



THE WILSON'S INTRIGUE

NEUROPLASTICITY

INTERVIEW WITH PROFESSOR JINDANI

HUMAN POWERED FLIGHT

SPACE TRANSPORTATION

Issue 2: Friday 14th February 2020

BIO-CHEMISTRY | ENGINEERING | PHYSICS

Introduction

We're back again and with renewed enthusiasm covering a wider range and more ambitious set of topics than we did in Issue 1. We hope you enjoy reading the articles in this magazine just as much as we enjoyed writing them, and so we would like to welcome you to the second issue of The Wilson's Intrigue, written by students for the students.

Our Mission:

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

Thanks to everyone who answered the survey in Issue 1 and we really appreciate your feedback. I hope you'll look forward to Issue 3 where I and the editing team will introduce a whole new design for the magazine. We would like to hear more from you, as the readers, as to what you like about the magazine and what more we can do to make the magazine more interesting, accessible and dynamic (and most importantly, to vote for your favourite articles). And so please visit <https://www.surveymonkey.co.uk/r/QR2Q5GX> where you will be asked to complete a short survey.

All references for each article can be found at the end of the magazine (from pages 61 to 63) which includes recommended websites, books and scientific articles for further reading/research, to explore a topic which has piqued your interest, beyond the contents of the article.

Thank you to Miss Banner, Mr Lissimore and Dr Whiting whose advice and support is very helpful in running the Science Magazine. Thank you also to Mr Carew-Robinson, Miss Ip and Miss Roberts for their help in confirming the scientific accuracy of the articles.

If you would like to write for the magazine and join a like-minded group of science and engineering enthusiasts, please email me at murugesand@wilsonsschool.sutton.sch.uk for more information.

The page outlining the Wilson's Intrigue Team (Editors and Writers) can be found on the next page.

The contents page outlining all the articles can be found on page 4 (please view this magazine on a PC for automatic hyperlinks from the contents page to the relevant articles)

Science knows no country, because knowledge belongs to humanity, and is the torch which illuminates the world.

Louis Pasteur

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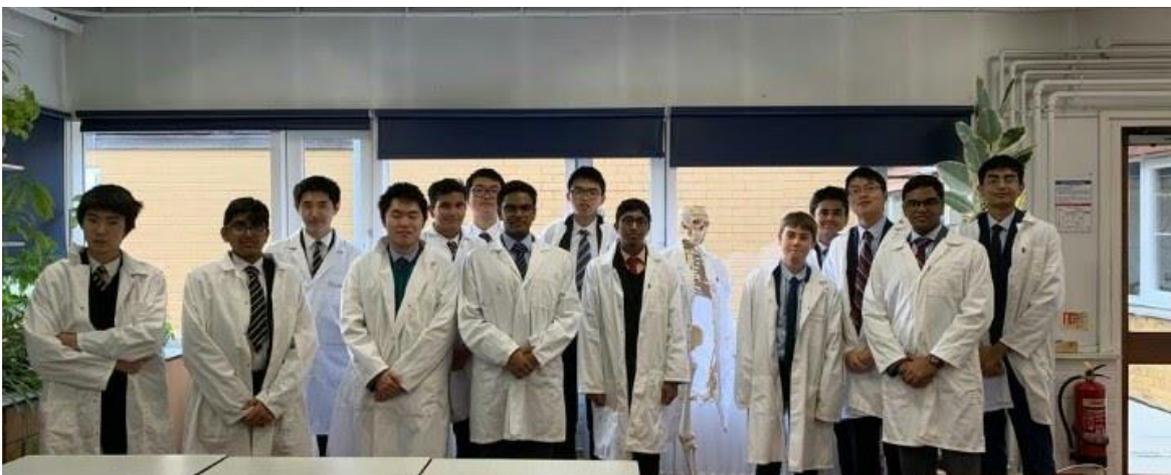
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BIO-CHEMISTRY- DID YOU KNOW THAT...

Coral reefs only occupy 0.1% of the area of the ocean but they support 25% of all marine species on the planet. Although scientists have only just begun to understand how reefs can contribute to medicine, already coral reef organisms are being used in treatments for diseases like cancer and HIV. However, about half the world's shallow water coral reefs are already gone, and without urgent action to address climate change, pollution, overfishing and destructive coastal development, these life-sustaining natural wonders could all but disappear.

- WWF

The Role of Neuroplasticity in the Viability of Hemispherectomy

by Anussan Nadarasa (Y12)

“Life with a half a brain”. It’s a rather open-ended series of words. It could be the heart-wrenching story of an individual who walks this earth, living only to survive, incapable of experiencing and unable to even voice their own suffering, but it also could be the title for a much anticipated comedy. Yet, it is neither. Amazingly, there are people who lead perfectly normal lives, like any other human being, with the only difference being that they have half a brain.

In severe cases of epilepsy, such as ¹**catastrophic epilepsy**, a patient’s seizures can become so persistent that they hinder the patient’s neurological development, to such a degree that ²**developmental regression** occurs. In such instances, with of aim of preventing further seizures, doctors may surgically isolate the affected hemisphere of the brain, in a procedure known as hemispherectomy, either by removing it entirely or by severing all connections to the functional hemisphere. It sounds too radical to ever even consider, much less to perform, yet hundreds of patients have undergone this procedure in the last century. This is testament to its success at achieving seizure freedom, as exemplified in a recent study which found that 86 percent of the 111 children who underwent hemispherectomy at Hopkins between 1975 and 2001 are either seizure-free or have non-disabling seizures that do not require medication. If undertaken during childhood, the patient may also retain incredibly high levels of cognitive, sensory and motor function – even lingual ability can reach near-complete recovery in patients who had their language-dominant hemispheres resected (cut out); the postoperative ³**neurologic deficits** are thus minimal.

This ability to recover normal function has been shown numerous times in multiple patient studies. For instance, a study published in *Cell Reports* studied six patients who had undergone hemispherectomy, at ages ranging from three months to eleven years. fMRI (functional MRI) scans of these patients showed that activity in the parts of the brain that control certain functions, such as vision, movement, cognition and emotion were extremely similar to non-hemispherectomy patients – the authors of the study determined this by comparing the fMRI scans to the dataset of 1482 healthy young adults from the Brain Genomics Superstruct Project. Not only this, but the long-term follow up of hemispherectomy patients at various hospitals throughout the globe have yielded similar results. To use the example of one John Hopkins hemispherectomy patient, Christina Santhouse finished her secondary education, being accomplished in both athletic and academic pursuits: in her senior year, not only was she was appointed the captain of her school’s varsity bowling team, yet she also came twenty-sixth in a class of two hundred and twenty-five, which allowed her to matriculate at the Misericordia University in Pennsylvania.

What allows, thus, for such an invasive procedure to be so viable, however, is the plasticity of the brain and, especially, the higher level of neuroplasticity in patients of such a younger age. ⁴**Neuroplasticity** allows certain cognitive functions that are distributed evenly between the two hemispheres, like language comprehension, to become more biased and lateralised to one side later on. In hemispherectomy patients, there is also increased connectivity between parts of different ⁵**functional networks** (“between-network connectivity”) across all networks in the brain, hence there is increased communication between networks. This increased between-network connectivity in hemispherectomy patients is reflective of an adaptive increase in network integration, which is necessary to support overall cognitive function, since hemispherectomy results in a loss of typically available brain structure that supports the functional organization of the brain. The increase in between-network connectivity, therefore, is suggestive of the functional reorganisation (ie. lateralisation of brain function) that occurs in patients’ post-hemispherectomy, due to the plasticity of the brain.

There is also a lot of redundancy in neural pathways that allows certain pathways to take over in case another pathway is defective. However, once the functional regions and neural circuits have become lateralised to one side, removal of that hemisphere can cause permanent, irreversible cognitive and/or motor damage. Thus, patients who have undergone hemispherectomy in adulthood are significantly less likely to regain full function of certain abilities, such as vision, audition, language/speech, cognition and motor function on the affected side – they may even become paralysed on that side of their body. Hence, if hemispherectomy is carried out before the age of two, patients are substantially more likely to retain normal function.

Nonetheless, cognitive abilities, such as reading, writing and language production are more likely to fully recover than vision and motor function, even if the operation is carried out at such a young age. For instance, if the left hemisphere was resected, the patient would have impaired motor control on the right side of their body and vision in the left side of both eyes would also be lost. This is because there is less compensation for motor and visual function than for cognitive function in the contralateral hemisphere, since there will be fewer connections between motor and visual networks and other networks and thus less lateralisation of motor and visual functions, even after intensive physical therapy.

Surprisingly, however, patients who have not had an ⁶**anatomical hemispherectomy** have higher likelihood of regaining full function of all abilities. This is most likely because the bilaterally distributed ⁷**functional regions** of the brain may still be interconnected, although less strongly, across the left and right hemispheres, thus some ⁸**(resting-state) networks** may still be intact. The existence of these preserved bilateral resting-state networks may be a result of the development of alternative interhemispheric connections, which occur as a result of neuroplasticity. Hence, neuroplasticity and the newly-formed connections and thus some of the intact resting-state networks may contribute to cognition, motor function and vision in hemispherectomy patients

The formation of new connections and the increase in connections between networks shows how the brain can adapt in extreme circumstances to enable cognition and motion, even in the absence of half of it. In the future, it may be possible to examine how these connections can help patients with damaged or missing brain tissue compensate while performing specific tasks. Understanding how atypical brains develop and reorganize themselves, thus, may help in the discovery of new treatments for people with brain injuries.

Edited by David Kuc

Key terms:

1. **Catastrophic epilepsy** – severe drug resistance epilepsy occurring daily, often multiple seizure types, and they lead to delayed cognitive development or regression
2. **Development regression** – when children who had previously exhibited normal development progressively loses lose certain developmental skills that they had previously successfully acquired.
3. **Neurologic deficits** – a neurologic deficit refers to abnormal function of a body area. This altered function is due to weaker function of the brain
4. **Neuroplasticity** – the brain's ability to reorganize itself by forming new neural connections
5. **Functional networks** – networks in the brain that are associated with certain functions, e.g. motor function, cognition, vision, etc.
6. **Anatomical hemispherectomy** – when the affected hemisphere is removed entirely
7. **Functional regions** – regions of the brain that are associated with certain functions, e.g. motor function, cognition, vision, etc.
8. **Resting state networks** – resting state networks, as imaged by functional MRI, are distributed maps of areas believed to be involved in the function of the “resting” brain.

An Investigation into Mycobacterium tuberculosis

by Devanandh Murugesan (Y12)

Incidence of Tuberculosis (Epidemiology):

Around 1.7 billion of the world's population carry TB Bacteria. It is estimated that at least 1 in 10 people develop TB at some point in their lives. The majority of the time this deadly pathogen may remain latent, hence you may be afflicted with a latent tuberculosis infection (LTBI) which is a 'state of persistent immune response to stimulation by *Mycobacterium tuberculosis* antigens without evidence of clinically manifested active TB' [WHO definition] i.e. you don't present any symptoms and TB remains at non-transmissible levels. The sinister nature about it is in that you don't even know it – "You have a ticking bomb waiting for your immune system to get weak," - Hayan Yacoub, MD, an internal medicine practitioner at Austin Regional Clinic in Texas. However, without swift treatment, 5-10% of people with LTBI will develop active TB as soon as their immune system's immune response begins to deteriorate. This may happen for several reasons with the most common being diseases which affect the immune system (HIV/diabetes), treatments which affects the immune system (chemotherapy) and malnutrition.

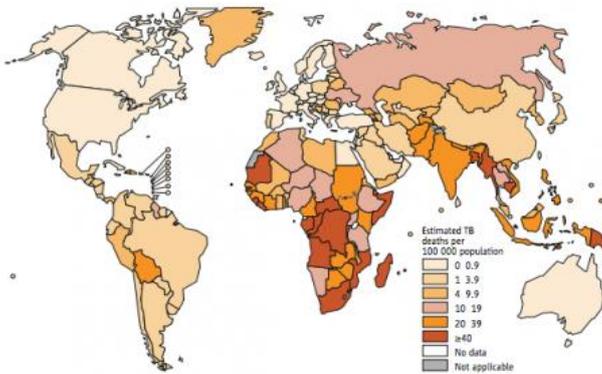


Figure 1: Global TB Mortality Rates excluding TB deaths among HIV positive people (2014)

Although tuberculosis mortality rates are decreasing, 5000 people still die every day - countries with high mortality rates due to TB are known to have a 'high burden' of TB. According to WHO, in 2018, the 30 High burden TB countries accounted for 87% of new cases, with India having the highest count followed by China and Indonesia. Moreover, figure 1 generally shows that TB-related mortalities transpire more frequently in LICs and NEEs (South-East Asia and Africa) than Western countries.

Pathology of Tuberculosis:

Mycobacterium tuberculosis is spread from an individual with active TB to an uninfected individual through the air (droplet infection). These droplets can be produced when a person coughs, sneezes or even speaks – and the droplets remain airborne for extended periods of time making TB a highly contagious disease.

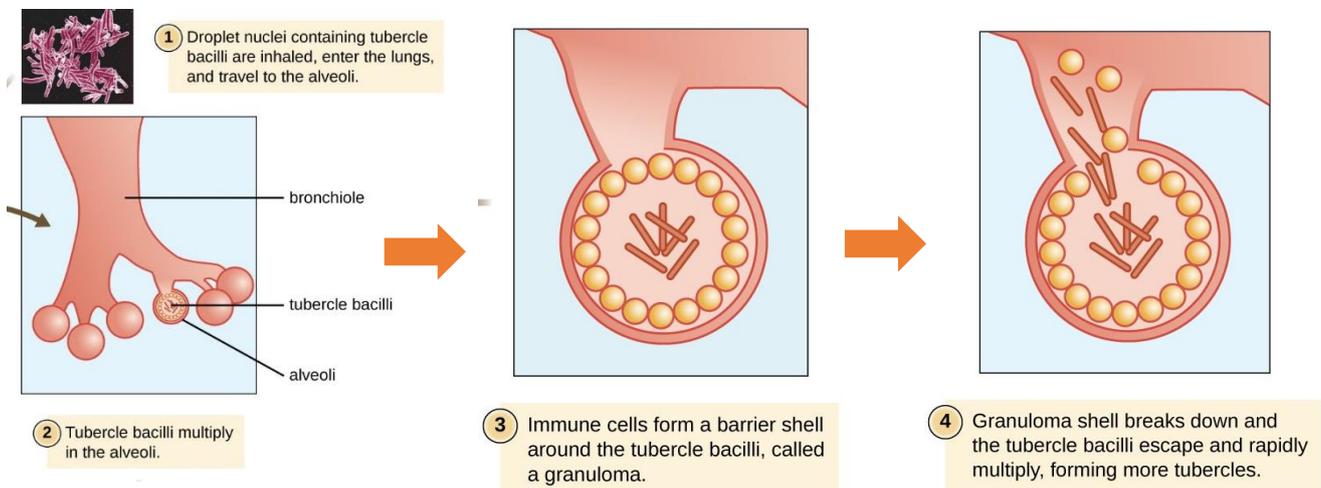


Figure 2: Pathogenesis of Mycobacterium Tuberculosis Simplified (Granuloma Formation in Alveoli)

Figure 2 outlines the process of pathogenesis of this bacterium after being inhaled by an uninfected individual. At the start of Stage 3, the body mounts an immune response and alveolar macrophages normally kill some of the bacteria by phagocytosis often leading to LTBI. Around this time, granulomas also form due to the accumulation of T lymphocytes and attempt to confine the growth of the remaining bacteria and the formation of caseous necrosis (a soft-cheese structure) with low oxygen levels, low pH, and limited nutrients. Even though further fibrosis and calcification of the tuberculosis lesions act to control the spread of the bacteria, they may begin to replicate rapidly if the infected individual's immune system deteriorates; the granulomas may be unable to contain the infection.

It is precisely this fact that makes HIV positive individuals more susceptible to active TB (an opportunistic pathogen) since the low CD4 (T lymphocyte) count means that granulomas can be more easily broken.

Perhaps more importantly, the contagious nature of *Mycobacterium tuberculosis* can be exacerbated by socioeconomic factors in LICs and NEEs. For example: rapid growth in the population due to increasing urbanisation inevitably leads to a high population density and perhaps poor sanitation, especially within slums. When coupled with poor/underdeveloped infrastructure (e.g. low investment in healthcare), densely populated communities can be easily overwhelmed and are simply not equipped to limit the spread of disease, which may explain why contagious, airborne pathogens such as TB are more prevalent in these areas.

The History of Tuberculosis:

The term 'tuberculosis' was coined by Johann Schölein (a German Physician) in 1834. It comes from the Latin word 'tuberculum,' meaning 'a small swelling'. This is due to the fact that when autopsies were conducted on TB patients, doctors discovered small white lumps within the lungs, also lending itself to be referred to as 'the white plague'. However, despite Dr Koch later isolating the bacteria which caused tuberculosis, it has been hypothesised that *Mycobacterium tuberculosis* originated 150 million years ago.

Despite the first documented evidence describing TB appeared in India and China 3300 and 2300 years ago respectively, Egyptian mummies dating back to 2400 BC reveal skeletal deformities pertaining to extra-pulmonary tuberculosis (where the bacteria has infected other organs in the body outside of the lungs). The Ancient Greeks called tuberculosis 'phthisis,' which describes a living body that shrivels with intense heat as if placed on a flame. In Medieval England, the term used was 'Scrofula/King's evil' and so people at the time believed it could be healed with the 'Royal touch' (a ritual frequently conducted by Henry VII where he touched the face of an infected individual and prayers from the Bible were chanted).

In stark contrast, the 1830s paved the way for new and creative approaches to the treatment of tuberculosis in the UK. A report published by Sir Edwin Chadwick prompted a reformation of living standards such as providing drainage on streets to deliver clean water and remove sewage. Though these actions were based on the belief that TB was caused by breathing foul vapours, they resulted in a gradual decline in mortality from tuberculosis in the United Kingdom from 1850 to 1960 (as shown in Figure 3).

Another notable idea was the establishment of sanatoria by George Bodington, who opened the first sanatorium in 1836 which served as a catalyst in the construction of tuberculosis sanatoria throughout Europe in the late 19th century onwards. This was based on the idea that 'a regimen of rest and good nutrition offered the best chance that the sufferer's immune system would "wall off" pockets of pulmonary TB infection' in the pre antibiotic era.

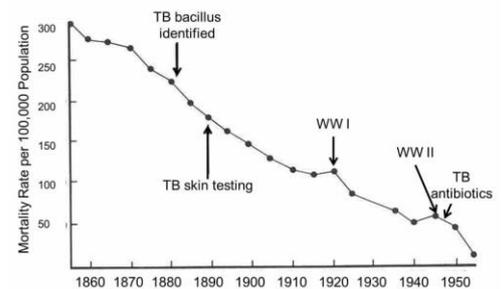


Figure 3: TB Mortality over time, UK



Figure 4: Sanatorium de Pen-Bron, France

Treatments for Tuberculosis:

Antibiotic	Mechanism of action
Isoniazid	Inhibits the synthesis of a fatty acid needed to make bacterial cell walls
Rifampicin	Inhibits bacterial RNA polymerase
Streptomycin	Binds to bacterial ribosomes to prevent binding of tRNA
Pyrazinamide	Not yet known, not the same mechanisms as the other 3 antibiotics

Figure 5: Antibiotics' for TB: Mechanism of Action Summary

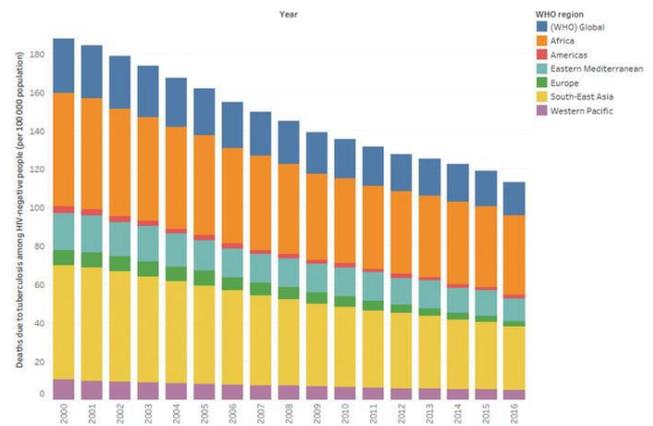


Figure 5: TB Mortality Rates per 100,000 people - WHO

Currently, TB is treated with a regimen involving a combination of 3 or 4 different antibiotics, each with different mechanisms of action (outlined in figure 5), usually lasting 6 – 9 months. However, this poses a problem as patients need to complete their course of antibiotics, remembering to take them everyday, otherwise this can lead to the development of drug-resistant TB which is much more difficult to treat. As a result, many scientists worldwide such as Professor Amina Jindani are working on clinical trials to reduce treatment duration to prevent an increase in TB resistant cases and decrease relapses post-treatment.

On the other hand, the BCG vaccine is the most popular vaccine for TB, with the vaccine being given to 100 million children per year globally as of 2004. It is derived from an attenuated strain of *Mycobacterium bovis* and was introduced in 1921 but has remained controversial in terms of its effectiveness differing across many clinical trials (depending on geographical locations). It has been reported to have variable efficacy in both low and high TB burden regions and more importantly, the efficacy of the vaccine nullifies after 15-20 years, demonstrating the brevity of immunity an individual could have to TB. Hence, more research is still required to create longer lasting vaccines that can be distributed more widely to eradicate TB.

Barriers to Treatment and Further Research:

One of the greatest issues, as outlined above, is the emergence of more antibiotic resistant strains of TB as patients are not finishing their course of drugs. Other problems relate to the clinical trials themselves where there is competition for research grants, then permission from ethical boards and participants dropping out of the trials culminating in difficulty creating and approving drugs. However these challenges are becoming overcome through the implementation of methods such as DOTS (Direct observation treatment system), where healthcare workers watch patients take their medication to ensure that they actually being taken. Moreover, increased funding is being put aside by the WHO and charities in order to support the research and innovation needed to discover and research new pharmacology interventions.

Prognosis/ Outlook:



Charities such as World Without TB, and initiatives such as World TB Day (24th March) strive to raise awareness of this disease and reinvigorate the efforts to combat it. Furthermore, The United Nations Sustainable Development Goals (SDGs) include ending the TB epidemic by 2030 under Goal 3 (Good health and well-being). *'Tuberculosis has plagued humanity for millennia. And it's still with us... TB knows no borders. Everyone is at risk. But it thrives where there is poverty, malnutrition or conflict... By the end of today, nearly 4400 people will have lost their lives to TB – including more than 600 children. Enough is enough. Now it's time to deliver. There has never been a better opportunity to make TB history.'* – Dr Tedros (UN General Assembly, 2018)



Interview with Professor Amina Jindani, Infection and Immunity, St George's University, London by Devanandh Murugesan (Y12)

Timeline of Achievements:

1960s - Coordinated the first East African/British Medical Research Council trial of short-course chemotherapy in Africa.

2003 –Elected as a Fellow of the Royal College of Physicians of London.

2004 – Joined St. George's, University of London as Honorary Senior Lecturer where she established the International Consortium for Trials of Chemotherapeutic Agents in Tuberculosis, known as INTERTB.

2006 – Established World Without TB as a civil society NGO to support research to meet the United Nations Millennium Development Goals for the eradication of tuberculosis by 2030.

2018 – Was awarded the Muslim News Ibn Sina Award for Medicine.

2019 – Was awarded the Princess Chichibu Memorial Global TB Prize by the Japan Anti-Tuberculosis Association and was awarded professorial status at St George's University

PRESENT – Is conducting the RIFASHORT trial to reduce treatment for tuberculosis from 6 to 4 months.

Professor Jindani conducts international, multicentre clinical trials on pulmonary tuberculosis with the objective of significantly reducing treatment duration with increasing doses of rifampicin, and, eventually, eliminating the disease. The trials are conducted in countries of South America, Africa and Asia where there is a high burden of tuberculosis.

- St George's University Profile

Q1) There are many diseases in the world, what inspired you to focus on tuberculosis specifically:

It was quite by accident, I went to work after qualifying and I gravitated towards Kenya, towards Africa. I went to work in Nairobi and they sent me down to a hospital called the Infectious Diseases Hospital where they treated TB and leprosy. I have never seen either before but that's where they put me and that's where I first developed my interest in tuberculosis. Leprosy is difficult to treat, but for TB, I felt I could do something as a doctor, I can cure patients and that's how I got into tuberculosis and have remained in [this field] until now.

Q2) You talked about curing tuberculosis in the Infectious Diseases Hospital. At that time, what was the treatment and what was the relapse rate after that treatment?

The treatment was for 12-18 months and relapse was very low, about 3%, provided that the patients took their drugs. But 12-18 months is too long and with tuberculosis, as soon as you took your drug for 3-4 months they started to feel well again, put on weight, their cough disappeared and they thought 'why bother?' So they stopped and then they relaxed. Although officially under trial (lab) conditions, the relapse rate was 3%, it was in fact in routine conditions (normal circumstances) much higher. So clearly the treatment duration had to be reduced. So that is what I have been working on all that time, eventually we got down to 6 months

It is quite an achievement

Yes, Indeed. Even that. You see, you have got to remember that tuberculosis is a disease [associated with] poverty. Poor people, living in overcrowded conditions, they get tuberculosis. You and I won't get

tuberculosis. [Since they are poor,] perhaps, they can't afford the bus fare to the clinic, in Africa they have to walk long distances to the clinic to get their medicine. So now the idea is to reduce treatment duration even further - even 6 months is too long, TB patients usually stop taking their medication at around 3-4 months. Sadly we don't yet have an effective vaccine, if we had a vaccine, our problems [would be] solved. But after a lot of research, people are still doing research on vaccines; we haven't yet identified a safe and effective one. So the way to go is to reduce treatment duration. So the trial I am doing now is testing two 4 month regimens. So 4 months, if it works, is a significant reduction and as patients tend to stop around 3-4 months... if it works, the majority can be cured in 3-4 months. Keep in mind, those affected by many diseases may relapse afterwards. But if that works, the next step is to see to get it down to 3 months and then 2 months and then one can even hope to have a one shot treatment in the future.

Q3) Why do you think that there is a lack of funding for normal strand tuberculosis compared to multidrug resistant tuberculosis?

You know there's a lack of funding for everything. I don't know if you have been listening but there's an election coming up and the Tories say they will fund this and the Labour party say that they will fund that and both promise to fund the NHS. Money is needed for so many projects, and so, tuberculosis is, in my opinion, probably not a very high priority, again because it is a poverty related disease. If rich people got tuberculosis, they would find a way to cure it very quickly. So essentially, you have to do something to improve the economic

situation of these people and that's a huge undertaking, particularly as things stand, there are too many people on the planet and most of them are unfortunately poor. So that's why I think people concentrate more on cancer - cancer research gets a lot of money. Heart disease, stroke, they get a lot of money but an infectious disease... look at what happened to Ebola, people threw everything at it because it was such a horrible disease; they made sure the Ebola epidemic was confined to where it began and they got rid of it. But they threw tons of money at it. So, one has to raise awareness, and that's what I try to do, raise awareness so that I can get the funding and see if I can reduce treatment duration.

Q4) When you were talking to TB patients, how did you overcome communication difficulties and language issues?

I speak a lot of languages: Swahili, Gujarati, Kutchi, French, Spanish and some Chinese so I have not had any problems in terms of communication with patients. The thing about communication is, you have to get patients and their families into the 'curing process'. Not as a doctor saying you must do this but say we are all in this together therefore the family gets involved and so the stigma gets removed; the family gets involved in curing the patients as well as the doctor.

Q5) So an effective support network is needed for treatment?

In my view, that is the way to practise medicine, not that 'you know, you get tuberculosis, go and sit in the corner'. Get everybody involved. It is like crime, if you don't reduce crime, eventually it will come to you. They'll get into your house. If you train them, educate them to get a job, they don't commit crime and you don't suffer for it. So to me, medicine is like that. If I can cure 100 patients like that, [it becomes] unlikely I will get it because I will have cured them. So the whole mindset has to be inclined that way and it has always been like that for me. It is not only putting on a white coat and a stethoscope and pointing 'you got to do this, you got to do that'. No, you got to get into it and work together to make the patient better.

So there's a lot of teamwork involved

Teamwork is essential but empathy is also essential. Certainly I need a microbiologist in my team but you got to have empathy. Not only have sympathy, but empathy with your patient, because one day that could be you. You see, it was only when I had my appendix out that I realised what people go through when they have their appendix out. So more than anything it's empathy, I think.

Q6) How could the cost of TB drugs be reduced in the future?

The cost of TB drugs is not very much now. The six month treatment costs £20 and when you think we had a seminar on melanoma this afternoon and one regimen of treatment for melanoma costs £85,000, you see the difference. So £20 is not a lot of money but again I have to stress that tuberculosis is a poverty related disease - the patients don't have £20. So in most countries in the world, in fact, in all of their treatment is free at the point of care - the drugs are given to them for free. The point is to make them take the drugs and yes, we have to reduce the cost of the drugs because national tuberculosis programs can't afford to buy so many drugs for so many sick people, so the cost of the drug has to come down as well. A pharmaceutical company could develop an effective TB drug, but they'll have done it at an enormous cost to them, so it has to be cost effective for them. They'll say, 'okay, we developed this drug, but you got to do the uptake, you got to buy the drug from us so that we can reduce the cost'. So the second thing is the uptake of the drug that would bring the cost down.

Q7) What do you have to do to set up a treatment centre?

Basically, I don't set up new treatment centres. I try to work with national treatment programs in that country, because they are the ones who see the TB patients. They are the ones who need to be trained in good clinical practice. They are the ones who need to be trained in ethically conducting experiments on human subjects. Because that's what clinical trials are - you are experimenting using human subjects and you have to be very careful. So any centre that has a high burden of tuberculosis and wants to participate. We go there, we train them, their practices improve, cure rates improve, but the cardinal thing is that if you are experimenting on people, you have to be ethically very, very aware that you are using human subjects.

Q8) What challenges do you face when trying to get your clinical trial approved by local and international ethics committees?

Challenges comes from the ethics committees and regulatory bodies of each country, since their requirements are not uniform. If they were uniform it would be different, but each country has different regulatory bodies from which I need to get approval. In one country I only have one body, in other countries I have up to four bodies, so those are the challenges I face and I have to convince them that it is in the interest of their population that this trial be done but they keep

tabs regularly on the conduct of the trial – how many are cured, lost, that kind of thing. They can't just approve anybody coming in (although this happens a lot).

Q9) How hard it is to gain research grants?

You have to hit the Euro Million jackpot. Everyone is winning millions of pounds there, it is a staggering amount. It is very hard to get money for tuberculosis except if you are researching drug resistant tuberculosis, - it is easier to get money for drug sensitive strains but for the population that I treat, it is really very hard. There is a lot of competition out there and not enough money, so even if you put in your grant application, you really have to show that your results will have very high impact - the impact factor is very important. And if the reviewers are convinced that your trial is successful it will have very high impact in eliminating that disease, than you will be likely to get the grant but otherwise it is hard work.

Q10) Why do you think there is a stigma around tuberculosis and receiving treatment for it?

The stigma is that you are not only going to spread the disease to me but that you are going to die. And eventually having spread it to me that, I am going to die - that is the stigma. Now we have to convince people that tuberculosis is curable. The stigma was there when there was no treatment and TB patients were put into ^[1]sanatoria and they remained in the sanatoria until they died. But now you have to convince them and tell them you can be cured. If the ^[2]isoniazid reduces colony count [significantly] within two days you know you are no longer transmitting the disease. So to all of this I say, you have to sit the family and the community down and explain all of this to them. They are not ignorant, they are just not well-informed. So when you do explain this to them they become very cooperative and I don't think there is a stigma for TB anymore. Once in South Africa, I think they found out that somebody had HIV and they stoned that person to death. But stigma is based on ignorance really; you just have to enlighten them and tell them the truth

Q11) How important is international collaboration in drug development?

You see, you do a trial, then the best way is to go to conferences and publish your results, that way you get International collaboration. Moreover you can publish the results in a high impact journal where anyone can see it, that way you can also get international collaboration. I have just come back from India as a matter of fact, where I spoke about one of my trials, but I have also been awarded a prize by the Japan Anti-tuberculosis association. So it's just putting the results out there and letting people criticise what you did or approve of what you did and from that trial you modify and do another one. If the results are bad, still publish it. Never ever hide. That is ethically wrong because again you have used human subjects to experiment on them so if the outcome is not so good, everybody should know so that they won't repeat the same mistake. So just put it out there for everyone to see and that's how you get international collaboration. I go to conferences all the time.

Q12) What would you say has been your greatest achievement to date?

If I were to have done anything at all, it would be to have reduced TB treatment duration. If I succeed in reducing treatment duration I hope it will be in the RIFASHORT trial.

Q13) Do you have a message you would like to say to the people reading this article?

When you mention tuberculosis, most people say 'Ah, tuberculosis, it's gone now,' but it has never gone away. It kills 2 million people every year. Please help us eradicate it. If it can be cured, it can be eradicated.

Q14) If you had one word to describe science (in general), what would you say?

Science equals life. Everything must be based on science. Even anything I eat I look at its scientific value. And it's not just about preserving life but prolonging a good quality of life. I still take the bus at the age of 83 because I think about my carbon footprint. I always believe that if you have a roof over your head and eat 1 meal a day, you are already doing better than most people in the world.

Further Information:

[1] 'an establishment for the medical treatment of people who are convalescing or have a chronic illness.' For more information on sanatoria please read 'An Investigation into Mycobacterium tuberculosis'

[2] Isoniazid is used in TB treatment as it has been proven under lab conditions to decrease the number of bacilli significantly in the first 2 **days** in a sputum sample to non-transmissible levels. However monotherapy is never used in TB treatment as the bacilli can quickly develop resistance. Rifampicin is used as it has late bactericidal activity and kills the 'persistors' — bacilli that remain **undetected** in a patient's body for an extended period of time. —undetected— (Latent TB)

For more information on previous trials conducted by Professor Jindani please visit:

<http://www.isrctn.com/ISRCTN55670677> (RIFATOX) <http://www.isrctn.com/ISRCTN44153044> (RIFAQUIN)

A crash course in Duchenne muscular dystrophy (DMD): causes, symptoms and possible treatments

by Ken Li (Y12)

1 in 3,500 children, mostly boys, suffer from this condition, showing symptoms of skeletal muscle degeneration^[1] from the early age of 3. As they get older, they get physically weaker and weaker; by adolescence, many are already confined to a wheelchair. Even with top-tier treatment, few make it past their thirties, passing away at this early age having been propped up on life support, and barely clinging onto life. Such is the reality of DMD – but why is this condition so devastating? How is it caused? Perhaps most importantly, how can it be treated, maybe even cured?

DMD is an X-linked recessive disorder, caused by a mutant variant of the gene that codes for the protein dystrophin, at **locus** XP21 on the short arm of the X chromosome. Since females have two X chromosomes, whereas males have one X and one Y chromosomes, females will need to inherit two copies of the mutant allele at XP21 on both chromosomes, whereas males will only need to inherit one mutant allele to have DMD on the X chromosome, as the Y chromosome is too short to accommodate an allele at XP21. Hence, males are much more likely to have DMD than females are - however this pattern of inheritance also means many females will not know they are a carrier until their son has DMD, by which time it is likely already too late.

Only 2/3 of all cases can be explained by inheritance, however, as the remaining proportion of cases are likely to have arisen from germ-line mutations in the normal dystrophin gene at XP21. Either way, this mutation causes a dysfunctional dystrophin protein to be produced, and in some rare mutations, not at all. The most common mutation of the dystrophin gene, however, is a deletion of one of the bases that leads to a **frame shift** in all of the bases following the deletion. Thus, the mRNA produced during transcription has an incorrect sequence, and when it is translated at the ribosome, not only is a different sequence of amino acids added to the polypeptide due to the frame shift in codons on the mRNA, but the stop codon (either UAG, UGA, UAA) also arises too soon, meaning the resulting polypeptide is too short.

In unaffected people, dystrophin is thought to be an anchoring protein, connecting the **actin filaments** to the **extracellular matrix** (via binding to the **dystroglycan complex** in the **sarcolemma** of skeletal muscle fibres (myocytes)). During muscle contraction, **myosin**, which runs parallel to the actin filaments, pulls the actin filaments along^[4] and thus leads to shortening of the length of the myocyte. Dystrophin is able to protect the sarcolemma of the muscle cell from strain and damage during contraction by anchoring the actin filaments and preventing them from being pulled along too much.

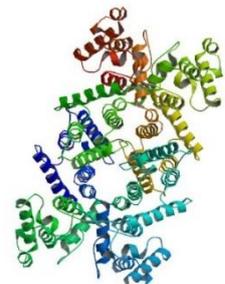


Fig. 1-The dystrophin protein

If the dystrophin protein is too short, it is missing the dystroglycan binding end (which binds to the dystroglycan complex), meaning it is not able to perform its role of anchoring the actin filament to the extracellular matrix, causing tears in the sarcolemma over time.

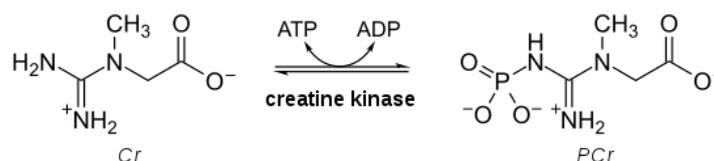


Fig. 2-the reversible conversion of creatine into phosphocreatine in the presence of ATP

This enables Ca^{2+} (which is in excess outside the myocyte) to diffuse freely through the gaps created, which would not previously have been able to pass directly through the **phospholipid bilayer** of the sarcolemma as a charged molecule. Excess amounts of Ca^{2+} in particular are very damaging to the cell^[2]. Firstly, since it is soluble, it can have an osmotic effect on the myocyte, and can lead to water entering the mitochondria, for example, which makes it burst, essentially destroying the ability to generate energy for the pulling of actin fibres in muscle contraction. Ca^{2+} is also an **enzyme activator**, and can activate too many dormant proteases through binding to **allosteric sites** on the enzyme, so that they begin to break down functional proteins in the myocyte.

Another effect of tears in the sarcolemma is that the enzyme, creatine kinase (CK), is able to leave the cell in vast quantities, ending up in the blood. This enzyme catalyses the conversion of the protein creatine to the phosphocreatine in the presence of ATP^[3]. The reaction can be reversed, meaning ATP can be rapidly regenerated from phosphocreatine, essentially rendering the presence CK as essential to facilitate energy storage for the myocyte. Lack thereof, therefore, in the myocyte means less energy is available for muscle contraction, weakening the muscle further. As a side note, elevated CK levels in the blood are also a useful indicator as to whether an individual has DMD or not.

All of these knock on effects means that muscle cells die and muscle degeneration and wasting occurs rapidly, faster than they can be regenerated. A typical patient will experience muscle loss initially from the pelvis and legs, meaning Gower's sign (when a person has to use their arms to help them stand up) in children is an early symptom of DMD. When muscles degenerate, fatty and fibrous tissue can also build up, also explaining the swollen calves in people with DMD. Other early symptoms also include a curved spine due to weak back muscles, arms swept back for balance and a protruding belly, as a result of weak abdominal muscles. As the patient gets older, the diaphragm and **myocardium** also weaken, eventually leading to death.

As of yet, there is no cure for DMD, and treatments revolve around therapy, medication and surgical intervention. However, an interesting recent innovation is the idea of gene therapy to directly manipulate and correct the process of making dystrophin. Conventionally, this involves using a viral vector to transfer the 'normal' dystrophin gene to the nucleus of the myocyte so the cell machinery is able to produce the functional protein. However this brings a variety of challenges, for example ensuring that the therapeutic DNA remains functional and that stability of the myocytes is maintained. Cells do divide, and this can make it quite difficult to successfully incorporate the new DNA into affected cells (since the new DNA is not incorporated into chromosomes before interphase) in the long term, meaning therapy may have to be carried out quite often. Another risk is the possibility of an immune response, which can be triggered if lymphocytes detect the presence of the viral antigen.

However, another method, exon skipping, is even more sophisticated in the sense that it removes the complication of having to introduce new genetic material into the cell. Instead, this method focuses on correcting the process of transcription and translation. When the faulty DNA for dystrophin is transcribed, pre mRNA is produced which has coding and non-coding sections, exons and introns respectively - the spliceosome then removes the introns in a process called splicing to leave the coding exons in the mRNA that will be translated. Premature stop codons, as discussed before, may be located on a particular exon wedged between 2 introns, in which case the spliceosome may be stimulated to cut out the two introns with the stop codon-carrying exon wedged in between^[5]. When the mRNA is then translated, the stop codon is encountered later on, and the resulting protein is longer, with both actin and dystroglycan binding sites present, but just a shorter **central rod**, thus restoring the function of dystrophin. This is meant to reduce the symptoms of DMD to prolong the lifespan and improve the quality of life for those with the condition.

These treatments are only in the early stages of development, and there have been cases where they have not worked or have worsened the onset of DMD. Because of the complexity of DMD, and the uniqueness of each case, it is understandable incredibly difficult to develop a universal cure that will suit all patients. However, these treatments do show promise and have the potential to become permanent cures, which is ultimately the Holy Grail for all DMD researchers. Only time will tell.

Edited by Ray Wang

Key terms:

1. **Frame shift:** When a substitution or deletion of bases in DNA leads to the reading frame, the sequence of codons, to be altered due to all the bases shifting along by one base.
E.g. TAC|CCG|GAT|GTG|ATT ...
following deletion of C in second codon, becomes: TAC|CGG|ATG|TGA|TT
2. **Actin filaments:** The long strands of protein that run the length of muscle cells, which serve the dual purpose of forming the cell's cytoskeleton and also muscle contraction. More on this in Y13 Biology.
3. **Extracellular matrix:** The network of proteins outside the cell that provide structural support and hold all the body cells together in tissue.
4. **Dystroglycan complex:** A transmembrane structure which connects the actin filaments via dystrophin to the extracellular matrix
5. **Sarcolemma:** The cell membrane of muscle cells
6. **Myosin:** The long protein with golf club shaped projections that latch onto the actin filaments and pull them along during muscle contraction. They run parallel to the actin filaments, and are a constituent of sarcomeres, the contractile units in muscle cells. Again, more on this in topic 7 of A Level Biology.
7. **Phospholipid bilayer:** The natural arrangement of phospholipids (polar molecules with a phosphate head and 2 fatty acid tails) when placed in water. The main constituent of cell surface membranes.
8. **Enzyme activator:** Any molecules that can increase/stimulate enzyme activity when binding to an enzyme.
9. **Allosteric sites:** A binding site on an enzyme that is not the active site.
10. **Myocardium:** Heart muscle tissue.
11. **Central rod:** The main section of dystrophin; the bridge between the actin filament and the dystroglycan complex.

Chess: Why you should start learning today

by Koushikk Ayyappan (Y12)

It may be evident that I am an avid chess enthusiast, having recently been awarded the Golombek medal. However, in order to avoid bias, I will be approaching the topic from a predominantly scientific background, in order to convince you to order a chess set today.

Physical Change of the Brain's Structure:

Chess, as with other intellectually stimulating activities, changes the structure of the brain after prolonged exposure. Firstly, it is important to understand the arrangement of a normal brain.

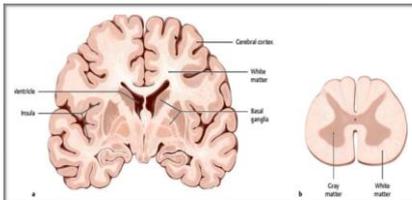


Figure 1: A diagram showing the distribution of white and grey matter in the brain.

The brain is composed of both white and grey matter. Grey matter is a significant component of the CNS (central nervous system), and refers to unmyelinated neurons, ¹glial cells, ²soma and various other cells, which are used in the control of muscles, emotions, senses, memory, etc. Many ³studies have proven a positive correlation between IQ and volume of grey matter. White matter, on the other hand, refers to areas of the CNS that are predominantly composed of myelinated axons, which are neurons enclosed by ⁴Schwann cells in a 'myelin sheath'. White matter acts as an information coordinator between areas of grey matter in the brain, where the decision making occurs.

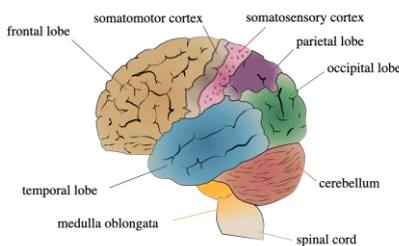


Figure 2: A diagram of the brain showing the four lobes' positions.

In proficient chess players, there are major differences in the volume and distribution of white and grey matter in the brain, with grey matter volume reducing over time. This is especially prevalent in the occipital-temporal junction, where the occipital lobe and temporal lobes meet, and is involved in representing objects and their relations to one another in our minds – visuospatial sketchpad. In addition, reduced radial ⁵diffusivity is present at the right side of the superior longitudinal fasciculus - a major white matter tract that transmits information from the 'visual' areas to the 'decision making' areas of grey matter.

On the other hand, at the left side of the superior longitudinal fasciculus, unusually, more diffusivity is present, implying that the overall distribution of white matter and synaptic connections had changed; this process is known as learning-based neuroplasticity. ⁶Cortical thickness was also reduced. These alterations in brain structure cumulatively result in an overall smaller brain volume.

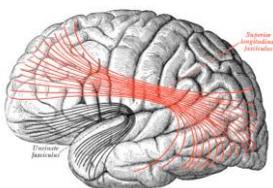


Figure 3: A diagram showing the location of the superior longitudinal fasciculus

However, this may not necessarily be detrimental to one's learning capacity or intelligence, despite the reduction in grey matter. The so-called idea that 'big brain = smarter' is false. For example, Einstein's brain weighed only 1,230g, compared to the average adult male's 1,400g. This indicates that there may have been other structural changes that may have contributed to his intelligence and effective thinking skills, such as the abnormal number of glial cells present - an observation from the dissection of his brain post-mortem.

In a chess player's brain, the reduced radial diffusivity may have been due to an increased efficiency in neuronal transmissions (due to increased myelination). The causal factors for these changes are still unknown, due to unavoidable variables not being controlled in these studies, such as the age at which one started learning chess. Perhaps practice during childhood contributes to increased neuroplasticity, during the maturation of the brain to adulthood, or perhaps this change in overall distribution of white matter occurs later in life - as a result of excessive practice. Studies are yet to prove the reason behind these complex variations in brain structure, but we have established that it is not necessarily damaging.

Impact on Academic Results:

Many scientific studies have proved that chess improves cognitive function as a result of long-term practice. Auditory memory was ⁷proven to be boosted, using the dichotic auditory-verbal test, which involves different sentences being played simultaneously on both the left and right ear, with the individual being required to repeat what was said in only one ear. There was a significant difference ($p=0.023$ vs $p=0.013$) between the scores for expert chess players and novices, proving that a correlation existed between chess and memory.

Following on from the idea that memory is strengthened, one would assume that excellent math scores would follow. This statement is surprisingly incorrect, but the very notion that chess ability and test scores are intrinsically interlinked is due to unreliable studies. ⁸This complex meta-analysis has been the source of almost all confusion, when it comes down to chess and mathematical ability. It concludes that there is a significant, positive effect on results for those who play chess. However, most of the studies included poor experimental design, and lacked an active control group. This often results in invalid results as a result of the placebo effect. A new ⁹study has attempted to use an active control group, who played checkers instead of chess and the results had shown no statistical difference between mathematical and chess ability.

Further studies would need to be conducted for ability in different subjects, although it would be more difficult to obtain a quantitative result for scores in English, for example, as it heavily relies upon interpretation. Personally, I believe that the benefit of a stronger memory alone would be enough of a motivation to start learning chess, despite no definitive improvement in subject results.

Chess is... a sport?!

Chess, with respect to biochemical and physiological aspects, is very similar to classical physical exercise. The reason why most of the world still do not consider it a sport is baffling.

Firstly, the energy needed for any brain activity is derived from glycogen stores in the brain, muscles and liver, followed by ¹⁰**adipose tissue**. Glycogen is broken down into glucose via a hydrolysis reaction, and this monomer is used for aerobic respiration and for the release of energy stored as adenosine triphosphate, also known as ATP. It is formed a result of the electron transport ¹¹chain and various other chemical reactions in the inner mitochondrial membrane, and the conversion of ATP to ADP (adenosine diphosphate) releases energy for our cells. The same biochemical process is used in classical physical exercise and sport.

Spirometric analysis (a test measuring airflow of the lungs) has shown that the respiratory exchange ratio had fallen in chess players from >0.89 to 0.75 between the start and end of a tournament, indicating that the source of energy had switched from carbohydrate to lipid oxidation (using the fat stores in adipose tissue), which is the same process that occurs during conventional sports, once glycogen stores are low.



Figure 4: How a spirometer works.

Moreover, energy expenditure during chess is similar to that during a marathon. In 2009, Robert Sapolsky of Stanford University stated that a chess grandmaster could burn up to 6,000 calories a day. This equates to over three times the average person's daily calorie consumption. Fabiano Caruana, who is commonly considered to be the second-best player in the world, normally weighs 135 pounds, commented that he had sometimes 'seen the scale drop below 120' after an intense tournament, suggesting that chess may even be used in a weight management scheme in a manner similar to physical exercise.

Physiological similarities are also present. Over an 18-day tournament in 1980, a ¹²study measured circulatory parameters, as well as 'autonomic excitability', referring to the autonomic nervous system, regulated by the hypothalamus - in control of our unconscious processes such as digestion and respiration.

The results had shown that the chess players were comparable to sportsmen of the 'light athletics' class, and in this sense, chess could very rightly be called a sport.

Heart rate increases by 75-86 beats per minute, primarily as a result of the mental stress. The player's adrenaline level can reach up to 8 times the normal value. Too many catecholamines (the group of hormones secreted by the adrenal glands, including adrenaline) can lead to severe physical pressure, and even heart attacks due to the increased chance of a blood clot forming as a consequence of hypertension - the leading cause of death in elite chess players. Therefore, it could be claimed that some physical fitness is needed in order to excel in competitive chess.

Overall, chess is a much easier alternative to other forms of physical sport, such as football or badminton, as it can be played almost anywhere, especially with the growth of chess apps and websites, and can even help control your weight, which is useful in the long term.

Chess as a protective factor against dementia:

Finally, chess can literally checkmate dementia - which is currently the leading cause of death in the UK. It can reduce the risk of the disease by over 74%, due to learning-induced neuroplasticity, which minimises the deterioration of neuronal connections. A meta-analysis¹³ study highlights the fact that any highly stimulating mental activity can reduce the risk of dementia, and chess - being one of them - can reduce the risk of Alzheimer's by 33% compared to the control group and can even delay the onset of dementia.

Ultimately, if you have some spare time, it is most definitely worth learning chess to reap these benefits, as it can even increase your life expectancy when it comes down to these terminal, neurodegenerative diseases. Chess does not have to be sitting by yourself in a dark room with a chess book, trying to solve a mate in 63, but can also be a very social hobby, especially if you join a weekly club. I hope that you will order a chess set today, or alternatively visit <https://lichess.org/learn/>. Enjoy!

Edited by Utkarsh Sinha

Footnotes:

1. **Glial cells** surround neurons to provide support and insulation e.g. Schwann cells, which we will visit shortly.
2. **Soma** are essentially neuronal cell bodies, which are connected to dendrites. These send information via electrical impulses to other neurons.
3. <https://www.nature.com/news/2004/040719/full/news040719-11.html> would be an example of such.
4. **Schwann cells** insulate neurons in order to speed up the rate of electrical impulses and the transmission of information, and form the 'myelin' part of the sheath. These fatty cells cause the 'white' colour of white matter.
5. **Diffusivity** refers to the diffusion properties of tissue or 'bushiness' of the brain.
6. Refers to the thickness of the layers of the cerebral cortex.
7. The study: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4715404/>, which also details biological reasons why this is the case.
8. Meta analysis study : <https://www.sciencedirect.com/science/article/pii/S1747938X16300112?via%3Dihub>.
9. Study which accepts the null hypothesis - that no correlation is present : <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5709436/>.
10. **Adipose tissue**, or fat, is a term for loose connective, made up of adipocytes. Glycerol can be converted to glucose in a process called gluconeogenesis.
11. This video explains the process very clearly : <https://www.youtube.com/watch?v=LQmTKxl4Wn4>.
12. <https://www.ncbi.nlm.nih.gov/pubmed/6775202>.
13. <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6617066/>.

How Dentists Are Detecting Oral Cancer Earlier

by Mohamed Ahmed (Y11)

Only 50% of the public are aware of oral cancer.^[1]

Oral cancer^[2]'s low survival rate (just 52% in five years after diagnosis) is partially due to delays in seeking professional advice (on average a four month delay). This makes this disease especially difficult to treat—survival rates saw no improvement at all from 1965 to 2015. While efforts to raise awareness over symptoms and risk factors (like alcohol and tobacco) are on-going, further improvements have also been made to the way we diagnose oral cancer as early as possible.



Figure 1: An oral cavity

But recent advances in this field mean that the survival rate may soon rise dramatically—which is why the World Health Organisation (WHO) has described it as an ideal choice for its goal to reduce cancer cases by a third—as the oral cavity^[3] allows for easy clinical inspections and mucosal changes (such the development of oral lesions and ulcers) to be examined, prior to the development of the tumour. These abnormalities can then be inspected to determine if they may be cancerous.

Through the process of tissue reflectance, visible light (ranging from wavelengths of 410 to 710nm) can be used to find abnormal cells. Cancer cells (especially epithelial) have larger nuclei—resulting in increased light reflectance when compared to normal cells. Hence, the observed colour of the lesion changes depending upon if it has the enlarged nuclei typical of cancerous epithelial cells. These methods are remarkably cheap at £25 for a single disposable stick and £250 for a reusable kit, meaning they are beginning to see mainstream use.

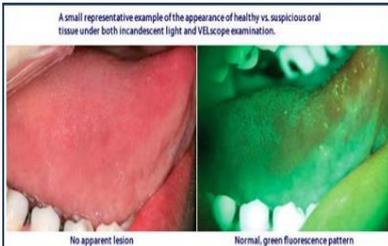


Figure 2: The use of tissue reflectance to identify the red suspicious oral tissue

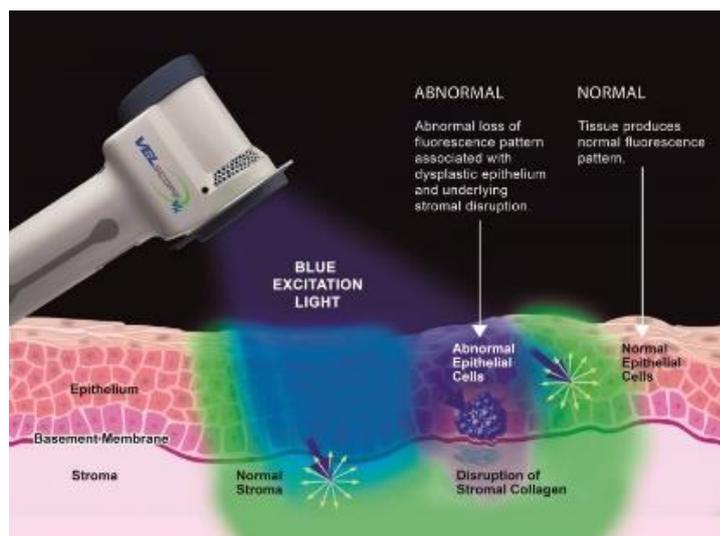


Figure 3: A diagram demonstrating how autofluorescence functions

Another method is autofluorescence, when ultraviolet light is also used to differentiate abnormal cells through observed colour. Human tissue contains a variety of fluorophores such as collagen, tryptophan, elastin, keratin and haemoglobin, which re-emit light at varying wavelengths during this process. The concentration of these fluorophores (and therefore natural light scattering and absorption) changes in cancerous conditions, causing a change in the spectral property of the oral mucosa^[4]. While this is more expensive (a visually enhanced lesion scope costs about £3,000), it can be used during procedures to define the surgical boundaries of the oral lesion.

While both techniques have their place in combating oral cancer, there are flaws that prevent them from becoming the final verdict in the diagnosis of oral cancer.

The biggest problem is in the sensitivity and specificity of the method: sensitivity is the probability of a test correctly identifying people with the disease (important in determining its use as diagnosis) whereas specificity is the probability of correctly identifying people without the disease^[5].

Tissue autofluorescence has sensitivity of 50% and a specificity of 81% leading to many false positives that need further testing. Tissue reflectance has 0% sensitivity but good specificity at 86%.

Overall, these tools are useful in referring patients to specialists but not as a final diagnosis of the disease.

Edited by Michael Lowe

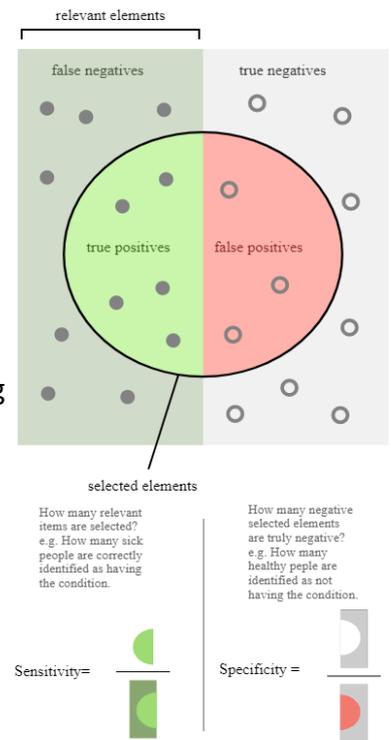


Figure 4: A diagram of how sensitivity and specificity is calculated

Footnotes and Key Terms:

1. according to a CPD on oral cancer at the London Dental Education Centre- presented by Dr Amit Rai BDS (Hons)
2. excluding cancers of the lip
3. the mouth
4. a membrane lining the inside of the mouth
5. See the diagram for further clarification. Specificity is an indicator of how many true positives there may be whereas sensitivity is an indicator of how many true negatives there may be.

The Challenge of Forensic Pathology

by Saishankar Vicknarajah (Y12)

In every crime film, there is always a scene where the forensic team is called to examine the death and provide a forensic report on the cause—but the process of producing the report is usually overlooked and disregarded. In this article, I am going to outline this process.

Forensic pathology^[1] is a sub-speciality within histopathology^[2], which involves discovering the cause of death, especially where the reason for death is seen to be unexpected or unnatural. A forensic pathologist investigates the cause of sudden and unexpected deaths using pathological methods and provides evidence in court that can be used in trials.

How a forensic autopsy is performed

At the scene of the crime, if police officials suspect that a homicide has taken place then they will call upon a 'Home Office Registered Pathologist'^[3]. This forensic pathologist would be called to the scene of the crime so that they can provide medical advice to the police about the cause of death and advise the officials on the recovery of the body, to ensure that no critical traces of evidence are lost while the body is moved to the mortuary.

Stage 1:

At the mortuary, the post-mortem^[4] examination of the dead body can begin. Initially the forensic pathologist conducts a detailed external examination of the body. The pathologist weighs and measure the dimensions of the body and records the subject's clothing, valuables and phenotypic characteristics^[5] such as hair colour and length, eye colour, ethnicity and sex. They would then remove the subject's clothes to examine the body for marks such as tattoos, scars, wounds, surgical incisions and evidence of lesions on the skin. X-rays may be used to reveal any bone abnormalities and ultraviolet light is used to detect specific residues left on the body.

While examining, the forensic pathologist collects any potentially useful samples (such as hair, fingernails and foreign objects found on the body), which might help to identify the cause of death. Throughout this procedure the forensic pathologist records all the evidence photographically and through written notes.

Stage 2:

After the external examination is complete, the subject's body is cleaned and prepared for the internal examination. For most standard autopsies, the pathologist usually dissects the subject's chest, abdominal and pelvic organs. However, sometimes the brain and other body parts such as arms and legs can be dissected if necessary. Before starting, the forensic pathologist places the subject's torso^[6] on a rubber block which extends the body's arch enabling greater access to the chest and abdomen. The pathologist then makes a Y-shaped incision from both shoulders joining over the sternum^[7] and then cutting straight down to the pubic region. The forensic pathologist would separate the skin and underlying tissues and then remove the ribcage by using a rib cutter to cut along the boundary between the ribs and the cartilage^[8] connected to the breastbone.

This allows access to the chest organs^[9] and also the abdominal organs^[10] so they can be dissected free. However, before removing the individual organs, the pathologist examines them with the naked eye, looking for changes, especially those caused by diseases such as atherosclerosis, cirrhosis of the liver and coronary artery disease. From this point on, the organs are removed, weighed and examined by further dissection to reveal any abnormalities like tumours and tiny samples are taken for examination under a microscope. Once the autopsy is finished, the organs are either returned to the body or are retained for

teaching and research purposes. The previous incision made in the body is sewn back. Even after the autopsy, bodies can still have an open casket funeral, as the incisions made are hidden.

Finally, once the autopsy is complete, the forensic pathologist uses the documentation to write a detailed report of their findings. They describe the autopsy procedure and any microscopic findings and conclude their verdict on how the death was caused. The report is presented to the officer dealing with the case and the forensic pathologist also has to give a witness statement to the police and provide the evidence in court.

Case Study: Dr Richard Shepherd

Dr Richard Shepherd was one of the UK's most distinguished forensic pathologists, who has built up a remarkable career, working in some of the most high-profile and tragic deaths that have taken place in recent decades. These include the 9/11 attacks, the 7/7 bombings and the deaths of Princess Diana and Stephen Lawrence. In an interview with the Guardian, Shepherd estimates that he has worked on over 23,000 dead bodies and said he was able to switch between the mortuary and his home, where he is a loving father and husband.

One of his most high-profile cases was involving Princess Diana, who died in a car crash on 31 August 1997. However, Shepherd reveals in the interview that "it was because she wasn't wearing a seatbelt" and that if she was strapped in "she could have walked away with a black eye". Shepherd's experience in his career has allowed him to make expert judgements on how the deaths were caused, but his career was not all highs.

In the interview, Shepherd admits that his previous mentality of him being "used to death [...] used to it for 35 years" was flawed. In 2016, Dr Richard Shepherd was diagnosed with PTSD (post-traumatic stress disorder), and this was due to the "build-up of emotional damage from putting 23,000 dead bodies under the knife".

Shepherd recollects how he had never noticed any mental effects on him, but now describes the process of getting PTSD as "like little fish—nibble, nibble, nibble—such tiny pieces go that you don't notice the individual bites". He reflects on how he only experienced the full effect of the problem in his sixties, the prime of his career. Shepherd depicts the trigger point for himself as being ice cubes in his drink, which reminded him of "his work after the Bali bombings of 2002, when there was no refrigeration for the piles of corpses". His career, although exceptional in his line of work, had destroyed him mentally and this also spilled into his personal life—resulting in the breakdown of his marriage in 2007. However, after receiving professional help from counsellors, Shepherd has returned to normal, although he has retired from forensic pathology and his name was removed from the Home Office list of forensic pathologists in 2017.

Reality:

Forensic pathology is a rewarding profession that comes with many benefits (such as being able to work on fascinating and challenging cases) but does also have its drawbacks, the main one being the psychological stress and pressures that forensic pathologists undergo. Although this can be argued for most professions within the medical field (i.e. doctors, surgeons), forensic pathologists are more susceptible to greater impacts and consequences. As seen from the case study of Dr Richard Shepherd, constant exposure to dead bodies—that have been abused, mutilated or even decomposed—can be mentally degrading to anyone, no matter how strong minded they perceive themselves to be. Unlike doctors or surgeons, who see a balance between happiness and trauma in their patients, forensic pathologists are only exposed to the trauma. They do not get the chance to help try and save the patient, but only to examine the lifeless body to find the cause of death. Therefore, I would argue that forensic pathologists have the most challenging profession within the medical field.

Edited by Michael Lowe

Footnotes and Key Terms:

1. **Pathology:** the study of diseases and its causes.
2. **Histopathology:** the study of disease through the examination of tissues/cells under a microscope.
3. **Home Office Registered Pathologist:** a forensic pathologist that is registered with the General Medical Council and holds a license to practice within the UK and is then added to the Home Office Register.
4. **Post-mortem:** a post- mortem examination (also referred to as an autopsy) is the examination of a body after death.
5. **Phenotypic characteristics:** the expression of genes in an observable way.
6. **Torso:** the trunk of the human body.
7. **Sternum:** a long flat bone, found at the front of the chest, which connects the ribs.
8. **Cartilage:** is the resilient and smooth elastic tissue that covers and protects the ends of long bones at the joints.
9. such as the heart, lungs, thyroid glands, oesophagus and trachea.
10. such as the intestines, liver, gallbladder, kidneys, urinary bladder, pancreas and reproductive organs.

How can we be allergic to everyday items?

by Shivank Khare (Y12)

Why are some people allergic to everyday items? How do they develop these allergies? How do we prevent allergies? Through this article I will show how allergies arise and what treatments are possible for them.

An allergy is a condition caused by the hypersensitivity of the immune system to typically harmless substances within the environment. A wide variety of allergies exist such as hay fever, nut allergy, egg allergy etc.

Most allergies that arise are Type 1 Hypersensitivity reactions. Type 1 Hypersensitivity is also known as IgE mediated hypersensitivity since it deals with ¹**IgE antibodies**, it is also sometimes called immediate hypersensitivity since the reaction takes place almost immediately (within a couple of minutes).

Allergies take place due to the reaction of specific molecules from outside the body. These molecules are breathed in or ingested. The molecules can also come in contact with skin. These specific molecules are usually known as antigens – but in this scenario they are known as ²**allergens** since they cause an allergic reaction. The first encounter with the allergen is the *sensitization stage* and is usually harmless, however, the *subsequent encounter* is much more serious and where the symptoms begin to show.

Most of the allergic reactions that occur are due to a change in certain genes causing their ³**T – helper cells** to be more hypersensitive to specific antigens (that would usually not be recognised as foreign). This means it is usually due to a genetic condition that is passed down and common within the family.

Sensitization Stage:

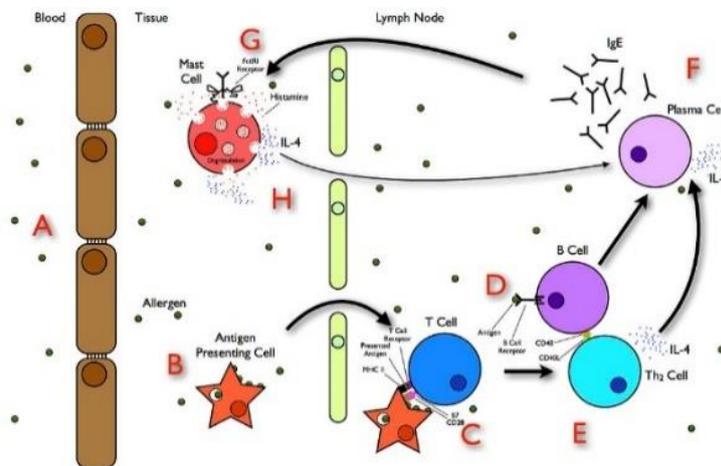


Figure 1: The Sensitization Stage

Let's take the example of nuts: A person could consume nuts without knowing he is allergic to them. The T – helper cells within the body are hypersensitive to specific molecules found on the nut making the molecule an allergen. The allergen is found in the membranes of the antigen presenting cells and needs to be transferred to the lymph nodes so immune cells ⁴**(antigen presenting cells)** transfer the allergen irrespective of whether the person is allergic to it or not.

⁵**Co stimulatory molecules** are found on the surface of the immune cells presenting the antigen which is required for an effective immune response. The (naïve) T – helper cell has never seen the antigen before despite being complementary to it. Once the T – helper cell binds to the co stimulatory molecule and the antigen then it differentiates and changes shape to form a T_{H2} cell. ⁶**Cytokines** present in the lymph nodes ⁷**(interleukin 4, 5 and 10)** help to change the T – helper cell's shape.

The changed helper cell also releases interleukin 4 which causes the antibodies on the B lymphocytes to change and hence start secreting IgE antibodies which are specific to the allergens found on nuts. The changed helper cell also releases interleukin 5 which activates eosinophils (a granulocyte which is a type of white blood cell) that releases toxic substances that can damage invading cells or even host cells known as ⁸**degranulation**.

The IgE nuts' specific antibodies have a high affinity for the receptors found on ⁹**mast cells** (a type of granulocyte) and hence bind to the surface of mast cells (they can also bind on receptors found on the cell surfaces).

Subsequent Encounter:

Let's say the person again consumed nuts for the second time (hence the subsequent encounter): The mast cell with the antibodies binds to the allergen. This signals the mast cells to degranulate and release many pro – inflammatory mediators such as ¹⁰**histamines**. The histamines can bind to H₁ receptors which causes the bronchi to contract leading to reduced effective ventilation. It also causes the blood vessels to widen and to become more permeable hence meaning increased blood flow. It also means more fluid leaks out and cause swelling known as ¹¹**oedema**.

Hence this results in inflammation. This is seen in people who have allergies since they usually complain about difficulty breathing and swollen lips. Other symptoms such as itchiness can also occur.

Treatments:

There are many ways to treat allergy symptoms, the most common being antihistamines. People perceive antihistamines as a drug to prevent or fully treat allergies but, they are used in order to reduce/ oppose the activity of histamine receptors and prevent inflammation from occurring. For example, histamines bind to H₁ receptors and cause the contraction of bronchi as previously mentioned. However, the antihistamines prevent the function of the receptors and hence the histamines can't bind to the receptors meaning no contraction of the bronchi.

However, this doesn't cure your allergy since if you were to have that substance again then the process would repeat and only be stopped once you've taken the antihistamine tablets. There are different types of antihistamine tablets - non drowsy or drowsy tablets and can be bought over the counter. Furthermore, there is a serious form of allergy known as ¹²**anaphylaxis** that is rapid and can cause death almost immediately. There are many symptoms of anaphylaxis (as shown in the image) such as skin rashes and itching.

Before being taken to the hospital sometimes an adrenaline auto injection is given to the patient. The release of adrenaline causes reduced vasodilation and reduces the permeability. This means less inflammation occurs and reduces the chance of the person from dying.

In the hospital steroids and antihistamines are given to try and reduce the reaction. Steroids target the site of inflammation and attempt to reduce it but are only given in very serious conditions.

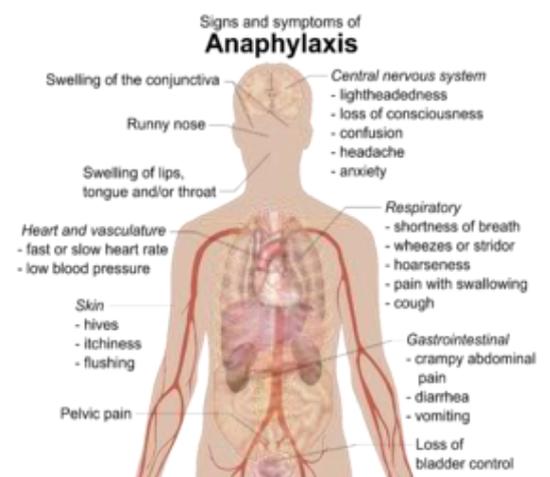


Figure 2: Symptoms of Anaphylaxis.

Another form of treatment is known as allergen immunotherapy. In this procedure small doses of the allergen are given, and the immune system reacts against it showing very minimal signs of symptoms. Then you start increasing the dosage and the immune system reacts to it better and better each time eventually making you immune to the allergy. However, this procedure can take a very long time.

Edited by David Kuc

Key Terms:

¹**IgE antibodies:** antibodies produced by the immune system as a result of hypersensitivity to an allergen.

²**Allergens:** an antigen that produces a significant abnormal immune response

³**T – helper cells:** A type of T cell (in an immune response) that recognises foreign antigens and releases cytokines to active T and B cells.

⁴**Antigen presenting cells:** Essentially immune cells that present antigens to the T helper cell.

⁵**Co stimulatory molecule:** Group of cell surface membranes that amplify the immune response.

⁶**Cytokines:** Substances secreted by certain cells of immune system that influence other cells.

⁷**Interleukin:** A type of glycoprotein used for stimulating immune responses.

⁸**Degranulation:** A process that releases toxic or harmful substances.

⁹**Mast cells:** A cell found in numbers in connective tissue for releasing histamine.

¹⁰**Histamines:** Organ nitrogenous compound involved in immune responses such as contraction of the bronchi

¹¹**Oedema:** Excess fluid within the body's tissues

¹²**Anaphylaxis:** An acute allergic reaction to an allergen due to the body becoming hypersensitive.



ENGINEERING- DID YOU KNOW THAT...

21st January, 2020 marked 50 years since the first commercial flight of iconic Boeing 747 in 1970. This feat of engineering was all designed and built in the space of just 18 months under an incredibly tight budget, yet the 747 was far ahead of its time and its enormous capacity revolutionised aviation to bring air travel to the masses. Today, 50 years on, the 747 is still a common sight at international airports across the world.

Human Powered Flight – Flapping your Arms doesn't Work

By Aditya Vishwanathan (Y12)

Flying is not an easy task – of the 70,000 species of animals, plants, fungi and bacteria in the UK, just over 600 are birds. However, we humans are trying our best – since the first airplane took off at Kitty Hawk in the USA to now, when aircraft can travel up to 17,000km non-stop at upwards of three times the speed of sound. But this feels like cheating – we *humans* are not flying, the aircraft is. The aircraft is the one designed to fly, not us – we humans are just passengers on board. Could we fly without needing an engine, motor or other form of propulsion?

Early attempts at self-powered flight did not end well. It is likely that people tried to imitate birds by flapping their arms in a doomed attempt to fly, but why did this not work?

There are four forces acting on a bird or aircraft: thrust (in the direction of travel), drag (against thrust), weight (down) and lift (up). Let us look at each force in turn.

The Thrust and Drag Problem:

Birds are able to fly through the design of their wings and muscles. Birds use their pectoral to flap their wings up and down. These muscles account for around 20% of a bird's mass so they are strong enough to enable the bird to fly; the human body does not distribute its mass in the same way. By angling their wings to flap not only down but also backwards, they push air down and backwards. The result is an equal force in the opposite direction (i.e. forwards and upwards), thereby producing thrust, propelling them forwards. If the bird is travelling at a constant speed forwards, its thrust is equal to the drag acting against it. The drag is given by the following equation:

$$\text{Drag Force} = 0.5 \times \text{Drag Coefficient} \times \text{Air Density} \times \text{Surface Area in contact with Air} \times \text{Velocity}^2$$

The drag coefficient is a value used to help with modelling the effect of the other factors affecting drag. It is calculated by dividing Drag Force by Air Density x Surface Area in contact with Air x Velocity². A low drag coefficient results in less drag force to overcome.



Figure 1: A Seagull in Flight

Humans have a problem with overcoming drag. The anatomy of birds is such that the amount of drag they encounter is minimal – they have beaks, they fold their legs in during flight, their heads are streamlined and although their wings have a large surface area when viewed from above, they are very thin when viewed from the front. This means the surface area in contact with the air (the fluid) is reduced. This means generating enough thrust to equal and exceed drag is easy for birds.

Humans, however, are not aerodynamic. We have large, blunt faces and nearly cylindrical arms and legs (which we can't even fold away). This means the drag force we encounter is immense.

The Lift Problem:

Birds (and aircraft) generate their lift through their wing shape. Their wings appear flat but actually, the top side is larger than the bottom side. This means air going over the wing has to travel further than air going underneath, so air above is faster than air underneath. Air travelling at a higher speed is at a lower pressure. Therefore, air above the wing is at a lower pressure, so the area of higher pressure (under the wing) will push the wing up, towards the area with lower pressure, causing the bird to experience lift. The problem for humans is our bodies are not aerofoil shaped. This means we cannot generate lift as birds can, confining us to the ground. However, generating lift is not our biggest problem.

The Weight Problem:

The main reason humans cannot fly is that we are too heavy. Our body is made up of dense, solid parts whereas birds have evolved to be as light as possible: their skeleton is often hollow, their feathers which help them get airborne are light, they do not have teeth, just a lightweight beak – even their diet is adapted since most birds tend to eat light, energy-rich food. All of this means that we are stuck on the ground, jealous of the birds above us.

An Example:

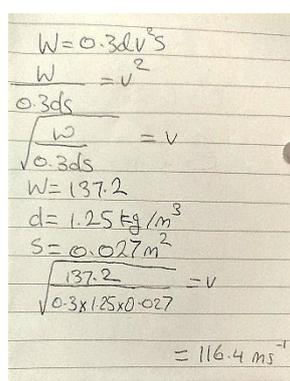
The human with the best chance is likely a child since they weigh less than adults. In this example I looked at how fast a child would have to travel to remain airborne.

The equation given focuses on weight since this is a much greater problem for humans to overcome – more so than the problem of drag. For level flight, weight must equal lift. The four forces affecting lift are angle of attack, air density, wing shape and airspeed and each component of the equation addresses one of these factors.

Weight = $0.3 \times \text{Air Density} \times \text{Velocity}^2 \times \text{Wing Surface Area}$ (or WSA) where 0.3 is a value related to the angle a bird or aircraft makes with the oncoming air, also called the angle of attack. The value 0.3 is related to an angle of attack of 6° which is roughly the angle of attack flying objects (both birds and aircraft) average on a long cruising flight. The WSA is measured by working out the surface area of the wing when viewed from above – not the underside of the wing.

Let us assume the mass of a child is 14kg. The weight of a 14kg child is equal to their mass times the acceleration due to gravity ($W = mg$). $14 \times 9.81 = 137.2\text{N}$ – the child weighs 137.2N. Values for the average surface area of a child varied since it depends a lot on height but average values were around 0.6 square metres. An arm is 9% of a person's surface area. To minimise drag, the child is going to be in a skydiver's position – torso to the ground, arms outstretched. WSA is only the surface area when viewed from above, so half the surface area of one arm, for two arms. $(0.6 \times 0.5 \times 0.09) \times 2 = 0.027$ square metres.

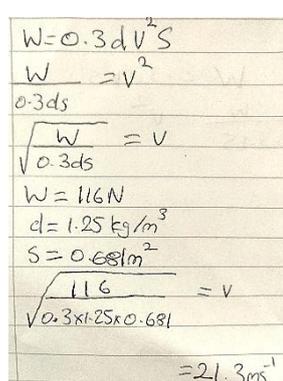
The values 0.3 and Air Density (let us take 1.25kg/m^3 , since that's the Air Density at sea level) are the same for both human and bird and for comparison with nature we will look at the Mute Swan, a bird with reasonably similar weight (116N) has a WSA of 0.681 square metres.



Handwritten calculation for a human child:

$$W = 0.3 d v^2 S$$
$$W = v^2$$
$$0.3 d S$$
$$\sqrt{\frac{W}{0.3 d S}} = v$$
$$W = 137.2$$
$$d = 1.25 \text{ kg/m}^3$$
$$S = 0.027 \text{ m}^2$$
$$\sqrt{\frac{137.2}{0.3 \times 1.25 \times 0.027}} = v$$
$$= 116.4 \text{ ms}^{-1}$$

Figure 2- Human Child



Handwritten calculation for a Mute Swan:

$$W = 0.3 d v^2 S$$
$$W = v^2$$
$$0.3 d S$$
$$\sqrt{\frac{W}{0.3 d S}} = v$$
$$W = 116\text{N}$$
$$d = 1.25 \text{ kg/m}^3$$
$$S = 0.681 \text{ m}^2$$
$$\sqrt{\frac{116}{0.3 \times 1.25 \times 0.681}} = v$$
$$= 21.3 \text{ ms}^{-1}$$

Figure 3- Mute Swan

Therefore, even if a child had arms in the shape of wings, they would have to travel at 418km/h to remain airborne. You, reading this article have no chance. Even if the rest of the child's body visible from above (and therefore counting towards the WSA) was considered, the surface area would only be 0.3 square metres, meaning the required velocity would have to be 34.9 m/s, 50% more than the swan.

The Solution:



Figure 4- The Gossamer Condor with pilot pedalling

Humans are an innovative species – since history began we have made tools and machines to aid our relatively physically weak bodies. Therefore, self-powered flight may be possible using machines that we power with our bodies; an aircraft truly powered by humans, since 1961 they have existed when an aircraft developed at Southampton University powered by a pilot using pedals to drive a propeller. In subsequent attempts, aircraft improved. In 1977 Bryan Allen flew the Gossamer Condor, developed by American engineer Paul MacCready in a figure-of-8, winning the Kremer Prize for human-powered flight and in 1979 flew the Gossamer Albatross across the English Channel in just under three hours.

Since then various competitions and experiments have occurred, but it will be a while before we can take to the skies by ourselves at will.

However, that day is getting closer.

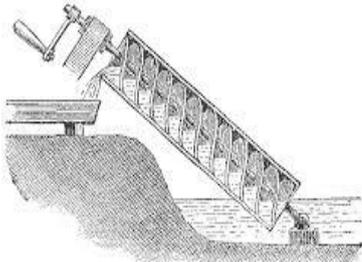
Edited by Ray Wang

Screws, Nuts and Bolts: Engineering the World

by Divy Dayal (Y11)



Chiselled and strong, groovy and curvy, lean and flat-headed. ¹The screw is the modern era's underdog, acting as the *cornerstone* of today's infrastructure, *twisting* its path into the best inventions of mankind, *putting together* fundamental topics of physics and engineering. This carbon steel corkscrew with a flat top simplifies today's engineering. Drilled, hammered and screwed, this simple machine literally puts the world together.



Archimedean Screw.

The humble screw's magic is down to its grooves, which wedge themselves into the material, thereby preventing it from falling out. The basic principles echo ²'Archimedes' Screw' (or the Egyptian Screw – named after those who used this technology well before his time to transport water from low-lying channels to irrigation ditches). The simplicity of the screw is perhaps what makes it so prevalent, since this easy to use, easy to produce and in high demand technology satisfies all economic objectives of a successful product.



It is difficult to say whether the screw's success is down to the global adoption of it, or the ingenious design since both appear to contribute equally to its importance. "Lefty Loosy, Righty Tighty" is perhaps the most famous and useful phrase that rings throughout the engineering world. The simple design makes it fool proof, and thus useful in any application, from a rake to a racing car, axe to an aeroplane.

Moreover, this small machine is coupled with the ³screw driver, and the two work in conjunction like a nut and a bolt. This synergy results in millions of uses, perhaps only surpassed by magnets and batteries.

While successful, the screw has stiff competition, primarily from the nut and bolt. Typically a hexagonal metal hoop, with the same Archimedean grooves on the inside, and a metal elongated cousin of a screw (with a flattened, groove free tail), this combo is perhaps more useful as a means of fixing something in. Developed near the first century, this evolution of the screw is rather remarkable since it doesn't require an external screwdriver (insertion may be achieved entirely by hand). This leads to their greater spread in DIY furniture.

While it's interesting to note on the evolution of the screw, something more remarkable is mankind's ingenuity over the ages. It may have taken 3,000,000 years for humans to evolve from *Ardipithecus Ramidus* (who made simple flint stones for meat cutting) to *homo erectus* (who made specialised flintheads for fighting) but only 58 years from flight to space. *Engineering has built its way up in the world.* The truth is that we live in perhaps the most successful time for mankind in history, and perhaps in the future too, and it's partly down to the humble underdogs of engineering – screws, nuts and bolts: engineering the modern world.

Edited Utkarsh Sinha

Key Terms:

1. **Screw** – a fastener which a helical groove that is used to fix and hold something.
2. **Archimedes' screw** – an ancient technology that was initially used to transport water p, by multiple helical paddles that rotated upon a man-powered axle.
3. **Screwdriver** – a simple machine which increases moment, grip and can have a magnet top – all of which make twisting the screw easier.

Personal Transportation – Where we’ve been and where we’re going

By Harsh Sinha (Y12)

For as long as humans have existed, we have needed to get places, be it in order to hunt for food or to socialise. However, in the last two centuries, the monopoly horses and camels held on the market has been shattered through the age of human innovation. In this article, I will be going through the various novel ways we have devised to make our movement as efficient as possible, with a focus on transport intended to carry one or two passengers.

The Ghosts of Transport Past: The Bicycle

The first verifiable example of a steerable, human-powered-and-balanced two wheeled contraption comes from Germany in 1817, when a civil servant to the Grand Duke of Baden came up with his *Laufmaschine* (German for “running machine”). The device was widely reported by press in both France and England and nicknamed the Draisine (after the maker, Baron Karl Von Drais). Drais proceeded to patent his design the following year, with production started in Germany and France, However, it took several iterations and years for the bicycle to look anything close to what we’re used to seeing on roads today. In 1889, an African American called Isaac R Johnson patented his own design for a folding bicycle, which used something called a *diamond frame* (figure 2). As an African American inventor before the equal rights movement in America, Johnson didn’t expect anything great to come from his invention. However, contrary to his expectations, the diamond frame has gone on to become the basis of almost all bicycles to this day.



Figure 1 – the Laufmaschine

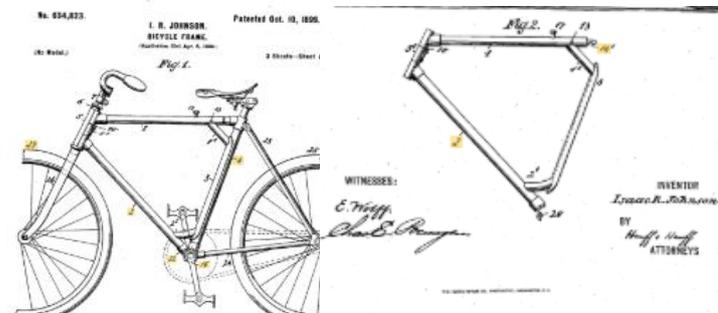


Figure 2 – Original drawings from Johnson’s patent

The Peel P50:

Three-wheeled, available in Daytona White, Dragon Red and Dark Blue (pictured above) and measuring only 134x98x100 cm, the Peel P50 had its initial production run from 1962 to 1965 in the Isle of Wight by the Peel Engineering Company, over which only 50 were made. Of that original run, about 27 are thought to still exist. Marketed at the time as “cheaper than walking”, giving up to 100 miles per gallon and costing only £199 to buy (£6,600 when you consider the cost with inflation today). It was powered by a tiny 49cc engine, producing 4.2 horsepower and capable of propelling the single occupant to a top speed of 38mph. Though barely any of these cars were sold, it seems to have been ahead of its time, with original examples now fetching upwards of \$140,000 at auction. The Peel P50 has had production restarted since 2011 at the original factory, with two new powertrains being introduced to meet new emission regulations. The Peel P50, a vehicle created in 1963, has the potential to be the perfect vehicle for the future.



Figure 3 – The Peel P50



Figure 4 – the P50 at speed

Where we're going: Teleportation



Figure 5 – Jetpack Joyride teleporter



Figure 6 – Star Trek teleporter

Teleportation has long been a commonly used tool in sci-fi culture to depict a future in which humans are much more technologically advanced than we are today, with the equipment required to transport wherever we want in an instant. However, it has long been the belief that teleportation of any kind is impossible, and that it breaks several fundamental laws of science and common sense. Thankfully, scientists have been working away despite this to try and crack teleportation and, in 2016, two separate teams of scientists in Canada and China succeeded at something called *quantum teleportation*. This isn't the teleportation of matter, but instead the teleportation of quantum information, with the state being transferred to a different physical location. This is essentially a high speed transfer of data, rather than the transport of matter, which may make it seem irrelevant when considering the end goal of achieving teleportation for humans.

However, a 1993 paper called "*Teleporting an Unknown Quantum State Via Dual Classical and Einstein-Podolsky-Rosen Channels*" written by Charles. H. Bennett and his team from IBM, described that the key requirement for teleportation to work is for every single piece of information about each particle to be transmitted perfectly and in relation with those around it. The quantum teleportation technology used by the scientists in China and Canada uses a simplified version of this technology which can, conceivably, be implemented in the future to lead to the transportation of physical matter. However, as it stands, teleportation requires the transfer of far too much data at impossible speed with invariably fatal consequences. Hopefully, the technology will mature enough in the future to make this sci-fi pipe dream a reality.

Edited by Ray Wang

Running on Uranium: a Look into Nuclear Powered Vehicles

by Ray Wang and Kevin Luo (Y12)

The year was 1954 and the US had just launched an absolute beast into its seas. It could lurk deep within the oceans for months on end and there was no way of knowing if it was halfway across the world or right on your doorstep. This was the USS Nautilus; the first ever nuclear submarine and it was set to revolutionise the capabilities of the submarine. Today, submarines that bear the most powerful nuclear weapons known to man within their hulls, are on constant standby, ready to strike, and bring destruction to millions at only moment's notice.

Just ten years before that, during the Second World War, submarines were hardly the threat we think of today. These submarines were powered by diesel engines and had on-board batteries to power the electrical systems. They could only remain underwater for a mere 48 hours before they had to resurface to refuel, recharge their short-lived batteries and take on air, severely limiting the operational capabilities of the submarines and sort of defeating its whole purpose, which was to stay undetected.

From this quite drastic comparison we can see the extent to which nuclear technology has revolutionised the ways submarines work. However, it begs the question, if nuclear propulsion has worked such wonders for the submarine, why isn't the same technology applied to other vehicles? Believe it or not, there was a time in history where we truly believed that every vehicle would go nuclear. Cars, planes, trains were all set to have their own built in reactors which would be able to power them to a virtually limitless range. There are a whole range of concept nuclear vehicles and prototypes that never made it to commercial production.

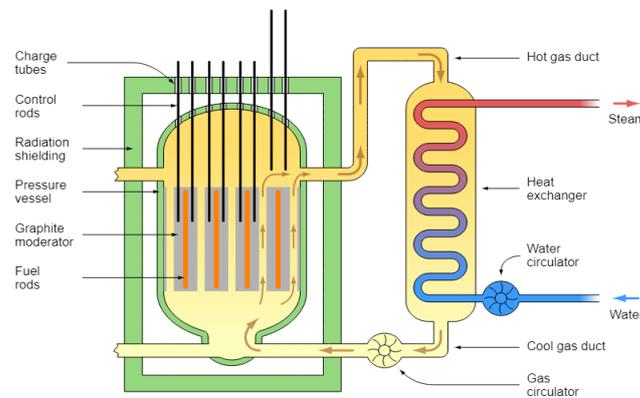


Figure 1: Interior of a Nuclear (Fission) Reactor

But before we delve into that, let's take a look at how a nuclear reactor actually works. Although the reactor may take different forms, the components which make each of them up have little variance. The process of nuclear fission is essentially where a heavy nucleus splits on impact with another particle giving off kinetic energy in the process which in turn increases temperatures in the reactor dramatically. In a nuclear reactor, fission begins by firing neutrons at some form of nuclear fuel, such as uranium-235, the most commonly used nuclear fuel. Nuclear fuel consists of materials with heavy fissile elements. These are elements with a heavier nucleus, meaning that the material is capable of sustaining a chain reaction.

The graphite moderator acts to slow the neutrons so that they can be absorbed by the uranium-235 nuclei causing them to fissure, breaking into two daughter nuclei and releasing energy and neutrons which go on to cause other nuclei to fissure. For this reason, nuclear fission is a chain reaction. If the graphite moderator was not present, the reaction would not be maintained as the neutrons would just bounce off the nuclei rather than be absorbed. The control rods act as a method to control the rate of the reaction by

lowering them down into the reactor due to them absorbing neutrons; this prevents nuclear reactions turning into an uncontrolled reaction (which is what happens in a nuclear bomb). When the control rods are fully lowered, the chain reaction stops as all neutrons are absorbed. A series of energy transfers then takes place: The kinetic energy produced by the process of fission raises the temperature of the coolant gas, which is then used to heat up water in order to turn it into steam. This is then used to turn turbines, powering generators transferring the thermal energy into electrical energy.

A nuclear reactor could quite easily be used to power various vehicles through different mechanisms. Conventionally, all these vehicles are powered by internal combustion engines of some sort, which use combustion of fossil fuels to generate heat, which in turn causes expansion of gases to turn a turbine-like mechanism (for example in cars, this expanded hot gas drives a piston attached to a crankshaft). Another way nuclear power could be harnessed within vehicles is by having what essentially would be a nuclear power station on board. Electricity would be generated from the nuclear reactors and turbines which would then be used to power electrical motors that provide the vehicle's propulsion.

The setup here shows how nuclear propulsion is used on board some ships. You'll notice there are two turbines; one turns the ship's propeller through a series of gears and a driveshaft while the other is connected to an electrical generator to provide electricity for the systems on board. The nuclear reactor here, has exactly the same setup as the one seen before in the power station. Conventionally, these turbines were powered by burning fossil fuels to heat up water into steam to turn the turbines.

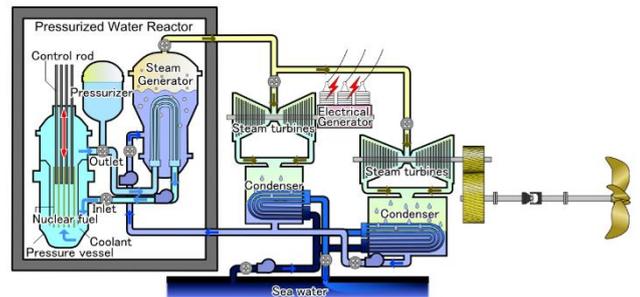


Figure 2: Nuclear Propulsion in Ships

So, if we could so easily power submarines and ships with nuclear systems, why haven't there been similar solutions implemented for other types of vehicles? There have been plenty of concepts such as the Ford Nucleon pictured here. This would work in the same way as the ship system seen above, but with a much smaller reactor located in the rear of the vehicle. It was proposed to drive two turbines, one connected to the wheels and another for electricity generation.



Figure 3: Ford Nucleon

Another potential vehicle was the X-12, a proposed behemoth of a train locomotive that would stand at fifty metres long and generate four times the horsepower of a conventional diesel loco. This system would work with the reactor driving turbines that linked to generators and the electricity was then used to drive motors for the wheels. But while these projects erred on the side of science fiction, the nuclear-powered plane was a very real possibility.

There were two potential ways in which a nuclear reactor could be incorporated into jet engines. Both worked by essentially replacing the combustion chamber of a conventional jet engine with a nuclear reactor, i.e. using a nuclear reactor to heat incoming air as opposed to combusting it. (If you'd like an in-depth explanation as to how jet engines work, check out my article on jet engines in issue 1) The first was the direct cycle engine whereby air would be drawn from the jet engine and pumped into the reactor core. This air would serve two purposes, acting as a coolant for the reactor core so that it doesn't melt while the core also heats up the air. The hot expanded air is then pumped back into the jet engine where it exits the engine as exhaust generating thrust.

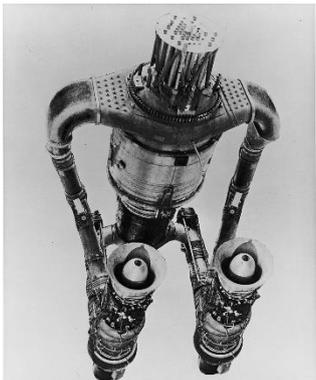


Figure 4: Direct Cycle Engine

The obvious issue with this setup is that pumping air through the reactor core would inevitably result in the air becoming irradiated meaning that the jet engine would leave a trail of radioactive fumes spewing out behind it which wouldn't do the people its flying over any favours.

The alternative method was the closed cycle system which uses a liquid to air heat exchanger to indirectly heat the air passing through the jet engine. In this setup, a molten salt is used as a liquid coolant, serving the same purpose as the air in the reactor core in the first method. The liquid coolant is heated by the reactor core while cooling it at the same time; the hot coolant then passes its thermal energy onto air passing through the main jet engine. As the molten salt is used in its own closed loop, the air passing through the jet engine would not get contaminated but it came at the price of a far more complex system with a much more plumbing and reduced efficiency as a result of the extra thermal energy transfer stage in which a lot of energy would be lost to the environment.

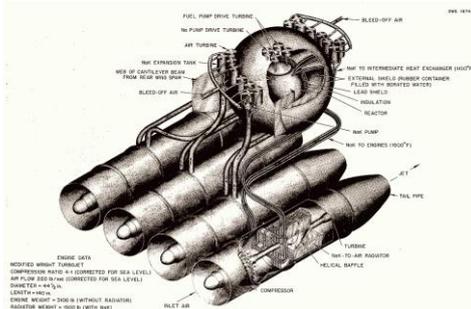


Figure 5: Closed Cycle System

There is a pattern you may have started to notice by now. It is the larger vehicles that have come further in development process than the smaller vehicles. It would appear that it is more difficult to implement a smaller nuclear reactor inside a smaller vehicle than larger reactors for larger vehicles and there are a few reasons for that.

Firstly, for any nuclear reactor to work there must be more than a minimum amount of nuclear fuel inside the reactor required for nuclear fission to take place, as for a chain reaction to happen, there needs to be an excess of neutrons. This is known as the critical mass.

This ties into another important factor to consider: the concentration of fissionable nuclei in the fuel. For a smaller reactor to have the same power output as a larger one, it must still have the same number of fissionable nuclei in its reactor core, so the concentration of fissionable nuclei must be higher. In other words, smaller reactors must use nuclear fuel that is more enriched and inherently a lot more expensive.

What this meant for nuclear planes was that the power to weight ratio of these engine systems was simply too low to get an aircraft off the ground. While nuclear fuels are incredibly energy dense, meaning nuclear reactors can provide enormous amounts of energy over their lifetimes, the power output of a reactor has a limited maximum. This is limited not by the process of fission itself but instead by the heat the cooling system can transport away from the reactor core before it reaches temperatures so high the reactor starts to melt and the control mechanisms get damaged (which is precisely what happened in the Fukushima disaster in 2011). It was therefore difficult to make a nuclear propulsion system powerful enough to generate enough thrust to be able to lift an aircraft off the ground.



Figure 6: NB-36H Atomic Plane

However, the most significant factor isn't to do with the nuclear fission process itself. It is actually quite easy to make a relatively small nuclear reactor, there was one in the first atom bomb and that fit comfortably on a plane. The difficulty lies in creating a reactor that is safe, controllable and can generate enough power. The nuclear reactor in an atom bomb doesn't need all the reflectors, blankets, shielding, coolants etc. that are required to make a controllable and safe reactor; while if we are using the reactor to power vehicles, these additional features are absolute musts.

Take the experimental NB-36H atomic plane for instance, while it never flew with power from its nuclear engines, it was designed to accommodate fully for its needs. In the image here, you can see the crew compartment of the aircraft was completely isolated from the main fuselage and encased in heavy lead and rubber to shield the crew from the radiation that would be generated from the reactor. In addition to that, there was a stainless steel and lead shell that encased the reactor itself.

This shows the lengths engineers had to go to in order to make the system safe and even then, it was deemed too much of a risk to turn the engines on. And they had a right to be cautious. Later, it was discovered that the Soviets, in an act of desperation, believing that the US were close to developing a fully operational nuclear aircraft, actually went ahead and flew a nuclear aircraft of their own during the 60s. The modified Tu-95 bomber with direct cycle engines and no shielding and it see worked, flying just as expected but the side effects on the crew were also just as expected. Within just three years of test flying, some of the crew had died from the radiation exposure due to lack of shielding.

This clearly shows the Achilles heel of nuclear power in vehicles, it was easy to make an aircraft that could work with a nuclear propulsion system, but making that system safe was another matter entirely.

All this talk of nuclear vehicles was over half a century ago and was born out of a massive arms race during the Cold War. With the increased development of ICBMs (Intercontinental Ballistic Missiles), the superpowers of the world could annihilate each other without having to fly a plane deep into enemy territory and with that there was no longer a necessity for a nuclear bomber with an almost infinite range. Additionally, the invention of aerial refuelling extended the range of conventional aircraft without the billions that would be required to develop a completely new propulsion system. As for other forms of transport, there were far more practical solutions for civilian vehicles. People simply didn't need to have a nuclear reactor under their bonnet, which could cause an all-out catastrophe in case of the all too common car accident, when gasoline worked perfectly fine.

However, it begs the question, after fifty years of technological advancements, could we really build nuclear powered vehicles in the future, especially as we are warned of the ever pressing threat of climate change and fossil fuels running out? Well, nuclear reactor technology has come so far in the past fifty years. We are now able to build reactors that are far smaller in size and examples of these have been used to power space projects. Advances in materials science also mean that materials required for shielding can be made to be a lot lighter and take up far less space while there are materials that can be used to withstand much higher heat allowing a higher power output from the nuclear reactor. There is also the potential to use other nuclear fuels that are more efficient and/or safer than Uranium-235 such as Thorium or molten salts or perhaps even fusion instead of fission in the future. Whether portable nuclear reactors will make an appearance in vehicles of the future is an exciting prospect with there already being plans to build nuclear trains as well as surveillance drones. Who knows what will be powering our vehicles in the years to come.

Edited by Devanandh Murugesan

Space Shuttle Challenger: The Disaster that Changed NASA

by Neel Patel (Y11)

On 28 January, 1986, the NASA Space Shuttle *Challenger* exploded 73 seconds into its mission, killing seven crew members; five astronauts; a payload specialist and a civilian schoolteacher. It was built to serve as a test vehicle for the Space Shuttle programme but unbeknownst to anyone, the disaster had several detrimental consequences.

Causes:

Rockwell International (an aerospace manufacturing company) began to build *Challenger* in November 1975, and it was then sent to Lockheed Martin (an aerospace technology company) on 2 April, 1978 for the purposes of structural testing and development. The first major threat to the shuttle's success was the lack of advanced computer systems to calculate the stresses on the shuttle during different points of its journey.



Furthermore, scientist Richard Feynman found the chance of failure of the shuttle to be around 1 in 100, a stark contrast to the 1 in 100,000 stated by NASA officials. He was able to investigate after the tragedy and learned that the rubber used to seal the rocket booster joints using O-rings, was unable to expand at temperatures at or below 0°C – this was the temperature at the time of the launch. To test his hypothesis, Feynman dropped a piece of the O-ring material (squeezed with a C-clamp to stimulate the actual conditions of the shuttle) into a glass of ice water. He reached the following conclusion:

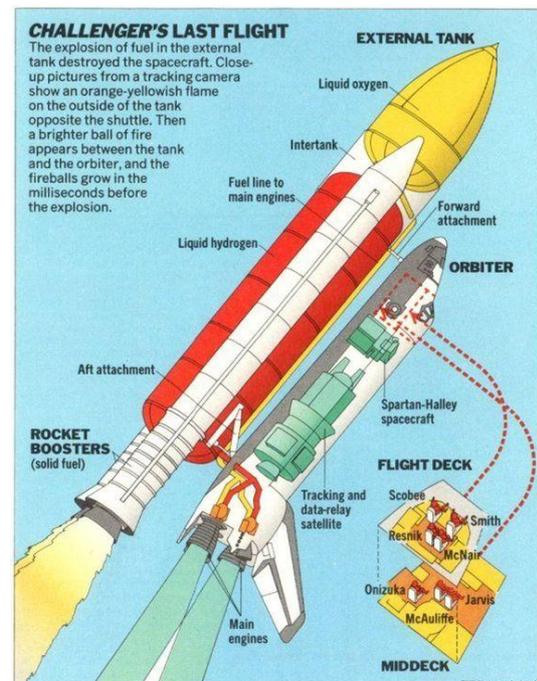
Since the O-rings could not expand, the gas was able to find gaps within the solid rocket booster joints, causing the gas to escape. The leak was visible as a small flicker, which soon transformed into a flame and began to heat the fuel tank, causing it to rupture. The fuel tank then released liquid hydrogen into the atmosphere, where it exploded.

Finally, to make each solid rocket booster, the Morton Thiokol factory (builder of the solid rocket boosters) built four hull segments filled with powdered aluminium (fuel) and ammonium perchlorate (oxidiser). They believed that O-ring seals in the solid-rocket boosters would perform adequately in the cold weather – an expectation which they sadly failed to meet. The O-rings were therefore never tested in extreme cold, and on the morning of the launch, the cold rubber became stiff and failed to completely seal the joint.



Effects:

On the morning of 28 January 1986, due to the freezing temperatures (0°C), engineers were concerned about the integrity of the seals on the solid rocket boosters. Nonetheless, *Challenger* took to the skies at 11:38 a.m. (EST). The presence of a civilian school teacher, named Christa McAuliffe, generated a buzz of media attention. However, the Space Shuttle *Challenger* tragically broke up precisely 73 seconds after its launch. Pieces of the shuttle fell from the sky into the Atlantic Ocean. Salvage crews spent several weeks recovering the pieces, and carefully preserving the remains of the crew. Identifiable remains were given to the families, while the rest were buried at Arlington National Cemetery on 20 May, 1986 in a monument to the Challenger crew. A presidential commission was convened to investigate the disaster, headed by a previous Attorney General and Secretary of State, William Rogers. It also included NASA astronauts Neil Armstrong and Sally Ride.



Results:

The commission report focused on the technical causes of the accident, pinpointing this to the cold weather degrading the seal on the solid rocket boosters designed to assist in bringing the shuttle into orbit. Moreover, it showed the cultural problems at NASA, such as the subpar communication and reporting of problems to the launch decision team. The commission also found that the proposed flight rate of the shuttle was unsustainable due to the size of its crew. In the wake of the Challenger disaster, NASA made many technical changes to the shuttle and also worked to improve communication within its workforce. The shuttle program (for which *Challenger* was a test) resumed flights in 1988. *Challenger's* explosion did however change the space shuttle program in several ways.



Firstly, plans to fly civilians in space were halted for 22 years, until McAuliffe's backup – Barbara Morgan – flew aboard *Endeavour* in 2007. Secondly, astronauts were no longer used to repair satellites, and thirdly the Manned Manoeuvring Unit was no longer flown as a result of a new focus upon astronaut safety. *Challenger* has left an educational legacy in the form of the Challenger Center for the Space Science Education programme, founded by members of the crews' families. It brings students on stimulated space missions. After the wreckage was examined, most of the pieces were buried and sealed in the abandoned Minuteman missile silos at Cape Canaveral Air Force Station, where they remain today.

Edited by Utkarsh Sinha

Tesla Inc. – Moving on and getting over

by Utkarsh Sinha (Y12)

“Buyers of the affected vehicles have become beta testers of half-baked software that renders Tesla vehicles dangerous if engaged.” An example of just one of the many demons facing Elon Musk (Figure 1) and his project, Tesla Inc., comes from a lawsuit filed by law firm Hagens Berman in April 2017. This criticism is aimed towards a feature that is arguably still the firm’s USP today, but was much more so two years ago; the driverless technology released as a novel but vital part of Tesla’s Model S platform was considered by an incredibly vocal opposition, to be nothing but a dangerous marketing gimmick. Tesla responded, claiming that this was an “inaccurate and sensationalistic” view of their technology.



Figure 1: Elon Musk

Since then, Musk has released hundreds of unofficial press statements via Twitter, and his company three developments of the “Autopilot,” software, which has reportedly worked successfully the vast majority of the time. During the years since the lawsuit, the story of Joshua Neally came to light – he suffered from a pulmonary embolism while on the way to Washington DC, rendering him incapable of driving. He relied upon the Autopilot software to drive him to the exit of the highway, allowing him to reach a hospital safely.

More criticisms can be found within a damning statement from Susan Rigmaiden, who had previously been an integral part of the health and safety team (and manager of the environmental compliance team) within Tesla Motors. She eventually left her role within the company due to her belief that there was a laissez-faire approach to safety and no desire to fix it; her conversations with key management staff often led back to the same name appearing repeatedly – that of chairman and CEO Elon Musk.

“Elon does not like the colour yellow,” she was told when she questioned the lack of (traditionally yellow) safety tape and marking. She finally reached the conclusion that there was little she could do to fix the issue as she watched multiple near-collisions between cars and people every day. The number of injuries within Tesla factories still far exceeds both the threshold required by the industry as well as the average within other similar businesses.

	2015	2016	2017
Tesla total injury rate	8.8	8.1*	6.2
Automobile industry total injury rate	6.7	6.2	Not yet available
Tesla serious injury rate	7.9	7.3*	5.2
Automobile industry serious injury rate	3.9	4	Not yet available

*Amended rate

Sources: Reveal analysis of Tesla Inc. injury reports and U.S. Department of Labor data.

CREDIT: GABRIEL HONGSDUSIT/REVEAL

Having emerged shamefaced but relatively unscathed from these, and other, controversies, Tesla Motors was renamed to Tesla Inc., as a reflection of the changing interests of both Musk and the company as a whole, which now include energy storage and production for households, and a general focus on cleaner energy production (see the Powerwall and Powerpack concepts).

In fact, Musk's business savviness is most evident outside of the automotive industry, where most people are used to seeing him; his plans to build a monumental "Gigafactory" were centred mostly around the idea that economies of scale would reduce the cost of production for the batteries in Tesla vehicles by up to 30% - savings that could then be passed onto consumers. Musk has claimed that 100 Gigafactories would be required to supply enough power for the entire human population, for one day, although it is difficult to confirm this due to the varying production levels of the three existing examples.

The Cybertruck:

Perhaps the most recent and relevant news for most readers is the release of the Cybertruck, an understandable development since Ford's F-Series pickups have been the best-selling vehicle in the United States for the last 42 years, throughout various iterations and financial climates. Indeed, the launch was largely successful despite the design reminding some of early video games, and an unsuccessful demonstration of the "bulletproof" glass windows. Tesla stock prices fell dramatically following the launch, but rose again following Musk's Twitter reveal that 250,000 people had placed deposits, which would hopefully be converted into sales once the pre-order period ended.

The Cybertruck is due to be released to the public with a starting price of \$39,000 for the least powerful of three variants. As with other offerings from the company, there is the option to add full "Autopilot" capabilities for a fee and similarly astonishing performance figures, with the most powerful variant apparently capable of reaching from 0 to 60 mph within 2.9 seconds. The affordable pricing range, aggressive marketing and current climate regarding environmental affairs, are likely to mean that the project is profitable, despite people like myself attempting to steer my own parents away from looking at Tesla's vehicles until recently.

What next?

Musk has already published plans to start a car-sharing service that allows Tesla owners to make money by renting out their personal vehicle to release more affordable vehicles to address "all major segments" and manufacture solar roofing panels that are attractive and effective for residential properties. Despite the controversies facing the company, it seems likely that Tesla Inc. will continue just as it has since its inception – moving on and getting over.

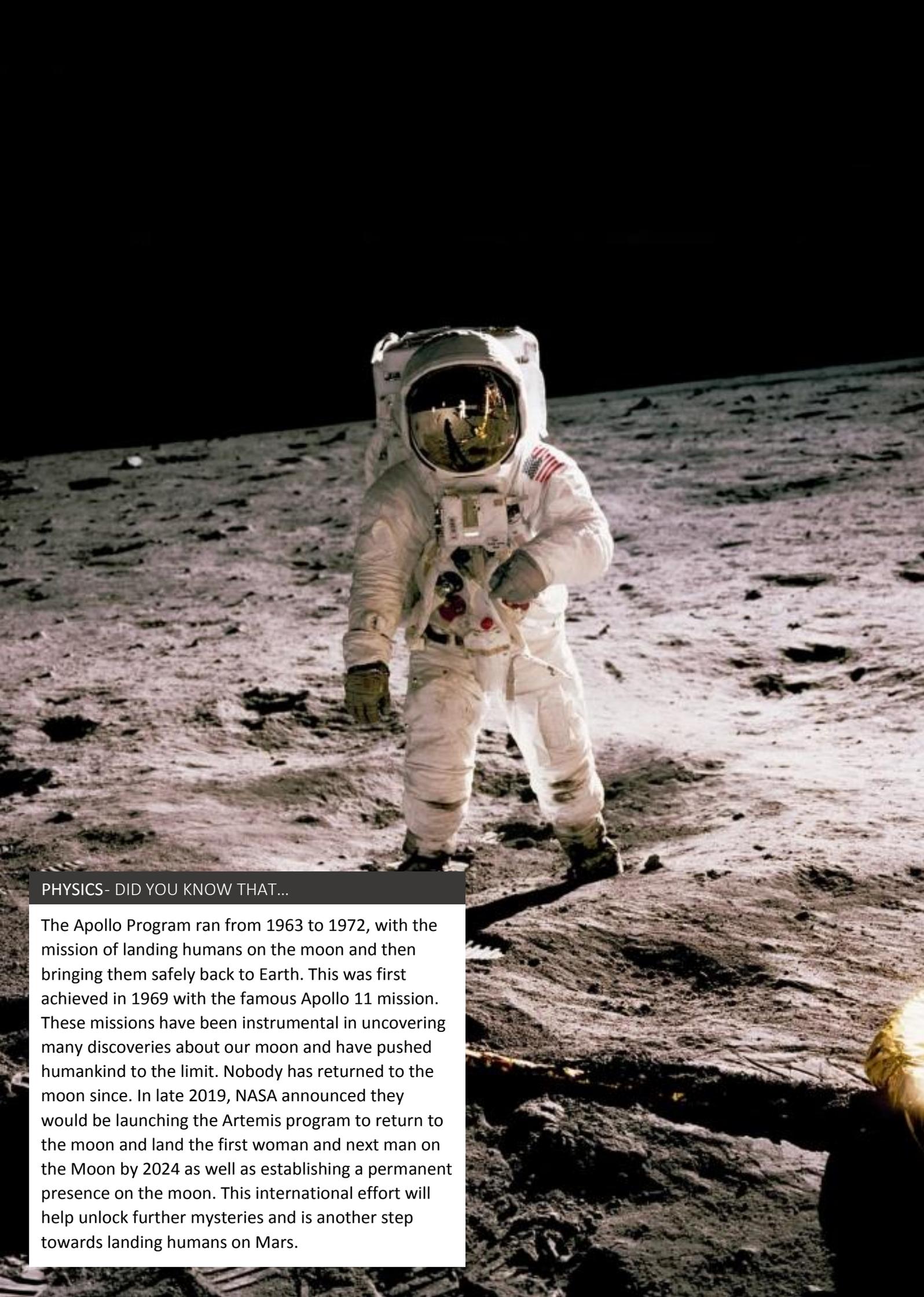
Edited by Devanandh Murugesan



Figure 2: Becca Farsace – Gigafactory, Nevada



Figure 3: Tesla - Cybertruck



PHYSICS- DID YOU KNOW THAT...

The Apollo Program ran from 1963 to 1972, with the mission of landing humans on the moon and then bringing them safely back to Earth. This was first achieved in 1969 with the famous Apollo 11 mission. These missions have been instrumental in uncovering many discoveries about our moon and have pushed humankind to the limit. Nobody has returned to the moon since. In late 2019, NASA announced they would be launching the Artemis program to return to the moon and land the first woman and next man on the Moon by 2024 as well as establishing a permanent presence on the moon. This international effort will help unlock further mysteries and is another step towards landing humans on Mars.

The Strange Nature of Everything

by Boyu Xiang (Y11)

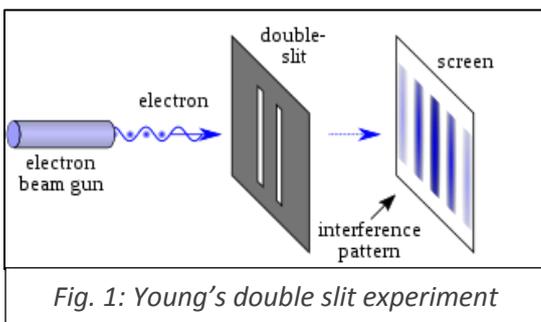
What is light? In school, we are taught that light is a wave that is detected by our eyes, enabling us to see. However, if you have read almost any book about quantum physics, or even glanced at the standard model of particles, you would be aware of “photons”, a particle which is responsible for light. With seemingly contradictory evidence, it makes one wonder about what the true nature of light really is - is light a wave or a particle? According to the wave-particle duality, it can be both.

A logical mind may be opposed to this notion - how can light be described in such fundamentally contrasting ways? Perhaps the knowledge that other “particles” have been described in a similar manner (that the wave-particle duality does not apply exclusively to light) would ease one’s mind. Electrons, for example, are well documented as having this wave-particle duality. In fact, everything with mass, as well as some things without mass (such as light - perhaps it would be more accurate to say everything with *momentum*) exhibits this quality. This even includes large objects such as planets, though their behaviour is so much like a particle that the “wave” aspect of its wave-particle duality is ignored for the most part. It is only in the subatomic mass range where wave-like behaviours can be observed.

For light to behave both as a particle and a wave, we need to first define how particles or waves should behave. The simplest explanation of a particle is to compare it to things which they all make up - physical objects. How do physical objects interact, then? Well, two objects cannot occupy the same space as each other at the same time - when forced to interact, they bounce off each other, exchanging momentum. Particles also have location that can be measured more or less accurately, waves however do not have a mass and do not occupy a space, you can find out roughly where it is by seeing how it affects other things, but do not have a set location as a particle does.

Waves are fundamentally different, since they do not have a mass. They are oscillations that carry energy which, instead of mass, is what provides their momentum. As they do not have mass, they can occupy the same space as each other (although they do not occupy space in the quite the same way as particles), but when they meet, they do interact on each other (which is called “interference”). As they don’t have a mass and don’t occupy a space (beyond the medium that they travel in), it is impossible to pinpoint an exact position that a wave is in - we can only know the direction it is going in, and the rough area in which it currently is.

The aforementioned logical mind that one might have would be extremely confused upon learning this - how could light both bounce off other light particles and not do so at the same time? How could we both know where light is, and yet be unaware of its location? “Particle-wave duality” seems impossible, or at least hopelessly obscure and unscientific.

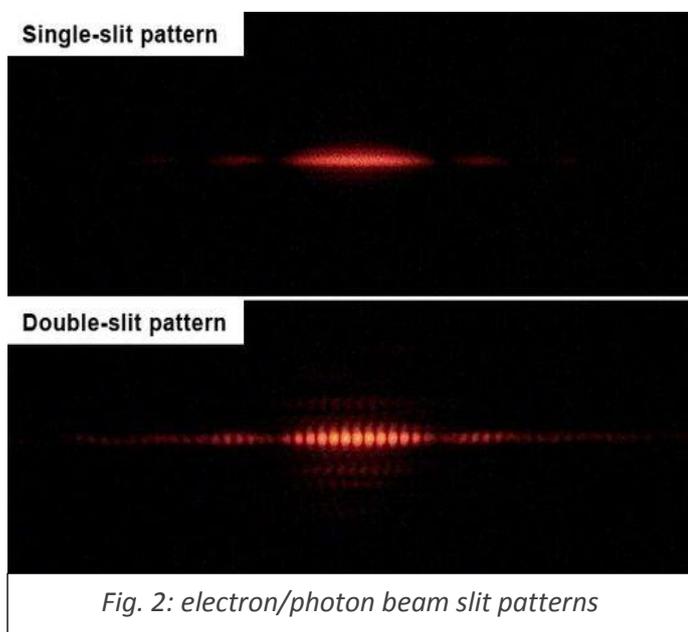


In actuality, the name “wave-particle duality” may be a misnomer, as it is not arguing that light is both a particle and a wave, but rather that in certain scenarios, light behaves like a particle rather than like a wave (or vice versa). Using this more accurate definition, it makes more sense that light can be both a particle and a wave.

Experimental data can hold up to scrutiny much better than theoretical propositions, so it would be amiss for this article to not touch on the plethora of experiments proving this fact.

Thomas Young’s famous and instrumental “double slit” experiment provided what seemed like conclusive proof that light was a wave. As shown in (Fig.1), light was shone through a screen with two thin slits. What was shown on the other side was *not* two clear lines as would be expected if light were a particle. This shows evidence of interference, as common of wave interactions as opposed to particle interactions.

In the diagram above, we can see evidence of “destructive interference”. To better explain this type of interference, we can imagine this: instead of two slits letting light through, we have two light sources. These sources emit light like two ripples in the water, and when they interact with each other, destructive interference occurs at specific locations. Destructive interference is when the crest of one wave coincides with the trough of another wave, cancelling each other out if they are of the same wave length. What we would see is a beam of light, separated by a few lines with no light where destructive interference occurs, like a block of cheese, cut precisely with a kitchen knife, which was the pattern observed in the experiment.



As you may have noticed, the diagram provided does not show that Young used a laser, but rather an electron beam used in tests done after the original. Interestingly, electrons acted in the same way the light did, showing that it is not just light that displays such peculiar behaviour. This has been done with larger “particles” as well, however it has become exponentially harder to yield similar results as the size of the object in question increases; the largest of such entities that replicated Young’s results were molecules composed of 810 atoms.

A rather more interesting phenomenon was also noticeable here - the photons were detected on the screen at a precise location, which, as stated earlier on, is a property unique to particles.

Despite this piece of contradicting evidence, light was widely regarded as a wave at the time. James Clerk Maxwell proposed that electricity and magnetism were two parts of the same force, later to be known as electromagnetism, one of the three fundamental forces. In 1862, he calculated the velocity of a carrier of electromagnetic force (or electromagnetic waves) to move at roughly the speed of light. Unwilling to disregard this as a mere coincidence, he postulated that light was a part of the electromagnetic spectrum, carried by *waves*.

With the evidence mostly pointing towards light being a wave, the question must be raised – what is the medium that carries light? All waves require a medium for it to travel in, as they have no mass of their own. Sound uses matter as its medium, while waves in the ocean use water. If light really were a wave, what would its medium be? The answer was not very forthcoming. Light travels in the vacuum of space, where there is nothing that could be used as a medium. Maxwell himself proposed a “Luminiferous Aether” as a medium, which was undetectable. The supposed undetectable nature of said medium was thoroughly tested, most prominently by the Michaelson-Morley experiment, but there was absolutely no evidence that it existed. It was like a ghost pervading the scientific community for a very long time, almost undeniably there, but was indeed undetectable.

After a series of requirements were placed upon the properties of aether after theoretical calculations, each more incredulous than the last, the final blow was dealt when Einstein’s special theory of relativity provided a much simpler reason, made far fewer assumptions and placed much less stringent requirements on the nature of the universe to explain the light’s nature (a classic example of Occam’s Razor - the theory that the explanation with the fewest assumptions is likely correct). The explanation was simple - light was a particle that exhibited wave-like attributes, and since particles do not need a medium, neither does light. This of course has evidence backing it up - most notably the photoelectric effect.

The photoelectric effect is the emission of photoelectrons from a material whenever an EM “wave”, such as light interacts with said material. These electrons are called “photoelectrons” (electrons released from the interaction of “photons” with metals, hence “*photoelectron*”). If light were a wave, this could be explained as energy from the wave giving the electron its pseudo-momentum (the energy contained in the wave), which would be transferred to the electrons as kinetic energy allowing it to overcome the electromagnetic forces from the nucleus, allowing it to escape from the atom. With this explanation, low intensity light should cause the photoelectric effect to happen more slowly (as energy is transferred slower), and high intensity light should cause it to occur more quickly. However, when observed, this was not the case.

Experimentally, scientists found that the frequency of light played a much larger role than previously thought. Lower frequency waves contain less energy, so the photoelectric effect should have occurred at a slower rate. However, the experiments showed that low frequency waves (any waves below a certain frequency, coined the frequency threshold) did not cause any electrons to be released at all, regardless of the intensity of the light, or the period of time. This would not be explainable if light was a continuous wave as the energy would eventually build up.

Einstein proposed that light was not a continuous wavelength, and that instead it was carried by individual packets of energy, or in other terms, particles with wave-like characteristics - photons. This is a definition which is still accepted to this day. Light sounds simple enough, but what about other “particles”, such as electrons? How can something with mass be both a wave and a particle? If it *is* a wave, what is its frequency and wavelength?

Copenhagen suggested that the wavelength of a particle is like a probability graph of all the places the particle might be the next time it is observed. This relies on Heisenberg’s uncertainty principle - that to know the position of an object, we must interact with it in some way, hence changing its speed and direction. As a result, the more we know about its location, the less accurate our knowledge of its speed and direction (momentum) becomes, as we need more interactions to find its momentum. Conversely, more energetic interactions would be required to have a more precise measure of its position, therefore affecting the particle’s momentum more strongly.

Momentum is also determined by mass, and so the more massive an object, the less it is affected by Heisenberg’s uncertainty principle, and therefore the more certain we can be of its future position - the range of probabilities of position is smaller the larger the object in question is. However, *everything* is affected by interactions, regardless of size, and thus everything has a wavelength.

This means that larger the momentum of an object, the smaller its wavelength is. Therefore, massless particles, like photons, have the greatest wavelength, while massive objects, like planets, have correspondingly short wavelengths. This is represented in the de Broglie equation:

$$\lambda = \frac{h}{mv} \quad \text{where } \lambda \text{ represents wavelength, } h \text{ represents Planck's constant, } m \text{ is mass and } v \text{ is velocity (} mv \text{ being momentum).}$$

To sum up - particle-wave duality simply refers to how light, as well as other substances, behave as a particle or as a wave depending on the situation. The larger the momentum of an object, the more it behaves like a particle, as you can more accurately predict its trajectory. Light and other elementary particles are a special case due to their low momentum, meaning the wave aspect of the particle-wave duality can be observed more readily, as well as having a noticeable effect on its behaviour. If anybody wishes to read any further into this topic, the links in the references section should be helpful.

Edited by Neos Tang (Y12)

Terraforming Planets and Colonising Exoplanets: The Future of Human Life?

By James Meehan (Y12)

As our human race develops and evolves into a more intelligent and inquisitive species, the idea of space exploration and colonisation of exoplanets is paramount due to our curiosity. The universe is considerably larger than the human mind could possibly comprehend, but nevertheless, the thought of living elsewhere or being able to interact with other intelligent species of life is extremely compelling. The idea of terraforming planets has grown as the Earth's population has increased exponentially in the last century, and as a result, the depletion of Earth's resources. Although the consequences of global warming and overpopulation provide pressure to expand mankind's colony, the very idea of travelling through space and living on another planet is enough to captivate NASA and other huge space companies to invest in ways of making this dream achievable.

Terraforming a planet involves permanently altering the environment found there. The word "terraforming" is derived from the Latin "terra", meaning "earth" or "ground", and "forming" entails changing of the terrain of a planet or region. The result of terraforming a planet is so that it can resemble the Earth more, and is capable of supporting human activity. Humans have been driven by their fascination of space to explore deeper into the universe, in the hope of finding a planet that could sustain life.

Within our solar system, there are endless possibilities to terraform planets. Mars is the main focus of development because it is abundant in the four main elements; Carbon, Hydrogen, Oxygen and Nitrogen. Mars has also been found to have water supplies and also has a weak atmosphere. This makes the potential for human life on Mars relatively high, however large scale projects to secure the inhabitability of the planet are a necessity. There are also many problems with Mars, such as the magnetosphere not being aligned, meaning that no magnetic field is present. In order to overcome this, a global warming event would have to be self-inflicted by humans on Mars in order to artificially create and sustain an atmosphere to accommodate human life.

Another planet that is being investigated is Venus, but the surface temperatures of the planet exceed the possible limit for humans to survive there, which is why people often allude to "hell" when referring to Venus. As a result, researchers look to the skies. 30 miles into Venus' atmosphere, Earth-like conditions can be found. This has pushed organisations such as NASA to hypothesise a life suspended in the atmosphere in airships, which could prove to be viable. In addition to this, although very unlikely, astronomers also theorise that places large-scale shields could help block out the Sun from Venus and reduce the temperatures found there, which can reach 462°C. A benefit of this colonisation over Mars is that it takes 4 months less time to travel to Venus than it does to Mars, which can take up to 300 days to travel to, depending on the orbit of earth and the other planets in the solar system around the Sun.

It is not only planets that are the target for terraforming, it is their moons that are also monitored closely to adjudicate whether or not they contain the necessary resources for human activity. Europa, one of Jupiter's moons, has been known to have frequent water eruptions similar to fissures that extend up to the upper bounds of its atmosphere, making them visible from space. Titan, (right) one of Saturn's moons, has been partly proven to have underwater oceans with elements similar to those found on Earth, providing some evidence that humans can develop colonies there. There are also many other benefits of locating there; Titan has an atmosphere that is already established and therefore an artificial atmosphere does not need to be generated, as well as containing an abundant supply of Nitrogen.

A key negative of locating on Titan or Europa (right) is due to distance from the Earth, taking 5.9 and 11.86 Earth years respectively. The very fact that it takes such a long time to travel to the moons can deter astronomers from developing their hypotheses about colonising these bodies, only dramatic improvements in space technology in the future will be able to cut the travel time and make these plans more viable. Our moon is therefore a more appropriate alternative, considering its' close proximity to the Earth, it falls into the "Goldilocks Zone". This term is used to describe celestial bodies that are within an inhabitable zone because their temperatures, water availability and atmosphere are all similar enough to that of the Earth's and therefore are sustainable to colonise.

Although planets and moons within this zone can be classed as "perfect", there are many flaws. Earth's moon has huge variations in temperature from day and night, -170 degrees Celsius at night and 120 degrees Celsius during the day, which can make it uninhabitable. However, these temperature difficulties can be minimised by colonising areas on the poles of the moon, where variations in temperature do not vary – which is not necessarily terraforming. In order to create an atmosphere on the moon (as it lacks one), "iceteroids" comprised of water frozen as ice would have to be directed to hit the moon and deposit water in order to culture algae and stimulate evolution of species, in order to create biodiversity within new ecosystems to supplement human activity on the moon. Unfortunately, the technology for this is not yet developed at an intricate enough level to achieve the manipulation of the comets required to supple the moon with water, but is being developed in the hope that these missions can be executed to terraform planets as soon as possible.

Rather than creating a new and permanent environment on another celestial body, planets have been located and studied to see if they could be inhabited without the process of terraforming. Planets that are extremely similar to the Earth in: distance from orbiting star, surface temperature, size, density, atmospheric pressure and water availability are all classified as "Earth Analogues" (or colloquially as "Earth's twin". Although not all exoplanets can be classified as such, there have been over 4,000 confirmed exoplanets discovered by astronomers globally. In addition to this, theories state that "if planets fall into a "Goldilocks Zone" as frequently as the Earth fits into the zone in our solar system around their stars, then there are over 1 trillion exoplanets in the Milky Way galaxy alone." The same theory also states that "orbiting each star in a solar system, there will always be one exoplanet." An exoplanet is classified also under the Earth Similarity Index (ESI) to adjudicate how Earth- like that planet is, with a maximum score of

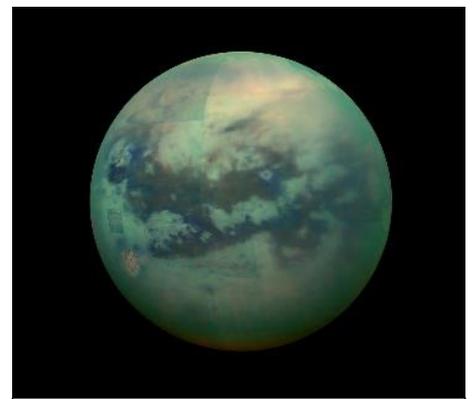


Figure 1: Satellite Image of Titan

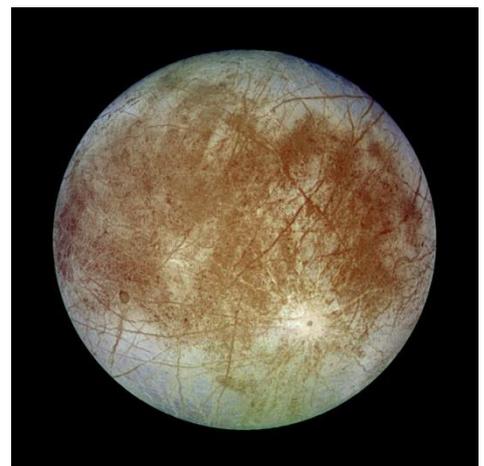


Figure 2: Satellite Image of Europa

1.0 (the Earth). A score of above 0.8 is considered extremely Earth- like, and within our solar system, no planet scores above that (Mars scores at roughly 0.7). In other solar systems however, a range of exoplanets have been found to be extremely similar to Earth.

Amongst the thousands of candidates to become the “next Earth”, Teegarden’s B became the most likely candidate to be the most inhabitable planet in July 2019. Teegarden’s B is located 12 light years away from earth and orbits an M-type star with surface temperatures of 2,900 C, but is not visible from Earth. The planet orbits the Teegarden’s star once every 4.9 earth days. Surface temperatures of Teegarden’s b are between 0- 60 °C, but estimates predict at around 28 °C, meaning that the planet could be habitable as water could be free flowing. Teegarden’s b scores the highest on the ESI, at 0.95 making it the most habitable planet (discovered by NASA). Teegarden’s star is the 24th nearest star to the Earth and the 4th nearest star with potentially colonisable exoplanets. As time continues, space probes will be used to monitor planets’ characteristics in greater depth to provide a wider understanding of the possibilities of creating a livelihood elsewhere than on Earth.

To take into account all of the necessary technology that is essential in terraforming planets as well as travelling to and colonising exoplanets, it is important to understand that such developments are most likely to not develop within our lifetimes, as the appropriate intricacy and efficiency of machinery and vehicular travel has certainly not been discovered. However it is inevitable though that the pure curiosity and will of humans will push us closer to colonising more of the universe than just our Milky Way Galaxy.

Edited by David Kuc

Reaching for the Stars: Space Transportation

by Kiran Lee (Y12) and Ansh Sharma (Y12)

The Future of Space Travel, and how anyone could become an astronaut in the decades to come:

With it having been over 50 years since the first human reached space, some might wonder as to why we haven't gone back regularly and maybe even why it has taken so long for space travel to become commercially viable. After all, within a few decades of constructing the first aeroplane we had not only national air forces which were thousands of aircraft strong, but also the beginnings of commercial aviation. So why is space travel still inaccessible to the vast majority of us, and how could we look to change that in the near future? In this article we will look at why space travel is so difficult, and two ways we could potentially make the journey into the depths of space much more easily in the next few decades.

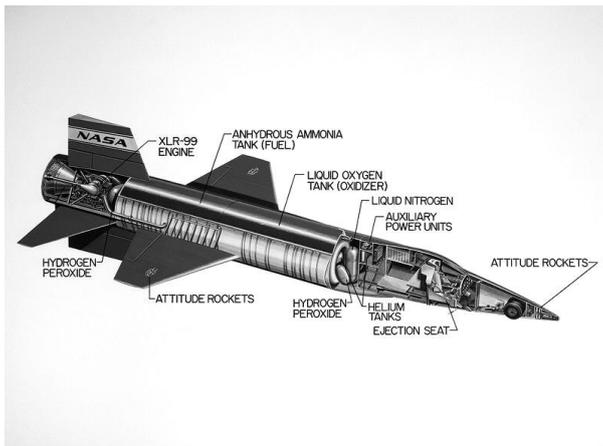


Fig. 1 - NASA's X-15 aircraft, despite only being around 15m long, managed to reach space on multiple occasions.

The first question we must ask when understanding how to make space travel easier is to think about why it is so difficult to achieve in the first place. This is actually a somewhat misleading line of questioning - getting to space is easy, relatively speaking. In fact, space is so close to the ground that you can get a person there with a rocket the size of a telephone pole. Outer space is around 100km from sea level, although definitions about the exact border between the atmosphere and space vary from around 80-120km. At that distance, somebody living in Milton Keynes is about as far from space as they are from the sea.

The problem with space travel is not simply reaching space, but actually staying there. For a spacecraft to remain in space after it gets high enough, it has two options: continue to burn fuel or accelerate so we can orbit the planet. The first option is to continue to burn fuel to resist the pull of gravity. Contrary to popular belief, this is actually very strong; the gravity at the edge of space is over 90% as strong as the surface gravity. The false perception may stem from the apparent weightlessness of astronauts at that altitude because the spacecraft is accelerating at the same rate as them. Resisting this pull would cause a spacecraft to run out of fuel *very* quickly. The second option is to accelerate to such a great horizontal velocity (parallel to the Earth's surface below) that it can orbit the planet. This results in spacecraft moving at astounding speeds (8km per *second* for the ISS, for example), and this is what most of the fuel in a rocket is used for as well: to accelerate to that velocity. Fuel is not used only for this, however - spacecraft need to reach thousands of kilometres into the sky or even higher, which means that they need to spend even more fuel rising to their target altitude.

Now, of course if we could keep ourselves in space without using fuel, then we wouldn't have to accelerate into orbit, which would save tremendous costs in fuel, increase cargo capacity, make space travel safer and therefore make it a much more realistic prospect for businesses and civilians. In the remainder of this article we will look at two potential ways of achieving this goal: the space elevator and the skyhook. Let's investigate the space elevator first, since it's the possibility that would require the most development from where we currently are.

Space Elevator:

A space elevator is more or less what it sounds like: a huge lift that could take cargo from the ground into space, thousands of kilometres high, if necessary. As we have already seen, a spacecraft needs fuel both to get it into space and to get it moving extremely quickly once it's there. A space elevator has a huge advantage over this more primitive method which we currently use, as it does not require the second stage, saving huge amounts in transportation costs. To understand why, think of the centripetal force (a force acting at 90 degrees to the direction of motion, causing the object to take a circular path of movement) that we can all investigate in our daily lives. If you spin a bucket on a string very fast, the string will stay taut - this tension is the centripetal force.

Now, imagine an ant walking along the string. While it is near your hand, it is moving very slowly, but by the time it reaches the bucket, the ant's linear velocity will have increased, even though the angular velocity stays the same. This is because the circumference of the circle formed at the end of the string is greater than the circle formed at the nearer end. The same principle applies to space elevators: by simply utilising centripetal force to provide the horizontal velocity, all we need to do is transport the cargo upwards using an elevator, and it will be moving horizontally by the time it reaches the top. Such a method would be slower, but it would be well worth the loss of speed in exchange for the safety, reduced cost and increased cargo capacity compared to a conventional rocket - possibly even cheap enough for tourists to go into space on a regular basis. Why have we not built a space elevator already then, if it has so many advantages compared to traditional space travel?

The main problem we currently face when trying to design a space elevator is what to make the cable out of. It needs to be light, inexpensive and stronger than any material we can currently produce on such a scale (although it is possible that carbon nanotubes will be strong enough, although not necessarily cheap enough). As such, making this elevator is not possible with current technologies, although it is very likely that it will become possible to build in the next few decades. A cheap elevator could make up for its upfront building cost after just a few launches (not to mention the capacity and safety improvements), so it is definitely something that humanity will continue to look into.

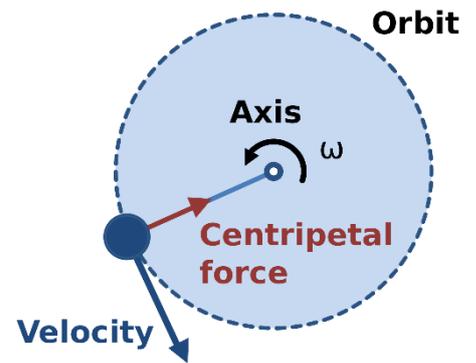


Fig. 2 - Centripetal force in action; this principle can help explain how the sky elevator works.

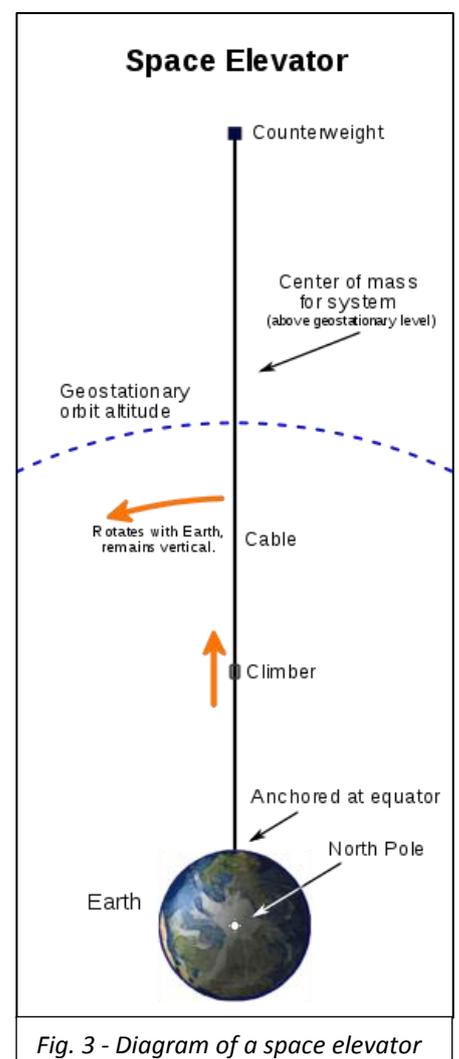
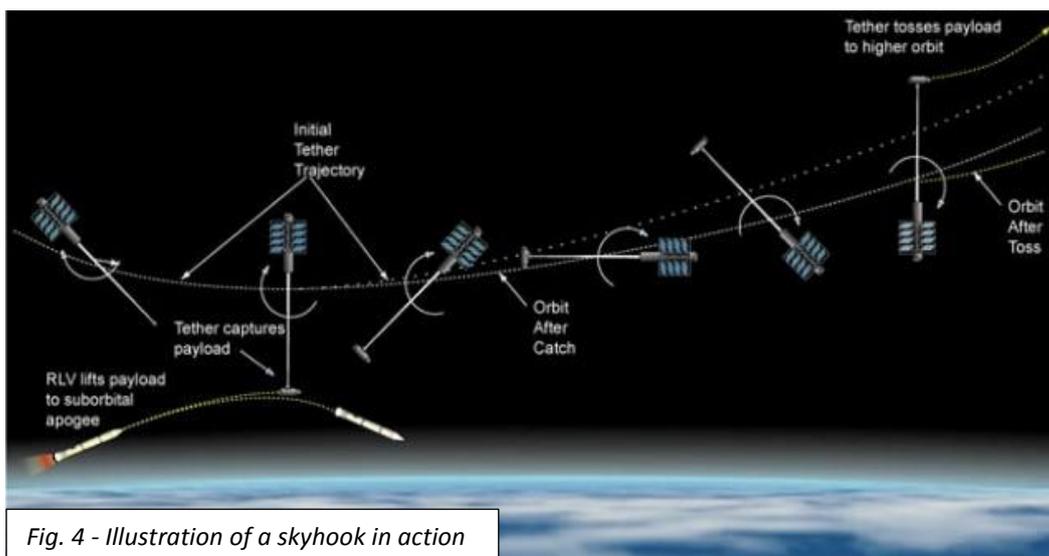


Fig. 3 - Diagram of a space elevator

Skyhook:

Now, we shall move on to the skyhook, discussing what it can do, and how it differs from the space elevator.

A skyhook is even simpler than a space elevator: at its core, it simply involves catching something at a lower altitude, and throwing it again at a much higher altitude. In order to achieve this, we could put the skyhook at a height of around 80km at its lowest point (high enough to avoid most atmospheric friction) and thousands of kilometres high at its highest point. Spacecraft would only need to reach the 80km point to then be captured by the tether and flung into a much higher orbit. In order to conserve the momentum of the skyhook, it could catch spacecraft coming in from outer space as well as sending spacecraft out - as long as the numbers going in each direction are roughly equal, the skyhook could maintain its altitude with relatively little input needed from any on-board thrusters.



This idea also improves on fuel costs, cargo capacity and safety compared to traditional spaceflight (and could allow for affordable holidays to outer space as well, eventually), although the concept cannot completely remove the fuel required to get the ships up to 80km to begin with. Unlike the elevator, it is also possible with current technology. Since in this case the tether doesn't have to be as strong, currently existing materials such as Kevlar and Zylon could be used to make it. The design has also already been successfully tested on a small scale, and in theory there is nothing stopping us from building a larger hook today. The only things we require now are the money, enough materials and the willing investment of governments and companies, and space travel via skyhook could become a way for ordinary (well, relatively ordinary) people to experience space in the very near future. Of course, only time will tell if skyhooks are ever used on a large scale, but they appear to be incredibly promising.

To recap, both the space elevator and the skyhook avoid many problems faced by traditional methods of getting into space, and thus allow us to overcome the barriers that have prevented space travel from being economically viable for anyone less rich than a billionaire, large corporation or government. Out of the two, the skyhook has a simpler design and could be built with current technologies, and thus we believe that it is the more likely method to be used for revolutionising the use of space travel in the decades to come.

Edited by Neos Tang

Computational Fluid Dynamics

by Leo Kavanagh (Y10)

The motion of water has mystified humans for many centuries, with its complex and elegant eddies and vortices; its turbulence and its unpredictability. From a scientific perspective, water often exhibits sensitive, chaotic behaviour, making it difficult to model and predict, which has been the goal of physicists and engineers ever since the behaviour of fluids was first analysed by George Stokes. Stokes decided to create a law of motion for fluids, an analogue to Newton's famous $F = ma$. To do this, he decided to rewrite $F = ma$, in the context of fluids, using the language of calculus:

$$\rho \frac{\partial V}{\partial t} = -\nabla p + \rho F + \mu \nabla^2 V$$

This was the result. Although the equation may look intimidating, it's really as simple as $F = ma$.

The first symbol, ρ (the Greek symbol *rho*) represents density, which you can think of as mass^[1]. The next part of the left-hand side, $\partial V / \partial t$, just means the rate of change of the velocity of the flow (V) as time changes: the acceleration of the fluid. Thus on the left side of the equation we have the ma of our $F = ma$.

On the right-hand side we find the forces that act on the fluid. $-\nabla p$ is the pressure gradient (i.e. how the pressure changes across the fluid). Fluids will tend to move into lower pressure areas (for example, air rushing into the vacuum of outer space, or air flowing towards low pressure areas on Earth to create wind). F is any external force being exerted on the fluid, such as gravity, and $\nabla^2 V$ is the gradient of the velocity, representing diffusion taking place in the fluid.

Using the equation:

Now, we need to find a way to apply these fluid equations to real world problems, such as the flow of water around a solid object (say for example a flat plate). Our goal is to be able to give this equation and some initial conditions to a computer and let it solve the equations, producing a visualisation of the flow and pressure in our scenario.

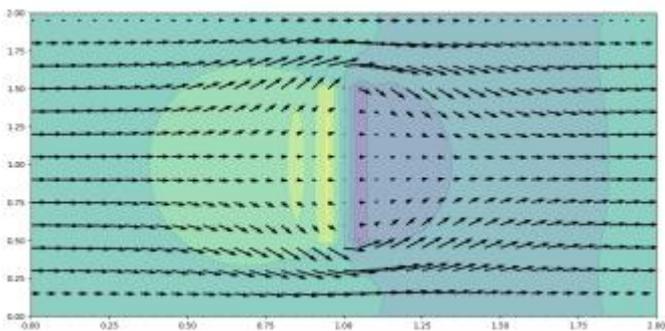
Firstly, we need to create the space that the fluid simulation is taking place in. We create a huge grid of points, each of which has a flow vector (a vector showing where the fluid is flowing at that point) and a pressure. We can then work out the quantities in our equation, such as the gradient of pressure, by comparing the different points.

For example, to find the pressure gradient at a point, we do the same as we would do if we were trying to find the gradient of a hill from a point. We take the pressure (imagine this as the height of the hill) of the point in front of it and then subtract the pressure of the original point from this value, and then divide this by the distance between the two points (you can also think of this as using the rise over run equation to find the gradient of a line graph^[2]).

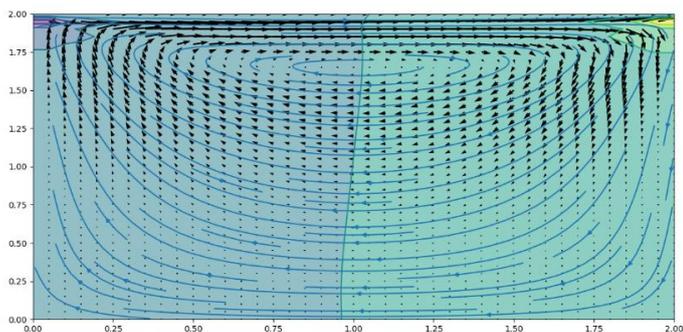
We repeat a similar process for all the other components of our equation on the right-hand side. This is because all the right hand side is equal to the density times the acceleration of the fluid, which means that we can then divide the right hand side by the density of the fluid to find the acceleration of the fluid, which can then be used to work out the new velocity (which is represented by our flow vectors) of the fluid.

We then use this to update the pressure, as the pressure at a point depends on how much fluid is flowing into it, which we plug into the equations again to update the velocity, which we use to update the pressures, and so on until it converges (i.e. starts to slow down in its change and approach a fixed state). We can then visualise the flow vectors and pressure to create a representation of the flow and pressure in our scenario.

I tried this myself using the Python programming language, modelling water flowing past a fixed, flat plate.



These were my results. The arrows represent the flow, or velocity of the fluid, around the flat plate located at $x = 1.0$. The colour represents the pressure, with brighter, yellower colours representing higher pressures. We can clearly see a realistic pattern of high pressure building up in front of the plate (including shockwaves: the two lines of very high pressure in front of the plate), as well as lower pressure behind it.



We can see that the fluid flows quicker around the edges of the pipe (where it is restricted into a smaller area), and then curls around into the lower pressure area behind the plate—as we would expect to see in reality. Having established that the model worked, I tried some other scenarios, such as convection, shown on the left.

Again, we can see a pattern typical of what we would expect in the scenario, based off our prior knowledge.

In conclusion, Computational Fluid Dynamics (CFD) is a powerful and effective tool that is possible to understand relatively easily. However, it requires a lot of computational power, and there are still mysteries as to the nature of the equations that we used. One of the most important open questions in mathematics and physics is finding out if the Navier Stokes equations will always yield a solution. The Clay institute offers a prize of one million dollars to anyone who can solve the problem ^[3]! There are other issues as well, such as the nature of turbulence—a tiny inaccuracy in the simulation might make the final results totally incorrect, with potentially disastrous consequences. Indeed, turbulence is an ongoing area of research in modern physics and there is still much more to explore.

Finally, I would like to close with a brief poem written by early fluid dynamics researcher Lewis Fry Richardson:

*Big whorls have little whorls
Which feed on their velocity,
And little whorls have lesser whorls
And so on to viscosity.*

Edited by Michael Lowe

Footnotes

1. Density is defined as mass per unit volume, so it is more applicable in this context than mass—the equations still work no matter what volume of fluid there is.
2. $m = \frac{\Delta y}{\Delta x}$
3. This was one of the seven Millennium Prize Problems set in 2000 which each have a reward of \$1million. Currently only one of these problems has been solved.



The Colonisation of Mars

By Neos Tang and Daniel Wan (Y12)

With the Earth becoming more and more crowded by the minute, the 'Red Planet' has been the subject of growing investigation as a possible candidate for colonisation. This may perhaps be fuelled by the idea that we may, at some point, be able to terraform it to sustain life from Earth. For a number of reasons, Mars is the prime target of space colonisation in the near-future. In spite of the array of problems a human settlement would face, space agencies and private enterprises are working intensely to bring us closer to this aim.

However, this whole concept raises several questions. Why is Mars first on the list for colonisation? How do we survive and thrive on a planet that, as far as we know, is completely lifeless? And ultimately, even if we can do this, what's the point?

Why Mars?

Many Sci-Fi films, such as *The Martian*, show humans colonising Mars, and we mainly hear stories about having to colonise Mars to combat overpopulation. But, why is it always Mars? Why not somewhere else? Well, there are some other candidates, but unfortunately they have some rather more dangerous setbacks. Venus does seem to be a good planet to build on, with its relative proximity to the Earth. Its atmosphere is also comprised of the mixture of gases which we need to survive, and its surface gravity (comparable to that of our planet) ensures that our bodies aren't crushed under our own weight as soon as we get near. However, a rather striking drawback is the 460°C surface temperature, which is enough to melt zinc, and, naturally, our flesh and bones. Another possibility is to take a look at Europa, one of Jupiter's moons. Its smooth surface leads to the belief that vast storages of water exist beneath, perhaps even supporting life already. In actual fact, the position of Europa situates it within a deadly radiation belt from the Sun - not ideal for human habitation.

Mars, on the other hand, has quite a few similarities to Earth, without drawbacks quite as terrible. Mars' and Earth's days are comparable, with Mars' days (or "sols") being only 37 minutes longer than that of Earth, while years on Mars are about double the length of those on our home planet. Mars reconnaissance also confirms the existence of water, in the form of ice. Water is, of course, essential to our survival, and its presence on our planet was one of the main factors facilitating life on Earth.

Surviving on Mars:

Astronauts may initially survive on Mars through the construction of a "space-base". Due to the far greater levels of radiation present (due to cosmic rays from the sun, which are high energy charged particles buffeting the planet, combined with the lack of a magnetosphere, which will be explained later on), this base must be able to absorb most of the radiation in order to keep everybody safe. There are many different designs for these kinds of bases, but an interesting (and now viable) concept is that of robots creating the base before the humans arrive. These robots could be sent over to Mars, who then proceed to 3D print, or "fabricate" a shield for the base. After the machines collect the soil, alongside other required components from Mars' ground, they will then expose this mixture to microwaves, creating a brilliant material to use for 3D printing. The robots would now fabricate just the radiation-protection shield - the living and working quarters within will all be inflatable modules, connected in a ring-like, circular formation.

This ensures that if one of the modules somehow becomes compromised, the rest of the base can still be accessed. In order to ensure the comfort of the astronauts, spacious areas will be provided, which keeps people happier (maintaining morale) when having to stay indoors. The base will be connected to a huge array of solar panels outside, to provide for power needs. Within the base, the astronauts will of course require oxygen, which can be formed through the electrolysis of water from the Mars ice (separating the hydrogen - “H₂” from the oxygen - “O” in water - “H₂O” through the use of electricity).

All of the astronauts’ culinary requirements can be met through the use of a greenhouse garden - a hydroponics bay of sorts, akin to that of *Star Trek*. An insect farm will also be extremely useful in providing all necessary proteins. Equipment for exercising must also be provided for the astronauts, in order to prevent the degradation of muscle and bone due to the low surface gravity on Mars (less than half of the Earth’s). The inclusion of a workshop will allow for the repair of robots, and the creation of anything new which might be needed. A research laboratory might also be important, investigating potential life on Mars, and our own origins.

Presumably we will eventually wish to expand our life on Mars - living in a base for all eternity would not be utilising Mars to its full potential, after all. If being inside is safe, and being outside is not, then making outside just like inside would be perfect. This is the process of “terraforming” - transforming the planet so that it can support human life.

Unlike the Earth, Mars does not have a magnetic field, which protects the planet from solar winds - the flow of charged particles from the Sun which tear away at the atmosphere. Without one of these, any terraforming we carry out on Mars’ atmosphere will just be demolished. Luckily for us, the Earth’s field occurs naturally due to the velocity at which it spins, and the liquid nature of its core. Unfortunately, Mars’ core is too cold to be enter a liquid state. Instead, we will construct an artificial magnetic field, by positioning a huge electromagnet between Mars and the Sun, the field from which surrounds and protects Mars.

Now that all of the changes that we make to Mars will remain, we need to warm the planet up from its current, chilly temperatures of -60°C. Mars has vast expanses of CO₂ ice caps, which reflect sunlight (so the energy from the sun is not retained within the atmosphere). Melting these ice caps would stop the reflection of sunlight, while also releasing CO₂ (infamous for being a “greenhouse gas” on Earth) in gaseous form - our aim is to artificially take Mars through global warming, which could be seen as a little ironic, considering the situation back home. The ice caps can be melted by orbital mirrors, which are huge reflective surfaces as satellites outside of Mars’ atmosphere, concentrating light and energy from the sun onto the glaciers.

Next, our goal will be to fill the atmosphere with the gases that we need to live. After Mars has reached 0°C, water will be able to take a liquid form, and rain will fall. Algae will then assist in transforming the CO₂-dominated atmosphere into a more breathable one through the process of photosynthesis, where they convert carbon dioxide into oxygen (although this will likely take about a thousand years). Ammonia can be imported, which will decompose into nitrogen and hydrogen. Nitrogen is required to decrease the toxicity and flammability of Mars’ new atmosphere, caused by the added CO₂ and O₂ respectively.



Figure 1: Christian Gruner – Mars Base



Figure 2: NASA, Growing food on Mars

Finally, we can develop the biosphere - the surface, where living organisms like us can live. Nutrient gel added to the soil will enable plants to grow. It would be best to import *arctic* trees and insects first, due to the still relatively low temperature, and then eventually move on to herbivores, such as sheep, and then arctic foxes and other animals.

And there we have it, a habitable Mars! There is no timeline for when Mars will be colonised as of yet, but there have been many propositions as to when and how we will travel to Mars. Space travel is a massive and incredibly fascinating topic (which unfortunately will not be discussed here), which is being heavily invested in. Elon Musk and SpaceX are aiming to land the first humans on Mars as early as 2024, just four years from now.

Should we do this?

With a huge level of financial and public support, the struggle for the colonisation of Mars is likely to continue. Could we set up bases on Mars? Yes. Could we terraform Mars? Probably yes, especially over a long period of time. Should we? Well... maybe?

There is little scientific consensus on the matter, with a spectrum of opinions ranging from its necessity, to being completely opposed to space colonisation. Perhaps the most widely-circulated argument for Mars' colonisation is that it is necessary for survival as a species; becoming an interplanetary species would increase the probability of survival if a catastrophe were to befall one planet. Consequently, a self-sustaining Mars base is the ultimate aim of almost all Mars missions. However, self-sustainability will take a while to achieve: if Earth were to be destroyed, the colony would need to maintain all of its equipment to recycle basic resources and continue to produce technology, particularly vital assets such as medicines and robotics systems. Additionally, but more importantly, this has the harmful implication that going to Mars will save humanity and absolve it from the self-inflicted problems on Earth. We are facing a period of dramatic change in the Earth's environment, for example, average global temperatures has risen by 0.85°C since the late 19th Century CE and the rate of loss of Antarctica's Ice has tripled within the last decade, to name a few worrying statistics. But if we are truly convinced that we can transform Mars into another Earth, then we should surely be capable of preserving the already-existing Earth. As Sir Martin Rees puts it in his book, *On the Future: Prospects for Humanity*, "Coping with climate change may seem daunting, but it's a doddle compared to terraforming Mars," particularly when taking into account that some of the most inhospitable locations on this planet, such as Mount Everest, are more suited for life. Colonising Mars does not need to come at the cost of ignoring Earth's problems.

But there are plenty of other reasons to colonise Mars, including the potential for massive developments in the search for extra-terrestrial life, especially through the use of high-tech laboratories. The recent discovery of methane on Mars by the rover Curiosity, a possible indicator of one of the building blocks of life, certainly supports this idea, and colonisation could provide the necessary space infrastructure to deliver much more than 6.8 kilograms of scientific equipment accommodated on Curiosity. Furthermore, development in the colonisation of Mars, and in space exploration as a whole, may lead to benefits in our daily lives as technology from the venture is repurposed for other uses. For example, an image analysis algorithm used for blurry images from the Hubble Space was later applied to obtain more accurate images for diagnosing breast cancer.

Issues with Colonisation

Venturing into this “new frontier” is also reminiscent of the ideas of colonising our own planet in the Age of Exploration, and has all of the complex problems surrounding it multiplied by two. The concept of the first humans born on Mars is scary, given that the effects of low gravity and exposure to radiation upon the reproductive system and pregnancy have not been studied.

Meanwhile, the social, legal and political dynamics of a colony does technically have the very modest scaffolding of the antiquated 1967 Outer Space Treaty. On the other hand, it was signed in the midst of the Cold War as a framework for dealing with space conflict, reflecting the fears of two superpowers. If this remains as the basis for space colonisation, then a colony cannot belong to a country, something that may encourage the development of a new form of space governance as current ideas of land rights and sovereignty are thrown out the window. The treaty is also ambiguous about private enterprises on Mars, which is unhelpful given how pivotal a role companies are likely to play in this process, but the solution remains unclear, and the ambiguity can equally be just as helpful. One proposition is “Cooperative Sovereignty”, where colonies have designated economic zones where development is incentivised, based upon international treaties regarding the seas and Antarctica. Others believe that capitalism will dominate as private companies expand, or that the people of Mars will emerge separately from humanity and establish themselves as an independent nation.

It’s conceivable to think of Mars as the inevitable next step in the growth of human civilisation, tapping into our rich history of exploration and expansion, and yet a number of important questions remain unanswered. Nevertheless, this is also not an insurmountable task. Provided that we don’t get so carried away that Earth is forgotten, humans have a good chance of making it to the Red Planet and turning it green.

Edited by Devanandh Murugesan

Red and Brown Dwarfs: The Forgotten Members of a Star's Life Cycle

by William Lu (Y11)

During our physics GCSE course, we learn about the fascinating life cycle of a star, yet two notable members have been omitted from our specification. Thus, we shall dive deep into the bizarre and obscure nature of these astronomical objects and explore what makes them so unique.

Brown dwarfs: failed star or massive planet?

Brown dwarfs have a rather misleading title. Firstly, they aren't brown, but rather a light magenta colour, and, secondly, they aren't stars despite being named a dwarf (which is common amongst small stars such as white and red dwarfs). In fact, there is lots of debate amongst the scientific community on what is or isn't a brown dwarf - as if it was not complicated enough already!

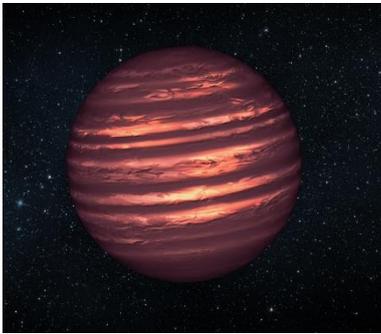


Fig. 1 - Concept art of a brown dwarf

Although the term 'brown dwarf' is commonly used to describe a protostar that failed to become hot enough to fuse hydrogen into helium, the International Astronomical Union (IAU) defines a brown dwarf to be balls of gas that can fuse deuterium (a hydrogen atom containing a proton and a neutron) into helium-3 (a helium atom containing two protons and a neutron). Now, the size threshold for fusing deuterium is around 13 times larger than the size of Jupiter, which means that very large gas giants can also fuse deuterium. This is at the heart of the brown dwarf controversy. However, there are some distinguishing features proposed by scientists to solve this conundrum (although not all scientists fully agree on these still).

Brown dwarfs do not share the same anatomy that planets do. They lack a solid metal core, for example. Differences such as this show us that the two are not alike. There are many other differences too, such as that brown dwarfs also have low metal content, and planets tend to have high concentrations of metal. One way that we can distinguish brown dwarfs from planets during detection is through the level of infrared radiation that they emit. "Warm" brown dwarfs tend to emit more infrared than a planet would, allowing us to tell them apart.

Red Dwarfs: how do they have such a long lifespan?

Red dwarfs are slightly larger than brown dwarfs, which means that they are categorically stars, and actually make up 70% of all stars. Despite being so numerous, however, you have never seen them on a starry night. This is due to two reasons: they are a lot cooler than other stars, and so only emit a faint red light (hence their name) and they are also only around 50-70% the size of our sun, making them even harder to detect. Therefore, we can only observe very nearby red dwarfs. However, this is actually not a huge issue as two thirds of our nearby stars are red dwarfs. The nearest star outside our solar system - Proxima Centauri - is also a red dwarf star.

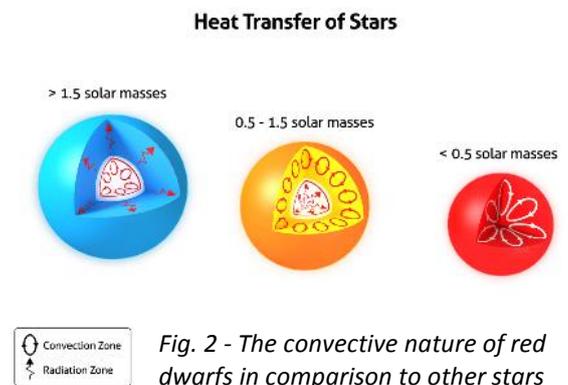


Fig. 2 - The convective nature of red dwarfs in comparison to other stars

Red dwarfs also have incredibly long lifespans - what makes these red dwarfs last over 10 trillion years before burning up? Well, unlike large stars such as our sun, red dwarfs do not accumulate their fused helium in their cores. Instead, they are convective, meaning that the hydrogen and helium within is constantly mixed together. This extends the star's lifetime dramatically as the convective nature prevents all of the hydrogen from being used up, preventing the formation of red giant, which would cut the lifespan of a star dramatically. Furthermore, their small mass means that they only need to exert a small pressure force to counteract the inward force of gravity, and so fuse atoms as a fuel at a slower rate than other stars to supply this force.

Because of this, the lifespan of a red dwarf ranges from 1 to 10 trillion years. The universe is currently approximately 14 billion years old, which means that so far, no red dwarfs have burnt up. So, as the universe begins to wane, these dim lanterns would be the only lights left in the universe. Once they have all finally been extinguished, the universe will be plunged into darkness. However, that is still eons away. But, what *does* happen when a red dwarf star burns up? It has been theorised that it might become a *blue* dwarf before returning to the well-known realms of the life cycle of a star, at which point it meets the same fate as stars similar in size to our star - the Sun. Its core will collapse into a white dwarf star (a very dense and small star made up mostly of helium-4 gas), which will then over time cool down. When the star has exhausted nearly all of its fuel, it has no alternative but to radiate off its remaining reserves, before finally meeting its fate at the end of the road as a black dwarf star.



Fig. 3 - Concept art of a White Dwarf Star

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