-2.7]

**EFFECTS OF EXERCISE ON COGNITIVE FRAILTY PARAMETERS
IN D-GALACTOSE-INDUCED AGEING AND NATURALLY AGED
SPRAGUE DAWLEY RATS**

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Background: Cognitive frailty (CF), which is characterised by physical frailty and mild cognitive impairment (MCI) that heightened one's susceptibility to unfavourable health effects, presents a significant challenge to the growing ageing population. However, effective interventions that could mitigate CF remain elusive. Whilst exercise has been increasingly explored for its potential benefits, previous research focused on cognitive impairment or physical frailty as separate entities rather than addressing the dual nature of CF. This study aimed at examining the effects of exercise on CF parameters in D-galactose-induced ageing (animal model 1) and naturally aged Sprague Dawley rats (animal model 2).

Methods: Animal model 1 (male, 26 weeks old; n=7/group) is a chemically accelerated ageing model, while animal model 2 (male; n=3-4/group) is a naturally aged model on which intervention was started at 20 months old (midlife) until 23 months old (aged). The rats were subjected to the motorised wheel running exercise regimen (>9 weeks) before physical (grip strength and rotarod) and behavioural [Morris water maze (MWM)] assessments.

Results: The exercise intervention significantly ($p<0.05$) improved the physical parameters of both animal models, indicating possible restorative effects of exercise on muscle strength and motor coordination. MWM test also showed significant ($p<0.05$) reduction in escape latency and increased time spent in the target quadrant by both animal models, indicating improved spatial learning.

Conclusions: These findings indicated the potential of exercise in improving CF parameters, hence warrants further investigations into the mechanisms underlying the reversal of CF, especially in relation to gut health.

Keywords: Cognitive Frailty, Exercise, Ageing, d-galactose, Sprague Dawley.

FREE ORAL COMMUNICATIONS

[SESSION 2: NEUROPHARMACOLOGY & RETINAL PROTECTION]

[OC-2.8]

THE EFFECT OF ACACIA HONEY ON MODULATING CUMS-INDUCED STRESS-DEPRESSION-LIKE BEHAVIOURS IN SPRAGUE DAWLEY RATS

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Stress-depression (SD) is characterised by persistently depressed mood, anxiety, and an inability to experience pleasure. Conventional treatment using antidepressants is often unsuitable for patients with chronic heart disease or liver damage, underline the need for natural alternatives like Acacia honey (AH). This study aimed to investigate the effects of AH on SD in Sprague-Dawley rats using a chronic unpredictable mild stress (CUMS) model. Forty-two male rats were divided into seven groups: normal control (NC), AH, amitriptyline (AMT), CUMS, CUMS+AH, CUMS+AMT, and CUMS+AH+AMT.

After 28 days of CUMS exposure, behavioral parameters were assessed using the open field test (OFT). Data were analysed using one-way ANOVA followed by Tukey's post-hoc test ($P < 0.05$). CUMS-induced stress significantly increased faecal count (3.67 ± 0.21), corner zone entries (23.00 ± 2.08), and immobility time ($184.28 \pm 5.04s$), while reducing climbing behavior (7.67 ± 0.67), distance traveled (5.09 ± 0.10 m), centre zone entries (1.60 ± 0.60), and time spent in the centre zone ($11.08 \pm 0.54s$). AH supplementation reversed these effects, improving stress-depression-like behaviors. In contrast, AMT improved only faecal count, climbing behavior, and centre zone time, while AH+AMT co-supplementation impacted only distance travel and centre zone entries.

In conclusion, AH effectively ameliorates stress-depression-like behaviors in CUMS-induced rats, suggesting its potential as a complementary therapy for stress-related disorders. Further studies are recommended to explore the underlying mechanisms of AH.

Keywords: Acacia Honey, Behaviour, CUMS-induced Stress-Depression Model.

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[SESSION 2: NEUROPHARMACOLOGY & RETINAL PROTECTION]

[OC-2.9]

**ENVIRONMENTAL ENRICHMENT ATTENUATES ASTROCYTE ACTIVATION
AND MODULATES GLUTAMATE LEVELS
IN STREPTOZOTOCIN-INDUCED DIABETIC RATS**

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Diabetes mellitus (DM) is a chronic metabolic disorder characterised by persistent hyperglycemia and is associated with various complications, including neuroinflammation and cognitive decline. Astrocytes, a type of glial cell, become activated in chronic diabetic states, contributing significantly to neuroinflammation. While environmental enrichment (EE) has been shown to enhance cognitive function and reduce neuroinflammation in several neurological conditions, its specific effect on astrocyte activation in diabetes remains underexplored.

This study aimed to investigate the impact of EE on astrocyte activation in streptozotocin (STZ)-induced diabetic rats and to evaluate the effects of EE and insulin on body weight, fasting blood glucose (FBG), and brain tissue glutamate levels. Thirty male albino Wistar rats were randomly assigned into five groups: Control, DM, DM+Insulin, DM+EE, and DM+Insulin+EE. Diabetes was induced using STZ in all groups except the Control. Treatment groups received insulin, EE, or a combination of both to assess their therapeutic potential.

The results demonstrated that both EE and insulin independently mitigated weight loss and reduced FBG levels, with the combination therapy showing the most pronounced effect. Immunohistochemical analysis revealed that EE alone reduced astrocyte activation and normalised glutamate levels in the brain; these effects were significantly amplified when combined with insulin treatment. In conclusion, EE exhibits neuroprotective effects by attenuating astrocyte activation and glutamate excitotoxicity in diabetic conditions. These findings highlight EE as a promising non-pharmacological adjunct therapy alongside insulin to mitigate neurological complications associated with diabetes.

Keywords: Environmental Enrichment, Diabetes Mellitus, Streptozotocin, Astrocyte, Glutamate.

FREE ORAL COMMUNICATIONS

[SESSION 3: ADVANCES IN CARDIOVASCULAR AND METABOLIC RESEARCH]

[OC-3.1]

**INFLUENCE OF PERIVASCULAR ADIPOSE TISSUE (PVAT)
AND ENDOTHELIUM ON THE RESPONSE TO HYPOXIA
IN RAT THORACIC AORTA**

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Background: Perivascular adipose tissue (PVAT) regulates vascular function and in obese subjects it can become dysfunctional. Obese PVAT is exposed to hypoxia which can modulate its function. This study aims to investigate how exposure to periods of hypoxia affects the regulatory activity of PVAT in both endothelium-intact and denuded arteries.

Methods: Thoracic aorta was prepared from male SD rats. Some rings were left with PVAT and endothelium intact and in other rings these were removed. Dose-response curves to phenylephrine (PE; 1nM to 30µM) were constructed under normoxic conditions or after exposure to hypoxia (30 mins with 95% N₂/5% CO₂).

Results: Under normoxia, the presence of PVAT had an anti-contractile effect against PE (134.71% vs 99.42% of KPSS contraction, n=5-6; p < 0.05). In contrast, under hypoxic conditions there was no significant difference in PE-induced contraction between endothelium-intact rings with or without PVAT (n=5-7). In rings with endothelium removed, the presence of PVAT significantly increased (p < 0.05) contraction on exposure to hypoxia (n=5-6). Paradoxically, in the absence of PVAT, rings with intact endothelium exposed to hypoxic conditions contracted significantly less to PE compared to control rings (p<0.05; n=5).

Conclusions: In rat thoracic aorta, the anticontractile effect of PVAT is sensitive to oxygenation and is reversed by hypoxia. Endothelium also has an anti-contractile effect under normoxic conditions which was augmented by hypoxia. Future work will study the effect of hypoxia on PVAT-derived mediators which could account for these effects.

Keywords: Thoracic Aorta, Perivascular Adipose Tissue, Endothelium, Hypoxia.

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[SESSION 3: ADVANCES IN CARDIOVASCULAR AND METABOLIC RESEARCH]

[OC-3.2]

PROFILING OF UPREGULATED miRNAS IN ENDOTHELIAL CELLS EXPOSED TO HYPERTENSIVE DISORDERS OF PREGNANCY: A BIOINFORMATIC ANALYSIS

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Offspring of mothers with hypertensive disorders of pregnancy (HDP) have an increased risk of developing endothelial dysfunction and cardiovascular disease (CVD) later in life. MicroRNAs (miRNAs), which are important post-transcriptional regulators of gene may play a role in the early development of endothelial dysfunction. However, studies on miRNA expression profile in endothelial cells from offspring of HDP pregnancies are still limited.

The aim of this study was to characterise the miRNA expression profile in human umbilical vein endothelial cells (HUVECs) isolated from newborns of mothers with HDP. HUVECs were obtained from both normotensive and hypertensive pregnancies. RNA sequencing identified eight miRNAs that were significantly upregulated in HUVECs exposed to HDP ($p < 0.05$). The potential target genes of these miRNAs were predicted using four databases: miRDB, TargetScan, DIANA-microT-CDS and miRWalk.

Subsequent analyses of gene ontology, pathway enrichment and protein-protein interaction (PPI) revealed that the predicted target genes are involved in critical biological processes and signalling pathways, including angiogenesis and cellular senescence, which are associated with endothelial dysfunction and increased cardiovascular risk. Among the differentially expressed miRNAs, hsa-miR-196a-5p showed the most marked upregulation. Its increased expression was further validated using stem-loop RT-qPCR and showed a 6-fold increase in hypertensive HUVECs compared to controls ($p < 0.01$).

These results provide new insights into the potential role of miRNAs in mediating vascular programming associated with HDP and suggest that specific miRNAs such as hsa-miR-196a-5p may serve as early biomarkers or therapeutic targets to prevent CVD in affected offspring.

Keywords: Endothelial Dysfunction, Human Umbilical Vein Endothelial Cells, Hypertensive Disorders of Pregnancy, microRNA, RNA Sequencing.

FREE ORAL COMMUNICATIONS

[SESSION 3: ADVANCES IN CARDIOVASCULAR AND METABOLIC RESEARCH]

[OC-3.4]

CARDIOPROTECTIVE EFFECT OF ETLINGERA ELATIOR FLOWER (BUNGA KANTAN) AGAINST ISOPRENALINE-INDUCED MYOCARDIAL INFARCTION IN A HYPERCHOLESTEROLAEMIC RAT MODEL

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Background: Acute myocardial infarction (MI) remains the leading cause of mortality worldwide, with elevated cholesterol levels and pro-inflammatory mediators playing key roles in its pathogenesis. *Etlingera elatior* (*E. elatior*) flowers exhibit *in vitro* anti-hypercholesterolaemic and anti-inflammatory properties, but their mechanisms of action remain unclear. This study aimed to evaluate the potential of an aqueous extract of *E. elatior* flower (AEEe) as a cholesterol-lowering agent and cardioprotective in a rat model of isoprenaline-induced MI under hypercholesterolaemic conditions.

Methods: Male Sprague-Dawley rats were divided into Control and Hypercholesterolaemic (HC) groups, with the HC group receiving a high-cholesterol diet for six weeks. Subsequently, HC rats continued the same diet and were subdivided into four groups: HC, HC-MI (MI induction), HC-MI + E1000 (Treated with 1000 mg/kg AEEe), and HC-MI + Ato20 (Treated with 20 mg/kg of atorvastatin) for another six weeks. MI was induced using isoprenaline (ISO) (85 /mg/kg, s.c) for two consecutive days at the end of treatment.

Results: Total cholesterol and LDL-cholesterol levels were significantly increased in all HC groups compared to control but were attenuated with AEEe. AEEe also significantly reduced inflammatory markers (IL-6 and TNF- α) compared to the HC-MI group. Histopathology examination revealed myocardial necrosis, inflammatory cell infiltration, and collagen deposition in the HC-MI group, which were improved with AEEe treatment.

Conclusions: *E. elatior* flower protects against isoprenaline-induced MI in hypercholesterolaemic rats, potentially through its cholesterol-lowering and anti-inflammatory effects. These findings support its potential as an adjuvant therapy to reduce MI risk.

Keywords: *Etlingera Elatior*, Myocardial Infarction, Inflammation, Interleukin-6, Tumour Necrosis Factor-alpha.

FREE ORAL COMMUNICATIONS

[SESSION 3: ADVANCES IN CARDIOVASCULAR AND METABOLIC RESEARCH]

[OC-3.5]

**ANTIHYPERTENSIVE AND VASCULAR PROTECTIVE EFFECTS
OF LIGNOSUS RHINOCERUS TM02® THROUGH ACE/ANG II/AT1R
AND NO PATHWAY REGULATION**

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Background: Hypertension is a risk factor for cardiovascular diseases such as coronary artery disease, heart failure and stroke. Lignosus rhinocerus (Cooke) Ryvardeen (also known as Tiger Milk mushroom), has been reported to exhibit a range of pharmacological effects, such as anti-inflammatory, anti-proliferative, antioxidative, immunomodulatory and anti-asthmatic activities. Previous studies demonstrated that L. rhinocerus exhibits in vitro angiotensin converting enzyme (ACE) inhibitory activity, and our recent work suggests its potential antihypertensive and vascular protective effects in vivo. However, the role of the renin-angiotensin system (RAS) pathway in these effects remains unclear. Therefore, this study investigated the involvement of the RAS pathway in the antihypertensive and vascular protective effects of L. rhinocerus TM02® sclerotia in Spontaneously Hypertensive Rats (SHR).

Methods: Plasma levels of angiotensin II (Ang II) and ACE, along with vascular ACE activity, and vascular (aorta) gene and protein expression of components in the ACE/AT1R/NO signalling pathway, were measured. Aortic tissue was analysed for structural changes using various staining methods.

Results: While plasma Ang II and ACE levels remained unchanged across treatment groups, L. rhinocerus TM02® significantly reduced vascular ACE activity. It also downregulated the gene and protein expression of ACE, AT1, NOX4, NFκB, and p-NFκB, while upregulating AT2, eNOS, and p-eNOS in the vasculature. Histological analyses revealed improved aortic structural integrity and reduced fibrosis in the treated groups.

Conclusions: Collectively, L. rhinocerus TM02® sclerotia exerts antihypertensive and vascular protective effects, likely through modulation of the ACE/Ang II/AT1R/NO signaling axis to reduce oxidative stress.

Keywords: Hypertension, Lignosus rhinocerus TM02®, ACE, Angiotensin II, Nitric oxide, Vascular protection.

FREE ORAL COMMUNICATIONS

[SESSION 3: ADVANCES IN CARDIOVASCULAR AND METABOLIC RESEARCH]

[OC-3.6]

**RESTORATION OF ENDOTHELIAL FUNCTION IN OBESE MICE BY
LIGNOSUS RHINOCERUS TM02[®] AND xLr[®] VIA
MODULATION OF OXIDATIVE STRESS**

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Background: Obesity-induced endothelial dysfunction is a significant risk factor for the development of atherosclerosis. *Lignosus rhinocerus* (Tiger Milk mushroom, TMM) has demonstrated various bioactivities, including anti-inflammatory, antioxidant and anticancer properties. However, its role in its potential in mitigating endothelial dysfunction associated with obesity remains unclear. This study investigated the effects of TMM supplementation, using uniquely cultivated *L. rhinocerus* TM02[®] sclerotia powder (TM02[®]) or its patented cold-water extract (xLr[®]) on vascular functions in an obese mice model.

Methods: Obese C57BL/6J mice, induced by high-fat diet, were supplemented with TM02[®] (500 mg/kg), xLr[®] (50 mg/kg) or aminoguanidine (200 mg/kg) orally for four weeks. The vascular reactivity in aorta was measured using a wire myograph, whereas the effects of TMM on the formation of atherosclerotic plaque, reactive oxygen species (ROS) production and nitric oxide (NO) bioavailability were assessed by histological and fluorescence staining.

Results: The results showed that both TM02[®] and xLr[®] oral supplementation significantly alleviated endothelial dysfunction in obese mice. Furthermore, supplementation also reduced plaque formation, reduced elevated ROS production and restored NO bioavailability.

Conclusions: These findings suggest that both TM02[®] and xLr[®] restores endothelial function in obesity by regulating oxidative stress, hence enhancing NO bioavailability. This study delineates the prospective of using TM02[®] and xLr[®] as natural adjunct therapies for preserving vascular health and managing obesity-related cardiovascular complications.

Keywords: Obesity, *L. rhinocerus* TM02[®], Cold-water Extract xLr[®], Endothelial Dysfunction, Oxidative Stress.

FREE ORAL COMMUNICATIONS

[SESSION 3: ADVANCES IN CARDIOVASCULAR AND METABOLIC RESEARCH]

[OC-3.7]

**SPHINGOSINE KINASE 1/SPHINGOSINE-1-PHOSPHATE RECEPTOR 2
AXIS MODULATES MACROPHAGE-MEDIATED INDUCIBLE NITRIC OXIDE
SYNTHASE EXPRESSION AND NITRIC OXIDE PRODUCTION
IN ADIPOCYTES**

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Background: In obesity, adipose tissue becomes chronically inflamed due to macrophage infiltration and this is associated with increased expression of inducible nitric oxide synthase (iNOS). Sphingosine kinase (SphK1) produces sphingosine-1-phosphate (S1P), which may regulate adipose tissue inflammation via the S1P-specific G protein-coupled receptor, S1PR2. Therefore, the objective of the present study was to investigate the role of the SphK1/S1P/S1PR2 axis in mediating iNOS expression and nitric oxide (NO) production in adipocytes.

Methods: RAW 267.4 macrophages were stimulated with LPS 100 ng/ml for eight hours in the presence and absence of PF543 (SphK1 inhibitor-100nM) and JTE 013 (S1PR2 antagonist-10microM) and conditioned media (CM) was collected. Cultured 3T3-L1 adipocytes were stimulated with CM for 24h. In some experiments, the CM was treated with PF543 or JTE 013 for 24h or the adipocytes were pre-treated with JTE013 prior to stimulation. iNOS expression was determined using western blotting and NO production in the supernatant was measured using a Sievers 280 analyser.

Results: CM induced upregulation of iNOS and NO production in 3T3-L1 adipocytes. SphK1/S1PR2 axis inhibition in macrophages decreased resultant iNOS expression and NO production in 3T3-L1 co-cultured with activated CM ($p < 0.05$, $n = 3$). Blocking S1PR2 receptors in 3T3-L1 also decreased iNOS and NO production following co-culture with LPS-activated CM ($p < 0.05$, $n = 3$).

Conclusions: Macrophage-derived S1P activates S1PR2 on macrophages and adjacent adipocytes, contributing to iNOS regulation in adipose tissue. The SphK1/S1PR2 axis may be a promising target for adipose tissue inflammation.

Keywords: Macrophage, Inflammation, S1P, Adipose, Tissue, Nitric oxide.

FREE ORAL COMMUNICATIONS

[SESSION 3: ADVANCES IN CARDIOVASCULAR AND METABOLIC RESEARCH]

[OC-3.8]

ASSOCIATION OF miRNA-126 EXPRESSION WITH DIABETIC RETINOPATHY: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Background: Diabetic retinopathy (DR) is a major cause of vision loss in diabetes. miRNA-126, a regulator of vascular and endothelial function, has emerged as a potential biomarker for early detection and monitoring of DR progression.

Objective: To systematically review and analyse the association between miRNA-126 expression levels and diabetic retinopathy, assessing its presence, severity, and potential as a biomarker through meta-analysis.

Methods: A systematic search of PubMed, Scopus, ScienceDirect, and PMC was conducted following PRISMA guidelines. Observational studies comparing miRNA-126 expression in diabetic patients with and without DR were included. Studies were screened using Covidence, quality assessed via the Newcastle-Ottawa Scale, and meta-analysis performed to estimate pooled effects. Heterogeneity and variability were explored through I^2 statistics and sensitivity analyses.

Results: The meta-analysis showed a general reduction in miRNA-126 expression among DR patients. Significant heterogeneity ($I^2 = 73.6\%–91.6\%$) was attributed to differences in study design and populations. Subgroup analysis indicated a stronger reduction in T2DM. Sensitivity analysis revealed the influence of outlier studies. One analysis showed a significant association (MD = -0.33, 95% CI: -0.62 to -0.04, $p = 0.0238$).

Conclusions: miRNA-126 is a promising biomarker for diabetic retinopathy, showing consistently reduced expression in affected individuals. Due to study heterogeneity, further validation with standardised methods and larger, well-designed studies is essential. Combining miRNA-126 with other biomarkers may improve diagnostic accuracy.

Keywords: Diabetic Retinopathy, miRNA-126, Biomarkers, Endothelial Dysfunction, Angiogenesis, Retinal Vascular Permeability.

FREE ORAL COMMUNICATIONS

[SESSION 3: ADVANCES IN CARDIOVASCULAR AND METABOLIC RESEARCH]

[OC-3.9]

**CONTINUOUS GLUCOSE MONITORING IMPROVES DIABETES
MELLITUS AWARENESS AND PROMOTES A HEALTHY LIFESTYLE
AMONG NORMAL HEALTHY ADULTS**

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Introduction: Continuous glucose monitoring (CGM), offered via a patch attached to the body, is an emerging follow-up tool for diabetes management. It provides real-time glucose monitoring by bluetooth-enabled mobile apps, recording diet, drug, and physical activity information also. CGM has gained popularity among the health conscious. This pilot study assessed satisfaction and experience of healthy individuals using a CGM device.

Methods: This prospective study of one month duration determined the experience of CGM-use among healthy individuals. Twenty-seven healthy male participants gave written informed consent, were instructed on the application and use of the CGM sensor and mobile app. The device collected glucose levels every 30 seconds, with a bluetooth-enabled mobile app. Participants logged their diet and physical activity in the mobile app. Participants' satisfaction was assessed using a modified version of the CGM Satisfaction Scale (CGM-SAT) tool.

Results: The participants (mean age of 29.1 ± 10.3 years) found that the device did not affect their daily activities, which they were able to perform freely, and recordings were easily interpreted. CGM gave them insight into their diet, physical activity they performed and their blood sugar.

Conclusions: This study revealed that the participants reported satisfaction with its use and ease of interpretation. It provided insight into the impact of participants food choices and physical activity. This suggests that CGM has applications beyond monitoring treatment in diabetic patients and is a well-received tool that may assist in personalised wellness plans for health-conscious individuals.

Keywords: Continuous Glucose Monitoring, Diabetes Mellitus, Satisfaction, CGM Satisfaction Scale.

POSTER PRESENTATIONS

[P-01]

CARPAINЕ SUPPRESSES ANGIOTENSIN II-INDUCED HYPERTROPHY IN H9C2 CELLS BY MODULATING CALCIUM RESPONSE

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Background: Angiotensin II (Ang II) is a key contributor to the development of cardiovascular diseases, promoting oxidative stress, cardiac hypertrophy, and calcium dysregulation in cardiomyocytes. Carpaine, an alkaloid extracted from *Carica papaya*, is known for its diverse pharmacological properties; however, its cardioprotective potential against Ang II-induced injury has not been fully elucidated. This study aimed to investigate the protective effects of carpaine on Ang II-induced damage in H9c2 cardiomyoblasts.

Methods: H9c2 cells were cultured in Dulbecco's Modified Eagle Medium (DMEM) and pre-treated with carpaine prior to exposure to Ang II. Immunohistochemical staining was performed to assess reactive oxygen species (ROS) levels and hypertrophic responses. Western blot analysis was used to evaluate the expression of proteins associated with hypertrophic signaling, and fluorescence-based assays were employed to measure intracellular calcium (Ca^{2+}) mobilisation.

Results: Our findings reveal that carpaine significantly reduced Ang II-induced ROS production, suggesting antioxidant activity in cardiomyocytes. Carpaine also inhibited Ang II-induced cellular hypertrophy, demonstrated by a decrease in cell size and suppression of hypertrophic marker expression. Mechanistically, carpaine treatment led to a reduction in the phosphorylation of extracellular signal-regulated kinase 1/2 (ERK1/2), a central mediator in hypertrophic signaling. Additionally, carpaine markedly attenuated Ang II-induced intracellular Ca^{2+} mobilisation, indicating its role in preserving calcium homeostasis.

Conclusions: Carpaine effectively protects H9c2 cells from Ang II-induced oxidative and hypertrophic stress, and calcium imbalance. These results support further exploration of carpaine as a potential therapeutic agent for Ang II-related cardiovascular conditions.

Keywords: *Carica Papaya*; Carpaine; Cardiomyocytes; Hypertrophy; Angiotensin II.

POSTER PRESENTATIONS

[P-02]

**EFFECT OF SEX, AGE AND ETHNICITY ON BLOOD PRESSURE
IN SEREMBAN DIABETES (SeDia) COHORT**

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Background: The SeDia Cohort is a collaborative study between the Ministry of Health (MOH) and IMU University, to investigate various factors that affect Type 2 diabetes mellitus (T2DM) in Seremban, Negri Sembilan. The objective of this study is to determine the prevalence of hypertension in the SeDia cohort and to identify demographic factors that influence systolic blood pressure (SBP) among individuals with diabetes.

Methods: Anonymous data was obtained from SeDia cohort, following IMU-JC approval. The data included various variables such as age, sex, race, BP, and presence of diabetes complications. The data analysed using SPSS.

Results: Among 549 participants of the SeDia cohort (Dec 2024), 87.6% are T2DM and 10.47% are non-diabetic (family members); 67.2% of diabetes and 37.7% of family members have hypertension. The mean SBP was 146.78 ± 19.45 and 128.45 ± 19.46 mmHg, in T2DM and family members, respectively. In the SeDia cohort, diabetes and the effect of age are the primary contributors to SBP. Further, age is significantly associated with increased SBP in men ($p < 0.001$) and women ($p < 0.001$). Among different T2DM complications, 74.4% reported having high cholesterol, 39.5% feet numbness, and 39.2% diabetic neuropathy.

Conclusions: In the SeDia cohort, rate of hypertension is 67.2% among T2DM and age is significantly associated with increased BP in total population and within both sexes, while sex and ethnicity showed no significant association. Therefore, age-related changes in BP are crucial in the Malaysian T2DM population.

Keywords: Type 2 diabetes, blood pressure, SeDia cohort, sex, ethnicity.

POSTER PRESENTATIONS

[P-03]

**EVALUATION OF ETHNIC VARIABILITY IN EPICARDIAL ADIPOSE
TISSUE THICKNESS AMONG TYPE 2 DIABETES MELLITUS
PATIENTS WITH CORONARY ARTERY DISEASE**

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Background: Epicardial Adipose Tissue (EAT) secretes pro-inflammatory adipokines and free fatty acids, exacerbating atherosclerosis and increasing Coronary Artery Disease (CAD) risk. Ethnic disparities in EAT distribution may also influence CAD risk, particularly in diabetic populations. SGLT2 inhibitors show promising cardiovascular benefits, making them a valuable therapeutic option for managing CAD in diabetic patients. This study evaluates SGLT2 inhibitor therapy's effect on EAT thickness and ethnic variations in EAT distribution among diabetic patients with CAD.

Methods: A single-centre quasi-experimental cohort study on diabetic patients with CAD divided into SGLT2 inhibitor users (n=151) and non-users (n=151). Ethnic variations in EAT thickness were analysed using echocardiogram at baseline and after six months.

Results: The study included 302 participants, predominantly male (70.2%) with mean age of 62. Ethnicity distribution was balanced, with 35% Malay, 32% Chinese, and 32% Indian participants. There were no statistically significant differences at baseline between study groups, although EAT thickness was highest among Chinese participants. After six months, EAT thickness significantly decreased in the SGLT2 inhibitor group compared to the non-SGLT2 inhibitor group within all ethnic groups ($p < 0.001$). However, the treatment response was not statistically significant across ethnicities ($p = 0.852$).

Conclusions: Our findings indicate SGLT2 inhibitors effectively reduced EAT thickness regardless of ethnicity. Although the treatment response did not significantly vary across ethnic groups, these findings support SGLT2 inhibitors' potential to reduce cardiovascular risk factors associated with EAT. Further research is needed to validate these results in larger, diverse populations.

Keywords: SGLT-2 inhibitor, Coronary Artery Disease, Epicardial Adipose Tissue, Diabetes, Ethnic.

POSTER PRESENTATIONS

[P-04]

EMPAGLIFLOZIN MITIGATES VASCULAR DYSFUNCTION IN A POST-MENOPAUSAL RAT MODEL

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Background: Cardiovascular disease risk increases significantly in post-menopausal women. This is partly due to oestrogen deficiency, which impairs endothelium-dependent vasorelaxation (EDV) by reducing endothelial nitric oxide synthase (eNOS) expression and nitric oxide (NO) bioavailability. This study aimed to evaluate the effects of empagliflozin, a sodium-glucose cotransporter 2 inhibitor with pleiotropic cardiovascular benefits, on vascular dysfunction in an ovariectomy (OVX)-induced postmenopausal rat model.

Methods: Forty female Sprague Dawley rats were randomly divided into five groups (n = 8): Sham, OVX, OVX + vehicle, OVX + empagliflozin (10 mg/kg/day, orally), and OVX + 17 β -oestradiol (20 μ g/kg/day, subcutaneously) for 28 days. On day 29, thoracic aorta was dissected for vascular reactivity studies using wire myography. The gene expression of eNOS in the aorta was evaluated by quantitative real-time reverse transcription polymerase chain reaction, while the aortic NO levels were measured via Griess assay.

Results: OVX rats showed increased contractile response to phenylephrine (P < 0.001), impaired EDV to acetylcholine (P < 0.001), along with reduced eNOS mRNA expression (P < 0.05), and NO levels (P < 0.001) compared to Sham group. Treatment with empagliflozin significantly improved EDV (P < 0.001), upregulated eNOS gene expression (P < 0.0001), and restored NO levels (P < 0.001) compared to untreated OVX rats. These effects were comparable to 17 β -oestradiol treatment.

Conclusions: Empagliflozin attenuates vascular dysfunction in OVX rats by improving EDV, upregulating eNOS gene expression, and enhancing NO bioavailability, indicating its therapeutic potential for vascular protection in post-menopausal women.

Keywords: Endothelial Nitric Oxide Synthase; Empagliflozin; Menopause; Nitric Oxide; Vascular Dysfunction.

POSTER PRESENTATIONS

[P-05]

IS BODY ADIPOSITY INDEPENDENTLY ASSOCIATED WITH BONE HEALTH? INSIGHTS FROM A MALAYSIAN COHORT

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Background: Body size, typically reflected by body mass index (BMI), is widely recognised to be positively associated with bone health. However, adiposity has been hypothesised to influence bone health independently of BMI. This study aimed to explore this hypothesis by examining the associations between various adiposity indices and bone health among Malaysians.

Methods: In October to November 2024, 320 participants were conveniently recruited during a bone health screening programme at a university hospital in Kuala Lumpur, Malaysia. Participants completed a questionnaire covering demographic characteristics and risk factors for osteoporosis. Height was measured using a stadiometer, body composition was measured using a bioelectrical impedance device, and hip and waist circumferences were measured using a flexible measuring tape. Bone health, indicated by osteoporosis index, was assessed using calcaneal quantitative ultrasound. Adiposity indices were calculated as per the convention. Associations between adiposity indices and bone health were analysed using multiple linear regression, adjusting for relevant confounders.

Results: After adjustment, BMI, body fat percentage, waist circumference, waist-to-height ratio, body roundness index, and visceral fat area were significantly and positively associated with the osteoporosis index ($p < 0.05$). However, these associations became non-significant after further adjustment for BMI ($p > 0.05$).

Conclusions: The positive associations observed between adiposity indices and bone health appear to be mediated by BMI or overall body size. Longitudinal studies are warranted to clarify the causal direction of this relationship.

Keywords: Adipose, Metabolic Health, Obesity, Osteoporosis, Skeleton.

POSTER PRESENTATIONS

[P-06]

**PROTECTIVE ROLE OF ETLINGERA ELATIOR FLOWER EXTRACT
AGAINST OXIDATIVE STRESS-INDUCED NEPHROPATHY
IN TYPE 2 DIABETIC RAT MODEL**

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Background: Diabetes mellitus (DM) is a major global health concern, often leading to complications such as renal disease. Medicinal plants are gaining interest due to their therapeutic benefits, particularly for their antioxidant properties. *Etlingera elatior* flower (*bunga kantan*) is traditionally used for its medicinal properties and shows antioxidant potential; however, scientific evidence in DM remains limited. This study aimed to evaluate the anti-diabetic, antioxidant, and renoprotective effects of *E. elatior* flower aqueous extract (EEAE) in a type-2 DM rat (T2DR) model.

Methods: The T2DR model was developed using a high-fat diet and streptozotocin (STZ) administration. Thirty-five male Sprague-Dawley (SD) rats were equally divided into five groups (n=7); normal, obese, untreated-DM, metformin-treated, and EEAE-treated at 1000 mg/kg. Treatments were administered orally for six weeks. In vitro antioxidant assays (DPPH and FRAP) and enzyme inhibition assays (α -amylase and α -glucosidase) were conducted. Fasting blood glucose (FBG) and blood pressure were recorded weekly. At the end of the study, biochemical parameters, antioxidant biomarkers, and kidney histopathology were assessed.

Results: EEAE demonstrated significant in vitro antioxidant activity and inhibited α -amylase and α -glucosidase. EEAE also significantly reduced levels of FBG and blood pressure and improved renal function compared to the untreated-DM group. Furthermore, EEAE significantly increased antioxidant biomarkers, including superoxide dismutase (SOD), catalase (CAT), and glutathione (GSH), and improved renal histopathology.

Conclusions: EEAE possesses antioxidant and anti-diabetic activities that contribute to its renoprotective effects in T2DR. These findings support its potential as a complementary therapy for diabetes-related renal complications.

Keywords: *Etlingera Elatior*, Type 2 Diabetes, Antioxidant Activity, Renoprotective Effects, Oxidative Stress.

POSTER PRESENTATIONS

[P-07]

THE ROLE OF APELIN IN THE PATHOPHYSIOLOGY OF POLYCYSTIC OVARY SYNDROME: A SCOPING REVIEW

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Background: Polycystic Ovary Syndrome (PCOS) is a complex endocrine and metabolic disorder affecting women of reproductive age. Adipokines like apelin have been associated with the pathogenesis of PCOS. However, apelin's specific role in PCOS remains incompletely understood. This scoping review aims to map the current evidence on the role of apelin in the pathophysiology of PCOS.

Methods: Following the PRISMA-ScR guidelines, a comprehensive literature search was conducted in PubMed, Web of Science, Scopus, and China National Knowledge Infrastructure (CNKI) to identify relevant studies published up to May 2025. Eligible studies included animal experiments, cellular models, and clinical research that examined apelin expression, signalling pathways, or physiological effects in the context of PCOS. Data were charted and thematically categorised according to study type, biological pathway involvement, and clinical findings.

Results: A total of 16 studies were included in this review, encompassing in vitro, in vivo, and clinical research. Most studies reported elevated apelin levels in PCOS models or patients, which correlated with insulin resistance, obesity, and reproductive dysfunction. Mechanistic findings suggest that apelin modulates insulin signalling, inflammatory pathways, steroidogenesis, and granulosa cell function through the APJ receptor and downstream effectors such as AMPK and PI3K/Akt. However, inconsistencies exist regarding apelin's exact role and whether its effects are compensatory or pathogenic.

Conclusions: Apelin is an important metabolic and reproductive regulator in the pathophysiology of PCOS. While current evidence highlights its potential as a biomarker or therapeutic target, further standardised and longitudinal studies are needed to clarify its clinical applicability and mechanistic role.

Keywords: Apelin, Polycystic Ovary Syndrome, Adipokines, Insulin Resistance, Granulosa Cells, Pathophysiology, Scoping Review.

POSTER PRESENTATIONS

[P-08]

ASTAXANTHIN NANOEMULSION ALLEVIATES MOTOR FUNCTION AND NEURONAL MARKER FOLLOWING ISCHEMIC STROKE IN RATS

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Background: Ischaemic stroke cases have been increasing lately worldwide. More natural products with neuroprotective potential have been discovered to reduce the ischaemic insults. Astaxanthin is a carotenoid that has potent anti-inflammatory effects but poor bioavailability. Hence, the advancements of nanoemulsion formulation might enhance its absorption, enabling it to penetrate the blood-brain barrier and potentially protect the brain against ischaemic stroke insult.

Methods: Eighteen male Sprague Dawley rats were randomly divided into three groups: stroke with astaxanthin extract (SE), stroke with astaxanthin macromolecules (SM), and stroke with astaxanthin nanoemulsion (SN). All rats received oral supplementation of their respective astaxanthin solutions at 1280 mg/kg body weight for seven days prior to and three hours after inducing permanent middle cerebral artery occlusion (pMCAO). After 24 hours, the rats were tested for neurobehavioral and motor function using the modified neurological severity (mNSS) score test, grid walking test, and rotarod test. Brain samples were then analysed for the neurological marker neuron-specific enolase (NSE) using ELISA.

Results: SN has the lowest neurological insult after pMCAO compared to SE and SM with improved motor function in mNSS test, grid walking test, and rotarod test. Furthermore, SN also showed the lowest level of NSE compared to SE and SM groups.

Conclusions: This study proved that supplementation of astaxanthin nanoemulsion might serve better neuroprotective effect compared to macroemulsion and extract, evidenced by the improvement in the neurobehavior tests and neuronal injury marker.

Keywords: Ischaemic Stroke, Nanoemulsion, Astaxanthin, Neuroprotective, Neurobehaviour, Neuronal injury.

POSTER PRESENTATIONS

[P-09]

UNDERSTANDING THE LINK BETWEEN CHOLESTEROL AND OCULAR DISORDERS FOR EFFECTIVE THERAPY

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Cholesterol is a lipoprotein molecule that plays its part in various physiological processes of the human body. However, excessively high cholesterol levels can lead to various kinds of health disorders, including cardiovascular diseases. Over the past few years, it has been discovered that there is also a link between cholesterol and eye disorders. This review investigates the relationship between ocular conditions and cholesterol in detail, outlining the potential mechanisms and manifestations of such conditions.

Different studies have established the fact that elevated cholesterol levels are capable of leading to the development of many ocular disorders, such as age-related macular degeneration (AMD), cataracts, and glaucoma. These conditions are characterised by degenerative changes in the retina, lens, or optic nerve which lead to defective vision or total blindness. Deposition of cholesterol in the ocular blood vessels can impair blood supply and thus, lead to retinal vascular diseases which adversely affect the delivery of nutrients and the elimination of waste products.

Further, high cholesterol levels result in yellowish plaques called drusen that are associated with the development of AMD. Further, cholesterol imbalances cause chronic inflammation and oxidative stress in ocular tissues. Inflammation is an important mediator involved in ocular diseases and results in blood-retinal barrier disruption and activation of a number of inflammatory mediators. Oxidative stress, on the other hand, leads to the production of reactive oxygen species that cause damage to ocular cells and tissues. These have been implicated in the etiology and pathogenesis of ocular conditions of AMD, glaucoma and cataracts.

Keywords: Cholesterol, Age-Related Macular Degeneration, Cataract, Glaucoma, Retinal Vascular Diseases.

POSTER PRESENTATIONS

[P-10]

**THE IMPACT OF METABOLIC SYNDROME ON DENGUE SEVERITY:
A REVIEW OF PATHOPHYSIOLOGICAL MECHANISMS
AND CLINICAL IMPLICATIONS**

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Dengue fever remains a pressing public health challenge, endemic in over 90 countries and placing nearly four billion individuals at risk globally. In Malaysia, 32,356 dengue cases and 46 deaths were recorded as of July 2025. Concurrently, metabolic syndrome (MetS) – a cluster of metabolic disturbances including abdominal obesity, hypertension, insulin resistance, and dyslipidemia – affects 36% of Malaysian adults, as reported in the National Health and Morbidity Survey 2024.

This review aims to examine current evidence linking MetS with increased severity of dengue infection, with a focus on the roles of systemic inflammation, endothelial dysfunction, and oxidative stress in disease progression. Recent literature suggests that patients with MetS are more likely to experience severe dengue outcomes, such as dengue hemorrhagic fever (DHF) and dengue shock syndrome (DSS).

Chronic low-grade inflammation and pre-existing immune dysregulation in MetS may exacerbate the cytokine storm induced by dengue infection. This inflammatory response contributes to endothelial injury and heightened vascular permeability – hallmarks of severe dengue. Additionally, oxidative stress associated with MetS impairs immune function and further damages the vascular endothelium, compounding disease severity. The coexistence of MetS significantly worsens clinical outcomes in dengue-infected individuals.

Early identification and management of metabolic abnormalities could reduce the risk of severe complications. Public health strategies and clinical interventions targeting inflammation, oxidative stress, and metabolic dysfunction are urgently needed, especially in dengue-endemic regions with rising rates of MetS.

Keywords: Dengue, Metabolic Syndrome, Inflammation, Endothelial Dysfunction, Oxidative Stress.

POSTER PRESENTATIONS

[P-11]

INVESTIGATING THE IMPACT OF PRENATAL EXPOSURE TO MITRAGYNE (KRATOM) ON THE DEVELOPMENT OF NEONATAL OPIOID WITHDRAWAL SYNDROME

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Background: Kratom is considered a safer alternative for pregnant women experiencing opioid-related issues. Nevertheless, prenatal kratom consumption in humans has shown that infants require pharmacological intervention due to withdrawal symptoms. Mitragynine, the principal alkaloid, has been detected in the umbilical cords of mothers who have ingested kratom, suggesting its ability to traverse the placenta and enter fetal circulation, potentially leading to neurodevelopmental disorders. Hence, this study aims to examine whether prenatal exposure to mitragynine induces neonatal opioid withdrawal syndrome (NOWS) in the rat's offspring.

Methods: The pregnant rats were orally administered with vehicle, morphine, low dose, or high dose of mitragynine from gestational day 11 until 18. Neonatal opioid withdrawal syndrome (NOWS) was assessed on postnatal day 1. The severity of NOWS was assessed by systematically observing and scoring the withdrawal-related behaviours, including mouth opening, stretching, face washing, jerking, tremors, and hyperactivity.

Results: The findings indicate that offspring exposed prenatally to a high dose of mitragynine exhibit neonatal opioid withdrawal syndrome (NOWS), as evidenced by significant alterations in certain withdrawal behaviours. The data further suggest that female offspring are more susceptible to the effects of prenatal mitragynine exposure compared to male offspring. Consistent with expectations, offspring prenatally exposed to morphine also demonstrated pronounced withdrawal behaviours.

Conclusions: Overall, prenatal exposure to a high dose of mitragynine (kratom) could lead to NOWS in neonates. Furthermore, the use of kratom during pregnancy might pose risks to the child, potentially resulting in neurodevelopmental issues and cognitive challenges that could continue into later life stages.

Keywords: Kratom, Mitragynine, Neonatal Opioid Withdrawal Syndrome.

POSTER PRESENTATIONS

[P-12]

THE EFFECTS OF TRIGONA HONEY ON SPATIAL WORKING MEMORY IN PRENATAL BPA-EXPOSED SPRAGUE DAWLEY RATS

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Bisphenol A (BPA) is an industrial chemical used in the production of polycarbonate plastics and epoxy resins. Prenatal BPA exposure disrupted the learning process by downregulating NMDA receptor subunit and synaptic plasticity proteins. Trigona honey, produced by stingless bees, has neuroprotective properties and enhances spatial memory.

This study investigated the effects of Trigona honey on spatial working memory in prenatal BPA-exposed offspring at age 21 days. Five mg/mL BPA was administered to pregnant rats from day 2 of pregnancy until day 21 to create a prenatal BPA exposure environment. Then the pregnant rats were allowed to deliver their offspring spontaneously. Eight male offspring were allowed to grow until day 21 and were randomly assigned into two groups (n = 4 each): Control and Trigona honey.

For the control group, each rat was administered 0.5 mL of RO water daily for seven days. For the trigona honey group, each rat was supplemented with a 1000mg/kg dose of trigona honey using oral gavage for seven days. At day 7, the T-maze test was conducted for each group for three days. Each rat performed four runs per day. The chosen arm for each run was recorded. The trigona honey-treated group ($75.0 \pm 9.2\%$) significantly exhibited a higher percentage of alternation than the control group ($58.5 \pm 9.8\%$) ($t(6) = -2.45$, $p < 0.050$). Trigona honey improves the spatial working memory function in BPA-exposed rats; further studies are warranted to gain a better understanding.

Keywords: Bisphenol A, Trigona Honey, Spatial Working Memory, T-maze.

POSTER PRESENTATIONS

[P-13]

**WI-FI EXPOSURE ALTERS MALE REPRODUCTIVE PARAMETERS:
EVIDENCE FROM A RODENT MODEL**

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With the rapid expansion of wireless technology, exposure to radiofrequency electromagnetic fields (RF-EMFs) has become nearly ubiquitous. As male gonads are particularly sensitive to environmental stressors, concerns have been raised about the potential adverse effects of RF-EMFs on male fertility. Therefore, this study investigates the impact of chronic Wi-Fi exposure on male reproductive parameters in rats, using 18 adult male Sprague Dawley rats divided into three groups: Control, 4-hour, and 24-hour exposure.

The exposure was conducted using a TP-LINK AC750 Wireless Dual Band Router Archer C20 placed at 20cm distance from the animal cages with only cage wall and its metal coverings as barriers. Exposure was conducted for 60 consecutive days as to represent one complete spermatogenesis cycle. No abnormalities were recorded on the gross morphology and weight of the testes. However, testicular histology revealed the presence of vacuolation within the seminiferous tubule and increased interstitial edema. Testicular testosterone level has significantly decreased in 24-hour exposed group compared to the Control group.

Furthermore, there was significant increase of testicular malondialdehyde level in 4-hour group. These detrimental changes have further contributed to the gradual and significant decrease in sperm concentration in both exposed groups. The results highlight the potential reproductive hazards associated with regular use of wireless devices and their possible role in male infertility.

Keywords: Testosterone, Malondialdehyde, Sperm Concentration, Wi-Fi 2.45GHz.

POSTER PRESENTATIONS

[P-14]

IMPACT OF PROLONGED EXPOSURE TO HIGH ENERGY VISIBLE LIGHT FROM DIGITAL DEVICES ON MEDICAL STUDENTS

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Background: Prolonged exposure to high-energy visible (HEV) light from digital devices may negatively affect the well-being, academic performances, and health of medical students. This study aimed to explore the extent of HEV light exposure and its potential consequences.

Methods: A cross-sectional study was conducted among 265 randomly selected medical students (Years 1–5) from Universiti Kebangsaan Malaysia. Participants anonymously completed an online questionnaire comprising demographic data, the Pittsburgh Sleep Quality Index (PSQI), the Warwick-Edinburgh Mental Well-being Scale (WEMWBS), and items on digital device usage (frequency, duration, timing). Sleep quality was classified as good (PSQI ≤ 5) or poor (PSQI > 5). Well-being was measured using WEMWBS on a Likert scale. Pearson correlation analysis was used to assess relationships between sleep quality, well-being, and device usage.

Results: The study examined the effects of prolonged HEV light exposure on sleep quality, well-being, and CGPA. Pearson correlation showed weak but significant negative correlations with sleep quality ($r = -0.207$, $p < 0.001$) and well-being ($r = -0.272$, $p < 0.001$), indicating poorer outcomes with increased exposure. No significant correlation was found with CGPA ($r = -0.016$, $p = 0.797$).

Conclusions: Blue light exposure may influence academic performance, well-being, and sleep quality. Further research with different samples is recommended for more comprehensive findings.

Keywords: Sleep Quality, Academic Performance, Well Being.

POSTER PRESENTATIONS

[P-15]

**EFFECTS OF TUALANG HONEY ON INTESTINAL FUNCTION
AND MICROBIAL COMPOSITION FOLLOWING REPEATED
PARAQUAT EXPOSURE IN RATS**

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Background: Chronic paraquat exposure has been linked to neurodegenerative diseases, with recent studies suggesting that the gut may play an early role in their pathogenesis. This study aimed to evaluate the effects of repeated paraquat exposure on colonic contractile function and gut microbiota composition, and the potential protective effects of Tualang honey supplementation.

Methods: Sixteen male adult Sprague-Dawley rats were randomly divided into four groups (n=4 each): control (N), Tualang honey (H), paraquat (PQ), and paraquat with Tualang honey (T). Groups H and T received oral TH (1.0 g/kg bw/day) throughout the experimental period. Following a two-week honey pre-treatment period, groups PQ and T were fed with a paraquat-containing diet (60 mg/kg food pellet) for 12 weeks, while Groups N and H received a standard diet. Lastly, faecal samples were collected for microbiota profiling using 16S rRNA sequencing. Proximal colons were also collected to assess colonic contractility using organ bath analysis.

Results: Paraquat exposure resulted in relatively lower maximal contraction and higher contraction frequency compared to the control group. Treatment with Tualang honey improves colonic maximal contraction and reduces contraction frequency when compared to PQ group. Paraquat exposure also showed reduced relative abundances of Campylobacterota, Verrucomicrobiota, Desulfobacterota, and Fibrobacterota, but an increase in Proteobacteria compared to Group N. Tualang honey supplementation was associated with higher relative abundances of Campylobacterota and Fibrobacterota, and lower Proteobacteria.

Conclusions: These preliminary findings suggested that Tualang honey may confer protective effects against paraquat-induced reductions in colonic contractility function and microbial dysbiosis.

Keywords: Paraquat, Tualang Honey, Gut Dysbiosis, Colon Contractility, 16S rRNA Sequencing.

POSTER PRESENTATIONS

[P-16]

PROPHYLACTIC POTENTIAL OF KELULUT HONEY AGAINST SARS-CoV-2 VIA ACE-2 BINDING INHIBITION

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Background: Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has caused a global health crisis. Despite vaccination efforts, the virus continues to evolve, decreasing the effectiveness of current vaccines. Angiotensin-converting enzyme 2 (ACE-2) is a crucial protein that facilitates SARS-CoV-2 entry into host cells. Our prior *in silico* docking and molecular dynamics simulation studies indicated that a compound called 4,4'-Stilbenedicarboximidine (SDC) binds strongly to ACE-2, surpassing MLN 4760, a known ACE-2 inhibitor. This study aims to assess the potential of local *kelulut* honey (KH) and SDC in inhibiting the spike protein-ACE-2 interaction.

Methods: An ELISA assay using the SARS-CoV-2 S1 RBD: ACE-2 Inhibitory Screening Assay Kit (BPS Bioscience, US) was performed following the manufacturer's instructions. KH and SDC were tested at multiple concentrations in triplicate, with MLN 4760 serving as the reference.

Results: KH, MLN 4760, and SDC significantly inhibited spike-ACE-2 binding, with an E_{max} of 82.66 ± 0.24 %, 59.81 ± 8.00 %, and 19.74 ± 0.40 %, respectively. The IC₅₀ values of KH, MLN 4760, and SDC were 0.17 % (v/v), 55.44 μM, and 19.20 μM, respectively. At 100 μM, SDC was found to inhibit spike-ACE-2 binding by 19.74 %, which was significantly lower (P<0.05) than MLN 4760 at 59.81%.

Conclusions: KH inhibits the interaction between the spike protein and ACE-2, supporting its prophylactic use against SARS-CoV-2. The higher inhibitory efficacy of KH in its absolute form compared to SDC is presumably due to the interaction of multiple bioactive constituents in KH, rather than the effect of SDC alone.

Keywords: Angiotensin Converting Enzyme 2, ACE-2, COVID-19, *Kelulut* Honey, SARS-CoV-2.

POSTER PRESENTATIONS

[P-17]

**PRELIMINARY STUDY ON THE MODULATORY EFFECT OF
N-ACETYLCYSTEINE ON RECALL ANTIGEN-INDUCED
IMMUNE RESPONSES**

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Background: Rapid reactivation of memory T and B cells is crucial for an effective recall response. However, it can produce excessive reactive oxygen species (ROS), impairing cell function and homeostasis. N-acetylcysteine (NAC) is an antioxidant that scavenges ROS and increases intracellular glutathione. This preliminary study aimed to assess whether NAC supplementation could modulate the recall response induced by tetanus toxoid (TT) in human peripheral blood mononuclear cells (PBMCs).

Methods: PBMCs isolated from a healthy, TT-vaccinated individual were cultured and divided into three treatment groups: TT alone (0.5 Lf/mL), NAC alone (1 mM), or in combination. Recall responses were measured at 24, 48, and 72 hours. B cell, T cell, and monocyte subsets were analysed using flow cytometry.

Results: NAC supplementation resulted in a significant increase in helper (CD4+) T cells, reaching 41.9% by 72 hours, while classical monocytes remained high at about 78.6 to 86.4% and intermediate monocytes were suppressed at 1.9%. B cells exhibited a moderate increase (27.2% at 72 hours), and cytotoxic (CD8+) T cells declined significantly from 64.2% to 37.2%.

Conclusions: NAC effectively reduces activation-induced oxidative stress in TT-stimulated human PBMCs, sustaining CD4+ T cell expansion and maintaining balanced monocyte subset distribution, with potential implications for B cell and CD8+ T cell responses. These findings highlight the importance of redox homeostasis in recall immune responses and support further investigation into antioxidant modulation of immune memory.

Keywords: Tetanus Toxoid, N-acetylcysteine, Peripheral Blood Mononuclear Cells, Recall Immune Responses, Oxidative Stress, Redox Homeostasis.

POSTER PRESENTATIONS

[P-18]

**RESEARCH PROGRESS IN THE ROLE OF PYROPTOSIS
IN THE DEVELOPMENT OF PERIODONTITIS**

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Periodontitis is a chronic infectious inflammatory disease characterised by progressive destruction of periodontal support tissue and is the primary cause of tooth loss in adults. Plaque microorganisms are the initiating factors of periodontitis, often acting in conjunction with various local or systemic factors to promote the occurrence of periodontitis.

During the progression of periodontitis, microorganisms and their metabolites cause tissue damage, stimulating the host's inflammatory response to resist microbial invasion. The interaction between plaque microorganisms and host immune inflammatory response is an important factor determining the degree of periodontal tissue damage, and overactivated inflammatory response can exacerbate periodontal tissue damage.

Therefore, it is particularly important to explore the inflammatory regulatory mechanisms during the occurrence and development of periodontitis. Pyroptosis is an emerging field in the study of inflammatory mechanisms. In recent years, research has found that various diseases such as osteomyelitis and periapical periodontitis are closely related to pyroptosis. Periodontitis, which is also an inflammatory bone destruction disease, may also be related to cell death. This article reviews the research progress on the involvement of pyroptosis in the occurrence and development of periodontitis.

Keywords: Pyroptosis, Periodontitis, Inflammatory Disease, Mechanisms.

POSTER PRESENTATIONS

[P-19]

**BREASTFEEDING AND THE BODY CLOCK:
A SYSTEMATIC REVIEW OF MATERNAL SLEEP AND FATIGUE**

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Background: The postpartum period involves significant physiological and behavioural changes, particularly among breastfeeding mothers who frequently experience sleep disruptions and altered circadian rhythms. These adjustments may contribute to increased fatigue and negatively impact maternal well-being.

Objective: This systematic review aims to synthesise current evidence on the associations between breastfeeding, sleep quality, and fatigue, with a specific focus on circadian rhythm disruption in postpartum women.

Methods: A comprehensive literature search was conducted across PubMed, Scopus, and Web of Science databases for studies published between 2015 and 2025. Search terms included “breastfeeding OR lactation,” “sleep OR sleep quality,” and “fatigue”. Studies were included if they addressed sleep quality and fatigue among breastfeeding women.

Results: A total of eleven studies met the inclusion criteria. The evidence consistently indicates that breastfeeding mothers, especially those involved in nocturnal feedings, experience significant sleep disruptions and elevated fatigue. Poor sleep quality was associated with higher levels of fatigue compared to those who reported good sleep quality. However, mothers who exclusively breastfed showed longer total sleep time and better sleep quality compared to mothers who used formula at night. Overall, both self-reported and measured sleep disturbances were strongly related to increased fatigue in mothers.

Conclusions: Breastfeeding mothers experience altered circadian rhythms, poor sleep quality and elevated fatigue during the postpartum period. A strong relationship was observed between poor sleep quality and increased levels of fatigue. These findings highlight the importance of implementing targeted postpartum approaches to improve sleep quality and promote the well-being of mothers.

Keywords: Breastfeeding, Circadian Rhythm, Maternal Sleep, Postpartum Fatigue, Chronobiology.

POSTER PRESENTATIONS

[P-20]

VISUAL LEARNING AS A TOOL FOR TEACHING RENAL TUBULAR FUNCTION: INSIGHTS FROM SECOND-YEAR MEDICAL STUDENTS

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Background: Visual learning is an effective strategy for enhancing comprehension of complex physiological concepts in medical education. This study evaluates the impact of diagram-based instruction on medical students' understanding of renal tubular function.

Methods: Second-year medical students participated in a structured, diagram-based learning session on renal tubular physiology. The intervention included a detailed visual representation of tubular function, followed by a formative quiz to assess conceptual understanding. A summative end-of-block test was used to evaluate overall comprehension. Students also completed a post-intervention survey to provide feedback on the visual learning approach.

Results: Seventy-three students from three academic batches took part in the intervention, with 64% (47/73) completing both the renal quiz and the feedback questionnaire. The average quiz score was 7.6 out of 10. In the end-of-block test, 72% of students correctly answered the question on renal tubular function. Survey responses indicated a positive perception of the visual approach: 79% of students reported that diagram-based learning made the topic more enjoyable, and 78% felt it improved their understanding of tubular physiology.

Conclusions: Diagram-based visual learning effectively supports student engagement and understanding of renal tubular physiology. Positive student feedback and strong assessment performance highlight the potential of visual aids as a valuable tool in medical education.

Keywords: Visual Learning, Renal Tubular Physiology.

POSTER PRESENTATIONS

[P-21]

BEYOND THE CHALKBOARD: TAPPING INTO THE POTENTIAL OF TECH-ENHANCED PHARMACOLOGY EDUCATION

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This pilot project describes the implementation of a multi-platform, multimedia teaching strategy aimed at enhancing student engagement and understanding during a pharmacology lecture on antitubercular drugs. Conducted with Year 2 medical students, the session incorporated a structured three-phase approach using interactive digital tools and animations. Pre-lecture engagement included a Quizizz quiz to assess baseline knowledge. A 60-minute lecture followed, featuring animated visuals to explain complex mechanisms of action, along with open-ended questioning to encourage participation.

After the session, students accessed a Padlet board containing lecture materials, additional notes, a Wordwall quiz for reinforcement, and an open column for questions or reflections. A short post-lecture survey using Mentimeter was conducted to gather student perceptions on the effectiveness of this approach.

This method facilitated both synchronous and asynchronous learning and demonstrated the feasibility of incorporating low-cost, accessible technologies to support more interactive and student-centered pharmacology education. While initial feedback and engagement were positive, a more formal evaluation is currently underway and will provide more conclusive evidence of its effectiveness for broader application.

Keywords: Pharmacology, Education, Teaching, Innovation, Mentimeter, Padlet, Quizizz, Wordwall, Animation, Interactive Learning.

POSTER PRESENTATIONS

[P-22]

THE POTENTIAL OF TOCOTRIENOL-RICH FRACTION SUPPLEMENTATION TO REDUCE THE IMPACT OF HIGH-FAT DIET-INDUCED OBESITY IN FEMALE MICE

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Obesity negatively affects the female reproductive system and reduces fertility. Tocotrienol-rich fraction (TRF) has potential anti-obesity effects, however, whether TRF supplementation could improve reproductive outcomes impaired by obesity is still unclear. Therefore, this study aimed to determine the effect of TRF supplementation on oocyte quality, as well as reproductive hormones in high-fat diet (HFD)-induced obese mice.

Thirty-two, six-week-old female mice were divided into four groups: Group 1: Normal control (given standard mouse chow diet); Group 2: HFD-induced obesity; Group 3: HFD-induced obesity (given palm olein as vehicle); Group 4: HFD-induced obesity + TRF supplementation. Mice were fed either standard mouse chow diet (10g/100 g fat) or HFD (45 g/ 100 g fat) for 12 weeks. Mice were given TRF for eight weeks via oral gavage daily (0.1 ml) at a dose of 150 mg/kg BW, four weeks after the induction of obesity. At the end of the treatment period, mice from all groups were superovulated and then euthanised and oocyte quality was assessed.

The reproductive hormones, namely FSH, LH, estradiol and progesterone were determined using an ELISA kit. The percentage of normal oocytes was lower ($p < 0.001$) in HFD-induced obese mice (Gp 2) compared to the control group (Gp 1), whereas TRF supplementation restored the percentage of normal oocytes in HFD-induced obese mice (Gp 4) towards control (Gp 1). Additionally, all reproductive hormone levels were reduced in HFD-induced obese mice ($p < 0.05$) compared to the control group; TRF supplementation restored these hormonal levels toward normal. These results conclude that TRF supplementation was able to reduce the negative impact of obesity on the reproductive outcomes.

Keywords: Tocotrienol-Rich Fraction; Obesity; Oocyte; Reproductive Hormones.

POSTER PRESENTATIONS

[P-23]

EFFECTS OF TUALANG HONEY SUPPLEMENTATION ON RATS' ILEAL SURFACE AREA FOLLOWING THREE-MONTH REPEATED EXPOSURES TO PARAQUAT

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Background: Morphometric analysis of small intestines, such as the ileum, provides information on the functional capacity of the gastrointestinal tract. Changes in the ileal surface area may indicate intestinal responses to various stimuli, including chronic exposure to environmental toxicants. This study aimed to evaluate the effect of repeated exposure to paraquat on ileal morphology and the possible protective effect of Tualang honey supplementation.

Methods: Sixteen male Sprague-Dawley rats were randomly divided into four groups of four rats each: normal (N), Tualang honey (H), paraquat (PQ), and paraquat with Tualang honey supplementation (T). Groups H and T were supplemented with Tualang honey (1.0 g/kg bw/day) throughout the study period. After two weeks of Tualang honey pre-treatment, groups PQ and T were fed with a diet containing paraquat (60 mg/kg food pellet) for 12 weeks, while groups N and H received a normal diet. At the end of the exposure period, the ileum was collected, post-fixed, and processed for haematoxylin and eosin (H&E) staining to evaluate the villus morphology.

Results: Repeated paraquat exposures caused a reduction in the villus height, width, and surface area when compared to the normal group. Daily supplementation with Tualang honey was shown to improve these changes when compared to the PQ group. However, the changes were statistically not significant.

Conclusions: Preliminary findings suggest that Tualang honey may exert a protective effect against paraquat-induced morphological changes in the ileum. Further investigation with a larger sample size is needed to confirm these findings.

Keywords: Tualang Honey, paraquat, Ileum, Villus Morphology.

POSTER PRESENTATIONS

[P-25]

NEUROPROTECTIVE EFFECTS OF FARNESOL ON MOTOR AND EMOTIONAL IMPAIRMENTS IN A 3-NITROPROPIONIC ACID-INDUCED HUNTINGTON'S DISEASE RAT MODEL

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Introduction: Huntington's disease (HD) is a neurodegenerative disorder marked by motor and psychiatric disturbances. This study aimed to investigate the therapeutic potential of farnesol in alleviating motor and emotional impairments in rat model of HD due to their antioxidant and anti-inflammatory properties.

Methods: Sixteen Sprague-Dawley rats were divided into four (n=4 per group): (i) healthy control, (ii) HD, (iii) HD+farnesol, and (iv) farnesol-only. HD was induced via intraperitoneal injection of 3-nitropropionic acid (10mg/kg) on day 0, followed by farnesol (100mg/kg) oral administration for 14 days. Motor performance (cylinder and four-limb hanging test) and emotional behaviour (Elevated Zero Maze [EZM] and Tail Suspension Test [TST]) were assessed on days 1, 15, and 22. Upon euthanasia, plasma catalase activity was measured, and brain striatal histology was assessed. ANOVA was used for analysis, with $p < 0.05$ considered significant.

Results: Improved motor performances were observed in the HD+farnesol compared to HD groups by day 22 in both four-limb hanging and cylinder tests, although insignificant in the latter. Anxiety- (EZM) and stress-like behaviours (TST) were also significantly lower in HD+farnesol compared to HD groups by day 22. Farnesol also resulted in markedly higher catalase activity and reduced striatal neuronal degeneration in HD-treated rats.

Discussions: Farnesol treatment mitigated oxidative stress and conferred partial neuroprotection, resulting in improvements of both motor and emotional behaviors. Farnesol may target multiple pathological mechanisms of HD, including inflammation and redox imbalance.

Conclusions: Farnesol shows potential as a complementary therapeutic agent for HD, addressing both motor dysfunction and psychiatric symptoms via neuroprotective mechanisms.

Keywords: Huntington's Disease, Neuroprotection, Psychiatric Disturbances, Farnesol.