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Department of Pharmacology

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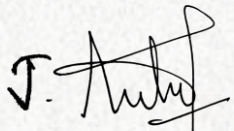
Editor's Note

Hello Readers !!!

This quarterly issue focus on recently published reports from different directions of health concerns globally. The current issue introduces "EcHo – Voice of Alumni", a new section that includes the informative contributions from our alumni. Apart from covering conventional informative scientific articles, the issue also features brain storming section for the readers, I encourage the readers to participate in it. I would like to personally thank Dr. Nagendra Babu & Dr. A Prashanth Saraswati for their contribution for this issue and to all other contributors for having put their thoughts and experiences into an engaging read.

For any queries, suggestions, feedback or submission of articles please do not hesitate to contact our team via fphpanpharmacon@gmail.com. We would love to hear from you and elevate the quality of the newsletter to serve you better.

Happy reading !!!



Dr. J. Anbu

Editor-Panpharmacon

Acknowledgement

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 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, an E – Newsletter. The Editorial Board is thankful to The Himalaya Drug Company, Bengaluru for sponsoring this quarterly issue. We also thank all our colleagues, well wishers and friends for supporting us in making this newsletter.

LEPROSY DRUG BECAME A HOME TREATMENT FOR COVID-19 !!

COVID-19

COVID-19/Corona virus disease is an infectious disease caused by a family of viruses that mainly affects the respiratory system causing common cold to severe diseases like Middle East Respiratory Syndrome (MERS) and Severe Acute Respiratory Syndrome (SARS).

LEPROSY

It is a curable infectious disease caused by *Mycobacterium leprae* which affects peripheral nerves, skin, eyes, and nose leading to muscle weakness or paralysis (hands and feet), numbness of the skin, and blindness (affecting facial nerves).
Clofazimine is an essential drug used to treat leprosy. It was discovered in 1954 and approved by the FDA and the World Health Organization's (WHO) list of essential medicine. Clofazimine is also a potent antiviral drug that acts by antagonizing the replication of SARS-CoV -2 and MERS-CoV and is now being widely used to treat SARS and MERS. It is also seen that Clofazimine in combination with Remdesivir has a synergistic antiviral effect in the treatment of COVID-19.

How the leprosy drug made its way in the treatment of COVID-19?

The scientists chose Clofazimine as a possible candidate for treating COVID-19 because of its previously reported anti-viral activity. It acts by inhibiting the viral glycoprotein induced cell fusion along with the inhibition of viral helicase (enzyme responsible for replication). Animal studies were conducted including SARS-CoV-2 virus infected hamsters which were later treated with Clofazimine. It was seen that Clofazimine prevented the SARS-CoV-2 viral infection by either blocking the entry of the virus into the host cell or by inhibiting the RNA replication.

"Clofazimine" has shown potent antiviral activity and also possessed broad spectrum Anti-CoV efficacy

Clofazimine has also been proven to inhibit the replication of MERS-CoV (type of coronavirus that causes Middle East Respiratory Syndrome in human lungs). More importantly, the drug did not show any adverse reaction in the animal.

Overall, Clofazimine showed potent antiviral activity and also possessed broad spectrum Anti-CoV efficacy. It antagonized the SARS-CoV-2 and MERS-CoV. The drug which is known for its anti-leprotic activity, is now known to act against COVID infection. Thus, scientists believe that it will act as a one of the potent weapon in overcoming this pandemic.

Reference:

Yuan, S., Yin, X., Meng, X., Chan, J.F.W., Ye, Z.W., Riva, L., Pache, L., Chan, C.C.Y., Lai, P.M., Chan, C.C.S. and Poon, V.K.M., (2021). Clofazimine broadly inhibits coronaviruses including SARS-CoV-2. Nature, pp.1-9.

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ARE HERPES VIRUS INFECTIONS LINKED TO ALZHEIMER'S DISEASE?

Introduction

Alzheimer's disease (AD) is a type of progressive dementia that causes memory loss, cognitive disabilities, and lack of verbal skills. Unfortunately, current therapies increase patient's life span and functioning abilities by reducing the rate of decline of cognitive functions. However, there are no therapies to prevent the progression of AD.

You will be surprised to know that the post-mortem analysis of brain tissues of AD patients showed an increased level of Human Herpesvirus (HHV-6A), Human Herpesvirus (HHV-7), and Herpes Simplex Virus 1 (HSV1) when compared to the brain tissues of healthy-aging individuals.

HSV1 can elude the body's immune responses, which are counteracted by the Immunoglobulin G3 (IgG3). Even in the presence of a high quantity of IgG3, the AD sera failed to neutralize the specific HSV1. It was reported that the continuous accumulation of AD biomarkers in the neocortex and hippocampus is seen after recurrent virus reactivation. Eventually, the cognitive impairments augment, which became irreversible after seven cycles of reactivation of the virus. HSV1 prominently triggered the amyloid-β_{42} formations and catalysed its aggregation. *In-vitro* studies proved that the distribution of amyloid precursor protein (precursor of neurotoxic Aβ)

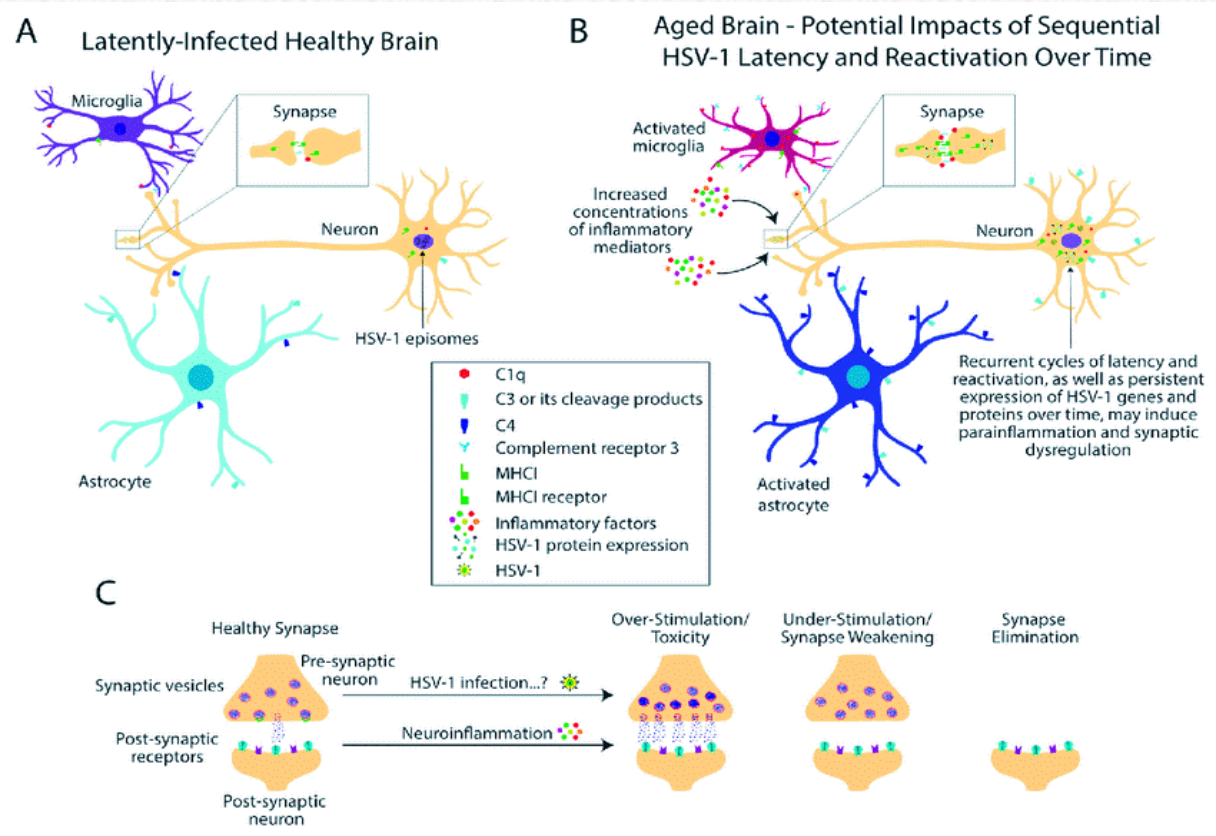

Fig: Recurrent cycles of HSV-1 latency and reactivation may cause aberrant neuroinflammatory responses and synaptic dysfunction with advanced age.

would be affected by HSV1 infection by increasing its phosphorylation and aggregation of Aβ. HSV1 was found in high levels in both control and AD brains after post-mortem, while HHV-6A was present in 70% of AD brain and 40% of control.

Pathogenesis underpinning the development of AD

- One of the virus's mechanisms in AD's pathogenesis is increasing the inflammation and thereby damaging the brain, which may happen in elderly individuals under stress or with a lowered immune system.
- Another possible mechanism is that the antimicrobial activity of Aβ against HSV1 leads to its accumulation, which in turn causes neurotoxicity and further develops into AD.

"Post mortem of AD patients showed increased level of herpes virus in their brain tissue"

- An imbalance in the formation and degradation of autophagosome due to HSV-1 and HHV-6A is observed in the AD patients, leading to accumulation of Aβ and dysregulation of metabolism of tau protein.
- Infection in the glial and microglial cells enhances the oxidant species formation, which in turn causes inflammation and eventually develops into AD.

Amyloid plaque-like structures were seen in the HSV-1 infected human brain-like tissue, strengthening the hypothesis mentioned above. Hence, we can conclude that HSV-1 and HHV-6A may be the causative factors and possible AD targets.

Reference:

Rizzo, R., (2020). Controversial role of herpesviruses in Alzheimer's disease. PLoS pathogens, 16(6), pp.e1008575.

Mangold, C.A. and Szpara, M.L., (2019). Persistent infection with herpes simplex virus 1 and Alzheimer's disease—a call to study how variability in both virus and host may impact disease. Viruses, 11(10), pp.966.

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3D-PRINTING OF HUMAN ORGANS CLOSER TO REALITY

3D Bioprinting is a method of additive manufacturing that uses cells and other biocompatible materials as “inks”, also known as bioinks, to print living structures layer-by-layer which mimic the behaviour of natural living systems. 3D bioprinting can be used to reconstruct tissue from various regions of the body. This technology can also potentially be applied to bone, skin, cartilage and muscle tissue.

Materials for 3D printing usually consist of alginate or fibrin polymers that have been integrated with cellular adhesion molecules, which support the physical attachment of cells. Such polymers are specifically designed to maintain structural stability and be receptive to cellular integration.

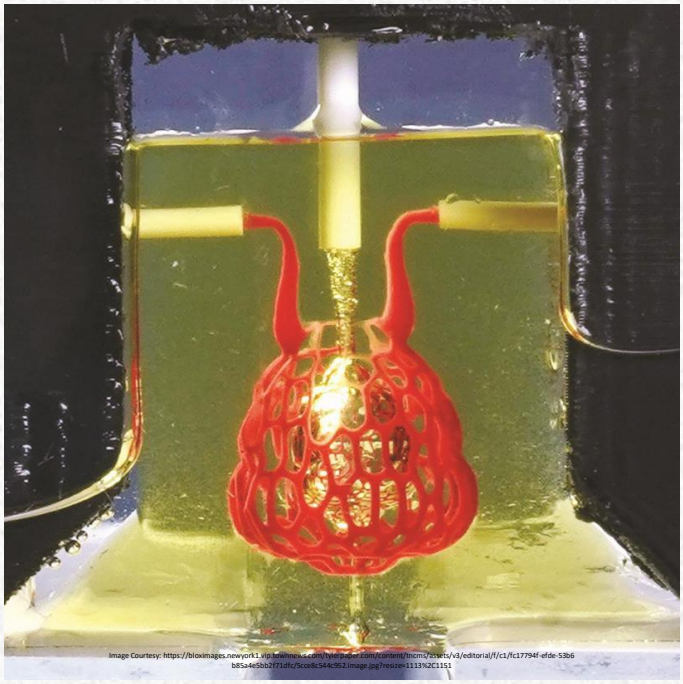

Image Courtesy: https://bloximages.newyork1.vip.townnews.com/tylerpaper.com/content/tncms/assets/v3/editorial/f/c1/fc17794f-efde-53b6-b853-4e5bb2f71dfc/5ccadc544c952.image.jpg?resize=1113%2C1151

Fig: 3D Printed Lung

Recently researchers at Lund University in Sweden have invented a new bioink i.e., a tissue-specific hybrid bioink, composed of a natural polymer, alginate, reinforced with extracellular matrix obtained from decellularized tissue (rECM).

Reconstituted extracellular matrix (rECM) has rheological and gelation properties useful for 3D bioprinting while retaining biologically inductive properties supporting tissue maturation *Ex-vivo* and *In-vivo*. This permits tiny human-sized airways which are 3D-bioprinted for the first time with the help of patient cells.

The 3D-printed models are biocompatible and assist new blood vessels into the transplanted matter. This is a significant step towards 3D-printing organs.

Chronic lung diseases are the 3rd main reason for loss of life globally with an European Union (EU) value of greater than €380 billion yearly. There is no cure for many chronic diseases and the final-stage option for the patients is lung transplantation. But to meet the clinical demand there aren’t sufficient donor lungs. Due to this reason, researchers are finding ways to increase the lung’s availability for transplantation by fabricating lungs in a laboratory by uniting the cells with a bioengineered scaffold.

“In the near future 3D printed organs can be used for organ transplantation”

In this process, the researchers have used the bioink to 3D-bioprint the small human airways which contains two cell types similar to human airways and this bioink can be used for any type of tissue or organ. They used a mouse model which closely resembles the immunosuppressant consumed patients who are undergoing organ transplantation. When it is transplanted, they noticed that 3D-printed models made by new bioink are well tolerated and assisted new blood vessels.

Based on the promising results, it can be said that, in the near future 3D printed organs used for organ transplantation can drastically minimise the time consumed for organ matching and rejection studies.

Reference:

De Santis, M.M., Alsafadi, H.N., Tas, S., Bölükbas, D.A., Prithiviraj, S., Da Silva, I.A., Mittendorfer, M., Ota, C., Stegmayr, J., Daoud, F. and Königshoff, M., (2021). Extracellular - Matrix - Reinforced Bioinks for 3D Bioprinting Human Tissue. Advanced Materials, 33(3), pp.2005476.

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DOORWAY SYNDROME

Ever wondered why you entered a room in the first place?? What did you want to get from the supermarket or suddenly forgot what you wanted to say?? Well, you're not alone. We all have faced this type of annoying yet common experience; as if the mind voids as the location changes. One thing we know for sure is that we forget things only because we don't pay enough attention. But this forgetfulness appears only when our brain is busy and this psychological phenomenon is termed as Doorway Syndrome or Location Updating Effect and it exposes the intricacy of human memory.

The doorway effect is seen when our attention shifts between the hierarchy of actions and reflect the dependence of our memory. The doorway effect occurs when there are changes in the physical environment, like moving to a different room via a door, and mental or meta-physical environment, like visualizing crossing a doorway or thinking about different things.

The "doorway effect" or location updating effect proposes that a location change establishes a need to update one's knowledge of the ongoing events. This doorway effect is also applicable to the virtual atmospheres as in computer games or even when an individual is asked to visualize passing through a door or threshold. When there are alterations in the physical atmosphere the mental environment gets modified as well.

"The doorway effect occurs when there are changes in the physical environment, like moving to a different room via a door, and mental or meta-physical environment, like visualizing crossing a doorway or thinking about different things"

A study comprising of 74 volunteers was carried out by a team of scientists led by Oliver Baumann, from Bond University, Australia; wherein they were asked to move across computer-generated 3D rooms and trying to remember certain objects from previous rooms, as they went on. After this experiment, it was noted that the participants remembered everything and the doorway effect was not observed. Further, the participants were asked to repeat the task of moving across the rooms and remembering certain objects along with counting backward. This time the volunteers started to forget things as overloading their memory made them susceptible to the doorway effect.

Other experiments were carried out where the participants were asked to walk through partitioned corridors and observing other people doing the same habitual tasks while completing memory assignments. The results of these experiments also didn't exhibit the doorway effect.

After analysing the results from all the experiments, the researchers predicted that walking past a door doesn't cause memory wipe but the moving from one location to a significantly different location causes a sudden and immediate change of scene which prepares our mind to receive something new, hence triggering the effect.

The brain's ability to sort and manage memories can be useful as it helps in making sense of the surroundings. Understanding how changes in the environment can reset working memory to some extent could be useful in managing the doorway effect. But an easy way to avoid the doorway effect is to prioritize and pay more attention to a task until it's done because a busy and overloaded brain will always fuel this effect.

Reference:

McFadyen, J., Nolan, C., Pinocy, E., Buteri, D. and Baumann, O., (2021). Doorways do not always cause forgetting: a multimodal investigation. BMC Psychology, 9(1), pp.1-13.

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SEMAGLUTIDE- A GAME CHANGER DRUG FOR OBESITY

Is Semaglutide a game changer drug to treat Obesity? Semaglutide acts by taking control over the regulating system of the body's appetite by transducing signals to the brain to reduce hunger and calorie intake. Obesity may lead to many life-threatening diseases like heart diseases, liver malfunctioning, type 2 diabetes mellitus, and various types of cancer. Moreover, it is one of the major risk factors of mortality in contrast to the COVID-19 virus.

Professor Batterham, UCL medical college, London reported the breakthrough for the development of the healthcare facility for the treatment of Obesity. It is observed that 75% of the patients who received 2.4 mg of Semaglutide showed a significant reduction in 10% to 20% of their weight.

"Semaglutide acts by taking control over the regulating system of the body's appetite by transducing signals to the brain to reduce hunger and calorie intake"

It was observed that Semaglutide not just decreased the body weight but also had beneficial effect on heart. It reduced the risk factors associated with diabetes and thus improved quality of life.

Professor John Wilding, University of Liverpool, England in his studies quoted a remarkable advancement in the treatment of Obesity. He reported that, the Glucagon-like Peptide 1 (GLP1) hormone which is structurally similar to Semaglutide acts by releasing in the blood from the guts in the post-meal period.

Accordingly, Semaglutide, which acts as a GLP1 agonist, can cause weight loss by reducing hunger, giving the filling of fullness, and hence reducing the calorie intake.

Phase III of the Clinical Trial took place in 129 sites across 19 countries involved 1961 patients, in which 2.4 mg of Semaglutide was given through a subcutaneous route. It was observed that there was a significant weight loss of 15.3 kg with a reduction of BMI of -5.54 while in the placebo group weight reduction of 2.6 kg was observed with a decrease in BMI of -0.92. It was also observed that Semaglutide reduces the risk factors for diabetes and heart disease as mentioned above. Thereby, Semaglutide can be considered as a potent game changer drug to treat obesity.

Reference:

Wilding John P.H., Batterham R.L., Calanna S., Davies M., et. al. (2021). "Once-Weekly Semaglutide in Adults with Overnight or Obesity. N Engl J Med. 384(11), pp. 989.

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MESSENGERS OF CELLULAR COMMUNICATION: Ca^{2+} SIGNALS THROUGH IP_3 RECEPTORS

Plasma membranes (PMs) – made of phospholipids – separate cells from each other and from the external environment. By forming a physical barrier between the cell and its external environment, PM protects the integrity of intracellular components, while also enabling communication with the outside world. Embedded within these PMs are receptor proteins, which serve as antennae that can sense several stimuli, both endogenous (e.g. hormonal - parathyroid, adrenaline, glucagon etc) and exogenous (e.g. sensory - light, smell and taste; and others). These receptors interact with stimuli and translate them into specific cellular responses using a range of proteins and signalling molecules.

Earl Sutherland, an American pharmacologist, was curious to understand how adrenaline, a hormone produced from adrenal glands during stressful situations, was able to cause production of glucose in liver cells. His work led to the discovery of an intracellular signalling molecule (cyclic adenosine monophosphate, cAMP - the first 'second messenger') and in process won him a Nobel prize in 1971. Cells employ several signalling pathways to decode external stimuli using these 'second messengers' (e.g. cAMP, IP_3, Ca^{2+}, DAG, cGMP). Such messenger molecules need to be rapidly delivered in necessary concentrations at adequate timescales and swiftly removed, to sustain essential life functions.

"Calcium (Ca^{2+}) ions are the simplest and the most ubiquitous, and versatile of all intracellular messengers"

Calcium (Ca^{2+}) ions are the simplest and the most ubiquitous, and versatile of all such messengers. Ca^{2+} binds a myriad of proteins within cells to regulate a wide variety of biological processes as diverse as fertilization, cell division, gene transcription, development, secretion, learning and memory and cell death with exquisite specificity. Hence, dysregulation in Ca^{2+} homeostasis within cells often underlies several pathologies including diabetes, hypertension, cancer and a number of neuro-pathologies. Ca^{2+}, being no more than a simple ion, cannot be synthesized upon need. Hence, to make it readily accessible for signalling needs, cells control its intracellular concentration by moving it across distinct membrane compartments (Fig. 1).

In order to achieve a tight control of cellular Ca^{2+} concentration ($[Ca^{2+}]c$), cells express an intricate ion transport machinery that includes ion channels, pumps and ion exchangers and several Ca^{2+} sequestering buffers (see Table 1). Ca^{2+} is stored at high concentrations in compartments within the cell and in the extracellular fluid from which, it can be rapidly delivered into the cytosol. Increases in $[Ca^{2+}]c$ can be rapidly achieved by the openings of Ca^{2+} channels resident within the PMs and the endoplasmic reticulum (ER).

These channels can be activated by ligands, voltage or by other proteins. Removal of Ca^{2+} from the cytosol is accomplished by several Ca^{2+} pumps and exchangers, while Ca^{2+} buffers help to regulate $[Ca^{2+}]c$ in distinct spatial domains within cells. While voltage-gated channels allow Ca^{2+} entry across the PM in excitable cells (e.g. neurons, skeletal muscles and cardiac muscle), receptor-activated Ca^{2+} release from intracellular stores is a main mechanism in non-excitable cells. Endoplasmic reticulum is the major Ca^{2+} store within cells, and forms a contiguous tubular network made of membranes.

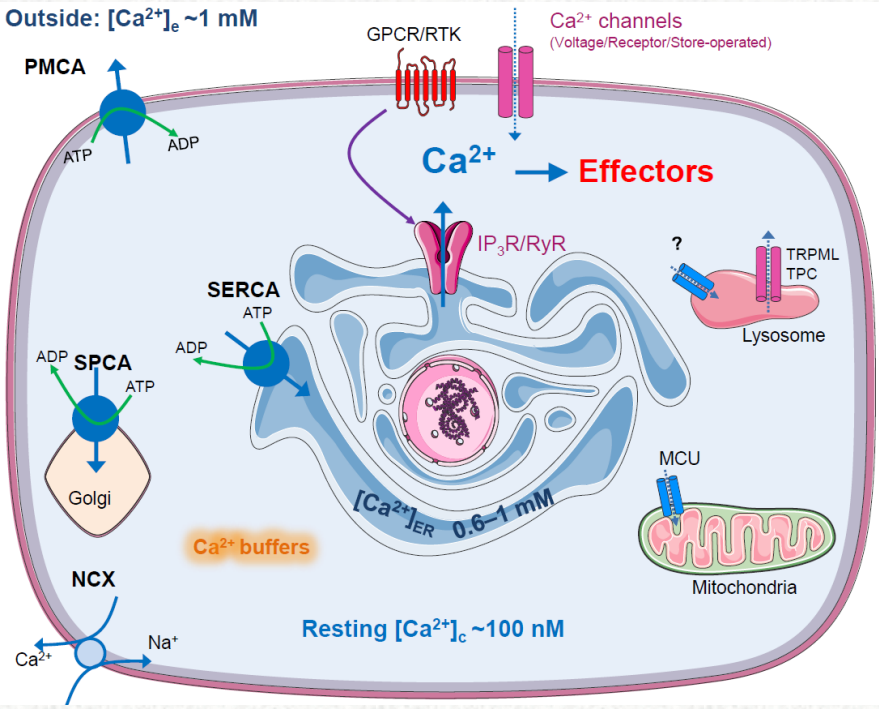

Figure 1. Cellular Ca^{2+} levels are tightly controlled. External stimuli are sensed by PM receptors and cause IP_3 synthesis. IP_3 and Ca^{2+} bind to, and activate IP_3Rs to release Ca^{2+} from the ER. Spatially distinct Ca^{2+} signals can regulate diverse cellular processes. Loss of Ca^{2+} from the ER is sensed by STIM1 which accumulates at ER-PM junctions and activates Ca^{2+} entry through PM Ca^{2+} channels.

Table 1: Summary of Ca^{2+} gain and extrusion mechanisms within the cell.

Ca^{2+} gain into cytoplasm		Ca^{2+} extrusion		Ca^{2+} buffers
Across the PM	From intracellular stores	Across the PM	Into intracellular stores/organelles	
L,P/Q,R,T-type Ca^{2+} channels nAChR TRPC, Orai	IP_3R RyR TRPML TPC	PMCA NCX	SERCA SPCA MCU unknown-lysosomal	Parvalbumin, Calmodulin, Calbindin-D28K Calretinin

Expressed within the ER are the inositol trisphosphate (IP_3) receptors (IP_3Rs) – the most ubiquitous Ca^{2+} channels across all animal cells – which are activated when they bind to IP_3 and Ca^{2+}. By linking extracellular stimuli, that activate G-protein coupled receptors (GPCRs) and receptor tyrosine kinases (RTKs) leading to production of the ligand IP_3, and a diverse range of cellular functions, IP_3Rs play a major role as a Ca^{2+} signalling hub within cells. Ca^{2+} release through IP_3 Rs results in loss of Ca^{2+} from the ER, which must be swiftly replenished to sustain signalling needs and other Ca^{2+}-dependent processes. Hence, cells use a Ca^{2+} sensor to detect loss of Ca^{2+} in the ER and activate Ca^{2+} entry into the cells through channels in the PM, by a mechanism known as 'store-operated Ca^{2+} entry' (SOCE) (Fig. 1). Such a tight control of $[Ca^{2+}]c$ is crucial for cells as small changes in $[Ca^{2+}]c$ can mean life or death for the cell. It is rather puzzling to think that Ca^{2+}, as a ubiquitous messenger, regulates cellular functions that are

'diverse yet specific'. How could Ca^{2+} signals be diverse and yet specific at the same time and what allows Ca^{2+} to achieve these apparently conflicting demands? It has long been proposed that Ca^{2+} signals are organized within cells both in space as well as in time which could help to achieve specificity. However, it is not completely understood yet as to how Ca^{2+} achieves spatial and temporal organization within cells.

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About Dr. Nagendra Babu

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Nagendra Babu Thillaiappan trained as a pharmacist and then held positions in industry before completing an MSc in Pharmacology at the University of Oxford, during which he worked on pharmacological tools to aid clinical diagnosis of Gaucher disease. He was then awarded a Cambridge International ship at the University of Cambridge, where he completed a Ph.D. in Pharmacology exploring the relationships between the geography and dynamics of IP_3 receptors and the Ca^{2+} signals they generate, using gene-editing and optical microscopy. His postdoctoral work at Cambridge with Professor Colin Taylor investigated the mechanisms that license IP_3 receptor activity. Presently, he is an Assistant Professor of Pharmacology at the College of Medicine, Qatar University.

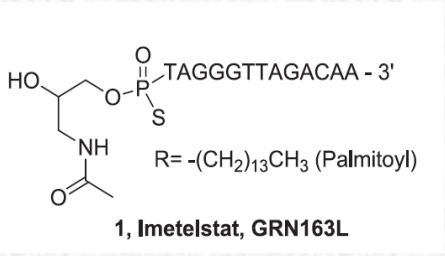
Nagendra Babu has expertise in calcium signalling, cell biology, advanced optical imaging and analysis including super-resolution imaging, molecular biology including gene-editing and laboratory-based teaching of *In-vitro* and *In-vivo* pharmacology. He managed an optical imaging facility in Cambridge and has a special interest in advanced optical microscopic methods including super-resolution microscopy. Nagendra Babu has published his research in high- impact journals and has been selected to speak at international conferences of The Physiological Society, UK and the Biophysical Society, USA. His research interests include calcium signalling with a special emphasis on IP_3 receptors, its contribution to health and disease and use of bioinformatics and artificial intelligence in biology. He is actively involved in teaching and research and is interested in public understanding of science through outreach activities.

TELOMERASE INHIBITORS: A PROMISING THERAPEUTIC TOOL TO FIGHT CANCER

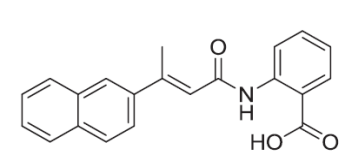
The 2009 Nobel Prize in Physiology or Medicine was conferred upon Elizabeth H. Blackburn, Carol W. Greider, and Jack W. Szostak for their influential work on telomere biology and biochemistry. Telomeres (often referred to as “biological clocks”) are protective ends of chromosomes that shield them from unwanted events such as DNA damage, recombination, end-to-end-fusion, and exonucleolytic damage. Telomere length is representative of the overall health status and viability of the cell. A typical eukaryotic telomere is composed of a double-stranded DNA with a Guanine-rich sequence, TTAGGG, and associated proteins. Moreover, it also contains a single-stranded 3’ overhang, which folds into the double-stranded telomere and forms the ‘T-loop’ structure. T-loop structure is responsible for capping the chromosomes and thus plays a protective role by preventing the loss of genetic information.

During every round of cell division, there is an incomplete replication of telomeres, leading to the shortening of telomere, termed as the ‘end-replication problem’. Telomere shortening occurs because of the loss of 50–200 nucleotides per round of replication. At a certain critical length (4–6 kb), the continued shortening is detected as DNA damage, leading to cell death events, such as senescence or apoptosis. Proliferative cells exploit and hijack the enzyme telomerase as a replenishing tool to overcome the end replication problem which leads to continued proliferation.

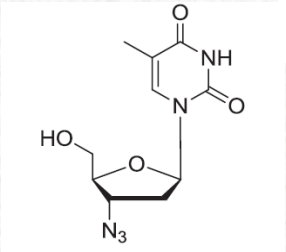
Telomerase (Reverse Transcriptase) is a ribonucleoprotein seen in mammalian cells that adds TTAGGG tandem repeats to the 3’ end of the telomere. The telomerase enzyme is upregulated in 80–90% of cancer cells and has been detected in various cancers, such as breast, colon, melanoma, ovarian, pancreatic, oral, prostate cancers, and cancers of soft tissues. Due to its increased expression in cancer cells, telomerase has been considered as an effective drug target and biomarker in cancer. Telomerase activity is higher in advanced and metastatic tumors, whereas in normal cells, it is minimal or undetectable. Therefore, utilizing the differential activity of telomerase in normal and cancer cells is essential for the development of novel anticancer agents.



1, Imetelstat, GRN163L



3, BIBR1532



2, Zidovudine

Fig: Representative structures of some telomerase inhibitors

Several approaches have been identified to inhibit telomerase activity, such as (i) targeting telomerase reverse transcriptase catalytic activity; (ii) targeting telomerase–RNA interactions; (iii) directly targeting the telomerase RNA component; and (iv) targeting dyskerin in ribonucleoprotein biogenesis. In this regard, many telomerase inhibitors have been developed and evaluated for their anticancer potential.

"Telomerase (reverse transcriptase) is a ribonucleoprotein seen in mammalian cells. Due to its increased expression in cancer cells, telomerase has been considered an effective drug target and biomarker in cancer"

These inhibitors include chemically modified oligonucleotides (e.g., imetelstat, 1), nucleoside analogs (e.g., zidovudine, 2, AZT), small molecules as synthetic mixed-type non-competitive non-nucleoside analogs (e.g., BIBR1532, 3), natural compounds (rho-docyanin, curcumin, and genistein), G-4 stabilizers (telomestatin) and vaccines. These inhibitors have shown promising results against cancer and paved the way for the development of potent and highly selective anticancer agents.

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Dr. A. Prasanth Saraswati
Postdoctoral Researcher
Universita Degli Studi di Milano, Italy

About Dr. A. Prasanth Saraswati

Dr. A. Prasanth Saraswati is currently employed at Universita Degli Studi di Milano as a Postdoctoral Researcher under the guidance of Prof. Sara Sattin. His research focuses on the development of novel antibacterial molecules aimed at combating antibacterial resistance.

In 2020, he was awarded a PhD in Medicinal Chemistry from the University of Siena as a Marie Curie Early Stage researcher, his research focused on the development of novel anticancer agents for treatment of cancers and rare diseases. He obtained an MS (Pharm) in Medicinal Chemistry from National Institute of Pharmaceutical Education and Research, Hyderabad after graduating from M. S. Ramaiah College of Pharmacy, Bangalore with a Bachelors in Pharmacy in 2013.

PRINCIPLES FOR SUCCESSFUL LIFE! – FOR ASPIRING ENTREPRENEURS

Department of Pharmacology, FPH, In association with RUAS Innovation Center, RUAS hosted a webinar entitled "Principles for successful life! – For aspiring entrepreneurs" on 5th - March - 2021

Mr. Meganathan Panchalan is a certified Passionpreneur mastermind and practitioner of Neruro Linguistic Programming (NLP) from global NLP training centre

About Webinar

Mr. Meganathan Panchalan delivered a motivational session that highlighted strategies including Action, Emotions and Acceptances oriented approach to overpower stress, fear, anxiety, depression, Job loss/insecurity.

He also emphasised the importance of being mentally fit by using following 5 techniques Increasing Consciousness Level, Expanding Context, Managing Thoughts, Taking risk and Celebrating failure.

Resource Person

Meganathan Panchalan

Passionpreneur.

UPCOMING EVENT

BASIC UNDERSTANDINGS OF AI & ML IN PHARMACEUTICAL INDUSTRY AND AS A CAREER PATH

Speaker

Mr. Manoj Kulkarni

Senior Executive

Formulation Development

Strides Pharma Science Limited Bengaluru, India

Speaker Profile

Mr. Manoj Kulkarni is a Senior Executive at Formulation Development in Strides Pharma Science Limited, Bengaluru, India. He was earlier associated with Apotex Research Private Limited, Formulation Development at Shilpa Medicare Limited, and Sanofi-Synthelabo Private Limited as Research Assistant & Research Scientist I, Executive and Trainee Scientist respectively. He has overall 8+ years of experience in Formulation Research and Development.

During his tenure, he was responsible for developing generic oral solid dosage forms for US, Canada, Europe and Australian markets. He is skilled in developing stable, scalable and bioequivalent formulation for highly regulated markets. He is trained to design, formulate and develop process as per Quality by Design employing Design expert software.

Convener

Dr. S. Bharath

Dean(I/C), FPH

Event Co-ordinator

Ms. Gouri Nair

Assistant Professor

Date: 15/May/2021

Time: 10:30 AM – 12:30 PM IST

Live Event Link:

https://cutt.ly/wvOYfmz

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For Queries: fphcologywebinar@gmail.com

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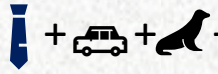
Mind Lab

Who am I ?!

1.  = I dilate your pupil
2.  = I reduce inflammation and relieve pain
3.  = I was approved recently to treat tumor
4.  = I am given for spinal muscular atrophy
5.  +in = I kill microorganism

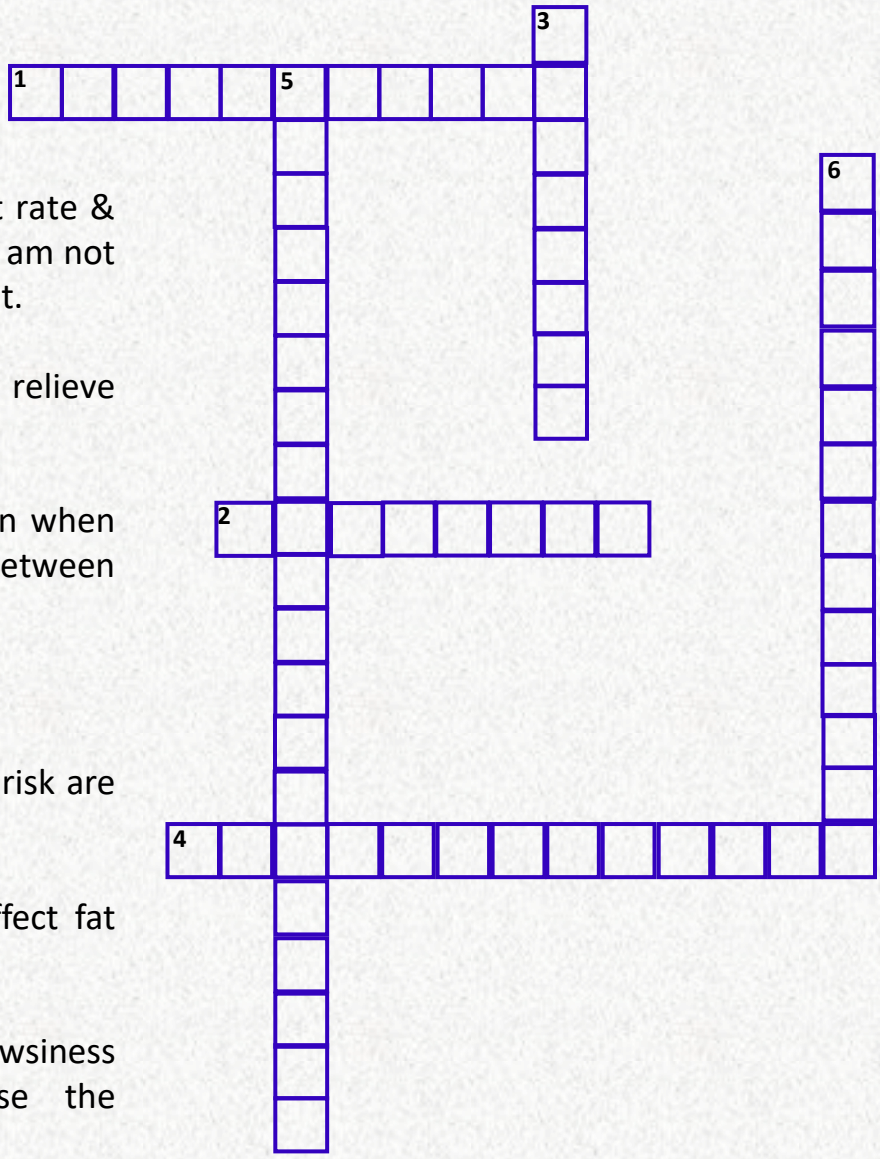
Find the word

T	A	B	V	A	C	A	B	A	P	N	A	P
B	P	Z	P	B	P	M	C	C	E	D	C	C
A	M	A	L	C	A	N	A	O	N	L	O	A
P	R	M	N	H	N	P	B	M	T	E	M	B
T	E	E	M	P	M	C	T	V	M	I	V	T
A	M	A	M	M	H	O	A	I	B	H	I	O
Z	D	I	B	L	T	A	Z	R	V	S	R	G
V	E	S	E	N	B	D	R	M	I	I	M	R
E	S	E	S	H	I	L	C	M	R	V	T	A
R	I	D	T	A	Z	E	A	P	A	O	P	V
I	V	Z	H	C	O	I	R	N	O	C	M	I
K	I	A	C	A	B	H	T	A	C	N	O	R
R	R	T	A	Z	V	S	O	P	M	H	R	N

Panpharmacon, Covishield, Cabtogravir, Remdesivir, Tazverik

Solve the Crossword

Win Exciting Gifts !!!



Across

1. I can decrease your heart rate & myocardial contraction but I am not suitable for asthmatic patient.

2. I work on your brain and relieve your pain. I make you crave

4. I don't like FOX. I'm given when ever there is imbalance between oxygen demand and supply

Down

3. Drugs with serious safety risk are put in this box

5. I am an orphan and I affect fat absorption

6.I cause lid retraction & drowsiness you use me to decrease the secretion

Terms and conditions

- Mind lab – II consists of **Three** segments, Solved answers to be mailed to fphpanpharmacon@gmail.com on or before **15-May-2021**
- Its mandatory to answer all the three segments to avail the prize
- Two Winners will be decided by drawing lot & Editorial board-Panpharmacon reserves all the rights
- Winners details will be announced in the upcoming issue
- Participation is restricted for Indian nationals only

Sponsored by The Himalaya Drug Company Bengaluru

Mind Lab - I Winners

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Assistant Professor
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Dayanand Sagar University

Deepanshi Garg
B.Pharm
Amity University Madhya Pradesh

Agnisha Sharma
Pharm.D
Faculty of Pharmacy
Ramaiah University of Applied Sciences

Recent Research Publications

- **G Nair,** G R Saraswathy, G N S Hema Sree., (2021) "Target mining and drug repurposing for hepatocellular carcinoma via bioinformatic and computational approaches", Annals of Oncology, 32 (1), pp s19

- Folitartha Roy, **Kesha Desai**, Parasuraman P, **Md Azamthulla.,** (2021) "A Brief Review on Novel Repurposing Candidates for the Treatment of Myocardial Infarction, International J of Engineering Applied Sciences and Technology, 5(10), pp 140-143.

- **Kesha Desai**, Austin Thomas, Shreya, Arushi, Pavan G and Lavanya., (2021) Preparation and Development of Poly-herbal Extract and its Evaluation for its Anti-obesity Property in Modified Diet Induce Obese Model in Rats, International J of Pharmaceutical Sciences Review and Research, 66(2),pp 30-35.

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Write your Feedback & Suggestions to

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