STUDENTS' NEWSLETTER | APRIL 2022

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Mélange UNITY IN DIVERSITY

GLOBAL DAY 2022 TEAMWORK







Zavia Evangeline Kitherian (2019bm36) Editor in Chief



Hey readers!!! How have you been?

I'm proud to present to you the 20th edition of our university newsletter. I'd like to thank you all for the constant support and encouragement we have received over the years. Our team has worked tirelessly despite their busy schedules to produce new and exciting content for all our readers and this edition is no different.

Join us as we delve deeper into the fascinating and engrossing world of prions, which are proteins which cause neurodegenerative diseases in a unique way.

As we all know, the fun summer season is quickly approaching. Apart from the dreaded heat and humidity, this season is perfect for trips to the beach and cooling pool parties. I love the feeling of having a nice cold ice cream on a hot and sunny day. Our friends have shared their thoughts on summer as well as their plans for this year's summer break.

Before I end, I'd like to share one of my favorite quotes. "You were born to be real, not to be perfect." Live in the moment and take your life one day at a time.

I hope you have an amazing day. See you next year!!!

Prions: The Molecular Zombies

By Dana M. Louai Al Akhras (2020bm06) and Danya Khan (2020bm21)

"HUMANS could catch Mad Cow disease from eating infected beef, the government will admit today"

This alarming newspaper headline, among many others, erupted in the late 1980's in Europe – particularly in England – sparking a wave of public fear and wary. A new, terribly terrifying and infectious pathogen has come to the picture: the prion.

In 1982, Dr. Stanley B. Prusiner was able to successfully isolate an infectious agent that was suspected to be the cause for Creutzfeldt-Jakob disease and other related neurodegenerative illnesses, and coined it as the prion. Dr. Prusiner later on received a Nobel Prize in Physiology or Medicine in 1997 for his discovery, which he had so sprucely addressed as "an entirely new genre of disease-causing agents".

Prions – short for proteinaceous infectious particles – are peculiar infectious pathogens, or proteins, that cause fatal neurodegenerative diseases by a very interesting and novel mechanism. They are responsible for the onset of a group of diseases known as prion diseases, or transmissible spongiform encephalopathies (TSEs). What is very peculiar about prions is that, unlike bacteria and viruses, they lack the presence of nucleic acids and are composed exclusively of a modified prion protein (PrP^{sc}); the normal, cellular protein (PrP^{c}) is converted to PrP^{sc} through a post translational process during which the new form acquires a high polypeptide sheets content.

Prion diseases can be brought about by three different ways: acquired, genetic, or sporadic. Acquired prion diseases are caused by a direct or indirect transmission of the prions between susceptible hosts through contaminated meat, via exchange of body fluids, or during medical procedures. A devastating incident involving an acquired prion infection occurred in Papua New Guinea, where the infection spread from a diseased person to the rest of the population due to a certain cannibalistic ritual practiced by the native people as a part of their funeral ceremonies. This endemic was later identified and reported as Kuru disease - the first prion disease - in the early 1950's. Bovine spongiform encephalopathy, commonly referred to as Mad Cow disease, also spreads from cattle to humans in a similar fashion.

Furthermore, genetically induced prion infections are a particular cause of concern since they are initiated by a mutation in the PRNP gene – the gene responsible for making the normal prion protein – and therefore it can be easily inherited by the next generation, making the prion disease familial in nature. Finally, sporadic



prion infections seem to be just that; randomly striking diseases without an identifiable cause. They mostly rely on spontaneous events that generate PrP^{Sc}, but still proceed in a similar manner to those diseases that are acquired or genetically induced. Observational studies of prion diseases have led to the understanding that transmission fluctuation and efficiency varies among different prions, primarily dependent on the strain, route of inoculation, and host genetics.

The molecular pathology of human prion diseases seems to be directly linked to their deleterious effects on the central nervous system. Since prions can cause a myriad of fatal neurodegenerative diseases, numerous interacting and/or independent cellular pathways – mainly affecting synapses, protein synthesis, and cell apoptosis – are compromised; ultimately leading to tissue damage. Prion diseases that affect humans include Creutzfeldt-Jakob disease (CJD) and variant Creutzfeldt-Jakob disease (vCJD), fatal familial or sporadic insomnia (FFI or sFI), Gertsmann-Sträussler-Scheinker syndrome (GSS), Kuru, and variably protease-sensitive prionopathy (VPSPr).

All prion diseases are caused by different versions of the same prion protein; since the PrP^c molecule can acquire a plethora of different deadly forms by abnormal folding. The PrP^c form of the prion protein is naturally found on the plasma membranes of nerve cells. However, an induced transformation of these proteins into their misfolded forms results in the formation of long fibrils which aggregate and hence can hinder the normal functioning of the brain, causing symptoms like rapidly developing dementia, confusion, and hallucinations.

The progress of any prion disease seems to follow a common course, where the main pathological condition that occurs is the progressive loss of neurons. Other characteristic diagnostic features of a prion infection include: a lack of classical inflammation, spongiform encephalopathy characterised by nervous tissue vacuolation, and deposition of abnormal conformers of prion protein forming amyloid plaques between nerve cells.

Although rare, prion diseases are incredibly fatal. This can be attributed to the peculiar ability of prions to induce abnormal folding in nearby PrP proteins, therefore contributing to the vast aggressiveness of the disease. The incubation period can take years as the prions spread "silently" within the brain, killing neurons in the process. Once enough damage is done, the patient begins to experience a series of symptoms that are associated with rapid cognitive decline, and eventually they succumb to the fatal disease within a few months.

Currently, the prospect of finding any treatment for prion diseases is far from reach. Nevertheless, there are some prominent efforts that have come up with astounding results; mainly proving the "protein only" hypothesis, which predicts that prions are capable of propagating individually without the intervention of nucleic acids. Such efforts include the efficient replication of prions in vitro by the PMCA in 2001, the production of infectious prions by de novo generation in 2007, and the creation of infectious material from recombinant PrP in vitro in 2010. These milestones mark the areas were research has successfully managed to make a dent in the challenging journey towards a better understanding of the nature of prions, which is laboriously relied on to pave the way for a promising a cure.

The Disease that Came About When People Ate People: Kuru

By Jaelyne Iona Tauro (2019bm12) and Nikita Sunil Binu (2021ph13) Editor: Diya Thakur (2021pt11)

Prion diseases are fatal neurodegenerative conditions that can arise sporadically, be hereditary or be acquired. One of these acquired prion diseases is kuru, a brain disorder that occurred at epidemic levels during the 1950s-60s in the highlands of Papua New Guinea. The term kuru comes from the Fore word to shake, "kuria", due to the body tremors that come with the disease. It is also known as the "laughing sickness" due to the pathologic bursts of laughter.

Kuru is largely localized to the people of the Fore tribe of Papua New Guinea and people with whom they intermarried. The first case appeared at Purosa in the 1930s. Researchers agree that Kuru first appeared in an individual due to a spontaneous change that created an infectious agent in the brain. The transmission of this agent is largely due to endocanabalism (eating of relatives), a core component of the South Fore's mourning ritual.

The Fore people believed that it would be better if their deceased relatives were consumed by family rather than becoming fodder for maggots. Primarily, adult women would cook and consume the dead body, especially the brain, as they were believed to be capable of hosting the dead body's spirit. This made women as well as children, more prone to contracting the infection in comparison to men, who preferentially consumed the muscles.

Through the consumption of deceased carriers of Kuru, the infectious agent got amplified within the community. This led to the Kuru epidemic of the 1950s-1960s.With bans on ritualistic cannibalism imposed in the 1950s, the incidence of the disease has decreased significantly, with the lingering of the disease in few due to a long incubation period. No deaths have occurred since 2010. Kuru belongs to a group of Human Transmissible Spongiform Encephalopathies (TSE) diseases, also known as prion diseases. A common characteristic of all TSE diseases was the presence of prions in the brain i.e. a group of infectious misshapen protein molecules. The prions would clump together in the brain tissue and is proven to change their shape as well as the shape of surrounding proteins of the same type.

Kuru has three clinical stages, which begin at the first onset of symptoms – ambulant (can still walk), sedentary (can only sit up) and terminal (unable to sit up by self). Once the first symptoms appear, life expectancy is an average of 12 months. Cerebellar ataxia and tremors are prominent signs. There may be a prodromal period characterized by headache and joint pain in the legs preceding the clinical stage.

At first, kuru begins with signs of titubation, tremors and an ataxic gait. The condition progresses with worsening tremors and ataxia. The infected individual exhibits signs of emotional instability and depression, yet bursts into sporadic laughter. At the end, the symptoms progress to include dysarthria, dysphagia, inability to respond to surroundings despite being conscious. The infected individual dies within two years due to pneumonia or other secondary infections.

In the CNS, neuropathological features are observed in the grey matter, such as spongiosis, neuronal loss and astrocytic microgliosis. Prion deposition is only observed in the CNS, and what differentiates kuru from sporadic CJD is the kuru plaques, which are spherical bodies with a rim of radiating filaments.

Currently, there is no known standard treatment and cure for Kuru, which is the same for other TSE diseases. The government's discouragement of cannibalism has mostly led to the decline of the disease.



Student Interviews

By: Sayesha Taneja (2020pcs18)

Yay! Finally summer is here. Summer vacation is that time when students can relax and enjoy their free time. It is a time to catch up on rest, spend time with family and friends, and explore new hobbies or interests. For many students, summer vacation is also a time to continue learning and improving their skills. We interviewed a few students about how they are going to spend their summer break. Let's have a look at them!



FAIZA TASNIM BADHON (2021bm40)

What's the best part of summer growing up?

The vacation is the best part of the whole summer. Even if I accept it or not, the summer is less problematic for me. Probably now we can't survive without AC but in the past, we didn't have to worry about getting sunstroke.

What tastes remind you of summer?

Probably, ice-cream or lemonade. Still I can't survive without having an ice cream everyday.

What are your plans for the upcoming summer break?

I have decided to go on a family trip with my family, visiting places in different cities. And also have a short trip to my home country because Bangladesh isn't as hot as UAE.



NUSRAT JAHAN PUSPITA (2020bm35)

What's the best part of summer growing up?

Growing up, I always looked forward to summer vacation as I could do anything without worrying about studies. We used to always have picnic, barbecue parties or go camping with family and friends. It used to be the most relaxing and fun time of the year. Sometimes we used to visit my home country,Bangladesh, meet my cousins, grandparents,relatives. We used to do all kinds of fun activities. Ah miss those days!!

Name a song that makes you think of a summer past and the reason.

There is no specific song that reminds me of summer. It's a season u can never explain with one song its a mixture of emotions and feelings.

What are your plans for the upcoming summer break?

I signed up for some electives this summer so I'm looking forward to that. Might try out something new that I have been thinking about for a while.

AKSHAY AHUJA (2020pcs05)

What are your plans for the upcoming summer break? I plan to go back to my home country, spend time with my family and join them to help people in need.

What's the best part of summer growing up?

The best part of summer is that I can polish my old skills and learn new ones. This helps me grow and that's why I look forward to summer breaks every year.

Name a song that makes you think of summer and why?

Summer of 69. It's an old classic English song that I heard during my summer vacation when I was 12 years old

MEENADARSHINI DHAMOTHIRAN (2021M038)



What are your plans for the upcoming summer break? I am planning to spend more time with my family and friends, reading books and meditating.

What's the best part of summer growing up? The best part of summer is to stay safe and stay hydrated and to plan out a proper diet.

Name a song that makes you think of summer and why? Summer Of Love by Shawn Mendes and Tainy.I always think about summer when I listen to this song because this song is about traveling and gives a summer vacation vibes

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Global Day - An Insider's Reflection

By Nazha Ayni First Year PharmD Student

This was my first time to attend Global Day at Gulf Medical University. I volunteered for decorating the tent, especially the photo booth for the Team India. It was a great experience for me and I got to know many people from different courses. Particularly, the Mr and Ms GMU competition was amazing. To be honest, I loved the general questions session where the participants answered brilliantly. I felt that was one of the good memories happened in my first year.

My suggestion for the next global day is to expand the timings and the space. Also, more volunteers for helping and cooperating for all the works to be done together perfectly is very important.

People On The Move *photo gallery by MSF*





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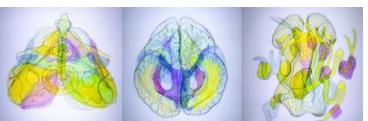
Mental Disorders as a cause of violence By: Layla Jameel (2019bm28)

Do you like extraordinary and unique ideas? Do you like art? There was an amazing Art Exhibition at the Gulf medical university hosted by Ms. Asmaa Elmongi. The art exhibition was conducted from 14- 18 March 2022, and the art title was " Mental Disorders as a cause of violence". The exhibition was organized by the communication department.

Ms. Asmaa Elmongi is an Egyptian visual artist and independent researcher who graduated from the Faculty of Fine Arts, Alexandria University, Egypt, She is an independent researcher who is practicing art through scientific research. Since graduation, Elmongi has participated in regional and international exhibitions and workshops and received multiple awards. She has publications in the Journal of Education and Social Sciences, Emergent Art Space website (a non-profit art foundation and an online platform for emerging artists), CIMAM website, and Research Gate.

In this Art exhibition, Asmaa Elmongi created impressive artworks using transparent paper, shading markers, colored ink pens, and backlight. The art exhibition contained 44 symbolic artworks that presented different mental disorders such as dementia, seizures, personality disorders, psychotic disorders, transmissible spongiform encephalopathies (TSEs), and much more. While childhood abuse and maltreatment, genetic inheritance, traumatic accidents, or brain infections are common reasons for mental disease.





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