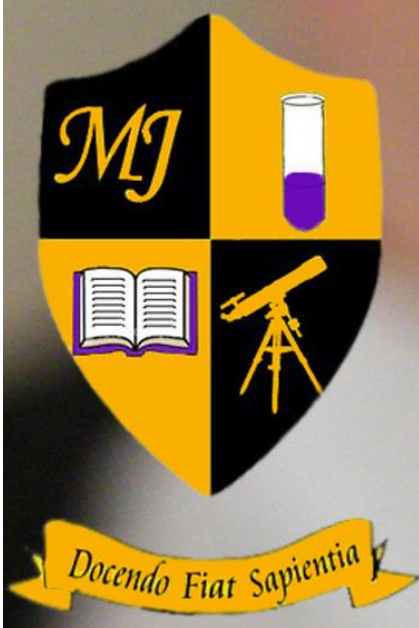


# *Quantum Ultimum*



*Moncrieff-Jones Society*  
2009 - 2010

# Foreword

Dr. Bruce Griffin (BSc, PhD, RPHNutr) is a professor at the University of Surrey, specialising in lipid metabolism, nutritional biochemistry and cardiovascular disease. He has also done extensive research into these fields with grants from numerous organisations, including the Medical Research Council, the British Heart Foundation and the Food Standards Agency.

“Each year I have been impressed by Caterham School’s science students on their visits to Surrey University. They consistently show a genuine interest in the topics which I present to them, asking intelligent and sometimes demanding questions. This year is the first time I have been invited to contribute to the Moncrieff-Jones society’s annual magazine and I am happy to continue my support for the school and its science community in this way.

The subject matter in the presentations this year covers an impressive range, from the sub-atomic world of *The Theory of Supersymmetry* to some of the most topical areas of research, including *Alzheimer’s Disease* and *The Telomere Theory* of ageing. It also yields an in-depth knowledge and interest which I look forward to sharing when I have the opportunity to read this publication.

I offer my congratulations to the members of the society and the students who run it for ensuring that this fine tradition can continue and progress so successfully.”

Dr. Bruce Griffin

Professor, University of Surrey



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# President's Introduction

Alex Hinkson has a place to read Medicine at St. Catherine's College, Oxford.

"Welcome to the 2010 edition of *Quantum Ultimatum*, the Moncrieff-Jones Society publication. This magazine contains a summary of each of this year's talks, which will give you an insight into the fascinating topics presented by our speakers.

This year has been particularly successful, with an increase of the society's profile within the school. The numbers of people attending talks has also increased and more members from lower years within the school are now attending regularly, which is no surprise, considering the quality of the presentations. I would like to congratulate all of this year's speakers for maintaining the standards that have come to be expected within the society and for sharing with us their passion and interest in their topics.

Finally, I would like to thank the Heads of Science: Mr Dannatt, Mr Keyworth and in particular, Mr Quinton, for the guidance they have given me in managing the society this year, as well as all those who attended the talks, asked questions and generally helped to make the society such a success. Good luck to those taking over next year, and I only hope you'll enjoy it as much as I did!"



## Vice President's Introduction

Alex Clark has a place to read Physical Natural Sciences at Robinson College, Cambridge.

"The Moncrieff-Jones Society has gone from strength to strength this year, not only in the quality of talks, but in the variety of topics also. From Speech to Supersymmetry, there has literally been a talk to interest everyone, from all branches of science. My thanks and congratulations go to all those giving talks, not only for their hard work and dedication in promoting science within the school, but also for all the effort that goes into preparing the talks too, including publicity and writing the articles for this magazine. You have all set the standard high for next year's speakers, and you have ably lived up to the society's motto, *Docendo Fiat Sapientia*.

As Alex has already mentioned, attendance of the talks has increased significantly in comparison to previous years, and it is very encouraging to see so many people present, many of whom are in the Fifth Form. Audience participation is crucial for these lectures, and it is encouraging to see so many asking thoughtful and insightful questions to challenge those who are speaking.

May I wish the best of luck to the incoming President and Vice-President for their year running the society; I hope you have a great year."



# Crohn's Disease – Eleanor Croft

Crohn's Disease itself is a fairly unknown, but incredibly interesting disease. The first question people ask is "What is it? How do people get it?" And these are two of the questions we're not really sure of yet. I aim to outline in this article where our current understanding is on what the disease is, and why we get it and then what we're doing to try to relieve symptoms, with the aim of curing it.

Crohn's Disease is an autoimmune disease-where the body's own immune system has a hyper-response in the gut. These diseases only occur in westernised countries, though the reason for this is unknown. It can affect anywhere along the gut, and causes inflammation, usually ulcers, but can also cause abscesses, fistulae, and joint inflammation. These cause symptoms such as diarrhoea, bleeding, anaemia, stunted growth, weight loss, and trouble moving.

Colonoscopy of a healthy small bowel (left) and that of a Crohn's sufferer (right).



These are not however continual symptoms and one of the mysteries of Crohn's Disease is that it is so different between people. Some sufferers will only have one or two episodes of these symptoms (known as flare-ups) during their lifetimes, whereas others will experience nearly continuous symptoms making it nearly impossible for them to live a normal life.

Not only do we not really understand why sufferers have such different symptoms but also we don't know why people get this disease. It occurs usually between the ages of 10 and 40, though children as young as five have been diagnosed, and we know that there is a genetic element, as those with a family member who has Crohn's are at increased risk. So the first step in looking for a cause was to isolate any genetic differences between sufferers and those who were not. As it is an autoimmune disorder, it was incredibly difficult to narrow down where to look, as about 29,000 of our 30,000 genes are involved in the immune response, so instead it was necessary to analyse any Single Nucleotide Polymorphisms across the genome. This was funded by The Wellcome Trust Case Control Consortium, and it was a huge project involving thousands of people across the world, and was successful in the case of Crohn's disease of discovering over 30 gene differences that can increase our susceptibility of getting Crohn's. It is very important to highlight that it is not a purely genetic disease. Studies with identical twins, have shown that although they are significantly more likely to both have IBD than non-identical twins this was still a relatively low percentage. It is thought that gene-environment interactions

lead to the development of Crohn's Disease, and that mucosal lining of the gut, bacteria (particularly *mycobacterium avium paratuberculosis*), smoking and possibly food types can influence those with genetic susceptibility.

Other than stopping smoking, at the moment doctors use three main types of drugs to try to reduce symptoms, and try to elongate the remission period, although 70% of those with Crohn's Disease require surgery at some point. A large proportion of attacks are treated with corticosteroids, either intravenously during severe attacks, suppositories if the inflammation is of the lower bowel, or orally if inflammation is mild across a large proportion of the bowel. However this is only to reduce the primary inflammation of the gut, and does not treat any secondary inflammation, nor does it help maintain remission. Also, over the long term, corticosteroids have very unpleasant side effects including: abnormal weight gain, deposits of fat around the chest, face and upper back, and also the adrenal glands no longer produce as much cortisol, so patients must be gradually weaned of these steroids. Another family of drugs used, particularly in more serious cases is immunosuppressants, which effectively stop the body's natural defences in the immune system by suppressing the activity of the lymph glands, so the patient is left with a very weak immune system over a long time. Thirdly they also are looking towards a new family of drugs monoclonal antibodies. Monoclonal antibodies are produced by injecting a mouse with the antigen of the specific cell you want to affect, and then taking the mouse's spleen cells and fusing them with immortal myeloma cells and finding. The cells which produce effective antibodies are then cloned and the antibodies produced are harvested. Those used in Crohn's are specific to human necrosis factor in humans, and prevent this molecule causing inflammation in both the gut and joints, but do not compromise the patient's immune system as much as immunosuppressants. It has also proved an effective way of treating some with severe active Crohn's disease who have previously not responded to other drugs.



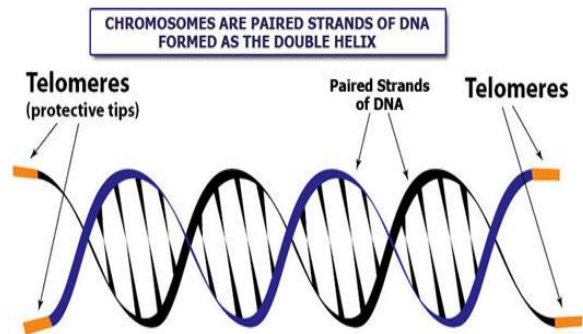
Adalimumab (an anti-TNF drug)

There are other incredibly interesting areas which could lead to new treatments. Incredible research across Europe has shown that autologous stem cell transplant, on those with very severe Crohn's, can be incredibly successful at maintaining longer term remission. However, this research is still in progress. Other slightly more obscure areas of research have shown hookworms greatly decrease the likelihood of getting Crohn's Disease, as it is thought they reduce the body's immune response in the gut, to enable them to grow.

# Why Do We Age? The Telomere Theory – Matt Fenton

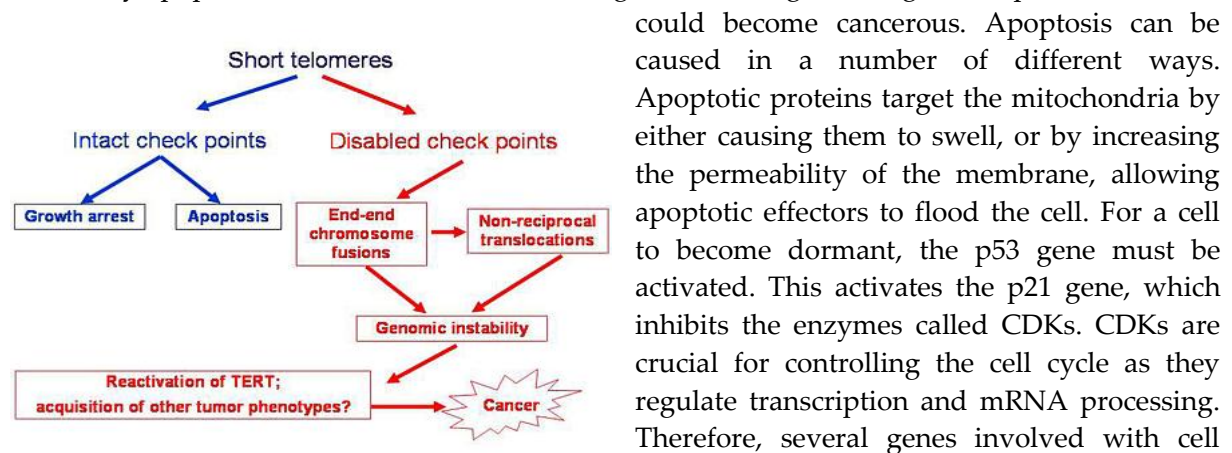
We all know that we are going to die eventually. Maybe we will be run over by a bus. Maybe we will die from myocardial infarction. But if we are lucky enough to avoid all these diseases and chance events, why can we not be immortal? Ageing is the accumulation of changes in an organism over time and these, eventually, lead to our death. The reality is that there is no one cause of senescence, but the Telomere Theory is the most widely accepted hypothesis today. I will also explore telomerase, an enzyme linked to cancer.

Telomeres are a repeated length of base pairs at the end of a stretch of DNA. In most animals the sequence TTA GGG (and consequently AAT CCC) is repeated over and over. Telomeres are roughly 9000 base pairs long at the end of each chromosome, with a single-stranded overhang of a few hundred bases on the 3' end. Elizabeth Blackburn helped to discover them in the late 1970s and likened them to the plastic on the end of a shoelace. She said that telomeres are there at the end of the DNA to stop it unravelling. Matt Ridley uses the analogy of a typewriter in his book 'The Human Genome'. He compares DNA replication to a "photocopier that makes perfect copies of your text but always starts with the second line of the page and ends with the penultimate line". When a cell copies its DNA, it cannot copy it all the way to the end. Therefore, every time the cell copies, part of the telomere is lost. The telomere is lost so that crucial genes at the end of the chromosome are not lost. Clearly, telomeres are only present in eukaryotes as we have linear chromosomes.



The next question is "What happens when we run out of telomeres at the end of our chromosomes?" A cell can usually divide about 50 to 70 times before the telomeres are in danger of becoming perilously short. If a cell tries to replicate without these telomeres, crucial genes may be lost from the ends of the chromosomes and this would be disastrous for the cell. This limit of the number of replications is known as the Hayflick limit.

When a cell has reached its Hayflick limit, one of three things can happen. The cell will commit suicide by apoptosis, become dormant, or if the genome does get damaged in replication the cell could become cancerous. Apoptosis can be caused in a number of different ways. Apoptotic proteins target the mitochondria by either causing them to swell, or by increasing the permeability of the membrane, allowing apoptotic effectors to flood the cell. For a cell to become dormant, the p53 gene must be activated. This activates the p21 gene, which inhibits the enzymes called CDKs. CDKs are crucial for controlling the cell cycle as they regulate transcription and mRNA processing. Therefore, several genes involved with cell



division are not expressed. The cell is said to be dormant, as it is not dividing but it is not dead. It is thought to be the build-up of these dormant cells that could lead to ageing.



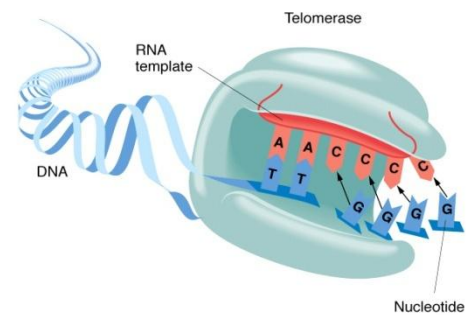
However, it is known that telomeres can be any length between 7000 and 10000 base pairs per end of the chromosome. The length of your telomeres is strongly due to inheritance, so if your parents and grandparents have lived long lives you have a good chance of having long telomeres. Jeanne Calment, who lived to be 121 years old, may have had many more repeats of TTA GGG, so her cells could divide many more times than the average human being. Her brother lived to be 97.

However, one may now be thinking “Surely if long telomeres are coded for in the genetic code, why hasn’t natural selection preferred the genes for long telomeres due to selective pressure?” Unfortunately human beings reproduce at the age of around 20-40 years old. At this point in life, there is no selective pressure to have long telomeres, as this affects our bodies long after reproductive age. There is no advantage to live to be very old, as you cannot still reproduce. Therefore natural selection has designed humans to live long enough to reproduce and then care for our offspring. This is why we cannot get rid of the genes that make us more susceptible to diseases such as Alzheimer’s and Parkinson’s.

Support for the theory comes from evidence in arterial walls. Arterial walls have to constantly withstand very high pulses of blood pressure throughout their lives. Therefore the cells in the wall need to be persistently repaired, which requires cell division. This is the reason we die from hardened arteries (atherosclerosis).

Telomerase is an enzyme that helps to reconstruct the telomeres. It is very similar to reverse transcriptase, as it contains an RNA template. Therefore any cell that expresses telomerase will live for far longer than a normal cell. Unfortunately, the TEP1 gene (coding for telomerase) is switched off in most cells of our body, except for those that it is vital to divide frequently, whilst still having a long lifespan. They prime example of this is the immune system.

The worrying thing about telomerase is that it equips cancer cells to be immortal. 90% of malignant cancer cells express active telomerase, and as a result they can divide continuously without trouble. There is hypothetically a way in which the cancer cell can exploit telomeres and telomerase. Genetic mutations block the signals that are usually emitted when a cell should either become dormant or commit suicide by apoptosis. The cell then continues to divide ‘normally’ as if nothing is wrong and as a result will mutate. If they obtain the necessary mutations, then the cell may become cancerous.



The fact that telomerase is present in 90% of cancer cells and that it is specific to cancer cells means it can be a real target for cancer treatment in the future. If one could use a chemical to inhibit the telomerase enzyme, then the cancer cells would wither and die or become dormant, whilst normal cells would not be affected. This seems like the perfect cure for cancer, as it is specific so doesn’t kill healthy cells and it covers a broad array of cancers. However,

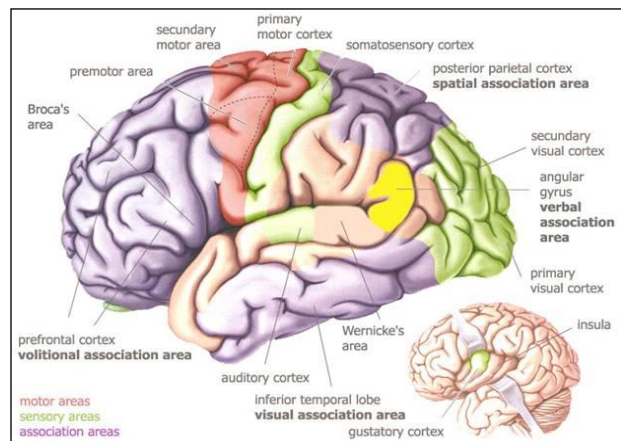
much more research needs to be done. We need to identify exactly which normal body cells actively possess telomerase and how crucial it actually is to them. If telomerase is unacceptably toxic to them, then this method will be null and void. However, whether this method of treatment or another, scientists are beginning to grow in confidence that we will have a cure for cancer in the near future.

# Alzheimer's Disease – Emily McCartney

Alzheimer's is a degenerate neurological disease that currently affects around 417,000 people in the United Kingdom alone, and by 2050 that number is expected to have quadrupled. Although the disease is far more prevalent in the elderly, it is a common misconception that only the older generations are affected by the disease. Generally people develop Alzheimer's from the age of 65. However, Early Onset Alzheimer's, which account for 5-10% of all cases of the disease, and in particular Familial Early Onset Alzheimer's, which refers to cases where a genetic disposition is the cause of the condition, can take hold in people from the age of 16.

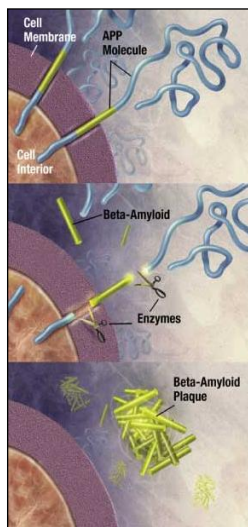
The disease primarily affects the temporal lobe, which is where the primary auditory cortex and hippocampus are located, so unsurprisingly the main symptoms of Alzheimer's affect communication and memory.

Although we know that the condition affects this specific area of the brain, we don't yet know for certain exactly what chemical processes happen to cause the changes and damage to this area. But there are three different main hypotheses that scientists have come up with, starting with the first and oldest hypothesis that was introduced in around the mid 1970s. That was the Cholinergic Hypothesis, in which scientists believed that the brain damage caused by Alzheimer's was due to



a deficiency of the neurotransmitter chemical acetylcholine. However, this hypothesis has generally been dismissed as more research into the neurotransmitter deficiency has come to the conclusion that it does not have a direct causal link with Alzheimer's, but is a result of the widespread brain damage caused by the disease.

The other two hypotheses are based not on chemical secretions but on protein misfolding, with the first being the Tau Hypothesis. Tau proteins form part of the microtubules within the neuron cells, and are responsible for stabilising these cells, but when they become hyperphosphorylated, meaning they become saturated with phosphate groups, they tangle together, causing the microtubule walls to disintegrate, so the neuron cell dies and electrical impulses can obviously no longer be sent along them.



However, once again this hypothesis isn't even nearly certain, and the most accepted hypothesis concerns amyloid plaques. These plaques form between the nerve cells within the brain, as opposed to within the nerve cells themselves. The most common plaques are formed from beta amyloid proteins which are peptide chains comprised of between 39 and 43 amino acids. These come from the breakdown of larger proteins called amyloid precursor proteins, which are necessary in the brain for neuron synthesis. The reason for the precursor proteins breaking down again isn't known, but it causes cell death of the surrounding neurons, and may cause pressure on the surrounding tissues too. This theory has the most support as the gene for amyloid beta precursor protein is located on chromosome 21. People with trisomy on chromosome 21, meaning they have three copies of that chromosome, causing Down's disease, almost all develop Alzheimer's by the age of around 40.

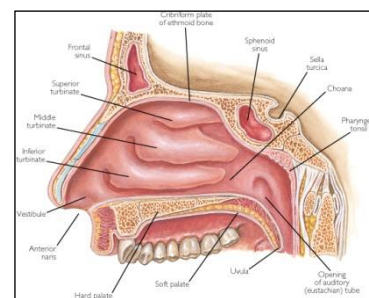
The first signs that a person may develop Alzheimer's disease in later years are often misinterpreted as purely being characteristics of ageing or stress. For example, they main begin to have trouble remembering meanings and recently learned information (semantic

memory) and encounter problems in planning ahead of a series of actions. However, these symptoms clearly do not always mean a person is going to develop Alzheimer's, but are just signs of Mild Cognitive Impairment (MCI). Only a small percentage of those who are diagnosed with MCI will go on to be diagnosed with Alzheimer's – around 10 to 15%. However, should the person go on to develop the disease, these early warning signs can be seen from as long as eight years before the condition and its symptoms become severe enough to be defined as Alzheimer's disease.

As Alzheimer's disease begins to become more apparent, the level of impairment of memory and learning of new facts increases. As well as this the patient may develop problems in speech, communication, and motor functions. Gradually, as these impairments worsen, reliance on other people increases, and the types of memory which were previously unaffected by the disease are steadily lost, and it's at this stage that the sufferer may start to forget names of family and close friends, and important past events. As a result of the person's increasing impairments, and loss of independence, they frequently become subject to many other psychological conditions, and become withdrawn, often due to their communication problems causing them a lack of confidence. In the final stages of Alzheimer's disease, patients lose all independence and become completely reliant on their carers, as they begin to suffer from complete exhaustion and communication is limited to simple phrases or even words only. However, although Alzheimer's disease is classed as a terminal illness, it is not Alzheimer's disease itself that will be the cause of death for the patients. Instead, it is the side effects of the condition, such as the motor skill impairments which can cause falls, or leave the patient bed ridden, leading to pressure ulcers, weight loss or urinary tract infections.

At present, no cure has been found for Alzheimer's disease, and as well as this no drug has been found which is capable of halting or even slowing the progression of the disease. Pharmaceutical treatment currently given to patients is very much just based on combating the symptoms of the disease, so three out of the only four pharmaceutical treatments currently manufactured for Alzheimer's disease are inhibitors of the acetylcholinesterase enzyme, which breaks down acetylcholine. These are Donepezil, Galantamine, and Rivastigmine. However, even these drugs still cannot be completely effective, and particularly in the most advanced stages of Alzheimer's disease, Donepezil is the only approved treatment out of these three drugs. As well as this, treatment for sufferers comes in the form of both psychosocial care (e.g. Art therapy and reminiscence therapy) and care giving by relatives or the NHS (in fact around 30% of hospital beds occupied by adults over the age of 65 are taken by people with some form of dementia, and between 62% and 74% of people in residential homes or some other form of care home are sufferers of dementia.)

Finally, there are various new advances in research looking at both the physiology and genetics of the disease itself, as well as treatment, for example as the research into stem cells continues for a wide range of different illnesses (including many neurological conditions) they also show potential to treat Alzheimer's disease. However, one of the main problems has been finding a way for them to reach the brain, as drilling through the skull to inject them would carry high risks, and injecting them into the blood stream would only carry low levels to the brain. Now, researchers believe snorting stem cells may be the answer, as the cribriform plate between the nasal cavity and the brain contains tiny perforations which would allow stem cells to fit through.



As well as this the drug Rember, which targets tau protein tangles by inhibiting tau protein aggregation, shows great promise; As a clinical trial of the drug just over a year ago with a group of 321 patients who were sufferers of Alzheimer's showed an 81% difference in rate of mental decline compared with those not taking the drug, Rember could be on the market as soon as 2012.

# Muscle Structure and Function – Leo Lobbes

This article is designed to give an overview of the structure, function, and contractile mechanism of muscles.

Muscles are necessary for survival. They are by definition force generators, i.e. a way of utilising chemical energy to generate motion in the body, which every organism needs to survive. Without muscles, we would not be able to walk, move, eat, reproduce, or appreciate our surroundings. Even essential actions we are not conscious of, such as breathing and pumping blood are dependent on muscles.

Being aware of the chemical and physical properties of muscles is therefore essential to understand the human body.

In the human body, there are approximately 650 muscles, making up 25-45 % of the body weight. They fulfil various functions, such as the contraction of blood vessels (enabling thermoregulation) and peristalsis of the gut (for digestion).

Muscles work by converting chemical energy into kinetic energy, causing the contraction of muscle tissue, which applies a mechanical force to parts of our body. This allows locomotion (i.e. moving parts of the body or the whole body), the movement of internal organs and facilitates the heartbeat. Each of these three functions requires a specific type of muscle:

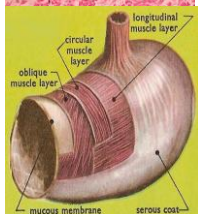
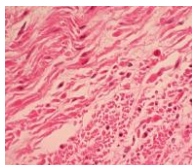
1. Locomotion – performed by *skeletal muscle*
2. Movement of internal organs – performed by *smooth muscle*
3. Heart Beat – performed by *cardiac muscle*

## Skeletal Muscle:



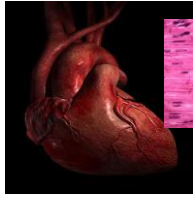
- Movement is voluntary, controlled by the CNS, done consciously
- Referred to as striated (structure: stripes across each muscle fibre, formed by alternating actin and myosin bands)
- Anchored to the bones via tendons
- Enable us to move (skeletal movement)
- Fast contractions

## Smooth Muscle:



- Found in the walls of inner organs
- Responsible for movements there
- E.g. peristalsis of the stomach and gut
- Structure different from skeletal
- Smooth, not striated (not the same regular arrangement)
- Different contractile mechanism
- Important: contraction involuntary, cannot be consciously influenced
- Longer, slow contractions

## Cardiac Muscle:

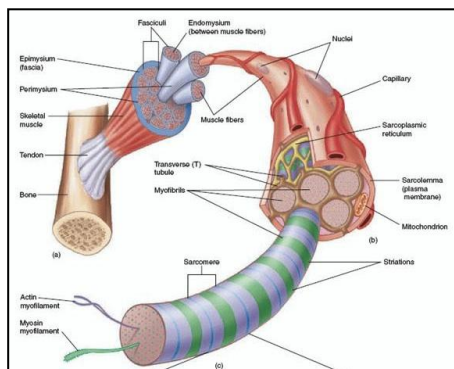


- also called Myocardium
- striated (similar to skeletal muscle)
- but has unique features found only in the heart
- contraction involuntary (myogenic, initiated by SAN, connection to Medulla oblongata which can influence, not stop or start, the heart rate)
- not connected to any bones via tendons

## Functional Anatomy of Skeletal Muscle

All skeletal muscles are made up of numerous fibres, which have diameters from 10-80  $\mu\text{m}$ , depending on the muscle. Each muscle fibre is in turn composed of successively smaller subunits:

- The Sarcolemma: Plasma membrane of muscle cells, which contains collagen and polysaccharides. It provides strength & elasticity, collects into bundles to form muscle tendons, and inserts muscle into bones.
- Sarcoplasm: Matrix inside the muscle fibre, contains nuclei and tremendous numbers of mitochondria.
- Sarcoplasmic Reticulum: Endoplasmic Reticulum with special organisation, contains Calcium pumps, and stores Calcium ions.
- Myofibrils: Threadlike fibrils, each muscle fibre has several hundred to several thousand, contain 3000 actin and 1500 myosin filaments each:
- Myofilaments: contractile proteins myosin (thick filaments) and actin (thin filaments); separated by the Z membrane which passes through the whole muscle
  - Myosin: filaments consist of 200 molecules, lie in apposition to actin filaments
  - Actin: polypeptide chain composed of 3 different proteins actin, tropomyosin and troponin; form double-stranded helix

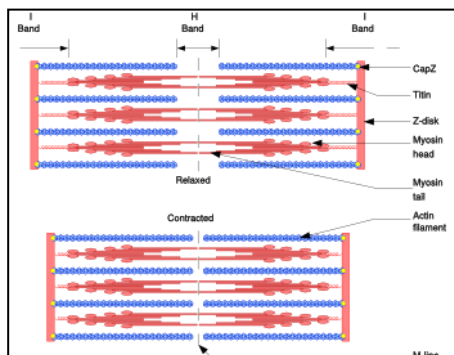


## Contraction Mechanism and Filament Interaction

During muscle contraction, the actin filaments in each myofibril slide into channels between the myosin filaments, shortening the whole muscle.

This happens because the filaments interact when calcium ions enter the myofilaments (Action Potential). The ions bind to the tropomyosin-troponin proteins on the actin, "opening" active sites. The myosin cross-bridges, which lie in apposition to the active sites on the actin, will now bind to the actin strand and pull it along. This process requires ATP.

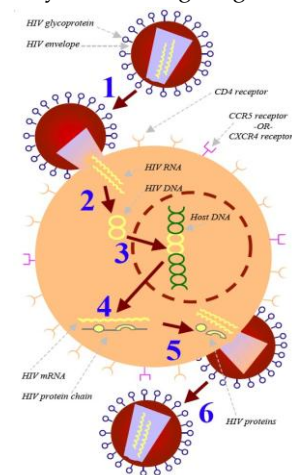
At the end of contraction, constantly active calcium pumps have removed the calcium ions from the myofilaments, so that the active sites on the actin strands are covered once again, and filament interaction stops, leading to the filaments sliding back to their original position. The degree to which a muscle contracts depends on the number of fibres stimulated by an action potential, and the number of action potentials (the actual intensity of an action potential is always the same).



# The HIV life cycle – Stella Hristova

There are probably no educated people, living in modern and developing societies, who have not heard of AIDS. It is caused by HIV (*Human immunodeficiency virus*), which affects major cells in the immune system (i.e. CD4+ T cells and macrophages). In order to develop a way of treating infected patients, scientists use their knowledge of the processes during the HIV life cycle that take place inside the human organism. Thus, they are able to find where and how the virus could be 'attacked,' so that its activity is reduced, and the period until the development of AIDS is increased.

The target cells of HIV are usually T-helper cells. The virus would affect one of them by introducing its genetic material to the cell. For that purpose the membranes of HIV and the T-helper cell fuse together. HIV has two major glycoproteins which take part in fusion - gp120 on the surface of the viral envelope and gp41 embedded in the lipid bilayer of the virus. CD4, on the other hand, is a glycoprotein found on the surface of T-helper cells, which serves as a receptor for HIV. Its shape is complementary to the shape of gp120, and therefore the two will bind together. This allows the virus to bind to a special set of chemokine receptors. There are two important chemokines to which the virus would bind: CCR5 and CXCR4. This binding results in a major change in gp41. The glycoprotein would project three peptide fusion domains, which hit and capture the bilayer of the target cell. These domains form structures which draw both membranes of HIV and T-cell together, and therefore make fusion easier. Finally, the whole virus enters the now host cell.



The chemokine receptors are crucial for the development of HIV infection. If, due to mutation, one lacks CCR5, then he is automatically resistant to infection. This suggests that by preventing the virus from binding CCR5 would decrease HIV spread, and studies in that area can result in an effective class of antiretroviral drugs.

When the virus is inside the T-cell it begins uncoating and its capsule dissolves. This results in the liberation of various viral contents in the cytoplasm, including the reverse transcription complex. It consists of viral RNA, lysine transport RNA (tRNA<sub>3<sup>lys</sup></sub>) and enzyme reverse transcriptase, and serves for carrying out reverse transcription. Reverse transcriptase transcribes viral RNA into viral DNA. This happens due to the presence of two catalytic domains in the enzyme: Ribonuclease-H active site and Polymerase active site. At the polymerase active site, the RNA of HIV is transcribed, and as a result an RNA-DNA double helix is formed. Then, at Ribonuclease-H, RNA is broken down, so that now the single DNA strand is transcribed at the polymerase active site. The result is the formation of a double viral DNA helix (cDNA). Various errors take place during this process and thus mutations can occur and respectively cause drug resistance. Reverse transcription can be blocked by using reverse transcriptase inhibitors.

In order for HIV to affect the T-cell, it will have to integrate its newly formed DNA into a chromosome of the host cell. As a result, a pre-integration complex (PIC), containing cDNA, integrase, and DNA-binding cellular protein HMGI(Y), is formed. It moves towards the nucleus using microtubules for directional movement.

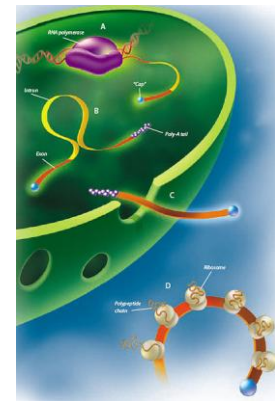
The first step of integration is when integrase enzyme from the PIC binds to both ends of the viral DNA, immediately after reverse transcription. This binding occurs in the cytoplasm of the host cell. Afterwards, still in the cytoplasm, integrase breaks each 3' viral DNA ends. Therefore, two nucleotides are lost, and now the end of each strand of HIV DNA is a 5' which 'hangs aside' and has a very reactive hydroxyl group. Following, the PIC has to enter the nucleus through the pores of the nuclear envelope, although it is twice the size of a pore. Scientists still do not know how this process takes place and find it the hardest part of HIV research, which, on the other hand, can be of great use for the development of new treatments.

As soon as PIC enters the nucleus, a host enzyme binds it and the host DNA in a way which is still not fully understood. Then, the hydroxyl group on the 5' end of viral DNA 'attacks' both strands of the host DNA. Integrase from PIC catalyzes this process. This results in the breaking of hydrogen bonds between bases and the phosphodiester bonds. Therefore, the hydroxyl group from viral DNA would bind the newly formed phosphate ends of the host DNA, thus joining them together and resulting in a new DNA molecule which begins to unfold. However, there still are areas of host and viral DNA that are unpaired and form the so-called 'gaps'. After repair

processes take place, a functional provirus is formed. This is the viral DNA, fully integrated into a host chromosome.

After integration a period of latency may follow. This is post-integration latency. It is of great clinical importance. During that time little virus is produced and we still do not know what exactly causes it. The viruses cannot be killed by chemotherapy during latency—they cannot be affected in any way. There is also pre-integration latency. After reverse transcription, the PIC can remain in the cytoplasm because there is not enough ATP to transport it inside the nucleus. When the T-cell becomes activated, more ATP is produced, and thereby the upcoming events of HIV life cycle can take place.

Next step of this cycle is transcription. At this point the proviral DNA is transcribed into mRNA by the host cell enzyme RNA polymerase II (RNAPII). The process is enabled by the action of a nuclear factor called NFκB, which becomes activated when the host T-cell is activated during an immune response. NFκB increases the rate of initiation of transcription, and only after it binds to the proviral DNA, will the process begin. As a result, short and stable transcripts, which remain in the nucleus, are synthesized. Also, small amounts of the HIV proteins Nev, Tat, and Rev are synthesised. As the protein Tat raises in quantity, it binds to regions of the LTR and the rate of further transcription of the proviral DNA increases too. Therefore, many different transcripts are formed, which are multiply spliced by the host cell RNA splicing machinery inside the nucleus, and finally encode for the viral proteins Tat, Rev and Nev. The protein Rev acts as a regulator of gene expression, which means that it thus can regulate splicing. This happens by interaction between Rev and the splicing factor ASF and its associated protein p32. If splicing is inhibited, this leads to the production of only singly spliced or non-spliced mRNAs in the nucleus. Rev also binds to the so called Rev Response Element (RRE), which leads to a significant increase in the number of protein molecules. Due to its many nuclear export sequences and its increased number, Rev enhances the export of singly spliced and non-spliced mRNAs from the nucleus into the cytoplasm.



Viral assembly is one of the last steps of the HIV life cycle. It takes place at the plasma membrane of the host cell. Three types of proteins are responsible for making up the virus. These are gp120, gp41 and gp160, which are all membrane protein complexes. Apart from that, two other, internal proteins Gag and Gag/Pol also take place in assembly. The Gag/Pol protein complex (or polyprotein) is formed as translation of viral RNA terminates at the POL gene instead of the GAG gene. This is a once in twenty transcriptions. All protein complexes aggregate at the cell membrane. The Gag/Pol protein draws two strands of viral RNA in the developing virus. Apart from that, the enzyme protease, which is a part of the polyprotein Gag/Pol, cuts itself free. As a result, protease begins to cleave Gag/Pol into smaller polypeptides, such as enzymes as reverse transcriptase, integrase, etc. In addition, protease also cleaves remaining Gag and Gag/Pol into structure proteins. Therefore, viral protease is vital for the life cycle of HIV, since viral proteins are not functional, unless they are separated. Gp160 is translated from a singly spliced mRNA. It is a membrane protein, as mentioned earlier, and it spans the membrane once. It passes from the Endoplasmic Reticulum to the Golgi apparatus, where it is cleaved by a host enzyme to gp120 and gp41. It differs from the other polyprotein as it is not cleaved by the viral protease. The cleavage of polyprotein is much more significant during budding and viral maturation.

All protein complexes, that were previously synthesised and assembled, associate with an area of the plasma membrane, known as lipid rafts. The lipid rafts are regions of the host cell's membrane with high concentration of lipids, cholesterol, and protein. The newly formed virus buds from this region. Consequentially, the viral envelope lipids would reflect the composition of the raft. When observed on an electron micrograph, the association with rafts appears darker. Later, the viral particle pinches off the host cell membrane. Thus, the plasma membrane of the CD4+ T helper cell is damaged; the cell becomes significantly weakened and dies. The virus is now released in the body, ready to infect other T helper cells. More cleavage takes place during this time, until the viral particle reaches maturation.

This marks the end of the HIV life cycle. The scientific knowledge we have gained until now has helped in developing new types of antiretroviral drugs. The effect of these is more than obvious for us today, as more HIV positive people live longer and develop AIDS later in their life (20 years or more). If we understand even more about the HIV life cycle, we will be able to develop other antiretroviral drugs and thus, give the opportunity for infected people to live until old age.

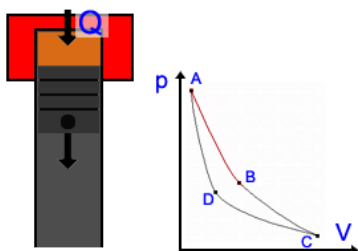
# The Basic Principles Behind Heat Pumps – Franz Richter

A lot of people worry about their cost of heating in their homes, especially after the latest news that the price of energy will increase by between 15 to 20 percent until 2020.

However, there is an option by which one can save money until 2020 and not have to worry about the prices rising. I am talking about a new heating system which uses our environment to warm up our rooms. This system is called a heat pump.

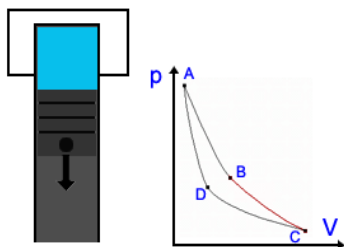
But how can this work? How can you warm up something warm with something cold? Surely it isn't impossible?

To explain this we have to look at the Carnot cycle and the basics of the Carnot cycle; the ideal gas laws.



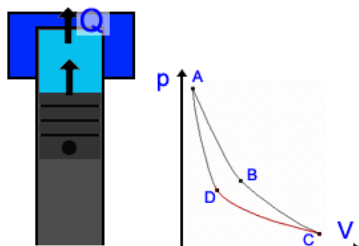
## Isothermal expansion

In this step we have an ideal gas (orange), a piston and a heat emitter (red). The gas absorbs heat from the heat emitter and expands. It does work on the piston. Therefore the volume of the gas is increasing: As a result of that, the pressure of the gas is decreasing (Boyle's Law), due to the fact that the temperature stays constant. The energy which is absorbed in form of heat is immediately transferred into work which is done on the piston.



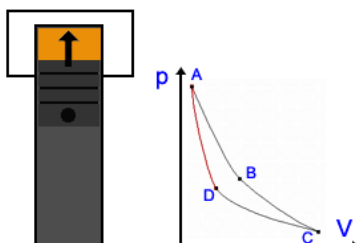
## Adiabatic expansion

An adiabatic process: is a thermodynamic process in which no heat is transferred to or from the working fluid. In the second step of the Carnot cycle, we remove the heat emitter. However the gas still expands. It does work on the piston, but this time the gas gets no energy from the heat emitter, so the gas cool down, due to the fact that the kinetic energy is transferred into work. The volume of the gas still rising and the pressure is still falling (Combined gas law).



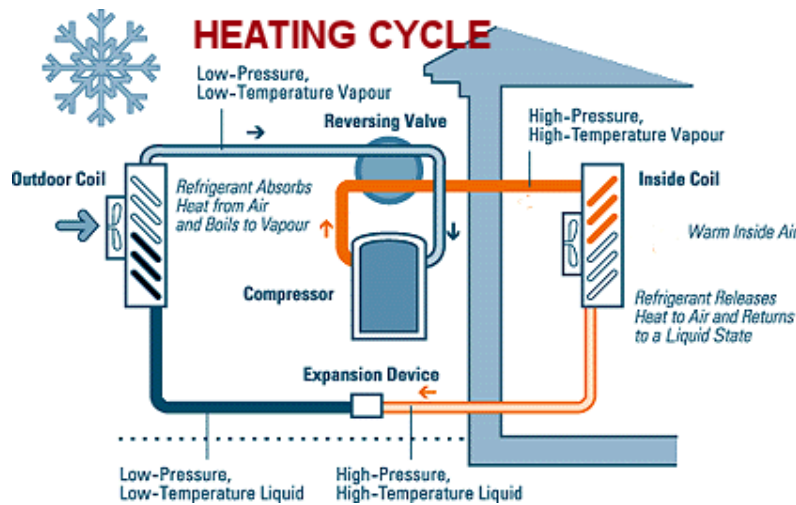
## Isothermal compression

In the third step we are starting to go back to our starting point. We know we have a piston and an ideal gas, but we also have a heat absorber. So in the third step, the piston does work on the gas, the energy gained through the work done on the gas is transferred into heat and emitted to the heat absorber from the gas. Therefore the temperature stays constant. As a result of this process the volume of the gas decreases and the pressure increases (Boyle's Law).



## Adiabatic compression

Now we remove the heat absorber. The piston does work on the gas; however we know the temperature of the gas rises because it gains kinetic energy from the work done on it. Furthermore, through the movement of the piston, the volume of the gas decreases and the pressure therefore increases (Combined gas law).



Now I will explain the heating cycle of a heat pump, which follows the reverse Carnot cycle.

We are starting at the outdoor coil which is equal to the evaporator. The refrigerant (liquid and vapour flowing in the cycle) goes into the evaporator with a temperature of  $-2^{\circ}\text{C}$ . There it gets warmed up with the heat from the air (when the air is colder than  $2^{\circ}\text{C}$ , the refrigerant is at a lower

temperature). It reaches its boiling point and goes into a vapour, and in the end it comes out of the outdoor coil with a temperature of  $3^{\circ}\text{C}$  (Isothermal expansion).

After this, the vapour goes into the compressor, which increases the pressure from 1.7 bar to 13.5 bar, which causes an increase in the temperature to  $73.5^{\circ}\text{C}$  (Adiabatic compression).

The third step is the condenser (Inside Coil). The refrigerant emits heat to the liquid which warms up the room. As a result of that, the temperature falls under the boiling point and the vapour goes into a liquid with a temperature of  $48^{\circ}\text{C}$  (Isothermal compression).

At the expansion device we are going back to the starting point. The pressure of the gas decreases from 13.5 bar to 1.7 bar. Therefore the temperature falls from  $48^{\circ}\text{C}$  to  $-2^{\circ}\text{C}$  (Adiabatic expansion).

Hydrochlorofluorocarbons are usually used as refrigerants.

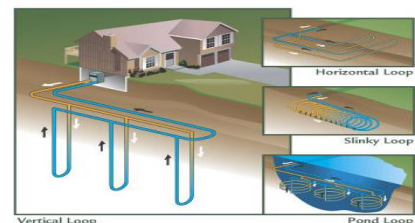
### Types of Heat Pump

There are two different types of heat pumps: air source and ground source heat pumps.



An air source heat pump uses the air to warm up the refrigerant. However, because of temperature changes in the air, the air source heat pump is less effective than the ground source heat pump, but still far more effective than normal heating systems. It is relatively cheap (£3000) with a pay-back time of fifth to sixth years and it needs only a small area outside.

The ground source heat pump is one of the most effective heating systems. It is four times as effective as normal heating systems. However a ground source heat pump is quite expensive (about £12000), the pay-back time is approximately 12 years and it needs a lot of space, as you can see on the picture. The heat exchangers shown can go up to 70 metres underground.

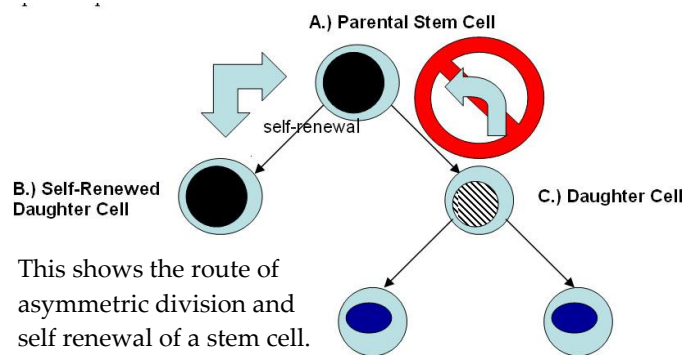


In the end we can say that a heat pump is a sensible investment for the future, due to the fact that it saves a lot of money after the payback time, and it will save energy. For example if you put one joule of electrical energy into the compressor of a ground source heat pump, you can move a volume of vapour containing four joules of energy. Even if you only live a short time in your home, you have still done something for the environment.

# Stem Cells: The Future of Medicine? – Eleanor Budge

Stem cells are the most primitive form of all cell types from all of the tissues and organs of a biological system. The word “stem” lends to the straightforward interpretation of the kinds of cells they really are; cells that are the origin or beginning of all other cell types, and cells in which all these other cell types eventually can arise from. Stem cells are often broadly classified as such, but many different kinds of stem cells exist, providing for a broad spectrum of proliferated progenitor cells that ultimately lead to the creation of every adult cell type necessary for sustaining life, whether it be a haematopoietic stem cell giving rise to all of the cell types typically found in blood, a cardiac stem cell giving rise to cardiac myocytes in the heart, a liver stem cell giving rise to hepatocytes in the liver, or a neural stem cell which can give rise to neurons.

Stem cells possess three important properties which differentiate them from mature cells. Self-renewal or proliferation is the ability of a stem cell to produce a progeny cell that is identical to the mother stem cell. When stem cells produce these clonogenic cells that will remain primitive and refrain from proliferation, the cells are said to have maintained ‘stemness’. The self-renewed stem cell is identical with regards to the expressions of cell-surface molecules, relative ‘stemness’, and the nature of the chromosomes amongst other things.



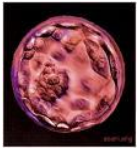
The second important property of stem cells is that of asymmetric division. This is when each stem cell produces two progeny cells that are initially the same as the parent, but one has the genes that give it the potential to continuously proliferate and become a more differentiated mature cell. The other progeny cell does not proliferate and remains identical to the mother stem cell, thus stem cells are able to maintain themselves in numbers.

The third and perhaps most impressive characteristic stem cells show is the ability to differentiate and “migrate” to where they are needed in the body. This is when a stem cell develops and becomes more specialised at each stage until it finally becomes a mature specialised cell in the body. Scientists are just beginning to understand the signals inside and outside cells that trigger each stage of the differentiation process. The internal signals are controlled by a cell's genes, which are interspersed across long strands of DNA, and carry coded instructions for all cellular structures and functions. The external signals for cell differentiation include: chemicals secreted by other cells, physical contact with neighbouring cells, and certain molecules in the microenvironment. The interaction of signals during differentiation causes the cell's DNA to acquire epigenetic marks that restrict DNA expression in the cell and can be passed on through cell division.

Every type of stem cell has a different type of potential ranging from the ability to be isolated and cultured to form a complete human being, to a limited potential of differentiation to a limited selection of specialised cells within a body system.

Embryonic Stem Cells (ESCs), taken from a morula (seven day old embryo), have the potential to develop into a complete human being including the umbilical cord, amnion and placenta. They are said to be totipotent.





ESCs removed from the inner cell mass from a blastocyst are able to develop into any type of cell but cannot form a whole human being if extracted and cultured in vitro. They are said to be pluripotent.

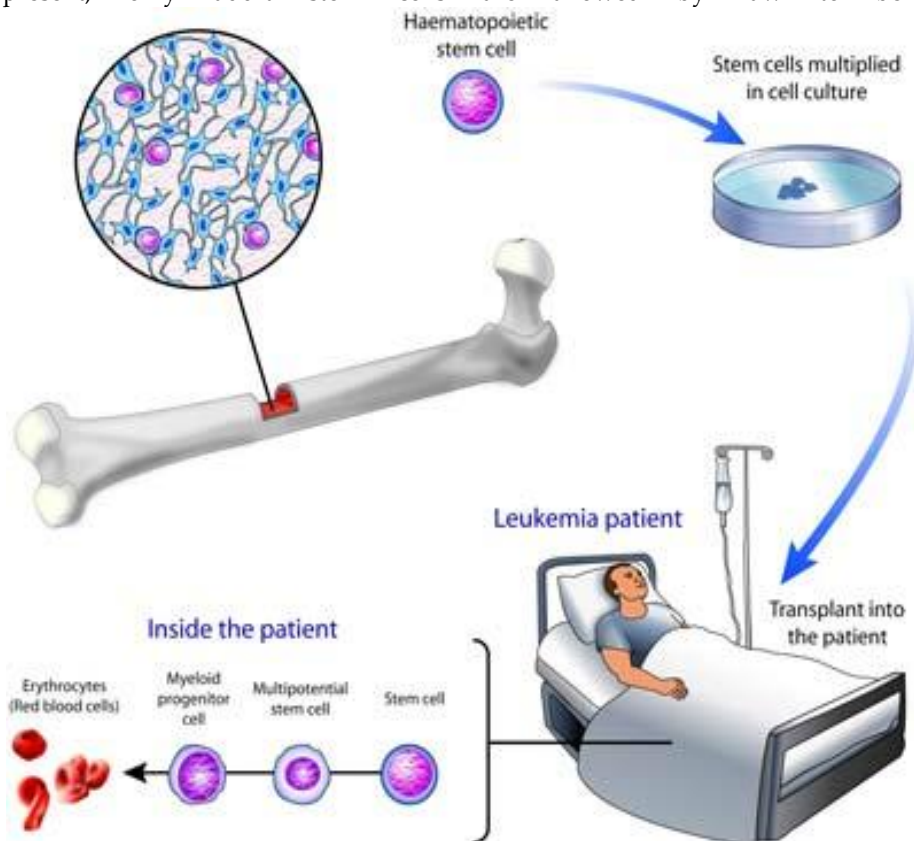
Fetal Stem Cells (FSCs) are responsible for the initial development of tissues before birth and are also therefore also pluripotent.



Blood taken from the umbilical cord of a new born baby will contain umbilical FSCs which are genetically identical to that baby and have a limited potency. These are said to be multipotent.

Adult Stem Cells reside in already developed tissues and direct the maintenance and growth of preformed cells. They are also multipotent.

By comparing the advantages and disadvantages of each type of stem cell, we can reveal the complexity of the task of finding a way in which these cells can be used in an ethical and effective manner: Umbilical cord stem cells do not promote tumours, unlike embryonic stem cells. Umbilical cord stem cells also do not cause immune reactions. Since these immature cells do not express adult tissue-type antigens on their surfaces, these proteins do not seem to cause either an immune reaction in the recipient, or a graft vs. host reaction against the recipient. This is not the case with adult stem cells, which do express adult tissue-type antigens. Unlike foetal stem cells, umbilical cord stem cells do not require the death of a human baby or foetus. At present, only adult stem cells are allowed by law to be used in treatments.

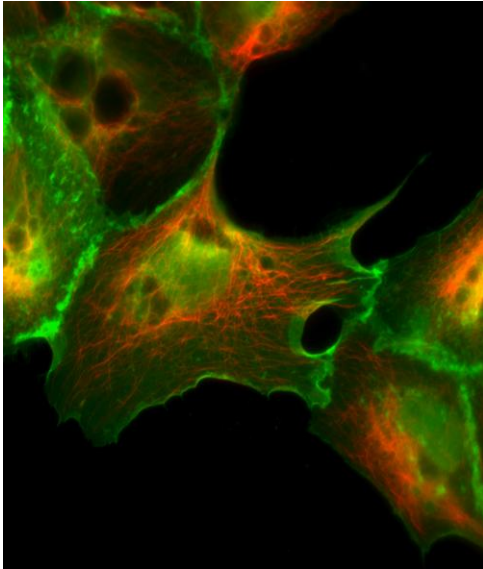


Above is an example of how stem cells are used to treat leukaemia patients.

Overall, stem cell treatment has great potential in preventing ageing, repairing tissues and structures in the body, and even provides the possibility to an infertile person of their own biological child through the use of manipulating stem cells to form gametes. All of these may be theoretical now, but who can say what will happen in years to come?

# The Cytoskeleton - Oliver Claydon

The cytoskeleton is a dense network of protein filaments held within the cytoplasm of the cell, and in



my presentation I discussed its structure and many important functions. Indeed the range of functions of the cytoskeleton is vast. Intermediate filaments, the first type of cytoskeletal fibre, are vital in stabilising, protecting, and maintaining the cell shape. This is especially important in epithelial cells on the skin where they play a large role in keeping the cells from rupturing when we put pressure on them. The second type, microtubules, controls a huge range of functions; for example the movement of flagella, the tail-like projections of sperm cells and such, and intracellular transport, the mechanism in which lysosomes and organelles are moved across the cell on demand. Finally, actin microfilaments help the cell to change the shape of its membrane, in processes such as phagocytosis, cytokinesis and cell crawling.

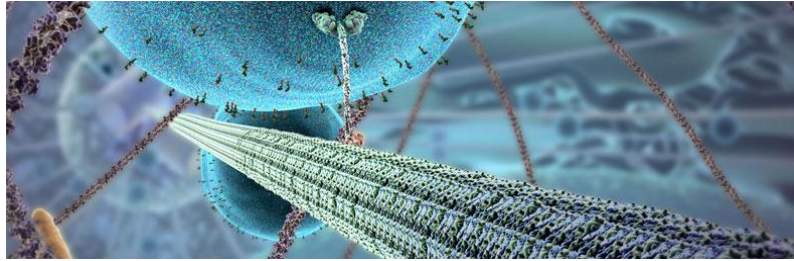
The basic subunit is an  $\alpha$ -helical strand of protein. Two of these strands then coil together in an antiparallel arrangement (both facing different directions) to form a dimer. Many dimers stack together to form an intermediate filament that stretches across the cell, connecting both sides of the plasma membrane. The protein from which the filaments are formed differs between different types of cell. Intermediate filaments in epithelial cells, for example, are made from a protein called keratin, whereas mesenchyme cells in connective tissue are made from a protein called vimentin.

This has important applications in the diagnosis and treatment of certain tumours. The doctor can extract cells from the tumour, separate the intermediate filaments and add chemicals that fluoresce in the presence of certain proteins, thus determining the intermediate filament protein and where the tumour originated. Treatment can then be especially designed to be effective against that type of tumour. The primary structure of amino acids in the protein keratin makes it exceptionally strong, and well suited for epithelial cells in the skin and gut. It contains high proportions of two small, uncharged amino acids, glycine and alanine, which means dimers can fit together closely, and the sulphur containing amino acid cysteine, which lets many strong disulphide bridges form between them.



The basic monomer of a microtubule is a dimer of two proteins called  $\alpha$ -tubulin and  $\beta$ -tubulin. These dimers then stack end-to-end, forming a hollow cylindrical structure. One of the most remarkable functions of microtubules is intracellular transport, such as when structures need to be moved across the cell. For example; a lysosome containing digestive enzymes can be moved from the Golgi body to the plasma membrane, to be released into the stomach. The lysosomes attach to a group of motor protein called kinesin, which, with the lysosome in tow, literally walk along the microtubule, moving the lysosome to where it needs to be. In this case, the microtubules act as tracks or pathways connecting different parts of the cell.

The 'hand-over-hand' motion is brought about by a series of interactions, involving the hydrolysis of ATP on the kinesin molecule to produce mechanical work. These motor proteins move at an astounding rate: around 4mm per hour, or 111 body lengths per second, considerably faster than a cheetah at full sprint, which moves at around 25 body lengths per second. It is this mechanism that allows microtubules to play important roles in other cell processes. In mitosis, microtubules act as the



spindle fibres that pull chromosomes apart. The kinesin protein walks along the microtubule with the chromatid in tow, whilst the microtubule depolymerises behind it, creating the impression that the spindle

fibres are contracting. In flagella in sperm cells, 10 pairs of microtubules stretch up the length of the tail. Kinesin proteins, attached to one of the microtubules, walk up the other microtubule, causing them to rub against each other. When this happens in all 10 pairs, it results in the bending and swaying of the flagella, propelling the sperm cell forward.

The network of actin microfilaments grouped around the plasma membrane allows the cell to change its shape, and move around in processes such as endocytosis, exocytosis, phagocytosis, cytokinesis and cell crawling. The monomer of an actin microfilament is a protein called globular-actin, which polymerises to form filamentous-actin, and two filaments twist together to form a microfilament. The polymerisation and depolymerisation of actin microfilaments is tightly regulated by the cell. When microfilaments need to be depolymerised, the protein cofilin is activated, which locks on to and twists the filament, helping to break it down. If it needs to be polymerised again the protein profilin is released, which helps to bind ATP to globular-actin subunits, changing their shape and making it easier for the subunits to bind together.

Like microtubules and kinesin, the actin microfilaments rely on a motor protein, called myosin, to produce the force that causes the cell to change shape. Myosin is very similar to kinesin, in that it hydrolyses ATP to produce mechanical work, and walks along the microfilament in a hand-over-hand motion. Kinesin is involved in a number of processes, one of which is cell crawling. This is the way in which cells are dynamically able to change their shape to move around their environment, for example a white blood cell moving towards a foreign body. The process starts with the rapid polymerisation of microfilaments on the leading edge of the cell, pushing the cell membrane forward. Next, the membrane locks onto the surface it's travelling on using specialised adhesion sites. Finally, myosin proteins walk along microtubules on the leading edge of the cell, whilst carrying another microtubule in tow, causing the overall contraction of the microfilament network. This results in the entire cell being pulled forward, and this process repeats to move the cell to its new destination.

This is just a summary of the structure and huge range of functions of the cytoskeleton. Although the cytoskeleton plays a role in many of the processes which take place inside living cells every second, it is still not fully understood. New research into the cytoskeleton, however, should bring about many medical advances, and give us a greater understanding of how we are built.

# Supersymmetry – Andy Paine

You will, inevitably, have heard about the Large Hadron Collider (LHC for short) at CERN in Geneva; also dubbed “the black-hole machine” due to predictions that, however low, there was a possibility that it may create a black hole. This collider essentially runs particles up to extremely high energies by blasting them around its 27km circumference, then smashing them into each other. Within the resulting mess, they hope to find the “God particle” – the theorised Higgs Boson, the particle which transmits mass onto all other particles. However important the Higgs Boson is, it is merely one part of a grander theory of the universe, started in 1960 by S. L. Glashow, which has evolved to become today what is known as the Standard Theory.

## Standard Theory

Three Generations of Matter (Fermions)				
	I	II	III	
mass →	2.4 MeV	1.27 GeV	171.2 GeV	0
charge →	$\frac{2}{3}$	$\frac{2}{3}$	$\frac{2}{3}$	0
spin →	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	1
name →	<b>u</b> up	<b>c</b> charm	<b>t</b> top	<b>γ</b> photon
Quarks	4.8 MeV	104 MeV	4.2 GeV	0
	$-\frac{1}{3}$	$-\frac{1}{3}$	$-\frac{1}{3}$	0
	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	1
	<b>d</b> down	<b>s</b> strange	<b>b</b> bottom	<b>g</b> gluon
Leptons	$\ll 2.2$ eV	$\ll 0.17$ MeV	$\ll 15.5$ MeV	91.2 GeV
	0	0	0	0
	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	1
	<b>ν<sub>e</sub></b> electron neutrino	<b>ν<sub>μ</sub></b> muon neutrino	<b>ν<sub>τ</sub></b> tau neutrino	<b>Z</b> weak force
	0.511 MeV	105.7 MeV	1.777 GeV	80.4 GeV
	-1	-1	-1	$\pm 1$
	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{2}$	1
	<b>e</b> electron	<b>μ</b> muon	<b>τ</b> tau	<b>W</b> weak force

Today, nearly all quantum theories revolve around the hugely successful Standard Theory. The theory dictates that there are 16 particles which make up all other particles in the universe, 12 fermions and 4 bosons (this is before the suggestion of the Higgs Boson). The 12 fermions were split into two groups, leptons and quarks. Leptons include electrons and particles called taus and are the particles which make up matter, whilst bosons such as the photon and the gluon transmit forces in the universe. The bosons therefore serve to explain three of the four fundamental forces of the universe – the strong, weak and electromagnetic forces. The gluon is involved in the strong interactions that hold neutrons and protons together. W and Z bosons transmit the weak force, responsible for nuclear decay, and photons are a massless particle, most famously used in transmitting light.

## Problems with Standard Theory

As previously mentioned the theory explains beautifully three of the four fundamental forces but unfortunately falls short on the fourth – gravity. As of yet, standard theory hasn’t quite managed to provide an explanation for this however the suggestion of a sixth boson, the graviton, may aid in finding a solution. Secondly there is the “hierarchy problem”, this is an inconsistency with the experimental and theoretical mass of the Higgs boson. Thirdly is a seemingly negligible difference, but in the early days of the universe when the lightest three elements were made (Hydrogen, Helium and Lithium) there should have been set amounts of the isotopes Lithium-6 and Lithium-7. However the measured amount of Lithium-7 is around about a third of the predicted value, and the prevalence of Lithium-6 is over 1000 times too much. Lastly and arguably the most important is the fact that Standard Theory is yet to explain dark matter – a mysterious type of matter which has been calculated to outnumber ordinary matter by 4:1.

## Supersymmetry

So what is Supersymmetry?

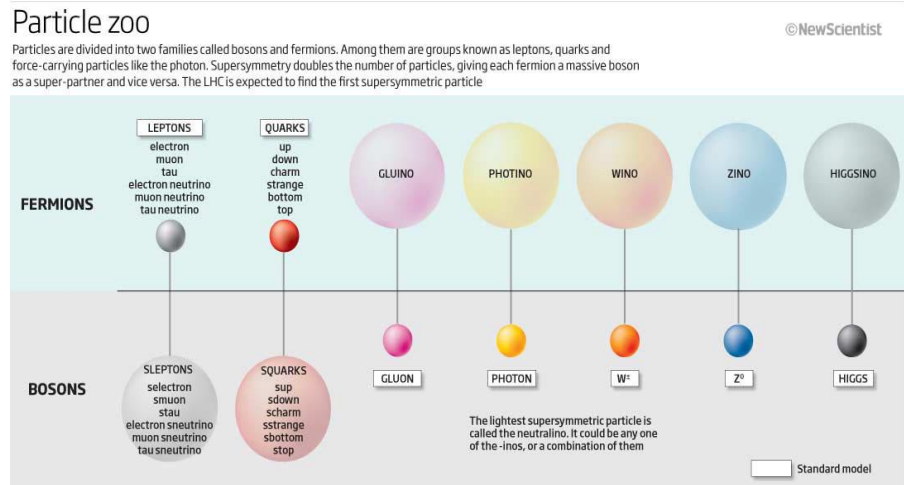
Supersymmetry or SUSY for short, is a theory dictating the existence of twice the number of particles in the universe. SUSY says that for every fermion, there is a giant boson partner

and that for every boson there is a giant fermion partner. This then produces fermions such as Photinos and Gluinos as well as whole groups of bosons called Sleptons and Squarks, from which all the super-symmetric bosons are formed (by placing an 's' in front of the name of each previous fermion).

### Supersymmetry's solutions

Thankfully, SUSY may provide us with some answers to the problems of Standard Theory. The "hierarchy problem" is one of SUSY's major selling points, due to the massive partners of our particles, the mass discrepancies of the Higgs boson drop straight out of the equations. SUSY also provides us with an answer to the Lithium issues – in the early seconds of the universe, gravitinos will have decayed in the primordial soup of light elements, throwing extra neutrons and protons into the fray, causing the Lithium-7 to form earlier than normal, and then decay under the higher temperatures, resulting in less Lithium-7. Also, these extra neutrons could cause a chain reaction of different nuclei decay to produce Lithium-6. In addition to this, staus, the super-symmetrical partner of the tau lepton, could catalyse the decay of boron-8 by binding to it and causing it to decay into two Helium-4 nuclei instead of Lithium-7, as well as this, staus could also bind to Helium-4 a few hours post-big bang. This new Helium-stau state fuses tens of thousands of times more efficiently than normal to produce Lithium-6, resulting in huge amounts more Lithium-6 than normally expected. Lastly and possibly most notably there is, as is often the case in quantum physics, a question. Where are all these super-symmetric particles today? The answer lies in the fact that all of these particles should have decayed into neutrinos (the lightest super-symmetrical particle) by now and, when the prevalence of these neutrinos is calculated, this super-symmetrical matter should outnumber traditional matter by 4:1. Sound familiar? Unfortunately SUSY cannot yet explain gravity, nevertheless it is a natural extension from the Lithium-7 solution that for our gravitinos to exist, there must be gravitons.

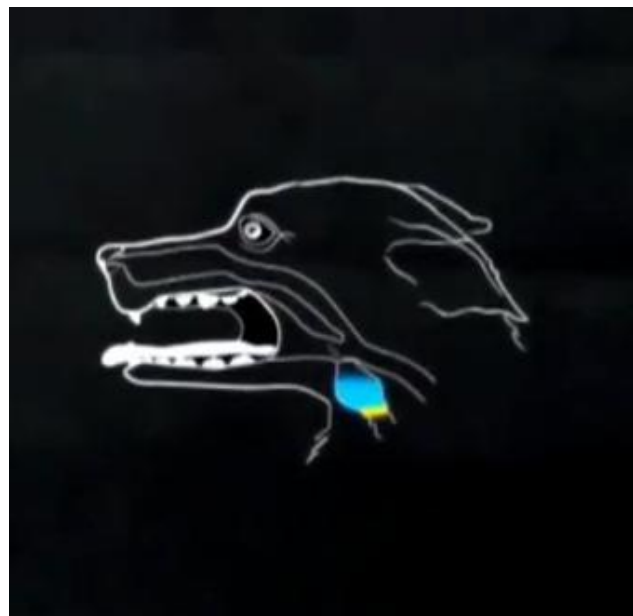
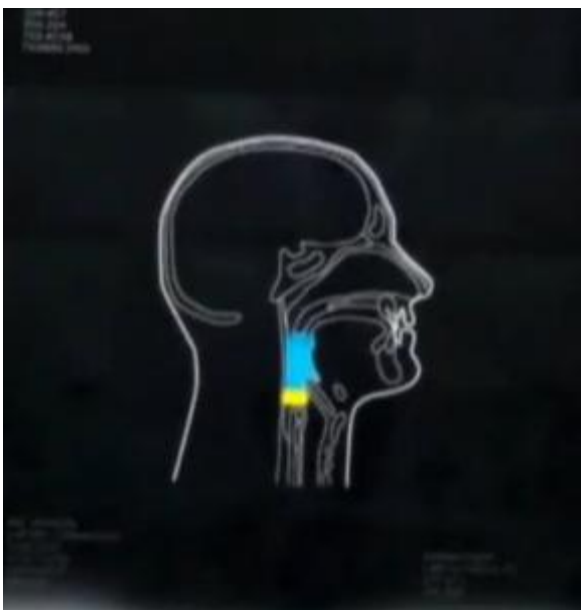
However convincing Supersymmetry may be, it is far from the answer to all our problems. The holy grail of physics is a grand unified theory of the universe; a rule that can explain all the interactions around us and the rest of the cosmos. Regardless of one theory's strengths it will always have weaknesses, it is up to other theories to fill in these gaps to reach that grail.



## Speech: Nature or Nurture? – Sally Ko

Is the way we use language an innate ability that we are born with already? If we had grown up without hearing a sound, would we still be able to develop our own language? Or, on the other hand, do we rely completely on what we inherit from our parents and learnt from our surroundings?

First of all, maybe the big question is: “Why are we the only species on earth that can talk, and produce a language?” Dogs can bark, lions can roar, but humans are the only ones to install grammar and order within these sounds. At first, it was thought that the reason for our ability to speak was because of the difference in position of the vocal tract. Our larynx is further down in our throat than other animals, making our vocal tract longer, and therefore able to produce the wide range of different sounds.



However, this proved not to be the answer to the question, as it was later discovered that when an animal would produce a noise, their larynx would lower, and the length of their vocal tract would extend, providing the ability to produce the sounds that we do. By process of elimination, it must be our brain that separates us from all other mammals.

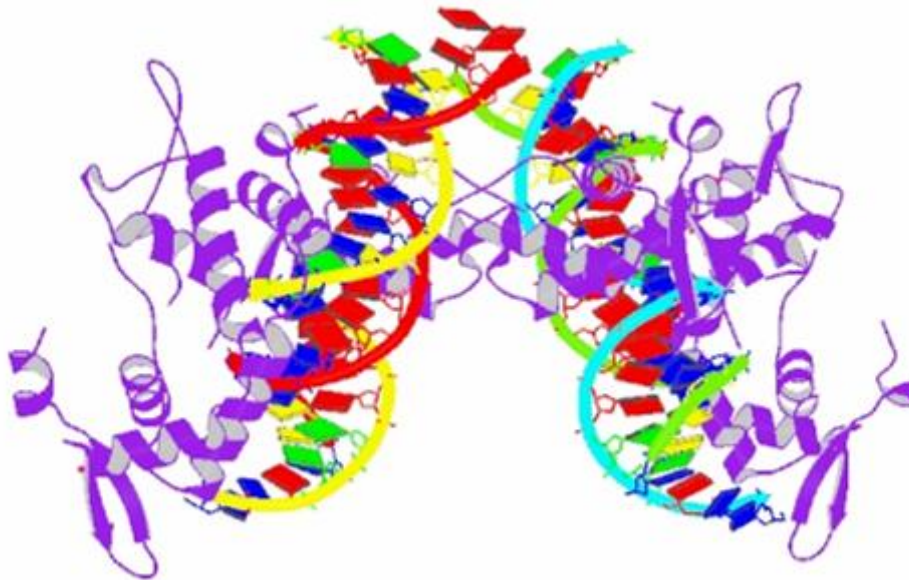
In general, the left side of the brain was most important for language, the front part of the brain was important for speaking, and the back part of the brain is most important for understanding speech. We need to use our memory, our senses, our precise motor control over our mouths and tongues to produce a fully fledged ability for language.

There are two major areas of the human brain that are responsible for language: Broca's area, which is thought to be partially responsible for language production (putting together sentences, using proper syntax, etc.), and Wernicke's area, which is thought to be partially responsible for language processing (untangling others' sentences and analyzing them for syntax, inflection, etc.).

So, is it the process of natural selection the reason why our brains have evolved in a way that provides with the ability to develop language? Language first appeared between 30,000 and 100,000 years ago in the species *Homo sapiens*. But how did language evolve? Many believe that language was an adaptation of some sort, to give us the advantage of communication and order, but some believe language to be the by-product of other evolutionary processes, not a special adaptation that arose by ordinary natural selection acting on mutations. They think that our ability for language was due to adapted neural structures formerly used for other functions.

It has also recently been discovered that language is not just a matter of learning, but is also instinctive. This shows that our ability to talk is really partly innate. There are genes involved in this innate instinct on chromosome 7. It is called FOXP2.

FOXP2 is a transcription factor - in other words it has the potential to affect the expression of an unknown, but potentially large number of other genes, and it is also known to be important in the development of the brain. It controls our unique ability to shape words, and the shaping of lower part of the face in order to be able to communicate, and the tiny movements that allow us to sequence the speech sounds that results in our fluent speech.



However, we do know that language is not completely innate, and it has to be learnt through our surroundings, it has to be taught to us. But how big is the influence of nurture, regarding speech? What would happen if we completely deprive a newborn baby from any sound at all? Unfortunately, the answer is still unknown, as it is a forbidden unethical experiment.

In conclusion, beyond our innate ability and our upbringing, there are more factors leading up to why we talk. Language is made up of many multiple different components. The innate abilities, the learnt abilities, what we inherit, and what we acquire, what we're given by our culture, our society, our parents, and what we get from our genes, all of those things need to be in place to have the full, fully fledged abilities with language.

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