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March 2022 | Issue #03

CATALYST

A Denovo: The Biotech Society Initiative

Cannibalizing Cancer Cells

Rethinking Treatment Strategies

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Ramaiah University OF APPLIED SCIENCES

De Novo THE BIOTECH SOCIETY

Issue 3
March 2022

From the Editor's Desk

On the 11th of March 2020, the World Health Organization declared the novel coronavirus outbreak as a pandemic, effectively shutting down global economies. Under the shadow of a looming pandemic, everything else took a backseat. What constantly plagued my mind, however, was how on earth I was going to take my grandmother to the hospital for her routine therapy. Her weakened immune system made her more vulnerable to the virus and this compounded my fear, leaving my mind fuzzy. I can only imagine what it did to the patients of such chronic illnesses themselves, who, more often than not, have to live with their debilitating conditions for life. Chronic illnesses do not have a cure. They are a silent pandemic that mostly evokes general sympathy from onlookers. While physical manifestations may differ, there is a universal undercurrent of anxiety and uncertainty.

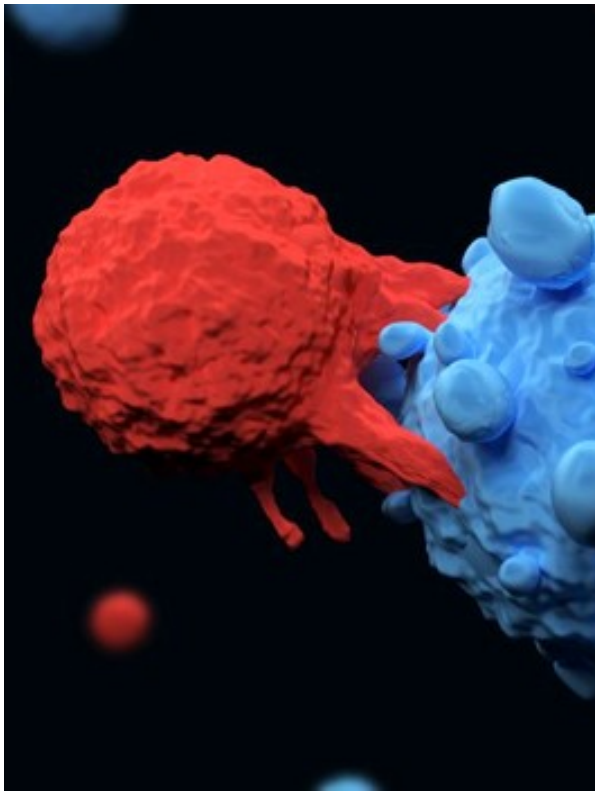
Cancer, diabetes, cardiovascular disease, hypertension and arthritis are the most prevalent chronic diseases that kill about 41 million people each year and require expensive treatment regiments, amounting to enormous financial and mental strain. Another hallmark of this silent pandemic is the increasing antibiotic resistance in patients. This drastically increases the cost of health care , augmenting the lethality of modern maladies. Other chronic illnesses like chronic fatigue syndrome, myalgic encephalomyelitis, fibromyalgia, Lyme disease — and now long-Covid-19 , whose diagnostics, treatments and severity are often unheeded , greatly reduce the quality of life.

Even prior to the pandemic, the healthcare system was inadequately poised to combat the rising cases of chronic illnesses. The white-knuckled grip of the virus on the healthcare system merely exposed its untenable cracks. The lockdown impeded the continuum of treatment for most patients, especially those belonging to vulnerable socio-economic backgrounds. Resource reallocations and breakdown of supply chains meant hospitals turned away most of these patients. Although some turned to telemedicine, many were left to fend for themselves which aggravated their situation even further. As we pass the two-year anniversary of the spiked virus, a scourge of new Covid induced complications and co-morbidities emerge. We are advised that this is the new normal, but we must not lose our sensitivity to the extremely high mental tax that these patients pay.

Therefore, in this issue, we strive to highlight some of the gaps and successes in this spectre. The bridge between research and medicine is weak and under-constructed. There is either little awareness or rampant misinformation regarding these illnesses. There is societal stigma, especially around mental health, which is inexplicably intertwined with these health scourges. However, strides are being made in cutting-edge medical technologies which are enabling patients with chronic diseases to live a life akin to normalcy.

I cannot conclude my piece without paying homage to the frontline workers and other health officials who continued to deliver health services, at the cost of their own physical and mental health in these trying times. And to each and every one of us, who rode out these daunting waves that have left us reeling with newfound fears, questions, and dare I say, hope.

-Sujanaa Sai
Editor-in-chief
B.Sc, 6th Semester



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CANNIBALISM IN CANCER

Tejal T,B.Sc, 6th Semester

Since the era of cancer chemotherapy began in the 1940s, cancer treatment and targeted therapy were revolutionized. With the advancements seen every day in the pharmaceutical industry, it comes as no surprise that cancer treatment has come a long way since those early days of nitrogen mustards and folic acid antagonist drugs. One such drug is Doxorubicin, an anthracycline.

The drug works by damaging the DNA inside the cancer cell in one of three ways: binding to the DNA by intercalation between base pairs on the double helix, preventing repair mechanism of DNA by inhibition of Topoisomerase II, and behaving as an iron chelator capable of forming iron-doxorubicin complexes that can bind both the cell's DNA and its membrane. However, even with these impressive attack strategies, a few clever cancer cells survive initial treatment, with Doxorubicin, resulting in a relapsed tumor. The cancer cells then transform into senescent cells- maintaining metabolic activity while also essentially being dormant. They are capable of releasing tumor promoters that encourage tumor growth.

A gene called TP53 is responsible for the production of tumor protein p53, also known as the "guardian of the genome". It is responsible for regulating the cell cycle, and thus acts as a tumor suppressor. A mutation in the TP53 gene is reported in 30% of breast cancers. These post-chemotherapy cancer cells were the subject of a recent study published in the Journal of Cell Biology. This team discovered breast cancer cells exposed to Doxorubicin or similar drugs transform into senescent cells, which then "cannibalize" neighboring cancer cells- irrespective of whether they were senescent or not. They used cells and tumors that expressed differently colored fluorescent proteins to observe interactions between cells in a treated tumor or culture. Confocal microscopy was then used to visualize a 3D representation of the image. It was beneficial in understanding how closely situated cells are oriented relevant to each other; under, on top of, or within one another, and time-lapse microscopy was used to generate a movie of these senescent cells cannibalizing other cells, providing definite proof of cannibalism in cancer

Another team of researchers discovered that the cells showing these cannibalistic tendencies activated a specific set of genes that is usually active in macrophages. RNA sequencing and Gene Set Enrichment Analysis were used to determine that tumors treated using chemotherapy, be it in-vitro or in-vivo, showed activation of macrophage genes that regulate engulfment and phagocytosis. The digested cells are processed using lysozymes and broken down, providing energy to the cannibalistic cell, which gains a survival advantage. Breast cancer cells are not the only ones this phenomenon has been observed in. Various other cancer types have the ability to activate macrophage genes and engulf neighboring cells. These cells survived significantly longer in culture than those cancer cells that did not cannibalize. The study also describes a lung cancer cell line and osteosarcoma (bone cancer) cell line that is also capable of phagocytosis after chemotherapy. Studies must be conducted to find if other cells have potentially cannibalistic inclinations, or if they risk falling victim to cannibalistic cancer cells.

Tumors, as we know, are not only made of cancer cells but also blood vessels, white blood cells, etc. Thus, there is no clear indication to what exactly the limitations to the type of cells that can be engulfed by post-chemotherapy cancer cells are. There is also no clarity on what advantages it lends to the cancer cell, and how this phenomenon contributes to cancer relapse, all of which need further study and experimentation. To avoid this "cannibalism" altogether, there is a need to formulate new therapies which can eliminate those cells that escape chemotherapy and become senescent. Further applications of this research can focus on discovering strategies to exploit the cannibalistic characteristics of these cancer cells.

Source

Sex Differences in Heart Disease: A Need for Targeted Therapy.

Shruthi, B.Sc, 6th Semester

A heart is a heart, or so we thought. It seems startling to think that there could be differences on a tissue level due to biological sex differences in human beings. Despite this, nearly all cardiovascular diseases exhibit sexual dimorphisms in occurrence, presentation, and outcomes.

Recent studies have shown that differences in sex chromosomes and the autosomes can lead to a surprising degree of variation in transcriptional and epigenomic factors between male and female hearts. Studies attempt to link these functional differences between the sexes to transcriptional bases, although, whether this link is causal, remains to be seen.

Genes that are commonly expressed in males and females may be targeted by factors unique to them. The sum of these regulatory biases leads to changes in the network structure which results in consequences for cellular responses to stress or disease. Hypothetically, this means that a particular disease in males and females results from different pathways and that therapeutic targets may be different too.

It has been observed that sex chromosomes can affect cardiovascular conditions like Aortic valve stenosis (AVS). Heart valves with XX or XY chromosomes differ in how they stiffen. Mostly, increased scarring, known as fibrosis, is observed in cases with XX chromosomes, whereas cases with XY chromosomes have an amplified build-up of calcium deposits.

It is partially true that sex hormones give rise to sex differences in valve tissue stiffening, a reduction in estrogen levels during menopause can aggravate heart fibrosis. But upon studying cardiovascular disease in XX and XY mice, scientists conclude that sex differences persist following the surgical excision of the reproductive organs producing the sex hormones.

To explain this phenomenon, we must understand X inactivation, cells that have XX chromosomes experience X-inactivation, where the X chromosome originating from one parent is turned off in some cells and the X chromosome inherited from another parent is turned off in other cells. X-inactivation occurs because people only need one X chromosome to be functional. Although around a third of the genes present on the inactivated X chromosome can escape inactivation, it is believed they are capable of disease regulation.

These differences in valve stiffening could possibly be explained by the action of genes that escape X-inactivation. To test this hypothesis, Scientist Brian Aguado and his team constructed Hydrogel scaffolds to recreate key aspects of the valve tissue microenvironment that could serve as a culture medium for sex-specific valvular interstitial cells (VICs) that are precursors to pro-fibrotic myofibroblasts. This system was used to cross-examine the intracellular pathways that are part of the activation and deactivation process of sex-dependent VIC-to-myofibroblast transformation.

The scientists found that the cells in the model successfully simulated the sex differences seen in valve tissue, that is valve cells with XX chromosomes exhibited more scarring than cells with XY chromosomes.

These findings confirm the need to develop optimized and personalised treatment strategies such as drugs that act on the specific target genes that escape X-inactivation and have a stronger effect on XX cells. Going forward, research must harness these cell sex differences to better understand cardiovascular disease and find inclusive solutions.

THE TELOMERASE CONNECTION: AGING & CANCER

Dr. Ekta Tripathi, Ph.D
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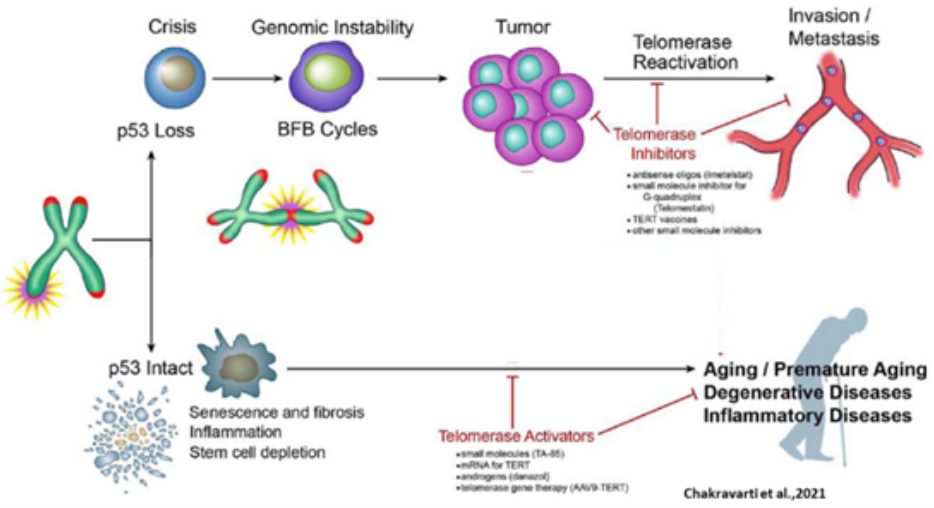


Figure 2: Telomere/telomerase in aging, cancer and potential therapy

In humans, telomere consists of repeat sequences and associated proteins (shelterin) at the chromosome ends which protect them against chromosomal fusion, recombination, and genomic instability. During the process of cell division, telomeres shorten as a result of the 'end-replication problem'. When telomeres become critically short (Hayflick limit), the cell activates the DNA damage response, which induces cell cycle arrest (replicative senescence) or underdgo apoptosis. Telomeres are therefore described as a 'biological clock' that determines how long a cell can contribute to cell population growth (Figure 1).

This is a tumour suppressive mechanism since it stops damaged cells from being propagated, however, in the absence of a functional checkpoint (p53), cells division continues with further shortening of telomere, leading to crisis. This is a stage of extensive genomic instability and cell death. Eventually, cells that acquire the telomere maintenance pathway either by activation of telomerase or ALT (alternative lengthening of telomeres) may survive, resulting in tumor formation. Thus, telomere biology is of great significance in our understanding of human aging and cancer development.

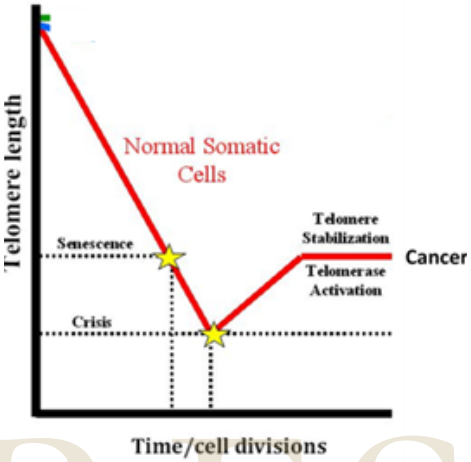


Figure 1: Telomere length and cell division

Beginning of the end story

The concept of telomere was given by Hermann Muller and Barbara McClintock in 1930s, when they found a unique structure at the chromosome ends in maize and Drosophila melanogaster and suggested that it was important to prevent chromosome fusion.

Later, in 1961, Hayflick and Moorhead demonstrated the presence of 'Hayflick limit' or replicative senescence in human cells and in the early 1970s, Olovnikov and Watson proposed the 'end replication problem'. In 1978, Blackburn and Gall sequenced the tandem repeats in Tetrahymena and later along with Greider, Blackburn uncovered a novel enzyme capable of extending telomere length, known as telomerase. These studies culminated in 2009 Nobel Prize in Physiology or Medicine to Elizabeth Blackburn, Carol Greider and Jack Szostak for their contribution in discovery of telomeres and telomerase

Telomerase Therpeutics

Shortened telomere length serve as a roadblock to cellular growth, but the loss of telomere coincides with both aging and cancer-inducing genome instability (Figure 2). The increased telomerase activity observed in most cancers has led to the development of antitelomerase therapies. Several approaches including antisense oligos, G-quadruplex stabilisers, vaccines and small molecule inhibitors have been developed to inhibit telomerase function, some of which are in clinical trials. Although telomerase is a promising target, but the time required to observe a clinical response after drug administration is quite long. Also, continued treatment leads to severe toxicity in patients. Therefore, alternative strategies to develop a telomerase inhibitor that kills tumor cells rapidly is a major challenge.In contrary to the potential applications of telomerase inhibition in anticancer therapy,

The link between telomere shortening and aging has catalyzed interest in telomeraserestoration therapy as a potential antiaging strategy. Such therapy rebuild or maintain telomere reserves by transiently inducing telomerase while avoiding its constitutive activation that can develop cancer. Telomerase activation has been achieved through natural molecules, synthetic molecules and genetic manipulation. TA-65 (cyclastragenol), a plant based compound that activate telomerase are provided as nutritional supplements (TA Sciences, Geron Corp). In addition, hormonal agents such as danazol and 5α dihydrotestosterone, which can increase telomerase levels, are being tested in patients with telomeropathies. Treatment of premature aging diseases such as Werner and Bloom syndromes would be an interesting application.

Future Perspectives

The telomere field exhibits an ideal system of convergence of basic and multidisciplinary science for our understanding of aging and the pathogenesis of cancer. In-depth understanding of many aspects such as genetic and epigenetic mechanisms involved in the regulation of TERT, non-canonical functions of telomerase and interplay between telomere dysfunction and human diseases is still lacking. Although not very clear whether telomere dysfunction initiates the disease or is only a participant, telomeres definitely play an integral role in human diseases. This elemental role encourages the development of efficient telomerase inhibitors as well as activators for the treatment of cancers and aging and age-associated diseases.

Self Portrait with bandaged ear by Vincent van Gogh

Understanding Schizophrenia: A Necessary Vignette

Ajay, B.Sc, 4th semester

Schizophrenia is derived from the root words σχίζειν(pronounced as “schizein”; meaning-” to split") and φρήν, φρεν- (pronounced phrēn, phren- respectively; meaning-"mind"). Contrary to the portrayal by media, schizophrenia does not involve a “split personality.” Rather it is a chronic psychotic disorder that disrupts the patient’s thoughts and affected patients experience deteriorating social relationships who find themselves unable to form meaningful new relationships as the condition acts as a deterrent in sustaining these relationships. With approximately 0.32% of people being affected worldwide, with a rate of 1 in 222 among adults, it is a rarity even among other mental disorders.

The disorder was first identified by Dr. Emile Kraepelin in 1887. He identified manic depression and dementia praecox, later termed schizophrenia, as distinct forms of psychosis. This differentiation was done on the consideration that manic depression was an episodic or periodic disorder that is not neurodegenerative, whereas schizophrenia resulted in permanent cognitive impairment.

Our poor understanding of how the brain works (we understand 10% of how it functions) acts as a hindrance to our understanding of the disorder. The inherent heterogeneity of schizophrenia has resulted in a lack of consensus regarding the disorder’s diagnostic criteria, etiology, and pathophysiology, but with the current advancements in science, we attribute its various phenotypes to multiple factors including genetic susceptibility and environmental influences.

Schizophrenia has a high heritability rate of 80%, but the genes responsible have remained elusive, until recently. Several papers in the 2000s described seven genes that may warrant the title “schizophrenia genes”. Apart from the hereditability of the disorder, most theories with regard to abnormalities in neurotransmission are centered around an excess or a deficiency of neurotransmitters, including dopamine, serotonin, and glutamate which are attributed as inducers of the disorder. In patients suffering from this disorder, the brain tissue itself appears to undergo detectable physical changes.

In individuals that are vulnerable to the disorder, environmental and social stressors like childhood trauma and discrimination respectively may play a role in the development of schizophrenia.

Individuals affected by the disorder may exhibit two types of symptoms, positive and negative. Positive symptoms are highly exaggerated ideas, perceptions of actions that show that the person is unable to distinguish between reality and imaginary like hallucinations and delusions. Negative symptoms on the other hand refer to an absence of normal mental function involving thinking, behavior, and perception. A schizophrenic patient may also exhibit cognitive symptoms, such as the inability to process thoughts and form decisions. But, it should be noted that some of the negative symptoms are common in depression as well. Effective diagnosis is a crucial factor in the treatment of either case.

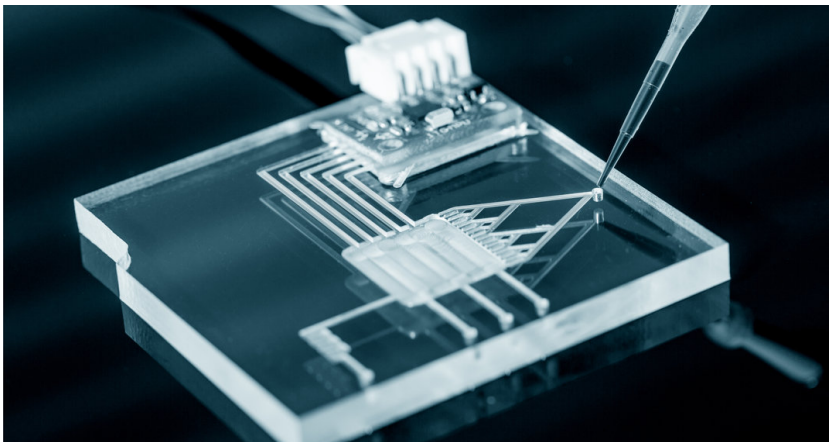
With regard to treatment, both nonpharmacological and pharmacological treatments must be used to optimize long-term outcomes. Psychotherapeutic approaches may be divided into three categories: individual, group, and cognitive-behavioral. Due to the nature of the disease, nonpharmacological treatments are incorporated in the treatment regime, and not merely as a substitute.

Sometimes paranoia and delusion may induce the patients to refuse medication, also called non-adherence hence, it is important to include individuals close to said patient in the treatment process, to speed up and smoothen the affected individuals' reintegration into society. SGAs are the pharmacological agents in the first-line treatment of schizophrenia, as they're associated with fewer extra-pyramidal effects. However, SGAs have metabolic side effects, such as weight gain and diabetes mellitus.

Although there is no cure for schizophrenia, as long as its symptoms can be suppressed through drugs in initial and maintenance therapy, the disorder can be kept in check. As we venture deeper and deeper into the science behind the functioning of the brain, we can hope for a final solution to such disorders in the future.

LAB ON A CHIP

Bhoomika Shetty, B.Sc, 6th Semester



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If it's taking too long to obtain samples from the lab, why not bring the lab to the samples? That was the thought of researchers at Stanford University, who recently developed what they call "a lab on a chip" based on CRISPR enzyme Cas12. About half the dimensions of a MasterCard, it contains a sophisticated network of channels smaller than the width of a person's hair and may deliver a coronavirus test's results in under half-hour.

Researchers say that the test might be modified to detect other infections, too, by recalibrating the CRISPR enzyme for a special gene. As the Covid-19 pandemic taught the planet, testing is the primary initiative in combating communicable diseases. With a lab on a chip, that testing is often done quickly, safely, cheaply, and more efficiently.

Lab-on-a-chip (LOC) technology was developed due to advances within the field of nanotechnology. LOC is engrossed with laboratory experiments administered on a really small scale. It can integrate several laboratory functions on a chip of size starting from a couple of millimeters to a couple of square centimeters. This helps achieve high-throughput screening and automation.

However, LOC is an emerging technology and features a few disadvantages. The physical and chemical effects like surface roughness, capillary forces, and chemical interactions between materials are more significant at the microscale level. This may often end in complications during LOC experiments.

Most of the research on LOC technology thus far has focused on its applications within the field of diagnostics like its use in diagnostic devices in medical offices or at sites that have limited or no access to laboratory facilities. Several applications in bioscience and medicine have also been explored thus far including potential use in protein crystallization studies and DNA or RNA sequencing.

The medical community, spanning decades of years , with over millions of people, has its fair share of scandals. Often , these are life threatening and involve heavy legal procedures where there is little respite to the victims.

Here are three of some of the most sensational scandals that shook the medical community.

Heparin adulteration

The US Food and Drug Administration announced a recall of heparin, which is a drug to prevent blood clots that were manufactured in China by Scientific Protein Laboratories. It was discovered that batches of the raw drug had been contaminated by an over-sulfated derivative of chondroitin sulfate and that the contamination had been deliberate due to its nature of imitating the effect of heparin. There were allegedly 81 deaths and over 700 other reports of severe injuries linked to the drug in question.

Mass Poisoning via Elixir Sulfanilamide

In 1937, several cases of poisoning and even fatalities due to the poisoning numbering at over 100 had turned up after an antibiotic called sulfanilamide was dissolved in diethylene glycol and was marketed as Elixir Sulfanilamide. Regardless of the reports indicating that DEG was harmful to humans , the final nail in the coffin came when the chief pharmacist at drugs manufacturer S. E. Massengill Company was oblivious to these reports.

The PIP Silicone Implant Scandal

Poly Implant Prothèse (PIP) a silicone implant company was found guilty in a gigantic scandal when French surgeons in 2009 began to report a large number of implants that had ruptured. The resulting investigation found that in 2001 PIP had begun using unauthorized, industrial-grade silicone instead of the medical-grade silicone that was supposed to be used which reduced their expense of manufacturing by a massive 90% with the only downside of the increased chance of rupturing which could result in a variety of problems such as inflammation, irritation and even the escape of toxic substances into the body. This crisis left thousands of women fearful and even drove the company to its fateful bankruptcy.

Aldo Emmanuel Pinto,
B.Sc , 4th Semester

A New Era.

Strategies

in Monogenic Disorder Therapy

Neha Rao,B.Sc, 6th Semester

There are a multitude of mechanisms by which our genome directly or indirectly contributes to disease or disruption of homeostasis. One such genetic disorder is monogenic disorder. Monogenic disorders are caused by variation in a single gene and are recognized by their familial inheritance patterns. Examples include sickle cell anemia, cystic fibrosis, Huntington disease, and Duchenne muscular dystrophy. Monogenic disorders have been designated the most straightforward and have been targeted in trials of directed gene therapy. Successes have been few, however sizable, such as remedies for retinal dystrophy, adenosine deaminase deficiency, and spinal muscular atrophy.

Monogenic disorders are caused predominantly by a lesion of a single gene, although the phenotypic manifestation may depend to various extents on additional genetic variants in the same or other genes, epigenetic changes, and environmental factors. Although most individual monogenic diseases are rare, together, they represent a high disease load in the population.

A new era of genomic medicine is on the horizon with the potential to treat a myriad of rare monogenic diseases with nucleic acid therapies. As a possible treatment for different monogenic issues, exogenously delivered mRNA is appealing because of its capacity to encode any sort of remedial protein, including cytosolic, intramitochondrial, transmembrane, and secreted proteins. By using the cell's endogenous translational machinery, mRNA allows for the natural production of such therapeutic proteins with the proper post-translational modification. Gene therapy and gene editing are immensely attractive due to their "one and done" approach, that is, administering a solitary portion of treatment that will be viable for a lifetime. Like mRNA, gene therapy and gene editing can be utilized to deliver or alter any sort of protein. Different clinical trials at various stages are as of now in progress for different uncommon monogenic problems, possibly yielding a flood of additional gene therapy/altering drug endorsements following

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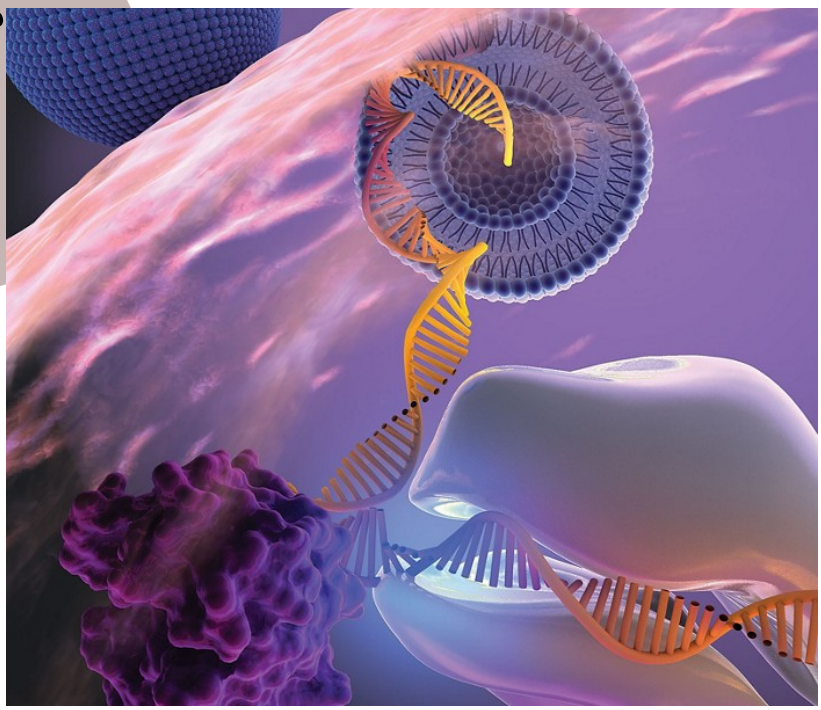

the positive outcome of voretigene neparvovec, a gene therapy to treat inherited blindness. Strategies have been tried for genetic disorders characterized by deficiency of a necessary protein. Protein replacement therapy is currently available for several disorders, but is deterred by immunogenicity of the substitution protein, as well as difficulties in conveyance of the protein. Endeavors to mask the substitution proteins from the immune system by pegylation have been met with poor success.

Organ transplant has been implemented with inconsistent results in certain diseases. Bone marrow transplant has furnished clinical advantage in patients with thalassemia. Nonetheless, the chance of graft-vs-host disease presents consequential outcomes, and restricted accessibility and availability of donors as well as the technological demands remains a challenge.

However, seemingly, the best technique is to eliminate a dysfunctional gene and replace it with a normal one, or to add a missing gene to the genome. While this may not generally forestall the production of protein-neutralizing antibodies, successful gene replacement may only need to be done once, and may not require ongoing medication.

The ethical and pragmatic technical concerns and difficulties for this approach have so far been sizable and restricting. There are moral reservations in regards to the chance of influencing germline DNA, with obscure results to offspring. There have been complications associated with viral gene delivery vectors and targeted delivery.

The therapeutic possibilities are striking. While further studies are needed to demonstrate the translatability into the clinic, mRNA therapy seems poised to be part of the next generation of promising therapeutics for the treatment of monogenic disorders.

Frederick Sanger, 'the father of genomics', was one of just four scientists to win two Nobel prizes and the only one to receive both in chemistry. He was born in Rendcomb, United Kingdom, in 1918. From an early age, he developed an interest in science from his father who was a physician. He received his bachelor's degree in biochemistry from St John's College, Cambridge, in 1939.

During WWII, he stayed at the University of Cambridge to research the nutritional value of lysine in potatoes with scientist Albert Neuberger. In 1958, he won his first Nobel Prize for discovering how amino acids are strung together in the protein insulin. Many people in the 1950's assumed the amino acids in a protein were arranged randomly, but Sanger proved that they form a unique sequence. He used a yellow dye to mark the ends of the different chains, then hydrolysed them to amino acids and recognised the marked amino acid in each case. He split the amino acid chain into defined pieces by using acids and enzymes, then tagged purified fragments with the dye and repeated the process and from that he was able to deduce the order of amino acids in insulin protein.

Sanger demonstrated his method by sequencing genomes of progressively larger sizes, first with a basic bacterial virus (5,386 nucleotides) in 1977, then the DNA in human mitochondria (16,569 nucleotides) in 1981, and finally the genome of bacteriophage lambda (48,502 nucleotides) in 1982.

Sanger was at his happiest at the laboratory bench, where he toiled diligently and single-mindedly. He solved incredibly complex problems with by using simple apparatus. As a result, he motivated younger scientists and drew some of the world's greatest biologists to Cambridge.

- Anjali Manoharan, B.Sc, 6th Semester

Frederick Sanger

13 August 1918-19 November 2013

"Scientific research is one of the most exciting and rewarding of occupations."

Airborne DNA from plants

Aryan Pradhan, B.Sc, 6th Semester

eDNA trapping could be the next big thing in the study of the plant community.

Mark Johnson, a graduate student at Texas Tech University, revealed as part of his work in the study of environmental DNA (eDNA) that trapping airborne DNA released by plants could be the effective replacement of countless efforts of collecting plant DNA. This will also be instrumental in understanding the effects of climate change on plant species. Just like aquatic DNA enjoys the center stage, eDNA might hint about invading species well in advance.

Studying plant traces is not new in the study of biodiversity. We all are familiar with how plants spread their life traces called pollen to reproduce, and pollen surveys have been conducted to study plant species for quite some time now.

However pollen surveys have certain boundaries, they can only identify pollen that is spread via wind. Other modes of pollination including animals, pollinating insects are not covered in the scope of pollen surveys. Johnson and his team took this limitation as a challenge and devised a method to capture DNA-bearing traces of plants that are not even pollinating or flowering.

Johnson and the team collected plant eDNA in dust traps from all the grass species in 27 100-meter transects every week for one year. They also took samples of the same transects using the traditional method. Surprisingly the eDNA technique was able to detect 91 distinctive species while the traditional methods only detected 80 species. The eDNA technique also revealed 13 new grass species which its traditional counterpart overlooked.

The airborne technique would be extremely helpful in detecting invasive plant species before they could unfold their evils. Since air trapping is a relatively newer technique, you might see a few modifications in the future. For example, since these traps depend on the flow of air to trap eDNA from host particles, in the future we might see filters with air fans to accommodate more particles.

Given the success story of aquatic eDNA, the future of air eDNA looks promising.

5

MYTHBUSTERS REGARDING HEALTH

PALLAVI, B.SC, 4TH SEMESTER

On a daily basis, we come across many misnomers, misconceptions, and superstitious social beliefs that mislead us about the reality and the sciences involved in common natural phenomena. And when it comes to our society, we hear and go through many non - scientific myths about health that are imposed upon us due to many reasons. Rampant misinformation on social media serves to cause confusion even further. However, not to worry, we have compiled the top five myths about health and laid down the truth behind each of them, all backed by science and research!!

1.SKIPPING BREAKFAST WILL MAKE YOU GAIN WEIGHT

Though, it's the most important meal of the day many tend to skip it. But skipping it to gain weight so that you overeat the other two meals of the day, was proven to be a myth, by a study published in the Journal of Nutrition Science in 2014.

2.READING IN DIM LIGHT WILL AFFECT YOUR EYESIGHT

While we read our books, newspapers, novels, etc at the end of the day, we unintentionally tend to read in dim-lights. According to Harvard Medical School, it does not damage your eyesight in any way. It just hurts your eyes as you are tiring and stressing them out, compared to reading them normally.

3.YOU DO NOT HAVE TO WORRY ABOUT HEART DISEASE IF YOU ARE A WOMAN

Although we have come across many heart diseases that usually tend to occur in men and not in women, the latest data from the Centre for Disease Control and Prevention proved otherwise. It was revealed that heart diseases in women are actually the major cause of their death. And that irrespective of gender, heart diseases can occur in both men and women depending on external factors like their lifestyle and their food intake.

4.DEODORANT CAUSES BREAST CANCER

There have been plenty of conflicts about the link between breast cancer and the use of deodorants or antiperspirants as they contain potentially harmful ingredients and are applied right near the breasts. Studies at the National Cancer Institute have not been able to prove this point as they do not possess any validating information or evidence as to the connection between the usage of deodorants and them causing something major that is, breast cancer in humans.

5.GREEN MUCUS IS AN INDICATION THAT YOU ARE SICK

It's natural for us to assume that we need to visit a doctor every time we catch a common cold along with green mucus, like the onset of a major viral infection or something serious. Yet again, the studies done at the Harvard Medical School gave us a confirmation that we cannot really rely upon the color or consistency of our mucus for suspecting an infection. In fact, the green mucus reveals nothing more than the fact that our body is perfectly doing its job by protecting itself.