RAMAIAH UNIVERSITY OF APPLIED SCIENCES

Faculty of Pharmacy

FPH

Panpharmacon

A Quarterly E-Newsletter

Department of Pharmacology

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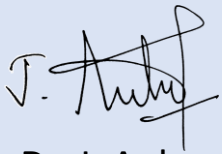
Editor's Note

Welcome to the Panpharmacon, I am delighted to bring you this quarter's newsletter. I am really encouraged by the feedback received from the readers of inaugural issue. This edition offers interesting scientific information, as well as an update regarding awards, achievements and departmental activities. I am looking forward to expand the scope of the Panpharmacon to include health related survey, behavioral (mental and physical) changes of parents and children due to pandemic-questionnaire analysis, innovative research ideas for short term UG and PG level projects and thrust areas for research, mini review articles etc. For any queries, suggestions, feedback or submission of articles please do not hesitate to contact our team via fphpanpharmacon@gmail.com



Dr. J. Anbu

Editor-Panpharmacon

Acknowledgment

Team Panpharmacon is very much thankful to RUAS management for providing a wonderful platform to explore and utilise our knowledge and skills. We wish to thank our Hon'ble acting Vice-Chancellor and Pro-Vice Chancellor for patronage and advising us on the importance of enhancing the visibility of workplace that stimulated us to come out with informative Panpharmacon E – Newsletter. We also thank all our well wishers and friends for supporting us in making this useful article.

Department of Pharmacology

Department of Pharmacology is one of the foundation departments of Faculty of Pharmacy, dedicated to the training of undergraduate, postgraduate pharmacy and doctoral students. The postgraduate program of the department was started in 2013 with intake of 15 students. The vision of the department is to become a top-ranked research and academic centre in the Pharmacology and Toxicology discipline in India that will be responsible for leadership in Pharmacology education, training, research, development and related services with the mission to contribute in the overall training of students, through enriching with the knowledge, attitude and skills needed to fit effectively and efficiently into both national and international arena. Department of Pharmacology is involved in various *in vitro* and *in vivo* research projects that expertise in the area of toxicological research, neuropharmacology, molecular pharmacology and cardiovascular pharmacology.

Key Features of the Department

- The Department of Pharmacology is well known for its research activities and its well-maintained animal house is approved by central government body CPCSEA
- The Department has well-qualified, experienced faculty with dedicated vision of research
- The laboratories at Department of Pharmacology are well equipped with instrumentation facilities including modern teaching techniques with computer assisted learning, library, audio-visual aids and other resources with all safety measures

- The laboratories provide facilities for the students to carry out all types of basic pharmacological *in vivo* and *in vitro* screening activities with computer simulated exercises
- The Department provides Hands-on experience on animal research with guidance from experienced faculty, technical skills and theoretical aspects of pharmacology individually
- Department provides an opportunity to students to interact with industry professionals through workshops, symposiums and seminars conducted in collaboration with pharmaceutical industries
- Keeping fit of postgraduates to make them competent in academics, research and industry with excellent placement support
- The department has legal Memorandum of Understanding with the reputed pharmaceutical industries to support our research scholars in performing advanced experiments and also understand the industrial climate

Effect of SARS-CoV-2 on CVS

The ongoing worldwide pandemic, COVID-19 is caused by the novel coronavirus (SARS-CoV-2). Reports suggest that subjects with pre-existing cardiovascular ailments are more prone to the severe effects of COVID-19. Few researchers also claim that the coronavirus can have long-lasting effects on the heart even after the infection is subsided. Symptoms such as shortness of breath, chest-ache and palpitations manifest again a month after recovering from COVID-19.

The proof of COVID-19's effect on the heart is derived from the research studying the impact of the SARS-CoV-2 on cardiac muscles. This virus starts impacting the respiratory system immediately upon entry into the lungs, thereby triggering an inflammatory reaction. This inflammatory reaction damages the cardiovascular system and heart undergoes collateral damage with severe immune response to the virus. Additionally, virus invades heart tissue, with the help of molecular elements known as ACE2 receptors.

ACE2 is a membrane-bound aminopeptidase that is highly expressed in the heart and lungs. It has a major role in the cardiovascular and immune systems. ACE2 is also involved in heart function and the development of hypertension. Some researchers found that the binding of SARS-CoV-2 to ACE2 can cause alteration of ACE2 signaling pathways due to which it causes acute myocardial infarction and lungs injury. COVID-19 is more severe in patients with Cardiovascular Diseases (CVD). This might be linked with the increased secretion of ACE2 in these patients compared with healthy individuals. Hypertension is powerfully associated with SARS-CoV-2 infection. This may be due to the pro-inflammatory state of this chronic illness and associated hypercytokinaemia that occurs in COVID-19.

Cardiac biomarkers that are linked with injury of the heart muscle are lactate dehydrogenase (LDH), creatine kinase (CK) and protein troponin I (TnI). Some studies revealed that the COVID-19 patients showed increased level of above biomarkers. The correct mechanisms of elevation of cardiac biomarkers among patients with COVID-19 have not so far been reported. The novel coronavirus and drugs used in its treatment may cause QT interval prolongation. Geriatric patients suffering from COVID-19 who also received a combination of the antiretroviral drugs: lopinavir and ritonavir, were more often seen to experience bradycardia. Patients exhibiting no heart ailment symptoms may start exhibiting the symptoms, unmasked by the virus. Fever and inflammation also make the blood more prone to clotting. They may also interfere with the body's ability to melt it.

Nowadays, doctors measure the cardiac bio-marker Brain Natriuretic Peptide (BNP) levels to check heart stretches. The increased BNP levels in COVID-19 patients are an indication of heart injury. According to the survey and researchers, the heart failure is an important cause of death in patients with COVID-19. This virus can directly damage the myocardium. There is a chance of increase in atherothrombotic events. These events occurs due to inflammatory destabilization.

Initially COVID-19 was assumed to be causing only lung disease, but further studies have revealed that the whole body gets affected. After all, it is still a new virus. More profound studies and research need to be carried out in order to understand the effects of the SARS-CoV-2 virus on the cardiovascular system and establish a correlation between them.

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Reference:

Kensuke M, Benjamin M. (2020). Impact of COVID-19 on the Cardiovascular System: A Review:J.Clin Med 9(1407),pp 1-18

:Background image source https://health.clevelandclinic.org/covid-19-can-also-damage-your-heart-not-just-your-lungs/

Brain Fog? Try Fasting!

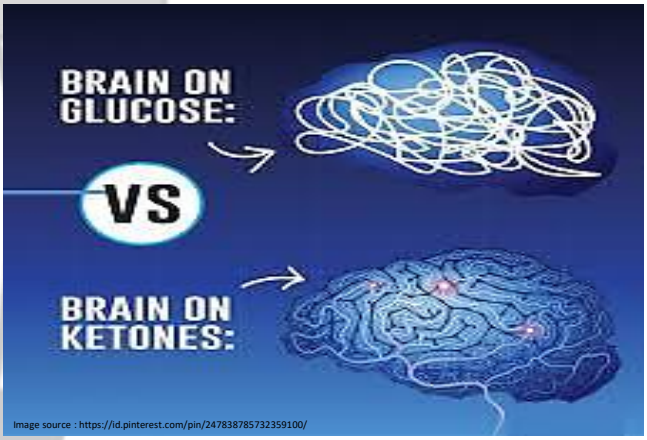
There are days when you feel you're not yourself or you're unable to think clearly or you might even have fuzzy thinking. A general term given to all these problems is "Brain Fog". So, what causes Brain Fog? There could be a number of causes ranging from nutrient deficiency, sleep disorders, overconsumption of sugar, chronic stress to hormonal changes, depression, anxiety or even medications. How do we focus or get rid of brain fog? Doctors and health coaches have a few recommendations to help us get rid of brain fog. They include, increasing physical activities, being an efficient sleeper, feeding brain with essential vitamins and minerals and engaging oneself in activities that increases mental acuity. Presently, many health experts suggest fasting holds potential to clear out brain fog.

Most of us skip meal and try to lose weight or possibly even abstain from eating in order to observe religious norms. What happens when we fast? Generally, fasting is a practice where one refrains from food for a particular period of time that elevates the ketone levels in our body. When one is not fasting, all the cells uses glucose as the fuel. Fasting exhausts liver off glucose and prompts the release fat from fat cells. This fat travels to liver, get converted to ketones that can be used as energy source. There is switch from the use of glucose as energy to ketone as energy source that usually happens after 10- 14 hours of not consuming food. And if one is active and exercises then the onset of this metabolic switch is accelerated.

So, how does ketones (formed due to fasting) effect our brain function? Many studies have reported the surprising benefits of this on cognitive function. It is seen that within few days of starting a fast, ketones become the favoured source of fuel to brain providing about 75% of its total energy requirement. Ketones not just constitute as the source of energy in muscle and brain but are also reported to enhance bioenergetics of neuron and cognitive performance.

Beta-hydroxybutyrate (BHB), a primary blood ketone plays a vital signalling function in hippocampal and cortical neurons via inducing transcription of brain-derived neurotrophic factor (BDNF). BDNF regulates neuron function that maintains the structure of synapse, increases the formation and survival of new neurons in the hippocampus and also increases resistance of neuron to injury and diseases that stave off neurodegeneration. The same has also been proved by animal studies. Additionally, fasting influences cell synthesis and degradation processes regulated by specialized protein kinases. Fasting affects fat metabolism by altering hormonal activities, it suppresses inflammation that helps in neuronal survival and healthy aging.

Are there any fasting regimens to follow to improve mood and hit peak cognition? Well, there are no hard and fast regimens to be followed. These days intermittent fasting (IF) is a rage, it involves restricting food intake over periods of time. Some people follow time restricted eating wherein they can go without food for more than 16 hours and eat only during the remaining 8 hours. While few can follow alternate day fasting with restricted calorie intake



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and eating normal meals on the other 5 days. Although, fasting is associated with a number of benefits but fasting might not suit all individuals and few may also develop fasting-related adverse effects like fatigue, nausea, headache, insomnia and dyspepsia. However, these effects can be avoided by maintaining a good balance between rest and exercise combined with adequate fluid and salt intake.

To conclude, it can be said that limiting the calorie intake for few hours in a day along with healthy eating habits could be a good way to help your brain health. So, for now, you might think twice about all the junk snack. Your brain might thank you later.

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Solianik, R., Sujeta, A., Terentjevienė, A., & Skurvydas, A. (2016). Effect of 48 h Fasting on Autonomic Function, Brain Activity, Cognition, and Mood in Amateur Weight Lifters. *BioMed research international*, 1503956.

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Background image source: https://medium.com/@PsychBehind/how-to-get-rid-of-brain-fog-with-3-steps-59677c5f707e

Healing Powers of Incy Wincy Spider!

Nature never fails to amaze us with how much it can contribute in our well-being. One such surprising creature is SPIDER. Apart from scaring us these spiders can aid us in pharmaceuticals, which has been discovered and experimented by researchers of different universities.

University of Bayreuth, Germany has found that spider silk has microbial repellent property against those microbes which are not killed by antimicrobials. So, silk used for building its home has been found to contain Fibroin 3 and 4 protein. The researchers engineered different recombinant spider silk proteins based on the consensus sequences of *Araneus diadematus* dragline – 2D biofilms & 3D hydrogels. These recombinant products form a barrier showing much better repellent activity as compared to the control materials. Along with this it showed mammalian cell attachment and proliferation which is better for healing.

Fig. Araneus diadematus

Fig.Venezuelan Pinkfoot Goliath tarantula

Not just spider silk but also its venom is found in medical use. The world's largest and poisonous spider Venezuelan Pinkfoot Goliath tarantula venom showed pain relieving properties. University of Queensland, Australia researchers screened around 28 spider venom but only this one showed promising results. It is observed that gut pain is challenging to manage compared to other pain and 20% world population suffer from this meanwhile the available drugs are ineffective.

There are particular sodium and calcium channels whose dysfunction causes chronic visceral pain. Two peptides which were isolated from this venom inhibits both the channels resulting in being potent at reducing sensory nerves of bladder, colon and visceral pain of Inflammatory Bowel Syndrome (IBS) model without interfering with channels on heart and other parts making it more specific and effective.

These findings showed promising results in its own way and definitely proved to have great potential in the future. Soon microbial infestation and pain from IBS is going to be history. So, turns out incy wincy spider isn't just for scaring but for exploring.

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Cardoso, F. C., Castro, J., Grundy, L., Schober, G., Garcia-Caraballo, S., Zhao, T., et al. (2020) A spider-venom peptide with multi-target activity on sodium and calcium channels alleviates chronic visceral pain in a model of irritable bowel syndrome. *Pain*, InPress.

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Impact of Gut Microbiota in Polycystic Ovary Syndrome

All of us are aware of the small microorganisms in our gut. A wide variety of fungal populations is there in our intestine and it varies with gender and life stages. Infants, children and females have a higher number of intestinal microbiota when compared to others. Do you know gut microbiota has other roles in our body beyond digestion? Well, there are many disorders such as Rett syndrome, Autism spectrum disorder, Polycystic Ovary Syndrome (PCOS) which disturb the homeostasis of the bacterial and fungal microbiota. PCOS is a common endocrine disorder that is characterized by tenacious irregular/missed periods, insulin resistance (IR), diabetes, obesity which are largely affecting women of reproductive age

How does Gut Microbiota affect PCOS??

In PCOS condition, gut microbiota like Tenericutes ML615J-28 and Bacteriodetes S24-7 was found to be altered. These stimulate inflammation in the GI tract which in turn causes infiltration of inflammatory factors into the bloodstream and leads to the inactivation of insulin receptors on our cells. An increase in the insulin and inflammatory factors in the bloodstream triggers the androgen synthesis in the theca cells of ovaries. Moreover, higher blood insulin level decreases Sex Hormone Binding Globulin (SHBG) leading to increased free testosterone in the blood. Studies confirmed that hyperandrogenism and elevated serum testosterone values can be associated well with PCOS.

Do you wonder if microbiota population alters in lean and obese humans? This could be the influence of microbiota in energy absorption pathway. Microbiota converts carbohydrates into simple sugars which further degrade them into CO2,H2, and short chain fatty acid then provide energy. In the PCOS, altered gut microbiota produces substances which cannot be utilized properly for energy but are only absorbed and thereby leading to obesity.

Obesity causes ovarian dysfunction, androgen production and thereby exacerbate PCOS.

Based on the above shreds of evidence it can be concluded that serum hormonal level and intestinal environment are closely associated with gut microbiota which in turn add to the occurrence of PCOS.

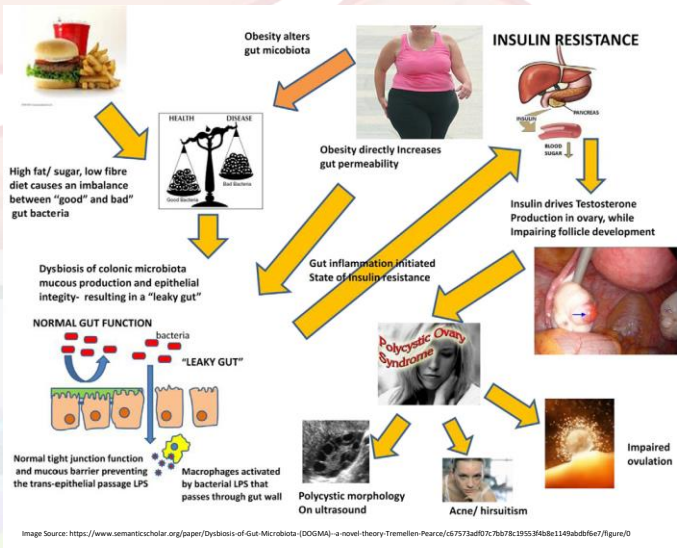

Fig. Pathogenesis of PCOS

Reference:

Zhao, X., Jiang, Y., Xi, H., Chen, L., & Feng, X. (2020). Exploration of the Relationship Between Gut Microbiota and Polycystic Ovary Syndrome (PCOS): a Review. Geburtshilfe und Frauenheilkunde, 80(2), pp.161–171.

Ms. Anusha Shrinarayan
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Common Pathological Features between Diabetes Mellitus and Alzheimer's disease

According to International Diabetes Federation, 463 million people are currently suffering from Diabetes mellitus (DM) worldwide. Diabetes mellitus related insulin modulation in hippocampus is reported to cause significant cognitive impairment. Diabetic patients are reported to be two-fold prone for Alzheimer's disease (AD) compared to non-diabetic individuals. This article intends to discuss certain common pathological features between DM and AD.

Amyloidogenesis: DM and AD are known to be amyloid forming diseases. The pathological hallmark of AD is the extracellular amyloid plaque. The primary component of this amyloid plaque is the Amyloid Beta peptide 1 – 42 (Aß). Aß is derived from the amyloid precursor protein (APP) by proteolytic cleavage. APP is the base of molecular pathology of which impairs glucose metabolism and tolerance in brain. The deposition of amyloidogenic peptide is also seen in the islets of Langerhans in DM in the same manner. The islet amyloid deposit is known as Islet Amyloid Polypeptide (IAPP) or amylin which is a 37 amino acid peptide.

Impairment in energy metabolism: Glucose is the main source of energy for the neurons in the brain. Thus any impairment in the utilization and glucose metabolism forms the pathological basis of DM. In total body glucose, 18-30% is consumed by brain. Disruption in the supply, transport or utilization of glucose can lead to neuronal damage and functional deficits in brain. Neuronal death always accompanies insulin resistance in developing human brains which may even cause permanent brain damage. This gives a clear indication that cognitive function is affected by disruption in glucose metabolism.

Oxidative stress and high level of advanced glycation end products (AGEs): Non-enzymatic glycation and oxidation of cellular proteins, nucleic acids, and lipids by reducing sugars leads to accumulation of AGEs in chronic hyperglycaemia. Accumulated AGEs binds to the receptor (RAGE) expressed on various cells such as microglia, vascular smooth muscle cells, mononuclear phagocytes and endothelial cells. AGEs interact with RAGE and triggers signalling cascade leading to increased oxidative stress by upregulating transcription factor NF-κB. This leads to increased secretion or production of inflammatory cytokines such as TNF-a and IL-6. Thus the interaction of RAGE-AGE is depicted as a major source of oxidative stress and inflammation which is correlated with diabetic complications as well as neurodegenerative diseases.

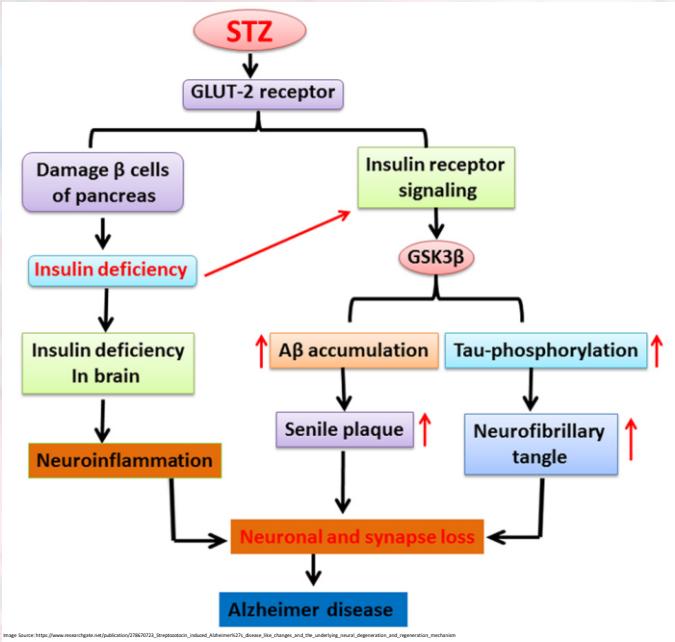

Fig. Mechanism underlying Diabetogenic agent Streptozotocin (STZ) induced Alzheimer's disease

Role of insulin receptor and insulin receptor substrates: Modulation of synaptic plasticity and cognition are the major functions of insulin receptor in brain. The abundant distribution of insulin receptors in cerebral cortex, amygdala, hippocampus are the evidences for their involvement in cognitive process and synaptic activity. Studies have shown that animals with increased amount of insulin receptor and insulin receptor substarte-1 in the hippocampal synaptic membranes show better spatial memory compared to the diabetic animals which showed reduction in hippocampal insulin receptor. Accumulated Aß competes with insulin to bind to the insulin receptor and interferes with insulin signalling. Impaired insulin signalling is reported to be a main cause of reduced clearance of Aß oligomers.

It is clear that AD and DM share a number of molecular processes that involves in neuronal degeneration. However, the exact molecular mechanism underlying the link between DM and AD still remains subtle. Although each of the factors mentioned could mediate its distinctive effect on AD pathology, it is likely that a combination of several of these factors acts in a synergistic way in promoting the development of AD.

Reference:

Sathiya R., Anita M., and Anbu J., (2018). Diabetes mellitus linked Alzheimer's disease–A review on sporadic form of Alzheimer's disease. Physiology and Pharmacology, pp. 141-145.

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Congestive Heart Failure and its Behavioural Effects in Mice

Myocardial Infarction (MI) is known as heart attack, is a medical incident where muscle tissue in the heart is damaged by lack of blood flow. Pain in the chest and upper body, trouble breathing, exhaustion and nausea are the most common signs, although MI can be entirely symptomless. If a significant portion of the cardiac muscle is destroyed, MI can lead to congestive heart failure (CHF), a condition where the heart is no longer able to pump enough blood to provide oxygen to all the body's organs and tissues. The severe prevalence and mortality rate of MI and CHF with increased risk factors in the population, makes scientific research extremely important. For such research, in place of human, mouse models are often used. Using mice incurs fewer ethical concerns, and being mammals their brain and behaviour are relatively similar to humans.

CHF and Depression

A study conducted in Germany in 2014 observed a marked depressive effect in mice with CHF induced by MI. The CHF mice appeared to experience anhedonia as shown by their reduced intake of sucrose compared to control mice. The worse the damage to each mouse's heart tissue, the stronger the observed reduction in its sucrose intake. The CHF mice were also less motivated and exhibited significantly less movement. Similar results were observed from a Japanese study published in 2016. Following a marked reduction in blood flow through the aorta, mice experienced heart failure and then exhibited what the researchers described as depressive-like behaviours. It is interesting to note that other conditions involving reduced heart function have also been associated with depression.

CHF and Anxiety

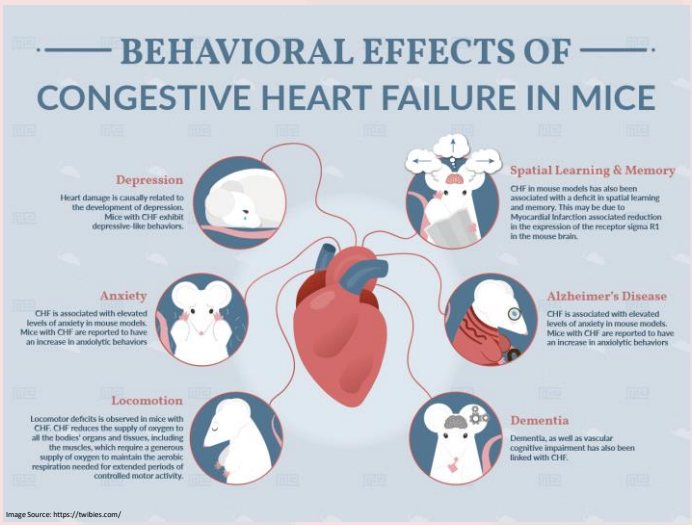
Interestingly, the researchers inferred that CHF was associated with elevated levels of anxiety in these mouse models. However, it is important to note that the several other studies focusing on hypertrophic cardiomyopathy also reported an increase in anxiolytic behaviours.

CHF and Locomotion

Since both depression and anxiety are assessed in mouse models behaviours that involve movement are central, it can be difficult to tease apart mood disorders from disorders of locomotion. If a CHF mouse moves less, does this mean that CHF is thus associated with depression, with motor deficits or both?. The results indicates that the diminished movement by CHF mice due to elevated anxiety could also be explained by a motor deficit where the mice are unable to move. A test which is more specific to motor deficits than to psychological states and which might help to tease apart effects of CHF on locomotion from its potential depressive or anxiolytic effects is the rotarod. A study did show a diminished performance by CHF mice on the rotarod test. This evidence strengthens the case for the presence of particular locomotor deficits in mice with CHF.

CHF and Spatial Learning & Memory

CHF in mouse models has also been associated with a deficit in spatial learning and memory. A study reported significantly worse performance in the Morris water maze test by mice with CHF following surgically induced MI. This study also reported deficits in a different form of memory as evidenced by a diminished performance of the CHF mice in an object recognition task. Reduced performance in the Morris water maze by CHF mice was also reported by the Chinese study. Furthermore, the Japanese researchers offer a molecular biological explanation for the memory deficit: an MI associated reduction in the expression of the receptor sigma R 1 in the mouse brain.



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Congestive Heart Failure and Alzheimer's disease

Brain needs a constant supply of blood in order to maintain optimal functionality. CHF has also been linked with the development of neurodegenerative disorders primarily associated with the aggregation of misfolded, cytotoxic proteins. There is some experimental evidence for a causal link between CHF and the development of Alzheimer's disease (AD). Some study showed alterations in the metabolism of beta amyloid, one of the proteins which when misfolded and aggregated is believed to underlie neuronal death in AD and also reported spatial learning deficits in the CHF mice, consistent with the memory loss normally seen in Alzheimer's disease. Several other studies have also reported apparent connections between CHF and AD-like pathology in mice.

Congestive Heart Failure and Dementia

Mice with CHF induced by expression of the signalling protein Gaq44 found a link between heart failure and vascular cognitive impairment. Vascular cognitive impairment refers to reduction in cognitive capacity resulting from insufficient blood flow to the brain and is less severe form of vascular dementia.

It is concluded that MI and CHF have philosophical effect on mouse behaviour and causal relationship between CHF and depression, anxiety, dementia, deficits in locomotion, spatial learning and memory. Hopefully, in the future, this knowledge will allow us to better understand and treat MI and CHF pathology in humans.

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Hay, M., Vanderah, T. W., Samareh-Jahani, F., Constantopoulos, E., Uprety, A. R., Barnes, C. A., & Konhilas, J. (2017). Cognitive impairment in heart failure: A protective role for angiotensin-(1-7). Behavioral Neuroscience, 131(1), pp.99-114.

Dr. Mohammad Azamthulla
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Webinar On Artificial Intelligence in Pharmacy Education and Research

Department Of Pharmacology, FPH, RUAS hosted a webinar entitled "Artificial intelligence in pharmacy education and research - a new paradigm shift" on 18th - October - 2020

The main agenda of the webinar was to emphasize on the use of various technological advancements related to Artificial Intelligence in Pharmacy Education and Research. Our Speaker, Dr. Haja Nazeer Ahmed briefed on the development of computational approaches in drug discovery, benefits of AI program in educating physicians about various medicines and ADRs, and training surgeons with the help of simulators for artificial surgery and development of computational approaches to carry out the task automatically.

It was interesting to know that, AI can be implemented in almost every aspect of the pharmaceutical industry, right from drug discovery and development to manufacturing and marketing. Pharma companies are increasingly adopting AI to improve & optimise the success rates of new drugs, create more affordable drugs, therapies, and most importantly, reduce operational costs. AI can be used to manage and improve all aspects of the manufacturing process, including Quality control, Predictive maintenance, Waste reduction, Design optimization, Process automation. So, AI can replace the time-consuming conventional manufacturing techniques, thereby helping pharma companies to launch drugs in the market much faster and at cheaper rates as well.

Recorded Webinar can be accessed through YouTube using the following link: https://youtu.be/oHGeAd7D64M

Resource Person

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Background Image source: https://enterprisetalk.com/tech-trends/ai/human-intelligence-can-fix-ai-shortcomings/

Faculty Awards & Publications

Faculty Awards

- **Mrs. Sathiya. R**, Asst. Professor from Department of Pharmacology, FPH have participated in AWSAR Research Story 2019 Contest by DST, Govt. of India. Her Research story titled **'Let us not give Alzheimer caregiver burden to our successors!'** based on the PhD work was selected for **AWSAR Award** as one of the top 100 research stories.

- "**Thathwamasi**" a short film mentored by **Mr. Damodar Nayak A** was Awarded **Best short film** in 1st Annual Kannada Rajyotsava celebrations of RUAS, Gokula Education Foundation(GEF). Short film can be accessed using the link - https://youtu.be/qEajgIg1bcQ

Research Publications

- Ramu, S., Murali, A., Narasimhaiah, G. and Jayaraman, A., 2020. Toxicological evaluation of Sargassum Wightii greville derived fucoidan in wistar rats: Haematological, biochemical and histopathological evidences. Toxicology Reports, 7, pp.874-882.

- Jyothi Lakshmi N, Anbu Jayaraman, M S Premakumari, Vaishnavi Balraj, Soumali Chakraborty, Moumita Mukherjee, Avinash Kumar. A Review on Multidirectional Holistic Approach and Impact of Adjuvants for the Treatment of Cancer. Int. J. Pharm. Sci. Rev. Res., 60(2), January - February 2020; Article No. 18, Pages: 107-120

- Moumita Mukherjee, Anbu Jayaraman, Soumali Chakraborty, Jyothi Lakshmi N, Vaishnavi Balraj. An update on the Current approaches for the enhancement of virility and the models for preclinical evaluation. J. Pharm. Sci. & Res. Vol. 12(5), 2020, 612-618.

- Soumali Chakraborty, Anbu Jayaraman, Jyothi Lakshmi N, Vaishnavi Balraj, Moumita Mukherjee. Migraine-A Review on Current Strategies in Diagnosis, Management and Evaluation Methods-An Update. /J. Pharm. Sci. & Res. Vol. 12(5), 2020, 691-697.

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