



THE WILSON'S INTRIGUE

STEM Issue 5: April 2021



BIO-CHEM

Cytokine Storms

ENGINEERING

The Flying - V

MATHS

Prime Tuples

PHYSICS

Muon Magic



Introduction

With a new editorial team taking over, we have brought some fresh ideas with us. We really hope you enjoy the new font, the new Maths section, the book reviews and the “Intrigued?” page in addition to the articles, which span from superhero science to quantum tunnelling.

The team of writers and editors is very proud to welcome you to the fifth issue of the Wilson’s Intrigue STEM, written for students by students.

Our Mission

- Expand your knowledge
- Contribute to the Wilson’s community
- Make complicated parts of science more accessible
- Popularise science and make it more interesting
- Inspire creativity through wider research

Acknowledgements

The magazine simply could not be produced with such finesse without the inquiring journalism of the writers and the multiple zoom calls the new editors have attended to learn and master formatting in Publisher. A massive thank you to all students involved for their contributions!

A special thanks must go to Mr Benn, Mr Carew-Robinson, Dr Cooper, Mr Jackson, Mr Lissimore, Miss Roberts and Dr Whiting for proofreading and verifying the accuracy of our articles and the magazine as a whole.

If you would like to write in the sixth issue of the STEM magazine to research and discover a new aspect to the subjects, please email me (Divy) at DAYALD@wilsonsschool.sutton.sch.uk for more information.

Founded by Devanandh Murugesan and his team of editors in September 2019

Front Cover: This majestic deep sea creature is known as a siphonophore. It is estimated that there are 175 different species of this class, and although it appears to be one large organism, it is in fact a colonial organism composed of two specialised zooids.

The Wilson's Intrigue Team

Editors

Divy Dayal (Chief Editor) Y12

Nabeel Abdul Rasheed Y12

Aditya Chougule Y12

Aditya Jain Y12

Atharva Narkhede Y12

Mann Patira Y12

Writers

Adam Ali Y12

Arya Narang Y11

Dulain Gamage Y12

Folaju George Y11

Jonathan Peter-Rajan
Y11

Junaid Ali Y12

Junayd Soobratty Y12

Kinshuk Jain Y12

Karun Kirubananthan
Y12

Matteo Cascini Y11

Mohamed Ahmed
Y12

Moksh Sachdeva Y12

Prabhas Vedagiri Y12

Sanuka

Gunawardena Y12

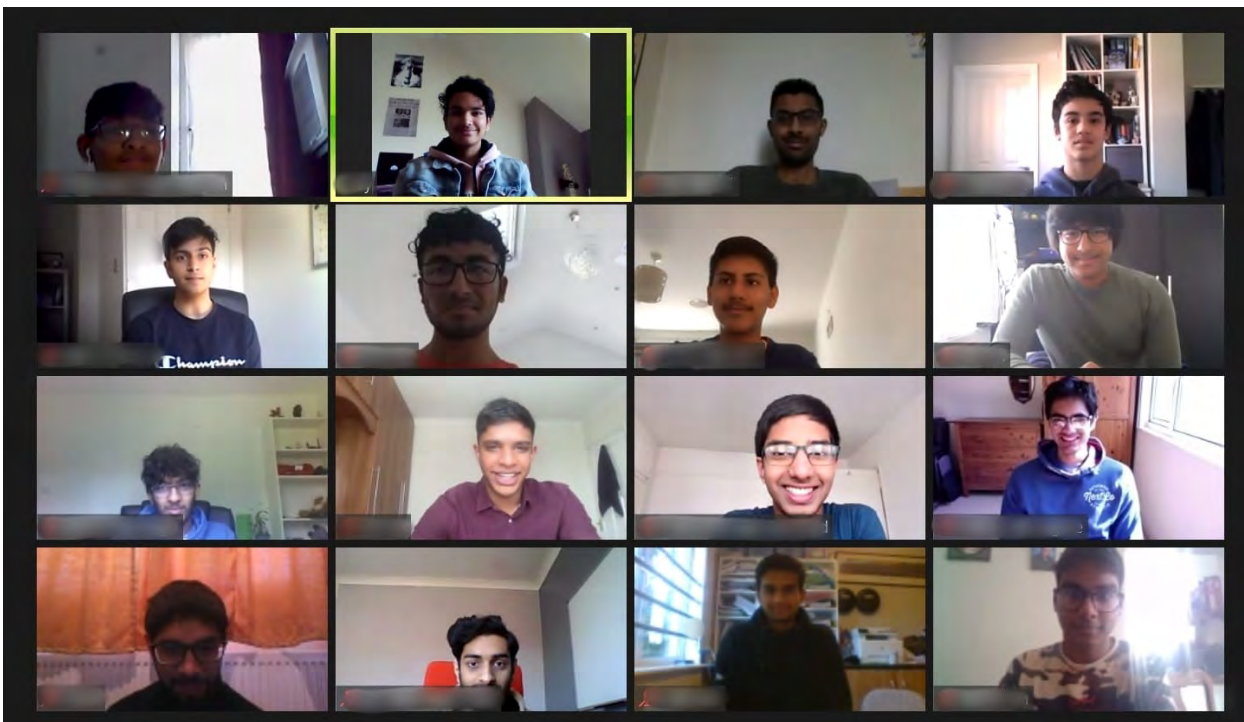
Shinujan

Saravanamuthu Y12

Syed Shah Y12

Tathushan

Subenthiran Y12



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DID YOU KNOW?

Dragonflies were one of the first flying winged insects (almost 320 million years ago). Today the biggest dragonflies have a wingspan of 14 cm, however, fossil records suggest ancient dragonflies had wingspans of up to two feet!

Bio-Chemistry Section

Inside the Cytokine Storm

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One vial (0.45ml) contains 5 doses of 30 micrograms of BNT162b2 RNA

entire complex in place while a peptide bond forms between the two amino acids through a condensation reaction. The ribosome then moves along the mRNA molecule, repeating this process until a stop codon (UAA, UAG or UGA) is reached. At this point, the polypeptide chain stops growing and detaches from the ribosome^[5].

The polypeptide later folds into its precise three-dimensional shape, specifically an antigen of the infectious agent that the vaccine is tailored towards. In the case of Covid-19, this antigen comes in the form of the crown-like spike protein which gives the coronavirus its name. As is typical during a primary immune response, the antigen is displayed on the surfaces of specific cells known as antigen-presenting cells or APCs (which include macrophages, dendritic cells and B cells). Here, it acts as a signal to trigger an immune response which eventually leads to immunological memory (through the production of B memory cells and T memory cells). In effect, the immune system can now respond much more quickly and on a larger scale if the actual pathogen were to enter the body^[1,6].

Benefits and Drawbacks

As you might imagine, a novel technology like the RNA vaccine comes with more than its fair share of risks and rewards. Chief among the advantages are:

Safety: Despite the government's best efforts, the issue of vaccine safety is one of continued public concern, as illustrated by the ever-growing anti-vaccine campaign. In the case of the RNA vaccine, there is no chance of infection because it is entirely free of pathogen particles. Once the antigen is 'memorised' by B and T memory cells, the mRNA is degraded by intracellular ribosome-degrading enzymes – a process likened quite aptly by

RNA Vaccines: The Copycats

How RNA vaccines are the key to future pandemics

By Nabeel Abdul Rasheed (Y12)

Many a GCSE biologist is well-acquainted with the traditional vaccine – memorised religiously and reproduced on exam paper as 'a dead or weakened form of a disease-causing pathogen'. But more recently, indeed in the last few weeks, scientific discussion has shifted away from these classical live-attenuated or inactivated versions and instead towards a newer, altogether more exciting prospect: the RNA vaccine.

Details & Mechanism

As is the case in all other types, the purpose of the RNA vaccine is to train your immune system to respond rapidly to a specific infection by mimicking the infectious agent. What distinguishes it from more established counterparts is the mechanism by which this agent is introduced into the body: rather than injecting the pathogen itself, the RNA vaccine comprises a messenger RNA (mRNA) strand that codes for a disease-specific antigen^[1]. This sequence is

enclosed within a special coating to protect the mRNA from extracellular RNA-degrading enzymes in the body (which detect and eliminate aberrant – meaning atypical or deviant – mRNAs as part of cellular homeostasis)^[2,3]. Once the mRNA reaches the target cells, it is thought to settle into small cup-like depressions in the plasma membrane known as caveolae (Latin for 'little caves'), before entering the cell through caveolar endocytosis in membrane-bound sacs called vesicles^[4].

Having entered the cell, the mRNA can then bind to a ribosome floating in the cytoplasm or one attached to the rough endoplasmic reticulum, ready for the familiar process of protein translation. Two transfer RNA (tRNA) molecules, each carrying a specific amino acid, align themselves next to mRNA codons which are complementary to their own anticodons – the start codon being AUG, which codes for the amino acid methionine. The ribosome holds the



ScienceNews to “a *Mission Impossible* message that self-destructs once it has been played” [2,7].

Efficacy: The most striking example of an effective RNA vaccine is undoubtedly the most recent one of them all. BNT162b2, as it is fondly named, was developed by biotechnology giants Pfizer and BioNTech and consists of a modified mRNA encoding a mutated form of the SARS-CoV-2 spike protein. Large-scale clinical trials involving more than 43,000 participants demonstrated a vaccine efficacy rate of 95%, measured from seven days after the second of two doses [8]. This was consistent across most demographics, so much so that in early December 2020, the UK became the first country in the world to approve the coronavirus vaccine – ordering over 800,000 within the first few days [9]. Up to now, the largely successful results of this project serves as testament to the potential of the RNA vaccine. Even so, in the context of vaccine trials, a sample size of 43,000 may not represent all demographics to the degree of validity associated with other, non-emergency use vaccines – thus paving the way for more extensive research in the near future.

Production: RNA vaccines have the added advantage of being produced much more rapidly and easily in standardised laboratory procedures than traditional vaccines. In fact, the German biopharmaceutical company CureVac (which specialises in mRNA technology) believes that it would only take two months to produce an RNA vaccine for influenza. Perhaps more importantly, the drastically lowered production period would enable swift rollout of vaccines during pandemics, once again illustrated by the invaluable efforts of Pfizer and BioNTech [1,2].

Equally, however, the problems associated with any medical treatment cannot simply be ignored. Just as there have been major reasons to celebrate, we are also faced with numerous challenges when it comes to RNA vaccines:

Side-effects: While the RNA vaccine may be free of pathogenic material, it can on rare occasion induce adverse effects on users, the most recent of which involved two NHS workers suffering allergic reactions to the Pfizer-BioNTech vaccine just one week after it was approved for use in the UK. Both suffered an anaphylactoid reaction (not to be confused with immune-mediated and life-threatening anaphylaxis), but were later treated and soon restored to full health. Some allergists and immunologists believe this type of reaction to BNT162b2 could be linked to the compound polyethylene glycol (PEG) contained in lipid nanoparticles used to package the mRNA [10]. They believe that a small number of people previously exposed to PEG may have high levels of antibodies against it, putting them at risk – however small – of an anaphylactic reaction to the vaccine [11]. Nevertheless, this theory is supported by limited evidence as of yet, and the general consensus is that initial side-effects are “common with new vaccines” (the words of Professor Stephen Powis, medical director for the NHS in England) [10].

Delivery: As mentioned previously, the RNA-degrading enzymes in the body make delivery of the vaccine to body cells quite challenging. The most common drug delivery system used to combat this are lipid nanoparticles, tiny spherical molecules which can carry lipid-soluble drugs in their cell membrane bilayer. They contain protruding surface proteins known as homing peptides which allow for specific, targeted drug delivery. What’s more, lipid nanoparticles ensure high encapsulation efficiency of mRNA, ease of transport and reduced risk to body cells and immune system compared to alternative methods [12].

Storage: Many RNA vaccines need to be frozen or refrigerated, owing to the instability of the RNA molecule itself and the temperature-sensitive nature of the lipid nanoparticles. The Pfizer-BioNTech vaccine must be stored at -70°C, almost four times colder than the average household freezer [7]! Such demanding requirements may be difficult to reach for countries with limited or no refrigeration facilities; even for those privileged with this infrastructure, the cost of procurement and transportation acts as yet another barrier to large-scale vaccine distribution [2].

The Future

Unsurprisingly, a strong focus of current RNA vaccine research is aimed at developing and improving the current mRNA sequences used in the COVID-19 vaccines – particularly minimising side-effects and making the molecule more suitable to everyday conditions. However, this looks likely to be overshadowed by the even more daunting prospect of new, emerging variants of the virus – growing areas of concern for governments and biotechnological companies alike.

Looking further ahead, RNA vaccines may well hold the key to further advances in multi-purpose treatment, providing protection for more than one disease at a time (as has already been achieved in conventional vaccines for a select few diseases – most notably the MMR vaccine which protects against measles, mumps and rubella) [3]. Research and clinical trials are also being carried out on RNA vaccines that could be used to encode cancer antigens and stimulate immune responses against malignant tumours [3]. This may even become a form of personalised treatment by tailoring the mRNA to the unique properties of the tumour cells of that particular individual [1].

Whatever the future holds, it seems increasingly likely that RNA technology will play a major part in it, making it of greater interest to us than ever before. Our role both as students and members of the public is to continue educating ourselves of the advances and the shortfalls encountered along the meanders of its development. Only then can we hope to leave behind a society rife with uncertainty and scepticism into one which offers hope and remedy for all.

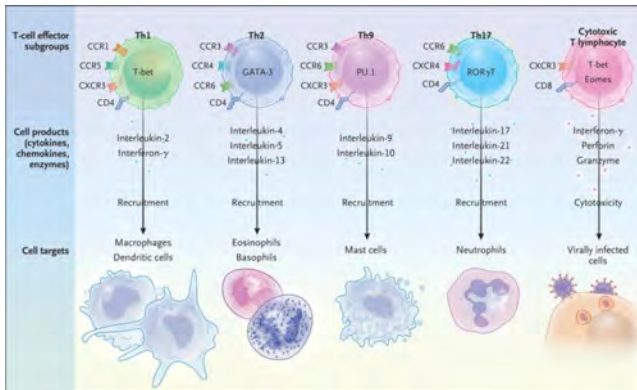
Edited by Aditya Chougule



Inside the Cytokine Storm

By Aditya Chougule (Y12)

Our immune system is both a blessing and a curse. Its intricacies allow for a phenomenal structured defence against invaders but these intricacies also mean a great deal more places for mistakes to be made. As such many diseases arise from malfunctioning of the immune system, the very organ that swore to protect us. One poignant example of this is the cytokine storm. Particularly relevant at the moment, the cytokine storm is a terrifying condition whereby an overabundance of cytokines can cause hyperinflammation and can lead to multi-system organ failure and eventually death.

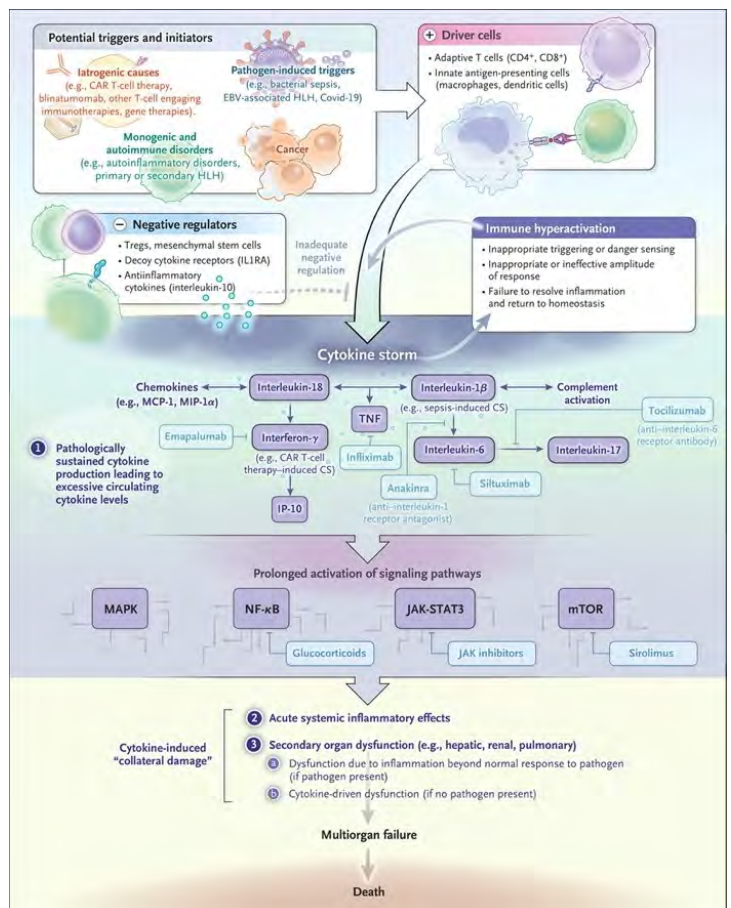


Cytokine distribution ^[1]

Key to the disease process of cytokine storms is (you guessed it) the role of cytokines; these tiny proteins are tremendously important in coordinating the immune response since they act as regulatory signals that activate effector cells and also help to resolve the immune response. There are a few cytokines that have a higher significance over others in the context of a cytokine storm- specifically interferon- γ , interleukin-1, interleukin-6, and TNF- and as such they are usually found in elevated levels in the midst of a cytokine storm. Interferon- γ is primarily secreted by activated T cells (Th1 and cytotoxic T lymphocytes) and natural killer cells and is a potent activator of macrophages, which engulf pathogens and cells by phagocytosis ^[1]. The interleukin-1 cytokines are a family of 11 cytokines that have a wide range of biological functions, including acting as a leukocytic pyrogen (mediator of fever) and activating macrophages and Th17 cells, the latter acting as helper cells that coordinate the immune response ^[2]. Interleukin-6 is one of the more complex cytokines since it is produced by and acts on immune and nonimmune cells across multiple organ systems- it is an important mediator of the acute inflammatory response and in increasing antibody production. And last but not least, TNF (tumour necrosis factor) is a proinflammatory cytokine with a vast array of functions; although its primary role is to induce fever, augment inflammation, and activate antimicrobial responses through interleukin-1 and interleukin-6 producing cells, TNF can also induce

cellular apoptosis- programmed cell death- and inhibit carcinogenesis and viral replication ^[3]. Essentially, all these cytokines play a wide variety of roles and communicate with a multitude of organ systems, making their betrayal even more frightening.

Those reading this will likely have heard of cytokine storm from the news in relation to COVID-19 but in fact there are quite a few triggers for cytokine storm besides a viral pathogen. Indeed, there are three types of cytokine storm: iatrogenic, pathogen-induced and monogenic. Pathogen-induced cytokine storm probably sounds more familiar than the other two since it is the most common cause for the condition; it results from naturally occurring microbial infections and viral invaders. In sepsis-associated cytokine storm (sepsis being a life-threatening reaction to an infection), very often the collateral damage caused by the immune response



as it attempts to clear the pathogen can be more deadly than the pathogen itself, which is why sepsis has such a high mortality rate^[4]. Iatrogenic and monogenic cytokine storms are quite different but equally as terrible. An iatrogenic trigger is that caused by medical treatment, for example, infusion of CAR T cells (T cells which have been genetically engineered to target cancer cells) has been shown to induce cytokine storm with excess levels of interferon- γ and interleukin-6^[1]. Additional iatrogenic causes of cytokine storm include gene therapies, immune checkpoint inhibitors, cardiac-bypass surgery and allogeneic stem-cell transplantation. Furthermore, monogenic disorders- genetic disorders caused by a single gene mutation- can also trigger cytokine storm, although this is rare, along with autoimmune and neoplastic diseases. For example, in patients with primary HLH autosomal recessive monogenic abnormalities this leads to defects in effector and regulatory mechanisms in the immune system, which results in overactive histiocytes and lymphocytes without a pathogenic trigger^[5]. In patients with secondary HLH, viral, autoimmune or neoplastic disorders lead to a cytokine storm. For example, patients lacking functional perforin, which is critical for resolving infections and inflammation, have prolonged T-cell production of interferon- γ and TNF, so when they are infected with a viral pathogen, secondary HLH-associated cytokine storm develops and it becomes very difficult to shut off the immune response; a self-reinforcing inflammatory spiral ensues, leading to multi-organ failure and likely death^[1].

In the COVID-19 sphere, a distinct positive correlation between the nasopharyngeal viral load and cytokine levels in ICU patients suggests that those with high viral loads are more susceptible to cytokine storm^[1]. Also, comorbidities such as hypertension, diabetes and obesity are associated with more severe cases of COVID-19, possibly because of the pre-existing chronic inflammatory state or a lower threshold for the development of organ dysfunction, thereby increasing susceptibility to cytokine storm in these patients. Surprisingly, it has been found that classic cytokine storm identification methods, such as the 2004 HLH criteria and the 2016 macrophage activation syndrome criteria, each missed at least 75% of COVID-associated cytokine storm patients, while classifying many others as false positives. Lacking an acceptable scale, scientists at Temple University in Philadelphia created their own Temple Criteria using patient data to determine whether a patient had cytokine storm from COVID-19; the method had a sensitivity of 0.84

(sensitivity measuring the proportion of positives that are correctly identified) and specificity of 0.73 (specificity measuring the proportion of negatives that are correctly identified) in patient trials- very encouraging figures^[6].

As for treatment, very often it simply involves supportive care to maintain critical organ function as well as control of the underlying disease- in the case of a bacterial trigger this would include antibiotics, for example- and immunosuppression, such as with the use of corticosteroids like dexamethasone, to limit collateral damage induced alongside the cytokine storm. Eliminating a particular cytokine that is elevated in the blood using treatments such as anti-interleukin-6, anti-TNF, anti-interferon- γ , or anti-interleukin-1 β antibody therapies may be effective in some instances. However, it may also propagate the underlying disease if given too soon, especially if the cytokine storm has been caused by a pathogenic invader. Hundreds of immunomodulatory drugs are currently under investigation for treatment of COVID-related cytokine storm; successful examples include the interleukin-6 receptor antagonists tocilizumab and sarilumab- drugs used to treat rheumatoid arthritis which have showed to reduce mortality from 35.8% to 27.3% when compared with standard care and found to significantly improve survival as well as cutting time spent in hospital by a week to ten days in patients with severe COVID-19 (REMAP-CAP trial)^[7].

I'd like to finish by lauding the work of healthcare professionals and scientists in helping the world get a grip on this pandemic. While there are many lessons to be learned from the past year, we must stop to appreciate the selfless work that these people carry out and what they are willing to risk to help others. So, THANK YOU!

Edited by Mann Patira



Can Stem Cells Help Cure Acute Lymphoblastic Leukaemia?

By Aditya Jain (Y12)

What is leukaemia?

Leukaemia is a cancer caused by the uncontrolled division of white blood cells. There are two categories of leukaemia: fast spreading acute leukaemia and slow spreading chronic leukaemia^[1]. Acute leukaemia further splits into two categories: acute myeloid leukaemia and acute lymphoblastic leukaemia. In this article I will focus on acute lymphoblastic leukaemia which, unfortunately, is one of the most common cancers in children.

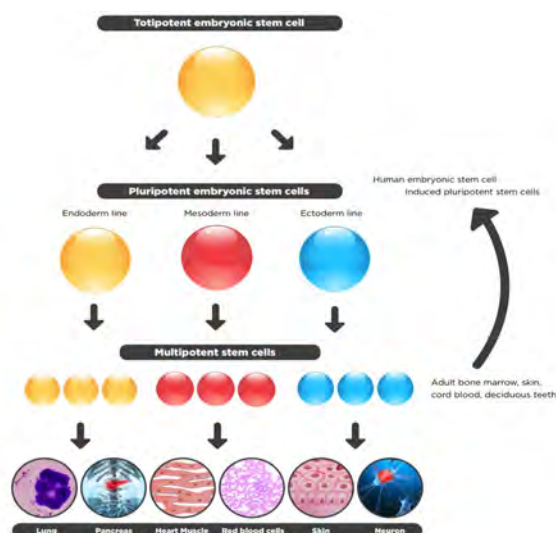
How does acute lymphoblastic leukaemia occur?

Acute lymphoblastic leukaemia is a condition that affects the bone marrow. The cells of the bone marrow have a mutation in which portions of two non-homologous chromosomes are switched. As a result, the cells are stuck in the early stages of development and are immature. This means that they do not function effectively, so lose their ability to properly produce mature blood cells and uncontrollably divide, overcrowding the bone marrow and causing cytopenia. These faulty chromosomes also cause the intracellular proteins of the cell to fold incorrectly and so the cells cannot function and divide as they normally would be able to. Acute lymphoblastic leukaemia leads to the release of large volumes of premature white blood cells. As a result, this means that the number of red blood cells and platelets are reduced (cytopenia) which can cause tiredness and breathlessness. Being premature, the white blood cells are ineffective in fighting off viruses and other infections, allowing the viruses to proliferate to other places like the brain and liver^[2].

What are stem cells?

Stem cells are undifferentiated human cells that are able to develop into many different cell types. This can range from muscle cells to brain cells. In some cases, they can also fix damaged tissues^[3]. Most of the cells in our body are not stem cells, they can only replicate through mitosis, creating copies of themselves. These therefore are very specialised and adapted to their function; they cannot just suddenly turn

into a different type of cell, for example, a skin cell cannot create a neuron. Our body does, however, have some multipotent stem cells left. They are stored in the bone marrow, brain and some other organs. There are three types of stem cells: totipotent stem cells – the best example of which is the zygote and can differentiate into any cell and create a whole organism; pluripotent stem cells – the ones in the embryo four days after fertilisation which can also differentiate into any cell in the body but not a whole organism; multipotent stem cells – found in the foetus and in specific tissues in the body and can only differentiate into those tissues^[4].



Stem cell potency hierarchy

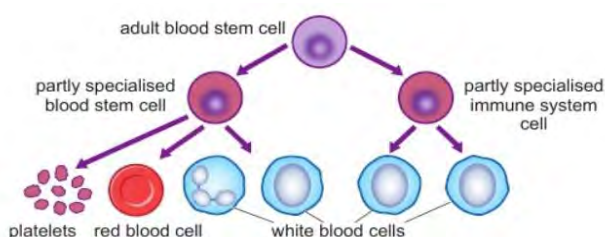
Why are stem cells vital?

The stem cells in our body help promote normal function and allow the body to produce new cells to repair minor damage, for example. The adult blood stem cell in our bone marrow can differentiate into different types of cells that are vital to normal body function as displayed in the diagram. Without the adult bone marrow stem cell, we would not have any white blood cells that protect us from pathogens or the red blood cells that are essential to allow aerobic respiration and not even the platelets that help stop bleeding. Therefore, stem cells are extremely necessary & vital to the body. When stem cells stop functioning properly, the patient's immune system is likely to be



compromised; the likelihood of catching infections, therefore, increases.

Blood stem cell differentiation



C Blood stem cells are found in marrow in the middle of long bones (such as the femur). They continue to divide throughout life to produce new blood cells.

How does stem cell therapy help cure acute lymphoblastic leukaemia?

Stem cells are extraordinary in our body considering their ability to develop into different cell types. They can, therefore, help to prevent or cure diseases such as heart disease, spinal cord injuries and diabetes, as well as cancer. As part of multipotent stem cell therapy, the cells used here form a specific germ layer after the differentiation of pluripotent stem cells, and can give rise to one or two specialised cells. This type of therapy is proven to be effective and has been used for decades in haematopoietic stem cell transplantation. Haematopoietic stem cell transplantation uses either allogeneic stem cells (stem cells from another individual) to replace the defective ones found in the bone marrow or autologous cells from the patient's own body that have the leukaemia cells removed. These cells will now produce the correct lymphocytes that function properly and do not uncontrollably divide.

This type of stem cell therapy involves the use of bone marrow as a treatment to haematologic and lymphoid cancers – mainly acute lymphoblastic leukaemia. The underlying cause of the cancer may be that the patient has faulty genes producing defective cells which can cause problems like the uncontrollable division of T-cells. The way this treatment works is that bone marrow from a healthy individual with matching antigens (to help avoid rejection) is transplanted into the bone marrow of the patient. Replacing the bone marrow means that healthy intracellular proteins are produced which yield healthy precursors, thereby, reducing the production of unhealthy and defective lymphocytes^[5]. The old cancerous cells are either already killed due to previous chemotherapy or surgery. These are replaced

with either healthy stem cells from a donor (allogenic) or the patient's own stem cells (autologous), which are purged and frozen for preservation, removing any leukaemia cells from the sample, and then infused back into the blood, after chemotherapy^[6].

This treatment can significantly improve the survival chances of cancer patients. In a study of 508 patients the eight-year survival average was 65% in patients less than 30 and 38% in patients less than 60^[7]. This treatment provides many benefits and forms of the treatment are already in use. For example, in Europe, more than 26,000 patients are treated with blood stem cells and this is only going to grow with time^[8].

Drawbacks of therapy

There are many potential concerns with stem cell therapy:

- Risk of cancer: the stem cells could divide uncontrollably and cause cancer once again; this means that it could actually harm the person even further, affecting their normal function and not actually helping them.
- Rejection: the immune system attacks and destroys these stem cells since they are foreign; to reduce risk of this the patient could take immunosuppressants and also take cells from a blood relative to help reduce the chance of rejection.
- Graft vs Host disease: transplanted stem cells or bone marrow contains cells from the donor's immune system; these cells can sometimes recognise your own tissues as being foreign and attack them.

Although stem cell therapy is still in its infancy, its benefits and widespread uses mean that it is likely going to flourish in the future. With further research and development, I believe stem cell therapy will be at the forefront of our fight against cancer, and will be the next major revolution in patient healthcare, just like antibiotics and vaccines were before it.

Edited by Aditya Chougule and Atharva Narkhede



A Vaccine and a Virus

By Moksh Sachdeva (Y12)

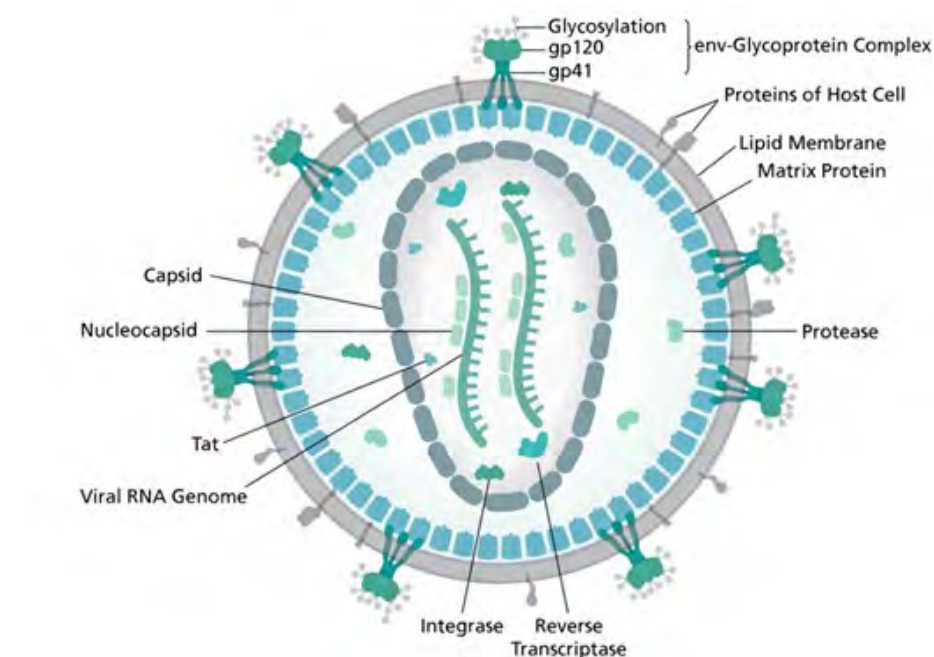
What is a virus?

A virus is an obligate parasite made of proteins and sometimes a membrane made of lipids containing single stranded or double stranded DNA or RNA. Viruses cannot replicate on their own however they can replicate in living cells. A virus can also affect the behaviour of the host cell. It is estimated that over one million different viruses infect invertebrates on earth. Viruses can be argued to be alive since they can persist independently, however some may argue viruses are not alive since they are unable to replicate independently^[1].

What is the structure of a virus?

Viruses don't have a specific shape since they display a variety of sizes and shapes otherwise known as morphologies. In general, they can be summarised into five main morphological virus types^[1]:

Icosahedral - these are most animal viruses however some animal viruses may be near



spherical with icosahedral symmetry.

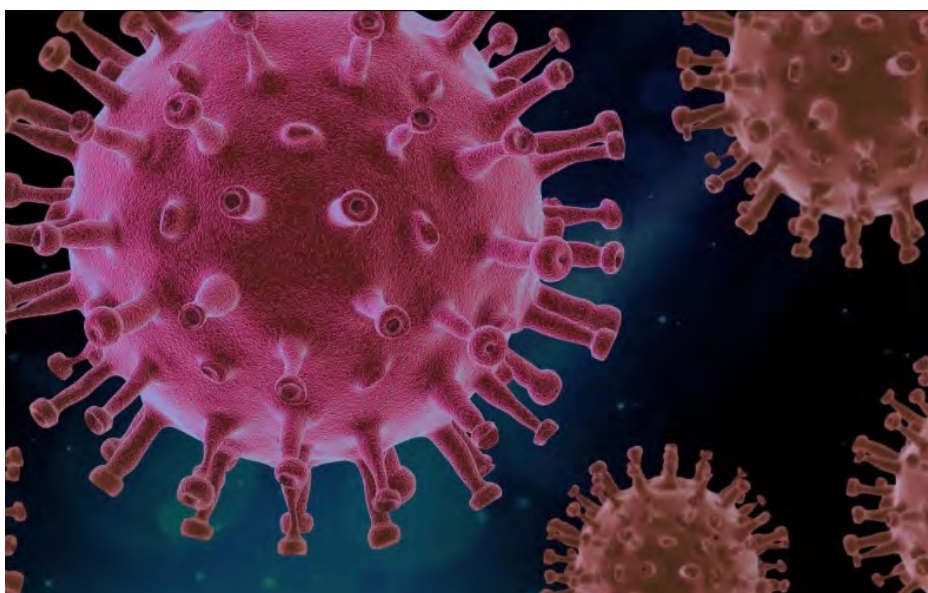
Prolate – this is a common arrangement for bacteriophage (a virus that infects bacteria) heads and consists of an icosahedron elongated along one axis.

Helical – this shape may consist of a central cavity or hollow tube and tend to form a helical structure since they tend to be stacked a central axis.

Envelope – these types of viruses envelop

themselves into a modified form of a cell membrane where either an internal membrane (such as the nuclear membrane) or the outer membrane (that surrounds the infected host cell) allows them to gain a viral envelope.

Complex – this shape possesses a capsid that is unlike the other shapes and may also possess other structures such as a complex outer wall or a tail of protein.



A virion, which is more commonly known as a complete virus particle, has a protective coat of protein called a capsid which surrounds the nucleic acid. The virus may also have gained a lipid envelope from the membrane of the host cell. Proteins encoded from the viral genome make up the capsid and the shape of the capsid allows for morphological distinction.

How does a virus work?

Viruses work by attaching onto specific molecules on the cell surface. A penetration virus will be endocytosed into the cell. The capsid will remove the exposing nucleic acid and then the replication of nucleic acid and synthesis of protein coats will occur allowing the virus to replicate.

Why is it so hard to control viruses?

Whilst a vaccine may help the body to produce antibodies to combat the virus, the vaccine cannot eradicate the virus from the general population since it is not feasible to have everyone vaccinated. Furthermore, over time a vaccine may no longer be effective for preparing the body to produce the required antibodies since the virus would have mutated. A virus mutates through either drifting or shifting. When a virus drifts, a minor change occurs in

the surface antigens due to point mutations. A point mutation is a genetic mutation where a single nucleotide base is changed, inserted or deleted from the DNA or RNA sequence of an organism. This results in the wrong amino acid being produced, thus altering the shape of the protein. Often, when a virus drifts a vaccine may still be effective. This is due to the change in the protein structure being minimal and so the virus is still recognised by the antibodies. However, when a virus shifts the vaccine is no longer effective. When a virus shifts, a major change in the antigens surface occurs accompanied by virus fusion. Virus fusion can be described as the process by which enveloped viruses enter the host cells. The shift leads to a major change in the antigens and so they are much less likely to be recognised by the antibodies which were produced in response to the vaccine. Thus, the vaccination is not very effective ^[2].

What is a vaccine?

A vaccine is a device that helps prepare the body to produce antibodies for a virus so that it can fight the virus when it encounters it. Vaccines are designed to prevent the replication of a virus in a cell rather than treat them. A vaccine injects a

weakened or inactive pathogen into the body where white blood cells then produce antibodies that would be able to combat the virus if it were encountered by traditional infection. Vaccines harness the natural activity of the immune system to better prepare it for the exposure of a particular virus ^[3].

The different types of vaccines ^[4]

The Oxford vaccine is composed of a weakened version of the common cold virus which has indeed been genetically engineered to produce proteins from the coronavirus. Nuclei acid (mRNA) host makes spike protein. These are safe and quick to develop but difficult to store and this is similar to the vaccine developed by Pfizer for Covid.

Edited by Aditya Chougule



The Hearts that Never Beat

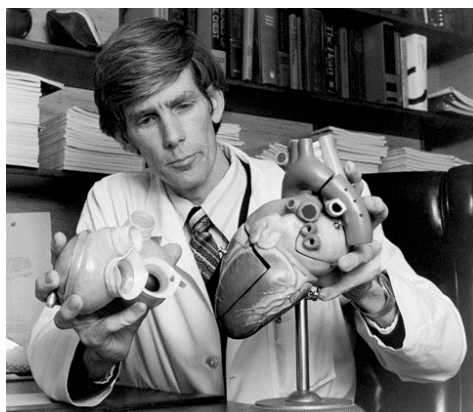
By Shinujan Saravanamuthu (Y12)

The heart is the driver of the body, with a simple function yet with such complex intricacies, giving it the strength to channel up to 9000 litres of blood a day. When the heart fails many face the only option of a heart transplant- receiving a donor heart is life changing, a second chance at life but granted at the expense of another.

Unfortunately, however, this is not accessible to everyone; as of July 2020 there are 343 patients in the UK who have been placed onto a waiting list for a donor heart ^[1]. Even though donor numbers are increasing, with people now having to opt out of being a donor, it is still not meeting demand with patients having to wait on average 1085 days for a transplant- one in six patients do not receive the heart they require in time ^[2]. Instead of a donor heart what else can be done...

permanently have someone strapped to a cardiopulmonary bypass machine? As well as being impractical, this machine does not maintain blood flow, can only sustain the body for 45 minutes and is only used as a stopgap measure during surgery. Maybe implant vibranium to magnetically control

blood? Not quite. However, mechanical hearts may be humanity's next evolution in our quest to become cyborgs; a hunk of titanium strapped to the chest with a motor spinning at 10,000 RPM seems to be the vital next step- impractical maybe- but lifesaving indeed, and in the medical world that will do.



Robert Koffler Jarvik

With all great inventions, there is a mastermind, pioneer and successor; for artificial organs it was Robert Koffler Jarvik, with the Jarvik-7. Born in Michigan, in 1946, he was a curious man: always either elbow deep into his research or a patient (quite literally) and inventing the surgical stapler just as a teenager. Working under the wing of Williem Johan Kloff- the inventor of the dialysis machine during WW2-

Jarvik began his work on artificial organs in 1971 and the Jarvik-7 was conceived around 1982 ^[3].

The Jarvik-7 was designed to completely replace the heart so the diseased heart had to be removed for its installation. It is composed of two bell-shaped plastic, shown to the right and titanium pumps that take the place of the two ventricles (the main chambers that deliver the final flow of blood to the lungs and body). It works by sewing on the cuff end of the pump to portions of the atria that remained when the heart was originally removed. After attaching the pump the chest is closed, leaving two flexible plastic tubes extruding from the torso, which are attached to an external air compressor which draws and pumps blood to the heart.



Jarvik-7



The first patient to receive the Jarvik-7 was retired dentist Barney Clark on December 2nd 1982. After the operation Clark awoke and was able to live another 112 days; the Jarvik-7 had extended Clark's life, but it could be argued that he was not truly living as he was mostly bed bound during this period, trapped within the confines of the hospital. The media documented Clark's condition on the road to recovery, however, it was soon clear that Clark was not to get any better with the major problem being that the site where the tubes were attached to the chest were prone to infection; this resulted in the lungs being compromised and significantly weaker so unable to support the artificial heart. This was in addition to the high velocity blood that, due to constant compression from the pump, was being damaged resulting in the blood clotting and leading to a stroke which left Clark paralysed [3].

This sequence of events led to the fall of the Jarvik -7 with the media seeing it as doctors playing God and extending life in return for one at a lower quality; which was also presented in the second patient, William Schroeder. Ultimately, the FDA discontinued the Jarvik pumps due to quality control resulting in the end for the 'bridges to transplant' [4]. Fast forward 18 years, to

2000, and the birth of the Jarvik 2000: a motor designed to fit onto the heart with a maximum output of up to seven litres per minute [5]. It was first implanted into a 61-year old man with cardiomyopathy- an inspiring procedure...

A skull pedestal, is attached to the head as the connecting point between the motor on the heart and the external battery. This is done because the scalp tissue is fixed closely to the skull so can reduce risk of trauma and damage to soft tissue- maceration.

Then a posterolateral thoracotomy incision- just below the shoulder blade- is made and a cruciate (cross-shaped) incision is made at the apex of the heart, which is then cored, removing a section of myocardium. When this occurs, the pump is then inserted to minimise bleeding. The right motor is connected at the now open ventricular apex and the blood is then run through a Gore-Tex sleeve which is connected to the descending aorta. During implantation, the power cable is carried from the apex up to the scapula where it is then connected to the pedestal described above.

This miracle procedure was performed by Dr Stephen Westaby as well as Bud Fraizer, who impressively improvised much of the operation, paving the way for future

implantations [6]. Finally, the 'bridges to transplant' could evolve to become the 'bridges to destination', where the pumps could take over the heart either giving enough time for a donor to be found or for the heart to recover again. With the leading causes of death in the UK since 2001 being ischaemic heart disease and cerebrovascular disease, complete artificial hearts demonstrate increasing potential in treating today's biggest diseases, and with upcoming inventions such as Total Artificial there is much more to look forward to [7].

Edited by Aditya Chougule



Can We Grow New Teeth?

By Mohamed Ahmed (Y12)

Translational dentistry (a collective term for regenerative dental medicine and dental tissue engineering) has been an active frontier of research over the past 100 years and it seems that it may soon be fit for use today ^[1].

To understand why dentists have been looking for ways to grow new teeth back, we have to ask why we cannot replace our teeth- like many reptiles and fish who regrow a new set of teeth every few months ^[2]! In fact, leopard geckos have been the ideal model organisms to use when researching mechanisms of teeth growth.



In humans and other mammals, the relevant stem cells needed to build up the adult teeth when we were children die later on in our lives whereas these geckos form new generations of teeth (from retained stem cells) which grow in size under the tissue, until they surface and displace the more mature tooth above the tissue ^[3]. This is much rarer in mammals due to the specialization of our teeth in response to more specific and evolved dietary needs. For example, carnivores and omnivores have developed canine teeth and molar teeth respectively to allow them to



effectively digest or obtain their food, which demonstrates that evolution has favoured well aligned teeth that are produced once.

While the process of tooth creation is complicated, it is luckily well understood: essentially, soft and connective tissue, nerves and blood vessels (all found in the pulp at the centre of the tooth) are bonded to 3 types of hard tissue: dentine, enamel and cementum. Using RNA sequencing, researches have deduced differentiation pathways of stem cells to odontoblasts and ameloblasts which are cell populations that give rise to dentine (the softer- but still harder than bone- tissue closest to the pulp) and enamel (a harder protective coating of the tooth) ^[4].

The fruits of this research have expedited efforts to replace missing teeth in places such as King's College London who have published their research in the Journal of Dental research where they are able to produce dentine, enamel and roots by cultivating adult human gingival tissue (from the gums) combined with embryonic tooth mesenchyme (loose multipotent cells in a protein and fluid mesh). The epithelial cells from

the adult gum can respond to 'tooth-inducing' signals from embryonic tooth mesenchyme in a way that is appropriate for in vitro (in a living organism) growth, which would contribute to tooth formation.

The issue we face today is in the ethics of obtaining embryonic cells for tooth replacements, which may be considered a frivolous use of the resource when extractions and other treatments are available and these cells could be used for more critical procedures (or certainly those who are ethically opposed to the use of the embryo in its entirety), and a lack of understanding in the cell dynamics- how the cell interacts with its environment- involved over the differentiation process. Research is currently being conducted to induce the same differentiation pathways possible in embryonic stem cells from adult stem cells and to understand how these cell populations respond to a variety of environmental factors.

Edited by Aditya Chougule



Intrigued? Organ Donation

Here at the Wilson's STEM Intrigue, we are very curious and this often leads to big discussions and debates of scientific news within the magazine writers.

Our new "Intrigued?" section hopes to shed some light on one of our most heated debates, while hoping to inform and update news you may have missed, and tackle the big questions with STEM.

We surveyed our editors and writers and below are some of the responses.

The New Law:

20th May 2020 marked a defining moment in the history of organ donation as the new opt-out system first came into effect in England. Also known as Max and Keira's law, this system means that all adults in England will be "considered potential donors unless they choose to opt out or are excluded" (gov.uk). Those excluded from the plans include: children under 18; people who lack the mental capacity to understand the changes for a significant period before their death; people who have not lived in England for at least 12 months before their death.

Great! From what I had heard, hospitals were always short on organ donors, despite the fact that so many people would happily donate their organs to save other people's lives. Having sensible defaults is important for anything, but especially where people's lives are involved.

Michael Y13
@Wilson'sIntrigueOrganDonation

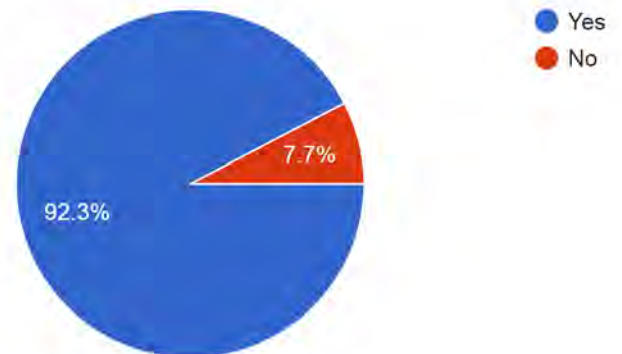
I believe that it was a valid decision to make, seeing as the majority will be willing to donate their organs. This would ultimately make life easier for many, whilst preserving the freedom to choose.

Ishan Y11
@Wilson'sIntrigueOrganDonation

Poll One :

Were you aware that the law had changed?

13 responses



Slightly queasy but it would help lots of people

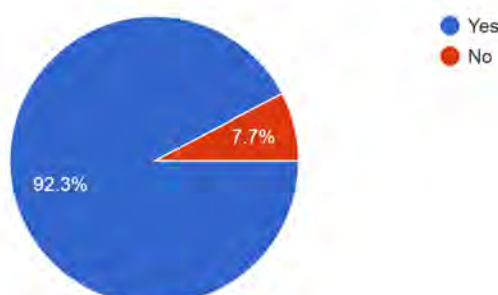
William Y12
@Wilson'sIntrigueOrganDonation

The idea is there and it is a good one, however, I feel that there has been a lack of effort by the NHS that the law has changed; yes I have seen and heard adverts to opt out but I still don't believe that it is sufficient. Thus it can lead to controversies and future problems for organ donation, however, it may have been daunting for people to think about organ donation especially during the current climate.

Shinujan Y12
@Wilson'sIntrigueOrganDonation

Poll Two: Would you be willing to have your organs donated?

13 responses



As a devout Muslim, my stance on most issues of morality is motivated by my faith. In the case of organ donation, many modern Islamic scholars differ in opinion. Although the most recent fatwā (Islamic scholarly opinion) is that organ donation is permissible in Islam following circulatory arrest, I would rather in this case err on the side of caution.

Nabeel Y12
@Wilson'sIntrigueOrganDonation



Arguments supporting this system include: 80% of people in England support organ donation but only 38% have opted in. This means that families are often left with a difficult decision when a loved one dies. Moreover, in 2019, 408 patients died in the UK on the transplant waiting list and as part of the law, the prospective donor's family will continue to be involved prior to organ donation.

Nevertheless, there are concerns that inaction in an opt-out system may lead to false positives, where individuals who do not wish to donate eventually become a donor. An opt-out system may also increase proportions of deceased donations, which are less likely to be suitable on account of possible brain injury, trauma, hypoxia or cancer prior to death. In the UK, it is estimated that only around 1% of patients who die do so in circumstances that allow organ donation to proceed. On top of this, some religious belief systems discourage or prohibit organ donation, attaching greater ethical implications to any potential false positives.

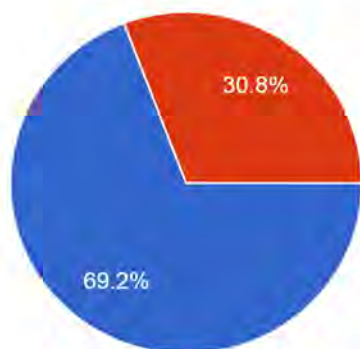
More information can be found at:

<https://www.organdonation.nhs.uk/helping-you-to-decide/about-organ-donation/fag/what-is-the-opt-out-system/>

<https://www.gov.uk/government/news/opt-out-organ-donation-max-and-keira-s-bill-passed-into-law>

Having read the extract and info, and considering personal opinions and preferences, is an opt-out donation scheme really better than an opt-in one according to you?

13 responses



● Opt-out donation scheme is better
● Opt-in donation scheme is better

We need more organs. Opt-in may prevent those pesky false positives, but there is always discussion with the donor's family beforehand whether or not to proceed with the donation. In most cases, the family should be aware of the donor's decision.

Koushikk



It is not worth giving people an opt in system when thousands of people are dying - action needs to be taken to help these people. Also, when people die, they wouldn't care whether they have their organs in their body or not so it shouldn't be a problem for them either. It's a win-win situation.

Arya

Although I am in favour of the opt-out scheme law, I believe that an opt-in donation scheme would be better, because it ensures that everyone who actually wishes to donate their organs are doing so. It eliminates the cases of uncertainty where it is unclear whether the donor actually wishes to donate their organ(s).

Shanjeev



It saves essential time, and therefore lives, for those who are in need of organs, which require extensive waiting lists which can go on for years. Opting out increases the availability for organs in the future. However, in the coming years use of stem cells and dying hearts are becoming increasingly promising and maybe the need for organs will decrease.

Shinujan

Control over our own bodies, dead or alive, should not be superseded by a need for organs in a hospital.

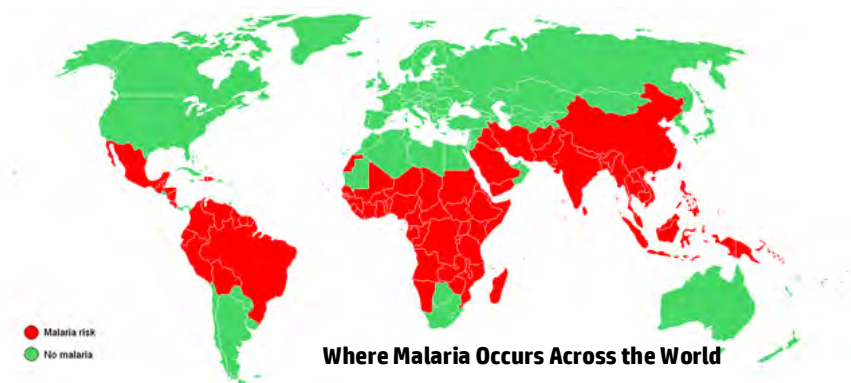
Haris

What do you think: OPT-IN or OPT-OUT?



Is Chloroquine an Effective Medication for the Prevention and Treatment of Malaria?

By Junaid Ali (Y12)



Malaria is a parasitic disease spread by female *Anopheles* mosquitoes. It is widely spread in many tropical regions, mainly areas in Africa, Central/South America, Asia and parts of the Middle East, because mosquitoes are able to reproduce and complete their growth cycle in areas with high humidity and high temperatures^[1]. Symptoms may begin any time from 7 to 18 days after being bitten by a mosquito that carries the pathogen. Symptoms can vary and include a high temperature (>38°C), headaches, vomiting, muscle pains and diarrhoea^[2].

Malaria is caused by single-celled eukaryotes known as *Plasmodium* parasites^[3]. *Plasmodium* species develop in blood-feeding insect hosts. When the mosquito bites the host's skin, the parasite enters their bloodstream through the mosquito's saliva. The parasite can then reproduce in the liver asexually. The new parasites then infect red blood cells and begin to multiply asexually. This causes the cell to burst, allowing the parasites to infect more cells and the cycle repeats.

There are 5 different species of *Plasmodium*: *P. falciparum*, *P. malariae*, *P. vivax*, *P. ovale* and *P. knowlesi*^[4]. *P. falciparum* is the most dangerous as it is the most common malarial parasite and

causes the most deaths worldwide^[5]. It is also capable of avoiding detection from the immune system.

Whenever a red blood cell is infected by *P. falciparum*, it expresses a molecule on its surface: *P. falciparum* Erythrocyte Membrane Protein-1 (PfEMP1). PfEMP1 is coded by var genes in *P. falciparum* and it is this protein that acts as the antigen which should be detected in an immune response. However, in a *P. falciparum* genome, there are 50-60 var genes and only one gene is expressed at a time^[6]. This means that the surface antigen is able to repeatedly change so that it is not detected by the host's immune system. In 2018, *P. falciparum* made up 99.7% of estimated malaria cases in the World Health Organisation (WHO) African Region, 71% of cases in the Eastern Mediterranean and 65% in the Western Pacific^[7].

Chloroquine is a medicine that is commonly used in the treatment and prevention of malaria. It is composed of a combination of various antimalarials and is typically taken with another medication called proguanil^[8]. It is able to prevent the spread of the malarial parasite by interfering with the processes carried out while it is in the host's red blood cells. During this phase of the lifecycle, the

parasite must reproduce. To do this, it requires energy for metabolic processes and amino acids to create new proteins. The parasite achieves this by breaking down haemoglobin. As well as proteins, haemoglobin is also made of a heme unit. The heme molecule is toxic to the parasite and soluble. To protect itself, the malarial parasite crystallises heme into hemozoin – a non-toxic molecule that forms insoluble crystals.

When chloroquine is taken into the body, it enters red blood cells by diffusion and it occupies the parasite's vacuole where the haemoglobin is digested. The vacuole is acidic, so the chloroquine gains H⁺ ions (protons) and it can no longer leave the vacuole by diffusion. Chloroquine is able to limit the amount of hemozoin molecules that the malarial parasite can crystallise. Therefore, after a certain point, the parasite can no longer continue to crystallise heme into hemozoin, so heme begins to build up. Heme binds to chloroquine to create an FP-chloroquine complex. FP-chloroquine is very toxic to the parasite and disturbs its usual membrane function. In the end, the parasitic cells break down and undergo lysis.

Chloroquine was first discovered in 1934 by Hans Andersag. Only in 1947 was



enough learnt about its properties for it to be incorporated into clinical practice^[8]. Chloroquine is now a part of the WHO's Model List of Essential Medicines and it is available as a general medication^[9]. The first recorded resistance of the parasite towards chloroquine was in the 1950s and ever since, chloroquine has gradually become less and less effective against *P. falciparum*^[10]. This is because the parasites have evolved due to natural selection and are able to channel chloroquine away from its vacuole. This means the drug is incapable of interfering with the parasitic cell's processes. Parasite cells that have developed resistance can drain chloroquine at 40x the rate of a cell without chloroquine resistance.

In places where chloroquine resistance has not yet been discovered amongst *P. falciparum*, chloroquine is still shown to be highly effective as a form of treatment. From 2008-9, a study was carried out in the municipality of Puerto Lempira, Gracias a Dios, Honduras, in order to evaluate the use of chloroquine. A total of 68 patients from ages 6 months to 60 years of age completed the entire trial and each patient was positive for malaria due to *P. falciparum* according to light microscopy. Each patient was given a dose of chloroquine of varying sizes over the first 3 days, depending on their body weight. Patients received supervised treatment until day 28. The data showed that the parasite density in most patients had dramatically decreased by day 3 and by day 7 all patients were parasite negative (shown by no detected asexual stages in the blood)^[11].

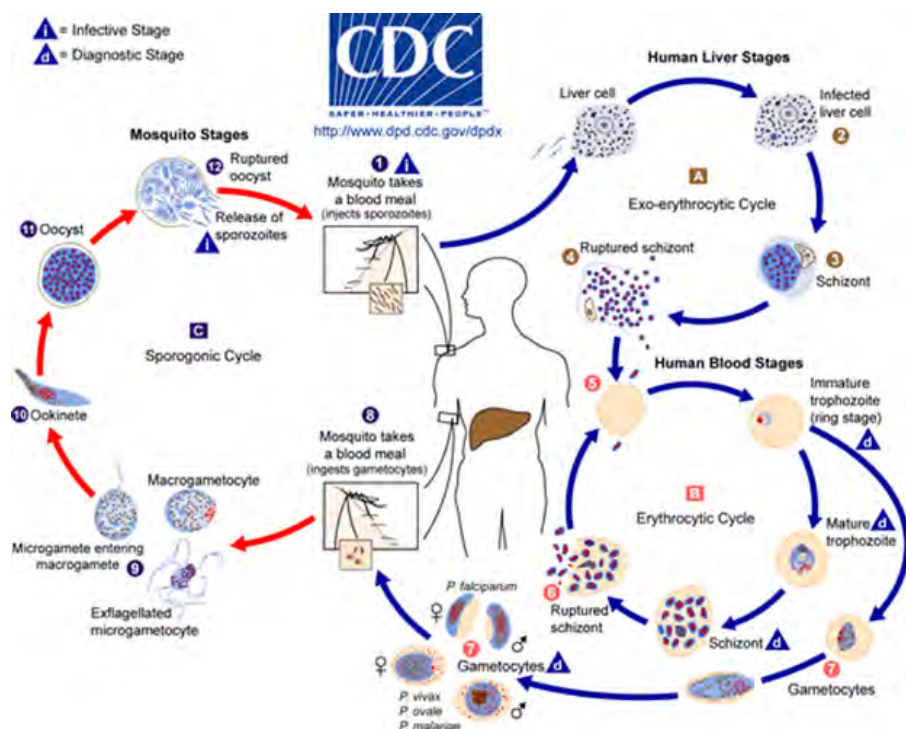
Chloroquine can have several side effects on the body, including nausea, vomiting, headaches and diarrhea. It also has several interactions with other drugs, which may be of

concern. For example, the interaction between chloroquine and another drug named cimetidine may lead to increased levels of chloroquine in the body due to the inhibition of chloroquine metabolism^[8]. Additionally, chloroquine in overdose has a 20% chance of death and can cause problems with vision, breathing, the heart or sleeping. Build-up of chloroquine deposits in the body can result in blurred vision and blindness.

Overall, it is clear that chloroquine is an invaluable medicine as it is extremely useful in the prevention and treatment of malaria, particularly malaria caused by *P. falciparum*, which is a highly deadly infection. However, it is also clear that resistance to chloroquine is becoming more widespread across the world with many countries opting to use other treatments such as artemisinin combination therapy (ACT) as the first-line drug for the treatment against *P. falciparum*. I believe that it is necessary for countries (at high risk especially) to be ready to use ACT if they are not already using it, as the WHO advises ACT to be used in areas where chloroquine-resistant *P. falciparum* are present^[12]. Small outbreaks of

malaria may arise if chloroquine-resistant *P. falciparum* develop in a certain area, which could lead to a wider spread if it is not controlled. Chloroquine should still be used, but I believe only under controlled circumstances for cases where there is shown to be no resistance to it.

Edited by Aditya Jain



What is the Effectiveness of the Use Of Antibodies when Treating Paediatrics Patients with Mild/Severe, Acute/Chronic Asthma Exacerbation?

By Junayd Soobratty (Y12)

An asthma exacerbation is distinguished by the swelling or inflammation of the airway^[1]. Respiratory difficulties are caused due to the narrowed bronchial tubes. The word exacerbation (more commonly referred to as an attack) denotes the event caused by a trigger such as an allergen leading to the airways constricting. Triggers may include, animal fur, pollen, dust or exercise where the trachealis muscles contract, producing more than standard levels of mucus. Asthma is the most common obstructive pulmonary disease in children affecting one in eleven in the UK^[2].

An asthma exacerbation can be categorised in two main ways which can further be subdivided into six classifications^[3]:

A) Intermittent asthma

- 1) Mild intermittent asthma: where the paediatric patient will only experience slight tightness of the airways on rare occasions
- 2) Moderate Intermittent asthma: where the asthmatic will feel some respiratory distress in exceptional cases
- 3) Severe intermittent asthma: where the person would feel significant tightness of the chest and pain sporadically

B) Persistent asthma

1. Mild persistent asthma: where the person would be wheezing or whistling when breathing, with slight swollen airways; symptoms occur more than twice a week but less than once a day
2. Moderate persistent asthma: wheezing to an extent of murmured speech, coughing, swollen airways, development of mucus in the airways, some chest tightness or pain; experience asthma symptoms every day
3. Severe persistent asthma: where the paediatric patient will feel significant respiratory distress. There may be a display of nasal flaring and paradoxical thoraco-abdominal movement; experience asthma symptoms every day and at least one night per week^[4].

Key characteristics of an asthmatic paediatric patient is the reversible airway obstruction, airway hyper-responsiveness and chronic airway inflammation typically with eosinophil infiltration. Eosinophilic asthma involves abnormally high levels of a particular type of white blood cell called eosinophils which are

part of the immune system produced in the bone marrow. However, high levels of eosinophils can cause inflammation in the airways, affecting the sinuses and nasal passages as well as the lower airways^[5]. The majority of asthmatic conditions can be controlled with conventional treatment such as inhaled corticosteroids (ICS) appropriate for their severity. However, some types of asthma, known as 'refractory asthma' which is when the patient experiences persistent symptoms, frequent asthma attacks or low lung function despite taking asthma medications as they do not respond to high doses of this conventional treatment. Some refractory asthma patients have to take oral steroids such as prednisone to manage their asthma.

In general:

Short-term treatments^[6]:

- Short-acting beta-agonists; first choice for quick relief of asthma symptoms as they are used as muscle relaxants so decrease the contraction on the trachea by the Musculus trachealis
- Anticholinergic agents; acts swiftly to instantly relax airways
- Oral and intravenous corticosteroids; relieve airway inflammation but can cause serious side effects when used long term

Treatment used in long-term:

- Inhaled corticosteroids; the most effective long-term control medicine usually
- Inhaled long-acting beta-agonists; open the airways by relaxing the smooth muscles around them
- Combination inhaled medicines; an inhaled corticosteroid along with a long-acting beta-agonist
- Biologics; target a cell or protein in the body to prevent airway inflammation
- Long-acting bronchodilators; possibly tiotropium (Spiriva) along with corticosteroids even though daily inhaled steroid is taken. Long-acting bronchodilators alone as a long-term asthma treatment is ineffective as it does not prevent and has short term effect for a long-term use



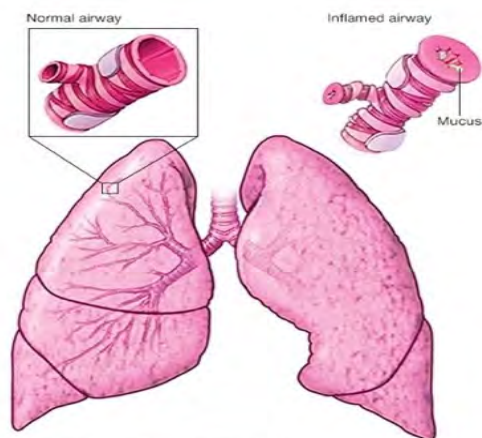


Diagram of normal vs. inflamed airway Mayo Clinic, 2020)

Other treatment:

Bronchial thermoplasty: this involves heating the tissue and reducing the amount of smooth muscle present in the airway wall; it is completed over a few procedures and has a permanent effect

Used for severe asthma where inhaled corticosteroids have no effect on the patient During bronchial thermoplasty, the doctor heats the bronchial tubes with an electrode The heat relaxes the muscles around the airway This in turn, moderates the discomfort and reducing possibility a severe asthma attack

IgE antibodies

In some severe cases of paediatric asthma 'refractory asthma', neither thermoplasty, corticosteroids, or any other treatment have any effect on the patient therefore distinctive measures are taken to help mitigate the consequences of an exacerbation.

Anti-IgE monoclonal antibodies are an innovative and unique method increasingly being used to treat refractory asthma. IgE (Immunoglobulin E) is naturally produced by B-lymphocytes. This antibody normally activates physical responses in an immune reaction. The anti-IgE antibody works by inhibiting IgE functions blocking free serum IgE and inhibiting their binding to cellular receptors. By reducing serum IgE levels and IgE receptor expression on inflammatory cells the allergic cascade can be controlled and reduced.

For some, exposure to allergens which are usually considered to be harmless, non-infectious substances (like dust or pollen) - can trigger the body to produce and release IgE. In these circumstances, IgE antibodies bind to the allergen and trigger an inflammatory response that can manifest the allergic symptoms mentioned above. When your body releases IgE, it is believed to bind to and activate several types of immune cells such as: basophils,

lymphocytes and mast cells.

Treatment

Treatment with anti-IgE is an approach that can be used along with bronchodilators or corticosteroids. IgE-levels helps to establish whether the patient will have a successful outcome with the anti-IgE antibody treatment. But a high level is not a requirement for this treatment as it may still reduce or alleviate any present symptoms in an exacerbation. Reducing IgE-levels is a more targeted method of preventing symptoms of allergic asthma than immunosuppression with standard steroids, which is the most common method of reducing inflammation and- although it works- has a lower success rate and less precision than anti-IgE, which has fewer side effects and avoids a sore mouth or throat and oral thrush seen in patients who take inhaled corticosteroids. Xolair (omalizumab) is an anti-IgE medication approved by the U.S. Food and Drug Administration (FDA) for the treatment of allergic asthma for paediatric patients over the age of six^[7].

Indications for the need of Xolair treatment include:

Severe or persistent asthma that is inadequately controlled with inhaled corticosteroids

A positive skin test or positive blood allergy test Chronic skin rash that is inadequately controlled with antihistamines

Xolair is given every two to four weeks as a subcutaneous (under the skin) injection as advised by a healthcare provider. Side effects can include injection site reactions, infections, and headaches.

Primarily, the main reason supporting the treatment of anti-IgE humanised monoclonal antibodies for the treatment of paediatric patients with asthma is the fact that many tests have verified its effectiveness in reducing the symptoms of an asthmatic exacerbation.

Summary of a medical trial

This trial was conducted and funded by Novartis where 246 randomized patients were tested (omalizumab, n = 166; placebo, n = 80)^[8]. Over the 24-week fixed-steroid phase, where only one drug was prescribed, omalizumab reduced the rate of clinically significant asthma exacerbations by 44% versus placebo 23% (in cases where symptoms worsened dramatically, a double dose of ICS was prescribed). Over 52 weeks, the exacerbation rate was reduced by 50% with omalizumab which had an acceptable safety profile, with no statistically significant differences in adverse events observed between



omalizumab and placebo ^[9].

This trial, upon preliminary inspection appears to be in favour of the use of the monoclonal antibody, as it is a randomised trial, allowing greater validity of results by removing any bias that may have been present otherwise, with $P = 0.047$. This p-value considers the probability of obtaining positive test results. A p-value less than 0.05 (typically ≤ 0.05) is statistically significant. A p-value higher than 0.05 (> 0.05) is not statistically significant and indicates strong evidence for the null hypothesis- that the anti-IgE treatment is not the most effective treatment- which means we would retain the null hypothesis and reject the alternative hypothesis, but the latter is not the case. Moreover, Xolair (commercial name for omalizumab) reduced the number of exacerbations by around half. Over the first 28 or 52 weeks of treatment in the first three studies, there were around 0.5 exacerbations per year in the Xolair group and around one per year in the placebo group. There has also been described greater improvement in quality of life for paediatric patients in particular. In a national health survey 5.5% of children aged 5 to 17 years old with symptomatic asthma experienced limitations in activity. After the omalizumab treatment, an 83% reduction in asthmatic exacerbations were observed. Furthermore, 73% of all paediatric patients with severe asthma felt limitations to physical activity had been alleviated as a result of Xolair providing evidence to suggest that this treatment has a high success rate ^[10].

On the other hand, antibody level can vary before treatment in each patient and IgE levels might be stagnant even if the patient has allergic asthma. A high IgE-level cannot confirm a diagnosis of asthma therefore high IgE can only suggest some sort of allergic disorder and anti-IgE may have no effect. In patients with allergic asthma aged 12 years and over, the most common side effects with Xolair- seen in one in ten patients of 100- are headache and injection site reactions, including swelling, redness, pain, dizziness and itching. Serious side effects from Xolair are not common, but they can occur involving inflammation in the blood vessels, chest pains, trouble breathing, skin rash, numbness, parasitic infections (causing pain in your belly /diarrhoea). In children aged between 6 and 12 years, the most common side effects- seen in more than one in ten patients- are headache and pyrexia (fever) ^[11]. Furthermore, Xolair is also not as cost efficient for the NHS, as if the patient is under 16 the NHS will have to pay the £256.15 (excl. VAT) per 150 mg vial. A single dose of 300 mg costs £512.30 and the cost for a 24-week course of treatment is £3073.80 (excluding VAT) ^[12].

This can be compared to the considerably cheaper inhaled corticosteroids; Ventilo inhalers ^[13]:

2-3 years

Volumatic with Mask = £6.70

Yellow Aerochamber = £8.02

3-16 years

Volumatic without Mask = £3.81

Blue Aerochamber = £4.81

In conclusion, we can clearly see that the anti-IgE monoclonal antibodies are practically efficient in reducing stress on the airways and this technology has revolutionised asthma treatment for the most severe and chronic cases. I believe the advantages to some extent do outweigh the disadvantages as we may have to allow and deal with the comparatively low clinical attainment rates in order to treat those with severe asthma based on current expertise, but in the future with more clinical trials and mass production of this antibody, treatment with anti-IgE can produce better outcomes than other current treatments for patients, along with fewer and less serious side effect. At this moment in time, anti-IgE does improve the quality of adjusted life years (QALYs) by two years but due to the current cost-ineffectiveness of this treatment, for the NHS to benefit and for the pressure of paediatric patients with severe asthma to be eased, only patients who experience no physical amendment in symptoms should be prescribed with Xolair alongside ICS.

Edited by Aditya Jain





DID YOU KNOW?

Logic gates in a computer system are created by combining various transistors in different orders. This is often the result of silicon semiconductors within the transistors and the electron properties (whether they have extra electrons or are deficient).

Computer Science Section

Neuromorphic Computing

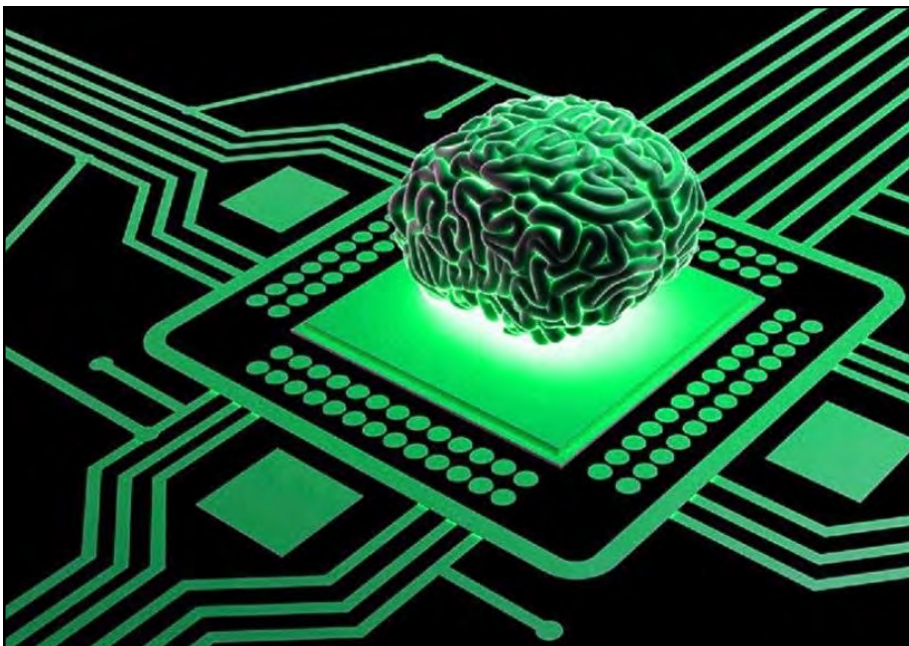
Mimicking the brain p24

Quantum computing

Computing in the quantum realm p26

"Computer Science is the operating system of all innovation"

- Steve Ballmer



Neuromorphic Computing

Can computers replicate the human brain?

By Jonathan Peter-Rajan (Y11)

With Moore's Law coming to an end and the ever-increasing importance of Artificial Intelligence, we are approaching a threshold that cannot be passed without making dramatic changes to our computing architecture - hence the need for neuromorphic computing was born. Its definition is much less complicated than it sounds: it is the designing and engineering of computer chips that use similar physics and computation to our nervous system. In essence, this will allow the creation of energy-efficient hardware, capable of carrying out highly

sophisticated tasks, especially machine learning.

The reason why scientists have looked towards our brains (which we have taken for granted) is because of their unparalleled performance whilst being extremely efficient. For example, the 86 billion neurons and one quadrillion synapses equate to 1 exaflop (10^{18} operations per second) consuming only 20W, the same amount it takes to power a lightbulb^[1]. To put that to context, traditional computers require 1.6 million processors, 1.6 petabytes, and 6MW to do the same^[6]. This is even translated in

some cases in our everyday life, where we can recognise images and patterns within seconds whereas computers are required to be trained on machine learning models for hours having been fed hundreds and thousands of data sets. Furthermore, the way our brain works provides a solution to the limitations of the Von Neumann architecture (made in the 1940s) such as the bottleneck created when having the CPU and memory separate. This results in the bandwidth of data transfer being limited causing latency and decreasing efficiency. Our brain avoids this issue since it has memory and processing embedded together; the cell body/soma can be seen as the CPU, axon as the data bus, and the synapse as the memory. Moreover, you can pass a gradient of understanding between neurons giving you a lot more computational options rather than your basic yes and no in binary.

The hurdle that we must jump is translating this biological computing to a solid-state device. For example, to mimic synapses (and communication via neurotransmitters), we need a single component that has memory however a standard resistor doesn't. A potential



solution to this problem is a memristor (as the name suggests it is a resistor that can retain memory - they behave in the same way as neurosynaptic cores) and so allows us to maintain the vital philosophy in neuromorphic computing that the CPU and memory should be very closely paired together. Therefore, as part of their solution for neuromorphic computing, engineers at the University of Michigan created a memristor-based computer [3]. This was created to resolve the memory bottleneck especially in AI and by applying neuroscience principles it also allowed them to dramatically improve the performance per watt.

Another implementation of neuromorphic computing has been done by IBM, with their TrueNorth chips [4]. These use neurosynaptic cores which have transistors to simulate programmable neurons. By 2013 they created a TrueNorth processor with 4096 cores, one million neurons, and 256 million synapses - all of this requires 70mW, four orders of magnitude lower than a conventional computer.

These two implementations give us a clear understanding of the practical advantages of using this architecture. To begin with, the memory and computation are integrated which reduces latency. They operate without a clock - this allows asynchronous operations resulting in lower power dissipation for a given performance level and the highest possible execution speeds [5]. Also, since every neuron is fundamentally a processor, each core can work both independently and in parallel which results in vast increases in performance and efficiency. Moreover, you can pass a gradient of understanding between neurons giving you a lot more computational options rather than your basic yes and no in binary. Finally, individual cores can fail, and the entire system can still function, improving scalability, mimicking the brain's neuroplasticity.

The progression in computing this technology can be applied to almost any field, from genome sequencing to very sophisticated simulations and even real-time machine learning. In the case of machine learning, neuromorphic computing would be incredibly significant as the neurosynaptic cores are essentially nodes in neural nets (ml models) represented through physical hardware instead of software abstraction which provides massive performance gains since the hardware architecture would be optimised for neural algorithms. The case for neuromorphic computing in AI is emphasised by the fact that a single neuron has thousands of connections (whereas a traditional CPU only has three); making a computer work (hardware) and think (software) like a brain will help us imitate the way humans learn and adapt quickly whilst unlocking the door to achieve perfect artificial general intelligence. Furthermore, the improved computing performance could be used for brain simulations allowing us to treat diseases such as dementia which we have little knowledge about currently. These simulations result in a positive feedback loop since an improved understanding of the brain will help us improve our neuromorphic architectures, which in turn improves our AI which then improves our brain simulations.

Ultimately, the irony in humans using their brains to make brain simulations to make brain-like hardware so that computers can think more like humans will potentially lead to many breakthroughs, far beyond the field of computer science, which we should all be excited about.

Edited by Mann Patira



Future of Computing: Quantum Computing

By Mann Patira (Y12)

What do we use now and why won't it work?

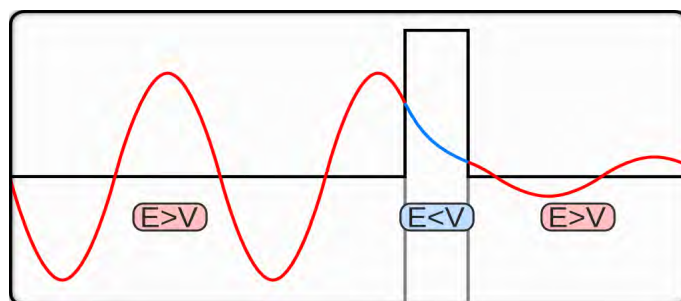
Computers are fast – incredibly fast. One of the many factors that help computers be so fast are transistors and how small they can now be manufactured. Transistors act as switches, storing the logical values one and zero as areas of high and low charge and the smaller they are, the closer together they can be, so it takes less time for signals to get from one part of the chip to the other, allowing for faster processing speeds.

The technological evolution of transistors on a silicon chip was predicted by a co-founder of Intel, Gordon Moore in 1965; he proposed that the number of transistors on a silicon chip would double every two years, and for the next 40 years, he seemed to be correct as progress followed his estimation as around every 18 months, the size of a transistor would halve^[1].

However, around 2010, Moore's law had begun to break down as transistors had become as small as ten nanometres, and around this size, classical physics no longer works, and we have to approach the problem using quantum physics. This means we have to understand that at these sizes, particles not only have particle properties but wave properties too, a phenomenon called wave-particle duality.

This begins to become a problem in computers because, as electrons approach a transistor that is meant to block all electric charge, since they exhibit wave properties, they still have a small probability of being on the other side of the gate. This is called quantum tunnelling and is greatly problematic as this electron leakage will cause errors in calculations since incorrect binary strings are provided to the processor^[2].

As a result, we seem to have reached a limit to how small transistors can be made and thus a barrier to how fast a classical computer can get. Therefore, an entirely new solution is required to allow technology to evolve and this is given in the form of quantum computers.



Quantum Tunnelling^[11]

How does Quantum Computing Work?

It is important to first note that a quantum computer is not just a more powerful version of our current computers, just like a light bulb is not a more powerful candle. You cannot build a light bulb by building better candles. A light bulb is a different technology, based on deeper scientific understanding. Similarly, a quantum computer is a new kind of device, based on the science of quantum physics^[3].

Unlike classical computers, where bits are stored as definite ones or zeros as high or low electric charge, quantum computing uses qubits instead such as an electron or a photon, which have a certain probability of either being a one or a zero but until they are measured, they exist in an undefined state. This is called superposition and is unlike anything we experience in the real world. The binary values no longer relate to electrical charge but correspond to properties of the qubit such as the spin of the electron or the polarisation of a photon. Different gates are used to alter the probabilities of a qubit relating to a specific value such as a Hadamard gate which sets the probability of each binary value to be 50%^[4].

Superposition is important since it frees us from binary constraints and allows for uncertainty so when a calculation is performed on a qubit, it is carried out on both a one and a zero in parallel, making the system much more powerful^[5].



Quantum computers also make use of a phenomenon called entanglement where multiple qubits exist in the same quantum state so changing the state of one qubit will instantaneously change the state of the other qubits no matter how far apart ^[6]. This concept isn't fully understood by scientists as even Einstein famously described it as "spooky action at a distance."

This helps execute processes quicker since the states of multiple qubits can be discerned by measuring the state of just one qubit because they are all entangled. Unlike a classical computer where the values of individual bits are strung together, in a quantum computer, it is the correlations that are brought together and described. As a result, as the number of qubits increases, the processing power of a computer increases exponentially to the point where to describe a system of 300 qubits, more numbers than atoms in the universe would be required ^[7].



Quantum Entanglement ^[12]

What can it do?

The power of Quantum computing has many uses, firstly quantum uncertainty from superposition could be used to create private keys for encrypting messages sent from one location to another so that hackers could not secretly copy the key. This kind of unbreakable encryption is already being tested by banks and other institutions worldwide ^[8].

Also, quantum technologies could transform healthcare and medicine. This is because the design and analysis of molecules for drug development is a challenging problem today, and that's because exactly describing and calculating all of the quantum properties of all the atoms in the molecule is a computationally difficult task, even for our supercomputers. But a quantum computer could do better because it operates using the

same quantum properties as the molecule it's trying to simulate ^[3]. So future large-scale quantum simulations for drug development could perhaps lead to treatments for diseases like Alzheimer's, which affects thousands of lives.

Challenges of bringing it to reality

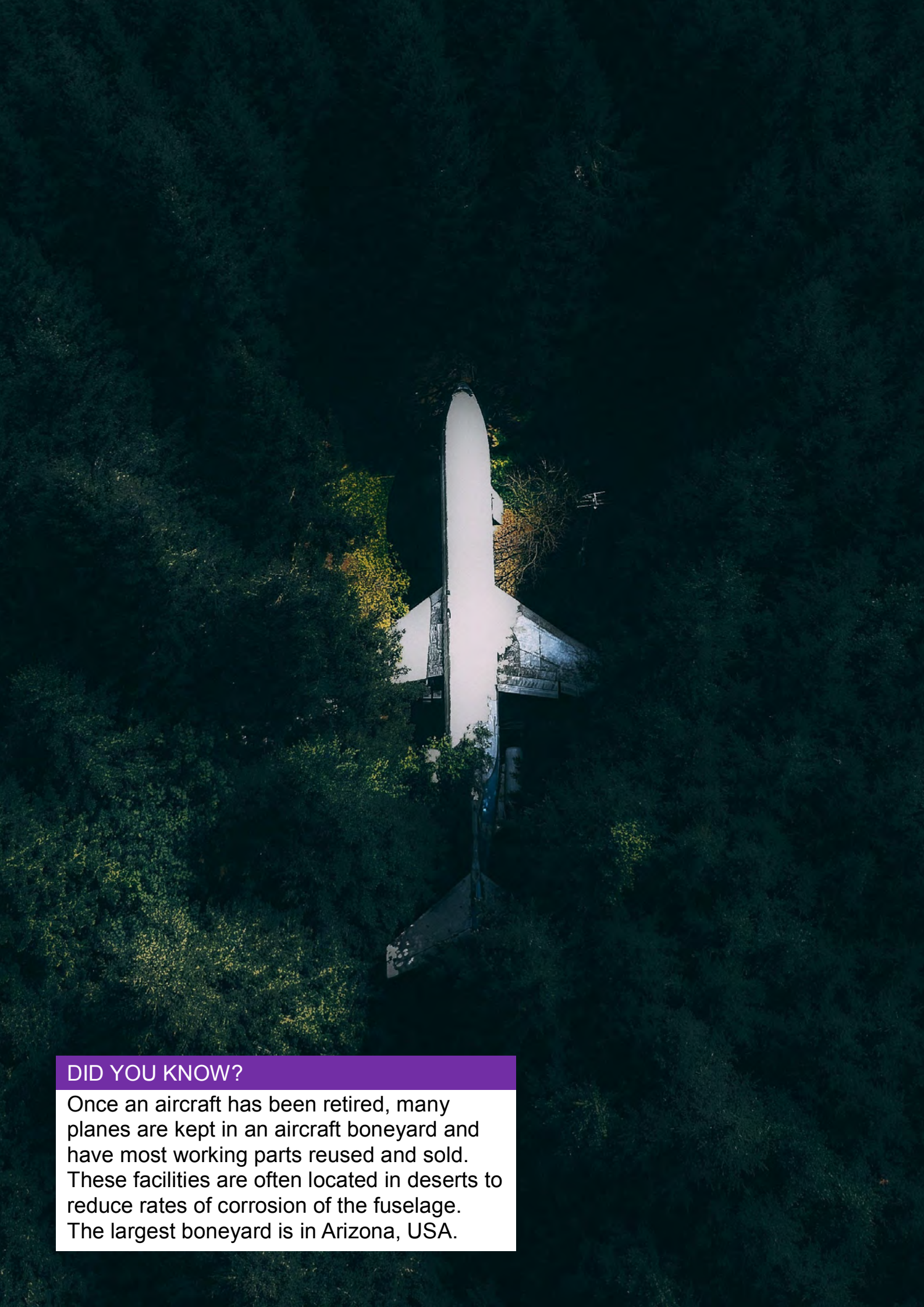
Quantum computers are extremely difficult to make, since for them to work, they need particles to act like qubits i.e., exhibit quantum behaviour and this is very problematic to achieve. This is because of decoherence, where qubits interact with their environment in ways that cause their quantum behaviour to decay and ultimately disappear and unfortunately their quantum state is very fragile. This means that the slightest change in temperature or vibration can lead to them breaking out of superposition before completing a process. To keep them in their quantum states, scientists have to store these computers at near absolute zero in supercooled fridges and vacuum chambers ^[9].

Conclusion

Quantum supremacy is where a quantum computer is created that is faster than the fastest classical computer and in only October 2020, Google announced that they have achieved Quantum Supremacy by creating a quantum computer that will process a certain task in 200 seconds and that the same task would take a state-of-the-art classical supercomputer approximately 10,000 years ^[10]. Despite this incredible achievement, Quantum computers still have many problems, as mentioned before they have to be stored at near absolute zero in a perfect vacuum ^[9] so it seems that it is years and years before all our computers are replaced with quantum technology, but it is important to note their potential and how much they can do for us.

Edited by Atharva Narkhede





DID YOU KNOW?

Once an aircraft has been retired, many planes are kept in an aircraft boneyard and have most working parts reused and sold. These facilities are often located in deserts to reduce rates of corrosion of the fuselage. The largest boneyard is in Arizona, USA.

Engineering Section

Marvel Nanotech

Nanotech used by superheroes p31

Autonomous Vehicles

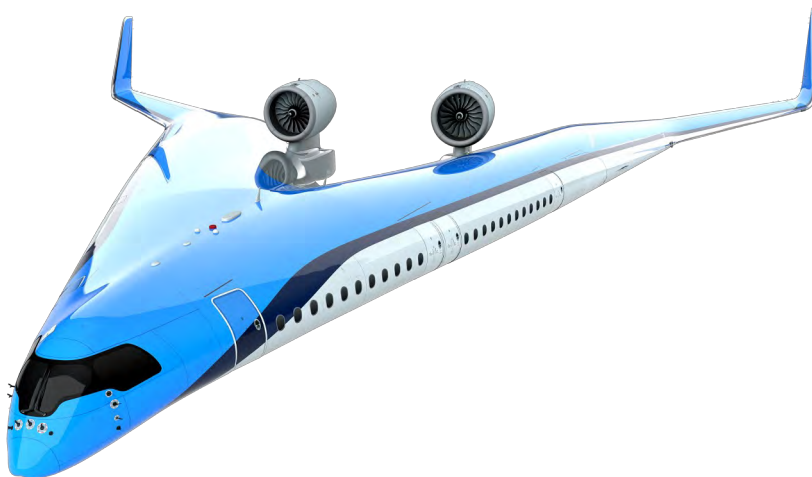
Dream come true? p33

Hydrogen or Electric?

Is one better? p34

Flash

Is it scientifically accurate? p35



The Flying-V

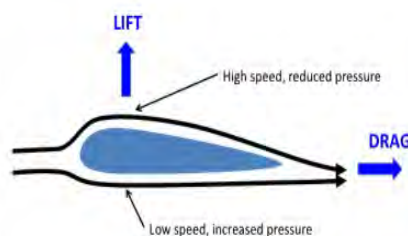
A new design for conventional aircraft?

By Atharva Narkhede (Y12)

How conventional planes fly and how the Flying-V flies differently:

Planes are able to fly when their aerofoil shaped wings generate lift exceeding their weight. How does this generate lift? Air travelling faster creates low pressure. Lift is generated when this aerofoil shape is introduced into the airflow, which induces pressure changes on either side of it – lower pressure above the wing and higher pressure below it.

The change in pressure acting on the area of the wing creates a resultant force in the



Cross-section of a conventional wing ^[4]

upward direction and lift with an angle of attack of 10 to 15° is generated ^[5].

This adheres to Bernoulli's principle, which states: as speed of the fluid increases, pressure decreases ^[6].

The Flying-V has a larger wing area than a conventional aircraft such as an Airbus A350, so when it pitches

upwards, it increases its angle of attack on take-off, so it generates more lift.

Design:

It is clear from the exterior that the V-shaped design will have the passenger cabin, cargo hold (where luggage is kept) and the fuel tanks all integrated into the wings ^[3]. KLM also promises that this state-of-the-art plane will be capable of accommodating up to 314 passengers. In order for it to be able to use existing airport facilities and infrastructure, engineers are ensuring that its length and wingspan are consistent with that of the Airbus A350 (an issue which Boeing 777-X engineers left unaccounted for and had to fix by creating a folding wing-tip mechanism).

Passenger comfort will be taken to the next level with many design ideas in consideration like group seats, flat beds, and lounge seats. Although, none of these have been confirmed by engineers, we know that the lightest possible materials will be used to achieve the kinds of aerodynamic efficiency that the aircraft proposes.

The materials used for the aircraft are very likely to be composites since the fuselages, wings, flaps, and other major components are



all comprised of composite materials in the latest aircrafts like the A350. Sandwiched versions of carbon fibre reinforced plastic (CRFP) are used in conventional aircraft, so it can be anticipated that the Flying-V will use these too^[7]. CRFPs are useful due to their high strength, low weight, and high corrosion resistance.

The Drag Coefficient and Link to Improved Efficiency:

This variable is used by engineers to help model the effect of all factors affecting the drag on a plane. It is calculated by rearranging the following equation for C_D :

Drag Force = $\frac{1}{2} \times$ Drag Coefficient \times Air Density \times Frontal Area \times Velocity²

$$F_D = \frac{1}{2} \times C_D \times \rho \times A \times v^2$$

The velocity and altitude of the plane during cruise are constant, the thrust equals the drag force, and the air density will also be constant at the same altitude. The frontal wing surface area of the Flying-V will be smaller compared to a typical wing since it is designed to have a smooth wing surface to reduce drag.

The drag encountered by the aircraft will be minimal since the whole body is incorporated into the wings and is all streamlined, thus minimising the frontal surface area in contact

with the air (which is the fluid). Drag is proportional to the dynamic pressure and the area on which it acts and therefore is said to be “a measure of the amount of dynamic pressure that gets converted to drag”^[6].

Consequently, it can be concluded that the Flying-V will experience less drag on average and so will have a lower drag coefficient in comparison to a conventional aircraft and this is desirable since an aircraft's efficiency and drag are negatively correlated.



Frontal view of the Flying-V^[8]

KLM claims that its Flying-V comes with a 20% fuel reduction^[3] purely from the aerodynamics of the aircraft and not from any new engine technology, although the most fuel-efficient jet engines will be fitted. Furthermore, with a higher efficiency, it can now be anticipated that the flight times will decrease, and its carbon footprint will be much lower, making it more environmentally than existing aircraft.

When can we expect to travel in this futuristic engineering feat? 2040. Indeed a long wait for aviation enthusiasts.

Edited by Mann Patira



THE SIMPLE SCIENCE OF FLIGHT by Hendrik Tennekes

Following a recommendation from the Cambridge Engineering Reading List and from Mr Carew-Robinson, I decided to approach this book. I was captivated immediately as it was said to be for aviation enthusiasts. It is informative, provides pleasant reading for anyone interested in aviation or is fascinated by flying—anyone who is an air traveller, bird spotter or dreams of learning to fly is easily inspired. After having read this book, I applied Tennekes' theory of flight from conventional modern commercial aircraft to that of the Flying V, a state-of-the-art concept!



It's Nanotech, You like It?

By Sanuka Gunawardena (Y12)



Iron Man Avengers Marvel ^[7]

The billionaire. The genius. The inventor. Tony Stark a.k.a. Iron Man has been a hugely successful hero in Marvel's MCU over the past decade. Now at iteration 85, it is easy to breeze over these world changing inventions that seem decades or even centuries ahead of our time, but have you ever wondered just how close we are? Well, pretty close, actually. In this article, I will explore one of Stark's most recognisable tech: his use of nanotechnology since Infinity War. Continue reading for not only an insight into the science and engineering behind nanotech in Stark's suit but explanations of some of the recent, exciting real-world innovations in this rapidly evolving field.

Nanotechnology helps solve the biggest problem facing modern technology, both in film and reality: fitting more into less. Since Avengers Infinity War, Iron Man has developed and used nanotech with the help of Wakandan technology. This unleashed a whole new type of

suit for Tony. He was able to use a reserve of billions of gold-titanium nanoparticles in his arc reactor to create shields, reconstruct broken helmets and suits, which seemingly emerged from nowhere. Other than this, Marvel has not disclosed much about Stark's nanotechnology, probably because this technology is novel and not yet fully understood.

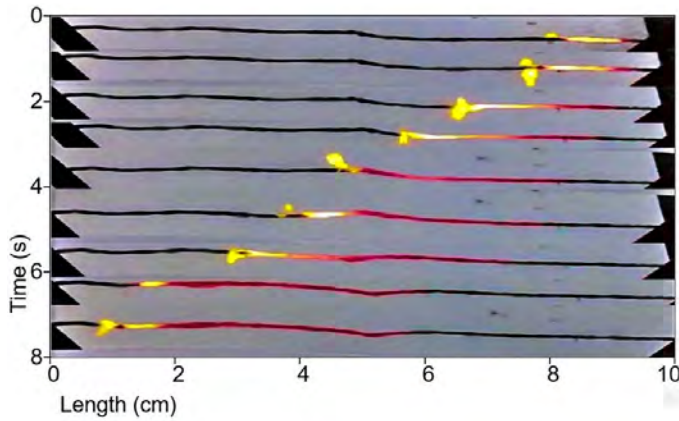
Nanotechnology's novelty, however, has not blocked many start-ups from success and the market is now worth \$60 billion annually. Before we get any further, it is important to note that nanotechnology covers small objects; less than 100 nm to be precise. That's about 100 times smaller than a human red blood cell!

You may already know that nanoparticles are used in sunscreens, either as titanium dioxide (TiO₂) or zinc oxide (ZnO) and that this new technology is used to waterproof t-shirts, shoes, and phones but these are just the beginning and recent innovations are looking, scarily at times, similar to Stark's own.

A new way of powering our tech:

If I asked you to name methods of generating electricity, you probably would have said fossil fuels, solar, wind, maybe even geothermal. One that I can almost guarantee did not come to your mind is thermopower. Yes, it sounds like a superhero power and it sure does feel like one! In 2011, researchers at MIT used nanotubes to generate electricity via the thermoelectric effect ^[1]. This is the conversion of a difference in temperature to voltage, allowing it to power devices. By covering the wire of carbon nanotubes in a combustible substance and lighting one end, causing a temperature gradient, the team was able to generate electricity ^[2]. The researchers used sucrose, or sugar, as the combustion agent rather than the harmful chemicals found in traditional lithium-ion cells in your phone. Currently, the cell can only be used to power small LEDs, but researchers pointed out that it took more than two decades for lithium-ion technology to be consumer-ready.





The researchers combusted the wire, made of nanotubes, like a fuse ^[2]

This technology, whilst in its infancy, has the potential to change the world of wearables and portables like never before. Phones, tools of great importance which we carry around with us everywhere, will see a surge in performance with more volume being dedicated to processing rather than battery. Not only this, but it brings us one step closer to building a functioning iron man suit. Since we first saw the billionaire soaring in his suit, one potential setback with our current technology has been its inability to store the enormous amounts of energy needed to power such suits. MIT's researchers may be close to solving that!

Nanotech armour:

Iron man would not be iron man, and certainly would not have survived all his combats throughout the years, without his armour. Traditional bullet proof vests like those commissioned to members of the armed forces use Kevlar, a material with a tensile strength 8 times greater than steel wire, to stop the bullet from penetrating and killing the wearer. This technology itself took decades of research into materials and is able to stop projectiles travelling at 1,800 mph. However, existing bulletproof clothing is not capable of spreading the impact force of the bullet enough to protect against blunt force trauma and damage to vital organs such as the heart. The solution: engineers have managed to use a composite of graphene, carbon nanotubes and traditional Kevlar to design a vest which is 90% better at preventing blunt force trauma and 50% better stab resistance ^[3], significantly reducing the chances of injury. The engineers settled for a beehive-like structure ^[4], held together by

strong covalent bonds, making them hundreds of times stronger than metals such as steel! These nanotubes are blended together to form nanofibers which allow for a light, durable and flexible vest that can protect against almost all projectiles and stabs. By using nanofibers, the researchers were able to considerably increase the elasticity of the material and its force absorption properties, helping the vests spread the impact across a greater area of the body ^[5]. The ability of nanotubes to be woven into nanofibers is due to the Van der Waals force. Graphene is used due to its high strength to weight ratio and its ability to almost 'self-heal'. The researchers have even plan to use a coating of this new material to protect satellites from micro-meteorites ^[6].

The improvements in protection stated above may not sound significant but it is important to note that this technology is still very new, and I strongly believe that we can expect, once larger companies and research institutions join this trend, armour that behaves similarly to Black Panther's Vibranium suit. Whilst this may not be the be all and end all, it is definitely a step in the right direction! Tony Stark's legacy has not only bought in billions of dollars of revenue for Marvel but has been a catalyst in inspiring thousands of engineers and scientists and has sparked a race to invent, improve and miniaturise incredibly useful technologies, such as nanotechnology, that are sure to improve quality of life around the world. It is just CGI in film, they say. Well, maybe one day you will be able to say otherwise. This is just the tip of the iceberg when it comes to what nanotech can do.

Edited by Atharva Narkhede



What Technology Will Autonomous Cars Use?

By Arya Narang (Y11)

Autonomous vehicles or self-driving cars are vehicles which are able to function by themselves with little to no human input. By the end of this decade, an astounding 10% of cars on the road could be autonomous ^[1]. This requires many different technologies in order to make these cars as safe as possible whilst also running at a similar or higher standard to normal vehicles. What technologies would be needed to meet these requirements and how will they operate to improve automation in driverless cars?

Firstly, autonomous vehicles will need highly reliable internet speeds and strong signals in order to keep up with live traffic, surroundings and provide entertainment. Therefore, 5G signals are almost a prerequisite to achieve this feat. One example where 5G would be needed is in an app or a server which shows all the information about live traffic to the most intricate details, such as whether a traffic light is green or red ^[2]. Imagine that you're travelling in a self-driving car and you approach a red light. In order for the vehicle to come to a halt, the car could be connected to the app or server monitoring live traffic information. Then the traffic light (which is connected to 5G broadband) would upload information onto the server stating that the light is red. Subsequently, this causes the approaching vehicle to receive this information almost instantly so as the car gets closer, it comes to a standstill. Once the light is green again, it will then forward this information to the server and when the vehicle receives this data, it then continues with its journey. In this example, if we were to use 4G, the task could be performed up to 100x slower which could pose a risk to the safety of the vehicle if it does not stop in time ^[3].

Moreover, 5G is necessary to interact with the rest of the surroundings, not merely for communicating with traffic lights. The Internet of Things (IoT) is a "system of interrelated, internet-connected objects that are able to collect and transfer data over a wireless network without human intervention" according to Aeriis ^[4]. In simpler terms, it refers to different devices communicating with each other via the internet; this could provide endless possibilities and opportunities to improve the efficiency of autonomous vehicles. Neighbouring vehicles could communicate with each other to improve safety. For instance, if there is a hazard on the road ahead which is detected by a particular vehicle, that

vehicle can then alert the car behind them of the looming danger so the car begins to adapt to the scenario promptly ^[4]. Despite the fascinating possibilities provided, a higher bandwidth allows for hackers to access data more easily because more information can be transferred in a shorter period of time so there are mild risks involved with using more advanced technology ^[5].

Furthermore, driverless cars require exceptionally robust sensors to ensure no accidents occur during journeys. As of yet, it has proven difficult to perfect the sensors of autonomous vehicles as they can still make many errors, such as the inability to differentiate between a traffic light and a black lamppost. Currently, self-driving vehicles rely on 4 types of sensors: lidar (light detection and ranging), video cameras, ultrasonic sensors and radars – however, all of these sensors have their flaws ^[6]. Video cameras are ineffective when there is poor visibility ^[6]. Lidar is limited by its short range and by heavy rain ^[6]. Radars are able to sense the relative speed of other vehicles and cyclists but they are restricted by their short range so as to ultrasonic sensors ^[6]. Thus, companies such as AdaSky have developed far-infrared cameras (thermal cameras) which can sense heat and this can distinguish people, animals and other surroundings ^[6].

These sensors are considerably more powerful due to their ability to detect objects over 100 metres away, even in adverse weather conditions ^[6]. Many companies are now considering integrating these sensors into autonomous vehicles, as most experts believe that far-range infrared sensors are the key to ensuring that self-driving cars are as safe and reliable as possible.

In conclusion, further research and technological development is needed before fully autonomous vehicles are available on the market. 5G broadband is one momentous step into making this possible, by enabling much faster connectivity. A breakthrough in sensors has been made, thanks to the invention of far-infrared sensors but more time is required until they become commercially available. It would appear that we are finally approaching the finish line as pioneers including Elon Musk strive to reach the end, meaning it is only a matter of time before driverless cars on the road become a norm.

Edited by Aditya Jain

Tesla Cars are renowned for their self-driving capabilities



Is Hydrogen a better Alternative than Electric for the Future of the Automotive Industry?

By Matteo Cascini (Y11)

“In order to have clean air in cities, you have to go electric,” Elon Musk, the CEO of Tesla, once stated in an interview with the CNN when questioned about the future of the automotive industry in China.^[1] However, whilst Tesla is currently leading in the production of sustainable energy vehicles, is electric really the future of automobiles?

Electric cars first became commercially available in 1996 when General Motors released the EV1. Indeed, over 1000 of these cars were mass-produced as part of the programme, but by 2003, the program was axed by GM due to the high development costs and the low 50-mile range. Consequently, most of these cars were removed from the roads and destroyed^[2]. Since then, however, new companies like Tesla, as well as already well-known car companies such as BMW, Audi and Jaguar, have begun to develop electric cars to replace the heavily polluting petrol and diesel cars on our roads today. Tesla began their journey in the industry in 2008 by releasing the Roadster – a luxury, sports car which used lithium-ion cells to power its reported 3.7s 0-60mph time and 244 mile range^[3]. This was an unprecedented development in the industry and helped to fuel the company's future as they later released the Model S (2012), X (2015), 3 (2017) and Y (2019) in addition to the Cybertruck and Semi-truck. Currently, Tesla has a global market share of roughly 18% amongst electric car producers, with the Model 3 being the most sold electric car ever (over 800,000 sold as of December 2020). Despite this significant development within recent years, as of 2019, electric car sales only accounted for 2.6% of global car sales^[4].

The main downfalls of electric vehicles currently include slow charging times, low range on a single charge and the lack of charging infrastructure across the globe. The recently announced Tesla Model S Plaid+, the EV with the longest range in the current market, only has a marketed range of around 520 miles. Whilst this is long enough for the average daily commute, it is

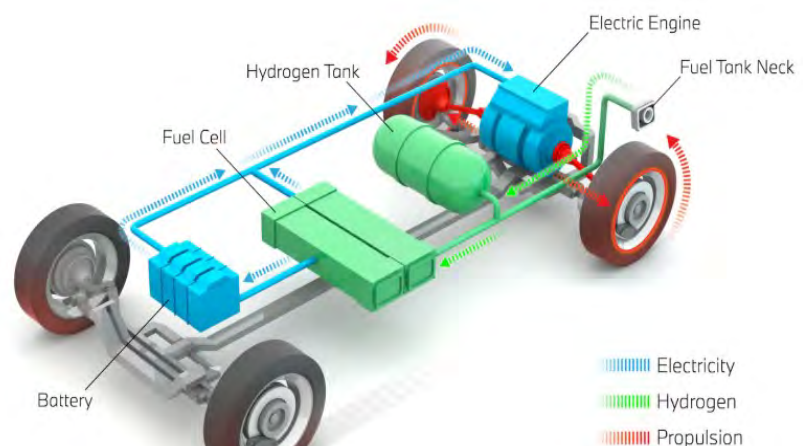
much shorter than the convenience of having over 800 miles of range with a more conventional petrol or diesel car. Furthermore, on average it takes 45 minutes to fully charge with the use of a 250kW supercharger^[5] whereas the regular 7kW charger can take over 8 hours to fill the car's battery. This is therefore a major disadvantage which the hydrogen-based car may be able to overcome.

In 1959, Francis Bacon, an engineer at Cambridge University, created and demonstrated the first hydrogen fuel-cell which could be used for an automotive. A fuel-cell vehicle (FCV) produces energy through the exothermic reaction of compressed hydrogen gas and oxygen in a process known as reverse electrolysis – a process where hydrogen gas is split into protons and electrons at the anode with a platinum catalyst before reacting with oxygen at the cathode, therefore creating an electrical current^[6]. This results only in the production of water and no other harmful gases, including carbon dioxide as well as nitrogen oxides or sulfur dioxide due to the low running temperature of the hydrogen fuel cell. Indeed, the process which occurs in the fuel cell is very efficient as there are no moving parts, like in an

internal combustion engine, and energy isn't lost as thermal energy to the surroundings. Not too dissimilar to most EVs, FCVs are currently limited to just over 300 miles of range^[7], but this can be scaled up in the future with larger hydrogen tanks. However, the largest current downfall of this zero-emission technology is the significant lack of refuelling stations – as of September 2020, there are only 80 stations in Germany and only 40 across the entire USA in contrast to over 20 000 EV charging stations^[8].

Other potential shortfalls of the hydrogen car include the potential carbon footprint of producing the required hydrogen gas and the possibly fatal dangers of pressurised flammable hydrogen, however Toyota claim that the low density of hydrogen gas will make it even safer. On the whole, both electric and hydrogen reduce pollution at point of consumption (both air and noise), and increase passenger comfort creating a luxury-feel. The future is exciting!

Edited by Aditya Jain



How the hydrogen and electric power systems co-operate



How Scientifically Accurate Is The Flash?

By Folaju George (Y11)

The Flash. The Scarlet Speedster. According to himself, the fastest man alive. For the purpose of this analysis, I will discuss the most popular Flash – Barry Allen. I will focus on the Barry Allen presented in the televised CW Arrowverse show, as his abilities and feats are most commonly displayed from this source.

To begin, I will discuss the manner in which this iteration of the Flash obtained his powers. In Season One, Episode One, it is shown that during the particle acceleration explosion, a stray bolt of lightning struck CSI Barry Allen, thrusting him into a cabinet of miscellaneous chemicals. The explosion formed a wave of dark matter which flooded the city, either killing or empowering those it made contact with – including Barry Allen. While his body was ionised, the content of the chemicals spilling on his skin connected him to the Speed Force – the otherworldly source of his extraordinary power. Unfortunately, this is astonishingly unrealistic. Although the design of the Star Laboratories particle accelerator appears similar to what one may find on a Google search, the physics behind this are unreliable and frequently change over the course of the series (most commonly in order to make things convenient for the writers and the plot). Additionally, so-called ‘dark matter’, which is the generic source of power for the meta-humans (as called in the series) is currently an abstract concept in physics and not a confirmed energy source which can be generated or concen-

trated. Furthermore, the most likely impact this will have on the human body would be to facilitate unimaginable and, likely, irreversible damage, though this is similarly unconfirmed as very little is known about the properties of dark matter. With regard to the lightning, the strike in question undeniably should have been fatal. Considering at the time he was holding on to large metal chains, which are good conductors of electricity, assuming they were made of stainless or galvanised steel, this strike should have been even more impactful on his body. A typical lightning bolt holds millions of volts of electricity and up to one billion joules of energy – the human body most certainly cannot withstand this. To add insult to injury, the various chemicals would likely be corrosive or otherwise harmful to the skin – such chemicals include ninhydrin, silver nitrate or cyanoacrylate which are often found in CSI laboratories similar to that of Barry Allen; he should have been dead and disfigured. But rather, he was knocked into a coma, which lasted nine months and it was here the first signs of his power were put on display.

The Flash is popular for having

some of the most amazing and fantastical powers and abilities across DC Comics, as well as in the Arrowverse, in relation to this televised iteration. For the purpose of simplicity and being concise, I will only discuss the two of the most frequently used powers on display across the series. These are superhuman speed and phasing. Clearly, superhuman speeds are physically unattainable. Certain episodes of The Flash show Barry Allen being able to move at speeds which narrowly miss the speed of light – this feat is first displayed on Season Four, Episode 15, titled “Enter Flashtime”. This title references the ability The Flash possesses to move at speeds so great the people around appear completely still by comparison. Though unconfirmed, the theory that this is nearly the speed of light comes from the YouTube channel ‘Comic Books vs The World’ in a video titled “The Flash Season 4: Barry Outruns a Nuke Calculated!”. I greatly recommend checking this out as the process by which he comes to this conclusion is a highly interesting watch. To move at even a fraction of this speed is, without a doubt, far too much for a human to withstand. To understand this, I will apply the use of G-force calculations. A nor-



mal, human can only withstand no more than 9G's of vertical acceleration, and even so for only a few seconds. When undergoing an acceleration of as high as 9 G's, your body feels nine times heavier than usual, blood rushes to the feet, and the heart can't pump hard enough to bring this heavier blood to the brain – a sustained period of this condition is unmistakably lethal. As for longitudinal G-force, Eli Beeding experience over 80 G's of force extremely momentarily and survived, demonstrating how much force the body can withstand. However, if a person were to accelerate to the 99.9% of the speed light in just 1 second, you would experience a G-force of approximately 30,000,000 G's. Frankly, that is insane. The Flash is shown doing these things and more, making the likelihood of such a man existing unquestionably impossible; to move at such speeds will irrefutably kill a person – they will vaporise before *increasing your speed a little bit is gonna let you do? Barry?* Generally, this means that simply moving faster does not enable someone to pass through the tightly packed atoms and particles of solid objects as the Flash suggests. It's simply not possible (regardless of how cool it looks on TV).

Now equipped with the facts required to understand how unrealistic this iteration of The Flash is and how unattainable his powers are, I will describe some further feats to cement this and further emphasise

that fact. One particularly noteworthy feat is The Flash's ability to produce tornadoes simply by running at high speeds in a circular motion. In reality, tornadoes form typically during thunderstorms when alternating masses of air meet, creating atmospheric instability. Ideal conditions require moisture in the air and high wind speeds. However, The Flash was able to form tornadoes from nothing which is highly questionable. Alternatively, one could consider the physics of stirring, by which (based on observational assumptions that the radius at which The Flash ran was 5 metres and his mass was 75kg). This would mean that, assuming he was running at Mach 2 as he was using his powers, it would take around 6 million newtons of centripetal force – even 40 MPa concrete would not support this!

Another thing The Flash was able to do that opposes real-life scientific laws was throw a bolt of lightning at himself. Upon obtaining a new suit, updated with a plethora of technological improvements and new features, which posed a great issue as he had come up against an adversary with the ability to manipulate technology to serve his will. This included The Flash's suit, which was set to self-destruct. It was decided, in that episode (Season 4, Episode 2) that the solution was to short-circuit the suit by throwing a lightning bolt at himself. The 'lightning' that The Flash is able to 'throw' is

not real lightning, but rather the physical manifestation of the energy coursing through his veins, which gives him his power. This is, generally, confusing and evidently unrealistic, as is most aspects of the existence of The Flash. However, the most significantly unrealistic detail of The Flash is his recurring ability to run back in time. This ability comes from being able to run at Mach 2, which is twice the speed of sound – I don't need to explain how absurd this is. By this erroneous logic, NASA's solar probe is equipped with enough speed to travel as far back as the Stone Age, reaching speeds one hundred and thirty-nine times greater than the mere Mach 2 required (though this is still very fast and it is definitely impossible to move at such a speed).

Perhaps there are some superheroes whose abilities are replicable, with realistic feats and sources of power – however, The Flash is most certainly not one of them. Though, blink, and you just might miss him.

Edited by Aditya Jain





DID YOU KNOW?

If we collected information about how far everyone thought they lived from the school in meters, and took the leading digit (i.e. the 2 in 20,000) then we would find that the number 1 appears the most frequently as the leading digit (30% of the time), which is counterintuitive as you would expect each number to appear 11.1 % of time. This is due to Benford's Law of anomalous numbers, where data occupies multiple orders of magnitude.

Maths Section

Euler's Number

The magic behind this transcendental number **p40**

Doomsday Argument

Maths of the apocalypse **p42**

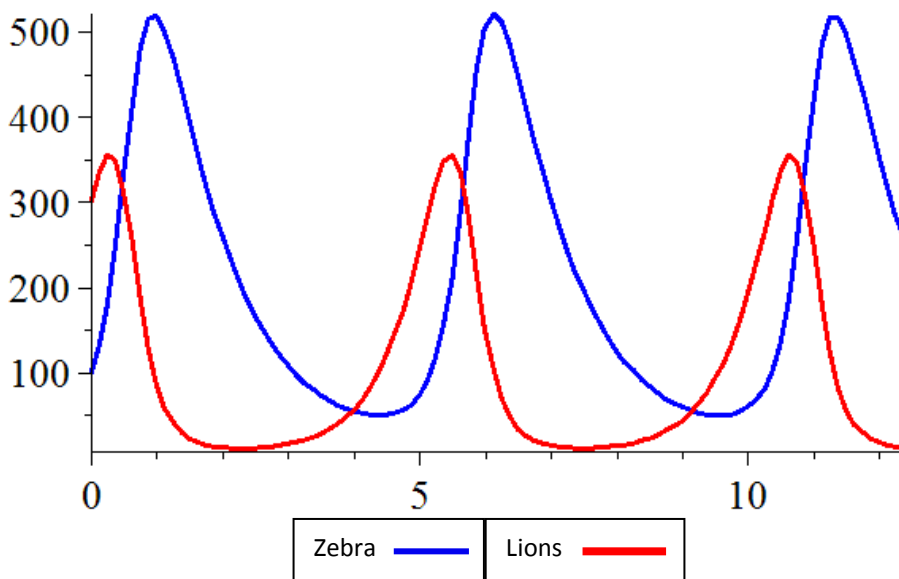
Prime Tuples

Modular arithmetic with primes **p44**

Infinite Series

How can they inform us about the real world? **p46**

Lotka Volterra Equation Plot



of volatility $(\gamma = \frac{d^2V}{ds^2})$. The $-\theta$, captures *ceteris paribus* (all other things being equal) and the generic fall in prices of goods and services with time. The right hand side explains that with respect to interest, the value of the share “V” should equal the start price “S” times the average increase in price “ δ ”. This is a surprising model to predict the future share market as with a few changes to the initial equation (such as starting price and volatility), the price of most shares can be predicted by solving for “V”.

This equation was critical to massive economic growth, since it allowed investors to invest in derivatives: the probability that a share price will rise; not the share itself. By 2007, the exchange of derivatives was approximated to one billion dollars a year ^[4]. However the predicting power of the equation was abused as the derivatives slowly became a product in themselves. This may not seem a problem until you realise that it's an unattainable prophecy: if the value “V” for a share is predicted to rise, then the value of the derivatives also rises. This then deters people from investing in that particular share, choosing to invest in the derivatives. This leads to a fall in price “V” and makes the prediction incorrect – given the assumptions made during the calculation of the prediction have now changed. This sets all who invested in the derivatives and the share up for a loss.

Although the model bases itself on multiple assumptions and approximations, it is near impossible to single out the impact of one individual on the share price in a system as complex as the share market with its millions of economic agents. With a model like this, it is important to remember that it only gives an indication to the price of a share, and this is better than no information at all for an investor.

Accuracy of Modelling

How accurate is mathematical modelling?

By Divy Dayal (Y12)

Modelling is a fundamental application of maths, given that mathematical instruments can turn a real-world situation into a predictable manageable model. Modelling provides data for future share prices, zebra population and even the weather. However, models cannot always be accurate and reflect the real world. It is incredibly difficult to decide what the most significant characteristics in a scenario are, and therefore assign a mathematical component to them in a model.

Black-Scholes Equation

Friedrich Hayek, while accepting his Nobel Prize for Economics, claimed how economists' confidence in their modelling “may have deplorable effects,” given how complex the real world truly is

^[1]. The heart of the modelling problem lies in the number of variables that need to be accounted for; Professor Michael Berry claimed that in order to predict the 16th shot in a billiards game, you need to account for the gravitational force of the people moving near the table and to predict the 56th, you would need to include the effect of every single particle in the universe ^[2]. One famous effort to simplify and mathematicise the market of the future shares is the Black-Scholes equation ^[3]:

$$\frac{1}{2}S^2\sigma^2\gamma - \theta = r(V - \delta S)$$

Once getting past the Greek letters, this complex equation can be simplified, where “ $\frac{1}{2}S^2\sigma^2\gamma$ ” the volatility of the share “ σ^2 ” adjusted for the change in the rate



Lotka-Volterra Equations

Another famous model that predicts populations of animals is the set of Lotka-Volterra Equations:

$$\frac{dy}{dt} = bxy - mx \qquad \frac{dx}{dt} = rx - axy$$

These may seem initially daunting but they are very intuitive: “ r ” is the birth-rate of a zebra and “ x ” is the number of existing zebras. “ a ” is how much lions eat and “ y ” is the number of lions. That means the first equation shows a change in zebra population, which is the number of zebras being born minus the number of zebras being eaten by lions. The second equation explores the change in lion population where “ b ” is the birth rate and “ m ” is their death rate. This simple model can be used to plot the change in each of their populations and can be used to make predictions given initial data.

$$\frac{dx}{dt} = rx$$

$$\Rightarrow \frac{dx}{x} = r dt$$

$$\Rightarrow \int x^{-1} dx = \int r dt$$

$$\Rightarrow \ln(x) + c = rt + c$$

$$\Rightarrow \ln(x) = rt + c$$

$$\Rightarrow x = Ae^{rt}$$

where A is the initial amount ($t = 0$)

If you were to imagine a savannah with no lions and only zebras e.g. a zoo then the first equation can be simplified to:

$$\frac{dy}{dx} = rx \Rightarrow x = e^{rt} + c$$

This would lead us to the conclusion that zebra population will grow exponentially given no predators and this is where the model fails. The model doesn't account for different predators, the season, meteorological events, the size of the animal and various other biotic and abiotic factors which influence populations. Ultimately, it is more a simplification to explain population growth and decline than an effort to pin down the exact zebra population in June 2021. It should be noted that when all factors are limitless, then population growth does in fact occur as predicted, as noted in Australia in 1860s' [5] when early colonists brought rabbits who had no natural predators in this vast continent. The rabbit population soared until 3000 km “rabbit walls” were put across Australia to prevent this unrestricted growth throughout the

continent.

In summary, forecasting is just an attempt to compute the complex world we live in into an understandable, manageable set of equations and code. The results of forecasting are always approximations, and depend entirely on the accuracy of the assumptions and data inputs. Where most forecasts fail is where there is over manipulation of data, and bias of those forecasting [1]. It often leads to a “fudge factor” within models to make the equations work with data attained; even Albert Einstein was guilty of using the “cosmological constant” to make his formulae work [6]. The next time it doesn't rain according to the forecast, spare a thought for the weatherman guessing the weather where you live.

Edited by Atharva Narkhede



Australian Rabbit Wall [7]



Wond-e-rs of 'e'

By Adam Ali (Y12)

Often in Maths we use letters to denote certain constants. More common ones include pi (π) or phi (Φ – aka the golden ratio).

However, one underappreciated letter you may not of heard of is 'e', or Euler's Number; and even if you have heard of it, you probably didn't realise how it uncannily appears in everyday life.

Leonhard Euler was an influential Swiss mathematician who contributed towards many branches of maths, including infinitesimal calculus and graph theory, as well as a range of other developments in physics, astronomy, geography, logic and engineering [1]. But more importantly, Euler was the one to develop the mathematical constant 'e'. But how exactly was it discovered?

"e" is equivalent to 2.718... or:

This means as "n" goes up to infinity

 $\lim_{n \rightarrow \infty} \left(1 + \frac{1}{n}\right)^n = e$

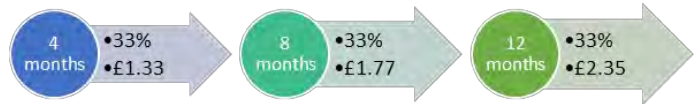
This is just one way of calculating 'e'. You can try this with your calculator – put in any number as "n", and you will get an approximation of 'e'. Notice that the larger "n" is, the closer you get to 'e' (2.718).

Appearances in Economics

Imagine you put £1 into a bank account that pays out 100% interest after one year, but you chose how often it compounds. For example, if you choose to compound every four months, you'll

be left with £2.35 in your account after one year.

But if you choose to compound every second, or a millisecond, or even smaller still a nanosecond (i.e. as time approaches $1/\infty$, it gets closer and closer to zero), you will be left with £'e' at the end of the year [2].



Economics application

Appearances in Statistics

Ever thought about playing the lottery? Let's look at the odds. Let P (winning the lottery) be one in one million ($1/1000000$). What is P (losing every time) if you play the lottery 1 million times?

Well the chance of failing is one minus the probability ($1 - 1/1000000$). Therefore, the chance of failing on your first go and your second go and your third go and so on is:

Look familiar? This equation right here is almost the same as the equation at the start of the article, except it is negative instead of positive, which is why you get the reciprocal of 'e'.

$$\left(1 - \frac{1}{1000000}\right)^{1000000} \approx \frac{1}{e} \approx 36.8\% [3] \text{ or as a more general form: } \left(1 - \frac{1}{n}\right)^n = \frac{1}{e}$$

Appearances in Mechanics

You'll soon learn that the derivative of displacement is velocity and the derivative of velocity is acceleration (i.e. for a displacement time graph, the



gradient is velocity, and for a velocity time graph, the gradient is acceleration).

In A-level Maths, we also touch upon the fact that the derivative, or the gradient at a certain point, of $y = e^x$ is also e^x . This means that if you were in a rocket, whose position was determined by

$$p(t) = e$$

where “t” is time, your position equals your instantaneous velocity equals your acceleration, because the derivative of $y = e^x$ is also e^x . So in 1 second, you would have travelled ‘e’ meters, be at a speed of ‘e’ meters per second, and have an acceleration of ‘e’ meters per second squared ^[4]!

Appearances in Pure Mathematics

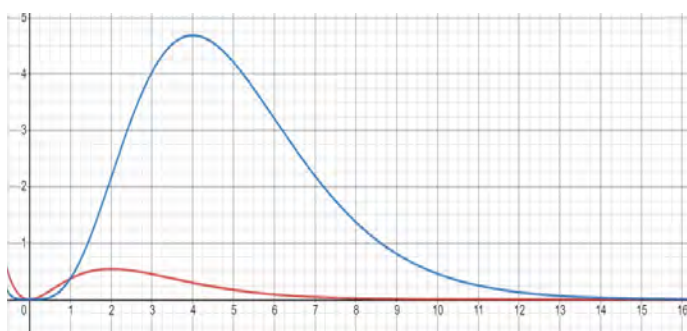
Perhaps the most intriguing appearances is in this area of maths, so I’ve saved the best for last. One notable application of ‘e’ is the “Gamma Function”. This is where we delve into the realms of A-level Maths, so you’ll have to bear with me here:

The blue line is $y = x^4 e^{-x}$, and the red line’s equation is $y = x^2 e^{-x}$. The area under the blue graph, between $x=0$ and $x=\infty$, is 4 factorial, and the area under

the red graph between the same bounds is equal to 2 factorial. In other words, if the power of ‘x’ is equal to ‘z’, the area under the graph is ‘z’ factorial! This is what’s known as the Gamma Function, and has many complex uses in fluid dynamics, astrophysics ^[5,6].

To conclude, ‘e’ appears more often than you think it does in life, and it goes to show that Maths actually has unique connections to the world that no other subject has. I hope this article has also enlightened you to the fact that there are other great mathematicians out there; so the next time you’re learning about Pythagoras and what a great mathematician he was, don’t forget Euler and may other influential mathematical thinkers.

Edited by Atharva Narkhede



$$y = x^4 e^{-x} \quad \text{and} \quad y = x^2 e^{-x}$$



“Humanity will be extinct in 15,000 years”

The Doomsday Argument: Fact or Fiction?

By Kinshuk Jain (Y12)

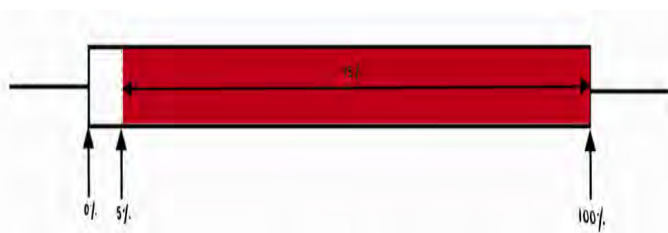
The Doomsday Argument (DA), proposed by theoretical physicist Brandon Carter, makes use of statistical theories to predict how many more humans will be born before extinction.

$$f = \frac{h}{H}$$

How does it work?

The first part of the argument estimates how many humans will ever be born, provided that humanity does not move beyond Earth. It makes use of the following terms:

- ‘ H ’ is the number of humans born from the start of humanity until its extinction
- ‘ h ’ is the birth rank (since the start of humanity) of any human
- ‘ f ’ is your fractional position along humanity’s timeline, defined as:



The last 95% of humans ever to be born

We assume that ‘ f ’ is uniformly distributed in the interval $(0, 1]$. So, we can say with 95% confidence that we are within the last 95% of humans to be born^[1] (red region):

$$f > 0.05 \Rightarrow \frac{h}{H} > 0.05 \Rightarrow 20h > H$$

Given that the most recent estimate of ‘ h ’ is in the region of 100 billion, we can say that:

$$H < 20 \times (1 \times 10^{11}) \Rightarrow H < 2 \times 10^{12}$$

That is, the total number of humans that will ever be born is 95% likely to be under two trillion.

The argument now considers the possibility of humanity expanding beyond Earth, which gives two possibilities for the future:

- Doom soon: the human race does not expand beyond Earth, which means the maximum number of humans to be born will be two trillion.
- Doom late: The human race spreads across the Milky Way, which means the total number of humans to be born will be in



excess of one quintillion (10^{18})

Suppose that a box containing numbered balls is placed in front of you. You are told that the box either contains ten balls (numbered one to ten) or 100 balls (numbered one to hundred), and you must decide which you think is the case. To help you, you will be able to pick out one ball only and see the number. If the ball you pick out has the number six written on it, the most logical choice would be to say the box contains ten balls – the probability of picking out six if there are ten balls is $1/10$, whereas, if the box contains 100 balls, the probability is $1/100$ ^[2,3].

We can now apply this analogy to the human race. Suppose that each ball represents a human, and the total number of balls is the total number of humans that will ever be born – 2×10^{12} (doom soon) or 10^{18} (doom late) ^[4]. The number on the ball represents the birth rank of each human, and the ball that is withdrawn is you (your birth rank is around 100 billion). This uses the self-sampling assumption (SSA) – “[reasoning] as if [you] are randomly selected from the set of all actually existent observers” ^[5].

Applying the same logic as in the ball scenario, what is more likely – that you are the 100 billionth person out of two trillion, or that you are the 100 billionth person out of one quintillion? The DA, therefore, reasons that it is extremely unlikely that the number of humans born will exceed two trillion. If we assume that the world population levels out at ten billion, with a standard life expectancy of 80 years, then the 1900 billion humans that remain to be born will be born within 15,200 years.

Problems

The first major objection is the self-indication assumption (SIA), which states that the probability of our existence actually increases with the total number of humans that will exist. For example, if there is a room with ten people in it, and a stadium with 1000 people, you are far more likely to be in the stadium than in the room. In the same way, the fact that we exist is testament to the idea that the total number of humans to exist is very high ^[6, 7].

The DA also makes a variety of assumptions, which are often the ground for rebuttals. For example, the argument uses the principle of indifference, which states that all outcomes should be assigned equal probabilities if we do not have any

information about which will occur. This is why ‘*f*’ is given a uniform distribution, but this is clearly an assumption that has no clear reasoning behind it.

Furthermore, the DA makes the assumption that we are not within the first 5% of humans to be born – but what if we are? Once again, there is no clear reasoning behind this assumption.

An example of the DA’s logic failing comes from the self-referencing rebuttal: using his own logic, when Carter came up with the theory, there was a 95% chance that he was within the last 95% of people to understand the theory, meaning a maximum of 20 people would understand the argument and therefore it is trivial, and should be ignored ^[1].

The bottom line

On the face of it, the Doomsday Argument appears to be a sound theory. However, digging deeper, it is easy to see that it is over-simplistic and simply makes too many seemingly unfounded assumptions. Whether you accept the DA also depends on whether you lean towards the SSA or the SIA. To me, the SIA makes a world of sense, whilst the SSA is, in comparison, somewhat questionable.

In conclusion, fear not! Humanity will continue to thrive for a while yet.

Edited by Atharva Narkhede



Prime Tuples

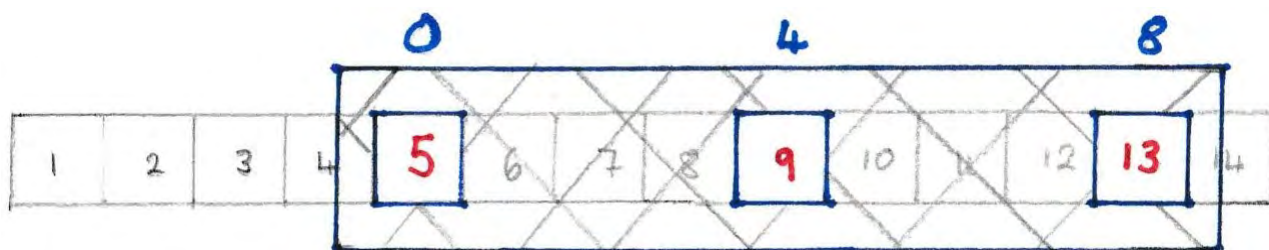
By Syed Shah (Y12)

Prime numbers have always fascinated mathematicians. They might seem to be the least interesting of all integers, given that they have the fewest factors, but it turns out that they reveal profound connections between parts of maths, and have many mysterious properties of their own. In this article, we introduce the Hardy-Littlewood Prime Tuple Conjecture, which is best known as one of its special cases: the elusive Twin Prime Conjecture.

Sets and the Punch-Card Analogy

First, some background knowledge. Consider the set $\{0, 4, 8\}$. In 'Closing the Gap', Vicky Neale represents such a set using a punch-card with holes at 0, 4, and 8^[1]. We can slide this punch-card along the number line and observe which numbers are visible ("the visible set").

For example, if we start the punch-card at 5, then the visible set is $\{5, 9, 13\}$, as shown in the figure.



Punch-card with the number line as the punch-tape^[2]

Admissible Sets and Modular Arithmetic

The problem with the set $\{0, 4, 8\}$ is that no matter where we slide its punch-card to, one of the visible numbers will always be a multiple of 3.

We say that a set is *inadmissible* if, for any prime, the visible set always contains a multiple of that prime.

On the other hand, admissible sets can conceivably have infinitely many positions where the visible set is all prime. However, it is not guaranteed.

We can determine whether a set is admissible by finding the remainders after dividing each element by every prime. Below, we do this for the prime 3 and the set $\{0, 4, 8\}$.

$$0 \equiv 0 \pmod{3}, 4 \equiv 1 \pmod{3}, 8 \equiv 2 \pmod{3}$$

If two numbers a and b have the same remainder r after being divided by some number

m , then we say that $a \equiv b \pmod{m}$. " a is congruent to b modulo m ." (This is equivalent to saying that m divides $a - b$.)

From this, we can deduce that $a \equiv b \equiv r \pmod{m}$.

Notice that the possible remainders after division by 3 are 0, 1, and 2 and that these are all found (as a congruent integer \pmod{m}) above in $\{0, 4, 8\}$.

The rules of modular arithmetic dictate that if $a \equiv b \pmod{m}$ and $c \equiv d \pmod{m}$, then $a + c \equiv b + d \pmod{m}$. In other words, we can add remainders when adding two numbers together.

Another way to think of the visible set is as equal to $\{n + h_1, n + h_2, \dots, n + h_k\}$, where n is the "position" along the number line, h_1 is the first element of the "punch-card" set and h_k is the last.

According to the rule above, regardless of the remainder of $n/3$, one of the elements in our



visible set will have a remainder of 0 when divided by 3. This element will therefore be a multiple of 3 and hence not prime, (unless it is 3 itself).

An example of an admissible set is {0, 4, 6}. For every prime p , there is always at least one remainder after division by p which is not found in the set.

$$0 \equiv 0 \pmod{2}, 4 \equiv 0 \pmod{2}, 6 \equiv 0 \pmod{2}$$

The possible remainder 1 does not appear.

$$0 \equiv 0 \pmod{3}, 4 \equiv 1 \pmod{3}, 6 \equiv 0 \pmod{3}$$

The possible remainder 2 does not appear.

You may have noticed that we have not tested every prime, (of which there are infinitely many!). If we think about it, we only need to test all the primes less than or equal to the width of the set. (The width is the number of elements.) This is because, for an integer m , there are only m possible remainders after division by m , so it is impossible for a set smaller than m to contain all of them.

Hardy-Littlewood Prime Tuple Conjecture

Before we said that, given an admissible set, it is not guaranteed we will find a visible set with all primes, but the Hardy-Littlewood Prime Tuple Conjecture predicts not only that there will be one such visible set, but that there will be infinitely many others as well.

In terms of the punch-card analogy, this means there are infinitely many positions to

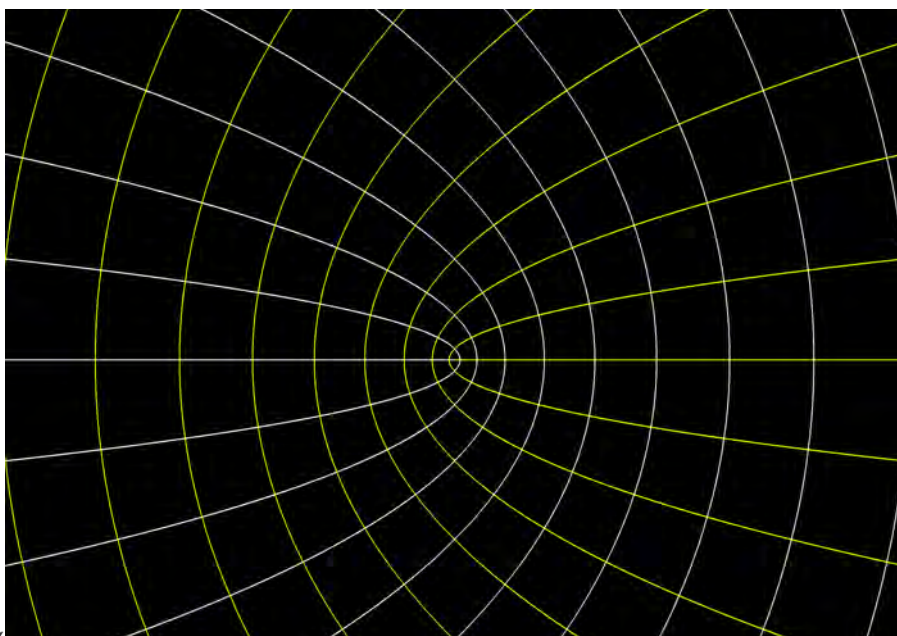
which we can slide the punch-card along the number line, such that all the visible numbers are prime.

The Twin Primes Conjecture is a special case of the Prime Tuple Conjecture where the admissible set is {0,2}. In other words, it predicts that there are infinitely many primes that are two away from an adjacent prime.

Much of the work towards proving this has involved finding narrower and smaller admissible sets. ("Narrower" means the set spans a smaller region of the number line, while "smaller" means the set has fewer elements in it.)

The first breakthrough was in 2013 when Yitang Zhang proved that there are infinitely many primes at most k away from each other, where $k < 70,000,000$ [3]. Zhang's focus was (evidently) not on finding the smallest bound for prime gaps, but rather on the finding of a *bound* itself. Since then, the Polymath8 project and the work of Terence Tao and James Maynard have been able to bring that down to 246. Assuming a strong version of the Elliot-Halberstam conjecture (currently unproven), the project has shown that this value is just 6 [4,5].

Edited by Mann Patira



The function $f : \mathbb{C} \rightarrow \mathbb{C}$, $f(z) = z^2$ represented as a transformation of the complex plane. The yellow lines were originally horizontal, while the white lines were originally vertical. Do you notice how the lines still meet at right angles?



What do Infinite Series tell us about the Real World?

By Karun Kirubananthan (Y12)

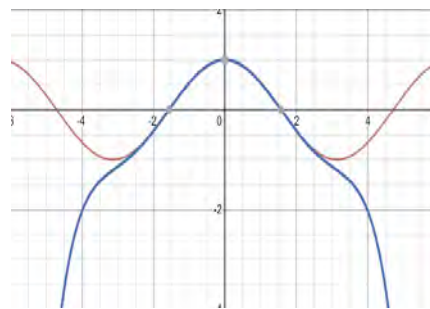
Imagine you have a pie. You eat half the pie and then eat half of the remaining pie, and then eat half of the remaining pie again. The more slices you eat, the total amount of pie you eat approaches the size of the original pie. The pie we eat can be shown by formula A, which is formally written as formula B. The total pie you eat ends up being 1 whole pie. Most people can grasp this, but what does this actually mean in the context of reality? What does adding infinite terms even mean? Does this result really have any application beyond a hypothetical pie?

$$\frac{1}{2} + \frac{1}{4} + \frac{1}{8} + \frac{1}{16} \dots$$

Formula A (Above)
Formula B (Below)

$$\sum_{n=1}^{\infty} \frac{1}{2^n} = 1$$

One key use of infinite series within Mathematics is the Taylor series. This series approximates a polynomial function $g(x)$ of $f(x)$ around the value of $x=a$, using the derivatives (first, second, third, fourth and so on) of $f(x)$ at $x=a$. For example, if we take a graph of $y=f(x)$ where $f(x)=\cos(x)$, there is no clear polynomial expression for the points surrounding $x=0$ at first, but if we take the derivative of $\cos(x)$ at $x=0$, the gradient of the line seems strangely close to the shape of the $\cos(x)$ curve at $x=0$. Currently, $g(x) = 1$, as at $x=0$, $\cos(x) = 1$. We can add more terms with powers of x , which help change this straight line into a curve that resembles the $\cos(x)$ curve. The coefficient of x^0 is determined by the y coordinate of $\cos(x)$ at $x=0$ which is one. The following coefficients are determined by the derivatives of $\cos(x)$ - x^1 from the first derivative (0), x^2 from the second ($-\frac{1}{2}$), and so on. Interestingly, this leads to a pattern to emerge with the coefficients. On the



$y = \cos(x)$ [red] &

$$y = 1 - \frac{1}{2!}x^2 + \frac{1}{4!}x^4 - \frac{1}{6!}x^6$$

[blue] [1]

curve to the side, you can see $y=\cos(x)$ (red curve) compared to

$$y = 1 - \frac{1}{2!}x^2 + \frac{1}{4!}x^4 - \frac{1}{6!}x^6$$

(blue curve). The blue curve approximates it very well until y reaches minus one. The coefficients for x , x^3 , x^5 and all the other odd powers of x are zero, because the "odd derivatives" of x are also zero. The Taylor series utilises this link between derivatives and coefficients, meaning we end up with this formula:

$$f(x) = \sum_{n=0}^{\infty} \frac{f^n(a)}{n!} (x - a)^n$$

where a is a real or complex number and $f^n(a)$ is the n th derivative of $f(x)$ at point a . The Taylor series is especially

useful when dealing with complicated formulae as it lets us approximate the formulae into a polynomial series, thereby making the problem much less complicated to use. For example, the formula describing angular acceleration in a pendulum is approximated from $\theta'' = \left(-\frac{g}{\ell}\right) \sin \theta$ to $\theta'' = \left(-\frac{g}{\ell}\right) \theta$ as $\sin(\theta)$ is approximately θ when θ is near zero.

Infinite series also do not have to be used for obscure pendulum problems either! In fact, series don't necessarily even need to be convergent (approach a number) to be useful. One example is Ramanujan's rather controversial take that $1+2+3+4 \dots$ is equal to $-1/12$.

Let's start with a series A, being $1-1+1-1+1 \dots$ it seems that the series goes from zero to one indefinitely, so there is no easy way to define what we get when A is evaluated. However:



$$A = 1 - 1 + 1 - 1 + 1 \dots$$

$$1 - A = 1 - (1 - 1 + 1 - 1 + 1 \dots) = 1 - 1 + 1 - 1 + 1$$

$$1 - A = A \text{ so } 2A = 1, \text{ so } A = \frac{1}{2}$$

Now let us consider a new series

$$B = 1 - 2 + 3 - 4 + 5 \dots$$

$$A - B = (1 - 1 + 1 - 1 + 1 \dots) - (1 - 2 + 3 - 4 + 5 \dots)$$

$$A - B = (1 - 1 + 1 - 1 + 1 \dots) - 1 + 2 - 3 + 4 - 5 \dots$$

$$A - B = (1 - 1) + (-1 + 2) + (1 - 3) + (-1 + 4) + (1 - 5) \dots$$

$$A - B = 0 + 1 - 2 + 3 - 4 \dots = B$$

$$A = 2B$$

$$\therefore B = \frac{A}{2} = \frac{1}{4}$$

And finally,

$$C = 1 + 2 + 3 + 4 + 5 \dots$$

$$B - C = (1 - 2 + 3 - 4 + 5 \dots) - (1 + 2 + 3 + 4 + 5 \dots)$$

$$B - C = (1 - 1) + (-2 - 2) + (3 - 3) + (-4 - 4) + (5 - 5) \dots$$

$$B - C = 0 - 4 + 0 - 8 + 0 - 12 = -4 - 8 - 12$$

$$B - C = -4(1 + 2 + 3 \dots) = -4C$$

$$B = -3C$$

$$\therefore C = -\frac{B}{3} = -\frac{1}{12}$$

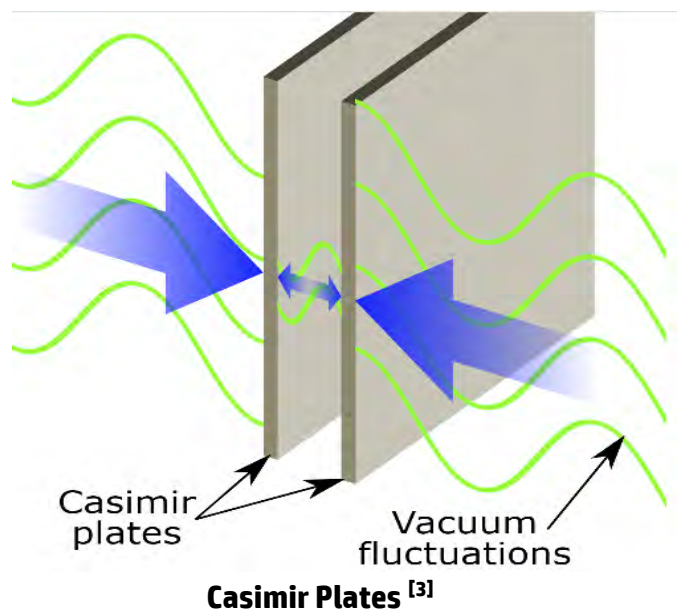
This result may seem absurd but it is certainly difficult to see any flaws in the reasoning. The Ramanujan Summation has had a huge impact on Physics, including being used in the “original” Bosonic String Theory as well as describing the solution to the Casimir Effect in which “given two uncharged conductive plates placed in a vacuum, there exists an attractive force between these plates due to the presence of virtual particles bread by quantum fluctuations. In Casimir’s solution, he uses the very sum we just proved to model

the amount of energy between the plates” [2].

Infinite series are mathematical devices that can tell us, often confusing and unintuitive truths about the world around us. Infinite series can be used as a tool for a range of problems, including simplifying trigonometric equations, developing string theory and allowing us to thus define the fabric of the universe around us. Anything requiring calculus, from basic integration to solving differential equations, all have one thing in common: infinite series are at the heart of solving these problems. The reason why infinite series are so useful is because of approximations: we hardly ever require exact evaluations of equations which is incredibly tiresome and complicated if they involve expressions with

non-polynomial functions – which can show up in fields like engineering, architecture and physics – instead infinite series let us see what such equations approximately yield without majorly changing the result. If a more precise result is needed, more iterations of the infinite series can be used. Perhaps the hypothetical pie we started off with was useful after all.

Edited by Atharva Narkhede





DID YOU KNOW?

The Soyuz spacecraft had a hard landing in 2008 which resulted due to the descent capsule not separating from the propulsion modules as planned. This led to the crew experiencing 8.2 G as they entered the atmosphere of Earth.

Physics Section

Muon Magic

A startling subatomic particle **p51**

Volumetric Displays

A startling subatomic particle **p53**

“What quantum physics teaches us is that everything we thought was physical is not physical.”

- Bruce H. Lipton



We can apply this idea when a particle approaches an obstacle. If the particle lacks the energy to break through the obstacle, the obstacle is a forbidden region that the particle cannot exist in. By using Schrödinger's equation, we can calculate the 'wavefunction' of the particle, which can be squared to find the probability distribution for the position of the particle. Under classical mechanics, there will be a 100% probability for the particle to be in a particular position and no chance at all for it to be anywhere else, even when the particle's position has not been measured.

Quantum Tunnelling

What happens during such a phenomenon?

By Tathushan Subenthiran (Y12)

Quantum tunnelling is a quantum phenomenon observed when a particle, headed at an obstacle without enough energy to pass through it, can sometimes simply appear on the other side ^[1].

This phenomenon is a direct result of the Heisenberg Uncertainty Principle, which states that the product of the uncertainties of two conjugate variables (variables, such as momentum and position or energy and time, with units that multiply to be the units of action, which is kgm^2s^{-1}) is always either equal to or greater than a certain number called Planck's constant divided by 4π ^[2]. Thus, a particle or

wave cannot possess an exact pair of conjugate variables simultaneously. For instance, a particle cannot have a precise momentum and position at the same time, nor can we make an exact observation of both variables in the present. However, the uncertainty principle is not simply an experimental shortcoming: it is an inherent characteristic of every particle.

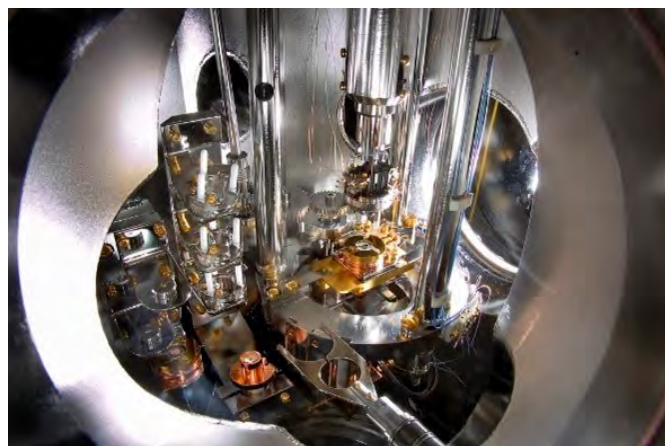
One example of the Heisenberg Uncertainty Principle is $\Delta x \Delta p \geq h/4\pi$, where x refers to position and p to momentum or $\Delta E \Delta t \geq h/4\pi$, where E refers to energy and t to time. Both these examples are versions of the Heisenberg Uncertainty Principle ^[3].

Under quantum mechanics, this is not the case. For us to have a sharp drop to zero in probability, we would need to be certain of the position of the particle which violates the Heisenberg Uncertainty Principle. Thus, the probability drops exponentially along with the obstacle but does not drop instantly to zero. If the obstacle is narrow enough, then there can be a probability that the position of the particle is on the other side of the forbidden region. The likelihood is minimal, but still present, and so particles are capable of 'tunnelling through' an obstacle. This can also be explained by the energy-time uncertainty inequality – for a short time, the particle can 'borrow' enough energy to jump over the potential barrier (the energy required to pass through the obstacle) and



then return it once on the other side. This doesn't always happen, but it can happen, so sometimes it does ^[4].

This phenomenon has practical uses too: in 1981, the Scanning Tunnelling Microscope was invented at IBM Zurich, which uses electron tunnelling to make images of objects as small as an atom ^[5]. A sharp metal tip is brought within a few nanometres of a sample of an electrically conductive material. The tip will have a slightly different voltage to the sample such that a potential difference is formed, and electrons are attracted to the sample but do not readily flow to it, due to the gap between them. However, some electrons will tunnel from the tip to the sample, producing a small, measurable current. The probability of tunnelling increases as the tip and sample gets closer, as the probability of the electron's position decreases along the forbidden region (and so the probability of the position being in the sample will too), so the rate of electrons tunnelling increases as well. Therefore, current increases as distance



Scanning Tunnelling Microscope

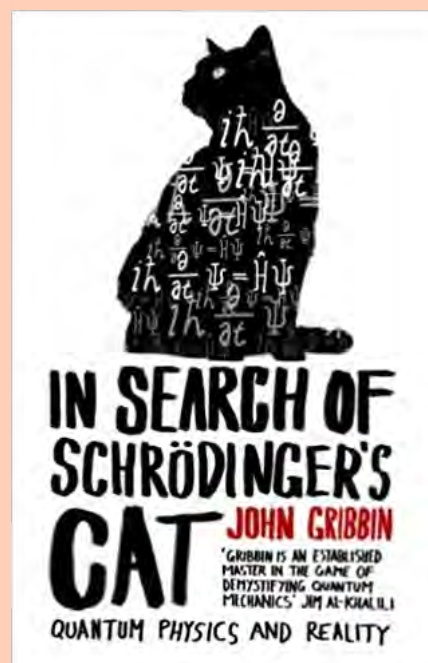
decreases – if something is physically sticking out of the sample towards the metal tip, we will get a spike in current as the distance between tip and sample decreases: we can study the current to map how the sample looks. This can be used to map atoms, study DNA, and even move around atoms – it allows for a new perspective of the nanoscopic world.

Edited by Mann Patira



IN SEARCH OF SCHRÖDINGER'S CAT by John Gribbin

This was essential to my understanding of quantum tunnelling, and a very enjoyable read. Gribbin wrote this book with the aim that a layman or a pre University physicist could have the best understanding of quantum physics they could whilst making sure the book was accessible - he does this by avoiding as much complex maths as he can and sticking to ensuring that the concepts he wishes to explore are fully fleshed out. In order to do this, he skilfully crafted his book to not only act as a source of information about different quantum physical phenomena but also as a history book, chronicling the different revelations different physicists (and chemists, teachers, etc.) have had in a way that maintains the interest of the reader. 'In search of Schrödinger's cat' is a fantastic read I would recommend to any budding physicist wanting to learn more physics, and anyone wondering about the true nature of reality and of the universe - the ideas Gribbin explores are counter intuitive (they don't fit what one would expect) and perhaps unsettling, but ultimately intriguing.



Startling Subatomic Particles

By Dulain Gamage (Y12)

Muons are elementary subatomic particles, similar to electrons with a charge of $-1e$. Muons are formed when cosmic rays interact with the nuclei of gaseous molecules in the atmosphere; typically, these collisions occur 10km above the surface of the Earth^[1]. The average lifetime of a muon is 2.2 microseconds, and the speed at which they travel is $0.998c$ where c is the speed of light. Even at these incomprehensible speeds, the muon's short lifetime restricts their maximum range of travel to only 660m yet, at the surface of the Earth, muons can be detected^[1]. To understand this phenomenon, we must first grasp the effects of special relativity.

To begin, we need to explore a concept called frames of reference. John is on a train that is moving at a constant velocity - there are no windows, the walls are soundproof and the only objects inside are a piece of string and a banana. How can John deduce whether he is stationary or moving? In short, he cannot. There is no acceleration, and thus John does not experience any force, so John believes he is stationary. Velocity is measured relative to other objects and if there are no objects to reference, our velocity is unknown. In John's frame of reference, he is stationary. "Frame of reference" can be thought of as "from their perspective"- if you were John, you would believe you were stationary, but if you were outside observing, the train and so John, is travelling at velocity v .

Now imagine a taxi with the driver and the passenger moving at velocity v towards an observer. All three entities move at the same velocity relative to the "stationary" observer outside - v . However, in the passenger's frame of reference, the driver and the taxi are stationary, and it is the observer that is moving at velocity v towards them. Another example is the earth which -relative to an observer in space - is orbiting the sun at 30km/s, and since humans are on Earth, we are also moving at 30km/s. However, since everything around us - the trees, houses, cars- are also moving at that speed, and since velocity is measured relative to objects around us, we register far slower speeds. A frame of reference where Newton's law of inertia holds true, is described as inertial frames^[2]. Thus if an object is not being acted upon by an external force, it will either be at rest or travel at a constant velocity.

Einstein began with two postulates— the laws of physics are the same for all observers in all inertial frames. The measured velocity of light in a vacuum c , is the same in all inertial frames of reference and is independent of the motion of the light source or the observer^[3]. The second postulate was a consequence of the Michelson Morley experiment producing a null result and establishing light's ability to travel without the need of a medium.

Time dilation arises as a result of the second postulate. This can be explained through by using a light clock. A light clock consists of two mirrors, stacked vertically a distance D apart. A beam of light, sent from the mirror A, is

reflected by the top mirror B, then set back towards to mirror A and the cycle continues. Each tick is registered when the light leaves A and returns to B.

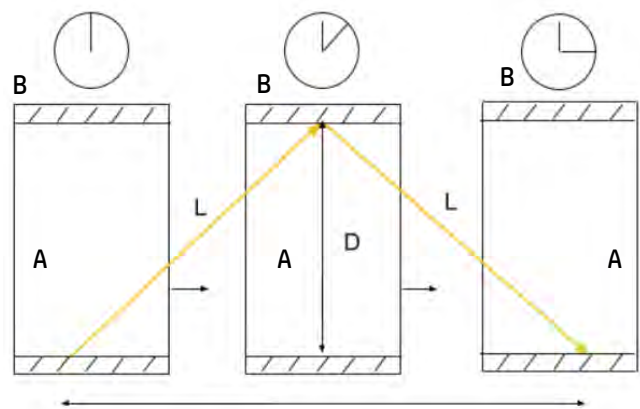
Molly is on a boat moving at a uniform speed of " v " -close to the speed of light. In her frame of reference, she is stationary and observes her light clock ticking every t_M . She believes she is stationary and everything around her is moving towards her. However, to a stationary observer, looking at Molly's clock, the light takes a triangular path (shown below).

$$t_M = \frac{2D}{c}$$

$$4D^2 = (ct_M)^2$$

$$t_J = \frac{2L}{c}$$

When the light leaves mirror A, mirror B (along with A) has already moved and so the light must take diagonal route.



One aspect that must remain consistent, is the light must reach the opposite mirror in both frames of reference. John is the stationary observer, and he has 2 clocks which are a distance vt_J apart. John must use two clocks in different places as, in his reference frame, the two events (the light leaving mirror A and the light returning to mirror A) happen a distance vt_J apart. In John's frame of reference, from the moment the light leaves mirror A and returns to mirror A, the time elapsed on his clock is $t_J = 2L/c$ as the light travels a distance $2L$ before returning to mirror A. Furthermore, in time t_J , the boat and therefore the clock, in John's frame of reference, has moved a distance vt_J . We can now calculate the distance L in terms of D and vt_J .

(Using Pythagoras theorem, we find L in terms of v , t and D)

We then substitute it into our equation for time elapsed on John's clock.

Multiply both sides by c and then square both sides.

We can then substitute for $4D^2$ using our previous equation.)

We can now rearrange to find John's elapsed time in terms of Molly's elapsed time. From this, we get the Lorentz factor.



$$L = \sqrt{\left(\frac{vt_J}{2}\right)^2 + D^2}$$

$$t_J = \frac{2\sqrt{\left(\frac{vt_J}{2}\right)^2 + D^2}}{c}$$

$$(ct_J)^2 = 4D^2 + (vt_J)^2$$

$$t_J = \frac{t_M}{\sqrt{1 - \left(\frac{v}{c}\right)^2}}$$

The Lorentz factor:

$$\gamma = \frac{1}{\sqrt{1 - \left(\frac{v}{c}\right)^2}}$$

The quantitative measurement of how much time and length changes for an object travelling at velocity, v . Examining the equation, we see that as v tends the speed of light, the Lorentz factor increases, and thus has a more noticeable impact.

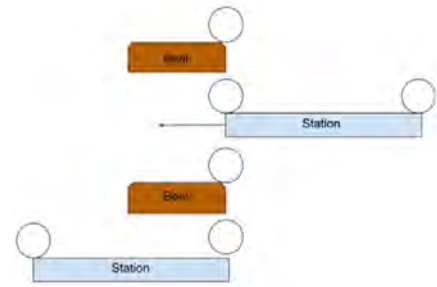
The Lorentz factor will always be more than or equal to one as the velocity of an object can never surpass the speed of light. Therefore, from John's frame of reference, he sees Molly's clock running slower than his, as more time elapses on his clock compared to her clock. A simple, common phrase used to describe this is "moving clocks run slow". The time that Molly measures is defined as the "proper time": which is the time difference between two events that occur in the same position in a reference frame and only requires one clock to measure [4]. In Molly's frame of reference, the two events -the light ray leaving mirror A and the light ray returning A - occurred in the same position. In John's frame of reference, the two events occurred a distance vt_J apart and so he does not measure the proper time. The velocities we experience and observe in our daily lives are far slower than the speed of light- in our equation for the Lorentz factor, we divide the square of our velocity by the square of the of the

speed of light, the result of this division is negligible and so the Lorentz factor is 1. Speeds, at which the Lorentz factor cause observable effects, are described as relativistic.

What does Molly see? From her frame of reference, she sees her clock running perfectly. The issue is seemingly symmetrical, except for one important detail: John uses two clocks to measure the time elapsed. This introduces a concept called the relativity of simultaneity. This is not required to understand the phenomenon with the muons. However, the effect of this concept is they both agree that John's second clock reads t_J .

We must next explore length contraction. The general equation for distance is velocity multiplied by the time. Molly and John want to measure the length of a docking station. Let us imagine Molly is on the same boat and travelling at the same relativistic speed. She is arriving to a docking station which can be simplified to a rectangular block. In her frame of reference, she sees the docking station moving towards her at velocity v . When her clock reads $t=0$, the front of the docking station has "reached" the front of her boat. After $t=t_M$ elapses on her clock, the back of the station has reached the front of her boat. She now knows the time taken and the velocity of the docking station and so calculates the length as shown in the first equation below. Remember, Molly's time is dilated. Thus, she measures less elapsed time, and so the length she measures is shorter. John who is on the station, uses two clocks as shown in the diagram. As John is stationary relative to the object they are measuring (the station), he measures the "proper length". Thus, John sees the docking station as longer than Molly does.

We can apply this to the case with the muons. Let us imagine a muon has an "internal clock".



$$l_M = vt_M$$

$$l_J = vt_J$$

$$t_M = t_J \sqrt{1 - \left(\frac{v}{c}\right)^2}$$

Subbing for t_J

$$l_{M=V} = t_J \sqrt{1 - \left(\frac{v}{c}\right)^2}$$

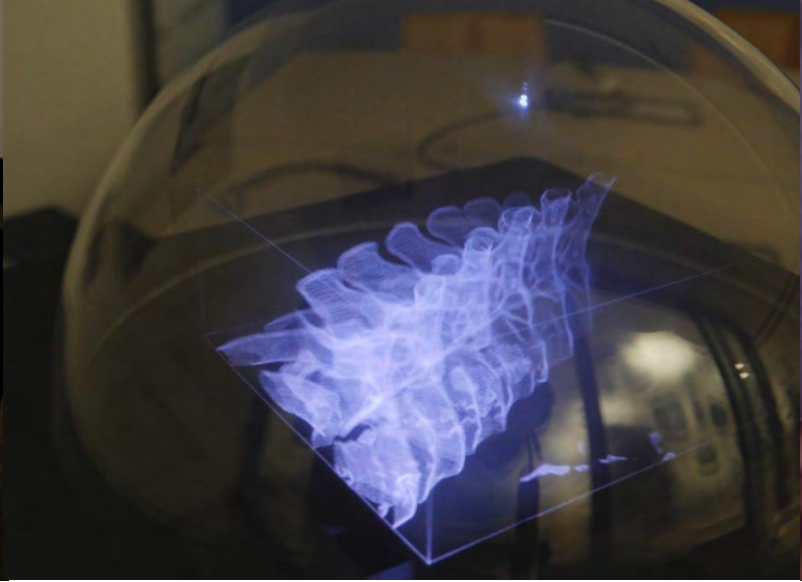
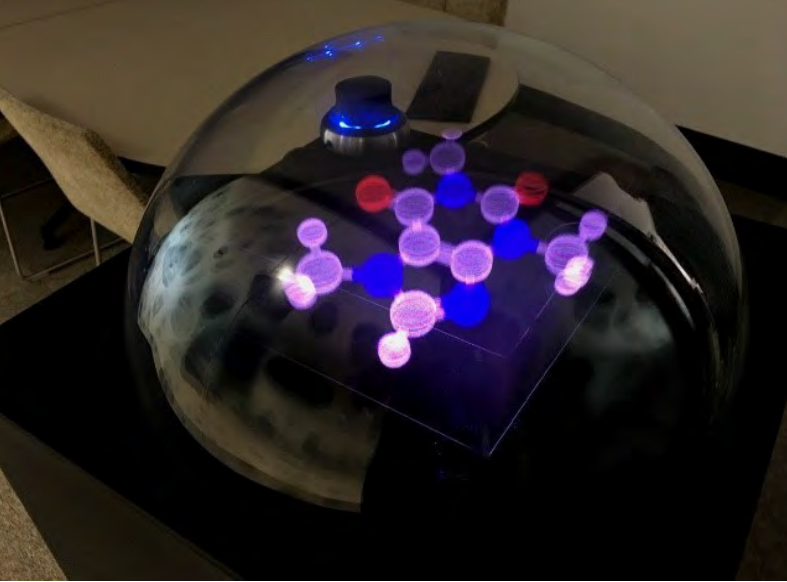
Thus,

$$l_M = l_J \sqrt{1 - \left(\frac{v}{c}\right)^2}$$

Remember: "moving clocks run slow". Observing from earth, we would see the internal clock running slow by a factor 1 over gamma where gamma is the Lorentz factor. As the muon travels at $0.998c$, the Lorentz factor is 15.8 . Thus, in our frame of reference, 34 microseconds elapses on our clock, when 2.2 microseconds elapses on the muon's clock. Multiplying this by the speed of the muon, the distance travelled by the muon is 10.4km . What does the muon see? In its frame of reference, the atmosphere is moving towards it at $0.998c$ and so its length will be contracted by a factor $1/15.8 = 633\text{m}$. Dividing this by the speed of the muon's velocity, we get 2.1 microseconds- which is less than its lifespan. Indeed, it does make it through the atmosphere without decaying.

Edited by Mann Patira





Volumetric Displays: From Science-Fiction to Science

By Prabhas Vedagiri (Y12)

Chances are you have never even heard of volumetric displays before, let alone know what they are. But it is likely that you have actually seen one already – maybe not in real life, but at least in movies. Volumetric displays are nothing more than what the layman refers to as a “hologram”. A “hologram” are those such as the ones in Iron Man – or, more famously, the “hologram” of Princess Leia, projected in Star Wars. I say “hologram” with quotation marks because, in fact, holograms aren’t the same as volumetric displays. While volumetric displays create real 3D images of 3D shapes, holograms merely appear 3D – they are virtual images, scattering light at a 2D surface ^[1,2,3].

Although holography was discovered all the way back in 1948, by a Hungarian scientist named Dennis Gabor, the first volumetric displays were only created in 2006 – almost a whole century after they were first postulated in science fiction novels ^[4]. A team from the Japanese National Institute of Advanced Industrial Science and Technology (AIST) collaborated with Keio University and Burton Inc. to create the first “real 3D images”. While this first volumetric display was primitive, to say the least, with no useful application – apart from creating a pattern of dots in the air, they showed one thing: there is indeed a solution to the “lightsaber challenge”. The

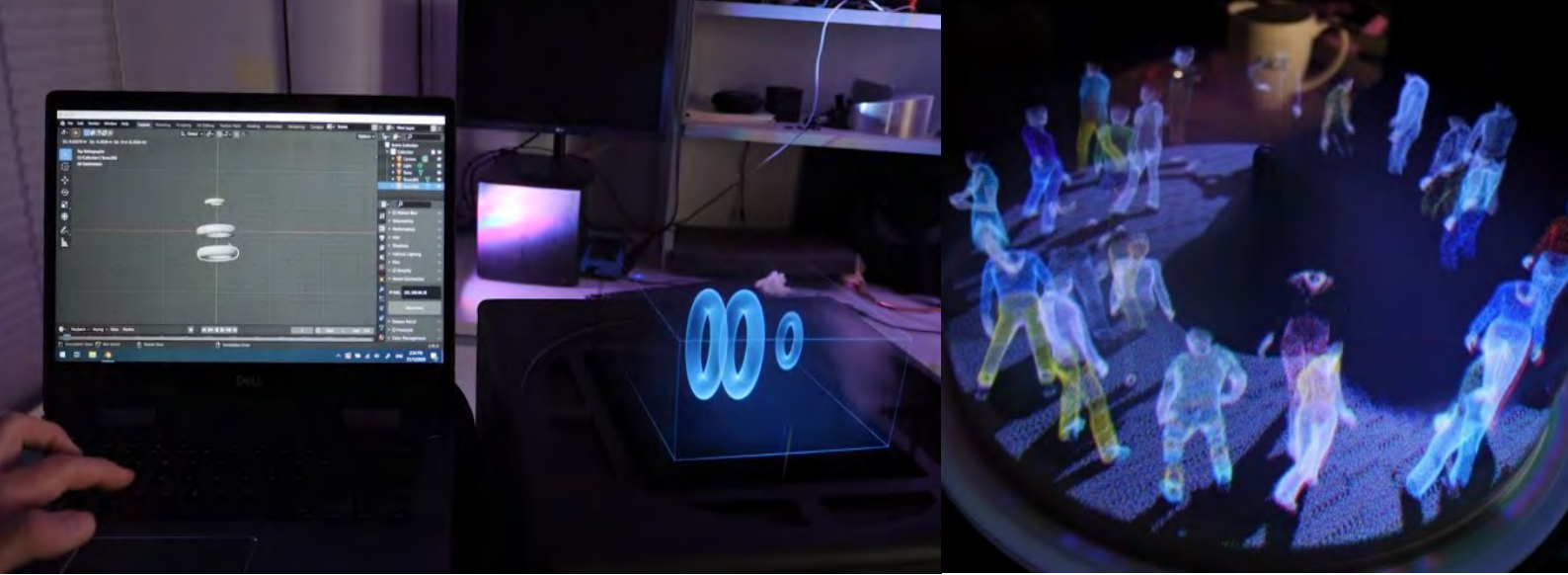
“lightsaber challenge” is the question posed by scientists: “how do you simply stop light from traveling in mid-air?” (As phrased by Dr Ricardo Figueroa, Rochester Institute of Technology) ^[2, 5,6].

Just as the team from AIST managed to break down air molecules into glowing plasma, to emit visible light, back in 2006, a team from Brigham Young University similarly managed to overcome the lightsaber challenge. They successfully created a high definition display in 2018 by “trapping” and “steering” a cellulose particle with a laser beam (known as an optical -trap display). While the display was merely a few centimetres in size, it demonstrated yet another advancement in volumetric display technology – the creation of high-resolution



Volumetric display of a butterfly from Brigham Young University ^[1]





images even in a free-space display (can project the display in air as shown in Figure 1). However, in reality, this display is only an illusion caused by the rapid movement of the particle. As David Smalley, PhD, from BYU says, “a good way to think about it is like a firefly or a sparkler”. What he refers to is the human persistence of vision, where images seem to fuse together as the human eye cannot process the images fast enough [2, 7, 8] [9, 10].

At this point, one is obliged to express their concerns for the viability of volumetric displays – if it took 12 years to get this far, surely at this rate competitors such as Microsoft’s HoloLens are much more likely to succeed, right? Wrong! Arguably, the most revolutionary development of volumetric displays came with the Voxon VX1 display table. At US\$ 9800 per unit, it offers much better value than Microsoft’s HoloLens 2 which, although only US\$ 4950, doesn’t allow multiple people to use a single device simultaneously [11, 12].

Voxon Photonics’ VX1 is a swept-volume display (and it operates by rotating emissive or reflective screens), with a resolution of 500 million voxels (essentially 3D pixels) per second. But it has more than just good graphics. The real commotion about the Voxon VX1 comes from its wide range of applications. The Australian start-up has already listed 37 use cases for the VX1 despite the novelty of the technology – and more is to follow [9, 12, 13].

Some of these uses, like playing games at over 4000 FPS, are simply for entertainment purposes. Others, however, have much more practical applications, such as 3D representation of molecules or instant 3D imaging using Blender, the CAD software. Demonstrations of volumetric displays for medical imaging and training have already taken place. They can even be used for 3D mathematical modelling, military visualisation, and, of course, video communications (as shown in Figure 2) [12, 13].

Despite all these existing uses for volumetric displays, there are still limitless possibilities for them. There is still the possibility of making volumetric displays responsive to touch – or the possibility to infuse AI to explore more applications – or the possibility to make the technology small enough to fit in a phone, or a watch. Actually, it seems I can hardly claim the development in volumetric display technology has even begun. And maybe it still is science fiction, and not quite science. But what I can say, is that the future looks bright.

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