Bang!

The Engineering Issue

Oxford Science Magazine
15th Edition
Michaelmas Term 2013

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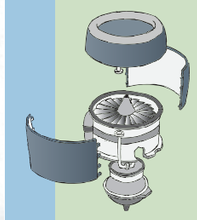

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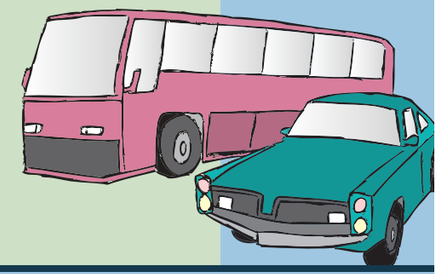

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Editorial

"Any sufficiently advanced technology is indistinguishable from magic" – Arthur C. Clarke, Clarke's third law.

Sometimes the inventions around us really do feel like magic. This is largely due to the wonders of engineering. A hundred years ago the marvel of the day was the telephone, now science is creating things that could be straight out of Hogwarts: invisibility cloaks and mind readers. This term's edition of *Bang!* explores some of this magic and celebrates the innovation of engineering.

Sophisticated engineering is being carried out all around us, especially here in Oxford. Our stunning centrefold shows the dramatic changes in Oxford architecture between the construction of All Souls in 1438 and the opening of the biochemistry building some 570 years later. As this shows, the magic for one age is the next generation's norm: cutting-edge engineering research is a glimpse of the future. In this issue we will show you some of this future, from artificial photosynthesis to electric cars. These innovations may seem magical now, but will be unremarkable in 50 years' time. And it's not all about completely new ideas; in engineering, you can teach old dogs new tricks: read our article on ceramics to see how this ancient technology has futuristic uses.

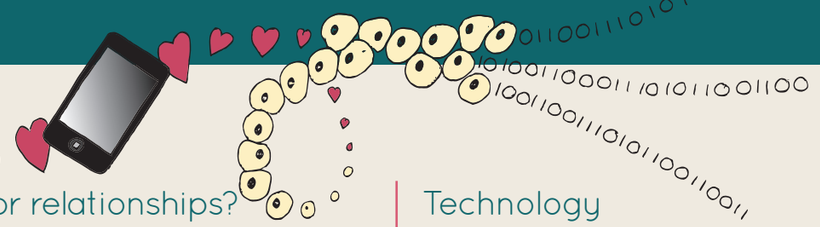
Engineering does not stay within defined subject boundaries, it affects all areas of life. Engineers aren't just building bridges and cars: biomedical engineers are working to develop radical new ways of treating disease and solving human health problems. You can read our interview with Dr Eleanor Stride about the innovative work she is doing with microbubbles to deliver drugs more effectively. It's engineering, but without the cogs and engines.

As well as focussing on engineering, we have articles about all the science you didn't know you didn't know, from why we have nails to how we develop morals. Science really does answer all the questions about the world around you that you may never have thought to ask!

So I hope you enjoy this edition of *Bang!*, giving you a peek at the world that engineering is creating while also finding out more about the world we live in now.

Iona Twaddell
Editor

News



Is texting good or bad for relationships?

New research conducted at Brigham Young University suggests that constant use of technology within committed relationships can lead to problems for couples, an issue particularly relevant for today's students!

Lori Schade and Jonathan Sandberg studied 276 young adults in relationships around the US. 82% of participants traded text messages with their partner multiple times a day. However, while expressing affection by text enhances a relationship, the researchers found that texting in other contexts was a negative indicator of relationship quality.

For example, many of the couples used texting for "relationship maintenance". While this is usually good relationship practice, when done via text it was often found to make things worse. Sandberg argued: "There is a narrowness with texting and you don't get to see the breadth of a person you need to see." Indeed, for female participants, using text messages to apologise, work out differences or make decisions could be associated with lower relationship quality. For men, lower relationship quality may result from too frequent texting. The researchers suggested that this could be the result of men disconnecting from conversation, replacing conversation with texting.

Increasing sophistication and reliance on communication technology has led to a change in the way relationships are maintained. While not necessarily negative, this study suggests that we should monitor how technology integrates into our social lives.

Next-Generation Sequencing in Advanced Cancers

This week, the Piper Cancer Center Clinical Trials at Scottsdale Healthcare (VGPCC), Arizona, have met with success in the use of new anticancer agents that target a single cell surface receptor, amplified gene product, or mutated gene when treating advanced cancers. Although patients' tumours were still found to eventually worsen following treatment, the research indicates that identifying a larger number of targetable vulnerabilities in cancerous tumours could provide a viable therapy.

In the study, researchers used next-generation sequencing technologies (NGS) to identify genomic events and expression changes in patients with advanced cancer. Results indicated that such methods were able to identify several targets on patients' tumours. In one case the exploitation of a discovered target and pathway identified by NGS resulted in a short-lived tumour reduction with significant reduction in associated pain.

The study also described a number of challenges involved in the designing of treatment plans based on NGS information. Examples of difficulties include communication of NGS results to out-of-state participants and their oncologists, as well as consideration of the ethics of handling fresh biopsy samples. However, while this pilot study was a slower process than anticipated, the team demonstrated the feasibility of using NGS in cancer patients for a brighter prognosis. This presents an exciting opportunity in the ongoing quest for novel cancer therapies.

Technology could be our downfall

A multidisciplinary team of researchers at the University of Oxford's Future of Humanity Institute has been set up to investigate the greatest global threats to the existence of the human race. The team has argued in a recent paper entitled "Existential Risk as a Global Priority" that policymakers must take species-obliterating risks seriously. The greatest of these risks, they suggest, is our own technological advancement.

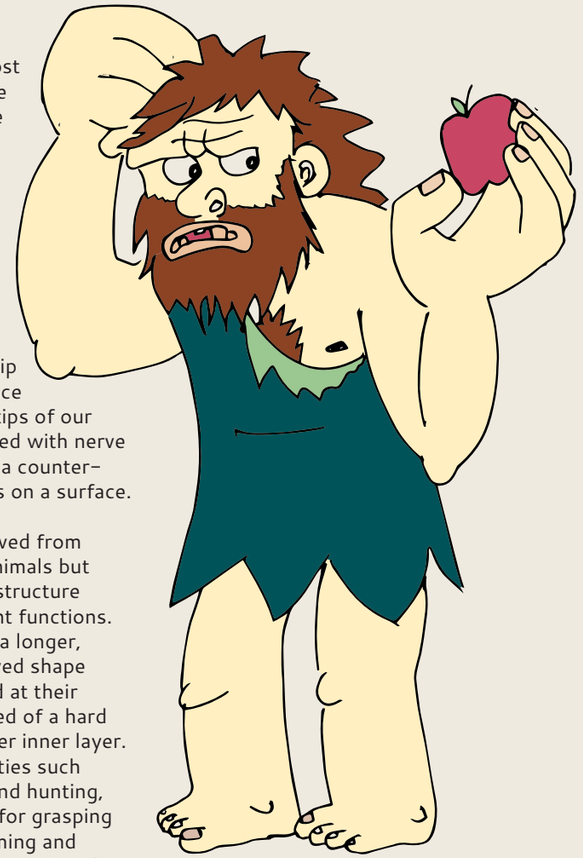
Dr Bostrom, Director of the Institute, argues that while pandemics and natural disasters might cause catastrophic loss of life, as a species we have already outlasted many thousands of years of disease, famine, predators, warfare and environmental change. Bostrom argues that even in a nuclear war, enough individuals would survive to continue the species. The team suggests that it is in fact new technologies that threaten our future most, since these are "threats we have no track record of surviving" according to Bostrom. They argue that "advances" in synthetic biology, nanotechnology and machine intelligence mask unforeseen consequences. While offering medical benefits, these areas could prove highly destructive if used for warfare. Similarly, exponential increase in the power of computing and artificial intelligence is becoming less controllable.

The Oxford group argue that while humans may well overcome natural disasters, our own technological advances could feasibly result in our extinction within a century. Therefore policymakers should perhaps take more notice of the risk to humanity that they can directly control, applying more stringent safety standards to research results that we are too often willing to blindly celebrate.

Patrick Roberts is a 1st year DPhil student in Archaeological Sciences at St Hugh's College.

Art by Iona Richards.

Bang! Explains: Nails



Have you ever wondered why we have nails? For fingernails, the answer seems obvious: they help us to perform manual tasks like gripping, tearing, and scratching. The same doesn't seem to be true of toenails, however. The explanations for how we came to have nails in the first place, and why we use our toenails less than our fingernails, lie in evolution.

You may not think there is much to your nails, but they actually have an intricate structure. Underneath the hard part of the nail, known as the nail plate, is the nail matrix, a tissue which contains nerves, lymph fluid and capillaries. It is visible as the white crescent (usually most prominent on the thumb) at the base of the nail. The nail plate is formed as new cells originate from the nail matrix and push old cells forward, compressing and flattening them. In this process the cells produce the tough protein keratin (the same protein that horns, hooves and hair are made from) and become translucent, which makes the capillaries in the nail bed visible, giving the nail its pinkish colour. The layers of dead compacted cells and keratin give the nail both strength and flexibility, while the underlying bone determines the shape of the nail. The tip of the nail is white because it is not attached to the nail bed and, like the rest of the nail, does not contain pigment.

The fact that toenails are used less than fingernails is reflected in their turnover rates. Fingernails grow at about 3mm a month and take three to six months to regrow completely, whereas toenails can grow up to four times more slowly and take as much as 18 months to regrow. Considering this, it is not surprising that it took the Indian Shridhar Chillal nearly 50 years to grow the longest fingernail ever, at 4 feet 3 inches!

Previously, the primary purpose of nails was thought to be to protect the fingertips and tips of the toes from injury. However, this theory has been dismissed by most scientists since people who permanently lose a fingernail or toenail find that the nail bed becomes tougher and provides perfectly adequate protection. Perhaps a more convincing theory is that we have nails because they improve the strength of our grip on objects, and enhance the sensitivity of the tips of our digits, which are packed with nerve endings, by providing a counter-force when they press on a surface.

Nails in primates evolved from the claws of earlier animals but have a very different structure and therefore different functions. Claws generally have a longer, thinner and more curved shape than nails, are pointed at their ends and are composed of a hard outer layer and a softer inner layer. While claws aid activities such as climbing, digging and hunting, nails are more useful for grasping small branches, grooming and manipulating fruit. Conventional wisdom held that nails first evolved approximately 30 million years ago with an increase in primate body size, since the flatter, broader tips to the fingers and toes that came with nails enabled larger primates to scramble through tree canopies by improving their grip on branches. However, recent research at the University of Florida revealed that nails have existed in primates for a much longer period. Palaeontologists Stephen Chester and Jonathan Bloch, who performed the study, analysed 55.8-million-year-old remains of *Teilhardina brandti*, an extinct six inch lemur-like primate, and discovered small nails on its hands and feet that helped it to move through the trees with greater agility.

So, if nails are useful mainly for gripping and manipulating objects, why do humans have toenails? The answer is that primates use both their fingers and toes for very similar tasks, such as picking up fruit and holding onto branches when climbing. As humans have evolved to walk upright, toenails have ceased to function as tools in the same way. This is why toes have become shorter than fingers and toenails smaller than fingernails. Toenails are therefore just an evolutionary leftover from the past that we may eventually lose altogether!

Lloyd Chapman is a final year DPhil student in Mathematical Biology at Lady Margaret Hall.

Art by Kate Williamson.

It's a Wild World

Animal ingenuity continues to amaze scientists

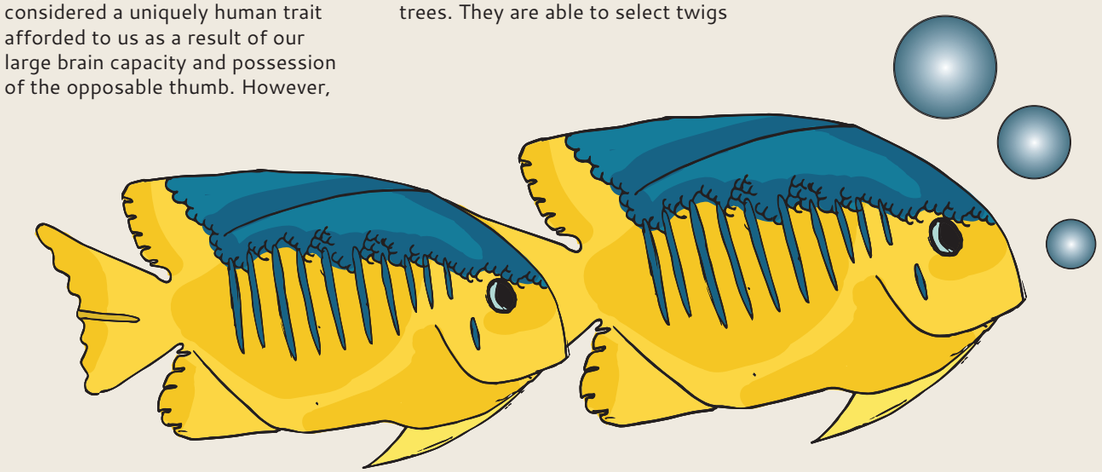
We describe our world as 'unnatural', as if our amazing cognitive abilities have somehow separated us from nature, allowing us to solve problems of survival in ways that no other organisms can. We have outfoxed Mother Nature with our ingenuity and believe that the world we have created for ourselves is no longer governed by her laws. If she had it her way there would be fewer than a billion people on the planet, and so she throws obstacles in our way in an effort to impede our progress and curtail our exponential population growth. But we believe ourselves to be cleverer than she; we use our opposable thumbs to create tools to negotiate her battlefields – building shelters to weather her storms, farming land to survive her famines and developing medicines to combat her diseases. But if we are as detached from nature as we believe ourselves to be, then why does nearly every strategy that we have developed in order to allow us to survive in our hostile world occur in a multitude of other organisms? Mankind should be proud of its achievements, but we must also recognise that we are not alone in our ingenuity and that we do not, and cannot, live outside of the natural world.

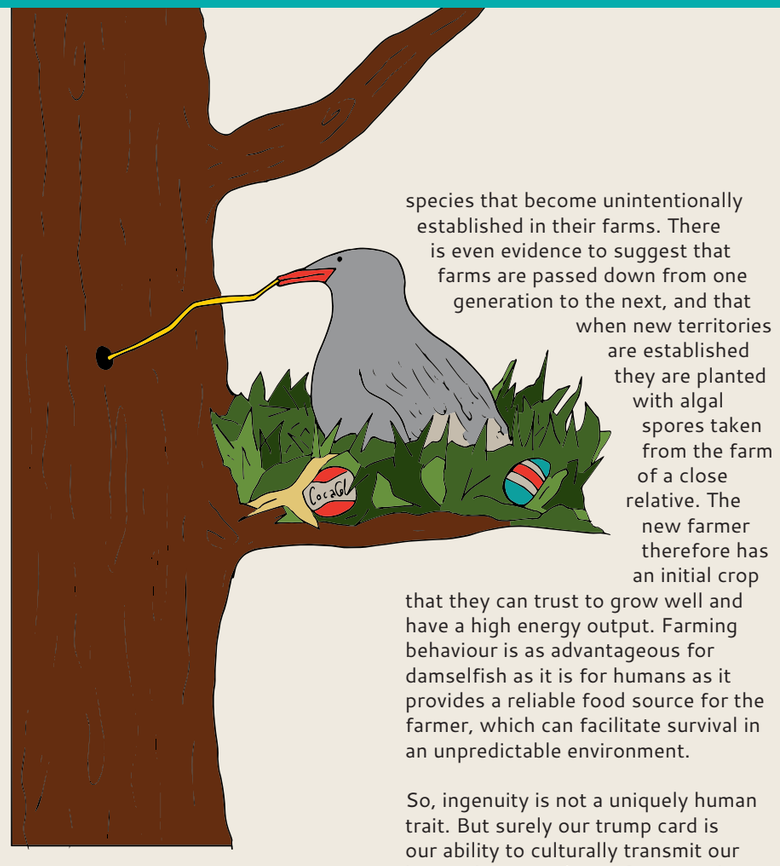
The ability to use tools was once considered a uniquely human trait afforded to us as a result of our large brain capacity and possession of the opposable thumb. However, observations of chimpanzees have shown that we are not alone in our ingenuity and that chimps' ability to alter the environment to suit their own needs is almost as impressive as our own. After Jane Goodall's discovery of tool use in chimpanzees in the 1960s, Louis Leakey notoriously wrote: "Now we must redefine tool, redefine Man, or accept chimpanzees as human." It has since transpired that the use of external objects to manipulate an individual's environment is widespread throughout the animal kingdom; from the humble sea urchin, which covers itself in small pebbles as protection against predators, right through to the majestic dolphin, which uses sponges as foraging aids. Tool-using behaviour allows animals to alter their surroundings in ways that would otherwise not be possible, meaning that they can live in inhospitable environments without coming to harm.

Perhaps the best example of tool use in occurs in the New Caledonian crow, which feeds off beetle larvae living in the bark of the island's Bancoulier trees. They are able to select twigs of the correct length and thickness and then manipulate them into an appropriate shape so that they can be used to extract their food from the interior of the tree. The New Caledonian crow's use of sticks has allowed it to expand its habitable range, as the individual will always be able to forage a meal regardless of the typical tree size of an area.

> "The bowerbird lacks no imagination, and amazingly complex structures have even been found to incorporate Coca-Cola bottle caps"

But surely our ability to use tools to build elaborate homes, and even have a penchant for interior design, must make us superior – again, it transpires that we are not the only ones. The male bowerbird is not much to look at, but he makes up for this by being an incredibly house-proud homeowner; the fancier his abode the more likely he is to attract a mate. He spends the majority of his time foraging for materials from which to build his palatial nest. The bowerbird lacks no imagination, and amazingly complex structures have even been found to incorporate such items as Coca-Cola bottle caps. The degree of animals' understanding of 'folk physics', the extent to which they comprehend the physical properties of the materials and tools that they are using, is currently unclear, but bowerbirds will often construct top-heavy nests supported by pillar-like structures, which surely indicates at least a rudimentary appreciation of the laws of physics. All male bowerbirds not only have the capability of building sophisticated homes for themselves and their families, but they also understand why they have to build them in a particular way – something that the average human male would almost certainly struggle with.





Cultivation of land has made the expansion of the human population possible, but we are not alone in our agricultural endeavours. Damselfish have evolved to aggressively defend their territories in which they grow their preferred algal food source. They grow algae in monoculture by actively excluding other unpalatable species that become unintentionally established in their farms. There is even evidence to suggest that farms are passed down from one generation to the next, and that when new territories are established they are planted with algal spores taken from the farm of a close relative. The new farmer therefore has an initial crop that they can trust to grow well and have a high energy output. Farming behaviour is as advantageous for damselfish as it is for humans as it provides a reliable food source for the farmer, which can facilitate survival in an unpredictable environment.

So, ingenuity is not a uniquely human trait. But surely our trump card is our ability to culturally transmit our innovations to others of our species, so that they, too, may outmanoeuvre Mother Nature. There are three elements that are common to all proposed definitions of 'culture': that behaviours are transmitted through learning and not inherited genetically, that behaviours are shared amongst many members of a social group but vary between members of different social groups, and that there is a degree of permanence in the behaviour over time. A fourth caveat that is used in many, but not all, definitions is that reproduction has to be able to occur between individuals of distinct cultural groupings, and that ecological differences in their respective habitats have to be such that the differences in behaviour cannot be considered to be adaptive.

A mounting body of evidence suggests that there are a large number of other animals which could be considered to be 'cultural'. Adjacent orangutan communities are behaviourally more similar to each other than they are to further-away communities – in humans the cultures of Spain and Portugal are much more similar than either is to the cultures of the Far East. Differences in orangutan behaviour have been shown to be cultural and non-adaptive as reproduction can occur between all of the sub-communities within the population and there is consistency in the environment in which all of these communities exist. Yet again, a peculiarity that we often consider to be a defining factor of humanity has been found to be pervasive throughout the animal kingdom.

While it is important not to undermine the ingenuity of man, it is equally important for us to shed our arrogance as a species and acknowledge our place within the natural world. None of our innovations and behaviours can be considered to be 'unnatural' – they have all emerged from a long evolutionary history of our ancestors interacting with their environment. If we can reconcile ourselves with the fact that we really are nothing out of the ordinary, and that Mother Nature really is in control, then maybe we can put the brakes on overexploitation and start to treat our planet with the respect it deserves.

> "While it is important not to undermine the ingenuity of man, it is equally important for us to shed our arrogance"

Max Bodmer is a 3rd year Biological Sciences student at St Peter's College.

Art by Nikolas Dion Susanto & April Hills.

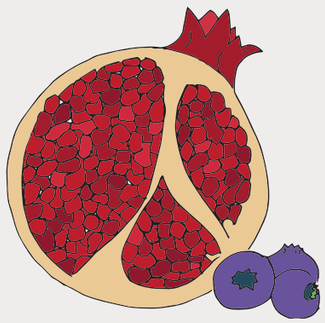
Superfoods

Too good to be true?

Lower blood pressure, fewer wrinkles and reduced risk of cancer. These are just a few of the extensive claims made about the health benefits of so-called "superfoods" such as blueberries, pomegranates and kale. Sounds great, but are they too good to be true? Many of these previously little-known foods have enjoyed a huge rise in popularity over the last couple of decades, ever since the term "superfood" was coined.

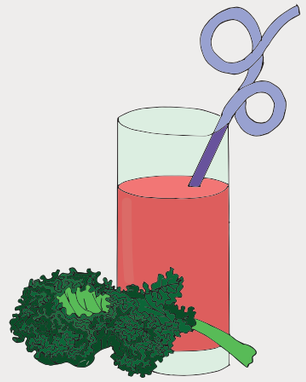
Blueberries are one of the most well-known superfoods, something that has undoubtedly caused a significant boost in UK sales of the fruit in recent years. The fruits are alleged to combat cancer and inflammation due to high levels of potent antioxidants. Antioxidant is a frequently used buzzword, but it is rarely explained what these molecules actually do. Put simply, their supposed benefits result from the way they interact with certain reactions within cells.

Redox reactions involve transfer of electrons between molecules and are essential for normal cell function. They also generate reactive oxygen species – molecules with unstable unpaired electrons – that react voraciously, swiping electrons from biological molecules such as lipids or DNA. This sets off a chain reaction that can result in damage like DNA mutations or enzyme denaturation. Antioxidants are reducing agents that remove these dangerous molecules by donating electrons. So what about those claims about antioxidants helping to fight cancer? Several studies have shown that antioxidants are in fact capable of inhibiting proliferation of tumour cells. In a 2005 paper, Akoh and colleagues report an experiment where anthocyanins (a family of red-blue pigments that act as antioxidants) were added to human colon cancer cells, resulting in a reduction in cell proliferation of more than 50% compared to when such antioxidants were absent.



Another study, this time on the effects of pomegranate juice, also showed positive results. Thirteen healthy volunteers supplemented their normal diets with 50ml pomegranate juice a day for two weeks. Subsequent analysis of blood samples showed a slight reduction in markers of reactive oxygen species activity that have been linked to development of atherosclerosis (literally "hardening of the arteries" – a condition intimately linked with heart disease and stroke). Similarly, a reduction in atherosclerosis was also seen in mice fed pomegranate juice.

Overall the results of these experiments sound positive, but they highlight many problems that arise when applying such evidence to the question of health benefits of certain foods. Firstly, many studies are performed on animals or *in vitro* cell cultures. While it is more practical to use a model system, it is entirely wrong to assume that an effect observed in such a model is applicable to whole, living humans. Humans are much larger and more complex than, for example, rodents. Such assumptions also disregard pharmacokinetics, which governs how an external substance acts within the body. For example, although antioxidants may inhibit proliferation of cancer cells, it is irrelevant if they are metabolised rapidly and never reach the tumour. Studies often measure the effects of a substance at very high concentrations or in quantities that are unlikely to be achievable as part of a balanced diet. Finally, there have been few studies to investigate long-term consequences of high concentrations of these compounds.



> "Unfortunately, a gallon of pumpkin juice will never 'offset' that bacon sandwich"

As a result, food standards agencies around the world have banned companies from using such unsubstantiated, misleading claims in their marketing. You might ask whether it really matters if it encourages people to eat more healthily. Like all things, it's about balance and common sense. Often, the way ingredients are processed can change everything. While green tea may be packed with antioxidants, if it is brewed with heaps of sugar, is it still healthy overall? Unfortunately, a gallon of pumpkin juice will never 'offset' that bacon sandwich. "Superfoods" may have real health benefits; they might even be harmful. Just as with any novel innovation in medical science, without rigorous evidence it is impossible to be sure, no matter how appealing its claims may sound.

Louise Thompson is a 3rd year Biomedical Sciences student at St Hugh's College.

Art by Kate Williamson.

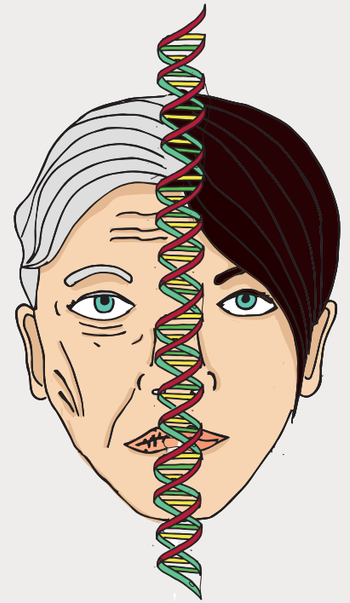
We're Gonna Live Forever?

The trade-off between ageing and cancer

Since time immemorial, mankind has been posing the question of whether immortality is possible. Although Nicolas Flamel didn't quite manage to share his secrets of the Philosopher's Stone with the rest of the world, scientists have made some incredible progress in understanding the process of ageing nearly 600 years after the alchemist's death. In a recent scientific review called "Hallmarks of Ageing" published in the journal *Cell*, Spanish researchers describe features that contribute to the ageing process.

It is striking to see that so many of the hallmarks described have a link to cancer. One of these hallmarks is genomic instability, which is found in cancer and occurs when DNA is damaged. This can lead to an abnormal chromosome number, changes in the base sequence of DNA and even translocations, where parts of two chromosomes are swapped around. Genomic instability is also seen in ageing, and there is some evidence that reinforcing genome stability decreases the speed of the ageing process.

Closely linked to genomic instability is telomere attrition. Telomeres can be described as protective bits of repetitive DNA at the ends of chromosomes. Every time your cells replicate, the ends of your chromosomes are chopped off. Your telomeres are there to stop them from becoming too short. However, over time, your telomeres can disappear altogether. When this happens, cells can no longer divide and replenish tissues. If this occurs on a large scale across many different tissues of the body, the result is ageing. Interestingly, cancer cells produce an enzyme which builds up the cell's telomeres after each division, so that they don't age. As a result, cancer cells are biologically immortal. As long as you provide them with nutrients, they will continue to divide forever, defying the ageing process.



Another hallmark linked to cancer is epigenetic alterations. These are changes to the epigenome, a system of chemical marks lying on top of the DNA that tell the cell which genes to switch on and off. When observing the epigenome of ageing cells, a specific marker increases, namely H4K16ac, which activates repair processes. In cancer, the opposite occurs and H4K16ac decreases, suggesting a possible mechanism and a link between ageing and cancer.

> "This latest research seems to imply that it is not possible, or is extremely difficult, to have cancer protection without having ageing as a side effect"

A normal, young and healthy cell faces many dangers in its day-to-day living. When these dangers result in DNA damage, the cell faces several possible fates: it can carry out a repair, it can kill itself, it can become inactive, or it can carry on going about its job despite being damaged. If the cell repairs the damage or if it undergoes programmed cell death (apoptosis), then we remain healthy completely oblivious to the damage our DNA undergoes (unless many cells die, as in radiation poisoning!). If the cell were to remain active despite the damage, genomic instability can result, which may lead to cancer. However, if the cell becomes inactive, it prevents further insults to the DNA from occurring that can send the cell on a pathway to cancer. It therefore seems a good defence mechanism to inactivate itself, since the alternative is the possibility of cancer.

At the heart of the phenomenon that links cell ageing and cancer is a protein called p53, which is known to be inactivated in over half of all tumours. Studies show that removing p53 from mice leads to early cancer, whereas studies that increase p53 in mice lead to cancer protection but early ageing. Other studies have shown that genetically modifying mice to express more p53 than usual, as well as another protein called ARF, leads to both cancer protection as well as an extended lifespan.

This latest research seems to imply that it is not possible, or at least it is extremely difficult, to have cancer protection without having ageing as a side effect. It seems that we evolved a way to remain healthy and relatively cancer-free in our youth, at the cost of driving cells towards ageing. But where does the balance lie? Can we stop ageing from occurring without developing cancer, or are the two just different sides of the same coin? We don't know yet for sure. Certainly, this new research can give us some answers, but the time-old question persists: is immortality possible?

Marco Narajos is a 1st year Medical student at Christ Church College.

Art by Nikolas Dion Susanto.

A Life Without Pain

Lack of pain: a curse, not a blessing

The sensation of pain is one which many amongst us wish we never had to feel, from everyday irritations like paper cuts to the agony of appendicitis. However, pain is in fact a protective mechanism, hence vital to human survival; a life without pain is very difficult indeed. A few unlucky people never have the sensation of pain. They suffer from CIPA – congenital insensitivity to pain with anhidrosis (reduced or absent ability to sweat). This genetic disease is so rare that only 300 cases have been documented worldwide.

> "CIPA patients are a danger to themselves as they are unable to tell when they are injured"

CIPA causes damage to nociceptive neurons, rendering patients unable to sense pain or temperature. 'Nociception' is the perception of unpleasant or painful stimuli. In CIPA patients, transmission of these signals is disabled. This means that CIPA patients are a danger to themselves as they are unable to tell when they are injured or hurting themselves. This is a particular issue for young children, who lack an adult's understanding of behaviours and situations to avoid, and don't know when to check themselves for damage. Several symptoms are found in the majority of patients due to self-inflicted injuries as toddlers. Corneal abrasions (cuts to the outer layer of the eye) are extremely common as patients scratch their eyes in their sleep. Severely mutilated lips, tongues, and fingers are also common as sufferers chew them incessantly during teething. Left unattended, a teething child with CIPA may chew through the entirety of their tongue or fingers to the point where amputation is necessary. Consequently, doctors may resort to extreme measures such as filing or removing teeth. A child with CIPA may rip out stitches, and even gnaw through plaster casts in order to chew on fingers. Also, CIPA patients do not feel hunger, as this sensation is transmitted via pain neurons, so it can be very difficult to persuade them to eat as infants. Nor can they sense when they need the toilet, so in later life means such as timer alarms are required to remind themselves to go.

Internal pain cannot be felt either; internal injuries or events such as heart attacks are often not detected until a very late stage, so are more likely to be deadly to a CIPA patient. Undetected broken bones may be disabling or fatal if they cause internal bleeding. Repeated bone damage may result in osteomyelitis (chronic bone infections), or Charcot joints (destruction of bone and tissue around joints), which are frequent causes of mortality in CIPA patients. CIPA patients may also place stress on their bodies by unknowingly sitting in strenuous positions, and are prone to early onset arthritis and joint damage, often needing physiotherapy. Anhidrosis vastly increases the risk of potentially deadly hyperpyrexia (extremely high fevers) and febrile (feverish) seizures, causing around half of CIPA patients to die before the age of three, and many others to be left with learning disabilities. Those that survive infancy rarely live past 25, due to their developing undetected injuries and infections.

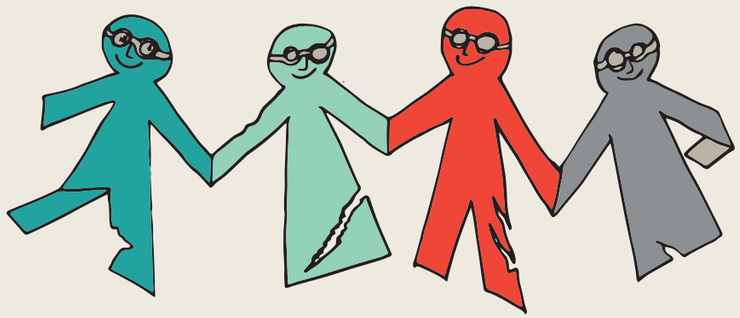
The mutations that cause CIPA are thought to be to the TRKa gene on chromosome 1. This gene codes for the receptor tyrosine kinase, a membrane bound receptor protein that binds the protein nerve growth factor (NGF). NGF is a neurotrophin, a type of protein responsible for the growth and function of neurons. NGF supports cell growth and survival. When NGF binds to the TRKa receptor this allows cell differentiation and survival by autophosphorylation. The mutations in TRKa in CIPA patients result in a loss of function of the receptor tyrosine kinase, causing the neurons to die prematurely. TRKa is present in nociceptive (pain-sensitive) neurons and thermoreceptive (temperature-sensitive) neurons, but not in other neurons that signal touch, since they express a different gene, TRKc, instead. Therefore, the TRKa mutation in CIPA results in a loss of pain and temperature perception but not of touch perception. The anhidrosis is caused by the death of neurons that innervate the sweat glands.

CIPA first came to media attention in 2005 with the documentary "A life without pain", following the life of the then three-year-old CIPA patient Gabby Gingras in Minnesota, USA, whose family had undergone a long struggle to find a diagnosis for their daughter, due in part to the disease's obscurity. Gabby showed many of the classic symptoms of CIPA. She had damaged her eyes so severely that they were sewn shut, however she ripped out her stitches and her left eye had to be removed when she was a toddler. At three she wore protective goggles at all times. Gabby had her milk teeth removed to prevent mutilation; her father Steve Gingras recalled: "She would chew on her tongue like it was bubble gum." Now aged 12, she is blind and has no teeth, having broken them all off as they came through after her milk teeth were removed. Unlike many CIPA patients, she has no learning disabilities, having escaped brain damage from anhidrosis, and leads a fairly normal life aside from the rigorous regime of daily checks for injuries.

> "The growing understanding of the genetic causes creates hope for a cure somewhere down the line"

Following the documentary, families of other children suffering with the disease began to share their strikingly similar stories. In the first months CIPA patients are "perfect babies" as described by the mother of 11-year-old patient Roberto Salazar: he "never cried, and would sleep 23 out of 24 hours a day." Roberto began showing signs of his disease at three months when he refused to eat. At eight months Roberto had a feeding tube inserted, which he still uses now. His anhidrosis meant he suffered frequent heat strokes that have left him with learning disabilities. Another young sufferer who received a great deal of media attention is 13-year-old Ashlyn Blocker, who, like Gabby, has not developed any intellectual disability, but had severe corneal abrasions, as well as many burns and injuries. Upon realising that they were not alone in coping with Ashlyn's disease, her family set up "Camp Painless but Hopeful", an American summer camp and support group for CIPA sufferers and their families.

There is no cure to enable a CIPA patient to feel pain. Treatments are predominantly designed with the aim of preventing injuries, or enabling their early detection. Methods include the use of protective clothing such as goggles to prevent eye scratching, acrylic mouth guards to prevent self-mutilation through biting, and restraints to prevent infants from augmenting their injuries. Routinely saying "ow" after a fall, and recognising that blood corresponds to an injury, are encouraged in order to increase the likelihood that injuries are discovered. Finding safe forms of exercise for CIPA patients is a challenge; however, swimming is excellent as it places little stress on the joints and the cool water prevents overheating.



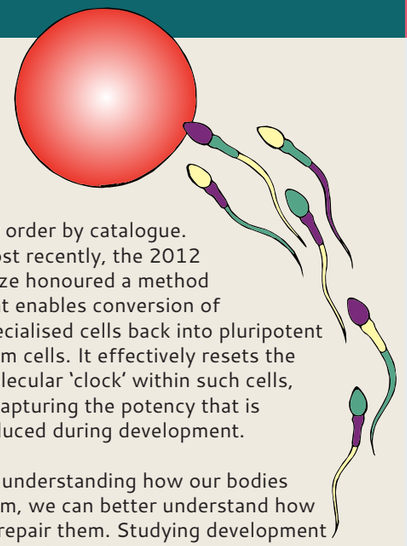
While CIPA patients still face huge daily challenges, advances in preventative treatment, and the increased capacity for sufferer's families to unite and share the load means that they are able to look forward to a brighter future. As Gabby's mother Trish states: "The media has been our best friend over the years... and our second best friend is Facebook!" The growing understanding of the genetic causes behind the disease also allows genetic counselling to be used in future, and creates hope for a cure somewhere down the line.

Zoe Evans is a 2nd year Biomedical Sciences student at Lady Margaret Hall.

Art by India Stephenson, Sophia Malandraki-Miller & Natalia Filvarova.

From One to Many

The study of embryology affects us all



Many years ago, you were a single cell, created when egg and sperm fused. Embryology is the area of science focused on understanding how that one cell became the trillions of cells that today form your body, a system capable of accomplishing complex tasks, such as reading this article! The study of embryonic development has yielded some spectacular discoveries, but also sparked its fair share of controversy.

That controversy often involves the use of embryos in stem cell research. The embryonic stem cells used are collected from an embryo four days after fertilisation, when it is a mere blob-like mass consisting of a few hundred cells. At this early stage, the cells are undifferentiated, unspecialised precursors to the specified cells we recognise in our bodies, such as oxygen-carrying red blood cells or electrically excitable nerve cells. Embryonic stem cells are pluripotent, hence can mature into a variety of more specialised cells.

This critical importance of embryonic stem cells is what makes the political hurdles blocking embryonic stem cell research worth overcoming. Many significant discoveries have already been made. The history of this work can be observed via several Nobel Prizes in Physiology or Medicine. The 1935 Prize was awarded to those who discovered the most basic principles of development. The 2007 and 2012 prizes were awarded for work in methodology; the 2007 winners developed the technique to modify mouse genes via stem cell delivery, creating so-called knockout mice. Today there are tailor-made mice designed for studying hundreds of disorders or systems, available for order by catalogue. Most recently, the 2012 Prize honoured a method that enables conversion of specialised cells back into pluripotent stem cells. It effectively resets the molecular 'clock' within such cells, recapturing the potency that is reduced during development.

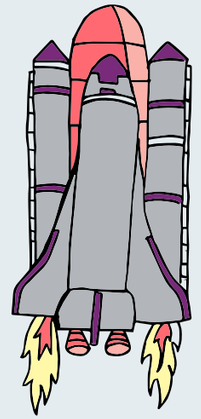
By understanding how our bodies form, we can better understand how to repair them. Studying development can feed into the path towards regenerative medicine – an extremely exciting prospect.

Sofia Hauck is a 1st year DPhil student in Zoology at St Cross College.

Art by Joy Aston.

Old Tools, New Tricks

The versatile uses of ceramics in modern engineering



Right now you're probably sat in a brick or concrete building, maybe holding a mug of coffee, and with porcelain fittings in your nearby kitchen or bathroom. These are obvious uses of traditional ceramics, but you may not have realised that your mobile phone, laptop and car also contain ceramic components. Ceramics may be among the oldest man-made materials but they are still used, mostly unchanged, all around us in our modern lives. So what exactly is a ceramic? A quick glance online informs us that a ceramic is an "inorganic and nonmetallic material that is formed by heating." The oldest known ceramic object is the Venus of Dolní Věstonice, a pottery figurine dated to around 27,000 BCE, and we know that ceramics became widely used around 6,000 BCE. In the past 200 years ceramic engineering has advanced well beyond the stereotypical uses of figurines and pots. Below are just three applications of some remarkable properties of modern ceramics.

Probably the most famous use of advanced ceramics is the tiles of the Space Shuttle. On re-entry to the earth's atmosphere, the friction between air and the spacecraft travelling at up to 18,000mph can create maximum temperatures of around 1,650°C, hot enough to melt steel. The structure of the shuttle can only withstand temperatures up to 173°C, so the surface is mostly covered in five-inch thick silica tiles, designed to protect the shuttle from the searing temperatures on re-entry. These tiles consist of silica fibres, but are 90% air. This trapped air insulates the space shuttle from these huge temperatures in the same way as a foam cup stops you burning your fingers (but not your mouth!) on hot tea.

> "It may surprise you to learn that ceramics are used for stopping bullets and bombs"

One point all traditional ceramics have in common is that they are brittle; if you drop them, they'll smash. So it may surprise you to learn that they are used for stopping bullets and bombs. Since the 1960s tanks have been made with layered plates of ceramic armour, known as Chobham tiles. The ceramics used, typically boron carbide, alumina, or silicon carbide, are extremely hard, and will bend and blunt missiles that can penetrate armour. The ceramic is still relatively brittle and will shatter, which creates ragged edges or a very hard abrasive powder. This makes the explosion act over a larger area than it is designed to, so the energy is less concentrated. Therefore it will not damage conventional metal armour underneath.

Modern developments are aiming to improve the toughness of Chobham armour, by creating composite tiles containing layers of rubber to absorb some impact energy. Also, the ceramic can be sandwiched between titanium plates to 'squash' cracks as they form. Research is ongoing into the use of nano-zirconia toughened alumina (nZTA) as armour. Zirconia is an interesting material, in that its crystal structure changes when a stress is applied to it. When a crack approaches a zirconia particle in nZTA, the stress causes a change in atomic crystal structure, which creates a small volume expansion. This applies a closing stress on the crack which stops it from growing further, toughening the ceramic. The higher the stress, the tougher the ceramic becomes.

The interior of a nuclear fusion reactor is probably the most inhospitable place on earth. Temperatures can reach 200 million °C in the centre of the plasma (the centre of the Sun is around 15 million °C), and materials are constantly bombarded with neutrons which knock atoms out of their normal positions. This makes them swell and deform, damaging the structure, and making some parts radioactive. Ceramics are now being developed to replace the metals which are normally used, as they are less susceptible to radiation damage. Silicon carbide composites are the most likely ceramic materials to be used in the next generations of experimental nuclear fusion reactors. Their atoms aren't displaced as easily as those in metals, they are mechanically stable at the operating temperatures of over 3,000°C at the reactor wall, and they don't create long-lived radioactive waste.

Hopefully you can now picture ceramics beyond your dinner plate and can appreciate some of the most advanced materials mankind has developed over millennia.

Alex Leide is a 3rd year Materials Sciences student at St Anne's College.

Art by Amber Barton.

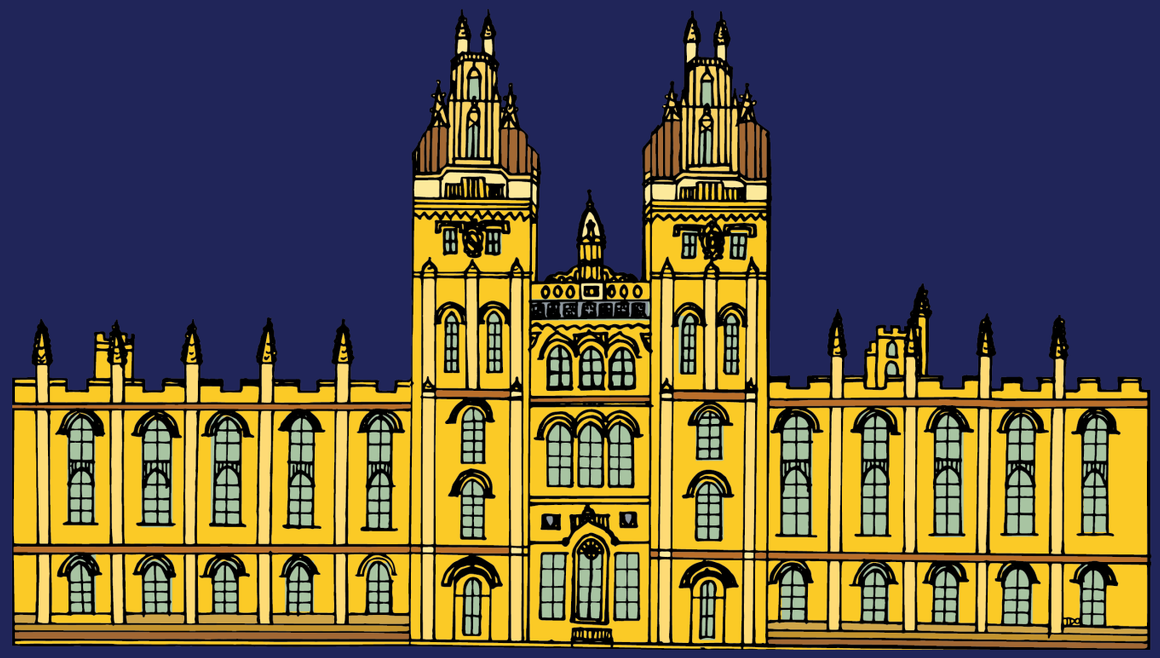
Oxford Architecture through the Ages

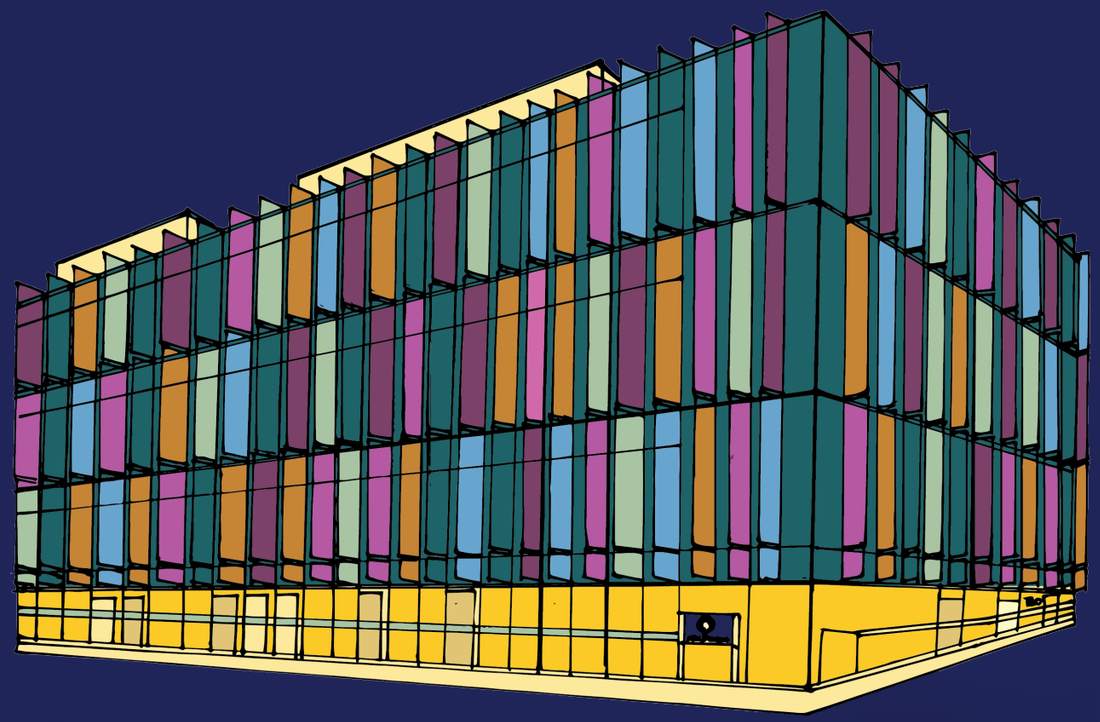
A tale of two landmark buildings

All Souls College (1438)

Construction of All Souls began centuries ago in 1438. Here, our image depicts the view from Radcliffe Square, which was built in the early 18th century. The exterior is gothic to match the rest of the College. The foundations and walls were built using ragstone from Headington.

Parts of the College have not changed in 500 years, though new parts have been added to keep up with the times, for example, 16th century battlements and Victorian windows. A blend of classic architectural styles gives All Souls its iconic appearance.





New Biochemistry Building (2008)

In stark contrast to All Souls' dramatic stone exterior, the New Biochemistry building is mainly made of glass, with coloured fins casting different patterns of light within the building.

New Biochemistry building's design epitomises an impression of space and transparency; indeed, researchers can be spotted at work through the glass panel walls.

Solar panels on the roof provide much of the building's electricity. The New Biochemistry building reveals how Oxford's character continues to develop through innovative and modern engineering.

Art by **Thao Do**

Fuel from the Sun

Artifical photosynthesis yields new engineering possibilities

In our current era of rapid worldwide industrialisation, alongside a growing awareness of our natural environment, the issue of how we should fuel civilisation has never been so important. The modern world has been running on coal, oil, and natural gas since the industrial revolution, but this practice is unsustainable. The amount of fossil fuels is limited, and the combustion of these fuels releases carbon dioxide, which is a direct contributor to global warming. Clearly, we need to replace fossil fuels with a renewable and clean alternative, and fortunately there is a constant source of energy shining down on us every day.

> "The amount of sunlight that reaches us in one hour is roughly the same as the global energy demand in one year."

Most of the fuels around us are actually forms of stored sunlight. The Sun provides energy for the photosynthesis process in plants to create carbohydrates. Even the fossil fuels we use today are the remains of organisms that lived millions of years ago, powered by sunlight. Might it be possible, then, to run our modern civilisation only on sunlight? The problem is not the quantity, since the energy from the Sun is more than enough for human needs; the amount of sunlight that reaches us in one hour is roughly the same as the global energy demand in one year. The problem is how to convert this into a form we can use.

One approach, solar cells, is already quite well developed. Current commercial modules can convert sunlight into electricity with 15–20% efficiency. Since solar cells cannot work at night, we need methods to store the electricity produced during daytime. Batteries can do the job, but they have low energy densities, especially when compared to chemical fuels. To illustrate the point, it takes a 50kg battery to store the same amount of energy that is in 1kg of gasoline. Therefore, as an alternative and complementary route to solar cells, we need to develop a method to store sunlight as fuels.

What fuel should we produce? Hydrogen is an attractive option, as it can be generated from water. With energy input, water can be split into hydrogen and oxygen. Using hydrogen, whether by combustion or by using a fuel cell, only leads to pure water as waste. Hydrogen can be considered as a totally clean fuel, but only on the condition that it was generated by a clean process. At present, most of industrial hydrogen is produced from natural gas, with carbon dioxide being generated as a by-product. The goal is therefore to replace this process with a solar-powered one using water as the source material. This process is called artificial photosynthesis.

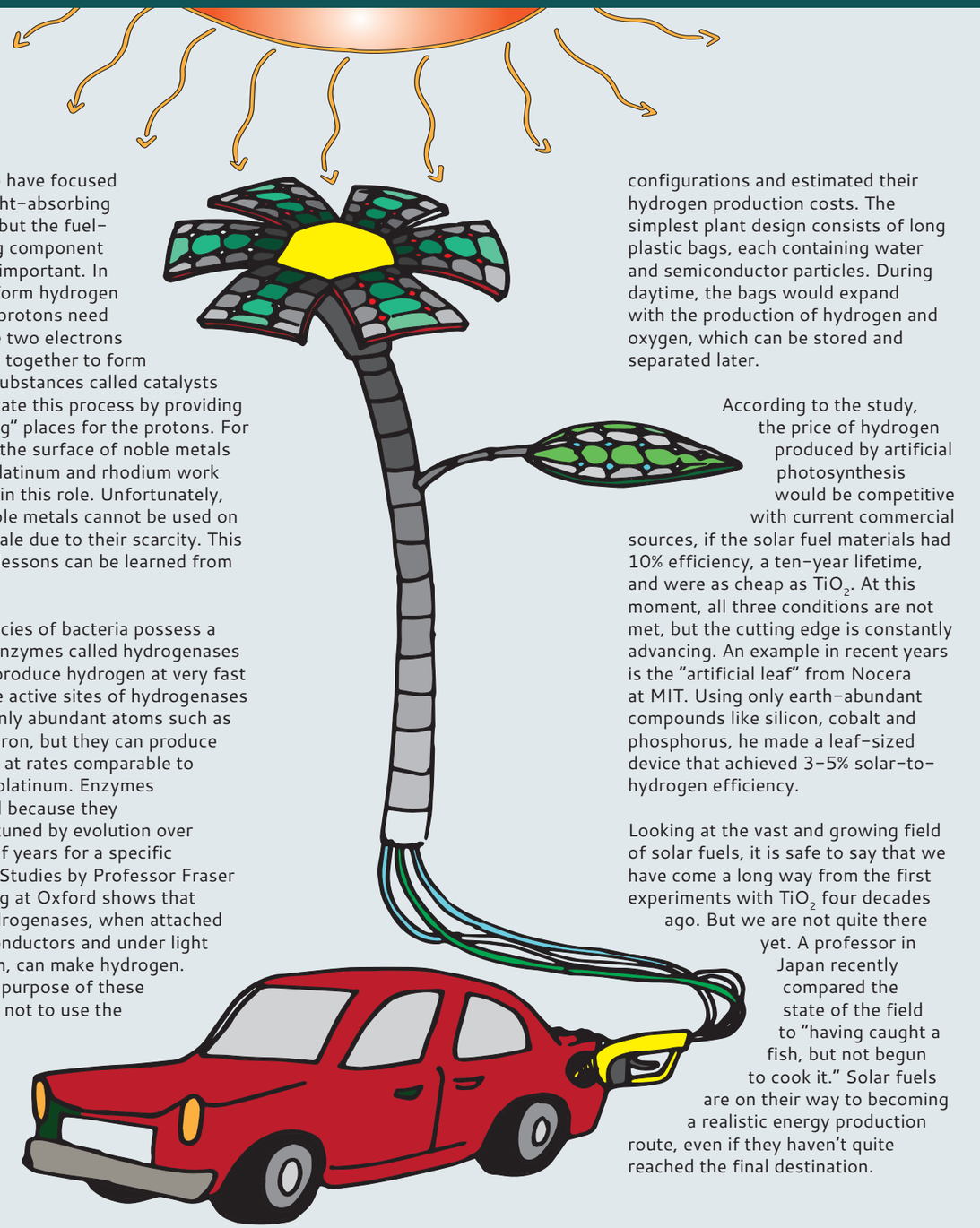
An artificial photosynthesis system is composed of two main components: the light-harvesting unit and the fuel-producing unit. The light harvester absorbs sunlight and creates high-energy electrons, which is passed on to the fuel producer in order to create hydrogen.

> "A professor in Japan recently compared the state of the field to 'having caught a fish, but not begun to cook it'"

Research in this field started 40 years ago, when a Japanese group demonstrated that light energy can actually be used to split water. They used titanium dioxide (TiO_2), a white-coloured semiconductor, as the light-harvesting material. While the work confirmed that solar water splitting is indeed possible, the amount of hydrogen generated was far too small for actual use. One reason is that TiO_2 can only absorb and utilise ultraviolet (UV) light, as is evident from its white colour (it reflects all visible light, making it look white). Since UV light comprises only a few percent of the whole solar spectrum, the solar-to-hydrogen efficiency of TiO_2 was low. For this process to become commercially viable, we therefore require new materials that can also absorb visible light.

In order to see what determines the range of light that a semiconductor can absorb, we must investigate its electronic structure. In a semiconductor, electrons can only exist at certain levels of energy, called bands. In order to jump up from a lower-energy band to a higher-energy band, an electron must absorb energy at least equal to the difference in band energies (called the band gap). The electron cannot receive energy smaller than the band gap because they cannot exist between bands. Thus, wide band gap materials (such as TiO_2) can only absorb light with high energy. Since sunlight comes in a spectrum, from UV (high energy) to visible and infrared (low energy), narrow band gap materials can absorb more of the solar spectrum.

One method to create a narrower band gap is to replace the oxygen atoms in the material with nitrogen or sulphur. These oxynitride, nitride, and sulphide materials come in a range of colours, from yellow to green to red, which shows that they can absorb visible light. Another method is to attach a dye molecule to the semiconductor, have the dye absorb visible light, then transfer the electron to the semiconductor.



So far we have focused on the light-absorbing material, but the fuel-producing component is just as important. In order to form hydrogen gas, two protons need to receive two electrons and come together to form a bond. Substances called catalysts can facilitate this process by providing "anchoring" places for the protons. For example, the surface of noble metals such as platinum and rhodium work very well in this role. Unfortunately, these noble metals cannot be used on a large scale due to their scarcity. This is where lessons can be learned from nature.

Many species of bacteria possess a class of enzymes called hydrogenases that can produce hydrogen at very fast rates. The active sites of hydrogenases contain only abundant atoms such as nickel or iron, but they can produce hydrogen at rates comparable to those of platinum. Enzymes work well because they are fine-tuned by evolution over millions of years for a specific reaction. Studies by Professor Fraser Armstrong at Oxford shows that these hydrogenases, when attached to semiconductors and under light irradiation, can make hydrogen. The main purpose of these studies is not to use the enzymes themselves on a large scale, because it takes some effort to purify even a small amount. Rather, it is to learn how these enzymes can produce hydrogen, so that synthetic chemists can produce analogues to replace noble metals. Again, man can learn a lot from nature.

So far, solar hydrogen production has been demonstrated at the laboratory scale. But is it possible to scale up? Will we see solar hydrogen factories in the near future? A recent study described several possible configurations and estimated their hydrogen production costs. The simplest plant design consists of long plastic bags, each containing water and semiconductor particles. During daytime, the bags would expand with the production of hydrogen and oxygen, which can be stored and separated later.

According to the study, the price of hydrogen produced by artificial photosynthesis would be competitive with current commercial sources, if the solar fuel materials had 10% efficiency, a ten-year lifetime, and were as cheap as TiO_2. At this moment, all three conditions are not met, but the cutting edge is constantly advancing. An example in recent years is the "artificial leaf" from Nocera at MIT. Using only earth-abundant compounds like silicon, cobalt and phosphorus, he made a leaf-sized device that achieved 3–5% solar-to-hydrogen efficiency.

Looking at the vast and growing field of solar fuels, it is safe to say that we have come a long way from the first experiments with TiO_2 four decades ago. But we are not quite there yet. A professor in Japan recently compared the state of the field to "having caught a fish, but not begun to cook it." Solar fuels are on their way to becoming a realistic energy production route, even if they haven't quite reached the final destination.

Bhavin Siritanaratkul is a 2nd year DPhil student in Inorganic Chemistry at New College.

Art by Sai Ulluri.

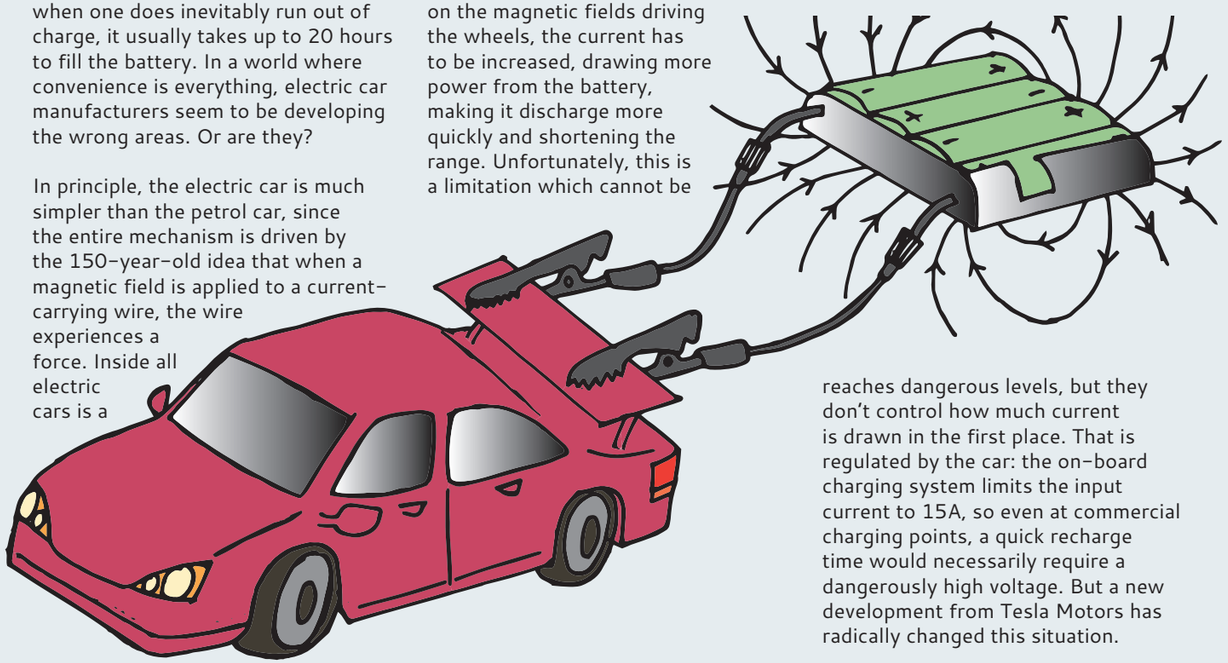
A Silent Road Trip

The developments and challenges for electric cars

Electric cars have come a long way since the very first Toyota Prius hybrid back in 1997. More and more companies seem to be ditching the internal combustion engine altogether, to create cars which run on just the power output of a battery pack. We have now seen electric sports cars hit the markets and, more recently, the electric Mercedes SLS has shown that even supercars can have their performance matched, if not surpassed, by their electric counterparts. This is a laudable achievement for an industry which has been subject to much criticism since its advent, but absolute performance has never been the biggest issue facing modern electric cars. It is still difficult, for example, to find an electric car whose range exceeds 200 miles, a distance which would be considered poor for a petrol engine vehicle, and, when one does inevitably run out of charge, it usually takes up to 20 hours to fill the battery. In a world where convenience is everything, electric car manufacturers seem to be developing the wrong areas. Or are they?

> "It is still difficult to find an electric car whose range exceeds 200 miles"

In principle, the electric car is much simpler than the petrol car, since the entire mechanism is driven by the 150-year-old idea that when a magnetic field is applied to a current-carrying wire, the wire experiences a force. Inside all electric cars is a battery pack; these drive current through wires which have essentially been coiled around the wheel axles. A strong magnet then acts on the wires, creating a force proportional to the amount of current. This force then causes the coil of wire to rotate, and since the coils are attached to the wheel axles, the wheels rotate and the car moves. By increasing the current, the force on the wires is increased, which makes the wheels move faster and the car accelerates. This is where the problem with electric car range lies: the magnets which drive the wheels can't be too strong, because they would then interfere with nearby electronics within the car. The size of the force on the wire, and therefore the speed of the car, depends on both the strength of the magnet and the size of the current. This means that, to compensate for the limitations on the magnetic fields driving the wheels, the current has to be increased, drawing more power from the battery, making it discharge more quickly and shortening the range. Unfortunately, this is a limitation which cannot be overcome altogether, although it can be pushed back. We have seen that, as the technology has improved over the past five years, the range of electric cars has increased fourfold.



One way this has been achieved is by increasing the amount of energy stored in the battery packs. However, this creates a second problem: recharge time. Most electric cars can be recharged at home – which at a glance seems simple, easy and convenient – but the 240V standard plug sockets are fitted with a 15A fuse. This limits the maximum power output to 3.6kW, so charging a 60kWh tank (barely enough to travel 200 miles) will take at least 17 hours. It's like trying to fill a swimming pool with a garden hose: it takes a long time because you are trying to do the job with equipment that is simply not designed to do it.

So why not remove the fuse? Fuses act as an emergency mechanism; they shut off the power when current reaches dangerous levels, but they don't control how much current is drawn in the first place. That is regulated by the car: the on-board charging system limits the input current to 15A, so even at commercial charging points, a quick recharge time would necessarily require a dangerously high voltage. But a new development from Tesla Motors has radically changed this situation.

Earlier this year, Elon Musk, CEO of Tesla Motors, unveiled plans for a network of 'supercharging stations' placed throughout the USA. These stations are able to bypass the charging system fitted to Tesla vehicles and significantly increase input power; as a result, they will charge electric vehicles to 80% capacity in around 30 minutes. They will also cost absolutely nothing to use. This may seem too good to be true, but there are already 31 of these stations up and running around the USA, and Musk has no plans to stop, claiming that: "You will be able to drive from Los Angeles to New York using the electric car by the end of 2013."

This is a notable improvement on the recharge times we are used to seeing, but half an hour is still much longer than it would take to fill a fuel tank. This issue can be easily resolved. Alongside the free recharge, superchargers also offer Tesla owners the option to pay $50-$65 for a 'battery swap'. This fairly self-explanatory scheme allows the customer to drive into a supercharger station and replace his or her empty battery pack with a fully charged one. The whole process takes less than half the time it takes to fill the tank of a petrol engine, and can be done without the driver even leaving the car. It is an elegantly simple solution which definitely brings electric cars into the same world of usability as their petrol rivals, and is a considerable step towards Musk's dream of transforming the electric car from a necessary inconvenience into a truly viable alternative.

> "It's like trying to fill a swimming pool with a garden hose"

So have we entered the future of electric cars? Probably not just yet. As with everything Musk announces, the superchargers are surrounded by impenetrable hype. People are very easily swept along by Musk's philanthropic charm; he has an air of 20th century ambition about him, and comes across as the embodiment of an era when anything seemed possible. For this reason, we are often guilty of forgetting to objectively analyse what he says, and when we do take a step back, the supercharging stations deliver a very clear message. People stare in wonder at a half-hour recharge time for a battery pack only capable of delivering half the range of an average fuel powered car, and the fact that this is considered a leap forward says a lot about how far electric car technology still has to come. Even the battery swap has unanswered questions, such as: how many battery packs will be fully charged and available at any one time? Are the supercharging stations likely to run out of batteries altogether? And when the batteries inevitably start to reach the end of their usable lives, how can the customer be sure the new battery will provide the 200 miles of range he or she paid for?

Musk and Tesla Motors have done a great amount of work on turning the electric car into a desirable product, and we shouldn't belittle this achievement. They have technology which is years, possibly even decades, ahead of their nearest competitor, but there is still a long way to go. The ideal electric car is still very much a technology of the future, but the superchargers do at least show us that they are possible, that change is coming, and that it comes in the form of Elon Musk.

A History of the Electric Car

1888: The first four-wheeled electric car is built

1897: Commercial electric cars are introduced into the New York taxi fleet

1900: Electric cars are the best-selling road vehicle: there are 4,000 on the road

1908: The Ford Model T is introduced as a cheaper petrol alternative

1920 Downfall: by the 1920s the electric car had almost vanished from the market

1996: GM created the EV1, an electric car that could go 80 miles before it needed recharging

2006: The Tesla roadster, a luxury electric car, is brought out

2010: The all-electric Nissan Leaf goes on sale. It can travel 100 miles per charge

Andrew Smith is a 2nd year Physics student at Somerville College.

Art by Sai Ulluri.

Bang! talks to...
Doctor Eleanor Stride

Doctor Eleanor Stride is a biomedical engineer, currently a fellow at St Catherine's College, Oxford. Her research involves finding novel strategies to transport drugs around the body using tiny bubbles.

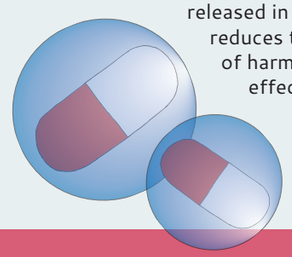
You work on engineering better drug delivery systems. Can you tell me more about your research?

So what we're really doing is vehicle design. We're developing means of getting the drug to target areas rather than just injecting it into the whole body, which is what's normally done now. We use microbubbles. These are tiny capsules that contain gas and the drug is in a shell around the gas surrounded by protective coating. This keeps the drug encapsulated in the bubble, and when the bubble reaches a target site we can release that drug. To release the drug, we are working on using ultrasound. It is a very similar technology to ultrasound scans but just at slightly higher energies. The ultrasound makes the bubbles expand and contract and eventually break open and release the drug.

> "We use microbubbles, tiny capsules that contain gas and the drug is in a shell around the gas"

What is wrong with current drug delivery methods?

Very powerful drugs, for example those used in cancer treatment, tend to be associated with very unpleasant side effects. Drugs are currently either be injected into the bloodstream or given in pill form so it goes through your entire body, all your cells are exposed to it. The bubbles localise the area that the drug is released in and that reduces the risk of harmful side effects.



Normally bubbles in the bloodstream are a bad thing, for example in the the bends. How are your bubbles different?

The reason bubbles are dangerous in the bends is that they can be quite large and they are formed in a completely uncontrolled manner so end up blocking blood vessels. So when we're manufacturing our microbubbles, we need to make sure they're very tiny, smaller than the smallest blood vessel in the human body, which is a couple of micrometres typically. And we also coat them with something to keep them that size, to stop them coalescing and causing a blockage.

How do you guide the bubbles to where they're meant to be?

They will just naturally float around the body, so they will probably pass a target site. It's possible to coat the bubbles with something that will make them sticky to a particular type of cell. For example, cancer cells express certain proteins that you can target, but that tends to be quite inefficient. You can actually use ultrasound: ultrasound is a wave, so you can use it to push the bubbles against the target site. Or one thing we're looking at is making the bubbles magnetic. So as well as having drug in the bubble coating you also have magnetic particles and that means that we can localise the bubbles using an externally applied magnetic field.

What kind of drugs would this be used for?

We're mainly looking at conventional chemotherapy drugs: they're already licensed for use, we're just trying to deliver them more effectively. But we're also looking at gene therapy. Gene therapy has got enormous potential for an awful lot of diseases but it's very difficult to get the DNA or your siRNA or whichever type of molecule to the right place. So we're hoping that the bubbles are going to be useful for that too.

What are your bubbles made of?

We try to use things that are either naturally present in the body or something that you regularly intake (foodstuffs). The main material we use is the same substance on all your cells. It's a phospholipid and you normally have a bilayer of that around all your biological cells. We use that same material but in a monolayer form to coat our bubbles.

How do you make these bubbles?

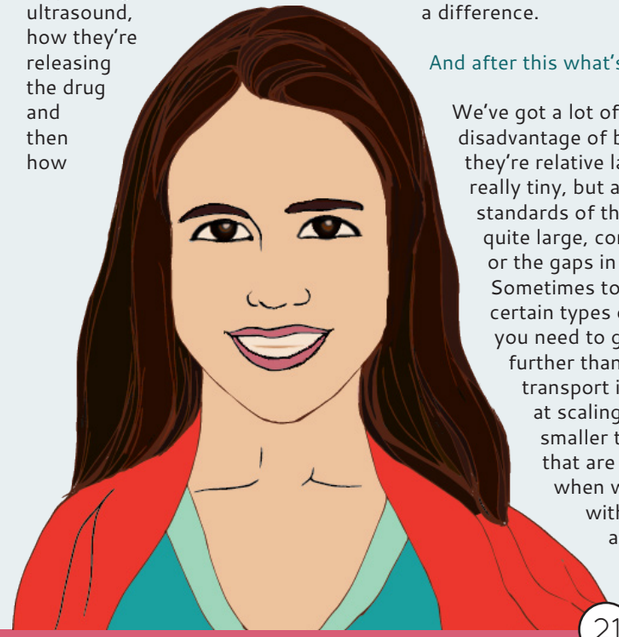
The industry standard is not very far removed from taking washing up liquid and shaking it really hard. That gives you loads of bubbles very quickly so it's very efficient, but you do get a very broad range of bubble sizes and you're not sure how much drug is in each bubble. So we've moved to looking at techniques using microfluidics that allow you to actually make the bubbles one by one, which is fantastic because we can control the size, how much drug is in there and we can predict much better how they're going to behave. The trouble is they're a little bit slow, so one of the engineering challenges is actually taking these techniques and scaling them up.

How did the idea of microbubbles come about?

The fact that bubbles make fantastic contrast agents in ultrasound scanning was discovered by accident about 50 years ago. The idea of loading drugs has also been around for a very long time, although admittedly not into a bubble. And the idea of the magnetic microbubbles was completely by chance: I was standing next to a colleague at a conference, he had a poster on magnetic nanoparticles, I had a poster on bubbles and we looked at each other and thought that would make an amazing combination.

What are the major problems you've faced?

Many. One is making sure the way we manufacture the microbubbles is something that could be scaled up and actually taken into pharmaceutical companies. And the complexity of the processes in the body are enormous. Understanding how the bubbles are responding to ultrasound, how they're releasing the drug and then how they're interacting with cells is very complicated but we need to understand those to make sure that it's going to work.



So what stage are you at currently?

We have lots of protocols for manufacturing the microbubbles, we have some very promising data with cells and also in small animal testing. My colleagues are taking a sort of beta version of ultrasound-mediated delivery into the clinic this year. That's not using microbubbles, it's just using capsules but we hope if that's successful, we'll then be able to integrate the bubbles into that and move into clinical trials of something with an already approved drug within the next two to three years.

When do you think we could see this being used in clinical settings?

We very much hope that bubbles combined with drugs will be around within the next five to ten years. The magnetic bubble idea is going to take a little bit longer, but I'm certainly hoping that within the span of my career that we'll really see this making a difference.

And after this what's the next stage?

We've got a lot of ideas. One disadvantage of bubbles is that they're relative large. I said they're really tiny, but actually by the standards of the body they're quite large, compared with cells or the gaps in blood vessels. Sometimes to treat particularly certain types of cancer tumour you need to get your drug even further than the bubbles can transport it so we're looking at scaling down to even smaller types of capsule that are liquid, but then when we insonify them with ultrasound, they actually become bubbles.

You studied mechanical engineering. What made you choose biomedical engineering?

I was doing my final year project on ultrasound imaging, but in oil pipes. We were looking at using bubbles as tracers to try and quantify how much oil and water is in a pipe coming off an oil rig in the ocean. And my supervisor happened to meet a clinician who said they had some friends who were doing something very similar for ultrasound imaging and might I be interested in looking into that further. To which the answer was yes absolutely! And that's how I ended up where I am.

How is biomedical engineering different to traditional engineering?

In terms of what we do and the concepts, it's not different at all. We're using thermodynamics, fluid mechanics, basic structural mechanics, all the same things that any other engineer would use. The only difference is the application, so rather than building cars or planes we're building tiny drug delivery vehicles. But the creativity and the mathematics and the physics are all the same.

You do a lot of science communication. Why do you think this is important?

People need to know why science is important. It's not just people in labs messing about, it really does have an impact on the world, particularly engineering. And the other thing is that most people do think of engineering as being about building bridges and cars, which it is, and those are very important, but it's a lot broader than that. Engineering affects all areas of human life.

Watch a video of the interview online at www.bangscience.org

Interview by Iona Twaddell.

Art by Thao Do.

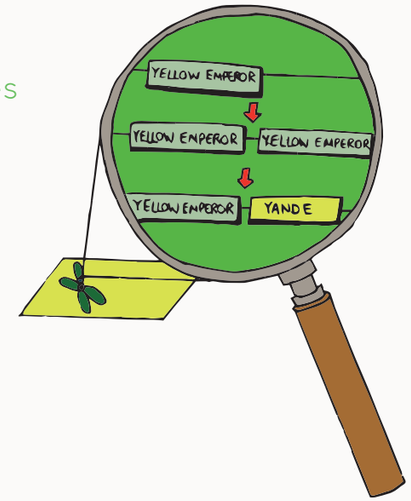
New Genes

How creating new genes creates different species

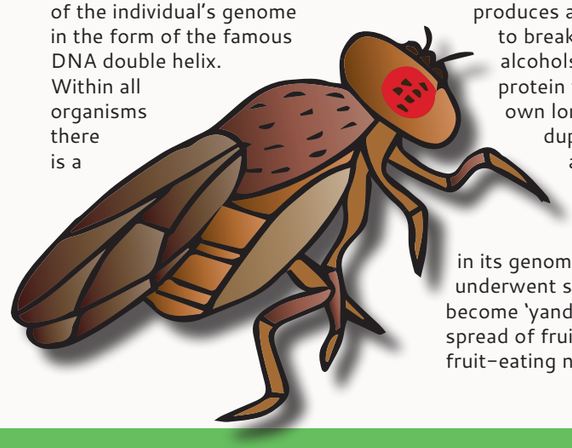
If comparisons are ever drawn between humans and bananas the emphasis is invariably placed on the genetic similarities between the two – it is a well-established fact that human DNA is 50% similar to that of bananas. Physiologically and morphologically, however, their differences are monumental; we find it astonishing that two such different life-forms can be so similar at the most fundamental level. But perhaps we are putting the emphasis in the wrong place, and what is truly astounding is the fact that a human is 50% *different* from a banana.

Life on Earth began four billion years ago and evolved from a single self-replicating molecule in the primordial soup. Evolution took that single entity and created the diversity of life that we see today. Despite the need for all living organisms to maintain a multitude of vital processes such as respiration and metabolism, evolution by natural selection has allowed a human to become 50% *different* from a banana – a truly astonishing feat. So the question that we really need to ask is how do two organisms with the same origin become 50% genetically removed from one another? The answer is through the evolution of new genes.

New genes can occur as a result of a number of different processes but one of the most common ways is through gene duplication events. Every cell contains a complete copy of the individual's genome in the form of the famous DNA double helix. Within all organisms there is a constant cycle of cellular death and regeneration, and the genome must be copied into all newly formed cells. This process of DNA replication is incredibly accurate, but copying errors are made. Occasionally two copies of a single gene will be produced and the second copy will become embedded in the individual's genome. Usually this second copy will be selected against as a result of the high energetic costs associated with its maintenance; however, every now and again having two copies of the same gene will prove to be beneficial and the second copy will be retained. Natural selection can operate on the two genes separately, through a process called subfunctionalisation, so that they acquire related, yet distinct functions. Descendants of the individual in whom the gene duplication event occurred therefore begin to build up genetic differences from their other ancestors that were not subject to the same duplication event.



Fruit-flies possess an alcohol dehydrogenase gene (ADH) which allows them to feed off fermenting fruit without getting drunk. This gene is made up of two separate components: 'yellow emperor' produces a protein with the ability to break down short-chain alcohols and 'yande' produces a protein with the ability to breakd own long-chain alcohols. A gene duplication event in the ancestor of the fruit-fly caused two copies of 'yellow emperor' to become embedded in its genome – the second copy underwent subfunctionalisation to become 'yande'. This facilitated the spread of fruit-flies into their new fruit-eating niche by allowing them to break down alcohols of any length – a privilege not afforded to their ancestors who possess only a single copy of 'yellow emperor'.



Gene duplication events create genomic speciation islands, portions of the genome where gene flow between parents is restricted. Under certain prevailing environmental conditions these islands have to be inherited by offspring in order for them to be able to survive. For example, any offspring of a fruit-fly must possess both 'yellow emperor' and 'yande' in order to be able to live off fermenting fruit. As new genes continue to emerge, gene flow becomes increasingly restricted until eventually a point is reached whereby the ancestor and the descendant are no longer able to reproduce – they are now classified as being two separate species. Genetic divergence continues in this manner as a result of the creation of new genes and genomic speciation islands until you end up with organisms that are as different as a human and a banana.

> "Fruit-flies possess a gene which allows them to feed off fermenting fruit without getting drunk"

Max Bodmer is a 3rd year Biological Sciences student at St Peter's College.

Art by Amber Barton.

Mirrors in the Mind

Why Botox could reduce your empathy

Botox is a popular anti-ageing treatment that paralyses facial muscles to help reduce wrinkles. In 2011 alone, over three million people worldwide received Botox injections. But could Botox have the unexpected side-effect of worsening the patient's ability to recognise the emotions of others? Neal and Chartrand (2011) found that individuals who had received Botox were impaired in classifying emotional expressions. Emotion recognition is critical to empathy – if we can't tell which emotion a person is experiencing, we are unlikely to understand and share how that person feels.

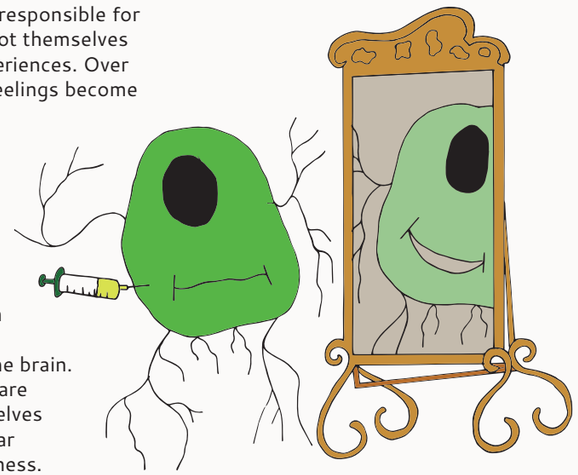
The reason Botox may impede emotion recognition is because it impairs our ability to imitate emotional expressions. When we see someone smile, we are likely to smile back. This act of mimicry helps us to identify with the feelings of others. That facial mimicry is important to emotion recognition is supported by the Neal and Chartrand study. It showed that unlike those with Botox, people with dermal fillers – an alternative anti-ageing treatment that leaves facial muscles untouched – were not impaired at classifying emotions. The facial muscles used for a particular emotional expression are also the ones crucial to the recognition of that emotion. In a 2007 study by Oberman and colleagues, participants were prevented from smiling by biting a pen. Recognition of happiness, but not other emotions, was greatly impaired. This suggests that it is the effect of Botox on facial mobility that causes impaired emotional recognition skills.

It seems there is a close connection between specific facial muscles and recognition of particular emotions. But how do external facial expressions relate to our internal identification with other people's feelings? One likely explanation is mirror neurons. These are brain cells in the frontal and parietal lobes of the brain that fire both when we perform an action and also when we observe others performing the same action. This property makes mirror neurons a good candidate for enabling us to feel empathy. If the same set of neurons are active when we express certain emotions as when we observe an equivalent emotion in other people, it could be part of the explanation of how we come to recognise what others are feeling.

> "Botox may impede emotion recognition because it impairs our ability to imitate"

Observing facial expressions triggers subconscious facial mimicry that in turn enhances mirror neuron activity for the corresponding expression. Mirror neurons are part of a motor network in the brain responsible for movements and do not themselves cause emotional experiences. Over time, however, our feelings become intimately associated with our physical expression of that feeling. For example, happiness is strongly associated with a smile due to connections between motor areas and emotional areas of the brain. The emotional areas are active when we ourselves experience a particular emotion such as sadness. Because of mirror neurons, these emotional areas are also activated when we observe others with a sad facial expression. Facial mimicry enables mirror neurons to link our own emotional experiences to those of others. This allows us to recognise – and empathise with – what others are feeling. An important feature of empathy is therefore reduced when Botox paralyses facial expressions.

However, facial mimicry may not be absolutely essential to emotional recognition. People who suffer from Moebius syndrome, a rare condition characterised by facial paralysis, are still able to recognise emotion successfully and show empathetic skills. Although this seems at odds with the theory of facial mimicry as a key to empathy, it does not have to be. Since people with Moebius syndrome are born with the condition, they never come to associate their emotions with facial expressions of that emotion. Instead, growing up with facial paralysis may give rise to compensatory strategies for emotional recognition, such as recognising emotion from voices or non-facial movement. In patients with Moebius syndrome, empathy may be achievable without facial mimicry, but it seems unlikely that this is the case for the rest of us.



Frida Printzlau is a 3rd year Psychology and Philosophy student at Christ Church College.

Art by April Hills.

Malarial Mapping

Tracking the genetics of a killer

Imagine walking into your friend's birthday party; you look around to see some recognisable faces, a few people you don't know, or perhaps some families with similar features. You wouldn't expect to see 20 identical people scattered around the room. But that's exactly the kind of scene Oxford Professor Dominic Kwiatkowski and a team of researchers stumbled across this year, while studying the genetic diversity of malaria parasites.

Through an analysis of hundreds of parasite genomes from malaria endemic countries in Africa and Southeast Asia, the researchers found that certain strains of malaria parasites in Western Cambodia were strikingly different to parasites everywhere else in the world. In fact, these strains were not only replicating faster than others, they shared unique genetic features that made the parasites easily distinguishable. It's as if you walked into that party and saw 20 identical people scattered around the room, and then another distinct group of 20 identical people, and then another. You might well stop at this point to wonder what on earth could be happening, or whether you should have had that last glass of punch... but in the lab, this unusual scenario provided a perfect springboard for an innovative research approach.

> "The collaboration has sequenced more than 800 parasite genomes, from small blood samples taken from infected patients"

These findings are the latest results from the MalariaGEN *P. falciparum* Community Project – a dynamic international collaboration involving researchers from 23 institutions across the globe. Led by Dominic Kwiatkowski (Professor of Genetics and Global Health at Oxford University and Head of the Malaria Programme at the Wellcome Trust Sanger Institute), the collaboration has sequenced more than 800 parasite genomes, using a technique they developed to extract parasite DNA directly from small blood samples taken from infected patients.

So why are Western Cambodia's rapidly replicating, genetically distinctive parasites so significant? Fascinatingly, each of the unique strains discovered pose a common threat – resistance to the world's frontline antimalarial drug, artemisinin. This is just the latest chapter in the continuing saga of antimalarial drug resistance.

For almost 300 years the most widely used treatment for malaria was quinine, but after malaria parasites started to develop resistance to the drug in 1910, its long reign was over. Chloroquine succeeded to the antimalarial throne in 1945 but, unfortunately, it took just 12 years for resistance to emerge. Sulfadoxine pyrimethamine resistance arose even more rapidly, less than one year after it was introduced in 1967. Mefloquine was similarly ill-fated, lasting just five years on the market before resistant parasites were first reported in 1982. Remarkably, many of these waves of antimalarial resistance first emerged in Cambodia.

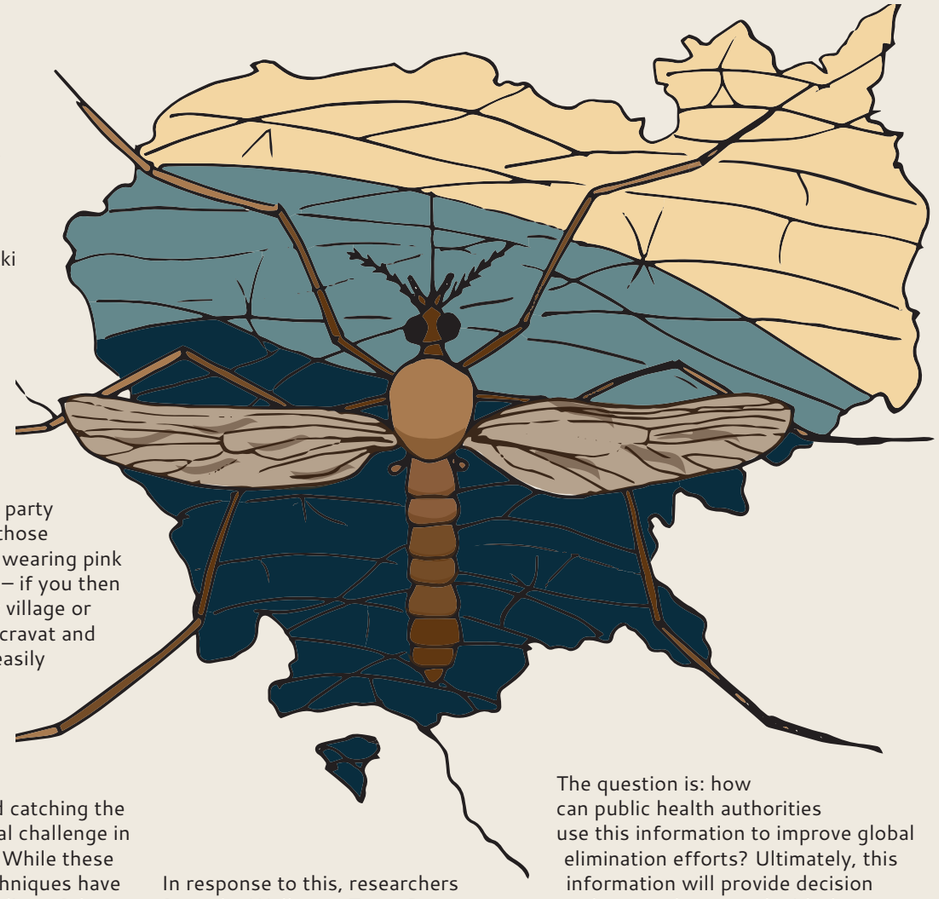
Fortunately, artemisinin, a highly effective antimalarial compound, was discovered in 1972 by Chinese government researchers. The potency of this antimalarial gained global attention in the late 1990s as a result of research from another Oxford University Professor, Nick White, and his group in Thailand. In 2006 they showed that artemisinin in combination with other antimalarial drugs – artemisinin-combination therapy (ACT) – was the most effective and fastest-acting malaria treatment. This persuaded the World Health Organization (WHO) to recommended ACT as the primary method of malarial treatment.

But while the introduction of ACT has contributed to a 25% reduction in malaria mortality and morbidity worldwide since 2000, it too is now being dogged by the threat of resistance, which would have devastating consequences across the globe.

In 2009, Professor White's group found that malaria parasites in Western Cambodia were taking longer than usual to clear from patients' blood – a worrying sign of drug resistance and an early confirmation that antimalarial resistance was again occurring in the region. In 2012, they showed that these resistant parasites had also emerged in Thailand.

Since resistance to the world's frontline treatment for malaria could lead to catastrophic losses of life, this news triggered a rapid response from the international community. For example, the WHO recently launched an emergency response to artemisinin resistance in Southeast Asia, and received 100 million US dollars in funding from the Global Fund to Fight AIDS, Tuberculosis and Malaria, to combat artemisinin resistance at its source.

But before you can combat resistance at its source, you must first identify where it is coming from. This is where MalariaGEN and Professor Kwiatkowski's discovery of unique strains of parasites in Western Cambodia comes into play. By stumbling across that figurative birthday party, and finding that the malaria parasites in Western Cambodia were strikingly different to parasites everywhere else in the world, they demonstrated that genetic analysis could be used to identify resistant parasites.



Crucially, the techniques developed by Kwiatkowski and MalariaGEN researchers means that they can now identify the genetic characteristics of particular resistant populations from a simple blood sample taken anywhere in the world. Going back to the party analogy, it's as if one of those identical groups were all wearing pink cravats and a bowler hat – if you then saw someone in the next village or next country with a pink cravat and a bowler hat, you could easily identify them as part of that group.

But identifying the killer is just half of the struggle; it's tracking and catching the killer that remains the real challenge in fighting drug resistance. While these genetic identification techniques have led to a better understanding of the specific parasites at the source of drug resistance, translating this information for use in the field is imperative if any real impact is to be made.

In response to this, researchers from the Wellcome Trust Sanger Institute and Oxford University are now working on designing simple and cost effective systems to send blood test information from the clinic to the lab for analysis, and then return the genetic analyses quickly back to the field, using mobile technology. Professor Kwiatkowski explains: "A small blood spot test should eventually be able to tell healthcare workers how many parasites are in a blood sample, what species they are, whether they are drug resistant, where they have come from, and whether that particular parasite is common to any other people in the area."

> "The question is: how can public health authorities use this information to improve global elimination efforts?"

As Professor Kwiatkowski explains: "If we can develop smart genomic tools that relay genetic information back to people working in endemic areas, we can help them to answer critical questions about the spread of resistance, such as: what should be done next, what the impact of previous interventions has been, and which areas need to be targeted first."

The question is: how can public health authorities use this information to improve global elimination efforts? Ultimately, this information will provide decision makers on the ground with the knowledge they need to target elimination resources, particularly to populations with a higher proportion of drug resistant parasites. Scaled-up interventions in these critical areas will assist in containing artemisinin resistant parasites and may slow, or even cease, the spread of resistance globally.

While the story is far from over, this research will undoubtedly improve global elimination efforts and lead us one step closer to ending the cycle of resistance once and for all.

Kate Fuller is a science impact development writer at the Nuffield Department of Medicine.

Art by Kate Williamson.

The Price of Altruism

The mathematics of evolution

$$\Delta\phi = \sigma\left(\frac{\omega_i}{\bar{\omega}}, \phi_i\right) + E\left(\frac{\omega_i}{\bar{\omega}} . \Delta\phi_i\right)$$

"The Price equation is to biology what $E = mc^2$ is to physics." At first I didn't believe my tutor, as such truisms in biology are often gross generalisations. However, I quickly realised that Price's equation is a very fundamental statement. The Price equation is a deceptively simple way of looking at the effects of selection. It describes how a certain trait changes between generations through natural selection, and the factors that affect this.

In biology, altruism is where one organism pays a cost to benefit another organism's fitness, or its contribution to the future gene pool. W.D. Hamilton's rule is a condition that when met keeps a certain trait favourable for natural selection. This rule essentially states that the cost to one's own fitness of performing an altruistic act must be neutralised by the recipient sharing sufficient genetic material with the actor. Helping a clone to reproduce is, in terms of genetic contribution, the same as reproducing yourself. It is often stated as an inequality, $rb - c > 0$, where: r is the relatedness between actor and recipient, b is the benefit to the recipient, and c is the cost to the actor. For example, if b would allow my sister to produce two offspring, but I give up energy equivalent to raising 'half an offspring', then: $b = 2$, $c = 0.5$ and $r = 0.5$. So $rb - c = 0.5$, $0.5 > 0$. In this case, natural selection would favour the altruism gene. The way r is calculated varies, but a simple way to interpret it is as the chance that you share the same altruism gene.

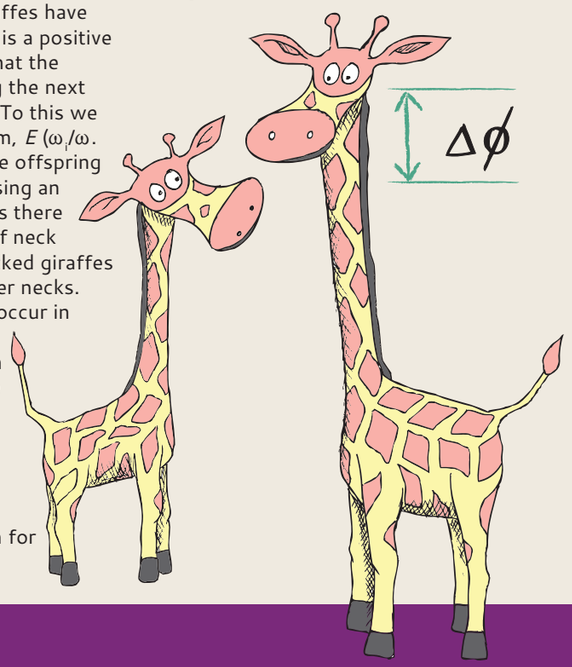
Price's equation extends the justification for natural selection to the mathematical level. Hamilton and Price corresponded during the development of Price's ideas. Hamilton realised the power of Price's equation and used it to recreate his rule for altruistic traits. The two published their respective papers side by side in *Nature*. Hopefully, you can see that the Price Equation has three major terms which can be described biologically. Consider a population of giraffes. Among bulls, neck length is used to determine dominance; more dominant males have better breeding success. Now let's interpret the Price Equation in this scenario. $\Delta\phi$, represents the change in average neck length between the giraffe population and their calves. This change has two elements to it, which are described on the right-hand side. Firstly, $\sigma\ (\omega_i/\omega, \phi_i)$ describes change due to selection and is expressed as a covariance (a measure of how two variables are associated) between the fitness of any one individual and its own trait value. If longer necked giraffes have more offspring, then there is a positive covariance, which means that the average neck length among the next generation will be greater. To this we add a general catch-all term, $E\ (\omega_i/\omega . \Delta\phi_i)$. This describes how the offspring differ from their parents using an arithmetic average. Perhaps there is some epigenetic effect of neck length that means long necked giraffes have calves with even longer necks. This additive effect would occur in the expectation (average) term. If calves were carbon copies of their parents, the term would be zero as the change in trait value, $\Delta\phi_i$, would be zero.

Price himself disliked the implications of his theorem for the logic of social actions, because it essentially reduces altruism to a cost-benefit analysis, a mere mathematical calculation. He worked hard to find a problem with the mathematics of his work, as well as seeking biological counter-examples. He opened his home to alcoholics and the homeless, to whom he donated all of his wealth. Some interpret his extreme altruistic behaviour as an attempt to defy the parameters of Hamilton's rule. Price was later evicted from his house and became depressed, which culminated with his suicide on 6th January 1975.

To many, reducing the complexity of life amounts to diminishing the sense of awe that we should have for the world. I would argue that we should celebrate our ability to identify the simple rules underlying life. The Price equation is nothing more or less than a definition. How we use that definition to increase our understanding of evolution, and by extension the Universe, is what is truly important.

Matishalin Patel is a 3rd year Biological Sciences student at Lady Margaret Hall.

Art by Iona Richards.



The Moral Compass

Is Morality Innate?

Imagine you're watching someone trying to climb a hill. One person is helping the climber up, while another is pushing the climber down, for their own obscure reasons. When the climber eventually reaches the top of the hill, you would be surprised if that individual walked over to the unhelpful person and avoided the helpful one.

It turns out that nine-month-old babies also understand that people express gratitude for helpful behaviour rather than mean behaviour. Paul Bloom and colleagues at Yale University showed nine-month-olds a version of this scenario with shapes (a ball was climbing the hill, helped by a square and hindered by a triangle). The infants were surprised if the ball approached the unhelpful triangle at the top of the hill. Even babies assume that the act of unnecessarily approaching somebody reinforces that person's behaviour. Moral beings reward helpers and punish hinderers, so it is morally coherent for the ball to approach only the helpful shape.

Children's surprise was inferred from the greater amount of time they spent looking at the unexpected (morally incoherent) event relative to the expected (morally coherent) event. Such research attracts much interest because babies have very little experience of a cultural environment. Therefore, their moral behaviour may be innate, reflecting a natural genetic predisposition for humans to be good.

However, scepticism surrounds the definition of morality adopted by this research. In order to scientifically test a concept as complex as morality, researchers focus on a single aspect of the moral sense and thereby ignore its other components. For example, can babies recognise that the morality of an action does not depend on who is performing it but does depend on their intentions? For example, amoral beings may help a climber because they want to. Yet only moral beings would help a climber given the belief that they ought to even if they don't want to.

In order to test whether an inborn ability to recognise this *helpful* behaviour extends to recognition of *moral* actions, researchers have presented older participants with moral dilemmas. These ask whether it is right to deliberately kill one person to save more; for example, to kill a dying person to distribute his/her organs across five people who need them immediately to survive? Although affected by the context in which the dilemma is presented, an overwhelming majority of people refuse to hypothetically kill one person to save five. Participants respond to such dilemmas quickly, with certainty, and are unable to consistently articulate the reasoning behind their decisions – researchers have inferred that the rules used are long, complex and abstract. These are all characteristics of intuitive knowledge which is not consciously learnt.

> "It is now possible to investigate the origins of morality at the level of genes"

However, the moral sense could be learnt implicitly (unconsciously) through imitation, reward and punishment. If so, we would expect variables such as nationality, age, sex, race, education and religion to predict differences in responses to moral dilemmas, yet they don't. What's more, children exhibit knowledge to which they have not been culturally exposed. For example, by the age of three, children can distinguish between moral violations (e.g. stealing) and violations of societal norms (e.g. wearing pyjamas to school). However, parents punish both of these violation types and do not tell children the difference between the two.

With advancements in technology, it is now possible to investigate the origins of morality at the level of genes directly. In 2011, Abigail Marsh and colleagues at Georgetown University found that participants with short (rather than long) versions of a gene were more likely to perceive killing one person to save five as morally wrong. This effect was attributed to a previously discovered link between the short version of the gene and reduced reuptake of the brain chemical serotonin. This interpretation is consistent with a previous finding: antidepressants which lower serotonin reuptake also reduce the perceived acceptability of killing one to save five. This suggests a link between genes and moral reasoning via the brain.

Altogether there is evidence that the ability to recognise good from bad and the need to reward right but not wrong is genetic. But none of the research above refutes the cultural need for positive role models and reinforcement to fine-tune the contents of children's moral knowledge. In the words of New York University psychologist Jonathan Haidt: "We're born to be righteous, but we have to learn what, exactly, people like us should be righteous about."

Robert Blakey is a 3rd year Experimental Psychology student at St Catherine's College.

Art by Haneesh Sidhu

Teetering on the Brink

How rising carbon dioxide levels will change our world

In 1958 a geochemist named Charles Keeling developed a method of measuring the concentration of carbon dioxide (CO_2) in the atmosphere at the Mauna Loa Observatory in Hawaii. His first measurement recorded a level of 315 parts per million (ppm). Fast-forward 55 years, burn a few billion tonnes of fossil fuels, and today the earth teeters on the edge of the first recorded measurements of 400ppm.

Although our atmosphere has held CO_2 for millions of years, scientists believe the last time the concentration reached 400ppm was during the Pliocene epoch, approximately four million years ago. The world then was vastly different to today – North and South America were separated by a narrow sea, alligators roamed Europe and Arctic temperatures were thought to be 19°C warmer than today. Such a high temperature today would melt hundreds of kilometres of arctic ice and seriously affect flora, fauna and sea levels. To give an idea of the scale of such changes, leading climate scientist Professor Maureen Raymo estimates that Pilocene sea levels were 10-40m higher than present. So what does 400ppm in the atmosphere today mean for us?

> "We can safely expect not to suffer global catastrophes such as those portrayed in films like 'The Day After Tomorrow'"

Possibly the most troubling aspect of the 400ppm measurement is the fact the International Panel on Climate Change (IPCC) have come to a broad consensus that it is "very likely" (more than a 90% probability) that the increase in CO_2 that has occurred since 1958 is the result of anthropogenic (human induced) greenhouse gas emissions.

Reaching a 400ppm CO_2 level is not going to have dramatic impacts on our day-to-day weather in the short term, but the warnings from palaeoclimatology tell us that, if the rise in CO_2 continues unchecked, we could be changing the planet in ways never seen before in human history.

However greenhouse gases should not be viewed entirely negatively. They positively contribute to regulation of the planet's stable temperatures and CO_2 is required for photosynthesis, a vital process for all living organisms. Furthermore, in an ecological sense, the difference between 399ppm today and 400ppm tomorrow is pretty negligible. Yet this doesn't mean it is not a cause for concern. Conservative estimates suggest that if trends in emission rates continue, global temperatures will increase by approximately 2°C by the end of the century. It has been estimated that these changes of this magnitude would lead 57% of the world's plant and 34% of animal species to lose over half their current habitat ranges by the end of the century.

It is important to also remember that more than just CO_2 influences climate change. Aerosols (small particles such as dust) as well as water vapour are thought to play big roles in atmospheric fluctuations. There is also further uncertainty about drivers of natural variability in the earth system and the limitations of our current set of climate models.

The latest IPCC report doesn't suggest an impending tipping point, which in this scenario would constitute a transition from one stable climate state to another; indeed not to Pliocene-esque conditions. However the picture in the long term remains uncertain.

Nonetheless, the 400ppm measurement is all not doom and gloom. The photosynthetic organisms that bloom for the northern hemisphere's summer help to regulate atmospheric CO_2, and we can expect the concentration to fall by around 6ppm by November. Furthermore, we can safely expect not to suffer global catastrophes such as those portrayed in films like "The Day After Tomorrow". Science fiction defies some pretty basic laws of physics, such as the Ideal Gas Law, which dispels any idea of a continent sized super-storm.

For those who don't want a pessimistic reminder from the world of climate science, there is hope. In 1987 in Montreal many of the world's nations agreed to address issues that are detrimental to our climate; after all, we no longer use chlorofluorocarbons to cool our fridges. This has gone a great way in helping reduce the hole in our ozone layer. Furthermore, the latest IPCC report provides renewed scientific rigour for the diplomatic agenda at the next UN climate change conference, Warsaw 2013. The arrival of the world's climate at 400ppm should provide fresh impetus for the next round of negotiations.

Ashleigh Ainsley is a 3rd year Geography student at St Catherine's College.

Art by Iona Richards.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