The Scholar

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thebrilliantclub.org

Were Victorian Asylums a Superior Solution to Earlier Methods of Treating the Mentally Ill?

+ 19 New academic essays from the young scholars of The Brilliant Club.

This issue

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Updates

News from The Brilliant Club

Researchers in Schools is Recruiting PhD Graduates and Schools

Alongside The Scholars Programme, The Brilliant Club also runs Researchers in Schools (RIS). RIS recruits, trains and places PhD researchers as part-time tutors and full-time teachers in non-selective state schools. Through RIS, PhD graduates become highly-effective classroom teachers, champions for university access and future subject leaders in the education sector.

Researchers in Schools is currently delivered in partnership with School-Centred Initial Teacher Training providers and has placed more than 150 participants in over 75 schools.

Current participants include Dr Alice Len, teaching physics at Cleve Park School in Sidcup. Alice completed her PhD at the University of Sydney and post-doctoral research on HIV with the Department of Infection and Immunity at University College London. She has acquired a £15,000 microscope for her school, donated by Professor Bill Richardson from UCL. Alice has also organised for a group of Year 12 students to complete work experience next summer at the university.

The RIS programme is now recruiting maths and physics PhD graduates to become excellent new teachers, starting the programme in August 2017. Schools that are interested in working with new subject specialist RIS teachers should also get in touch.

To find out more about the groundbreaking work of Researchers in Schools, please visit www.researchersinschools.org.

The Scholars Programme Launches in Scotland and Wales!

Last term saw the launch of The Scholars Programme in Wales. Four schools and colleges from different areas of Wales are pioneering the programme, which connects state-school pupils with PhD researchers. Participating pupils attended a launch trip at the University of Cardiff, which was a resounding success. Pupils enjoyed a tour of the University's campus, asked questions of current students and admissions staff, and completed their first tutorials with their PhD tutors.

The Brilliant Club is also working with The Seren Network, which supports high-performing sixth form pupils from across Wales to secure places at top universities. Our PhD tutors have delivered master classes to pupils and training to schools on how to deliver university-style learning in the classroom. The Brilliant Club and Seren will also be holding a series of conferences, the first running from 15th - 16th March, that will see over a thousand pupils and their teachers participate in workshops designed to support pupils to develop the skills, knowledge and confidence needed to obtain places at highly-selective universities.

This term, The Scholars Programme officially launches in Scotland! The programme will be piloted in partnership with the University of Strathclyde and Glasgow City Council and will be delivered in two schools in Glasgow.

Monitoring and Evaluation Gets a Boost

The Brilliant Club is committed to the rigorous monitoring and evaluation of its programmes to ensure that we deliver consistent outcomes for the pupils we work with. To this end, since September, we have recruited three members of staff to the Monitoring and Evaluation Department, more than doubling the size of the team! This will enable the department to evaluate the impact of The Brilliant Club's programmes from beginning to end.

A recent evaluation of the university destinations of pupils who completed The Scholars Programme in summer 2015 revealed that, of a total cohort of 325 pupils, 77% applied to a highly-selective university and 71% received an offer from one. This means that nine out of every 10 pupils who applied to a highly-selective university received an offer from one! There were also 15 pupils who accepted offers from Oxford and Cambridge.

These achievements demonstrate both the academic calibre of the pupils who participate in The Scholars Programme and the efficacy of the programme in developing the knowledge, skills and ambition necessary to secure places at highly-selective universities amongst its participants.

The Monitoring and Evaluation Department has also designed a competency framework that details how we will support pupils to develop the knowledge, skills and ambition needed to progress to highly-selective universities. This is built upon a series of skills that research shows have a positive impact on academic attainment. The Brilliant Club has identified six competencies that we believe will be effective in supporting our pupils on their journey to excellent universities. These are:

1. Written and Verbal Communication
2. Subject Knowledge
3. University Knowledge
4. Motivation and Self-Efficacy
5. Meta-Cognition
6. Critical Thinking

We look forward to sharing updates as we use the competency framework to track and evaluate the progress made by pupils on The Scholars Programme.

Guest Article

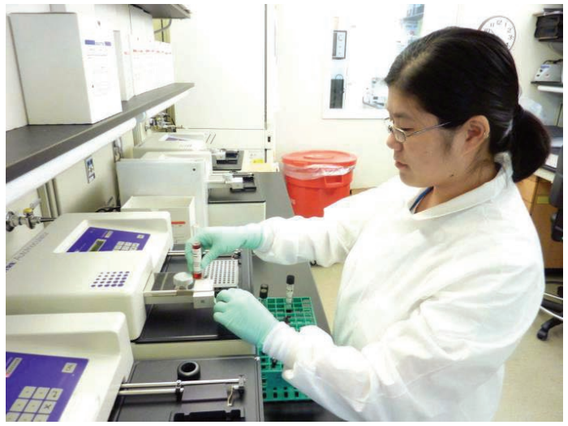
Scientific Publishing: From the Lab to the World

Dr Joana Osório
Chief Research Highlights Editor
Nature Research,
Springer Nature

A scientist has a great idea, obtains funding to research it, does a lot of hard work with their team and finally finds something interesting we didn't yet know about. Exciting! But that's far from being the end of the story. For these research findings to be useful to other scientists and to society in general, knowledge needs to transition from the lab to the outside world. Without this process, even the most brilliant discoveries will become dust at the bottom of a drawer.

Science communication is changing, but the most typical process of bringing new discoveries into the open is still the publication of a scholarly article in an academic journal. Nature is one such journal – and one of the most well-respected and high-profile scientific publications in the world. In addition to articles written by scientists, Nature publishes a broad variety of content. Research Highlights, for example, are short editorial pieces that summarise and highlight the context, findings and implications of a recently published research paper. They are 'snacks' of important new scientific knowledge, made accessible to a broad audience of people interested in science – not only to scientists working in a specific field. The partnership between Springer Nature, the publisher of Nature, and The Brilliant Club is a wonderful opportunity to communicate exciting new developments in science with the talented high-school pupils who read and write for The Scholar.

Londell McGlone, M.P.H. preparing specimens for testing during an investigation of pertussis-like illness in Ohio, USA



Microbiologist performing antimicrobial susceptibility testing of Streptococcus pneumoniae

So, where does the process of science communication begin, and who plays a part in it? First, the researchers write their scientific article – the 'paper' – where they describe the context of the research, the methodology used during the investigation, the main results of the study, the conclusions that can be drawn from the results and the overall implications of the work. Then, they send their paper to the journal they feel is the most appropriate venue for their work.

Laboratory technician adding solution to an enzyme-linked immunosorbent assay (ELISA) at the Instituto de Salud Pública de Chile

The number of scholarly journals currently available is huge. Some journals are multidisciplinary, some are dedicated to a specific area of knowledge. Some only publish articles that are considered major advances in the field, others are dedicated to publishing all research, regardless of novelty, as long as the work is sound. Some publish articles that are free to read, some require readers to pay for access. Regardless of these differences, scientific papers sent to a journal are evaluated by either professional editors or external experts who are qualified to assess work done in a specific research area.

These people make decisions on whether to reject, ask for revisions, or publish the papers. Usually, decisions on publication are also guided by the opinion of peer-reviewers, researchers working in the same field as the authors. The peer-reviewers comment on the accuracy, validity and, for some journals, interest level of the work.

Many professional journal editors dedicate their time solely to assessing, orchestrating the peer-review process and making publication decisions about research papers. However, the job of an editor can vary from journal to journal. Some editors attend conferences and visit laboratories and other places where they can meet and talk to scientists and keep on top of what is happening in a research field. Some editors identify emerging or controversial topics or select important papers that merit further discussion and invite experts to write an opinion piece or a review about them. Some editors help authors improve the text and images of their articles. Some editors write news pieces, commentaries and editorials about specific discoveries or other science-related topics. Some write Research Highlights.

Nature's mission statement, dating from 1869, mentioned two main objectives: "FIRST, to place before the general public the grand results of Scientific Work and Scientific Discovery, and to urge the claims of Science to a more general recognition in Education and in Daily Life; and, SECONDLY, to aid Scientific men themselves, by giving early information of all advances made in any branch of Natural knowledge throughout the world, and by affording them an opportunity of discussing the various Scientific questions which arise from time to time." The Research Highlights published at Nature fit within this mission statement. I hope the readers of The Scholar will find our selection fun to read, a source of inspiration, and fuel for a continued interest in scientific research and communication.

Dr Joana Osório

Laboratory photos from Centre for Disease Control Public Health Image Library
Photo of Dr Osório author's own

Guest Article

The Scholars Programme: A Teacher's Perspective

Lucy Hemsley
Head of Higher Education and Upper Ability Provision
Cheltenham Bournside School and Sixth Form Centre

Widening access to elite universities is something about which I am so passionate that I explored it for my Master's thesis; I am therefore always on the lookout for meaningful ways to support students on their journey towards higher education. Yet, having now seen two cohorts of students complete The Scholars Programme, I can confidently state that The Brilliant Club is one of the best initiatives in which I have been involved.

Why we decided to take part

The Scholars Programme immediately appealed to me for its academic rigour, its links with top universities and its ability to deliver a programme that not only takes students into universities, but also brings PhD researchers into schools, so that students can experience university-style tutorials. What's more, for busy teachers, the fact that the work is planned, delivered, marked and moderated by The Brilliant Club is an added bonus. Even the trip paperwork, letters and risk assessment are kindly provided!

The flexibility of the programme to deliver tutorials ranging from Years 6 to 12 is another real strength in an arena that is so often limited to specific cohorts at specific times of the year. Reflecting on the higher education provision in my school, I felt that, despite the wealth of opportunities and support in place from Year 9 upwards, we could do more for younger students to start thinking about, and preparing for, top universities.

After an assembly launch of the programme, students were asked to write a letter of application to the Headteacher – we were overwhelmed by the response! We selected students based on their letter of application, their prior attainment and contextual information (eligibility for free school meals or the Pupil Premium, or if their parents had not been to university).

What our students did

We have run two Scholars Programmes for Year 8 students this year: one Philosophy course ('Do the ends justify the means?') led by Anil Awesti (University of Warwick) in the Autumn term, and a Maths course ('Could the stars float in the bath?') taught by Scott Harper (University of Bristol) in the Spring term. In addition to the programme itself, students visited The University of Oxford (Wadham College and Lady Margaret Hall) for their launch trip and had a graduation ceremony at the University of Warwick. At graduation, all of our students were accompanied by a parent, which was particularly powerful given that a significant number of parents had never been to a university before. For me, this was the most memorable part of the programme: seeing students proudly collecting their certificates, wearing their mortar boards, surrounded by their parents celebrating their university-style learning.

The benefits for our students

The Brilliant Club compiles a detailed report on the impact the programme has on your students, making it easy to quantify both in terms of attainment and aspirations. 100% of our students said that they now aspire to study at one of the top universities. Beyond the statistics, the benefits of the programme for our students are clear. Hannah (Year 8) said: 'I really enjoyed speaking with and being taught by a PhD tutor as it let me visualise what university is like.' Isabel (Year 8) also commented on the value of the academic work itself, for example 'the structure of an essay'. Max (Year 8) thought that: 'The course was hard but it's supposed to challenge me. I think it has made me more confident because I know how to deal with harder things so I'm less fazed.'

For many of our students, the university visits were the highlight of the programme. Following his university visit, Michael (Year 8), now thinks that 'those are the kind of places I want to go.' Another student, who is in receipt of the Pupil Premium, now wants to apply to Oxford and both her teachers and her parents have seen how her involvement in the programme has increased her motivation at school.

As with all schools who take part in The Scholars Programme, two students from each cohort received a Distinction for their final assignment. However, every single one of our students showed increased confidence in both their academic ability and in their potential to study at a top university and, for that, The Brilliant Club receives a Distinction from me!

Lucy Hemsley

Guest Article

Brilliant Science

Dr Annalisa Alexander
Head of Outreach
Imperial College London

As we approach Christmas, the campus at Imperial starts to buzz with excitement as our undergraduates go home after a busy term and the university settles down into the swing of academia after the hectic summer school season. This year, we welcomed over 500 pupils from Year 9 to Year 12 on to campus, keen to learn more about science and get to grips with the more difficult aspects of these often-misunderstood subjects.

Imperial is a unique university in that all we do is science. A passion for learning about, experimenting with and understanding science underpins everything we do and that translates across our work with postgraduates, undergraduates, teachers and school pupils of all ages. Our summer schools showcased some of the most exciting cutting edge science, whilst also dealing with the more mundane, yet important areas that will get you through the exams and into the career of your choice.

Science often gets bad press at school. It can be very challenging, sometimes boring, and can seem pointless to those who are studying it – but trust me, it isn't. When you see how new drugs to treat cancer are created or learn how spectroscopy is used every day by analytical chemists on a much larger scale, it suddenly becomes so much more interesting and exciting.

Many of our school programmes are taught by young postgraduate students from Imperial who use the techniques and skills they are teaching in their everyday work. This makes the subjects more relevant and opens up a whole new world of career options. We encourage our undergrads and postgrads to talk to pupils about their love of science and what inspired them to study it further. It's a common misconception that you should only study science if you want to become a doctor, vet or engineer but did you know that a lot of science degrees lead to careers in the city, or within industry or even in schools or universities? Choosing to study science at A-level and beyond can open the door to a huge range of opportunities and can often lead you down the most unexpected and exciting career path!

I always loved biology at school but I was sure I wanted to become a primary school teacher like my mother (and in fact every female member of my family for the past few generations). However, some good advice from a teacher at my secondary school meant that I decided to study biology, chemistry and maths at A-level, to keep my options open. I wanted to study music as well but realistically I couldn't fit in all the hours of practise that were required. So, I dropped music but kept my instruments up as a means of stress relief and having fun. My sage teacher suggested that I might like to study biology at university rather than train to become a primary school teacher as I could always re-train later and I should harness my passion. I hadn't a clue what to do with biology. Be a biology teacher, right? But then I spent a summer working in a research lab at my local university. My dad was an academic there and he encouraged me to send a few letters (we used pen and paper in those days... e-mail didn't exist!) and I was invited to spend three weeks working in an ecology lab. It was incredible and very eye-opening. I found out about career options in research and academic administration at universities. As a result, after completing a degree in biology and then a PhD in ecology, I realised that it was the university environment I loved and so, 15 years on, I am thoroughly enjoying myself working with a crazy, passionate team of scientists in Outreach at Imperial – inspiring other pupils to love science the way we do and opening their eyes to the vast range of career options out there. And guess what? I get to teach biology in our schools' lab to primary and secondary school pupils, so you could say that I went full circle.... But I certainly wouldn't have had the wealth of experiences and options I have had if I hadn't listened to that teacher all those years ago.

Annalisa Alexander

The Brilliant Club

What is The Brilliant Club?

The Brilliant Club is an award-winning charity that exists to widen access to highly-selective universities for under-represented groups by mobilising researchers to bring their academic expertise into state schools. We do this through two core programmes:

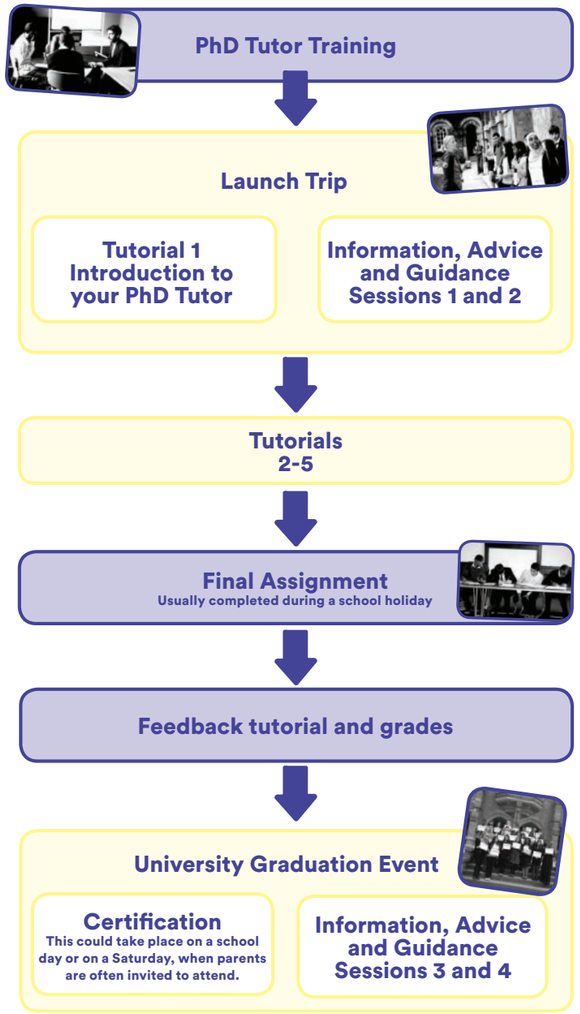
The Scholars Programme trains PhD and postdoctoral researchers to deliver university-style courses with tough academic challenges to groups of pupils. These courses begin and end with information and guidance trips to highly-selective universities. Researchers in Schools is an initial teacher training route for researchers to become classroom teachers and in-school champions of higher education and education research. Both programmes are designed to support pupils to develop the knowledge, skills and ambition necessary to secure places at highly-selective universities.

The Brilliant Club is building a national movement to mobilise PhD researchers to engage with state schools serving low HE-participation communities. At present, we are working with over 400 schools, placing over 600 PhD tutors a year to work with more than 8,500 pupils across the UK.

Through The Scholars Programme, our PhD tutors deliver courses of university-style learning to pupils from Year 5 through to Year 12. The courses they deliver focus on fascinating topics ranging from, 'Tackling Heart Attacks with Bubbles' to 'What Language do Humans Speak? An anthropological voyage through cultural and linguistic relativism'.

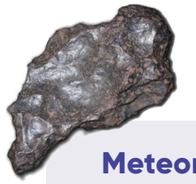
As the diagram to the right shows, The Scholars Programme consists of trips to highly-selective universities, a series of tutorials and the completion of university-style assignments. It is the best of these university-style assignments that are debuted here, in The Scholar.

We are delighted to showcase our pupils' work and celebrate their achievements in the country's only academic journal dedicated to publishing university-style assignments authored by school pupils. Publishing original work is an important component of academia and it is exciting for us to introduce our pupils not only to the world of research but also to the next stages of circulation and response from the academic community.



Highlights

nature research



Chemistry

Meteorite makes good catalyst

An iron-based mineral from a meteorite can catalyse a chemical reaction that splits water into oxygen and hydrogen, which can be used as fuel.

Some naturally occurring metallic minerals are known to have catalytic activity. Kevin Sivula and his colleagues at the Swiss Federal Institute of Technology in Lausanne studied pieces of the Namibian Gibeon meteorite, which was identified in the nineteenth century. They tested how efficiently the mineral could catalyse the oxidation of water, and found that it performed as well as synthetic iron–nickel catalysts and remained stable for 1,000 hours.

The catalytic performance emerged only after about 10 hours of operation, when a layer containing concentrated nickel, iron and cobalt with a unique 3D structure formed at the material's surface. Natural materials could inspire the creation of new kinds of catalyst, the authors suggest.

Energy Environ. Sci. http://doi. org/brsp (2016)

Animal Cognition

Bees learn and 'teach' others

Bumblebees can learn to manipulate objects — and can pass their knowledge on to other bees.

Lars Chittka at Queen Mary University of London and his colleagues presented bumblebees (Bombus terrestris) with a disc that had been filled with sugar water and placed under a transparent sheet of Plexiglas. To get at the disc, the bees had to pull on a string attached to it (pictured). Just 2 bees out of almost 300 worked out how to do this on their own; most needed stepwise training, after which more than 80% of bees were successful.

When untrained bees watched other bees getting the sugar water, they were able to learn the trick. Seeding untrained colonies with a single trained 'demonstrator' and then pairing bees from the colony with the disc apparatus eventually resulted in roughly half of the foragers learning the task. None of the foragers in the control colonies could pull the disc out.

PLoS Biol. 14, e1002564 (2016)

Neuroscience

Hunger overrides other motivations

Hungry mice will seek out food in fearful situations that they would normally avoid, and researchers have pinpointed the neurons in the brain that seem to control this behaviour.

Michael Krashes at the US National Institutes of Health in Bethesda, Maryland, and his colleagues stimulated appetite-regulating neurons in the hypothalami of mice that had recently been fed, and observed their behaviour in various settings. They found that the animals were more willing than non-stimulated ones to enter open, unprotected spaces or areas infused with fox odour in order to obtain food. Hungry or brain-stimulated males also opted to pursue food rather than spend time with a female mouse. Future studies could reveal how these neurons suppress competing drives such as fear and sociality.

Neuron http://doi.org/brbf (2016)

Astronomy

How black hole obscures itself

A supermassive black hole at the core of a distant galaxy is hiding in a cloak of its own making. Supermassive black holes are shrouded by doughnut-shaped rings of gas and dust, but scientists are not sure where these come from. A team led by Jack Gallimore of Bucknell University in Lewisburg, Pennsylvania, used the Atacama Large Millimeter/submillimeter Array in Chile to observe galaxy NGC 1068, 14.4 million parsecs (47 million light years) away. They saw hot, ionized clouds of carbon monoxide gas flying away from the galaxy's black hole in opposite directions.

This suggests that the gas originates from the disk of material swirling around the black hole and is flung off by its spinning magnetic field. The findings could alter theories of how black holes interact with their host galaxies.

Astrophys. J. 829, L7 (2016)

STEM

Volcanic Hazards - Interactions of people and volcanoes

Year 9, Key Stage 4

The Ferrers School, Northamptonshire
A. Johnstone, supervised by N. Jordan, University of Leicester

Task: Which volcanic hazards can you identify at Ruapehu, Merapi and Yellowstone volcanoes and how do they differ from each other? Based on their location and eruption history, which one of them is the most dangerous?

Introduction to the volcanoes

Mt. Ruapehu is situated in New Zealand; it is one of New Zealand's most active volcanoes and forms the highest peak of the North Island (VolcanoDiscovery, 2015a). Ruapehu began erupting at least 250,000 years ago and its magma source might be anywhere between 1 and 5 km below the crater (GNS, 2016). When an eruption from the crater occurs, it can drain the crater lake and cause extremely dangerous lahars (VolcanoDiscovery, 2015a). Lahars are strong mudflows that form when magma hits water on the surface of the volcano (such as snow). Often, when Ruapehu erupts, the crater lake causes magma to cool extremely quickly and to fragment (explode) suddenly and violently into tiny pieces, which leads to fine ash falling over the countryside. When this volcano erupts, I think the lahars will greatly affect the nearby population and cause lots of damage and masses of ash to be produced (GNS, 2016).

Mt. Merapi is north of Central Java's capital, which is called Yogyakarta and it is Indonesia's most active volcano (VolcanoDiscovery, 2015c). On average, Merapi erupts every five to ten years. The eruptions produced by Merapi are greatly feared because of the deadly pyroclastic density currents created. They can travel up to 20 km away from the crater, causing devastating effects for the people around the volcano. When this volcano erupts, I think that the most-feared hazard should be the pyroclastic density currents and clouds because they will destroy everything in their path and cause fires everywhere they can. Many tonnes of pyroclastic materials will then fall on nearby villages and cause great devastation in the area.

Mt. Yellowstone is one of the largest known volcanoes in the world and is the largest volcanic system in North America (VolcanoDiscovery, 2016). The volcano is above an intra-plate hot spot that has been feeding the magma chamber underneath Yellowstone for at least 2 million years (VolcanoDiscovery, 2016). An intraplate hotspot is when the hot molten magma from deep within the Earth rises through the crust to reach the surface, sometimes happening in the middle of a tectonic plate. When a hotspot forms in the middle of a plate, the hotspot remains constant while the plate moves over it. The result is that a trail of volcanoes is left behind, with older volcanoes moving away from the hotspot, and newer ones forming over top of the hot spot (Geology For Kids, 1998-2016). Yellowstone has a large caldera - a depression resulting from the explosion or collapse of the centre of the volcano - which is the product of the large-scale collapse of the crust after three 'ultra-Plinian' or 'super-volcano' eruptions (Volcano Discovery, 2016). These are large explosive events that erupt several hundred to a few thousand cubic kilometres of magma. These eruptions occurred 2.1, 1.3 and 0.6 million years ago (VolcanoDiscovery, 2016). Yellowstone is also the world's largest hydrothermal system. It contains 182 geysers, mud pools and fumaroles (VolcanoDiscovery, 2016). A hydrothermal system is a natural area under the volcano where water gets heated within the Earth's crust due to the magma that is present there. If this volcano erupts, I think that it could cause extreme lahars because of the mud pools and geysers near by and a big explosion because of the fumaroles meeting the magma upon eruption.

Fig. 1 Geyser at Yellowstone

Comparison of the volcanoes

The volcanic hazards that Ruapehu could cause differ from the volcanic hazards that could occur at Merapi because, at Ruapehu, lahars would occur because the ice covering the peak of Ruapehu, would melt and create hot water and mud which would sweep up pyroclastic material and become an intense lahar. In contrast, at Merapi, pyroclastic density currents would be caused, which are clouds of hot gas and ash that can burn and 'suffocate' surroundings. On the other hand, there are a few similarities between the two volcanoes. The natural hazards they both cause are greatly destructive and will destroy anything in their paths and cannot be stopped. When I compare Ruapehu to Yellowstone, I find quite similar natural hazard consequences, as they both would create extremely dangerous lahars. However, I can also see that Ruapehu doesn't have any fumaroles and, therefore, could not create a hazardous gas explosion like Yellowstone. When I compare Merapi and Yellowstone, find great differences between their natural hazardous effects on their surrounding areas. For example, the main consequence of Merapi's eruption would be large pyroclastic density currents, which means that it would give out lots of gas and ash, but the main consequence of Yellowstone's eruption would be a lahar caused by all of the water near Yellowstone and it's hydrothermal system, which would sweep in lots of directions, caused by nearby water heating and pyroclastic materials that for various reasons couldn't create a pyroclastic density current – for instance, the pieces could be too heavy or there might not be enough heat generated.

I think that Mt. Ruapehu is in the most dangerous location compared to Yellowstone and Merapi because many civilians were affected by the 1996 eruption. It is situated in a big national park which would take a lot of the knock-on effects of the eruption as it would flood and most of the lahar would stay in the surrounding area. However, the eruption in 1996 caused many problems such as producing at least seven million tonnes of ash, with 2.3 million tonnes falling on Lake Taupo, which contaminated drinking water, therefore leading to people and animals, particularly in rural areas, being affected (Waikato Council, n.d.). Ash also got into waterways - which reduced the purity of the water and even made some of it acidic – which was certainly not suitable for consumption. Even a few structures and vehicles were damaged by burning ash. All tourism activities were disrupted, having a bad effect on the economy. In addition, dangerous levels of sulphur dioxide (SO2) were found on the mountain and the ash in Tongariro River resulted in damage to the Rangipo Power Station (Waikato Council, n.d.). Ash fall also lowered visibility, disrupting air travel at times, as the plume (which could be seen by satellite) travelled northeast to the coast and across the Bay of Plenty (Waikato Council, n.d.). However, it has been erupting regularly since around 1995 and, therefore, I believe that the effects will be known to the public and won't cause many civilian problems due to the extensive knowledge of the dangers and effects.

Fig. 2 Mount Merapi

Looking at its location, I think that Mt. Merapi is in the least dangerous location because there is a lot of unused space around the volcano. There are small villages and towns nearby but they will be able to evacuate easily, considering there are only a few citizens in the area. In a previous eruption in 2010, lots of citizens were treated for severe burns and respiratory problems after a large pyroclastic cloud covered their villages. Whole areas appeared to be in flames and over 100 people were killed, roughly 75,000 residents had to be evacuated, leaving everything they couldn't take (BBC, 2010). The Indonesian President announced that the government would buy all the cattle from farmers in the affected villages to keep farmers from risking their life when going back to their farm and trying to help their animals during the crisis (BBC, 2010). Many children died, mostly from the village called Argomulyo, which is 18km away from the crater of Merapi (BBC, 2010). However, this was an unusual eruption (eruptions are normally smaller) and I believe that as the volcano erupts so frequently, the citizens in this area will be used to eruptions.

Based on Mt. Yellowstone's location, I believe that it could be very dangerous to the area around it because the eruption would cause dangerous lahars. The lahars would spread to the surrounding lakes (see Fig. 8) and end up flooding large areas of land and affecting many citizens nearby. Although there aren't many citizens too near Yellowstone, the lahar can travel lots of kilometres without the extra water from all the surrounding lakes adding to the devastation. By looking at its eruption history, I can tell that this eruption could be a lot worse than Ruapehu or Merapi because this volcano hasn't erupted since (roughly) 70,000 years ago, which means that lots of magma will be inside the magma chamber of Yellowstone. When it does erupt, many scientists have said that this eruption will be huge. It is not the most dangerous based on location, but because it hasn't erupted for 70,000 years, many people won't know what to do, what is happening or where to go, which compounds the danger.

Looking at the topography of Mt. Ruapehu, I can see that there are not many points where the magma and lahars would collect, meaning that the area affected will be greater in size than if there were many hills and valleys nearby. You can easily see where the lahars will travel down the slopes of Ruapehu as there are very deep 'tracks' which show the direction of the path they take after Ruapehu erupts.

In comparison, from the topography of Mt. Merapi I can see that there are extremely deep valleys on the sides, which were probably created by the pyroclastic density currents and now control where they go. Therefore, I think that the actual pyroclastic density currents will hit the same area almost every time. Also, there is another mountain next to Merapi, which means that the pyroclastic density current cannot travel through it, reducing the area it can hit on that side but increasing the amount of pyroclastic material that will hit the other side of the volcano.

Fig. 3 Mount Ruapehu

When I think about Yellowstone's topography I think that there are a lot of other mountains nearby that would restrict the flow of lahars and in some places, force them to or into bodies of water. The water would react with the pyroclastic material and make ash clouds.

I conclude that Mt. Yellowstone is the most dangerous compared to Mt. Ruapehu and Mt. Merapi because of the lahars created by Yellowstone and then the flooding that they would cause because of the geographical surroundings of Mt. Yellowstone. Mt. Yellowstone's eruption could cause many citizens and animals to drown and completely flood areas of land. This would affect the areas greatly because the areas would need a lot of investment to rebuild and what government would decide to invest in an area that has been flooded because of the consequences of an active volcano's eruption?

It would ruin areas whereas, when Mt. Ruapehu erupted in 1996, a lot of problems occurred, but they were minor and were easily fixed with a small investment. If Mt. Yellowstone erupted, a very major investment would be needed. Mt. Merapi is nowhere near as dangerous as Mt. Yellowstone in my opinion as Mt. Merapi causes severe pyroclastic density currents and pyroclastic clouds but the local area would be easily evacuated compared with the huge area that Mt. Yellowstone could affect.

References

BBC (2010). Dozens die in new Mount Merapi eruption in Indonesia [online]. Available from: http://www.bbc.co.uk/news/world-asia-pacific-11699945/ [Accessed 20/03/2016]

Photo Credits

Figure 1 Geyser at Yellowstone - Yellowstone by jevx (2014) https://www.flickr.com/photos/ericvaughn/16276833918/

Figure 2 Mount Merapi – Dikala cerah sang Merapi by yudha aria (2011) https://www.flickr.com/photos/71960670@N04/6576541443/

Figure 3 Mount Ruapehu – P1100319 by seesix (2011) https://www.flickr.com/photos/c6/6884733182/

Geology for Kids (1998-2016), Volcanic Hot Spots [online]. Available from: http://www.kidsgeo.com/geology-for-kids/0048-volcanic-hot-spots.php/ [Accessed 19.03.2016]

Global Volcanism Program (2007). Report on Merapi (Indonesia) – February 2007. Smithsonian Institution [online]. Available from: http://volcano.si.edu/showreport.cfm?doi=10.5479/si.GVP.BGVN200702-263250 [Accessed 19.03.2016]

Horizons Regional Council (2016). Wangaehu Tangiwai webcam [online]. Available from: http://www.horizons.govt.nz/HRC/media/Data/WebCam/Tangiwai_latest_photo.jpg?width=1280&height=960&ext=.jpg [Accessed 19.03.2016]

Stanmeyer, J. (not dated). Living with volcanoes [online]. Available from: https://mountmerapi.wordpress.com/living-with-volcanoes/ [Accessed 31.03.2016]

VolcanoDiscovery (2015a). Ruapehu Volcano, VolcanoDiscovery [online]. Available from: https://www.volcanodiscovery.com/ruapehu.html [Accessed 19.03.2016]

VolcanoDiscovery (2015b). Merapi volcano [online]. Available from: https://www.volcanodiscovery.com/merapi.html [Accessed 29.03.2016]

VolcanoDiscovery (2015c). Merapi Volcano, VolcanoDiscovery [online]. Available from: https://www.volcanodiscovery.com/merapi.html [Accessed 19.03.2016]

VolcanoDiscovery (2016). Yellowstone volcano [online]. Available from: https://www.volcanodiscovery.com/yellowstone.html [Accessed 29.03.2016]

Waikato Council (not dated). Mount Ruapehu erupts. Waikato Council [online]. Available from: http://www.waikatoregion.govt.nz/Services/Regional-services/Regional-hazards-and-emergency-management/Volcanic-activity/Mount-Ruapehu-erupts/ [Accessed 19.03.2016]

Yellowstone National Park (2016). Watch Geysers Erupt in Yellowstone [online]. Available from: http://www.yellowstonepark.com/watch-geysers-erupt/ [Accessed 19.03.2016]

About the author

A. Johnstone was in Year 9 at The Ferrers School in Higham Ferrers, Northamptonshire, when he did the course with Dr Jordan (he is in Year 10). Dr Jordan is a volcano researcher at the University of Leicester.

PhD Tutor's note

A. Johnstone is a very keen student and I thoroughly enjoyed his input in class. His essay is unique in using an abundance of maps and aerial photos, an idea which he came up with himself! He received a mark of 68 out of 100 (a high 2.1) for this work, which means he is already performing to a good standard at A-levels.

The Perfect Human Virus

Year 9, Key Stage 4

Abbey Grange Academy, Leeds

E. Stewart, supervised by G. Manley, University of Sheffield

Viruses use humans in many ways to replicate themselves. I will explain how viruses use cell organelles to replicate viral particles and the ways viruses use humans to replicate.

Enveloped viruses replicate by binding to proteins on the outside of cells; the particles can only bind to cells with specific proteins on the outside.[1] The cell will try and break the virus down when it enters by creating a vacuole with a high pH; this breaks the particle down into proteins and genetic information, or DNA. The proteins then create a circle around the DNA; the cell allows the proteins out of the vacuole into the cell because it no longer recognises them as a threat. The proteins then release the DNA and the DNA travels through the cytoplasm to the nucleus of the cell, where it is converted into RNA. The RNA leaves the nucleus and travels to the ribosome where the RNA is then converted into amino acids that form new viral proteins. The virus uses the cell to make lots of virus particles, so that the particles can leave the cell and travel to more cells and infect them. When they leave, they take some of the cell's membrane with them, this is called budding.[2] There are other ways a virus can get in and out of a cell to replicate: some replicate by bursting the cell, meaning that only a certain amount of viral particles can be made by each cell.[3]

The Visna Virus that infects sheep can undergo antigenic shift

The immune system for our body has two parts, the innate immune system and the adaptive immune system. The two immune systems both have many ways that they try to stop viruses. One of the methods the innate immune system uses is antiviral molecules that are created to generally target all viruses. A method used by the adaptive immune system is antibodies that are unique to each type of virus and bind only to specific virus particles. Viruses have found ways to evade these two immune systems: proteins that stop parts of the innate immune system, and antigenic shift and drift, which slows the process of making antibodies by changing the viruses' DNA.[4]

In our innate immune system, we have interferons. These tell your cells to turn on their antiviral state and release antiviral molecules.[5] There are many types of antiviral molecules that prevent viruses from replicating in the cell, one is APOBEC3G.[6] APOBEC3G is put into every new virus particle the cell makes, and when these particles infect new cells, it changes the viral genes, mutating the virus so that it will not work, therefore preventing the virus spreading.[7] One virus that has developed a way to stop APOBEC3G is HIV-1 (Human Immunodeficiency Virus). HIV-1 uses a protein called Vif.[8] Vif stops APOBEC3G by preventing the antiviral protein from entering the virus particle, while the particle is budding from the Cell. This allows the particle to replicate in other cells.[9] When a virus evades the innate immune system it means that the virus can continue using the human host to replicate, allowing the virus to spread.

Another way viruses have adapted to be able to evade the adaptive immune system and replicate efficiently in humans is by changing their genetic properties, through antigenic shift or drift. Antigenic drift is where the proteins in a virus, called antigens, genetically mutate. They do this slightly so that B-cells will need to produce new antibodies for the new form of the virus as the body does not recognise the slightly different form.[10][11] The virus will do antigenic drift regularly to keep evading the adaptive immune system.[10]

An example of a virus that can do antigenic drift is HIV.[12] When two types of the HIV virus infect a host cell; parts of each virus join to create a new form of HIV that is unique because it is a random combination of both viruses' genetic information.[13] The change in genetics mean the drugs to treat - but not cure - HIV no longer work, as a new type of antibody will be needed for the new form of the virus. This means that the new form of the virus will be able to replicate more in the human as the designed treatments will not work, allowing the virus to spread to other people and replicate.[14]

Antigenic shift is where a major and sudden change occurs in the virus' antigens, meaning that a completely new antibody will need to be made by the B cells to fight the virus particles.[11][12] In influenza, when antigenic shift occurs some new proteins are created on the virus so that the B-cells have to make new antibodies. In some cases, antigenic shift may be the result of two subtypes of a virus forming a new one, one can be an animal subtype and the other human.[15]

A virus that can antigenic shift is the Visna Virus that infects sheep and causes ovine progressive pneumonia. In 1933 there was an outbreak in Iceland when an imported sheep spread the virus; however, it was not until 1939 when the symptoms first appeared because the virus replicated slowly and mostly in macrophages.[16] In 1977 they tried to give the sheep an inoculation to the virus, however there had been an antigenic shift in the virus, resulting in the inoculation not working in some sheep.[17]

Using a host is another effective way for a virus to use humans to replicate. The virus can use a human to spread to more humans and animals. An example of this is when a mosquito bites an infected human. The mosquito can then catch the virus. The female mosquitos need human blood to lay eggs and reproduce, meaning that as they bite more humans they spread the virus.[18] The infected human will have caught the virus from an infected mosquito biting them; this means that as hosts pass on the virus, more people and mosquitos become infected. The virus can also spread using humans as its way of spreading virus particles to other animals and humans.[19] Being bitten by a mosquito is common in countries with hot climates meaning that it is easy to infect people because it is very hard to know if a mosquito is infected. Although it is not a virus, one disease that uses this method to replicate itself is Malaria. Malaria can only be spread by mosquitoes passing the disease onto humans or vice versa but it cannot be spread from human to human.[20] In Malaria the female Anopheles mosquitos spread the disease to humans.[21] Viruses that are transmitted through hosts are effective, as it can be hard to know if the animal has been infected as sometimes the animal is too small or there are no symptoms. An example where symptoms are visible is Bovine Foot and Mouth Disease, which has obvious symptoms such as frothing at the mouth and weight loss, showing someone that the cow is infected.[22]

Coughing, sneezing, vomiting, diarrhoea and rashes are all symptoms of viruses, but they are also ways viruses use humans to transfer the virus to new hosts. People with a virus can spread viral particles to other people by touching surfaces with infected particles on their hands. For example, an infected person coughs on their hand and then touches a door handle. Someone else then touches that door handle and then their face, catching the virus.[23] Many viruses can live in the air or on a surface for long periods of time, allowing them to pass their particles onto more people and use the new host's cells' organelles to replicate and spread further.[24]

Coughing and sneezing is both a symptom and transmission mechanism of many viruses

Whilst viruses use symptoms such as coughing and sneezing to spread themselves and replicate, some viruses use symptoms that do not affect humans as much, allowing the person to carry on as normal.[25] This allows the virus to have the chance to spread to more people, whereas viruses that have symptoms like vomiting cause the person to stay at home meaning they are less likely to spread the virus further. An example of a virus that is both good and bad at spreading is Croup. Croup is spread by coughing and sneezing however, its symptoms are a strong bark like cough. [26][27] The strong cough informs the host that they are very ill. The host will limit their contact with people more than they would for a normal cold, limiting the virus' ability to spread, but the cough will put Croup particles into the air; increasing the chance that the virus will transfer to a new host.

The immune system uses symptoms as a way to remove infected particles from the body; however, this causes the virus to spread to more people and replicate itself.[25] An example of a virus that uses this default in the immune system to spread and replicate is Rubella, as it is spread through people coughing and sneezing. This is where droplets of mucus and viral particles are released into the air where they can be inhaled by other humans.[28]

The perfect human virus I have created is Big Bertha 3.0 (BB3.0). BB3.0 has small viral particles, containing few viral proteins and DNA, giving it the ability of speed when replicating, but this means that the virus is not deadly.

It does this by binding to BB3.0's double stranded RNA and preventing it from binding to the antiviral proteins OAS1 and PKR. NS1 stops these from activating, letting the virus use the cell to replicate and spread.

The immune system uses symptoms as a way to remove infected particles from the body; however, this causes the virus to spread to more people and replicate itself.[25] An example of a virus that uses this default in the immune system to spread and replicate is Rubella, as it is spread through people coughing and sneezing. This is where droplets of mucus and viral particles are released into the air where they can be inhaled by other humans.[28]

The perfect human virus I have created is Big Bertha 3.0 (BB3.0). BB3.0 has small viral particles, containing few viral proteins and DNA, giving it the ability of speed when replicating, but this means that the virus is not deadly. It doesn't have the infectious proteins that are needed to make a deadlier virus. Ebola is a virus that does have these deadly proteins, meaning that the virus is bigger and therefore slower at spreading [29]. When in a pH vacuole, BB3.0's particles are able to be broken down quickly meaning it can infect cells faster than Ebola[1]. With BB3.0 infecting cells faster, it can spread throughout the body faster without killing the host.

Coughing and sneezing are the symptoms, and also the way Big Bertha 3.0 spreads. When an infected person sneezes or coughs they release virus particles into the air, these can be inhaled by someone else. They live in the air for up to five hours.[32] In comparison Human T-Lymphotropic virus or HTLV-1 is spread by sex, blood to blood contact or breast feeding, these are harder for someone to catch because they are more intimate ways of transferring between hosts. This means that while BB3.0 can be spread to many people at once, HTLV-1 can usually only be spread between one human and another at any time, making BB3.0 better at infecting lots of people.[33] Big Bertha's symptoms allow the infected individual to continue being as active as normal because coughing and sneezing is a common symptom that does not worry people. Whereas, if someone had a more serious symptom, like shingles, they would stay away from people in order to prevent the virus transferring. Shingles has more conspicuous symptoms than coughing and sneezing, meaning the virus has fewer chances to spread.[34]

When someone inhales Big Bertha 3.0 the viruses will split off: half of the particles will stay in the mucus and replicate in nearby cells so that the immune system responds by coughing and sneezing to remove the virus particles, thus spreading the virus further. With these virus particles constantly replicating, it means more can be spread to other people. The other half of the BB3.0 particles try to spread throughout the whole body, making the body more tired and presenting a weaker immune system, as there is less energy. Conversely, Visna Virus in sheep stays mostly in the macrophages, meaning it generally does not spread to many other types cells in the body. This means that Visna Virus spreads less, and more slowly because it is only infecting one type of cell, unlike BB3.0 which infects all cells of the body.[16]

Big Bertha 3.0 has genetically adapted to become able to evade the human adaptive immune system for longer, because of antigenic drift, meaning the body needs new antibodies when the virus re-infects the body and it takes longer for the body's adaptive immune system to fight off the virus, allowing more time for BB3.0 to spread.[10]

In conclusion, I think that Big Bertha 3.0 is the perfect human virus because it is amazing at infecting people. Even though my virus does not have proteins that prevent antiviral molecules, like Tetherin or APOBEC3G, making my virus only effective at combating some antiviral proteins, I think that it is still very effective at infecting lots of people because of its size, speed and inconspicuousness.[7]

References:

[1]- Garoff, H., Hewson, R., and Opstelten, D., E., 1998, Virus Maturation and Budding, Microbiol Mol Biol Rev, 62(4), Abstract only.
[2]- 'Steps of Virus infections', https://www.boundless.com/biology/textbooks/boundless-biology-textbook/viruses-21/virus-infections-and-hosts-137/steps-of-virus-infections-552-11762/ [3]- 'Viral Exit', https://www.boundless.com/microbiology/textbooks/boundless-microbiology-textbook/viruses-9/positive-strand-rna-viruses-in-animals-124/viral-exit-642-1079/ [28th March 2016]
[4]- 'How it works?', http://www.immunologyexplained.co.uk/HowItWorks.aspx [28th March 2016]
[5]- 'Interferons', http://www.encyclopedia.com/topic/interferon.aspx [28th March 2016]
[6]- Brian, M., 'How Your Immune System Works', http://www.saanendoah.com/immunesystem.html
[7]- 'Restriction factors', http://www.shmoop.com/microorganisms-viruses/restriction-factors.html
[8] - 'HIV: Why is the virus so 'successful'?', http://www.bbc.co.uk/news/health-30282147[9]- 'Viral Infectivity factor', https://en.wikipedia.org/wiki/Viral_infectivity_factor [28th March 2016]
[10]- 'What Are Antigenic Drift and Shift?', http://coldflu.about.com/od/coldflutermsae/fl/What-Are-Antigenic-Drift-and-Shift.htm [28th March 2016]
[11]- Alberts, B., et al., 2002, Molecular Biology of the Cell, 4th ed., New York: Garland Science.
[12]- 'Antigenic drift', http://www.britannica.com/science/antigenic-drift [26th March 2016]
[13]- 'Viruses and Evolution', http://www.historyofvaccines.org/content/articles/viruses-and-evolution
[14]- 'HIV treatment', http://www.tht.org.uk/myhiv/HIV-and-you/Your-treatment/HIV-treatment
[15]- 'Influenza Epidemics and Pandemics', http://www.rapidreferenceinfluenza.com/chapter/B978-0-7234-3433-750010-4/aim/introduction [27th March 2016]
[16]- 'Visna Virus', https://en.wikipedia.org/wiki/Visna_virus [26th March 2016]
[17]- Narayan, O., Griffin, D. E., and Chase, J., 1977, Antigenic shift of visna virus in persistently infected sheep, Science, 197(4301), Abstract only.
[18]- Mercola, 'How and Why a Mosquito Bites You?', http://articles.mercola.com/sites/articles/archive/2015/08/08/why-mosquitoes-bite.aspx [26th March 2016]
[19]- 'Mosquito-borne disease', https://en.wikipedia.org/wiki/Mosquito-borne_disease[20]- 'Malaria-Causes', http://www.nhs.uk/Conditions/Malaria/Pages/Causes.aspx
[21]- 'Malaria - the disease', http://www.bupa.co.uk/health-information/directory/m/malaria-disease
[22]- 'Foot-and-Mouth', http://www.thecattlesite.com/diseaseinfo/243/footandmouth/ [23]- 'Office Germs: Viruses Spread Everywhere in Just Hours, Study Shows', http://www.livescience.com/47730-virus-spread-offices.html [27th March 2016]
[24]- James M. Stueckelberg, M. D., 'How long do cold and flu germs stay alive after infected people cough or sneeze?', http://www.mayoclinic.org/diseases-conditions/flu/expert-answers/infectious-disease/faq-20057907 [27th March 2016]
[25]- 'The science-of-sneezing', http://chealth.canoe.com/channel/Cold-and-Flu/Prep-for-cold-flu-season/The-science-of-sneezing [27th March 2016]
[26]- Woods, C. R., 'Patient information: Croup in infants and children (Beyond the Basics)',
[27]- 'Croup - Symptoms', http://www.nhs.uk/Conditions/Croup/Pages/Symptoms.aspx
[28]- 'Rubella', http://www.who.int/mediacentre/factsheets/fs367/en/ [27th March 2016]
[29]- 'Ebola virus disease', http://www.who.int/mediacentre/factsheets/fs103/en/ [1st April 2016]
[30]- Hale, B. G., Randall, R. E., Ortin, J. and Jackson, D., 2008, The multifunctional NS1 protein of influenza A viruses, J. Gen. Virol., 89(10), Abstract only.
[31]- Hatada, E., Saito, S., and Fukuda, R., 1999, Mutant Influenza Viruses with a Defective NS1 Protein Cannot Block the Activation of PKR in Infected Cells, J. Virol., 73(3), 2425-2433.
[32]- Delaney, A., 'Centers for Disease Control (CDC) Removed Information On Coughing and Sneezing from Ebola Q&A', http://www.globalresearch.ca/centers-for-disease-control-cdc-removed-information-on-coughing-and-sneezing-from-ebola-qa/5411083 [1st April 2016]
[33]- 'Human T-lymphotropic virus 1', https://en.wikipedia.org/wiki/Human_T-lymphotropic_virus_1 [34]-

About the authors

E. Stewart is a Year 9 pupil at Abbey Grange Academy in Leeds. G. Manley is a PhD student working on viruses and the immune system at the University of Sheffield.

PhD Tutor's note

Teaching at Abbey Grange Academy was a real joy. I was extremely impressed with all of the pupils I taught, they all picked up complex information really quickly and showed great enthusiasm in every tutorial. All of the viruses the students came up with showed great imagination and creativity. E. came to every tutorial really eager to learn and worked really hard on all her assignments, as you can see from her final essay. Her essay really impressed me, not only because it was written extremely well and contained lots of detail about all the topics we covered, but also because she researched everything independently and found an astonishing amount of additional information, including examples of obscure viruses I hadn't heard of before!

A Zooarchaeological Case Study: analyse an assemblage using zooarchaeological techniques, and produce an interpretation of animal exploitation on the Roman site of Yewden, Buckinghamshire

Year 9, Key Stage 4

Birley Community College, South Yorkshire
L. Dart, supervised by T. Fraser, University of Sheffield

Introduction - Background information for the site

The assemblage examined in this work has been excavated from a site known as the Yewden villa complex, located in the south of the village of Hambleden. Hambleden is in the south of Buckinghamshire, England, and is on the banks of the River Thames. The site was excavated by Alfred Cocks in 1912[4] and through the information gathered from this excavation, we know Yewden was a Roman villa complex, occupied from the early 1st century AD until at least the end of the 4th century AD. Yewden was ideally positioned for links to places like Dorchester, Verulamium (St. Albans) and Silchester [4]. Half a kilometre away from Yewden, was a smaller villa at Mill End, which has not yet been excavated but has been identified. It is thought that the two villas were connected through ownership, however, Mill End is assumed to have had a different function to Yewden. The complex is thought to have been surrounded by a wall, consisting of a main dwelling villa, two other dwelling structures and a fourth small house. The main villa (or first house) was not built until the 4th century and the second house was a living area but later became a workshop; the third house was also a living area [11]. The fourth house was likely to have been a small shrine or temple [11]. Also, in 2010 archaeologists proposed the idea that the site was a brothel, following the discovery of the remains of 97 new born babies [7].

Image 1:
Map to show the location of Hambleden

Methods

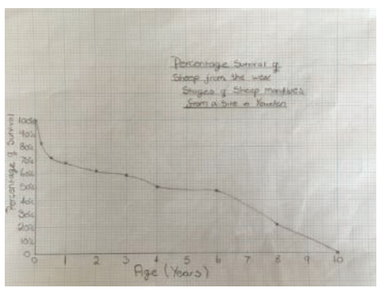
In my investigation, I used various zooarchaeological techniques. I produced a graph by calculating the MNI values for each specie by referring to a table containing the MNE values and choosing the largest statistic for each specie. For example, the largest statistic for cattle was 30, which was for right calcaneum bones; these must have come from 30 different cattle species, therefore we know the minimum number of cattle on site was 30. I repeated this process for each specie then plotted my results as a bar chart. I produced this graph because it is useful to know roughly how many of each specie was on the site as it gives us an insight into the lifestyles of the people who previously lived there, including useful information like which animals were used for meat and which were used for other purposes, like transport and milk.

The next method used was analysing bone fusion to chart the percentage survival of pigs and cattle. There are three bone fusion stages in pigs and four in cattle. Using information that specified when each bone is fused in both species, I worked out the percentage survival for each. This is useful because it allows us to see the percentage of animals that are killed off at a certain age since different bones fuse at different times but this varies from specie to specie. For example, if the remains of a pig are found and the pig's pelvis bone had not been fused, we can assume that it was under 12 months old because the pelvis bone is in fusion stage 1, and would be fused when a pig is between 6 and 12 months old. I conducted this research because it is interesting to see the age at death as it can teach us about what an animal was killed for and can indicate roughly the lifespan of an animal.

The final method used was dental ageing, which I used to produce Graph 1 that shows the percentage survival of sheep. The wear stage of a mandible can indicate the age at death because we know that the more worn a mandible is, the older the animal. I worked out the percentage survival for sheep by looking at a table containing the dental ageing data for sheep mandibles and this had been collated using Payne's work [6]. I used this information to plot a line graph showing the results. As mentioned previously, age at death is useful because it can inform us about the lifestyles of the people who lived on the site.

Wear Stage	Number of Jaws
A	42
B	27
C	10
D	15
E	7
F	22
G	7
H	64
I	51
Total	245

Table 1:
Dental ageing data for sheep mandibles.



Graph 1:
Percentage survival of sheep from the wear stages of sheep mandibles.

Results

Element and Specie Frequencies

My results show the most frequent animal found on the site was sheep, and the least frequent was horse. The most common element found was the sheep left mandible and the elements that weren't found were the sheep pelvis and astragalus, the horse humerus, the dog astragalus and phalanxes 1, 2 and 3. The overall most frequent element found for all species was the left mandible and the least was the left pelvis.

Percentage Survival Rates

Bone fusion analysis shows that cattle survival dropped drastically at the first bone fusion stage, from 100% to 47%, when they were aged between 7-10 months. Then the percentage survival dropped at smaller rates throughout the remaining 3 fusion stages, leaving 36% of cattle in the assemblage living to at least 36 months. The same analysis showed that only 17% of pigs did not reach bone fusion stage 1; the largest drop in survival was at fusion stage 2, as survival dropped by 41%, when the pigs were aged between 14 and 28 months. By fusion stage 3, only 31% of pigs had survived, therefore 31% of the pigs lived to at least 30 months.

The analysis shows that 32% of sheep in the assemblage were aged under one year old when they died. 52% of sheep were under five years old and none of the sheep lived past ten years. The biggest drop in survival was between six and eight years, when percentage survival dropped by 26%. The smallest drop was between four and six years when percentage survival dropped by 3%.

Butchery marks

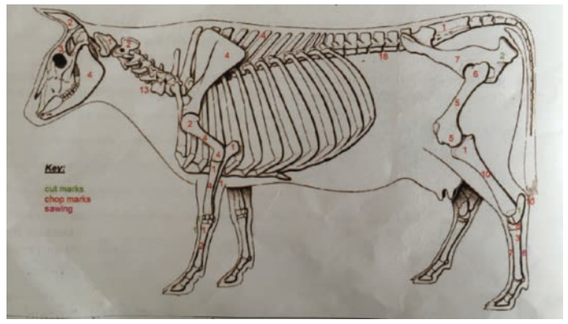


Fig. 1: A diagram showing the distribution of butchery marks found on cattle bones.

Figure 1 shows that there were 138 chop marks and five cut marks found on cattle bones. The distribution of chop marks on cattle were mainly concentrated on the legs, predominately the hind legs. Ten chop marks were found on the hind tibia and ten on the hind tarsal joint. The highest amount of chop marks was found on the lumbar vertebrae, where 16 were located. Also, there were a few chop marks found around the head area and 13 were present on the cervical vertebrae. However, few cut marks were found on the cattle bones; two were found on the ischium and two around the foreleg carpal joint.

There were six chop marks and nine cut marks found on pig bones. The distribution of chop marks on pigs was similar to that of cattle: many were found on the hind legs. The highest amount of chop marks was on the hind ilium, where two were found. One chop mark was observed on the lumbar vertebrae. However, there were more cut marks: three on the hind sacroiliac joint, one on the mandible and five along the foreleg.

Interpretation

I think my results indicate that the cattle on the site were predominantly used for meat, milk, leather and labour. The fact that they were commonly used is supported by the fact that they were the second most frequent species found. Bone fusion analysis shows that only 47% of cattle survived to 10 months; this suggests that there was a high death rate for calves. This could be due to infant mortality, malnutrition, disease, exposure to extreme weather conditions and problems at birth, [1] since the Romans didn't have modern medical technology. The analysis also shows that the percentage survival rate does not decline drastically from age 12 to 35 months. This is because cows are kept alive for milk production and oxen for types of farm labour such as pulling carts and ploughs.[10] Then the survival slowly decreases since the cattle would be slaughtered for meat when they are no longer of any use.[10] In Roman times, cattle were killed aged between four and six.[10] The cattle would not be completely killed off because some needed to be kept for reproduction, as would all the other species. Also, the percentage survival graphs for all species would not start at 100% due to infant mortality. The butchery marks found on cattle bones imply that the legs were a popular source of meat since many chop marks were found at leg joints. Also, the 13 chop marks found on the neck show that the cattle's head was often removed as it was not used. The 16 chop marks on the vertebrae suggest that the hind quarter was commonly used for meat. Finally, the cut marks perhaps were unintentional since the bone could have been caught with a knife when the animal was skinned.

In terms of pig exploitation, I think the Romans on the site did not keep as many pigs compared to sheep and cattle; the MNI for pig was 24 whereas for sheep it was 136. My results suggest that some of the pigs on the site were used for meat, however cattle were a more popular source. The amount of butchery marks found on the bones correspond with this as a few marks were found on pig but many more were found on cattle. The fact that the chop marks were in similar places to cattle shows that pigs had a comparable purpose. Graph 10 also supports this since it shows survival gradually decreased with only 31% surviving past 42 months. Another interpretation would be that the 17% drop in survival at under 6 months was because Romans often ate piglets as a delicacy.[2]

My interpretation of sheep on the site is that they were the most frequent animal, because sheep have the highest MNI out of all species found. However, unlike cattle and pig, I don't think that sheep were kept predominately for meat; the figures fit the wool kill-off pattern graph structure meaning that the sheep were most likely used for wool production. I think the high number of sheep indicates that the Romans depended on wool for their clothing and bedding, as well as a food source since sheep would be killed for meat.

In my opinion, the horses on the site were probably only used for transport, since the MNI for horse was 8. I think this is because the MNI would be much higher if they were used for meat. Also, Romans did not include horse meat in their diet.

Finally, I think that the dogs found on the site were used for hunting wild animals such as hare and deer [3]. Romans thought that dogs were fast and strong and therefore useful in hunting.[8] The data for dogs was similar to that for horses as they both had a small MNI so I think that dogs were also not used for meat.

Taphonomy

My results could have been affected by many taphonomic processes, including hunting, butchery, burrowing, food remains from animals living on the site, choice of excavation site, choice of recovery methods, outbreak of disease and weather conditions. Yewden is close to the River Thames so some bones could have been washed away or brought to the site during a flood. These processes may have affected my results in many ways. For example, maybe not as many pig bones were found because pigs could have been sold and butchered elsewhere. Some animal bones could have been burrowed deep into the ground and archaeologists might not have dug deep enough to find them. These processes show that my results may not be an accurate reflection of the lifestyles of Romans in Britain.

Comparison to the Ipplepen, Devon assemblage

The results for Yewden are similar to that of other Roman villa excavation sites such as Ipplepen in Devon. Like Yewden, it is thought that cattle in Ipplepen were used for labour, milk and then meat.[5] There is no evidence of horse consumption for either sites.[5] Cattle were very frequent on both sites, being the most common in Ipplepen and second in Yewden.[5] Horse and dog were found to be in low frequencies on both sites.[5] This shows that cattle were a useful commodity to the Romans but horse and dog were not. Since the two sets of data agree with each other, this ties Yewden in with a wider Roman picture. However, there is an anomaly: pigs were of a high frequency in Yewden but the data for Ipplepen suggests that there was a much lower number there.

Future work

To make my results and interpretations more accurate I could widen my research, improve my knowledge, expand the excavation site which would result in more bones being found and therefore give more accurate data. I could also compare my results to excavations in Italy to see how Roman lifestyles have been brought over to Britain and compare the sets of data. I could carry out another excavation in Yewden since archaeologists would have improved technology and better knowledge.

Appendices

Image 1: A map to show the location of Hambleden. Source: Wikipedia
Graph 1: Percentage survival of sheep from the wear stages of sheep mandibles.
Table 1: Dental ageing data for sheep mandibles.
Figure 1: A diagram showing the distribution of butchery marks found on cattle bones.

Bibliography

[1] Bruce,B ,Kvasnicka,B and Torell,R. (1999). Perinatal calf weakness. Available: http://www.cabnr.unr.edu/resources/cattlemens/1999/06.htm. Last accessed 1st April 2016.
[2] Barrow,M. (2013). What foods did the Romans eat?. Available: http://www.primaryhomeworkhelp.co.uk/romans/food.html. Last accessed 1st April 2016.
[3] Cross,N. (2006). Roman food in Britain. Available: http://resourcesforhistory.com/Roman_Food_in_Britain.htm. Last accessed 1st April 2016.
[4] Fraser,T. (Unknown). A zooarchaeological analysis of the faunal assemblage from the Roman villa site of Yewden Buckinghamshire. Available: https://www.academia.edu/19607516/A_zooarchaeological_analysis_of_the_faunal_assemblage_from_the_Roman_villa_site_of_Yewden_Buckinghamshire. Last accessed 25th March 2016.
[5] Fraser,T. (Unknown). Report on the Faunal Assemblage from Ipplepen, Devon. Available: https://www.academia.edu/19607656/Report_on_the_Faunal_Assemblage_from_Ipplepen_Devon. Last accessed 1st April 2016.
[6] Payne,S. (1973). Kill Off Patterns in Sheep and Goats: The Mandibles from Asvan Kale. In: Unknown Anatolian Studies,23. Unknown: The British Institute at Ankara. p281-303.
[7] Unknown. (2010). Baby deaths link to Roman 'brothel' in Buckinghamshire. Available: http://www.bbc.co.uk/news/10384460. Last accessed 1st April 2016.
[8] Unknown. (2015). Dogs of Roman Britain. Available: https://en.wikipedia.org/wiki/Dogs_of_Roman_Britain. Last accessed 3rd April 2016.
[9] Unknown. (2016). Hambleden. Available: https://en.wikipedia.org/wiki/Hambleden. Last accessed 1st April 2016.
[10] Unknown. (2007). The Romans in Britain. Available: http://www.iadb.co.uk/romans/main.php?P=5. Last accessed 1st April 2016.
[11] Unknown. (Unknown). Villa reconstructions. Available: http://www.chilternarchaeology.com/villa_reconstructions.htm. Last accessed 1st April 2016.

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PhD Tutor's note
I particularly enjoyed working with Birley Community College as the pupils immediately took to the subject material and all produced excellent final assignments. However, I enjoyed reading this assignment in particular as L. went beyond what I had taught to produce extra results and a more detailed interpretation. She used the zooarchaeological evidence very effectively to produce a very thorough conclusion, which would not be out of place at degree level.

Dual Tasking During Gait, Brain Function and Diagnosis of Neurodegenerative Disease

Year 9, Key Stage 4

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Abstract

Dual-tasking is the simultaneous execution of two tasks; these tasks include two elementary cognitive tasks or secondary tasks. Main cognitive functions, attention and executive function specifically, integrate within dual-tasking due to the utilisation of such functions in secondary tasks. The process of walking integrates a variety of main cognitive functions as this process composes human gait. In people suffering from neurodegenerative diseases, such as Parkinson's disease or Alzheimer's disease, gait changes may occur. Gait abnormalities or a deviation from a normal gait pattern, include: slowness of pace, variability of step length and width and postural instability. During the process of dual-task walking these deviations in gait are revealed. Neurodegenerative diseases are the effects of the onset of neurodegeneration; the loss of neurons. There is no known direct cause for this to occur. Dual-task walking can aid in diagnosing neurodegenerative diseases - Alzheimer's disease in particular. Alzheimer's patients will show marginal difference to gait controls when single-tasking.

However, when subjected to dual-tasking conditions – gait changes become apparent. The importance in improving current diagnosis of Alzheimer's disease is fundamental, as current tests are expensive and can misdiagnose patients. Once current diagnosis is improved to the point where early diagnosis can be given, the pathologies of such disorders will be recognised in a shorter time span. This can improve a patient's quality of life as well as that of those around them. Gait analysis through dual-tasking is becoming an increasingly important subject to study for researchers, professionals and neurologists alike.

Essay

Dual-tasking is the simultaneous execution of two tasks, (McArdle, 2015) these tasks include two elementary cognitive tasks (ECT), or secondary tasks. ECTs are basic tasks which require a small number of mental processes (Caroll, 1993). Dual-tasking is the combination of two ECTs. Main cognitive functions (MCFs), integrate with dual-tasking due to the utilisation of such functions in ECTs – specifically in executive function (EF) and attention. EF includes complex mental processes such as: problem solving, planning, and dual tasking (Zelazo, 2002). Attention encompasses mental processes: the ability to concentrate on people, actions, objects, and impulsive action restraint. Gait relies specifically on attention and EF. Gait is a person's manner of walking. It refers to the repetitive movement of human limbs to acquire locomotion. Changes in gait – a 'normal' gait pattern deviation or gait dysfunctions (Sudarsky, 2012) - can be revealed through dual-task walking (Kressig, Herrmann, Grandjean, Michel, Beauchet, 2008). Postural instability, variability of step length and width, shown in a study by Borges et al (2015), are aspects of gait abnormalities. This can show an impact in areas of the brain, which control gait, or MCFs. This essay will aim to discuss dual-tasking, its effect on gait and the brain, and argue that dual-tasking is fundamental for the early diagnosis of neurodegenerative disease; this essay will aim to discuss the importance of diagnosing early for Alzheimer's disease (AD).

How can dual-tasking change gait?

Dual-tasking can influence changes in gait, for example: bradykinesia (slowness of movement) and postural instability are results of changes in gait. These symptoms are shown in Parkinsonian gait dysfunction. Parkinson's disease (PD) is a progressive neurodegenerative disease (Thomas, Beal 2007). The pathology of a progressive disorder is that it gradually worsens as a patient grows older. In addition, as the disease develops – multiple neurons inside the patient's brain will eventually perish as connections and signals are lost between each neuron. When this happens, patients gradually lose one main cognitive function and in turn most neurological processes. Processes that are affected include motor control in gait, leading to difficulties in walking. This process is referred to as 'neurodegeneration' (Ontario Brain Institute, n.d.) and there is no known prevention or cause of the process. Neurodegeneration can affect a patient's ability to dual-task as parts of the brain that control EF or attention may be affected due to loss of neuronal signals necessary for the execution of dual-tasking and holding attention.

Furthermore, motoric gait dysfunction becomes apparent in a PD patient through an asymmetrical, or irregular, gait. An asymmetrical gait (or antalgic gait) is shown through 'shuffling' movements during walking. 'Shuffling' movement is quick small steps, where a PD patient acclaims a tendency to run or quicken their pace. This is known as 'festination' (Giladi, Shabtai, Rozenberg, Shabtai, 2001). The result of a festinating gait and motor blocks (a form of akinesia or loss of movement) is that patients are subject to frequent falls (Giladi, McDermott, Fahn, et al. 2001). Falls are common in the later stages of the disease - however, it is uncertain when this stage occurs in every patient.

The above symptoms of gait dysfunction are also present when a patient is single-tasking. This refers to focusing on one task until completed, rather than sharing attention to another task and simultaneously trying to complete both (Jayne, n.d.). However, patients inflicted with PD, subjected to dual-task conditions, are seen to have an increase in festinating steps and a worsened state of bradykinesia (Brauer, et al. 2011). Furthermore, patients with the disorder, while dual tasking, demonstrate increased variability in step length and width, leading to asymmetry in gait and frequent motor blocks compared to walking alone (Brauer, et al. 2011). Therefore, it can be argued that gait in PD worsens once a patient is subject to dual-tasking conditions.

Dual tasking can reveal issues with gait

In addition, PD is only one of several neurodegenerative diseases that have effects on gait. AD is another progressive neurological disorder, which leads to changes in gait as the disease progresses. Dementia related gait dysfunctions, include a decrease in gait pace. This is caused by stride length decreasing, which then affects variability. However, motor disorders are found in the later development stages of dementia (Beauchet, et al. 2008) and therefore, may not be noticed once mild cognitive impairment (MCI) becomes apparent. MCI is often seen as a pre-dementia syndrome; it is also a transition stage between a normal cognitive state and dementia in older adults (Petersen, 2004). The transition between a normal cognitive state and dementia is defined by the loss of one main cognitive function (McArdle, 2015). However, when MCI becomes apparent and is diagnosed, the dementia is already in its later stages of development. This means that it has already become severe by the time of diagnosis and the patient's quality of life may already have been affected.

Finally, AD patients – when subjected to dual-tasking - reveal changes in gait. Patients with any dementia or walking abnormality, walking without any interference from a secondary ECT show marginal change compared to the normal gait for their age group (Muir, et al. 2011). Once a patient combines a secondary ECT with their normal gait – symptoms are revealed. This helps to classify disorders. Gait abnormalities that dual-tasking reveals for patients with AD or other dementias include: slowness of pace, variability of step length and width (Borges et al 2015).

What can the discussed changes tell us about brain function?

Dual-tasking has an impact on areas of the brain, which control EF and attention. Both MCFs are closely linked as both have integration in dual-tasking. EF is controlled, primarily, in the prefrontal regions of the frontal lobe (Kringelbach, 2005) with neuronal connections to brain stem areas, such as the cerebellum and medulla oblongata. Attention is controlled in the four lobes of the cerebral cortex (parietal, occipital, temporal, and frontal). It encompasses many regions of the brain and is fundamental in most daily tasks. An example of the daily utilisation of MCFs is walking. The act of walking is a task that is performed daily, which involves complex processes. These processes require the ongoing comprehension of visual information, information from the muscles and other organs and required sensory information. One's joint positions must be controlled, feedback from the terrain one is walking on should be understood and the environment one is present in must be comprehended (Burskens, Bock, 2012). Multiple areas of the brain are utilised in receiving and comprehending sensory information - the occipital lobe and parietal lobe, for example. Additionally, everyday life integrates walking with other activities – or ECTs (secondary tasks), for example talking or using a mobile phone. When an ECT is integrated with the act of human locomotion it is termed 'dual-task walking' (Kressig, Herrmann, Grandjean, Michel, Beauchet, 2008).

Furthermore, dual-tasking reveals severity levels and onsets of mild cognitive impairment (MCI) syndrome. This syndrome is commonly diagnosed in later stages of dementia patients. MCI is separated into two types: amnestic MCI (a-MCI) and non-amnestic MCI (na-MCI.) A-MCI is a condition where patients have memory inhibitions more severe than normal for their age and education – however, it is not serious enough to affect daily living ('Amnestic Mild Cognitive Impairment (MCI) Doubles Risk of Death 2012.) A-MCI is not considered to be a type of dementia. Rather, a patient with either type of MCI is more likely to develop a type of dementia. Na-MCI includes difficulties with EF – problems with dual-tasking, judgement, and/or organising. Therefore, researchers and practitioners can infer that: if a patient has problems with dual-tasking, then it could be a sign of na-MCI. This then leads to an earlier diagnosis of dementias (like AD.) In a study by Petersen et al (2013), an estimated fifteen percent of the population, between ages 70 and 90, experience MCI. From this percentage of individuals, research studies show that 10 to 15 percent of elderly cohorts who had a-MCI developed dementia - usually AD.

As previously stated, dual-tasking reveals changes in gait. This change shows an impact in areas of the brain which control gait or motor skills. This enables researchers and practitioners to infer brain function from dual-tasking and if it shows a risk for clinical disorders. In addition, gait requires a collaboration of MCFs – attention and EF, specifically. Human gait also requires the motor processing functions of areas of the brain, such as the motor cortex, basal ganglia and cerebellum. As these areas of the brain begin to be affected by neurodegeneration at the onset of any neurodegenerative disease, this leads to a loss in MCFs and to a decline in gait (Buracchio, Dodge, Howleson, Wasserman, Kaye, 2010.)

How is dual-tasking fundamental to aid the diagnosis of AD?

As mentioned previously, gait changes in AD cannot be identified through single-task walking alone. This is because the patient, while single-task walking, will display a marginal difference compared to control or a 'normal' gait pattern (Bridenbaugh & Kressig, 2014.) Dual-task walking can reveal these changes, which then can lead to early diagnosis of the disease. Gait abnormalities and changes are markers for the onset of the development of AD (Strobel, n.d.) Once these dysfunctions in gait are found, it is easier to diagnose the correct type of dementia.

There are many types of dementia – examples include Dementia with Lewy Bodies and AD, amongst others. ('Types of Dementia' n.d.) The symptoms of each dementia are similar to one another – therefore, this makes it difficult to determine which dementia a patient is afflicted with. Furthermore, other neurodegenerative diseases display changes and abnormalities during the process of walking as well (Snijders, Warrenburg, Giladi, Bloem, 2007.) Dual-task walking could also aid professionals and doctors to differentiate a dementia from disorders like stroke or PD due to the patterns of gait in a patient's walk. Consequently, this would reduce the risk of misdiagnosing a patient.

Furthermore, the attributes of AD are similar to depression. When subjected to dual-task conditions, individuals with Alzheimer's performed significantly worse than people with depression and healthy older cohorts (Kaschel, Logie, Kazén, Sala, 2009.) This further suggests that dual-task analysis will aid in diagnosing for AD as it can differentiate between neurodegenerative disorders and mental illnesses.

Why is it important to improve early diagnosis of neurodegenerative diseases?

It is fundamentally important to improve the current diagnosis of neurodegenerative diseases to enable patients to be diagnosed earlier. Although this is a strived for idea it may take a considerable amount of time before changes will start to impact the lives of patients. Improving early diagnosis of neurodegenerative diseases may improve the quality of life a patient has while afflicted with their disorder. It may be possible to give them, their family or their acquaintances advice early in the onset of the patient's disease. This could help in supporting the patient during their life with the disorder and aid them in coping with a neurodegenerative disease.

One can argue that the diagnosis of such diseases must be improved as soon as possible. Annually, a large amount

of money is spent on the treatment and diagnosis of neurodegenerative diseases – AD in particular costing £26 billion per annum ('Dementia now costs £26 billion a year' 2014). The diagnosis of these disorders is done through paper tests -the 'Self-Administered Gerocognitive Exam' or SAGE test for AD patients (Scharre, n.d.) - where a patient may become agitated while taking it and not produce an accurate result. Furthermore, these tests can be perceived as unreliable – as information may correlate too closely to other dementias and misdiagnosis may occur. These tests also take a considerable amount of time to complete.
Therefore, it can be argued that gait analysis with dual-tasking is more suitable and produces more accurate results compared to paper tests used to diagnose AD. However, professionals must consider that every individual has their own specific gait pattern and stages of neurological development. Neurologists and researchers alike must strive to improve diagnosis for the benefit of current and future patients.

Conclusion

Dual-tasking plays a fundamental role in the study of neurology and the diagnosis of neurodegenerative diseases such as PD and AD. When utilised to diagnose gait, symptoms and abnormalities can be discovered to signal the onset of disorders months before the disorder becomes severe (Muir, et al. 2011). The brain is affected throughout the duration of a neurological disease: a process named neurodegeneration begins to develop. Through this process, parts of the brain become unresponsive and eventually will cease to carry out their respective activities, or MCFs, leaving the patient without the ability to do daily tasks, including speaking and walking. In some cases the quality of an AD patient's life is jeopardised due to the inability to accurately diagnose the correct dementia, which could then affect those around them. It is fundamentally important that more research is carried out into dual-tasking as it can aid the economy in using less paper tests and can serve as an accurate way to diagnose patients earlier.

References

Amboni, M., Barone, P., & Hausdorff, J. M. (2013). Cognitive contributions to gait and falls: Evidence and implications. Movement Disorders Mov Disord., 28(11), 1520–1533. doi:10.1002/mds.25674
Amnestic Mild Cognitive Impairment (MCI) Doubles Risk of Death | Albert Einstein College of Medicine. (2012). Retrieved April 25, 2016, from http://www.einstein.yu.edu/news/releases/810/amnestic-mild-cognitive-impairment-mci-peter -risk-of-death/
Beauchet, O., Allali, G., Berrut, G., Hommet, C., Dubost, V., & Assal, F. (2008). Gait analysis in demented subjects: Interests and perspectives. NDT Neuropsychiatric Disease and Treatment, 155. doi:10.2147/ndt.s2070
Beurskens, R., & Bock, O. (2012). Age-Related Deficits of Dual-Task Walking: A Review. Neural Plasticity, 2012, 1-9. doi:10.1155/2012/131608
Borges, V., Aquino, C., Gazzola, J., Doná, F., Silva, S., Ganança, F., . . . Ferraz, H. (2015). Changes in postural control in patients with Parkinson's disease: A posturographic study. Physiotherapy. doi:10.1016/j.physio.2015.08.009
Brauer, S. G., Woollacott, M. H., Lamont, R., Clewett, S., O'Sullivan, J., Silburn, P., . . . Morris, M. E. (2011). Single and dual task gait training in people with Parkinson's Disease: A protocol for a randomised controlled trial. BMC Neurology BMC Neurol, 11(1), 90. doi:10.1186/1471-2377-11-90
Bridenbaugh, S. A., & Kressig, R. W. (2011). Laboratory Review: The Role of Gait Analysis in Seniors' Mobility and Fall Prevention. Gerontology, 57(3), 256-264. doi:10.1159/000322194
Bridenbaugh, S., & Kressig, R. (2014). Quantitative Gait Disturbances in Older Adults with Cognitive Impairments. CPD Current Pharmaceutical Design, 20(19), 3165-3172. doi:10.2174/1381612811319666 0688
Bridenbaugh, S., Kressig, R. (2010) Laboratory Review: The Role of Gait Analysis in Seniors' Mobility and Fall Prevention Bridging the Gap between Clinical and Behavioural Gerontology Part I: Promoting Late-Life Mobility and Independence DOI: 10.1159/000322194
Buracchio, T., Dodge, H. H., Howieson, D., Wasserman, D., & Kaye, J. (2010). The Trajectory of Gait Speed Preceding Mild Cognitive Impairment. Arch Neurol Archives of Neurology, 67(8). doi:10.1001/archneurol.2010.159
Carroll, J. B. (1993). Psychomotor Abilities. A Survey of Factor-analytic Studies Human Cognitive Abilities, 532-541. doi:10.1017/cbo9780511571312.014
Dementia now costs £26 billion a year. (2014). Retrieved April 26, 2016, from http://www.lse.ac.uk/newsAndMedia/news/archives/2014/09/Dementia.aspx
Giladi, N., Mcdermott, M. P., Fahn, S., Przedborski, S., Jankovic, J., Stern, M., & Tanner, C. (2001). Freezing of gait in PD: Prospective assessment in the DATATOP cohort. Neurology, 56(12), 1712-1721. doi:10.1212/wnl.56.12.1712
Giladi, N., Shabtai, H., Rozenberg, E., & Shabtai, E. (2001). Gait festination in Parkinson's disease. Parkinsonism & Related Disorders, 7(2), 135-138. doi:10.1016/s1353-8020(00)00030-4
Hausdorff, J. M., Doniger, G. M., Springer, S., Yogev, G., Simon, E. S., & Giladi, N. (2006). A Common Cognitive Profile in Elderly Fallers and in Patients with Parkinson's Disease: The Prominence of Impaired Executive Function and Attention. Experimental Aging Research, 32(4), 411-429. doi:10.1080/03610730600875817
Jayne, A. K. (n.d.). Single-Tasking vs. Multitasking. Retrieved April 26, 2016, from http://smallbusiness.chron.com/singletasking-vs-multitasking-32781.html
Kaschel, R., Logie, R. H., Kazén, M., & Sala, S. D. (2009). Alzheimer's disease, but not ageing or depression, affects dual-tasking. Journal of Neurology J Neurol, 256(11), 1860-1868. doi:10.1007/s00415-009-5210-7
Kressig, R. W., Herrmann, F. R., Grandjean, R., Michel, J., & Beauchet, O. (2008). Gait variability while dual-tasking: Fall predictor in older inpatients? Aging Clinical and Experimental Research Aging Clin Exp Res, 20(2), 123-130. doi:10.1007/bf03324758
Kringelbach, M. L. (2005). The human orbitofrontal cortex: Linking reward to hedonic experience. Nature Reviews Neuroscience Nat Rev Neurosci, 6(9), 691-702. doi:10.1038/nrn1747
Kringelbach, M. L. (2005). The human orbitofrontal cortex: Linking reward to hedonic experience. Nature Reviews Neuroscience Nat Rev Neurosci, 6(9), 691-702. doi:10.1038/nrn1747
McArdle, R (2016) Alzheimer's Disease and Mild Cognitive Impairment. Powerpoint slides: 8, 9, 15, 17.
McArdle, R. (2016) Watch Your Step: How Does The Brain Influence Walking? Powerpoint slides: 17, 19, 21, 23.
Montero-Odasso, M., Verghese, J., Beauchet, O., & Hausdorff, J. M. (2012). Gait and Cognition: A Complementary Approach to Understanding Brain Function and the Risk of Falling. Journal of the American Geriatrics Society J Am Geriatr Soc. doi:10.1111/j.1532-5415.2012.04209.x
Muir, S. W., Speechley, M., Wells, J., Borrie, M., Gopaul, K., & Montero-Odasso, M. (2012). Gait assessment in mild cognitive impairment and Alzheimer's disease: The effect of dual-task challenges across the cognitive spectrum. Gait & Posture, 35(1), 96-100. doi:10.1016/j.gaitpost.2011.08.014
Ontario Brain Institute. (n.d.). Retrieved April 26, 2016, from http://www.braininstitute.ca/homepage
Petersen, R. C. (2004). Mild cognitive impairment as a diagnostic entity. J Intern Med Journal of Internal Medicine, 256(3), 183-194. doi:10.1111/j.1365-2796.2004.01388.x
Petersen, R. C., Aisen, P., Boeve, B. F., Geda, Y. E., Ivnik, R. J., Knopman, D. S., . . . Jack, C. R. (2013). Criteria for mild cognitive impairment due to alzheimer's disease in the community. Annals of Neurology Ann Neurol. doi:10.1002/ana.23931
Pinel, P.J. (2011) Chapter 10: Brain Damage and Neuroplasticity. Biopsychology (8th Ed) pg. 251-252
Sauer, A. (2014). What Your Walk Says About Your Risk for Alzheimer's. Retrieved April 25, 2016, from http://www.alzheimers.net/9-2-14-alzheimers-gait
Schäfer, S., Huxhold, O., & Lindenberger, U. (2006). Healthy mind in healthy body? A review of sensorimotor–cognitive interdependencies in old age. Eur. Rev. Aging.Phys. Act. European Review of Aging and Physical Activity, 3(2), 45-54. doi:10.1007/s11556-006-0007-5
Scharre, D. (n.d.). SAGE: A Test to Detect Signs of Alzheimer's and Dementia. Retrieved April 26, 2016, from https://wexnermedical.osu.edu/brain-spine-neuro/memory-disorders/sage
Siddiqui, S. V., Chatterjee, U., Kumar, D., Siddiqui, A., & Goyal, N. (2008). Neuropsychology of prefrontal cortex, 50(3). Indian J Psychiatry. doi:10.4103/0019-5545.43634
Snijders, A. H., Warrenburg, B. P., Giladi, N., & Bloem, B. R. (2007). Neurological gait disorders in elderly people: Clinical approach and classification. The Lancet Neurology, 6(1), 63-74. doi:10.1016/s1474-4422(06)70678-0
Strobel, G. (n.d.). Diagnosis of Early Onset Alzheimer Disease | ALZFORUM. Retrieved April 26, 2016, from http://www.alzforum.org/early-onset-familial-ad/diagnosisgenetics/diagnosis-early-onset-alzheimer-disease
Sudarsky, L., & Viswanathan, A. (2012). Balance and gait problems in the elderly. Handbook of Clinical Neurology Ataxic Disorders, 623-634. doi:10.1016/b978-0-444-51892-7.00045-0
Thomas, B., & Beal, M. F. (2007). Parkinson's disease. doi:10.1093/hmg/ddm159
Types of dementia. (n.d.). Retrieved April 26, 2016, from https://www.alzheimers.org.uk/typesofdementia/
Yogev-Seligmann, G., Hausdorff, J. M., & Giladi, N. (2008). The role of executive function and attention in gait. Movement Disorders Mov Disord., 23(3), 329-342. doi:10.1002/mds.21720
Zelazo, P. D., & Bauer, P. J. (2002). Journal of cognition and development. Mahwah, N.J: Lawrence Erlbaum Associates.

Programme Officer's note

I am so impressed by this article. The abstract is clear and concise, and the vocabulary used throughout is of the highest level. The sources used within the text are carefully referenced, showing the greatest of respect for the ideas of others. The author has achieved a rare balance – sounding like an expert, whilst explaining the concepts clearly so that they are understandable for all. Really well done!
—Dr N. Day, Programme Officer for the North East and Yorkshire, The Scholars Programme

Is Ebola the Next International Pandemic?

Year 9, Key Stage 4

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Introduction

Ebola is a severe haemorrhagic fever caused by viruses belonging to the genus Ebolavirus in the family Filoviridae, as classified by the International Committee on Taxonomy of Viruses.[1] Ebola infects eukaryotic cells. Viruses are complicated 'packages' of protein, carbohydrates, lipids and nucleic acids (DNA or RNA). As all viruses are pathogenic, they must invade a living cell to multiply and spread. The virus binds to the host cell surface using its envelope proteins and then injects its genome into the host cell. The virus takes over the host cell and uses it to replicate its genes. Viruses hijack cellular 'machinery' to replicate their own genetic information and create viral copies. The new viruses kill the host cell, causing harm to the whole host, which we observe as symptoms. The symptoms of Ebola are: fever, intense weakness, headache, sore throat, muscle pain, vomiting, diarrhoea, internal & external bleeding, rash, and impaired kidney and liver function. Since viruses can only multiply in host cells, it is not as easy for them to spread as it is for bacteria. However, they can still be very contagious. They can spread by one of three main ways: bloodstream, airways and other bodily fluid. Ebola could be transferred to a human through human-to-human transmission or direct contact with the blood, secretions, organs or other bodily fluids of infected animals, as well as indirect contact with environments contaminated with such fluids. The first cases of Ebola occured between 1st September and 24th October 1976, in Northern Zaire, where 318 people were diagnosed. The outbreak was centred in the Bumba Zone of the Equateur Region and most of the cases were within a diameter of 140km of Yambuku. The first case was first mistaken for malaria. The patients were even given chloroquine injection at Yambuku Mission Hospital. The patients at Yambuku Mission Hospital who received injections contracted Ebola haemorrhagic fever, now called Ebola virus disease (EVD) because the needles used were not sterile.[2] Until now the virus has spread considerably but is it the next international pandemic? Ebola is currently endemic to Western Africa, meaning it is found in a specific area. If a disease is prevalent across a whole country or the world then it is classified as a pandemic e.g. Tuberculosis, Polio and Influenza.

Ebola prevention in less developed regions and bioethics

Another West African country, Mali, became the sixth country to be affected by the Ebola outbreak. Security and quarantine actions made so far by Ebola-hit African countries prove that, in the region's less developed countries, the Ebola virus is still hard to contain, prompting fears of an uncontrollable spread to other continents. Ebola virus continues to spread and Asia is at great risk [3]. People in Asia are under greater risk from the Ebola virus because billions of people live in poverty and public health systems are often very weak as there are a lot of less developed countries. The source from which this information was taken is not reliable because it's not a well-known site and does not include the name of its author. Also it has not been peer reviewed. However we can trust some facts as they have also been mentioned in the fourth source.

Barack Obama has said that a global effort is needed to stop the spread of Ebola and blamed it on weak and overwhelmed health systems in West Africa. He said that the disease, which has killed more than 11,000 people in Liberia, Sierra Leone and Guinea, can be controlled and stopped with the right resources. One more case was confirmed in Nigeria. Some African counties have weak public health systems. They weren't able to identify and isolate cases quick enough and as a consequence it spread more rapidly. The US is working with European countries and the World Health Organisation (WHO) to respond to the crisis. A drug, which is being tested on an American health worker who contracted Ebola and has flown back to the US, could be distributed in West Africa. [4] Although one limitation is that there have not been many cases of Ebola in the US to test the health services there. If the affected countries had stronger public health systems, then the disease would have been identified and stopped before it quickly spread, killing a lot of people. However, it's quite obvious why the public health systems are weak. The affected West African countries are LEDCs, which means that governments do not have enough money to supply better medical care. The information contained in this source is more reliable because Chris McGreal, who worked for British Broadcasting Corporation (BBC) before, is a senior writer for Guardian US. The article has a lot of key points, which are based on an interview with an important and powerful figure. However, Obama's speech could be politically biased, to try and make the US sound helpful.

On the other hand, the fifth article contradicts the fourth article as it says, "the international community has made almost zero response. The leaders in the West are more interested in protecting their own countries and closing airlines than helping contain the crisis that has now claimed more than 1200 lives." De la Vigne said, "the solution is not that complicated but needs political will to do so." Dr Gabriel Fitzpatrick said," if this Ebola outbreak happened in a western community in London then you'd get a few cases and that would be it. The main objective here is not to increase the person's chance of survival; it's to contain the spread".[5] This shows that nothing like what Obama said has been done to stop the disease from spreading. The long incubation period of 2 to 21 days allows people to leave the infected countries without the virus being detected, which can make a pandemic much more likely. By closing all of the airlines the pandemic can be prevented. In addition, the government can work together to stop Ebola from spreading. This is not a very reliable source because it lacks factual information and does not have many opinions from medical professionals. I immediately recognised what Dr Gabriel Fitzpatrick said as incorrect because even though England is an MEDC, if Ebola outbreak happened in London then it would not stop that quickly because no cure has been found for the disease yet. It is also biased because all of the information is negative (against the government) whereas the fourth source consists of both positive and negative consequences. I think the fourth source is more reliable.

Economy

The virus is having a shocking impact on the economies of Guinea, Liberia and Sierra Leone. Sierra Leone's Agriculture Minister Joseph Sam Sesay told the BBC that the economy has been deflated by 30% because of Ebola. The agricultural sector is the most impacted in terms of Ebola because the majority of the people of Sierra Leone, about 66%, are farmers.[6] This shows that not much agriculture is happening, which will create food shortages and pressures on food prices. If there is a shortage of food then it can affect the health of locals because people will not get enough nutrition. Less nutrition can lead to more illnesses and possible deaths, which means that there will be an increase in death rate. It also means that farmers (producers) cannot sell food, which will lead to no cash-inflows. This can affect all three West African nations because Guinea, Liberia and Sierra Leone are already poor countries, but the Ebola outbreak could make them even poorer. This means that their Human Development Index (HDI) rate will decrease. Also, they will not have money to improve their public health systems meaning that an Ebola infection could become inevitable and spread further, resulting in a pandemic. The BBC is a reliable web source because it is a site with a good reputation. It includes the name of the author: Richard Hamilton. Only members of the BBC News team can edit the content. This source has been peer reviewed by an expert in the field before publishing it to the public, which makes it very reliable. This is an advantage because it's a scientific article with the aim of being scientifically accurate; if the source is peer reviewed then it means the facts are more accurate and therefore we can trust this source.

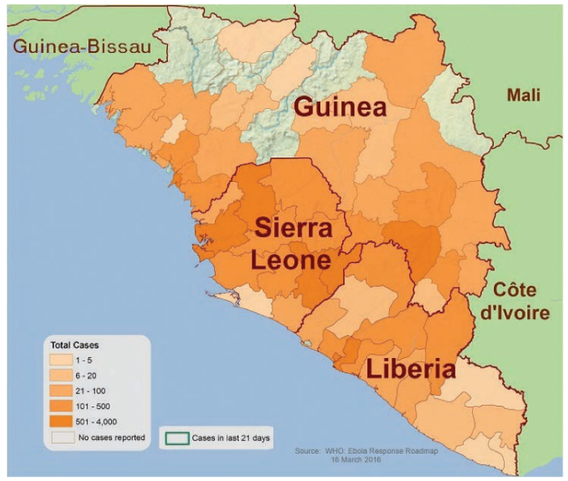
Wildlife

People are at risk from a range of deadly diseases. A new re-emerging virus capable of killing animals can also be the first indication of a human pandemic such as human immunodeficiency virus (HIV), acquired immune deficiency syndrome (AIDS), severe acute respiratory syndrome (SARS), Middle East respiratory syndrome (MERS) and Ebola. All of these diseases spread from animals to humans. Pathogens infecting wildlife are twice as likely to jump over to humans as those without wildlife hosts.[7] This information came from an article in National Geographic magazine, which is a reliable source because it has been around for over 100 years and has some of the world's top scientists, researchers, and photographers working for them. They're widely recognised by everyone, have good publications and accurate facts.

Ebola travelled to Asia inside wild African animals. There have been rumours that wealthy people in the Philippines who own private estates in the rainforest have been importing African animals illegally and releasing them into the Philippine jungle for hunting. If Ebola is present in African animals then it might have travelled to the Philippines.[8] This is not very reliable information because there is no robust evidence to support this claim.

Immunity, ethics and vaccination

The immune system is our defence against infections. The first lines of defence are physical barriers e.g. skin. Viruses must bypass these through air/water/food/bodily fluids to enter the body and infect us. When Ebola enters the body, it targets dendritic cells in the immune system. Normally, when a virus is detected, these cells tell other cells to produce antibodies via major histocompatibility complex (MHC) signalling. Ebola prevents that signal getting out. As far as the immune system knows, everything inside the body is fine. Ebola then begins replicating rapidly. It then spreads into the bloodstream, infecting the whole body. Cells start to break up and die in huge numbers. This finally triggers the immune system, which reacts too aggressively and launches a cytokine storm. Pathogens contain chemicals that are foreign to the host body called antigens. Antibodies are produced by the host immune system to help detect antigens and destroy the invading pathogen. Antibodies have a 'chemical fit' to a certain antigen. Antibodies (made by B cells) bind to antigens. Antibodies act like a 'tag' alerting the killer cells, T cells and macrophages to attack and destroy the foreign material e.g. antigen/cell.



Map showing incidence of Ebola Virus in 2014 West Africa breakout

Several vaccines are being tested but none are available for clinical use. It has been difficult to make a successful Ebola vaccination because of different strains of virus, instability of the viral genome and the fact that it is dangerous to work with. Also, obtaining viral samples is difficult from remote areas of Africa. In August 2014, a group of medical ethicists met to discuss whether an experimental drug should be made more widely available to those suffering from Ebola. The only way to find out if these treatments really work is to test them in this epidemic. However, this is a controversial decision. The point of ethics is to ensure the safety of people and the soundness of the research. Worrying about the safety of people during an epidemic that has no cure may seem unnecessary, but considering that Ebola is not, in fact, 100% fatal (mortality rates vary from 50%-90% depending on the strain) means that the apparently compassionate supply of experimental therapies could do more harm than good.[10] This source is reliable because Dan O'Connor is Head of Humanities and Social Science at the Wellcome Trust. He directs the trust's funding of bioethics research and was formerly a member of faculty at the Johns Hopkins Berman Institute of Bioethics. This means the writing is likely accurate and the source has been peer reviewed.

Vaccines are a way to show your body a virus or pathogenic bacteria so it can remember and fight it in the future if you become infected. Vaccines can contain dead or live fragments of virus which are injected into the body. The injection triggers the immune system to start making antibodies against the virus. An Ebola vaccine is in development by the Vaccine Research Centre (VRC). It contains no infectious material and cannot replicate inside cells. However, it encourages an immune response and so could be used to treat or prevent Ebola. Plans are underway to begin phase 3 trials after the recent success of phase 2. A recent test in Guinea proved 100% effective and is likely to bring the West African epidemic to an end.[11] The WHO believes however that the percentage could change between 75% and 100% as they receive more information.[12] During the tests, 4123 patients were given the new treatment followed by another 3528 three weeks later.[13] At the time of writing the vaccine has still not been licenced.[14]

Conclusion

In conclusion, it is still difficult to say whether Ebola virus will be the next pandemic or not. Better health care, education and economy in Western countries can prevent the pandemic. The epidemic is still located in West Africa and is not spreading at an extremely high rate as it is still a blood borne infection. No licenced vaccine for EVD is available. Patients can frequently dehydrate and will require oral hydration with solutions containing electrolytes or intravenous fluids to relieve symptoms. It is important to be hospitalised for intensive supportive care and to be quarantined. Several vaccines are being tested and new drug therapies are being evaluated. If a cure isn't found, if the virus spreads to more countries or is found on another continent, or if the virus mutates from blood borne to air borne, then Ebola could definitely be the next international pandemic, since even Western healthcare systems may be unable to cope with the spread of infection.

Bibliography

[1] The 2014 Ebola virus outbreak in West Africa, G. Gather: The journal of General Virology, 2014.
[2] "Ebola virus disease", http://www.who.int/mediacentre/factsheets/fs103/en/[30/03/2016]
[3] "Ebola virus continues to spread, Asia at great risk", http://www.dailysabah.com/asia/2014/10/26/ebola-virus-continues-to-spread-asia-at-great-risk, October 26, 2014.
[4] Chris McGreal, "Obama pushes for 'global effort' to combat spread of Ebola", the Guardian, [5] Lisa O'Carroll, "World Leaders 'failing to help' over Ebola crisis in Africa", the Guardian, 19th August 2014.
[6] Richard Hamilton, "Ebola crisis: The economic impact", http://www.bbc.co.uk/news/business-28865434
[7] Mark Strauss, "How Healthcare for Wild Animals Could Stop the Next Pandemic, National Geographic", http://news.nationalgeographic.com/2015/06/150616-connecting-dots-disease-ebola-mers-pandemics/, published 17/06/2015.
[8] Richard Preston, 1994, The Hot Zone: the chilling true story of an Ebola outbreak, 336
[9] Tom Cheshire, Ebola outbreak: why is the virus so deadly? http://news.sky.com/story/1353981/ebola-outbreak-why-is-the-virus-so-deadly, 30/03/2016
[10] Dan O'Connor, "Terrifying as the Ebola epidemic is, we must not lose our research ethics", The Guardian,
[11] Sarah Boseley, "Ebola vaccine trial proves 100% successful in Guinea", http://www.theguardian.com/world/2015/jul/31/ebola-vaccine-trial-proves-100-successful-in-guinea, 30/03/2016
[12] James Gallagher, "Ebola vaccine is 'potential game-changer'," http://www.bbc.co.uk/news/health-33733711,
[13] NHS Choice, "Scientists hail '100% effective' Ebola vaccine", http://www.nhs.uk/news/2015/08august/pages/scientists-who-hail-100-percent-effective-ebola-vaccine.aspx ,
[14] WHO, "Ebola vaccines, therapies, and diagnostics", http://www.who.int/medicines/emp_ebola_q_as/en/, 30/03/2016

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J. Altaf is now in Year 10 studying for her GCSEs at Oasis Academy, Oldham, Greater Manchester. J. Oliver is in the final year of his PhD, studying the genetics of rheumatoid arthritis treatment at the University of Manchester.

PhD Tutor's note
I was very impressed with J's final assignment and her work ethic and attitude during The Brilliant Club course studying the biology of the Ebola epidemic. I have no doubt that she will continue to work hard and achieve great success in her GCSEs and whatever she sets her mind to in the future.

The Future of Technology and Us

Year 9, Key Stage 4

Huntcliff School, Saltburn-By-The-Sea
E. Fowler, supervised by M. Kundegorski, University of Durham

Technology influences our everyday lives hugely, from the moment we wake up with our alarms, to the moment we switch our lights off to go to bed. A clock was the first spark of technology back in 1656.[1] In this essay, I will discuss the possible effects that medical technology may have on us in the future and how mobile phones and social media affect teenagers and the world around us. I have decided to write about these subjects because they surround us wherever we go. In addition, I am a teenager and I was brought up using a phone and social media.

The development of medical technology over the past 50 years has been greater than its development over the previous 2000 years. This means that we will see even greater medical advancements in our lifetime. Advancements in medical technology over the past 50 years include the following: 1976 brought the first commercial PET scanner; 1985 the first surgical robot from Yik San Kwoni; 1988 the first laser cataract surgery of Patricia Bath; 1998 the first stem cell therapy; 2001 the first tele surgery; 2007 the first bionic eye and 2013 the first laboratory-grown human liver.[2]

The progression of medical technology has made a positive difference in the world because it has increased life expectancy, provided cures and improved preventive care. In general, quality of life has been made better and people can make more of their lives. Pace makers, artificial kidneys, insulin pumps, hearing aids, joint implants and income health monitoring systems are widely used in modern society. In 2050, 25% of our population will be considered of old age [3]. Therefore, tele-health tools like smart homes, wearable monitoring systems, health apps, e-visits and electronic health record systems must be explored and developed [4]. I believe that, in this way, technology can further improve health care and make it accessible and affordable to our growing and ageing population.

I believe that medical research is the most important aspect of medical technology. Due to advanced medical research, scientists have been able to protect people against life-threatening diseases such as malaria, polio, MMR and hepatitis. We are given vaccinations containing antibodies of the diseases so that if we contract them, our bodies can fight the diseases more easily. At school, we received the HPV vaccinations to prevent cervical cancer. This vaccination was available from 2006.[5] According to the World Health Organisation, vaccinations save approximately 3 million lives a year. Furthermore, due to medical nanotechnology and existing knowledge of molecular and cellular biology, scientists believe that in the future, medical technology will be more accurate; and even quicker diagnosis techniques will be developed.[6] For example, cancer will be diagnosed earlier and may have a higher survival rate.

Medical science has made a significant leap forward in reproduction and fertility. It began in July 1978 when Louise Brown was the first successful test tube baby created through a fertility treatment called IVF.[7] Scientists have recently started to explore CRISPR (Clustered, Regularly, Interspaced, Short Palindromic, Repeat).[8] If CRISPR is a success and is developed further, it means that we will be able to genetically modify people. Consequently, many complex ethical issues will arise and some people refer to the technology as 'playing God'.

Social media is having profound effects on the way we think and act

The example I will be using is 'designer babies'. The term 'designer baby' refers to a baby whose genetic makeup has been artificially selected by genetic engineering and combined with in vitro fertilisation to ensure the presence (or absence) of particular genes or characteristics.[9] Designer babies mean that we can choose what gender a baby is and which genes they will inherit. Some advantages of this method are that it reduces the risk of babies getting genetic diseases and medical conditions that could affect them later in life. This may result in an increased lifespan. There is also a better chance that the child will succeed in life and there will be a better under-standing of genetics; for example regarding the prevention of passing on certain diseases or characteristics. As well as being able to choose which genetics a baby has, we may be able to pick and choose what behavioural traits they have; musical or athletic ability for example. However, although the thought of being able to pick and choose what our future generation will look like may seem like a brilliant idea, there are serious ethical issues to consider. How might it affect the relationship between parents and children, with the knowledge that the parents have chosen what the children look like? Eventually, everyone could look the same, be the same and act the same. Nobody will be an individual and there could be a loss of variety. We may as well be robots programmed to do exactly the same thing.

If we do have designer babies in the future, only the rich would be able to afford them. There are other, more important issues in the world, such as child poverty and high mortality rates in LEDCs. We should be investing our money in adopting existing children who have become orphaned by war, or children who live in poor countries, or children like me who have been adopted so that they can have a better life. Therefore, I believe that the future advancement of reproduction and fertility will make us forget the more important things in life, like looking after the people who are already here.

When the first mobile phone was made in 1973, Martin Cooper was the first to use it to make a call on the 3rd April that year. The phone weighed 1.1kg, was 23cm long, 13cm deep and 4.45cm wide.[10] Not many people had a mobile phone then because it was a luxury, whereas it is now seen as a necessity. It was during the mid-1980s that the mobile phone gained popularity. Nowadays, more people have a mobile phone than a toothbrush.[11] Mobile phones are an example of our neediness for every new advancement in technology. The most telling example of this is 'Apple'. Almost every year 'Apple' brings out a new model of the 'iPhone' and each version has brand new features - a 'must have' for every phone buyer. Phones are becoming thin and touch screens are becoming bigger. Therefore, metals which are present in touch screens are in higher demand than ever.

Indium is a metal found in tablet and smartphone screens. Indium, a byproduct of zinc mines, was investigated by Dr William S. Murray in 1924.[12] If the demand for zinc declined it would become hard - maybe impossible to obtain indium.[13] Because of this, mobile phones for our future generations would be very costly if we do not find an alternative resource. Therefore, we must learn to recycle mobile phones more efficiently and consider alternative phone electronics. For example, Fairphone is a promising social enterprise that is building a new movement for fairer electronics.[14] They aim to have a social impact on mining, design, manufacturing and recycling. It is comparable to the fair-trade system in food and clothing. I believe that there should be more awareness and education about this possibility and hopefully, as a result, many more people will buy a 'fair phone'.

Mobile phones also have a negative effect on our eyes. Research has shown that squinting at your phone, could make you susceptible to digital eye strain.[15] Originally, the phone was designed as a form of communication from one house to another but it has now become a whole new type of technology. It appears people would rather text each other than have a face-to-face conversation. Excessive use of mobile phones may be causing illness. One such illness is known as 'Phantom Vibration Syndrome'.[16] Phantom vibration syndrome is where you feel like your phone has vibrated in your pocket but it has not. Another danger of mobile phones is the risk of cancer due to high levels of electromagnetic radiation.[17] Children are at greater risk because their brains are still developing and their skull is very thin. In the UK 90% of children between the age of 8-13 have a mobile phone and 52% of the children have their first phone by the age of 10.[18]

Most people who own a smartphone, have their phones linked to one or more social media accounts. Social media has changed the way people think, act and live. It affects our emotions, decision making and attention-spans. There are many examples of social media: Instagram, where people post pictures; Tumblr, where you can make your own blog; Snapchat, where you post ten-second pictures that are automatically deleted; Twitter, where you can interact with celebrities; and Facebook, the most popular, where you can chat to your friends and family and post on each other's timelines with messages and photos. Facebook is the most popular form of social media and has 1.59 billion monthly active users. Facebook was invented by Mark Zuckerberg, an undergraduate at Harvard University early in 2004.[19] Social media is now a modern way of becoming 'famous'. We post pictures of ourselves trying to get as many likes as we can and it makes us feel good about ourselves. Social media is like a drug; it is addictive and we crave more and more of it. However, it possibly has many negative effects on teenagers including sleep disorder, depression, anxiety, addiction, isolation, cyberbullying, insecurity and FOMO (Fear Of Missing Out).[20]

Teenagers no longer daydream because they are preoccupied with their smartphones. This causes them to not use the parts of their brains used for self-reflection and reflecting on other people.[21] This may lead to becoming isolated and the inability to read nonverbal language. With excessive chatter and texting, combined with too few real-world relationships, social skills will weaken.

What makes social media so addictive is that it releases a feel-good chemical into our body called 'dopamine', particularly when we talk about ourselves online.[22] Not only has our generation become obsessed with social media but now many of us also believe that the most important values are fame and fortune, whereas in the past they were more centred on family and community. Children would now rather stay in on a warm summer's day, watching TV and playing on iPads and phones than go outside to play sports. In addition, indoor activities like arts and crafts and family games are not as popular as they used to be. I believe that smartphone technology has changed the world that we live in and that it is a big distraction from the most important things in life: family, friends and education.

In conclusion, I believe that the advancement of technology overall is a good thing because, although we may not realise it, technology saves our lives. In hospitals, we are attached to lots of machines to keep us alive and well. Alt-hough, some advancements may seem like they are stupid or outrageous - such as designer babies, they are all being developed for our benefit. It is our responsibility to use these in a mature way to get rid of diseases that have been passed on through generations, or, if we use it in a selfish way, to make children look how we want them to. The same applies to smartphones and tablets. We can use them in a way that it is beneficial or we can let them take over our lives. After all, can you blame younger generations for being on phones too much when they have been brought up surrounded by advancing technology? We must learn how to manage it sensibly and make time for real-life issues. This is our responsibility.

References:

[1] History of timekeeping devices
https://www.en.wikipedia.org/wiki/Hi [8 April 2016]
[2 Timeline of the history of medicine and medical technology.
https://en.wikipedia.org/wiki/Timeline_of_medicine_and_medical_technology
[6 April 2016]
[3] Advantages And Disadvantages Of Longevity Sociology Essay, 23 March 2015.
https://www.ukessays.com/.../advantages-and-disadvantages-of-longevity..
[6 April 2016]
[4] Mayo Clinic Staff,'Telehealth: When technology meets health care'
www.mayoclinic.org/healthy-lifestyle/consumer-health/in-depth/telehealth/
[9 April 2016]
[5] HPV vaccines
https://en.wikipedia.org/wiki/HPV_va [9 April 2016]
[6] University Polytechnic de Catalunya,June16,2009,'Advances In Medical Technology:What does the future hold?'
https://www.sciencedaily.com>2009/06
[7] Louise Brown
https://en.wikipedia.org/wiki/Louise_Brown [9 April 2016]
[8] Ekaterina Pak,'CRISPR: A game-changing genetic engineering technique '...
sitn.hms.harvard.edu/.../crispr-a-game-changing-genetic-engineering-tec
[9 April2016]
[9] Designer Babies Pros and Cons | Gene Therapy | Genetic ...
www.futureforall.org/bioengineering/designer-babies.html [9 April 2016]
[10] History of mobile phones - Wikipedia, the free encyclopedia
https://en.wikipedia.org/wiki/History_of_mobile_phones [10 April 2016]
[11] Jamie Turner,18 October 2011:'Are There Really More Mobile Phone Users Then Toothbrushes ' [10 April 2016]
60secondmarketer.com/blog/.../more-mobile-phones-than-toothbrushes/
[12] "indium' corporation'
mobile.indium.com/metals/indium [10 April 2016]
[13] Rachel Nuwer,'What is the worlds scarcest material?'
www.bbc.com/future/story/20140314-the-worlds-scarcest-material [11 April 2016]
[14] www.fairphone.com [11 April 2016]
[15] Ed Yourdon,'5 Reasons Why Cellphones Are Bad For Your Health'
www.medicaldaily.com/5-reasons-why-cellphones-are-bad-your-health-[11 April 2016]
[16]Tim Locke,'Do You Have 'Phantom Vibration Syndrome'?' - WebMD
www.webmd.com/news/20160111/phones-phantom-vibration [10 April 2016]
[17] Dr.Edward Group,'5 Ways Cell Phones Harm Your Health'
www.globalhealingcenter.com/health/5-ways-cell-phones-harm-health/
[10 April 2016]
[18]Bianca London,'Over HALF of children under 10 have a mobile phone'| Daily ...
www.dailymail.co.uk/.../Over-HALF-children-10-mobile-phone-15-food..
[10 April 2016]
[19] Facebook - Wikipedia, the free encyclopedia
https://en.wikipedia.org/wiki/Facebook [10 April 2106]
[20] The Positive & Negative Impacts of Social Media on Our New
www.khaama.com/the-positive-negative-impacts-of-social-media-on-our
[10 April 2016]
[21] '5 Crazy ways social media is changing your brain right now,ASAp science
https://youtube/HffWFd_66JO [9 April 2016]
[22] Molly Soat,' Social Media Triggers a Dopamine High'
https://www.ama.org/publications/.../Pages/feeding-the-addiction.aspx
[10 April 2016]

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Programme Officer's Comment:
The writer of this article successfully argues that technology is hugely influential in all aspects of life, and tracks the accelerated growth and importance of medical technology in recent decades. Her persuasive and thoughtful style reinforces her key points, which are also well-supported with suitable sources. The focus on smartphones and social media in the second half of the article is especially interesting, as it is a topic of great relevance to young people today. It contributes to the writer's convincing conclusion that technology is a great benefit to human life, as long as it is managed sensibly.
—Dr N. Day, Programme Officer for the North East and Yorkshire, The Scholars Programme

Why do Health Inequalities Exist in the UK?

Year 9, Key Stage 4

Ark Globe Academy, London
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Over recent years, the gap between the rich and the poor has been increasing - and so has the gap in health between them. Health inequalities are still a problem in the UK despite advancements in healthcare, as those who are more disadvantaged have poorer health than those who are more advantaged.[1] This issue has been the subject of intense debate in government, focusing on why these inequalities exist in a developed country that should enable all of its population to lead healthy lives. This essay will discuss various factors that contribute to health inequalities, including more pragmatic explanations such as material and psychosocial factors and theories that have been discredited or criticised, such as the indirect and direct 'selection' theory. The essay will also analyse suggestions that have been proposed in the Marmot Report and the Acheson Report to reduce health inequalities in the UK.


Many studies link deprivation with poor health choices

Inequality in health is an important matter in the public health sector and this controversy was fuelled by the Black Report, which was published in 1980.[2] It highlighted that people who were in lower socioeconomic classes had a lower life expectancy than those who were in higher classes. Evidence from OPCS supported this point, as in 1971 men in unskilled occupations were more likely to die younger than men who worked in higher-skilled jobs.[3] The release of the Black Report could be seen to shed negative light on the Conservative government in 1980 because it exposed the underlying disparities in the quality of life among social classes - and it is likely that the government would have not wanted to attribute this to the lack of funding that could have supported deprived communities. Since the 1980s, there have been notable advancements in health and science, along with greater efforts by the public health sector to improve health in less developed areas. However, it has been shown that health inequality is still a recurring problem in the UK. The Marmot Report, for example, showed that there is a health gradient in regards to the link between an individual's socioeconomic status and their quality of health. The Office of National Statistics showed that those in more technical, managerial jobs have a lower mortality rate per 1000 than those in less advanced levels of employment (bear in mind that the health gradient is not the difference between the most privileged and the least privileged, but is instead the relationship between varying levels of social status and health [4]) and that these observations were more prominent in the North East than the South West.[5] These findings are shocking, mainly because the UK has a free healthcare service, the NHS, promoting "equality through the services it provides" and paying particular attention "to groups... where improvements in health... are not keeping pace with the rest of the population". [6] As the NHS has been active since 1948, it would only seem logical that everybody, regardless of socioeconomic class, should have an equal quality of health.[7] This has been shown to not to be the case. As a result, there have been several explanations proposed in various studies concerning the continued presence of these inequalities.

The 'selection' theory, considered in the Black Report, suggests that these dramatic health inequalities are caused by the genetic disposition to illness and the innate qualities of an individual.[8] The direct selection theory dictates that if an individual was 'naturally ill', the person's socioeconomic status would fall as a result, affecting them in social aspects of their lives as well as financial (for example being employed in menial jobs). The indirect selection model claimed that an individual's innate personality influenced their social standing as their qualities affected their likelihood of being employed and their lifestyle choices.[9] This theory may have been supported by observing that parts of the UK with "more fit populations" had an economy where "job opportunities were better".[10] Despite this, the 'selection' theory has been widely discredited. Firstly, it assumes that there is a high occurrence of mobility between social classes. Such fluid movement has not been observed in recent history. People in the upper classes have more choice of different types of employment, are kept financially secure by means of inheritance, and have better access to social support such as excellent healthcare - therefore, it is unlikely that ill health will automatically render them poor.[11] Marmot disagrees with the notion that health inequalities are wholly caused by genetic makeup or other uncontrollable factors as in the report it mentions that health inequalities are the prime result of "social and economic inequalities in society". [12] This theory gives no consideration to other important determinants such as education and income, which can affect the type of occupation a person is employed in - it blames people for something that they have little control over. Therefore, the 'selection' theory cannot be a direct cause of health inequalities in the UK.

The behavioural / cultural explanation has also been used to explain health inequalities in the UK caused by behaviour. It states that in general, people who are more deprived tend to adopt unhealthy, damaging behaviours, such as smoking and the over-consumption of unhealthy products whilst those who are not as deprived are more likely to engage in health-promoting behaviours, such as daily exercise and the consumption of nutritious foods. Therefore, this explanation holds people responsible for their own health. In 2012, men and women in the most deprived areas of England were several times more likely to smoke than people in the least deprived areas [13]. We often see stereotypical representations of such individuals in the media - perhaps viewing them as irresponsible and careless. [14] However, we cannot just blame poor quality of health on ignorant behaviours - it is vital to examine other linked factors, such as stress management, that can contribute to the way a person acts in regards to their health. For example, people living in a deprived area where unemployment is rife are less likely to be able to afford healthy foods and turn to less healthy food, which is cheaper and more readily available (which is becoming increasingly common as a result of a greater concentration of takeaway food outlets).[15] The 1987 Hilary Graham study serves as an antithesis to the 'victim blaming' idea observed in the mass media (this idea is even observed in studies that discuss the matter). This study aimed to perform a deeper analysis of the experiences of working-class women who smoke. Working-class women between 1972-1982 were more likely to take up smoking and were less likely than upper- class women to quit smoking.[16] It was observed that the primary reason for this was to help cope with the stress that comes along with childcare and financial problems that they faced.[17] Those who are more economically secure have increased access to social / material support, meaning they are less likely to be dependent on a recreational drug to deal with stress. They may be better informed of the dangers of smoking, including the effects that it will have on their children as they can afford a better education. However, these privileges are often not available to those who are struggling financially and thus, they may turn to damaging habits.

A more universally accepted cause for health inequalities is the material explanation. It says that health can be affected by material, tangible factors such as income and living conditions. The income that a person receives in the UK is usually enough to cover their absolute needs - such as shelter and some form of nutrition - which contrasts well with other less developed areas of the world. However, the amount of income that a person receives can have a knock-on effect on their relative needs, such as the quality of housing and the quality of the food and drink they consume. For example, a person with a low income may be unable to afford nutritious food - meaning that in the long term, it will be likely that they will suffer from a condition that may be caused by a lack of nutrients. This links to behavioural factors as money can determine whether a person has a wide range of goods or services to choose from or not. In addition to this, housing and the quality of the environment can be included as a material factor. A low disposable income forces people to move into poorer-quality housing - meaning that their health can be affected by factors such as infestation and mould (which can affect the respiratory system).[18] From 2001-2006, it was shown that deprived areas with the least favourable environments (for example poor air quality or proximity to a landfill) had a greater proportion of diseases and conditions than less deprived areas.[19] A strong, direct link has been observed between material factors and health. Therefore, we can say that material explanations for health inequalities are realistic and reasonable. Psychosocial explanations for health inequalities link material factors, such as income, to a person's mental and physical health. The main psychosocial explanation states that a person's income (from the occupation that they are employed in) can influence the level of stress that they have - whether they do or do not receive social/material support to help deal with their stress can influence their physical health (particularly the cardiovascular system). Those who have a lower income are more likely to be stressed (as was observed among single working-class mothers in the Graham study [20]) - affecting their outlook on life and their physical health. This can be worsened by the use of drugs (especially depressants like alcohol) or by the lack of mental health services.[21]

We cannot truly say that health inequalities are caused by just one of these factors - instead, we are more likely to take a 'social determinants approach'. The WHO defines social determinants as "the conditions in which people are born, grow, live, work and age... shaped by the distribution of money, power and resources." This can include gender, age, place of residence, level of education and social standing. [22] These factors are influenced by the way the government uses its power to support (or not support) certain groups in a society. The social determinants approach has strong links to the material / psychosocial explanations because they both examine underlying factors, such as income, to explain observations and behaviours. On the other hand, however, we would expect these social determinants to be a major factor in developing countries - not much so in the UK, where the government strives for health equality by means of the NHS.

In conclusion, there have been several theories on why there are health inequalities in the UK, with some stemming from or being supported by reports such as the 1980 Black Report, the 2010 Marmot Report and other notable studies. Five main explanations have been discussed concerning the occurrence of health inequalities in the UK: the 'selection' theory, the behavioural / cultural, material and psychosocial explanations and the social determinants approach. However, it can be said that the material explanation is the most plausible because there is a clear link with factors such as housing and environmental pollution with health. The selection theory has been discredited because it assumes that there is a lot of movement between social groups (which is not true) and says that a person's social status (and ultimately health) is determined by innate physical and mental characteristics.

There have been several suggestions outlined in relatively recent reports such as the 1998 Acheson Report and the 2010 Marmot Report. The Acheson Report stresses the importance of education as a means of providing the qualifications that adults need to increase their socioeconomic position (helping them materially as they get a higher income from higher job occupations), and ensuring that children and young people have the knowledge and skills to lead a health-promoting life.[23] [24] However, the quality of education may be hindered by environmental factors such as vandalism and littering and psychosocial factors (such as stressed parents being unable to support their children and the level of education

the parents have). This can be particularly stressful on the staff working in schools in disadvantaged areas.[25] Therefore, Acheson recommends that schools be provided with "additional resources... serving children from less well-off groups to enhance their educational achievement".[26] It may be effective because it can help schools in deprived areas to deliver a higher standard of education, which can help pupils in the long-term. However, on the other hand, it does not directly help the parents. In the Marmot Report, it recommends that schools should play a major part in supporting families and communities by incorporating social and emotional development and health into the curriculum.[27] This can be effective because more young people are aware of matters that can affect their health, making them less likely to engage in health-damaging habits.

The public health sector must close the gap between socioeconomic classes in the UK caused by health inequalities and enable everyone to have access to good health - meaning that they can lead productive lives.

References

1. David Matthews, 'Social Class and Its Influence in Health', http://www.nursingtimes.net/clinical-subjects/public-health/social-class-and-its-influence-on-health/5091017.fullarticle [27th March 2016]
2. BBC, ' Higher Bitesize Modern Studies- Wealth and Health Inequalities part 2: Revision', http://www.bbc.co.uk/bitesize/higher/modern/uksociety/health/revision/1/ [27th March 2016]
3. Occupational Mortality 1970-72. (Microfiches and 1978, P-37)
4. Richard G. Wilkinson, M. G. Marmot, (2003), Social Determinants of Health: The Solid Facts, page 10.
5. Siegler V, Langford A and Johnson B (2008), Regional Differences in male mortality inequalities using the National Statistics Socio-economic Classification, England and Wales, 2001- 03. http://www.statistics.gov.uk/downloads/theme_health/HSQ40-winter-2008.pdf
6. NHS England, 'The principles and values of the NHS in England-NHS Choices', http://www.nhs.uk/NHSEngland/thenhs/about/Pages/nhscoreprinciples.aspx [29th March 2016]
7. NHS England, 'About the National Health Service (NHS) in England- NHS Choices', http://www.nhs.uk/NHSEngland/thenhs/about/Pages/overview.aspx [29th March 2016]
8. Sir Douglas Black, 'The Black Report' (1980), chapter 6 (Explanation for Health Inequalities), paragraph 7.
9. Sir Douglas Black, 'The Black Report' (1980), chapter 6 (Explanation for Health Inequalities), paragraph 7.
10. Sir Douglas Black, 'The Black Report' (1980), chapter 6 (Explanation for Health Inequalities), paragraph 8.
11. Tami Luhby, 'The New Inequality: Health Care', CNN, 18th December 2013, http://money.cnn.com/2013/12/18/news/economy/health-inequality/ , [30th March 2016]
12. Marmot M, 'Fair Society, Healthy Lives: A Strategic Review of Inequalities in England'(2010), page 10.
13. Integrated Household Survey, 'Smoking Rates by Area Deprivation' (2012) [31st March 2016]
14. John H. McKendrick et al., 'The Media, Poverty and Public Opinion in the UK'(2008), chapter 3 ('Reporting Poverty in the UK News')
15. Denis Campbell, 'Fast Food Takeaway Shops Grow More Rapidly in Deprived Areas of UK', Guardian, 2nd April 2015, http://www.theguardian.com/society/2015/apr/02/fast-food-takeaway-shops-grow-more-rapidly-in-deprived-areas-of-uk [31st March 2016]
16. Office of Population Censuses and Surveys, 'Cigarette Smoking: 1972 to 1984', OPCS Monitor, GHS 85/2, OPCS, London, 1985
17. Hilary Graham, 'Women's Smoking and Family Health'(1987), page 52.
18. Jake Eliot, 'The Three Housing Problems That Most Affect Your Health', Guardian, 8th August 2014, http://www.theguardian.com/society-professionals/2014/aug/08/housing-problems-affect-health [1st April 2016]
19. Department For Environment, Food and Rural Affairs, 'Populations living in areas with, in relative terms, the least favourable environmental conditions, 2001–6'(2007)
20. Hilary Graham, 'Women's Smoking and Family Health'(1987), page 52, under 'Smoking And Caring'.
21. Vijaya Murali & Femi Oyebode, 'Poverty, Social Inequality and Mental Health' (2004), page 219, under 'Alcohol and Substance Misuse'
22. WHO, 'What Are Social Determinants of Health?'(2012), http://www.who.int/social_determinants/sdh_definition/en/ [2nd April 2016]
23. Sir Donald Acheson, 'Independent Inquiry into Inequalities in Health Report (Acheson Report)', 1998, Part 2: Education
24. Sir Donald Acheson, 'Independent Inquiry into Inequalities in Health Report (Acheson Report)', 1998, Part 2: Education
25. Sir Donald Acheson, 'Independent Inquiry into Inequalities in Health Report (Acheson Report)', 1998, Part 2: Education
26. Sir Donald Acheson, 'Independent Inquiry into Inequalities in Health Report (Acheson Report)', 1998, Part 2: Education
27. Marmot M, 'Fair Society, Healthy Lives: A Strategic Review of Inequalities in England'(2010), page 10.

PhD Tutor's Comment:
In her essay, K. demonstrated a sophisticated understanding of socio-economic inequalities in health and significant critical engagement with a number of different explanations for the existence of such patterns. She successfully grasped several new and complex concepts and skillfully applied appropriate literature to support her arguments throughout her paper. I was struck by the quality of her written work and the way in which she was able to construct such a coherent pathway through this material. I would be surprised if many undergraduate students could craft something this good in that length of time! – Dr R. Ponsord, London School of Hygiene and Tropical Medicine.

How Do Nutrients Affect Metabolism and Predispose Us to Disease? A Case Study on Sugar and Obesity

Year 10, Key Stage 4

Hatch End High School, London
K. Pattni, supervised by N.E. Lara-Pompa

Sugar. It is everywhere, in our food and in our drink, whether it's written as glucose, fructose, honey or dextrose. The list is endless, yet all of them are made of the same thing. I have noticed a lot of speculation around the impact of this carbohydrate on many different health conditions, like heart disease, diabetes and obesity. The theory that intrigues me most is the theory that sugar can lead to obestity, as we always hear about lipids being the main cause of the epidemic. This topic is relatable being a child myself and being exposed to a variety of unhealthy foods and drinks with high sugar levels. This is why I have chosen to research the link between obesity and sucrose. Therefore, I will be searching for evidence from various scientific sources to determine if sugar plays a part in the obesity epidemic.

Childhood obesity is a major epidemic that is sweeping the nation and is affecting many lives as it can have serious long-term health effects. As children and teenagers have the ability to purchase and consume sugary produce, many children are now becoming overweight. It's not just us children that are exposing ourselves to additives and sugar but adults as well who are misled by false advertising and handing their kids toxic amounts of the carbohydrate. According to Bupa, throughout the world over 42 million children under five were overweight in 2013 indicating that if these trends continue, we will have 70 million overweight or obese children by 2025.[1]

I carried out a literature search on sugar and its impact on obesity and found several news articles and case studies. Keywords that I felt were relevant were 'obesity', 'sugar' and 'child' and these helped me as I input them into my search engine to find five reliable sources, four of them from Google Scholar to ensure reliability and scientific relevance.

The first article that I decided to use as a source was a news article from The Guardian on sugar being the 'deadly villain' in the obesity epidemic.[2] The piece covered the findings of an American paediatrician specialising in the treatment of overweight children in San Francisco, Dr Robert Lustig. Lustig has spent 16 years studying the effects of sugar on the central nervous system, metabolism and disease. He believes that sugar is addictive and toxic, and blames insulin for the problem. Insulin is a hormone that is produced to regulate the concentration of glucose in the blood. He believes foodstuffs that raise the insulin levels in the body are the main causation for 75-80% of obesity cases. This is because too much insulin will allow lots of glucose to pass through cell walls and these large amounts of sugar in the blood will rush to the liver. The liver will not be able to process all that glucose at once and this will overload the organ, causing it to convert the excess sugar into fat. Thus leading to the major health problem, obesity.

High quantities of sugar are often added to foods that target children

Lustig's larger message was that "kids have access" to the harmful foodstuff and that it's included and added to produce that does not even require the presence of sugar. When lipids and fat were identified as the main cause of obesity and overweight conditions, the food industry began to introduce low-fat products. However, to make these more palatable, sugar was added, causing even bigger problems. Lustig identified that evidence of dietary effects on the body is very difficult to estimate and that a randomised control trial would be almost impossible as individuals may revert to a normal eating pattern after a few days.

The second source was from the NHS website and backed up most of the ideas from the first article.[2] [3] The piece spoke about a group of specialists called Action on Sugar (AOS) that is concerned with sugar and its effects on health. AOS believes that there is a link between obesity and high sugar consumption and want the food industry to reduce the amount of sugar that they are adding to children's food in particular. AOS wish to stop the targeting of children with massive advertising for high calorie snacks and soft drinks. According to the British Dietetic Association (BDA), added sugar is not necessary for a healthy diet and most of the foodstuffs with added sugars do not contain many of the other nutrients such as proteins, vitamins and minerals. High consumption of these foods could contribute to becoming overweight, thus increasing risk of other health conditions such as heart disease or type 2 diabetes.[3] However, the BDA says that small amounts of added sugars to sweeten foods are not too harmful if limited. It's the overall amount of sugar and the frequency of sugary food snacking that matters. Sugar is added to many foods like cakes, chocolate, sweets and some fizzy or juice drinks. Sugar is often added in large quantities i.e. a can of Cola can contain up to 35 grams of sugar, which is the equivalent of seven sugar cubes.[3]

For my third source I found a scientific analysis of the relationship between consumption of sugar-sweetened drinks and childhood obesity.[4] This journal was written by three authors, Dr David S. Ludwig, MD, Karen E. Peterson, ScD and Steven L. Gortmaker, PhD. This journal cites their findings and methodology for the prospective observational analysis they carried out to test the relationship between sugary drinks and childhood obesity. They enrolled 548 diverse schoolchildren from public schools in the Boston, Massachusetts area and watched over these individuals for 19 months from October 1995 to May 1997. Their trial resulted in the evidence to show that each additional serving of sugar-sweetened drink consumed by the children resulted in their Body Mass Index (BMI) and odds of becoming obese increasing.

The fourth source was a systematic review of the many trials and studies carried out on the impact of sugary drinks consumption on obesity in children and adolescents. These articles were found through a key word search through the MEDLINE database.[5] 'They found 264 citations using key words such as 'soda', 'sugar-sweetened beverage' and, 'weight gain' and 'obesity'. This narrowed down to 72 potentially eligible articles. These were screened further for relevance and eligibility and was narrowed down to 30 studies (4, 7-33), 15 cross-sectional (7-21), ten prospective (4, 11, 14, 22-28) and five experimental (29-33), two of which were Randomised Control Trials (29-33). This source has many references to various studies that the authors had found through searching and filtering the many studies published from 1966 to May 2005.[5] The source reviewed all of these studies and their findings and commented on the results from these. The majority of the results agreed with the theory that sugar causes obesity.

Source five was an evaluation of a longitudinal cohort study and had focused on the impact of sugar-sweetened drinks on African-American preschool children.[6] 365 low-income African-American children were involved in the study, aged three - five at the beginning of the study. The children were examined at a dental clinic in 2002-2003 to test for the effects of sugary drinks and the dietary information of the subjects were collected through the Block Kids Food Frequency Questionnaire. BMI was recorded at the beginning of the investigation and again at the end of the study two years later at the same dental clinic, classifying children in the range of the 85th-95th percentile as overweight and any child with a BMI above the 95th as obese. The results of the trial showed a significant association between sugar-sweetened drinks and risk of childhood obesity and an increase from 12.8% of the children being overweight to 18.7% after the 2 years.[6]

The prospective observational analysis journal seems very reliable as the trial was carried out by experienced professionals with expertise in the areas of study.[4] They managed to carry out an observational study designed to

get accurate and reliable results that would convince people that the link between sugary beverages and child obesity exists. I believe that the longitudinal cohort study did the same but it only focused on one racial group.[6] These two sources (4, 6) were very reliable as they included the authors' details and references to other sources. The most impressive source was the review as this summarised the findings of many different types of study designs, including two randomised control trials (29, 33) which are one of the most accurate and expensive study design types.[5] The NHS source was also reliable as the National Health Service website forum is run by experts and professionals and is supported by the government.[3] The article from the news was also quite reliable as the author had interviewed an expert doctor and The Guardian is regarded as an informational newspaper.[2] All of the findings from each of the sources I studied (2-6) agreed with each other and have helped me to come to a conclusion on the impact that sugar has on obesity.

From my research I have been able to distinguish a link between sugar and obesity in children. The higher the intake of glucose and sugar, the higher the production of insulin, which causes fat production. This in turn will cause the BMI of the individual to increase and the individual will become overweight or obese. Children are exposed to a lot of sugar and this is why childhood obesity is becoming more wide-spread.

The sources that I trust the most are the analysis which describes the findings of the multiple studies and the randomised control trials (RCTs) (29, 33) that were carried out by professionals.[5] This is because this type of study design effectively controls variables and tests the theory. There was also a lot of information on how the test was carried out and it gave the expected results. Also because RCTs are expensive, I find this source more reliable as money wouldn't have been invested unless the team and experiment were credible.

After analysing all of my sources, I have found limitations in the evidence as the prospective analysis was observational and cannot prove causality.[4] Another limitation may be that the prospective analysis has limited statistical power as it used a very small cohort.[4] Also the children may have made mistakes while filling out the forms if they had forgotten information. This could have also been the case with the studies mentioned in the review and the cohort study as the food frequency questionnaires may have been incorrectly filled. The Longitudinal cohort study also only focused on one racial group and the preschool children aged three - five could have moved into the different age group of five - seven years of age. This means that the lifestyles of the children would have changed and it would be difficult to determine the reliability of the results. Also the parents and carers of the children may not be able to track what their children eat and the results may be biased as children often follow the example of their caregivers.[6] The lack of any physical activity data is also a limitation as this can be a major factor in the child's BMI. Furthermore, with some of the other sources (4,5) there were studies in varying age groups such as children and adolescents, in which the measures for obesity were indirect and they could not control changes in the body caused by puberty or fitness training.

The review of the 30 studies states that there were multiple articles that had found that there was no association between the consumption of sugary carbonated drinks and the BMI of American children and adolescents (12, 13, 34).[5] The news article could also be considered biased as the content only focused on one doctor's findings. Nevertheless, the majority of the sources I looked at recognised sugar as a key contributor to the epidemic of obesity (2-6). Therefore, the news article (2) was supported by the scientific studies (4-6) and after analysing these sources, I am convinced that high sugar consumption can lead to obesity.

References:

1. Bupa Health Information Page – Obesity in Children. http://www.bupa.co.uk/health-information/directory/c/child-obesity
2. The Guardian News Article on Sugar Being the 'Deadly Villain' In the Obesity Epidemic. http://www.theguardian.com/society/2013/mar/20/sugar-deadly-obesity-epidemic
3. NHS Heath News Page On Sugar and the Obesity Epidemic. http://www.nhs.uk/news/2014/01January/Pages/sugar-obesity-qa.aspx
4. Relation between consumption of Sugar-Sweetened Drinks and Childhood Obesity: A Prospective, Observational Analysis – The Lancet Article. http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(00)04041-1/abstract?cc=y
5. Intake of Sugar-Sweetened Beverages and Weight Gain: A Systematic Review – The American Journal of Clinical Nutrition Article. http://ajcn.nutrition.org/content/84/2/274.full
6. Obesity and Sugar-Sweetened Beverages in African-American Preschool Children: A Longitudinal Study – Wiley Online Library Epidemiology Article. http://onlinelibrary.wiley.com/doi/10.1038/oby.2008.656/full
7. Energy and fat intakes of children and adolescents in the United States: data from the National Health and Nutrition Examination Surveys. Am J Clin Nutr 2000. http://ajcn.nutrition.org/content/72/5/1343s.

About the authors

K. Pattni is a Year 10 student at Hatch End High School. N.E. Lara-Pompa is a paediatric dietitian currently completing her PhD at the Great Ormond Street Institute of Child Health, University College London, where her research focuses on the use of body composition measurements and malnutrition screening tools for the nutritional management of paediatric patients with chronic conditions.

PhD Tutor's note

K.'s essay shows a deep understanding and critical analysis of the use of different studies and sources to provide the evidence underpinning nutritional recommendations. He performed a thorough search of the literature, summarised and critically analysed high-quality scientific sources and came to an informed conclusion on the strengths and challenges of research in this field. This essay was clearly above the expected skills of his current year. Working with all the pupils at Hatch End High School was a very rewarding experience. All students showed a high commitment to the program, actively participated in class discussions and submitted high quality essays, demonstrating skills that no doubt will enable them to succeed in their future academic activities.

Future Applications of Ultrasound in the Diagnosis of Prostate Cancer

Year 10, Key Stage 4

Lady Bridge High School, Manchester
J. Howie, supervised by D. Miguez, Manchester Metropolitan University

Introduction

The implications of ultrasound, like those of other imaging techniques, are vast in the medical world and hold great potential for use in the diagnosis and treatment of innumerable diseases, injuries and physiological phenomena. Within this, although several forms of it are already widely used, there is great prospect for further development and research into future applications of ultrasound in the diagnosis of prostate cancer.

Medical Imaging Techniques

At present, a variety of different medical imaging techniques, each with their own advantages and disadvantages, can be utilised depending on the circumstance. Examples of these include ultrasound, radiography, CT scanning, MRI and EMG. These are all medical applications of waves (whether sound or light), which are usually painless and non-invasive, however some are better for diagnosing specific conditions than others.

Radiography

When X-rays are passed through the human body, mediums of different densities absorb different amounts of radiation. As a result, a detector can build up an accurate 2D representation of the body's internal structures based on the amount of radiation it detects from different areas. Computed tomography (CT) uses a number of X-ray images to generate a 3D representation, however, whilst conventional radiography takes just minutes, CT takes considerably longer. All forms of radiography however, are useful for the diagnosis of certain diseases, including some cancers and infections, as well as breaks and fractures to bones. On the other hand, exposure to the ionising X-ray radiation used in radiography does slightly increase a person's risk of developing cancer in the future.

Magnetic Resonance Imaging (MRI)

As the human body is largely made up of water (containing hydrogen), magnetic fields can be used to manipulate the body's hydrogen atoms to find the density of the material in which they are found. Firstly, a magnet causes the hydrogen atoms to align with its magnetic field and a second magnetic pulse causes them to move back into their former position. The time that this takes depends on the density of the medium in which the atoms are found, therefore a detailed 3D image can be produced, which uses no ionising radiation. Unfortunately, however, slight movement can distort the image so (due to the movement caused by breathing) it cannot be used for abdominal scans and it can be a very lengthy and noisy procedure. It is also expensive so radiography is often used as a more cost-effective method.

Ultrasound

As part of a high-frequency wave is reflected when it hits a medium of a different density, when ultrasonic waves are passed through the human body, the proportion of the wave that is reflected and the time that this takes can be used to create an accurate live image of the body's internal structures without using potentially damaging ionising radiation. As the image generated by the scan is live, movement (such as that of a foetus or the contraction of muscles) can be easily observed, however, it takes a considerable amount of skill to interpret an ultrasound scan and you cannot use it to generate an image of what is behind particularly dense mediums such as bone. Finally, factors, ranging from the frequency of waves used to the patient's body size can affect the quality of the image produced.

Prostate Cancer

What is the Prostate?

The prostate, a gland of the male reproductive system, is located under the bladder and is about the size of a satsuma. It is, in part, responsible for the production of semen which, of course, leaves the body, making the prostate an exocrine gland. [1][2]

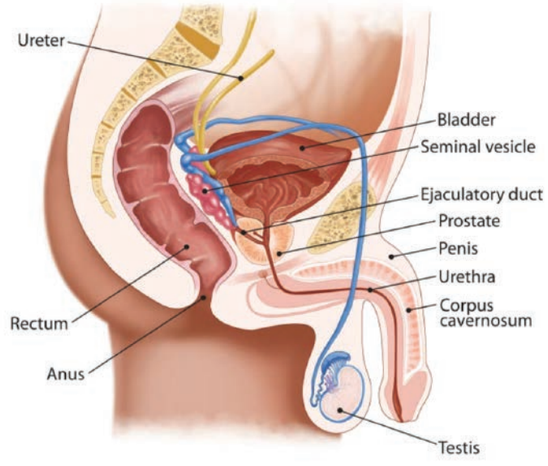

Diagram of the location of the prostate gland [5]

The Causes and Symptoms of Prostate Cancer

While the exact causes of prostate cancer remain largely unknown, certain factors are known to increase the likelihood of a man developing it. Of these, one of the main contributors is age, as prostate cancer usually develops in men of over 50. Lineage and genealogy can also affect one's chances, for there is a slight increase in risk for men who have a first degree relative with the cancer. Also, for reasons which are as yet unknown, the cancer is more prevalent in males of African or African-Caribbean descent and less prevalent in those of Asian descent.

As prostate muscle fibres contract and relax to control the flow of urine through the urethra (the tube which carries urine

from the bladder to the penis), many cancer symptoms do not become apparent until the prostate's tumour is large enough to affect the urethra. These symptoms include a need to strain to urinate, an increased need to urinate and, after this, feeling that the bladder is not fully empty, however, the development of the cancer is slow and these symptoms may not become apparent until a man has had the cancer for many years, sometimes a lifetime. [3][4]

Current Techniques Used in the Diagnosis of Prostate Cancer

Transrectal Ultrasonography

Transrectal ultrasonography is currently used to investigate a man's results from a digital rectal exam that could be potentially indicative of cancer. It is also used after a blood test that has shown elevated levels of a prostate-specific antigen (PSA). This is an antigen produced by the prostate gland and can indicate any growth to the prostate, which could be a sign of cancer

In transrectal ultrasound however, an ultrasound phase array transducer is inserted into the rectum where it uses ultrasonic waves to generate a picture of the prostate gland. Any abnormal growths to this could be a sign of cancer. However, as prostate enlargement in men over fifty is often a natural part of ageing and does not necessarily mean that a tumour is forming, imaging techniques must be used with other tests in order to diagnose a cancer.

Transrectal ultrasound is also used to help guide needles into the rectum so that a small amount of tissue from the rectal gland can be removed and used to complete a biopsy. A biopsy involves removing a small amount of tissue so that it can be analysed and tested for a disease or condition, in this case, prostate cancer. [6] [7]

MRI

Like ultrasound, MRI can be used to give a clear picture of the prostate and its surrounding tissues to assess for cancer and, if one has been found or previously diagnosed, find out the degree to which it has spread. To give a clearer picture, a contrast material such as gadolinium is often injected into the veins to contrast further the difference in colour between the prostate and its surroundings.[8]

Radiography

CT scans are usually preferred to a standard x-ray because of the detailed cross-sectional 3D images they can produce. Although they are not as useful as MRI for viewing the prostate itself, CT is sometimes used to map the spread of a tumour and see if it has reached nearby lymph nodes. These are an important part of the immune system and only become visible when they are swollen due to an infection or cancer. [9][10]

Bone Scans

Although they cannot be used to diagnose cancers, bone scans are often very helpful when mapping the spread of a tumour as most cancer spreads to the bones first. To do this, a small amount of low-level radiation is injected into the body which will settle in the bones where there is damage. A detector will detect the radioactive waves and use them to generate a picture of the skeleton. Bone biopsies are also used to analyse the spread of a known cancer. [11][12]

Development of Current Diagnosis Techniques

As no single technique can reliably be used alone to diagnose prostate cancer, a variety of techniques, including some which use ultrasound, are employed. This is because growth to the prostate gland, which may seem abnormal and could be indicative of cancer, is often a natural part of ageing, particularly in men over fifty. In addition, other diseases and infections can cause a growth to the prostate so it is necessary to use a wide range of different tests to ensure an accurate and correct diagnosis before treatment. Also, tests such as transrectal ultrasonography and MRI are often uncomfortable or invasive as a probe is often inserted into the rectum. With MRI, this is not always the case and this method is widely used to generate a clearer image of the prostate.

Future Diagnosis – Ultrasound

Ultrasonography, unlike radiography and bone scans, uses no ionising radiation, which very slightly increases a person's risk of developing cancers in the future as a result of their exposure. As a result, it is completely safe and is also painless. Though the equipment used in ultrasonography is very expensive, individual scans are relatively cheap to conduct and, as the image produced is live, the transducer can easily be moved to view the prostate from a variety of different angles without multiple scans. Standard greyscale ultrasonography is already widely used as a diagnosis technique for prostate cancer, however, variations of ultrasound have great potential as future techniques.

Future Diagnosis – Doppler Ultrasound

What is Doppler Ultrasound?

Doppler Ultrasound makes use of the Doppler Shift to determine both the direction and relative velocity of bodily structures (usually blood). Of course, if the blood is moving away from the probe, a drop in the frequency of the wave will be detected when it is reflected and if the blood is moving towards the probe, the wave will increase in frequency. The degree by which the frequency has changed is also used to determine the relative velocity of the blood flow as the more that the frequency changes, the faster the blood is travelling. There are three forms of Doppler ultrasound.[13]

Colour Doppler

The change in the frequency of the ultrasonic waves that are detected can be converted into different colours to indicate the velocity at which the structure is travelling with different colours indicating different velocities and directions.[14]

Power Doppler

Power Doppler is much more sensitive to slight changes in frequency than Colour Doppler so it is widely used in cases where the movement of a structure is slight. However, it is non-directional so although the speed of the structure can be measured with great accuracy, there is no way of detetcting its direction.[15]

Spectral Doppler

This type of Doppler uses graphical means to display Doppler measurements. It calculates the distance travelled by the structure per unit of time so that its velocity can be calculated. A positive velocity will indicate movement away from the ultrasound probe and a negative velocity will indicate movement towards it. Often, this form of Doppler will be converted into audible sound that can be heard as a distinctive but synthetic pulse with each heartbeat. [16]

Implications of Doppler Ultrasound in the Diagnosis of Prostate Cancer

As hypervascularity (a significantly increased velocity) of blood flow is often caused by nearby tumours, Colour and Power Doppler ultrasonography can theoretically be used to help diagnose cancer. As some cancers are isoechoic (the cancerous tissue is very similar to normal tissue) [17] they may not be diagnosed if only conventional ultrasonography is used and trials have shown that measuring blood flow has helped to diagnose some isoechoic cancers not seen with greyscale ultrasound. As Power Doppler is more sensitive than Colour Doppler, it can detect abnormal blood flow in smaller vessels and therefore should be able to help diagnose more cancers of the prostate. If tumours can be pinpointed accurately using this method, the areas where biopsies will be more successful can also be located. [18]

Future Diagnosis – Elastography and Ultrasound Contrast Agents

Elastography involves applying pressure to a body structure using an ultrasound array transducer. As tumours are denser and therefore typically stiffer than normal tumour, the degree by which tissue compresses because of elastography can be used to distinguish between ordinary and cancerous tissue. [19]

In MRI, to ensure better definition between body structures, a gadolinium contrast agent is often used. It is possible that similar contrast agents could be used in ultrasonography and those which are currently used have many advantages over the gadolinium-based agents used in MRI and CT as patients are often allergic to these contrast agents. Ultrasound contrast agents use microbubbles containing gas which are administered through the veins of the patient. They have a high degree of echogenicity (ability to reflect ultrasonic waves) so a great contrast between tissue and the microbubbles' gas is apparent. [20][21]

Colour Doppler Ultrasound (CDUS) and Magnetic Resonance Imaging (MRI)

There is much debate as to which of the above imaging techniques is more useful in the diagnosis of prostate cancer. Of course, MRI is notoriously more expensive and time-consuming while CDUS images are real-time and scans are considerably cheaper. However, MRI is considerably more sensitive in the outer regions of the prostate, where 70% of cancers are known to begin [23], when determining the hypervascularity of blood flow that is often caused by prostate cancer. Currently, CDUS is slightly more sensitive than contrast-enhanced MRI in the innermost regions of the prostate however there is potential to improve the sensitivity of MRI in these regions. As both techniques can be used to detect hypervascularity, both are useful for detecting areas where biopsies will be most effective, however, because of its sensitivity, MRI is probably more useful for this. [24]

Conclusion

All in all, there is great potential for both new and improved ultrasound imaging techniques to contribute to the diagnosis of prostate cancer in the future. Whilst MRI techniques may currently have advantages over ultrasound, future development of the latter may result in their routine use for diagnosis of prostate cancer. These techniques include both Power and Colour Doppler, elastography and microbubble contrast agents to be used alongside standard greyscale ultrasound.

Bibliography

1, 3 – Prostate Cancer, http://www.nhs.uk/conditions/cancer-of-the-prostate/Pages/Introduction.aspx 12/03/16
2, 4, 5 - Christian Nordqvist, Prostate Cancer: Causes, Symptoms and Treatments, http://www.medicalnewstoday.com/articles/150086.php 12/03/16
6 - Prostate-Specific Antigen (PSA) Blood Test, http://www.webmd.com/prostate-cancer/guide/psa 12/03/16
7 - Prostate Cancer: Prostate Ultrasound and Biopsy, http://www.webmd.com/prostate-cancer/guide/ultrasound-biopsy 12/03/16
8 - Tests for prostate cancer, http://www.cancer.org/cancer/prostatecancer/detailedguide/prostate-cancer-diagnosis 12/03/16
9 – Tests for Prostate Cancer, http://www.cancer.org/cancer/prostatecancer/detailedguide/prostate-cancer-diagnosis 22/03/16
10 – Siamak N. Nabili, MD, MPH, Swollen Lymph Nodes, http://www.medicinenet.com/swollen_lymph_nodes/article.htm 12/03/16
11 – Tests for Prostate Cancer, http://www.cancer.org/cancer/prostatecancer/detailedguide/prostate-cancer-diagnosis 22/03/16
12, 14, 15, 16 – General Ultrasound, http://www.radiologyinfo.org/en/info.cfm?pg=genus
12 – Siamak N. Nabili, MD, MPH, Swollen Lymph Nodes, http://www.medicinenet.com/swollen_lymph_nodes/article.htm 12/03/16
http://sperlingprostatecenter.com/mri-vs-color-doppler-in-detecting-prostate-cancer/ 12/03/16
13, 14, 15, 16 – Dan Sperling, M.D., MRI vs. Color Doppler in Detecting Prostate Cancer, http://sperlingprostatecenter.com/mri-vs-color-doppler-in-detecting-prostate-cancer/ 26/03/16
17 – U Patel, Department of Radiology, St George's Hospital, UK, TRUS and prostate biopsy: current status, http://www.nature.com/pcan/journal/v7/n3/full/4500728a.html 22/03/16
17, 18 - General Ultrasound, http://www.radiologyinfo.org/en/info.cfm?pg=genus 12/03/16
19 – Sung Il Hwang and Hak Jong Lee, The future perspectives in transrectal prostate ultrasound guided biopsy, http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4286726/ 22/03/16
20 – The Basics, http://asecho.org/contrast-zone/the-basics/ 22/03/16
21 – Dr Henry Knipe and Dr Marcin Czarniecki, Contrast-enhanced ultrasound, http://radiopaedia.org/articles/contrast-enhanced-ultrasound-2 26/03/16
22 – Image accredited to Dr Leonard Gomella, Thomas Jefferson University, Kimmel Cancer Centre who gave permission for his image to be used. Ultrasound Contrast Agents Help Diagnose Prostate Cancer, http://www.cancernetwork.com/articles/ultrasound-contrast-agents-help-diagnose-prostate-cancer 22/03/16
23, 24 - Dan Sperling, M.D., MRI vs. Color Doppler in Detecting Prostate Cancer, http://sperlingprostatecenter.com/mri-vs-color-doppler-in-detecting-prostate-cancer/ 26/03/16

Programme Officer's Comment:
This is a fantastic article that shows a high level of subject knowledge about the applications of ultrasound. The concepts expressed throughout the article are clearly articulated while maintaining a style that is easy and pleasurable to read. The author has demonstrated a good knowledge of the subject matter and a high-level style of scientific writing. — L. Adamson, Area Director, North Midlands and West, The Scholars Programme

The Memory Impairments in the Clinical Case of Patient H.M.

Year 10, Key Stage 4

Oaks Park High School, Essex
J. Yogarajah, supervised by S. Buck

In this assignment, I will be discussing the memory impairments of Patient H.M. and what he has contributed to the world of brain science by helping us develop a greater understanding of how memory works and the functions

within our brains. I will also include the story of how he came to be an important asset to the history of neuroscience and contributed to great discoveries.

The case of Henry Gustav Molaison most commonly known as Patient H.M., has been hailed as one of the most important patients and case studies in the history of neuroscience. Without the somewhat unfortunate events that occurred during Henry Molaison's life, our current understanding of the brain, specifically the memory function, would not be as good. At an early age, Molaison had a bike accident, which led to a series of unfortunate events. After a few years, H.M. began to experience seizures that overtime gradually got worse and worse. It is unclear if H.M.'s epilepsy was a direct effect of that accident but by the age of 27, his seizures were so severe, they rendered him unable to work.

In 1953, H.M. had an experimental procedure performed by Dr William Beecher Scoville, which involved the removal of part of the brain, specifically the medial temporal lobe structures, including the hippocampus. It was thought that this could possibly alleviate epileptic symptoms.

The surgery controlled the epileptic seizures but left H.M. with unexpected side effects (Milner, B., Corkin, S., et al. 1968). The side effects were unknown to Scoville and his team. Molaison could recall his childhood clearly, along with his family name and history, even the Wall Street Crash in 1929, but struggled to recall events from a few years before (B., Carey, 2008). This is commonly known as retrograde amnesia, which is the inability to recall events that occurred before a trauma. He was later discovered to suffer from anterograde amnesia; the inability to form new declarative memories and therefore was referred to Dr Brenda Milner who also encountered two other patients (P.B. and F.C.) who were also severely amnesic but treated following removal of the medial structures of their left temporal lobe (L. R., Squire, 2010).

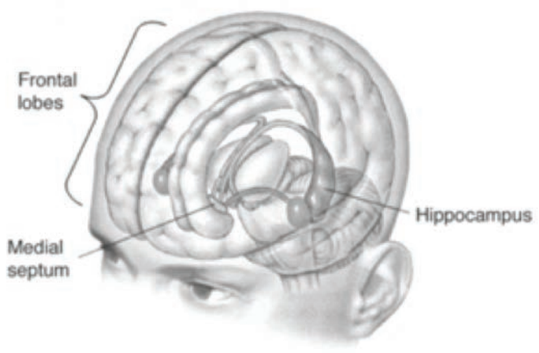

Fig. 1 Parts of the hippocampus were removed from patient H.M.'s brain

Dr Milner conducted trials with H.M. and her most notable discovery was that in the trials, she had asked H.M. to draw a line between two outlines of a five-pointed star whilst watching his hand and the page in a mirror. Dr Milner noticed that even though she asked Henry to repeat the task several times on different occasions, every time Henry could not remember having completed the task before but his performance of it improved. This discovery demonstrated that although he was not conscious of it, Henry had the ability to learn new motor skills by repeated practice. It was also revealed up that he could somewhat form associations between different things, for example first and last names, despite not being able to remember either, or the layout of a house. However, when questioned how he could remember, he often stated that it just felt familiar with no additional explanation. Milner could conclude that this form of memory, motor learning, was distinct from the type of memory that records facts and experiences. She could then conclude that this form of memory was located in a different part of the brain, unaffected by H.M.'s surgery, which meant that we have multiple memory systems located in different parts of the brain. With our knowledge, we can deduce that H.M. could build implicit memory but not explicit memory; meaning that he could not learn specific facts or events, but could still learn new skills or become familiar with his surroundings without specifically remembering them (BigPicture, 2013).

H.M. was also studied by Suzanne Corkin, a former student of Dr Brenda Milner. She had spent 46 years with H.M., however for Henry it always felt like the first time. She discovered that H.M.'s memory loss was quite specific, to the point where he forgot all his experiences after the operation within 30 seconds, and how for more than 50 years, he was only able to acquire the tiniest fragments of self-knowledge (T., Adams, 2013). From this we know that short-term memory only lasts 30 seconds to one minute as H.M. could never transfer these memories to his hippocampus meaning they became lost and forgotten.

H.M. had an encoding impairment linked to his anterograde amnesia. The connections between the hippocampus and cortex, which include the thalamus, hypothalamus and the surrounding cortical structures, were destroyed therefore making it impossible to encode or store memories. Whenever new information was processed, it was forgotten almost immediately since it could never make it to the regions of the brain which end up storing remote long-term memories. Therefore, H.M. could not form new long-term memories, specifically explicit memories, which resulted in him feeling that nothing around him ever changed.

H.M. also had a problem in retrieving long-term memory that had occurred after his surgery, which was linked to retrograde amnesia and consolidation. When new information has been processed and transferred to the hippocampus, the synapses form connections solely between the cortical modules and the hippocampus and, as time passes, each time the memory is reactivated or consolidated, these cortical modules form connections among themselves, meaning they are no longer dependent only on the hippocampus. Therefore, meaning that the older the memory, the better it is preserved and the sharper the recollection of it.

This is interrupted for H.M. due to the surgical removal of his hippocampus, severing those connections before they had a chance to become redundant; leading him to lose some memories. Episodic memory was more severely affected than semantic memory since it had not been fully consolidated and was therefore vulnerable to loss compared to semantic memory, which is everyday facts and knowledge making it more consolidated. However, for H.M., those memories that had not been thoroughly consolidated were lost following the removal of his hippocampus and cortical modules. H.M. allowed us to discover through tests and trials that the medial temporal lobe was not the ultimate storage site for previously acquired knowledge. Despite this, H.M. produced well recited autobiographical memories (unique events, specific to time and place) from when he was 16 years old or younger, therefore demonstrating that H.M.'s remote memory impairment extended back 11 years before his surgery; however, this changed as H.M. began to age. After 20 years, H.M.'s memories consisted more of facts than specific episodes and he couldn't recite an event that may have occurred during a specific time or place. Conclusions were made that the medial temporal lobe was needed for long-term memories over the course of decades to make the memories persist and not be forgotten (L.R., Squire, 2010).

Patients who suffer from the severity of anterograde amnesia that H.M. suffered, witness a huge, life-long impact on their quality of life. This is due to the fact that they cannot live alone or care for themselves as they would forget what they had done an hour ago, which could lead to problems occurring. For example, they would not be able to do simple tasks, such as making food, as they would forget what they were doing and why they were there. Not being able to perform simple life tasks would mean they could die and therefore they would always need to be cared and catered for throughout the day to make sure they are safe. However, those who suffer retrograde amnesia alone will not be as affected as those affected by H.M.'s anterograde amnesia as they would be able to perform tasks and live their life quite normally except that they may have a blank in their memory for a specific time and moment. They have the possibility to live a good life. However, for those with the severity of anterograde amnesia that H.M. had, it would be difficult to have a good quality of life as they would be rendered unable to work, etc.

I believe it is vital that we should continue to study and research H.M.'s conditions due to the fact that this study led to a great discovery in how memory works in the brain. The common misconception was that the memory function was scattered throughout the brain. H.M. helped us realise that it was not and to reassess the whole structure of the brain. It was always recorded previously that the hippocampus, along with the surrounding region, were very important for long-term memory, but never that it was solely responsible for it, allowing us to form the fundamental basis for modern neurology. If it were not for H.M., the knowledge behind neurology would not be as vast and exciting as we have recorded to this day and that it why it is vital to not stop but to keep striving as amazing discoveries from Henry Molaison have led to saving people worldwide.

Even though he tragically died in 2008, he will be recognised and remembered as one of the most important patients in the history of neurology. His unfortunate condition helped to reshape such a vast and intricate field of science. Even though his issues were completely a matter of chance, he still left behind a formidable legacy and contributed to the development of science itself.

References

Milner, B., Corkin, S., et al. (1968). Further Analysis of Hippocampal Amnesic Syndrome - 14-Year Follow-up Study of HM. Neuropsychologia, 215–230.
http://www.rise.duke.edu/apep/pages/001006.html
Benedict Carey (2008) H.M., an Unforgettable Amnesiac, Dies at 82, The New York Times, nytimes.com/2008/12/05/us/05html?
Larry R. Squire (2010). The Legacy of Patient H.M. for Neuroscience – NCBI
Big Picture Education (2013-2014), Inside the Brain, Neuroscience, 'Undergoing Surgery' http://bigpictureeducation.com/brain-case-study-patient-hm
Tim Adams 2013, The Guardian, Henry Molaison: The amnesiac we'll never forget https://www.theguardian.com/science/2013/may/05/henry-molaison-amnesiac-corkin-book-feature
Franklang & Bontempi (2005). The Organisation of Recent and Remote Memories. Nature Reviews Neuroscience, 6, 119-130

About the authors

J. Yogorajah is a very motivated student from Oaks Park High School who demonstrated good understanding of the neuroscience topics we went through in The Brilliant Club tutorials. He had great participation in class and his essays were very well written throughout the weekly assignments.

S. Buck is a PhD student at University College London (UCL) working on a research project investigating memory in children who suffer from epilepsy.

PhD Tutor's note

J. Yogorajah's essay is very clear and structured and demonstrates excellent knowledge and understanding of the topic. He showed great ability to make comparisons between different concepts learned in the course. It has been a pleasure working with Oaks Park High school and the science teachers there.

Should Research in Alzheimer's Disease Focus on Genetic or Epigenetic Disease Susceptibility?

Year 10, Key Stage 4

Fredrick Gent School, Derbyshire
J. Plumb, supervised by K. Boden, University of Nottingham

Essay Abstract – Alzheimer's disease is a neurodegenerative disease that affects 1 in 14 people over the age of 65 and 1 in 6 over the age of 80. This paper will explain the roles of genes and the epigenome and the part they play in Alzheimer's disease, and whether research should focus on genetic or epigenetic disease susceptibility.

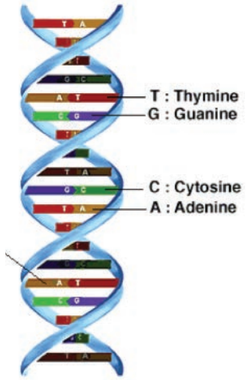

Fig. 1 DNA structure showing nucleotides

Base Genetics

A gene is an ordered sequence of nucleotides, which contain the coding for making a specific protein. They can make enzymes, such as amylase and protease and others made in the pancreas, as well as the proteins that form the base of a human being, which make muscles, organs and hair.[1] Genes create your base phenotype (what you will look like), without factoring in any environmental factors. Genes are made up of DNA (deoxyribonucleic acid). An allele is a different variant of a gene, resulting in a varied phenotypic trait. The recessive allele will be overridden by the dominant and result in the dominant being shown in the phenotype of a child.[2] The DNA molecule is a double helix and has a sugar phosphate backbone. It is twisted to protect the bases inside of it. The bases are pairs and only ever appear in the same base pairs; adenine with thymine and guanine with cytosine (shown in Fig. 1).[3] DNA is found within the nucleus of most cells in the human body. The molecules are packed tightly around proteins called histones to make chromosomes.[4] There are 23 pairs of chromosomes in every cell, half from each parent. They can be XX or XY (female and male respectively). Many diseases and medical conditions are caused by extra chromosomes, such as Down's Syndrome. This is also the case with dominant and recessive alleles. People can also carry genetic diseases; this is why a genetic test is required to see if a person is a carrier and if their child will get the disease.

Gene expression

Gene expression is the process by which the nucleotide sequence of a gene is used to direct protein synthesis and produce structures of a cell. They are expressed as proteins or as ribonucleic acid, which is a single strand, rather than a double helix and its base pairs differ from DNA, with uracil replacing thymine.[5] The promoter region on the DNA strand starts transcription.[6] The transcription factor determines whether DNA can be transcribed into RNA (ribonucleic acid). In this process the DNA is used by RNA polymerase to create mRNA. The mRNA can now leave the nucleus and it goes into the cytoplasm.[7] The gene is then expressed.

Epigenetics

The word epigenetics is derived from 'Epi', meaning above, so epigenetics means above the genome.[8] As individuals grows up, the body triggers chemicals and hormones to activate and deactivate sections of the human genome.[9] Epigenetics is the study of how environmental factors and chemical reactions affect them. It does not involve changes to the underlying original DNA sequence, meaning that a change in the phenotype (what you look like) can happen without a change in the genotype. For example body building is an epigenetic change. Epigenetics hold the answers to a lot of health based questions such as why do some foods cause health problems and others make us healthy and why is it that the older we get, the more likely it is that age-related illness will strike us?[10] Another way of proving epigenetics causes health problems is by looking at two twins, imaginatively named Tim and Jim. They are genetically identical and both leading the same sort of lives in the same space. However like many siblings in modern society, they grow further apart. Jim becomes extremely active, he is healthy, does not drink or smoke and he has a good job. He also has an active social life, along with a wife and children. He continues to stay this way into old age. Tim takes a turn for the worst; he starts smoking and becomes slowly addicted to alcohol. He is obese, unenthusiastic and has no social life. This results in a lack of brain stimulation. Tim then gets Alzheimer's disease at the age of 45.[11] In this example you can clearly see that his disease and health problems were caused by epigenetic changes because Jim was unaffected. His disease was caused by his lifestyle choices and environmental factors. Epigenetic changes may be stable and passed onto offspring.[12] Most biological aspects are affected by epigenetics.

Methylation

Methylation of DNA is an epigenetic mechanism to control gene expression. Methylation can 'turn off' genes.[13] Methylation involves adding methyl groups (C1 H3) to parts of DNA. When this happens, the gene does not produce proteins. Methylation occurs in cytosine, which is paired with guanine and will end up with the methylated cytosines being opposite to each other on a strand of DNA.[14] The problems are caused when genes that control important bodily functions are turned off. It also stops gene expression by stopping the transcription factor from bonding with the promoter. Methylation can come in the form of hypomethylation, a decrease in methylation; or hypermethylation, an increase in methylation resulting in less gene expression.

Alzheimer's disease

Alzheimer's disease is named after the doctor who first described it, Alois Alzheimer. He found that the brain tissue of a woman contained amyloid plaques and tau. Before she died she suffered from language problems, memory loss and erratic behaviour.[15] The amyloid cascade theory suggests that if the Amyloid precursive protein isn't broken down properly, β-secretase 'cuts' the APP in half. γ-secretase (PSEN) then gets rid of the good protein, leaving only Aβ 40/42 (a type of Aβ) resulting in plaques, causing inflammation, which the brain worsens with an immune response that changes the conditions within the brain, destroying cells in the process and creating a large build-up of proteins that may result in Alzheimer's disease, leading to neurodegeneration.[16] This is avoidable if the body properly breaks down the sAPP-α, which is cut by α-secretase, then again by γ-secretase, making p3 and is then degraded and does not cause any plaques.

Alzheimer's Disease is thought to also be caused when the amyloidβ protein secreted and Tau is phosphorylated by a secreted form of kinase A. Phosphates are added to tau, which hypo phosphorylates tau and it starts to clump it together, causing tangles in the brain.[17] The mutation in the APP gene will result in Alzheimer's disease. Genetic factors cause early onset, and late onset is epigenetic (less mobility etc.). The amyloid proteins cannot simply be destroyed as they are vital for life, and we cannot yet distinguish between good and bad, and at the time of writing, we can only target the whole protein.

The first symptom of Alzheimer's disease is the loss of short term memory (the start of dementia), then come speaking problems. The next stage is extreme speech difficulty, loss of some motor skills such as walking at speed or getting out of bed. Long term memory also starts to become affected, which impacts on the sufferer's family hugely as childhood memories and knowledge of family are lost. Angry outbursts become prevalent and the sufferer may become delusional and incontinent. At this stage, full-time care will be needed. Towards the end, as the brain deteriorates even more, sufferers 'forget' how to speak in complete sentences and are bedridden. The brain is shutting down each section of the brain, eventually forgetting how to breath and keep muscles working, resulting in death.[18] [19] [20] Inside the brain, the plaques/tangles start in the hippocampus. It destroys brain cells (neurons), stopping memory slowly. Its spreading around the brain causes the stages of the disease. Balance, co-ordination and breathing are the last to go, resulting in death. Alzheimer's shuts down the brain bit by bit, killing neurons, shrinking it, shrivelling it, leaving the brain of an Alzheimer's sufferer significantly smaller.[21] The disease tends to affect people aged 65 and over (for late onset, early onset is 50+). When an affected person is above this age the rate of risk doubles every five years.

Epigenetic Disease Susceptibility

Environmental exposures early in life have a large impact in later life. Many of these exposures can be permanent and therefore a person may not have any choice, but factors such as gaining weight or being unwilling to perform exercise may become hard habits to break and can result in diseases such as type 2 diabetes and cause problems throughout later life.[22] Some of these environmental effects may be passed on to subsequent generations. In the case of Alzheimer's disease, epigenetics are at fault for causing plaques via a mutation in Aβ peptide by directing processing down Aβ 40/42 route which are 'cut' by β and γ secretase. When the APP and β-secretase are over expressed the chance of getting Alzheimer's will be increased. When γ-secretase is over or under expressed there is also an increase in the chance of getting Alzheimer's. These four factors contribute to late onset Alzheimer's disease, which affects 90% of Alzheimer's disease sufferers and isn't obviously linked to genetic defects.[23] It currently affects people aged 65+ and is also linked with the 14th chromosome. Late onset is currently being researched second to early onset and genetic susceptibility, due to people dying earlier and losing time with their family while some members of it are young (children).[24] Also, late onset is thought to be less important due to people living longer. One of the earliest symptoms of Alzheimer's disease is dementia, which is often misdiagnosed as vascular dementia, and therefore there is often a smaller window of time in which the patient can be helped or at least prepared, as there is no current cure for dementia, vascular or Alzheimer's. Although early onset takes priority currently, 850,000 people have Alzheimer's in the UK alone, most of which is late onset and caused by epigenetic factors and therefore action is being taken to find the cause. However, since late onset is an epigenetic disease, exercising more often, eating healthily and keeping the brain active will prolong and reduce the chance of getting late onset Alzheimer's disease.

Genetic Disease Susceptibility

It is thought that Alzheimer's disease can be inherited via certain genes, and this will cause early onset Alzheimer's disease and is called familial Alzheimer's. It is caused when one of three genes (amyloid precursive protein, presenilin 1 and presenilin 2) mutates. When these mutations happen, the sufferer will get Alzheimer's between the ages of 30 and 40.[25] A total of around 510 families have mutations in these genes making it a very rare disease. A genetic mutation in the apolipoprotein (APOE) gene in chromosome 19 will result in a higher risk of Alzheimer's. APOE ε4 is an increased risk, while APOE 3ε is an average risk, and APOE ε2 has slightly less risk.[26] These genes have only been consistently linked to later onset of Alzheimer's. Hypophosphorylation of tau is still the main cause of early onset Alzheimer's disease, and affects the larger population of early onset Alzheimer's sufferers.

My Opinion

Alzheimer's is a disease that mostly affects the elderly population, but no matter who it affects, the sufferer will die from it. In the end, not being able to fend for myself, having to rely on carers and being bedridden without being able to enjoy life to its fullest concerns me most about Alzheimer's disease. On one hand, research focusing on early onset would allow future sufferers to be able to spend more time with their families and do what they enjoy. On the other hand, being able to grow old (as the average life span is increasing) and watch grandchildren grow is also important, and being able to interact with them while maintaining a good quality of life. For me, research in Alzheimer's disease should focus on epigenetic factors, so that the elderly can enjoy a good quality of life as health care improves. This will go hand in hand with helping reduce Alzheimer's, because the longer a person is able to perform exercise and keep their brain stimulated, the less prevalent Alzheimer's will become. This may leave the minority of early onset patients out, but the 90% majority should take precedence.

Bibliography

[1] http://www.bbc.co.uk/schools/gcsebitesize/science/21c/genes/genesrev1.shtml)
[2] https://en.wikipedia.org/wiki/Allele
[3] http://www2.le.ac.uk/departments/genetics/vgec/highereducation/topics/dnageneschromosomes
[4] http://www.nature.com/scitable/definition/histone-histones-57
[5] http://www.news-medical.net/life-sciences/What-is-RNA.aspx
[6] https://en.wikipedia.org/wiki/Promoter(genetics)
[7] http://www.britannica.com/science/transcription-factor
[8] http://www.superconsciousness.com/topics/science/interview-dr-bruce-lipton
[9] http://learn.genetics.utah.edu/content/epigenetics/
[10] http://www.zymoresearch.com/learning-center/epigenetics/what-is-epigenetics
[11] Tutorial 2, double trouble
[12] http://www.zymoresearch.com/learning-center/epigenetics/what-is-epigenetics
[13] http://www.news-medical.net/life-sciences/What-is-DNA-Methylation.aspx
[14] http://www.nature.com/scitable/topicpage/the-role-of-methylation-in-gene-expression-1070
[15] http://www.dementia.co.uk/alzheimers/what-is-alzheimers-disease
[16]http://wiki.iop.kcl.ac.uk/default.aspx/Neurodegeneration/Amyloid%20Cascade%20Hypothesis.html
[17] http://www.ncbi.nlm.nih.gov/pubmed/21869458
[18] https://en.wikipedia.org/wiki/Alzheimer%27s_disease
[19] http://www.alz.org/alzheimers_disease_what_is_alzheimers.asp
[20] http://www.health.com/health/gallery/0,,20416288,00.html
[21] Tutorial 3- via my notes
[22] http://www.nature.com/nrg/journal/v8/n4/abs/nrg2045.html
[23] http://www.rightdiagnosis.com/l/late_onset_alzheimers/intro.htm
[24] http://www.ncbi.nlm.nih.gov/pubmed/8498809
[25] https://www.alzheimers.org.uk/site/scripts/documents_info.php?documentID=168
[26] http://labtestsonline.org.uk/understanding/analytes/apoe/tab/test/
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PhD Tutor's note
It was a joy teaching at Fredrick Gent School as all the pupils were fantastic. When it came to marking the essays at the end of my placement I was absolutely blown away by the quality of all of the assignments submitted! J.'s essay was particularly impressive, while I was marking it I remember thinking this is better than the essays I was writing during my first years at Uni! He is very deserving of having it published. I am very proud of how hard he worked both during the tutorials, which were often very challenging, and in writing up such a wonderful essay.

Can You Help Cure Cancer?

Year 10, Key Stage 4

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H. Mead, supervised by O. McGinn, University of Cambridge

Humans are made up of around 37 trillion cells[1], each with a different role and different phenotype that allows them to carry out its specific job. A phenotype is any observable characteristic possessed by a certain cell, which is a result of its genotype interacting with its environment[2]: Blues eyes are an example of a phenotype. Every cell's phenotype is essential to its function as without the correct phenotype, the cell would be unable to carry out its job effectively; for example, a red blood cell's job is to carry oxygen to muscles and organs around the body. In order to do this, all red blood cells have a bi-concave shape that allows them to carry large quantities of oxygen to the muscles.

Like all other living things, cells will age and become less effective at carrying out their purpose; due to this, all cells have a specific life span after which they will die and be replaced by another, newer cell. The process of this pre-programmed death is referred to as apoptosis: this is when the cell begins to shrink into itself and form blebs - round growths on the surface of the cell. Proteins within the cell are consequently triggered to break down the components of the cell; enzymes will also begin to break down the nucleus, after which the cell will release a signal summoning a type of white blood cell, called a macrophage. Having contacted the macrophages, the cell will then break down further into much smaller components containing parts of the cells and the destroyed nucleus. Finally, the macrophage will arrive and remove the broken down pieces of cell from the body.

While the process of apoptosis is vital in order for our bodies to continue to function correctly, if cells died without being replaced, we would undoubtedly die also - this is where proliferation comes in. Proliferation is the process in which cells replicate themselves to replace dead cells: they will recognise that another cell is needed to replace the dying cell and will subsequently begin to grow and take in nutrients; they will reach the restriction point where they will either begin to replicate their DNA and go on to divide or will simply die. If they do go on to replicate their DNA, the cell will then prepare to divide and undergo mitosis, producing two genetically identical daughter cells to replace the dead cell.[3]

Both of these processes will occur in normal body cells, however, proliferation will occur at a much faster rate in cancerous cells: cancer is a disease resulting in a malignant growth or tumour due to the uncontrollable division of abnormal cells. There are over a hundred different types of cancer and it has most probably affected humans for as long as they have lived - the earliest record we have of cancer dates back to 1600 B.C. in ancient Egyptian manuscripts.[4] However, the word cancer has been translated from the Greek word carcinos, which literally means crab - it is thought that cancer was named after crabs by the Greek physician Hippocrates as cancerous tumours appear to have multiple crab-like legs stretching outwards.[5]

Nevertheless, cancer has become much more prevalent over the past hundred years for several reasons: newer technologies that we have never used before, such as sunbeds, are known to cause certain types of skin cancers; furthermore, the life expectancy of humans is much longer now than it has ever been, meaning there is more time for our cells to mutate and cause cancerous tumours. On top of this, pollution levels are also higher than they have ever been before and this is creating holes in the ozone layer, which leaves us unprotected from harmful UV rays emitted by the sun, which also cause cancer. Moreover, we now have more advanced technology that can detect cancer - previously, someone may have died from cancer but doctors may have been unaware because they didn't have the medical equipment to diagnose the cancer.

Every form of cancer behaves in a certain manner that allows them to have advantages over normal body cells- these behaviours are referred to as 'hallmarks' of cancer. In January 2000, two researchers, Douglas Hanahan and Robert Weinberg,[6] published a paper based on these behaviours titled, 'The Hallmarks of Cancer'; both authors believe that all types of cancer share certain 'hallmarks' that allow them to undergo changes that transform them from normal cells into cancerous cells - some of their phenotypes have mutated and allow them to perform certain tasks that they wouldn't have previously been able to. The first of the 'hallmarks' described is resisting cell death: this is when cancerous cells ignore signals sent by their local environment informing them to undergo apoptosis. Because they are able to ignore these signals, they are can continue to grow and mutate - unlike other normal cells that would have to perform apoptosis when instructed to do so.

Similarly, cancer cells are also able to sustain proliferative signalling: they do not require a signal from their local environment, like other cells, in order for them to be able to proliferate- this too gives them an advantage above normal cells as they are able to proliferate without a signal or a reason meaning they can divide and replicate at a much faster rate. Cancerous cells are also able to proliferate infinitely (enabling replicative immortality), again, allowing them to have the upper hand against normal cells. Most cells can proliferate only a specific, pre-destined amount of times before they die whereas cancerous cells are able to proliferate limitlessly

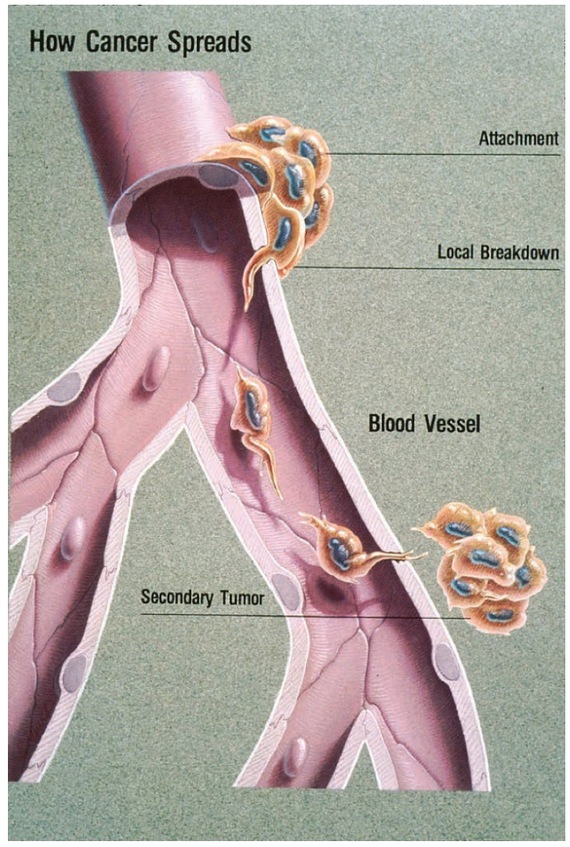
The third hallmark discussed in the paper has been labelled 'inducing angiogenesis': cancerous cells are able to produce blood vessels that will provide them with a direct link to the blood stream. They will use the blood vessels to absorb nutrients from the blood, and to remove waste products, which will then be used to help them to grow and mutate further. This is performed by cancerous cells in order for them to have an ample supply of nutrients and an effortless way of disposing waste. This gives cancer cells an advantage over normal cells as a sufficient supply of oxygen and nutrients is essential to the survival and growth of a cell and, as the cancerous cells are able to absorb these nutrients directly from the blood stream, they will grow much more than a normal cell.

Another hallmark is activating invasion and metastasis: this is the cancer cells' way of spreading and invading other organs and tissues. After inducing angiogenesis, the cancer cells will use the blood vessels to travel into the blood stream and, henceforth, to other organs and tissues where they may grow and cause the development of a second malignant tumour - referred to as a metastasis. This benefits the cancerous cells as, if a normal cell mutated, it would remain in one area and therefore make it easier for the immune system to locate and destroy it whereas, because the cancerous cells can spread around your body, it's much harder for your immune system to locate all the metastases and destroy them - therefore allowing cancer cells to have a better chance of survival than normal mutated cells.

The final hallmark highlighted in this paper describes the cancer cells ability to, once again, ignore signals sent to them by their local environment, ordering them to stop growing - it is referred to as 'evading growth suppressors'. Cancerous mutations will proliferate, and therefore grow, at a much faster rate compared to normal cells; when the brain sends these mutated cells a signal informing them to stop growing, it is simply ignored - something that cannot be done by normal cells. This allows them yet another advantage over normal cells as they are able to grow at an uncontrollable rate without needing to obey the commands of the brain.

In 2011, Weinberg and Hanahan suggested four new behaviors in their updated paper, 'Hallmarks of Caner: the next generation', that all cancer cells share that may too provide them with benefits that normal cells do not have. Deregulated metabolism is the name given to the behavior of the cancerous cells that allowed them to use 'abnormal metabolic pathways' to produce energy; this benefits the cancerous cells as they are able to generate their own energy whereas normal cells would have to use nutrients taken in by the body to create any energy.

The second update to the paper refers to the ability to avoid immune destruction: cancerous cells are capable of hiding from the body's immune system meaning they cannot be located, and therefore destroyed, by the immune system. This is a massive advantage for cancerous cells as it allows them to further grow and mutate without the possibility of being discovered.



Cancer becomes very difficult to treat once it has metastasised.

Furthermore, cancerous cells have many chromosome abnormalities that will worsen the tumour's mutation - this has been labelled mis-matched repair. Due to an environment that promotes further gene destabilisation, the cancer cells will mutate wildly unless treated; this is an advantage of kinds to the cancerous cell as it means that some treatments used to kill the cancer cells will be ineffective.[7]

Finally, the last hallmark discussed proposes the widely accepted theory that chronic inflammation can induce some types of cancer - branded tumour promoting inflammation. While inflammation doesn't necessarily benefit cancer, it can cause it: when a tumour first grows, it is relatively small and can therefore usually find enough oxygen and nutrients to keep it alive - however as it grows, demand for nutrients begins to overtake supply. As the tumour begins to mutate and develop more genetic faults it will, eventually, send chemical signals summoning macrophages from the immune system. The immune system uses inflammation as a way of combating invading bacteria. However, when the macrophages begin to induce

inflammation, they will release molecules that cause the growth of blood vessels that usually help to destroy bacteria and repair any damage - in this case however, inducing angiogenesis allows the cancerous tumour to link directly with the blood stream and therefore provides it with a sufficient source of energy and nutrients, thereby causing the growth and further mutation of the tumour.[8]

Because cancer has existed for as long as humans have, cancer therapies were developed thousands of years ago; the first known cancer therapy was a fire drill. This was a type of surgery that was used 5000 years ago and consisted of using a heated stick to drill into the skin and destroy the tumour- along with a lot of the patient's skin. Obviously, this method of treating cancer was extremely painful and incredibly difficult to perform as the patient was unlikely to have kept perfectly still; because of these two factors scientists have been looking for different ways to numb the pain of surgery for hundreds of years and, in 1846, William T. G. Morton was the first person to demonstrate the use anesthetic for surgical purposes.[9]

Nevertheless, surgery cannot always cure cancer. Firstly, if the cancer has metastasised, it is often too dangerous to operate on and remove the primary tumour as well as the metastases. In addition to this, it isn't always possible to access the tumour, due to our body's complex anatomy. For example, it is incredibly difficult to operate on a blood cancer that has metastasised. When using surgery to treat cancer the cost is, compared to chemotherapy and radiotherapy, reasonably small, however, as previously stated, its effectiveness will vary depending on whether the cancer has metastasised. A possible way to improve surgical therapy could be to identify the cancer before it has metastasised.

Other therapies used to treat cancer include radiotherapy: radiation is fired at the affected cells in order to destroy the genes that govern proliferation - because the genes have been destroyed, the cancer can no longer proliferate and will therefore die. Radiotherapy was first discovered by a German physics professor, Wilhelm Conrad Roentgen, in 1896 and was revealed to the rest of the world in a lecture; Roentgen labelled his discovery the 'x-ray', with x being used to represent an unknown number.[10]

While radiotherapy does have its advantages (shrinking inoperable tumours, controlling the symptoms and relieving pain of advanced cancers) it does have some major downfalls: because your body has a three dimensional structure, when the radiation is fired at the tumour, it may also hit and destroy healthy cells. The main disadvantage of radiotherapy is that, in large doses, it may actually cause further cancerous mutations. In fact, the woman who pioneered radiation died from the cancer that the radiation caused: Marie Curie managed to isolate radium in 1902 after years of working with large quantities of uranium ore; while she knew it was incredibly radioactive, she did not realise that the side effects of working so closely to this radiation would eventually kill her. In 1934, Curie died of a bone marrow disease that was most likely caused by the radioactive material she worked with[11]. Radiotherapy can be quite expensive, with some treatments costing between £15,000 and £18,000[12]; considering it cannot be used to treat metastases and may cause further cancerous mutations, radiotherapy isn't always the best option when treating cancer. One way to improve radiotherapy would be to find a way to get the radiation to the cancerous tumour without damaging the surrounding healthy cells.

Chemotherapy uses a combination of toxic drugs to poison the cancerous cells and is the only main type of cancer therapy that can be used to treat metastases. It was discovered in World War Two after soldiers exposed to mustard gas were found to have abnormal changes to their bone marrow; the US army was studying several chemicals related to or used in mustard gas and, during the course of this work, discovered a compound called nitrogen mustard that was found to work against cancer cells. This discovery prompted further research into similar chemicals and it was eventually found that these toxic drugs were able to destroy cells that proliferated at a faster than normal rate, such as cancer cells. However, because it kills cells which proliferate extremely quickly, one of the most common side effects of chemotherapy is hair loss - as in order for them to grow as much as they do, hair cells proliferate very quickly too. [13]

Nevertheless due to the fact that it destroys only cells that proliferate quickly, chemotherapy is one of the only cancer therapies that doesn't destroy a large number of healthy cells - with the exception of hair cells. While it is incredibly effective in treating cancer, each course costs around £90,000 meaning people in low income countries simply cannot afford this treatment.

Targeted therapy is one of the most recently developed cancer therapies and seems to be one of the most effective. It uses precision medicine to attack the specific genes and proteins that allow cancer cells to proliferate and metastasize therefore, healthy cells aren't damaged in this specific type of therapy as the drugs attack only the cells that have the characteristics of cancer cells. Nevertheless, targeted therapy can cost up to £6890 a month and there are only two targeted therapy drugs that have been licenced for use.[14]

A specific type of targeted therapy, labelled 'antibody drug conjugates,' (ADCs) is being researched by scientists across the globe that are looking for ways to improve the safety and effectiveness of targeted therapy. These biopharmaceutical drugs are able to destroy the cancerous cells without damaging healthy cells; the ADCs consist of a target 'monoclonal' antibody that is biologically connected, using a stable linker, to a cytotoxic drug that will hunt, infiltrate and kill the cancerous growth and its metastases. The stable linkers control the distribution and delivery of the cytotoxic agent so it can pass through the circulation for a pro-longed period of time without harming healthy cells. Once the monoclonal antigen has located the cancerous tumour, it will bind to the target antigen on the surface of the tumour and be internalised into the cancer cells through the process of receptor-mediated endocytosis. Having been internalised, the ADC will undergo lysosomal degradation and release the cytotoxic agent into the cancer cell - the cytotoxic agent will initiate cell death.[15]

The main advantages of ADCs are that they are able to combine the toxic power of the cytotoxic drugs with the targeting ability of the monoclonal antibody to ensure that the maximum amount of cytotoxic agent can reach the cancer cells with minimum collateral damage. Despite this cancer cells can become resistant to ADCs which, obviously, limits their effectiveness and means that more research is needed to develop different types of ADCs .[16]

Scientists are trying to improve this therapy by developing more stable linkers so more potent cytotoxic agents can be used and by enhancing their knowledge of cancer biology and pharmacology.

In order to treat cancer, I would focus on the prevention of invasion and metastasis, as having induced angiogenesis, the cancer cells will use the blood vessels to move into the blood stream and travel to other organs and tissues where they may grow and cause the development of a second malignant tumour. A real world analogy for this could be a flock birds increasing in numbers to the point that some of the birds have to fly south to find a new food supply, leaving the rest of the birds to eat the original food supply. Having flown south, the birds now use a farmer's crops as their food supply. There are a number of ways to solve this problem such as: placing a huge barbed wire dome over the crops that has holes in big enough to allow sun and rain in but too small for the birds to get through. Further solutions include: poisoning the crops, shooting the birds, not planting any crops or to use a scare crow to frighten the birds.

If the first solution, using a barbed wire dome to protect the crops, was to be translated back into a cancer therapy, it would consist of engineering cells to surround and encase cancerous cells before they begin to invade the rest of the body or even before they induce angiogenesis. The cells could work together to form one giant cell membrane type dome around the tumour which wouldn't allow nutrients to come in or waste to go out.

Adult stem cells could be engineered into these cells that would have similar properties and phenotypes as a regular cell membrane; these cells could be connected to antibodies (that would carry them to the tumour) in a similar way to how cytotoxic agents are connected to antibodies to form ADCs. Once the cells have become specialised and have been stably linked to the antibody, they would be injected into the blood stream so they could act as quickly as possible. Once the tumour had been isolated and surrounded, the cells would control which substances could pass in and out of the tumour - obviously, anything that would help the tumour to grow would be prevented from entering and anything that the tumour needed would be allowed to leave. When the tumour had been starved of enough nutrients to kill it, the engineered cells could work alongside the immune system to surround any remaining cancerous mutations or could simply be used to limit the reproductive capabilities of viral or bacterial infections until the immune system was able to develop an antibody to destroy it.

However, there are several downfalls to this therapy: it would most likely be very costly to research and develop methods that would allow scientist to engineer cells into anything they wanted. Furthermore, because the cells will have been genetically engineered, they may have the capability to mutate and cause another cancerous tumour which, because of the nature of the therapy, would surround the original tumour, potentially causing serious complications. In addition to this, the engineered cells would be most effective at starving and killing the cancer before it has induced angiogenesis and metastasised meaning this therapy may only work on patients who have an early stage cancer.

Due to complexity and expense I think it unlikely that this form of therapy would be developed in the immediate short term, probably requiring 10 to 20 years to develop. Given that 7.9 million people die of cancer globally[17] each year, if this therapy were to be successfully developed in the next decade, 79 million people could have already died of cancer.

Nevertheless, this therapy does have its advantages: because the cells would starve the cancer of the nutrients it needs - without the use of potentially harmful drugs- the cancerous cells would die, achieving the same outcome as other, more dangerous therapies, such as chemotherapy and targeted therapy that use poisonous substances. To build on this, because no harmful drugs would be required, this therapy would not cause damage to other healthy parts of the body like most other therapies would. Likewise, because this therapy, in theory, wouldn't be harmful to the body, it could be used to destroy metastases. Finally I believe that this solution could be reasonably cost effective if scientists are able to reduce the cost of genetically modifying cells as the therapy could kill the cancer quite quickly and would be able to attack any metastatic growths.

Bibliography

1 ROSE EVELETH 'THERE ARE 372 TRILLION CELLS IN YOUR BODY',http://www.smithsonianmag.com/ist/?next=/smart-news/there-are-372-trillion-cells-in-your-body-4941473/ [2ND APRIL 2016]
2 OXFORD UNIVERSITY AND R.E.ALLEN (1990) THE CONCISE OXFORD DICTIONARY, PAGE 893
3 Brilliant club and Owen McGinn (spring 2016) can you help cure cancer? Page 11
4 'History of Cancer,' Ancient and Modern Treatment Method', http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2927383/, [2ND APRIL]
5 Dr Ananya Mandal, MD,' Cancer History', http://www.news-medical.net/health/Cancer-History.aspx , [2nd APRIL]
6 'HALLMARKS OF CANCER' https://en.wikipedia.org/wiki/The_Hallmarks_of_Cancer [2ND APRIL]
7 'The Hallmarks of Cancer' (UPDATES) https://en.wikipedia.org/wiki/The_Hallmarks_of_Cancer#Updates [2ND APRIL]
8 'Feeling the heat – the link between inflammation and cancer' http://scienceblog.cancerresearchuk.org/2013/02/01/feeling-the-heat-the-link-between-inflammation-and-cancer/ [2nd April]
9 Brilliant club and Owen McGinn (spring 2016) can you help cure cancer? Page 18 [2ND APRIL]
10 Brilliant club and Owen McGinn (spring 2016) can you help cure cancer? Page 19 [2ND APRIL]
11 'Marie Curie the scientist' https://www.mariecurie.org.uk/who/our-history/marie-curie-the-scientist [2ND APRIL
12 http://www.theprostatecentre.com/prostate-centre-services/prostate-cancer-treatments/radical-radiotherapy/
13 Brilliant club and Owen McGinn (spring 2016) can you help cure cancer? Page 20 [2ND APRIL]
14 'Targeted Therapies Offer Promise, But Are They Affordable?' http://www.medscape.com/viewarticle/810147 [2nd April]
15 What are Antibody-drug Conjugates (ADCs)?' https://www.youtube.com/watch?v=GD0gcZoqtcM&feature=youtu.be [2nd April]
16 Terry Chapman, 'ADCs: Pros and Cons' http://mabstalk.com/2015/01/27/adcs-pros-and-cons/ [2nd April]
17 'World Cancer Day 2013 - Global Press Release' http://www.worldcancerday.org/world-cancer-day-2013-global-press-release [5th April]

Programme Officer's Comment:
This is an engaging article that grabs the reader's attention from the first sentence. The article is written in a good scientific style throughout and demonstrates an in-depth understanding of the subject. It is a pleasure to read such a great article that clearly demonstrates the pupil's engagement with the course and high-level subject knowledge. —L. Adamson, Area Director, North, Midlands and West, The Scholars Programme

The Lab Report: Testing the Serial Position Effect

Year 10, Key Stage 4

Preston School, Somerset
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Abstract
A test on 12 students has managed to identify both the primacy and recency effect. That is, the first few and last few items on a list are easier to recall than the words in the middle. This is due to short-term and long-term memory, either retaining a group of words for a small amount of time or retaining a group of words for an extensive period.

Introduction
The human mind is a vast, complex web of components that together make the most sophisticated natural computer the world has ever seen. Incidents in the past have demonstrated just how important the mind is to our lives and how there are still many things we haven't come across yet. Patient H.M. had surgery at the age of 27 in 1953 (Steinvorth et al, 2005). Since then, researchers have spent more than 50 years discovering how important the hippocampus is when forming new memories. Patient H.M. was unable to remember anything beyond the first 27 years of his life. Meanwhile, whilst he was studying at college, Rajan Mahadevan managed to recall the first 31,811 digits of pi (Thompson et al, 1993). He averaged 3.5 digits per second. These cases are able to show that the mind processes information into quick memorisation and permanent storage. The purpose of this research is to test how well the mind can remember things and exactly what it remembers.

There are several key terms to understand before reviewing the results of this experiment. According to the three-stage model by Richard Atkinson and Richard Shiffrin (1968) there are three components to memory: Sensory, short-term and long-term memory. Sensory memory briefly holds incoming sensory information (Passer & Smith, 2009). Short-term memory (STM) is a memory store that temporarily holds a limited amount of information, whereas long-term memory (LTM) is our vast library of more durable stored memories (Passer & Smith, 2009). However, scientists now deem STM too passive and prefer the term 'working memory' - a limited-capacity system that temporally stores and processes information (Baddeley, 2007).

The Serial Position Effect refers to the findings that the chance of retrieving an item is dependent on the item's position in a list (Passer & Smith, 2009). There are two aspects to the Serial Position Effect: the primacy and recency effect. The primacy Effect describes the ability to quickly encode the first few items on the list and transfer them to LTM; the recency effect describes the ability to encode the last few items of a list, due to the fact that they're stored in the STM and aren't challenged by any more words needing to be memorised.

This experiment was designed to put the Serial Position Effect to the test. It was hypothesised that the first few and last few words of the list presented would be recalled more accurately compared to the words in the middle. This would be due to the effects of STM and LTM. The independent variable was the list of words presented and the dependent variable was how many words could be recalled correctly and in the right order.

Material and Procedure
Ten words were presented, each one taken from a similar experiment by Baddeley, Thomson and Buchanan (1975). The words appeared in two different orders, with the first order being the following: Puma, Measles, College, Peru, Blackpool, Kettle, Physics, Utah, Essay and Carbon. The words themselves were shown in a PowerPoint presentation. In total it took fifteen seconds for all the words to be shown, during which time the participants were not given any equipment to be able to write the words down; by writing the words as they appeared the participant would not only be defeating the objective of the experiment but would also be missing other words appear as they glanced at their paper. Each word was presented for one second; there was also a 300 millisecond delay between the words. Participants had two minutes to recall the material in the correct order - this was a serial recall. The two groups saw slightly different orders of the words to control for the effects of individual words. For the first part of the experiment the students had no equipment - once the presentation was finished, the students were given a pencil and a blank piece of paper to write down as many words they could remember in the order they were presented.

Scoring
In order to fully distinguish the primacy and recency effect, the words were divided into three groups according to their position. The first three items of the test were chosen to identify the primacy effect, whereas the last three were chosen to identify the recency effect. Performance for the first and last three words was compared to performance for the middle words.

An item was scored as correct depending on two criteria: if it was the right word and if it was in the right position. For example, if the word 'Blackpool' was recalled but wasn't in the right position it was not counted as correct. However, if the participant scored five out of ten words but the last two in his or her list were the last two of the presentation, they were scored as correct.

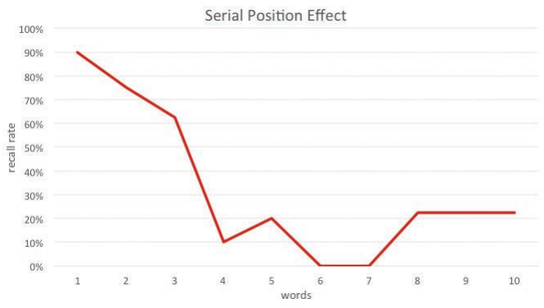

Fig. 1 Recall rates for the ten words as a function of word position. The findings show a clear primacy effect. A recency effect was also observed.

As the graph shows in Figure 1, the primacy effect had a clear recall rate along with a notable recency effect. The results perfectly support the idea that people are more likely to recall the first few and last few words of a list, due to the LTM cementing earlier information and the STM receiving later information.

The very first word to appear had the highest recall rate of 90%, since it had remained in the working memory longest and was the first to enter the declarative LTM. The fourth word had a significant drop in recall rate (down to 10%) from the third word (60%). Thus, despite the fifth word being slightly higher, perhaps meaning an inconsistency in STM and LTM - this was the beginning of the participant failing to fully remember and recall the words. The last three words - all showing a recall rate of just above 20% were an improvement and shows that some participants were able recall them.

Discussion
The results supported the hypothesis. There is no doubt that there is a primacy effect, although the recency effect is significantly lower despite being identifiable. However, the fact that the recency effect wasn't as high as the primacy effect suggests that the brain can be too busy processing a few words at the beginning to accurately recall the rest of an entire list. Therefore, the results suggest that the primacy effect is the more dominant of the two in this study.The reason for this could be that although the words in the STM are the first to perhaps be recalled, the brain can only concentrate on one task at a time, which is the retrieval of the information from LTM.

The delay of 300 milliseconds may have also caused the primacy effect to dominate the recency effect. Some studies suggest that a short delay improves rehearsal. However, a delay can cause some items to decay in memory over the time of the whole-list presentation. The fifth word is an interesting result and it can be seen as an exception. The primacy effect clearly ended around the fourth word and the next three words were expected to be incredibly low as they were the middle set of words. The fifth word has the highest recall rate of all the middle words and is almost at the same percentage as the words in the recency effect. This might be where the experiment is dependent on the individual participants. It's as if one or two participants realised that they hadn't membered the fourth word during the presentation. Upon noticing their failure to remember the word they gave more focus to the next word - the fifth one - before becoming overwhelmed by the rest of the sequence and failing to remember the middle words. This could also be another explanation for the recency effect being lower than expected - the participant is too conflicted by the idea that they missed the fourth word that they leave themselves lingering on the fifth word and do not give the rest of the sequence enough attention. Words six and seven had a recall rating of 0%. It is at this point that the participants were having the greatest struggle in encoding the last half of the sequence.

This experiment manages to reflect the idea that people's attention can only be controlled in one area. Perhaps, trying to give your mind more than one or two things to think about will result in the mind failing to focus on all of its required tasks.

The test that was carried out mirrors that of Glanzer and Cunitz (1966). Their test showed that without a delay, the primacy and recency effect are almost as strong as one another. They also performed their test a second time, but this time with a deliberate delay and the recency effect was quite low.

In the future, this experiment could be improved by following Glanzer and Cunitz (1966) more closely - that is, to perform the test again without a delay. One may also try different variations of the test, by changing some of the conditions. For example, the experimenter could show the presentation in darkness, with the lights off, and get participants to recall the words with the lights on. Here, one could also be testing the encoding specificity principle. The principle states that recall is better if the retrieval context and the encoding context are the same. This new, modified test will explore how much the two contexts can differ for recall to be affected. A second variation might be to show a series of pictures and get the participants to recall the images as words - forcing them to process the word from the image before recalling it. A delay on this second variation might be vital to give a participant enough time to process the image to the word. The brain will be encoding through visual cues and recalling through phonological cues. This means that the visuospatial sketchpad and the phonological loop will be put to the test. The visuospatial sketchpad briefly stores visual information whereas the phonological loop stores acoustic codes (Passer and Smith, 2009).

In conclusion, this experiment manages to support the idea that the brain processes information, which is stored as short-term or long-term memories. As this study shows, one is more likely to recall the first few and last few items of a list when immediate recall is required. The longer something remains in our memory, the easier it is to remember.

References
Atkinson, R. C., & Shiffrin, R. M. (1968). Human memory: A proposed system and its control processes. In K. W. Spence & J. T. Spence (Eds.), Advances in the psychology of learning and motivation: Research and theory, 2. New York: Academic Press.
Baddeley, A. (2007). Working memory, thought, and action. London: Oxford University Press.
Baddeley, A.D., Thomson, N., and Buchanan, M. (1975). Word length and the structure of short-term memory. J. Verb. Learn. Verb. Behav. 14, 575–589
Glanzer, M., & Cunitz, A. R. (1966). Two storage mechanisms in free recall. Journal of Verbal Learning and Verbal Behavior, 5, 351–360.
Passer, M. W., & Smith, R. E. (Eds). (2009). The science of mind and behaviour (4th Ed.). New York: McGraw-Hill.
Steinvorth, S., Levine, B., Corkin, S. (2005). Medial temporal lobe structures are needed to re-experience remote autobiographical memories: evidence from H.M. and W.R. Neuropsychologia, 43:479–496. doi: 10.1016/j.neuropsychologia.2005.01.001.
Thompson, C. P., Cowan, T. M., & Frieman, J. (1993). Memory search by a memorist. Hillsdale, N.J: Erlbaum.

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PhD Tutor's note
S. Finlayson's work shows strong evidence of critical thinking and in-depth understanding of the concepts introduced throughout the course. His writing is coherent and organised and his discussion extends the readers' knowledge on the subject. Overall, it is an outstanding piece of academic work. As a tutor, it is always nice to work with pupils that are eager to learn. This pupil engaged actively in discussions during the tutorials and showed great interest in the subject. Although the course was very demanding, all pupils rose to the challenge and successfully completed the course. It was a great pleasure and an inspiring experience working with them as a Brilliant Club tutor.

In Search of Maximum Coin Sorting Efficiency, Through the use of K-Means Clustering on 45 Coins of Nine Different Types with Information on Diameter and Mass of the Coins

Year 12, Key Stage 5

Primary Investigator: G. Mullahasani-Dula, Group Members: S. Mehta, D. Burke, B. Farkhas, Supervised by D. Birrenkott

Introduction

The issue that I am trying to solve in this project is how can you sort a random assortment of coins using a machine learning algorithm, more efficiently than a human could. A machine learning algorithm is an algorithm executed by a computer which adjusts its performance through experience and by learning from any mistakes it makes on the way. One type of machine learning is supervised machine learning, which is where the computer uses information it is given and makes a prediction for an output of data given an input of data. The input of data being information the computer uses, given by the user, about a certain situation or task and performs or displays the appropriate information to the user, which is the output. Another type of machine learning is unsupervised machine learning. This is where an input of data is given to the computer (such as the diameter and mass of a coin, in our case) and the computer categorises and groups the data into clusters, which are then shown or output to the user. The machine learning algorithm that will be used in this project is K-means clustering. This algorithm is an unsupervised machine learning algorithm that will be used to sort the coins in their correct clusters. This type of machine learning is being used because the coins are being grouped, so it is appropriate to be using unsupervised machine learning.

Coin sorting is a crucial ability for banks and large company organisations such as charities to have, so the they can manage the currencies that are given to them and sort the coins to perform easy exchange between different valued coins.

During the 19th Century, coins used to not only be sorted and inspected by tellers, but they had to be counted by the teller.[1] Machines were developed in 1919 to increase the speed of the sorting process making them equal in speed to the old timers of banking fame.[2] However, in the late 1920s, the counting process became mechanised by developing a machine called the Federal Bill Counter, which tallied the number of coins and notes that were sorted by the tellers. This prevented the need for the tellers to count and so reduced the error of the number of coins and notes counted, since humans are more likely to make the mistake of miscounting. It also allowed increased speed in the sorting process since the Federal Bill Counter was much faster at counting than a normal human.[2] Later, during the 1960s and 1970s, a new technology was developed which sorted coins and authenticated them using ultraviolet light and magnetic sensors. However, this technology was still unable to distinguish between unfit notes and coins and legitimate ones.[2] This is where a revolutionary development was made in the 1980s with the introduction of the Fed's first computerised currency counting equipment, the REI High-Speed Machine. This was not only able to recognise whether notes and coins were legitimate or not, but it also increased the speed of sorting and accuracy. From then on coin sorting changed completely and was made much more efficient.[3]

The way coin sorting is performed by some mechanical machines is very simple and effective. They use the size of the coin and have varying sized holes that are in ascending order, so as the smaller coins go through, they are separated earlier before reaching the larger sized holes, meant for larger sized coins.

Machine learning will not only further speed the process of coin sorting but also sort the coins with maximum accuracy.[4] Machine learning has been used in coin recognition with a Rotation-Invariant Neural Pattern Recognition system that observes the coin at different angles. The coin is observed on its reverse side and obverse side and so coins that are similar in size and weight are very accurately differentiated by the machine learning system after it has had a bit of experience with the data. The aspect of accuracy is a very important factor and reason as to why the K-means clustering algorithm is being proposed in this project for coin recognition.[5]

Some pattern recognition systems, when sorting different currencies, have a recognition pattern set, (RPS) and any coins that don't belong to that set are rejected. This is a very useful technique when there are coins that could be fake versions of those different currencies, this helps to reject those coins. The major reason that machine learning is such a viable option for coin sorting is its accuracy, especially after undergoing many iterations (large amount of past experience).[6] Even experts in their own fields are being taken over by machine learning, with the large amounts of data that machine learning can use to make better judgements on certain tasks and hence have better precision and accuracy than human beings. This can be crucial when it comes to coin sorting and determining whether a coin is fake or legitimate and what type of coin it is.

The hypothesis for this project is that with the appropriate use of a machine learning algorithm, sorting will be done much more efficiently than by a human, by increasing the speed and accuracy. K-means clustering will be used with data such as the diameter of the coin (mm) and the mass of the coin (g). It is expected that the machine learning algorithm will have an error rate close to 1 as the K-mean clustering algorithm is so effective, especially with enough iterations. The algorithm should be able to classify almost all coins in their correct clusters, with the exceptions of some coins that might be measured with an error caused by factors such as dirt on the coin increasing the mass.

Method

The machine learning algorithm that will be used is the K-means clustering. This is an unsupervised machine learning algorithm, which is a method used to categorise different data sets into groups based on similar characteristics. This is done by reducing the Euclidian distance between each point and the ideal centroids for each cluster for every repetition. In the case of coin sorting, the method will be using data for the weight of the coin and the area of the coin, so that the same coins are clustered into a group if they have similar weights or areas.

A weighing scale will be needed to measure the mass of each coin. A Vernier scale is used to measure the diameter of each coin. The programming language that will be used is Octave, which is appropriate since it does specialise in numerical methods, so that the K-means clustering algorithm can be performed more quickly by the computer. Firstly, the values for the diameter of each coin is recorded on a table (the values are under the variable name) and the values of the weight of each coin (under the variable name.) Then, appropriate points for the centroids must be selected (the X value being under the variable name and the Y value being under the variable name.) Afterwards, the following calculations for each coin for one of the centroids is performed:

These calculations are repeated for every other centroid. When the Euclidian distance has been calculated for each of the coins between each centroid, the coins are then grouped to clusters. If the coin has the smallest Euclidian distance between centroid 2 then its X and Y values are recorded in a table called cluster 2. If it has the smallest Euclidian distance between centroid 1 then it is recorded in a table called cluster 1, etc. The new X and Y values for each centroid are then calculated.

Once the new centroid points are found, this entire process is repeated. It is repeated until there is no change in any of the centroids, and so once it stops performing the K-means cluster algorithm, the coins will have been grouped and so it will be known which coins are which, based on the final cluster that they are in.

When the results for the coin data are received using the K-means clustering algorithm, the error rate of the results is found.

The number of correctly classified coins is found by looking at the results of the algorithm and comparing it with the known data that has been collected and seeing whether each coin type has been grouped in the correct cluster or not. (The pseudo code for the algorithm is at the end of the report.)

Results

In this experiment the results collected were very accurate with minimal error made in the denomination of the different coins. From the algorithm, the amount of coins that were correctly classified was 42 out of the 45 coins in total. This meant that it had an error rate of 0.933 which means that 93.3% of the time, it correctly classified the coin sample which is what was expected in the hypothesis (for the error rate to be near 1). The set of coins that were being classified were the following:

- US 1 cent
- US 5 cent
- GB 1 penny
- GB 2 penny
- GB 5 penny
- CAN 5 cent
- CAN 10 cent
- EURO 1 cent
- EURO 5 cent

The coins that were incorrectly classified were 3 Canadian 5 cent coins. They were classified by the algorithm as being European 5 cent coins. This is most likely due to it having a very close diameter and mass measurements of the European 5 cent coins. It could also be due to an error in the measurement of that particular coin, since the measurements did look like overestimates when compared to the diameter and mass of other Canadian 5 cent coins. So the errors weren't necessarily due to the algorithm, instead it might have simply been due to human error when measuring the different coin characteristics.

Figure 1 is a graph plotted by the algorithm on the programming language Octave, showing the points of each coin and the centroids.

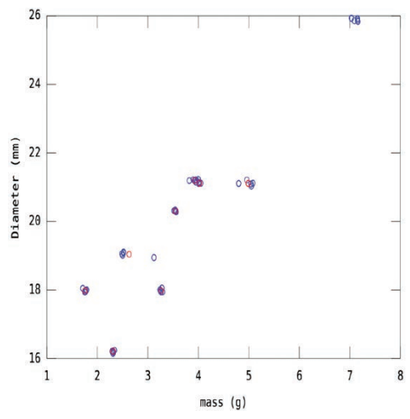


Figure 1: The graph is showing plots of all the different coins with their diameter's and mass' (they are plotted as blue circles.) The centroids have also been plotted as red circles to visualize the grouping of all the different coins.

Not only has the machine learning algorithm produced very accurate results, but the speed at which the coins were classified is a major advantage. The experiment took a mere two seconds once the algorithm was run and this would have taken a human being hours to perform.

Discussion/Conclusion

The hypothesis in this report was, if a machine learning algorithm (the K-means clustering) was used to sort a group of coins, that it would do it more efficiently than a normal human. The results showed that the error rate for the algorithm was 0.933, which agrees with what was expected in the hypothesis (for the error rate to be near 1), even for a large number of clusters and coins. Moreover, the speed at which the algorithm performed the sorting was only two seconds. This would normally take a human hours to do,

which overall, shows it is the more efficient option when it comes to coin sorting. These results clearly support the hypothesis by demonstrating that the important factors of coin sorting (speed and accuracy) are all exceeding a human's ability with the use of machine learning (in particular unsupervised machine learning: the K-means clustering algorithm).[7]

The next step in this project will be to implement a calibrated camera, that way, the computer not only has data on the diameter and mass but also visual aspects of the coin. This will give the computer and machine learning algorithm a better way to differentiate between the very similar coins. It also helps to better identify fraudulent coins by analysing any visual features that might separate it from coins with similar diameters and masses.[8] The machine learning algorithm will also use the density of the different coins, found by dividing the mass of the coin by its volume. Density is found to be a great discriminator of coins as each coin will have their own unique make up of alloys. This will further increase the precision of the machine learning algorithm's sorting and grouping process of each of the coins.

Overall, what has been learnt is that the process of coin sorting can be performed much more efficiently with the appropriate application of machine learning algorithm than by humans who are much more prone to error and are leagues behind in computational power. The next steps in this project will only further improve the machine learning's already impressive performance and so lead to near perfect results for coin sorting.

Bibliography

[1] W. Kaempffert (ed.), "Counts and wraps coins quickly and accurately," Popular Science Monthly, vol. 94, no. 2, pp. 68, Feb. 1919.
[2] History Of Currency Counting At The Federal Reserve Bank Of Philadelphia (n.d.). Federal Reserve Bank Of Philadelphia. (Online). Available: https://www.philadelphiafed.org/education/teachers/resources/history-of-currency-counting#1960s/70s Accessed: 2016
[3] How Do Coin Counters Work? (n.d.). Thomas McNish on eHow. [Online]. Available: http://www.ehow.com/how-does_6172153_do-coin-counters-work_.html Accessed: 2016
[4] Minoru Fukumi, "Rotation-Invariant Neural Pattern Recognition System With Application To Coin Recognition", IEEE Transactions On Neural Networks, vol. 3, no.2, pp. 272-274, March. 1992.
[5] Talbot H., Ourselin S. and Adriaansen T., "Dagobert – A New Coin Recognition and Sorting System", pp. 10-12, Dec. 2003
[6] Y.S Abu-Mostafa, "Machines that think for themselves," Scientific American, vol. 307, no. 1, pp. 78-81, Jul. 2012.
[7] V.Murino and E. Puppo (Eds.), "Counting Turkish Coins With A Calibrated Camera", Department of Electrical and Electronics Engineering, Anadolu University, Eskisehir, Turkey, pp. 216-226, 2015.
[8] TTD plans to install coin-sorting machine. (n.d.). The New Indian Express. [Online]. Available: http://www.newindianexpress.com/states/andhra_pradesh/article245073.ece?service=print. Accessed: Nov 18, 2011.

Programme Officer's Comment:
This is an impressively well-researched and detailed article. It combines both high-level subject knowledge with a clear explanation of the subject and methods used. The abstract provides a concise overview of the article and, along with the thorough introduction, gives even a non-expert reader a clear understanding of machine learning. In the more technical sections of the article the author manages to continue to communicate in an accessible manner. The article is carefully referenced and demonstrates an impressive command of the subject. Well done!
—D. Jones, Teaching and Learning Manager, The Scholars Programme

Pseudo-Code

```
import coinData and centroidsOld
count = 0
meanVal = 1
WHILE meanVal > 0.0001 THEN
        FOR n = 1 to LEN(coinData) THEN
                FOR m = 1 to LEN(centroidsOld) THEN
                        euclidean(n,m) = SQRT((coinData(n,1) –
centroidsOld(m,1))^2 + (coinData(n,2) – centroidsOld(m,2))^2)
                ENDFOR
        ENDFOR
        FOR i = 1 to LEN(euclidian) THEN
                [~, clusterIndex(i,1)] = MIN(euclidean(i,:))
        ENDFOR
        storePoint = []
        FOR q = 1 to LEN(centroidsOld) THEN
                FOR p = 1 to LEN(clusterIndex) THEN
                        IF clusterIndex(p,1) == q THEN
                                storePoint(end+1,:) = coinData(p,:)
                        ENDIF
                ENDFOR
                centroidsNew(q,:) = MEAN(storePoint)
                storePoint = []
        ENDFOR
        centroidsOld =SORTROWS(centroidsOld)
        centroidsNew = SORTROWS(centroidsNew)
        meanVal = MEAN(MEAN(centroidsOld – centroidsNew))
        centroidsOld = centroidsNew
        count = count + 1
ENDWHILE
DISP(clusterIndex)
DISP(centroidsOld)
FIGURE
SCATTER(coinData(:,1), coinData(:,2))
XLABEL('Diameter (mm)')
YLABEL('Mass (g)')
HOLD ON
SCATTER(centroidsOld(1:8,1), centroidsOld(1:8,2), 'r')
HOLD OFF
```

Arts and Humanities

Were Victorian Asylums a Superior Solution to Earlier Methods of Treating and Containing the Mentally Ill?

Year 9. Key Stage 4

Lampton School, London
H. Bhatti, supervised by L. Neff, Royal Holloway

In this essay, I will compare and contrast the care and treatment of the mentally ill in Victorian asylums with methods used in earlier times, stretching right back to the Neolithic age. My aim is to show that, although the Victorians made huge strides in this area, their treatment of the mentally ill in asylums was not necessarily better, outside of a few progressive pockets.

Until the 20th Century, the mentally ill were treated with a fear and loathing that arose from a basic misunderstanding of their condition. This attitude was largely fuelled by religion and superstition. For example, Christians and Jews believed that mental illness was the result of divine retribution for past sins.[1]

Hindus believed in the concept of 'nazar' or the 'evil eye' and blamed mental conditions on it. [2] Muslims believed in Djinns, largely mischievous spirits who have the power to possess humans. Often, these beliefs helped religious authorities push their own agenda by both helping to explain mental illness in purely spiritual terms and by scaring the public into following religious rules and behaving 'appropriately', to avoid being cursed too.

Even before organised religions developed, belief in witches and demons was widespread in societies all over the world. In Neolithic times, people believed mental health issues were the result of evil spirits inhabiting a person's head, while the ancient Persians thought that adequate hygiene, and purity of the mind and body helped ward off evil spirits. Certain societies, including many in Europe, held the idea that mental illness was hereditary and passed on from one generation to the next through a 'bad bloodline'. By contrast, in China, people thought the mentally ill suffered from a 'bad fate' that was contagious and so they were best avoided.

Such beliefs lead to a range of cures and treatments being developed. The most common was simple confinement of the patient, usually in their own home. While this could result in kind and sympathetic treatment of the ill by close family members, it more often resulted in the opposite, because of the shame, neglect and social stigma associated with mental illness. Patients were hidden away in cellars, caged, starved and abused or left to be looked after by neglectful servants. Indeed, custody and confinement of mentally ill patients was officially sanctioned, as can be seen from this quote from the Greek philosopher Plato in his book, The Laws:

"If a man is mad he shall not be at large in the city, but his family shall keep him in any way they can." [3]

If home care was not an option for them, or they were considered harmless, then patients were often cast out and left to wander the streets alone. While this may have saved the mentally ill from domestic abuse and given them a certain level of freedom, it also meant they were exposed to abuse from strangers and forced to beg for scraps of food in order to survive. It was a very different story for the most extreme cases, though, as Allison M. Foerschner says in her essay on the topic:

"Those who were deemed dangerous or unmanageable, both in family homes or on the streets, were given over to police and thrown in jails or dungeons, sometimes for life.".[4]

Other, much more gruesome, treatments were also used to deal with or treat the mentally ill. For instance, trephining (also referred to as trepanning), which involved drilling a hole in a patient's skull to release evil spirits, was used from Neolithic times right through to the Middle Ages. In ancient Mesopotamia, priest-doctors used magic, incantations and other mystical rituals, as well as threats, bribery and punishment, to drive out bad spirits. Through the ages, religious institutions have offered amulets that contained astrological symbols, St. John's Wort or prayers on rolls of paper that could be worn around the neck, to ward off malevolent spirits.[5] If these methods failed, then the religious could arrange for the mentally ill to be exorcised, to drive out any demons possessing them.

A trepanned Neolitic skull

It should be stressed, however, that not all ancient treatments and cures were brutal. Progressive methods were developed as far back as Egyptian times. They encouraged the use of music, dancing and painting to relieve symptoms and achieve some sense of normality in the lives of sufferers. They were also the first people to identify that mental problems stemmed from the brain, an idea developed further by the Greek physician Hippocrates many centuries later. [6]

The Roman physician Galen introduced the concept of the four essential fluids of the human body: blood, phlegm, bile, and black bile, the combinations of which produced the unique personalities of individuals. His theory endured into the Middle Ages. Physicians of that era believed that mental illness resulted from an imbalance of these humours and could be cured by emetics, laxatives or leeching/blood-letting.

Later, in the 18th Century, German physician Franz Mesmer used hypnosis and his theory of 'animal magnetism' to cure the mentally ill.[7]

In addition, civilizations as diverse as the Greeks and the Arabs developed various herbal concoctions and special diets for treating mental health issues. After the discovery of the New World, tobacco imported from America was popularly used to induce vomiting as a cure. Although the ideas they promoted were often unhelpful, religious institutions played a crucial role in the care of the mentally ill too. Before the advent of official asylums, especially in Europe, churches and monasteries often took in and cared for the mentally ill.

The provision of care and treatment for mentally ill patients did not alter substantially until the Victorian era. Increasing industrialisation and urbanisation, particularly in Britain at the time, resulted in social dislocation and lead to an explosion in the numbers of the mentally ill; it is estimated that their numbers increased tenfold between 1800 and 1900.

In response, the Victorians first tried to leave the provision of asylums to market forces, but this lead to uneven standards. For example, William Cowper was so well looked after at Nathaniel Cotton's St. Alban's asylum that he hired one of the staff there as his personal servant. He says:

"I received nothing but praise and care from the doctors."
He added that the staff were:
"...ever watchful and apprehensive for my welfare."[8]

Contrast this with Dr John Conolly's description of life in an asylum for the poor in his book 'A History of Penal Methods':

"They were but prisons of the worst description. Small openings in the walls, unglazed, or whether glazed or not, guarded with strong iron bars, narrow corridors, dark cells, desolate courts, where no tree nor shrub nor flower nor blade of grass grew. Solitariness, or companionship so indiscriminate as to be worse than solitude; terrible attendants armed with whips... and free to impose manacles and chains and stripes at their own brutal will; uncleanliness, semi-starvation, the garotte, and unpunished murders – these were the characteristics of such buildings throughout Europe."[9]

Similarly, Roy Porter states the following in his book 'Madness: A Brief History':

"Asylums varied widely in quality. Reformers exposed many as abominations, riddled with corruption and cruelty, where whips and chains masqueraded as therapeutic."[10]

When it became clear that free-market provision was failing, Victorian governments started to intervene and regulate asylums to ensure common and minimal standards. At the same time, the medical profession started to become involved, gaining exclusive powers to confine, diagnose and treat mental illness, giving birth to the new science of psychology in the process, but reducing family involvement, which was traditional up until then. The Victorian era also saw a huge increase in the development and use of new drugs to treat or subdue mental illness.

Due to continued scandals involving corruption, neglect and abuse of patients in Victorian asylums, and the inevitable public anger these caused, progressives such as Robert Gardner Hill and John Connolly, who built on the pioneering work of William Tuke, Philippe Pinel and William Battie, briefly gained attention with their theory of moral management, which argued that the mentally ill were best treated with kindness, patience and humane treatments and conditions.

Unfortunately, as asylums clogged up with difficult, long-term patients who could not be discharged easily, and new wonder-drugs failed to cure as promised, public opinion began to turn against Victorian reformers. Attitudes hardened: governments soon reverted to using asylums as dumping grounds for the mentally ill; critics of moral therapy began to argue once again that insanity was hereditary, ingrained, chronic and likely incurable; prurient photographs depicting inmates as freakish and abnormal began to circulate; curative approaches in asylums were replaced by formal drills and a more austere environment.

It is little wonder then that a century of reform started to unravel by the end of the Victorian era and the quality of care began to deteriorate. A setback that, arguably, wasn't corrected until the 20th Century.

Having compared Victorian and pre-Victorian approaches to mental health, it is obvious that both had pros and cons.

In ancient societies, attitudes and beliefs towards mental illness were largely shaped by superstitions or religion. Most of these were irrational and resulted in treatments and cures that were often equally irrational, violent or inhumane, such as prayer, exorcism, trephining and leeching.

Asylums barely existed in these times, and there was no formal provision for the mentally ill. Patients were usually confined to their homes or, alternatively, exiled, and forced to wander the streets begging for their food. Either way, they were often the victims of neglect and abuse. Mental illness was a source of great shame for families and many believed it to be hereditary or the result of 'bad fate'.

However, the picture was not all negative. Families were the primary carers of the mentally ill in pre-Victorian times, meaning there was a personal connection between carers and patients so patients were often looked after with more care and sympathy than they were in the profit-making asylums that were common at the start of the Victorian era. This was also often the case with religious people such as monks and priests, who were motivated purely by a sense of religious duty and not money. In addition, even when they were banished from home, the mentally ill were often left alone, and so had a degree of freedom and choice that they could never exercise in a Victorian asylum, no matter how progressive.
The Victorians also engineered many advances in mental healthcare, and all in just a few decades, rather than over the course of centuries. They introduced the regulation and state provision of asylums. They also developed earlier ideas about the need to treat the mentally ill humanely into the theory of moral management, an idea which resulted in much more agreeable asylums and higher cure rates. Equally importantly, they applied rational ideas and the science of medicine to develop new drugs and therapies to combat mental illness. This innovation resulted in the development of a new discipline called psychology, which went on to transform our understanding of mental health in the 20th Century.

Despite the enormous advances in mental healthcare during Victorian times, asylums were not of uniform quality. Rich patients enjoyed luxurious facilities in private asylums that resembled retreats, while the poor were usually imprisoned on doctors' orders in squalid institutions that were corruptly run and where they were often abused or tortured. Worse, after progressive theories fell out of favour, curative treatments were replaced with a lifetime of hard labour in workhouses or state-run asylums. Both the state and the medical profession promised more than they could deliver in Victorian times, resulting in a public backlash and a change in attitudes that took decades to reverse.

As we've seen, both Victorian asylums and earlier methods of caring for and treating the mentally ill had distinct advantages and disadvantages. Therefore, we cannot argue that one era or approach was superior to another, even considering the many remarkable innovations by the Victorians in this field. There has never been a single best solution for dealing with mental health issues, and that is still the case today.

Bibliography

1.http://www.academia.edu/4073569/SCHIZOPHRENIA_FROM_A_CHRISTIAN_PERSPECTIVE_A_CRITICAL_INVESTIGATION_OF_THE_CAUSES_OF_SCHIZOPHRENIA_IN_NAIROBI_C
2. http://wiredspace.wits.ac.za/bitstream/handle/10539/9949/Hindu%20Psychologists'%20Perceptions%20of%20Mental%20Illness_MAThesis_2010.pdf?sequence=2
3. Roy Porter, Madness: A Brief History, Chapter 5, Locking up the Mad (page 89)
4. http://www.studentpulse.com/articles/283/the-history-of-mental-illness-from-skull-drills-tohappy-pills (main source)
5. https://en.wikipedia.org/wiki/Hypericum_perforatum
6. http://www.britannica.com/biography/Hippocrates
7. http://www.britannica.com/biography/Franz-Anton-Mesmer
8. Roy Porter, Madness: A Brief History, Chapter 5, Locking up the Mad (page 99)
9. http://wellcomelibrary.org/item/b24917060#?c=0&m=0&s=0&cv=99&z=0.4354%2C1.0544%2C0.4501%2C0.2277
10. Roy Porter, Madness: A Brief History, Chapter 5, Locking up the Mad (page 99)

Programme Officer's Comment:
This essay manages to combine a fascinating overview of historical approaches and attitudes to mental health with sharp analysis of their advantages and disadvantages. I am particularly impressed with the essay's nuanced approach to discussing methods of treating and containing the mentally ill; H. has avoided the temptation to paint history in black and white and has recognised that we cannot always neatly categorise into right and wrong or better and worse. In addition to H.'s impressive analysis, the use of well selected sources and high-level language demonstrates an impressive command of the subject material. Well done!
—D. Jones, Teaching and Learning Manager, The Scholars Programme

Writing the Short Story

Year 10, Key Stage 4

Eastbury Community School, London
S. Awoyemi, supervised by I. Nedelcu, University of Edinburgh

A short story is, by definition, 'a brief tale that can be read in one sitting'.[1] This loose definition has created a literary platform with a large variety of styles and methods of storytelling. In this essay, I will explore the way in which narrative voice and its closeness are used to alter the reader's experience of the characters in a short story. More specifically, how the distance of the narrative voice, being 'close' or 'far', affects the evocation of a reader's emotions towards and their understanding of a character.

When the narrator is close to the story, such is the case in Lappin and Lapinova by Virginia Woolf, the reader feels more empathic towards the character that the narrator is close to. So in this story, the reader 'feels sorry for' Rosalind because we see the world from her perspective, whereas, if the story was told from the viewpoint of Ernest we may regard Rosalind in the same way he does, as crazy. We see brief moments of this alternate interpretation as the style of narration is, ultimately, third person so the perspective cannot be limited to just that of Rosalind. An example of a moment when the reader sees both the views of Rosalind and Ernest is towards the end of the story:

"I thought my rabbit was dead!" she whimpered. Ernest was angry.
"Don't talk such rubbish, Rosalind," he said. "Lie down and go to sleep."[2]

As the narrator has been close to Rosalind, the reader feels sympathy for her as Ernest treats her so harshly, but, had the narrator been close to Ernest or to neither of them, then the reader may feel the same way as Ernest does. In this way, the author, Virginia Woolf, has used the narrative to orchestrate the emotions of the readers into defending Rosalind. A chief reason for this may be because Woolf herself suffered from mental illness, similar to Rosalind in the story. Stephen Trombley described Woolf as being 'a woman who is the victim of male medicine', referring to the lack of understanding towards mental illness during Woolf's time.[3] So, in a way, having a close third person narrative is how authors are able to create pity for their characters because it allows the world, and its unfairness, to be clearly put across through the medium of the protagonist's thoughts.

However, it could be argued that a close third person narrative is not needed to create an emotional response to characters in a narrative, which is proved by the short story Hills Like White Elephants by Ernest Hemingway. In this text, a 'fly on the wall' narrative style is instead used, but it still manages to produce a similar reaction from the reader; much like the previous text. As the narrator only says what is on the surface, it is up to the reader to infer most of the background information. Consequently, Hemingway has made it easy to work out details in the story, examples of this being the referral to the characters as a "man" and a "girl" showing the age gap between the two characters in the relationship.[4] Moreover, phrases such as "It's an awfully simple operation" and "once they take it away, you never get it back" make it clear that the topic of their conversation is an abortion.[5] Whereas the man is sure that this abortion is necessary, the girl is not so sure and this garners sympathy from the reader. The clear naivety of the girl's responses during the conversation shows how she is unprepared to deal with this sort of issue. She also seems to drink frequently, which isn't something a pregnant woman should do. Maybe, Hemingway did this intentionally in order to show that even though this issue more concerns the girl than the man, he still holds the power, ultimately, to make this decision. It could also show, from the viewpoint of the reader, how this girl is being forced to make a decision that she may not be ready to make. In Lappin and Lapinova Rosalind fixates on details, such as the features of Ernest's parents' home and their similarity to animals, whereas the girl in this narrative is erratic in her nature and her characteristics often shift dramatically, potentially because of her distress because of the situation. But an alternate interpretation could be that the girl is more mature than she seems, though because of the narrative style, we can never know for sure what she is thinking. This can also make the reader pity her since she might have something worthwhile to say about the situation but the man doesn't regard what she says as important so he doesn't truly listen to her.

In summary, both of these narrative styles allow the reader to understand the characters fully and to feel emotions, particularly sympathy, towards them. However it is not only

the narrative that creates this effect, it is only when they are paired with other techniques that the full effects are felt. For example, in Lappin and Lapinova, the reader may not have felt the same way towards Rosalind if the narrative had been solely focused on her for the duration of the text. It was the brief interjections of the opinions of the other characters in the story that allowed Rosalind to be portrayed as a sort of 'maiden in distress' that appealed to the solicitude of the readers. Similarly, in Hills Like White Elephants, the troubled surface of the relationship between the man and the girl, coupled with the hidden undertones that the girl is more mature than she seems allow her to get sympathy because we are the only ones who can see that the girl is constantly put down by the man and what she says is regarded as unimportant. If the narrative styles of these two texts had been flipped, then it may not have been as effective as the reader wouldn't see inside Rosalind's mind and there would be no surprise of the girl being, potentially, more sophisticated than initially thought.

Bibliography:

1. Edgar Allen Poe, 'The Philosophy of Composition', 1846
2. Virginia Woolf, (1938) Lappin and Lapinova
3. Virginia Woolf, https://en.wikipedia.org/wiki/Virginia_Woolf [3 April 2016]
4-5. Ernest Hemingway, (1927) Hills Like White Elephants

My Short Story

Sakura

The man and woman wished with all their might as they planted the tree in the ground outside their house. Both were tired of this fruitless struggle, trying over and over again for a child yet failing every time. It was of no fault of their own, but something like that takes a toll over time, making you feel old and tired before your time. And so, the couple knelt in the dirt, meticulously scooping handfuls of dirt around the base of the young tree; all the while praying that they might someday get the chance to have the child they both longed for. It was the dead of winter, probably the worst time to plant, but their desperation was so abundant, they were ready to try anything.

About a year later, a beautiful baby girl was born to the couple; she was everything they had wished for and more. She had the same bright blue eyes as her parents, the same naturally tanned skin, the same full pink lips; which were now pulled into a grimace as she reached out for comfort. But, whereas her parents both had thick, dark hair, the baby had soft pink hair just beginning to grow, the same colour as the cherry blossoms that had begun to bloom on the tree just earlier that day. Because of this, the man and woman decided to name her Sakura, after the tree that seemed to have finally caused their prayers to be answered. However, what the man and woman didn't know was that the life of their child was in fact linked to the cherry blossom tree they had planted just a year ago.

As Sakura grew into a toddler, the tree continued to grow with her, never shedding its blossoms, even in the colder months. During the first years, Sakura's parents continued to tend to the tree and made sure it was healthy all the time. It was their way of saying 'thank you' to the universe for giving them what they had desired for so long. Though, as time went on and they got busier, the health of the tree began to falter as no one tended to it anymore. And as the tree's health declined, so did Sakura's. At first it seemed as if it was only a small cold, then the flu, but as weeks and months passed and Sakura didn't get any better, her parents finally realised that the tree was the key to keeping Sakura healthy. So they went completely overboard and made sure the tree was kept in its best condition. They also kept their daughter in the house all the time, never allowing her to go outside where they couldn't protect her. For a while, it seemed as if everything was back to how it was during those first few years of Sakura's life; when she and her parents were happy and just like a normal family. But eventually, as she grew into a young woman, Sakura began to feel trapped in her own home. Her parents meant well, but they had kept her in the house for nearly 15 years! She had asked them a plethora of times if she could finally be allowed to go out, but to no avail. Sakura knew that they would never let her leave, so drastic measures had to be taken. Sakura planned to run away.

It took nearly a month, but finally, Sakura had all she needed. She had been secretly taking small amounts of food and knew exactly where her parents had hidden the key to the door. So, under the cover of night, when silence had settled like a blanket over the house, Sakura left home for the first time in over a decade. She knew that eventually her parents would know that she had left, but hoped that she could fool them for at least a day before they came looking for her. Sakura headed deeper into the city from her home in the suburbs. She had seen the lights of the city's skyline from her room's window many times and had always felt the pull of the city's unlimited possibilities inside her. However, it was nerve wracking being out alone for the first time in so long. But Sakura knew that there was no way she would ever go back.

And so, Sakura created a new life for herself in the heart of the city that had been almost on her doorstep all her life. It pained her to know that her parents had been withholding all of this from her. She was experiencing so many new things: work, parties, friends and even... love. Yes, Sakura had fallen in love and it was better than she could have ever imagined. There had been a time when she had questioned whether she would ever have a relationship as perfect as her parents' had seemed when she was young and now she knew that is was possible. Sakura lived like this for almost a year, until things began to go wrong.

Sakura woke up one morning feeling seriously ill. It was similar to how she had felt what seemed like a lifetime ago when she was very young and her parents weren't looking after the tree. With a start she realised what was happening. Nothing had happened to her to make her feel so terrible, so it must be that someone was deliberately cutting blossoms off the tree to harm her. She never thought her parents would stoop so low, but she knew that if she didn't succumb to their will, she would soon be too weak and ill to even do anything. So, with a heavy heart, Sakura packed up her things and left, without so much as a goodbye to her love or her friends.

When back home, Sakura was shocked to see that there were only a couple of blossoms left on the tree. Were her parents trying to kill her? Her parents welcomed Sakura back with their faux love but kept her too weak to ever run away again. That is until Sakura's love came up from the city to find her. Sakura had told him her story a while ago and he knew that he couldn't bear to think about her parents holding her captive for the rest of her life. He walked up to the tree and gently plucked off the last few blossoms. Up in Sakura's room, her hair turned dark as the last bits of life left her body. She was sorrowful that she had to leave this world but found comfort in the fact that she had lived a full life for a while. Meanwhile, Sakura's love treasured those last few blossoms for the rest of his life, and they never died.

Inspirations:

Identifying trait – Ernest's twitching nose, Sakura's pink hair
Symbol – white elephant, cherry blossom tree
Close third person narrative – Lappin and Lapinova
Protagonist kept in submission – Hills Like White Elephants, Lappin and Lapinova

About the authors

S. Awoyemi is in Year 10 at Eastbury Community School in the East London Borough of Barking & Dagenham. I. Nedelcu has completed her PhD in Creative Writing at the University of Edinburgh.

PhD Tutor's note

It was an absolute joy to work with S. during The Brilliant Club tutorials on English Literature & Creative Writing. I was particularly impressed with his outstanding level of engagement with the theory, his comprehensive essay writing skills, his captivating creative writing artfulness and constant generosity in sharing ideas when contributing to tutorial discussions. No doubt S. would be an invaluable presence to any undergraduate programme.

To What Extent Was Friendship the Bond That Led the Roman World Together?

Year 10, Key Stage 4

Holy Trinity Catholic School, Birmingham
M. Mahmood, supervised by J. Kemp, University of Warwick

The Roman world had some aspects of amicitia, or friendship, which could be the force that kept the Roman world together. Cicero, Seneca and Pliny the Younger had stated the different characteristics of friendship in their writings. Some of these aspects were: benevolentia, munera, beneficia, amicitia, or friendship. These were portrayed through the friendships of the kings, the emperor, the republic and the empire with foreign nations.

Julius Caesar offered munera to Ariovistus on the behalf of the Senate and Rome. In his speech he states, "By the Senate that gifts (munera) had been sent most lavishly."[1] This shows the extent he would go to maintain this friendship with Ariovistus. However, this source could not be reliable because it was written by Julius Caesar, himself, who could be biased and this affects the reliability of the source. This wouldn't be reliable as Caesar would want himself to be portrayed as a positive and good person. Munera was an attribute of Cicero's criteria of an ideal friendship. Caesar did this in order to uphold his position in Rome and to gain land in Gaul, this shows that friendship did hold the Roman world together, even if 'amiticia was a weapon of politics.'[2] P.A. Brunt pointed out that the word amicitia came from the word amo, Latin for love, which must have had connotations of affection and love.[3] I agree with this because there was mutual respect between people and they would do favours for each other, be generous and gift things to each other.

The Senate gave Octavian honours out of goodwill and respect, which was a part of Cicero's characteristics of an ideal friendship 'if you remove goodwill from the very name of friendship is gone;'[4] Octavian was given these honours so there would be an emperor that kept the Roman world going and developing. This showed that by gaining Octavian's friendship by giving these honours to him, the Roman world had someone to care for it and the people. Furthermore, Pharasmanes, a ruler of the Iberian kingdom, had a strong friendship and alliance with Antonius Pius, who was Hadrian's successor. Cassius Dio, who was writing in the 2nd and 3rd centuries, had stated that Pharasmanes went to visit Antonius in Rome and 'Allowed him to offer sacrifice to the Capitol and set up an equestrian statue', which shows how much respect and love they had for one another, unlike the friendship between Pharasmanes and Hadrian.[5]

There were many friendships between kings, the Roman emperor and the Roman people. This shows that if Rome ever needed help with anything, they would always have an ally to help them. The SHA describes the friendship: 'The Parthians always regarded him as a friend'.[6] By gaining people's friendship and trust, the Roman emperor and the people would always have someone there to help them and this is what kept the Roman world together. In the empire, coins were minted to show respect, this differed when in the republic they would give ranks, positions and honours. Coinages from the times of Antonius Pius portrayed that he is crowning someone from the Quadii tribe.[7] The coins are showing the emperor's friendship with foreign people which reinforces that there would always be someone looking out for them and helping them in a time of crisis, which is one of Cicero's aspects: 'For generosity is of two kinds: doing a kindness and requiting one'.[8] Furthermore, the coinage depicts the crowning of a new Armenian king.[9] This showed it to be a good thing as he was making more alliances and more friends, which allowed him to be safer, guarded and protected. This suggests that friendship, to a certain extent, was the feature that held the Roman world together.

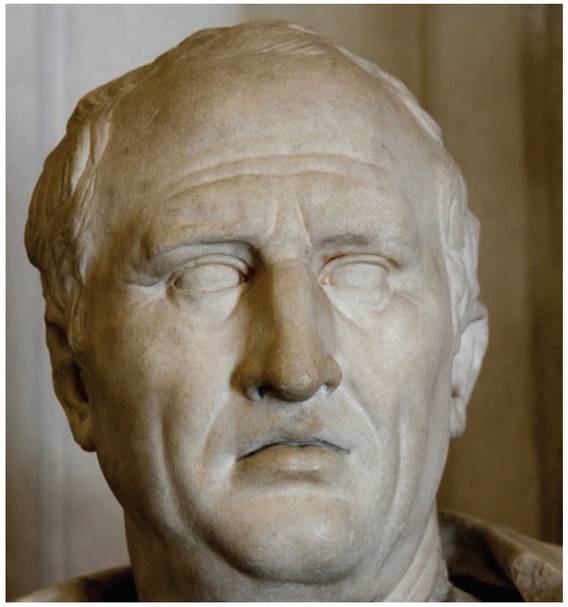
However, there were bad aspects to friendship that showed that it was not the thing that held the Roman world together. For example, the friendship between Pharasmanes and Hadrian was not as good as that between Pharasmanes and Antonius, when they 'mock the gifts' that they gave each other.[10] Mark Antony and Octavian formed an alliance and to seal this alliance, Octavian gave his sister Octavia's hand in marriage; this was recorded in the Treaty of Brundisium. Coins were also minted to show their alliance and friendship. Although, Mark already had a wife, he went to Egypt and married Cleopatra. As a result of this, Octavian had waged war against Mark Antony and Egypt, which resulted in deaths and chaos. This is a clear example that

showed amicitia was not the attribute that held the Roman world together.

Furthermore, the use of bribery and money was possibly the thing that kept the Roman world intact as Cassius Dio stated: 'This best explains why he lived for the most part at peace with foreign nations, but received money besides'.[11] Cassius' writing implies that alliances and friendship were not needed to keep peace and order. Money and wealth were very important in the period as they enabled people to have a higher authority and more luxuries. Money was also used by Marcus Aurelius to keep Rome's enemies at bay when they were at war: 'these received a gift of money'.[12] R. Syme had also said that amicitia was a weapon of politics, 'not a sentiment based on congeniality'.[13] This agrees with Taylor's idea of amicitia being used as a weapon of politics and attention.[14] I agree with Taylor because amicitia was used for politics and power by Julius Caesar when he married his daughter to Pompey in 59BC, as a way to seal their alliance, in the same way Octavian had done to seal his alliance with Mark Antony. However, this source's reliability is not certain because it is not a primary source and therefore would have a different view on things.

Amicitia meant alliances and friendships with many people, which would be considered a good thing, however if one alliance had failed or went wrong, it not only impacted the parties in the alliance but other friendships as well. The Republic would converse through common friends, such as finding someone that they were both friends with and then befriend each other. However, if there was any dispute between the two friends, people would have to choose sides, which could cause a lot of hatred and anger towards one another. When Octavian was betrayed by Mark Antony, Pompey was also betrayed: 'Pompey was betrayed by the simulacrum of a peace'.[15] This showed that having friendships with a lot of people was bound to end in a disastrous way. If people had to choose which side they were on, there would be a lot of chaos and rage between the two groups, and this would result in violence. This showed that amicitia was not a good thing and did not keep the Roman world together.

Hadrian was very well prepared when it came to his army, which underwent 'such a long course of training'.[16] He never took any chances and ensured his army was in the best shape possible. He would go to any extent to make sure that they were prepared for any situation. If friendship was the bond that held the world together, then Hadrian wouldn't need a reason to protect his empire. Friendship was only a word and it was not valued as much as an armed force. If amicitia was the bond that held the Roman world together then Hadrian would not need to worry about what could happen, with all these allies by his side. Furthermore, Hadrian had to mention Trajan's name in order to gain the army's support: '...deified Trajan, my model'.[17] The use of 'model' showed that Trajan was almost Hadrian's and the army's example to follow. They had Trajan as a system that they could imitate and Hadrian wanted the army's support, so he was talking about Trajan in order to gain all the support he needed. Calling Trajan his model could suggest that there was not really any friendship between the two and Hadrian thought Trajan to be better than him,



Philosopher Cicero articulated the key characteristics of friendship, which influenced the idea of amacitia.

which was not a part of Cicero's ideal friendship; 'although this mutual interchange is really inseparable from friendship'.[18] However, this source may not be reliable because there are a lot of gaps, which could suggest that there are parts missing and important information that could be helpful in order to fully understand what was going on at that time. Furthermore, it was translated from Latin to many different languages and if the translation of a word was incorrect then this would massively impact the reliability because there would be a lot mistakes. On the other hand, this source could be reliable because it was recorded at the time in question, which makes it a primary source.

In conclusion, amicitia played a vital part in the Roman world as it was one of the means used to make sure that the Roman world was developing and working efficiently. The Roman world had a lot of allies, which benefitted them in case of any war breakouts. In my opinion, amicitia, to a certain extent, did hold the Roman world together through the means of making allies by munera, beneficia, gratia and favours. However, other factors also had a significant part in keeping the Roman world composed and protected such as: money, bribery, the army and having alliances.

References

[1]Julius Caesar, De Bello Gallicio, 1.43.4-7
[2] Taylor, L., 1949, Party Politics in the Age of Caesar, page 12
[3] Brunt, P., 1965, "Amicitia' in the Late Roman Republic' in PCPS, Vol. 191, pp.1-20
[4] Cicero, De Amicitia, 19
[5] Cassius Dio, Historia Romana, 69.15.3
[6] SHA, Hadrian, 21.10-14
[7] REX, QUADIS DATUS (RIC III, no.620)
[8] Cicero, De Officiis, 1.48
[9] REX ARMENIIS DATUS (RIC III, no.619)
[10] SHA, Hadrian, 17.10
[11] Cassius Dio, Historia Romana, 699
[12] Cassius Dio, Historia Romana, 71.11-13
[13] Syme, R., 1939, The Roman Revolution, (Oxford: Oxford University Press)
[14] Taylor, L., Party Politics in the Age of Caesar
[15] Tacitus Annals, 1.10
[16] Speidel, M., 2006, Emperor Hadrian's speeches to the African army.
[17] Speidel, M., 2006, Emperor Hadrian's speeches to the African army.
[18] Cicero, De Amicitia, 26

Bibliography

Cassius Dio, Historia Romana, 69.15.3
Cassius Dio, Historia Romana, 699
Cassius Dio, Historia Romana, 71.11-13
Cicero, De Amicitia, 19
Cicero, De Amicitia, 26
Cicero, De Officiis, 1.48
Cicero, De Amicitia, 26
Julius Caesar, De Bello Gallicio, 1.43.4-7
REX ARMENIIS DATUS (RIC III, no.619)
REX, QUADIS DATUS (RIC III, no.620)
SHA, Hadrian, 17.10
SHA, Hadrian, 21.10-14
REX, QUADIS DATUS (RIC III, no.620)
Tacitus Annals, 1.10
Speidel, M., 2006, Emperor Hadrian's speeches to the African army- A New Text, (Mainz Verlag des Römisch Germanischen Zentralmuseums)
Syme, R., 1939, the Roman Revolution, (Oxford: Oxford University Press)
Taylor, L., 1949, Party Politics in the Age of Caesar, page 12, (Berkeley, Los Angeles and London: University of California Press)
http://penelope.uchicago.edu/Thayer/E/Roman/Texts/Tacitus/Annals/1A*.html#10
https://www.numisbids.com/n.php?p=lot&sid=1424&lot=317
http://www.history.co.uk/biographies/julius-caesar
http://www.dirtyoldcoins.com/roman/id/Coins-of-Roman-Emperor-Antoninus-Pius.htm
http://www.livius.org/sources/content/plutarch/plutarchs-caesar/caesar-and-pompey/

Programme Officer's comment:
"I have really enjoyed reading this article, what a fantastic ambassador from Holy Trinity School!! This pupil has made real progress on their Scholars Programme journey, I hope that they feel inspired by working with the PhD tutor, Joanna, who said that she was really grateful for the sparky contributions made in class. Reading this article has made me really proud; it was clear to see how much time and effort has gone into it. Well done to this pupil and best of luck in their future studies!"
-Annika Rowbury – Harrison, Programme Officer (West Midlands, South West & Wales)

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—A. Rowbury - Harrison, Programme Officer, West Midlands, South West and Wales, The Scholars Programme

Social Sciences

Consequentialism and Deontology: Do the Ends Justify the Means?

Year 8, Key Stage 3

Friesland School, Derbyshire
L. Ford, supervised by L. Earl

Euthanasia is said to be "the bringing about of a gentle and easy death for a person suffering from a painful incurable disease" (Oxford Paperback Dictionary 1988, p.274). There are three different types of euthanasia: voluntary, non-voluntary and involuntary. Voluntary is when someone personally requests it, non-voluntary when a person is unable to make the decision and finally involuntary is when a person wants to go on living. Depending on these different circumstances, I believe that consequentialists, deontologists and I would have different views on whether euthanasia is morally right or not. In some cases, particular events or conditions may make someone believe that the 'ends justify the means', whereas in a separate situation, they may disagree.

One example of where a person may want to undergo assisted suicide is if they were diagnosed with a terminal illness. Subsequently, they may be condemned to a life of discomfort where the patient is forever traipsing between hospitals, which in the end has no effect as they are still going to die. Therefore, this patient may decide that rather than carrying on with their life in pain, they may want to end it there without going through any suffering. Some consequentialists would have no objection to this as this is the person's own choice and therefore should not be influenced by anybody else. Furthermore, this may also help others in the long run. The patient has been diagnosed with a terminal illness. They will never get better, only worse. Therefore, this person's health will deteriorate, which might make them reliant on life support and / or other people. On average, it is said that a person's life support in the last year of their life, costs around £7,500 (Doughty 2012). This is a large amount of money that could be used to help actually save someone's life. As it is certain that the person is going to die anyway, and the support will only extend their life, a consequentialist would agree with the patient and allow them to take part in assisted suicide. Moreover, the time that is added on to the patient's life will almost certainly be miserable, as they will be condemned to suffering.

On the other hand, a deontologist would go against the idea. They would say that taking someone's life, for whatever reason or morals, is always wrong. They would argue that by killing a person, even on their request, you are responsible and therefore taking their life. To them, this is against our human rights and should not be done. They may also think that only killing a few people for severe reasons would lead on to more things. By killing the odd person, you are opening a door to a whole other world where people will just start killing each other for no apparent reason. What started as a minute number of voluntary euthanasia cases would soon escalate to non-voluntary, and who knows where that could lead? Over time we would be killing each other so inhumanely that we would be turning into wild animals. Furthermore, people with illnesses may feel pressured into euthanasia so as to not be a burden on their friends and family (NHS 2014).

Likewise, a utilitarian would not permit the assisted suicide, as it does not create a large amount of happiness. According to the Hedonic Calculus, the death of the patient should not be undertaken. For example, one of the criteria is extent – how many people will benefit and get the most happiness from an action. I believe this to be quite low as the only true person to be happy about the result is the patient, but they will be dead. Of course, some people may say that the patient's family would be happy for the end of their suffering, but I think they would be more relieved for the end of the patient's suffering, and sad that the patient has gone. A further example is repetition- how will the action produce more happiness? Again I think the score will be quite low, as no one else would be affected by the happiness and it will therefore not spread.

Personally, I agree with the consequentialist. In some cases, the choice of the patient's death should be up to them as it is their life – such as when they have a terminal illness. It is up to them how they want to end their life and if they do not want to suffer, then so be it. Furthermore, the money and resources that would be saved could help others who may benefit more. What is the point of keeping someone alive who is quite blatantly suffering and would much rather die? I believe that if a person is going to die anyway, they should not have to suffer just for a few extra years. Perhaps we could offer support to the person beforehand, and try to talk them out of the idea, but in the end, it is their choice and if it is what they want, why should we stop them? However, I think it is down to the end result on how we determine whether we should kill the person. If a patient is in severe pain now, but there is a high prospect of them living a happy life after the illness, then I think that the person should perhaps not be assisted in dying. They have a whole life waiting ahead of them, and it would be illogical to take that away from them. I think we have to weigh up the current situation, and what is likely to happen in the future. We must compare how the person is now, look at how much pain they are in, and decide if what they are going through now justifies the end result.

An example of involuntary euthanasia is when someone is facing the death penalty. They would have committed some crime that the nation says should be punished by death. Consequentialists would argue that for severe crimes, the death penalty should be permitted. One reason for this is that it embeds fear into potential criminals. If a person knows that death would be the punishment of their crime, then surely they would not commit it? Of course, they don't exactly want to be in prison, but the prospect of the death penalty is a lot more intimidating and should therefore stop people from committing the crime. Subsequently, this should lower the number of crimes and save a lot of people who may have been victims of murder or other crimes. Between 1990 and 2010, in America, research has shown us that the amount of murders committed in a year has deteriorated in death penalty states. However, further results have told us that perhaps the decrease in murder rates has not got much to do with the death penalty because the amount of murders committed is substantially lower in non-death penalty states (Amnesty International, n.d.). This perhaps suggests to us that maybe the death penalty is not working the way that people would think. Some consequentialists may disagree with the others and go against the idea of the death penalty. A reason for this is that cases without the death penalty costs on average $740,000, while cases with the death penalty costs about $1.26 million (Death Penalty Information Centre 2015). Clearly, the death penalty is much more expensive than a life sentence in prison as it requires more time and lawyers. A death penalty case can typically last 6-10 years (Lawyers.com, n.d.). This means that the prisoner must be kept on death row, which is more expensive than a prisoner in the general prison population. Moreover, this is perhaps not as effective as we originally thought. By keeping someone in prison you are forcing him or her to think about what they have done, whereas if you were to kill them then they are in a sense getting out of their punishment, as they do not have to live through a miserable life.

Deontologists would be against the death penalty as they believe that no matter what a person has done, it is still wrong to kill someone. They would say that life in prison would be more morally correct and that the death sentence cannot be justified. What if someone had been arrested for murder, a crimes we as a society have ruled unjustifiable, but surely if we were to sentence someone to the death penalty we ourselves are murdering them and therefore committing the same crime as them. How can it be that when they do something it is deemed wrong but when in turn we do it to them it can suddenly be justified?

I am against the death penalty as we are spending more money on someone who is not really going to be punished, as they will be dead. Furthermore, if a mistake is made and is not discovered until sometime later then the death penalty is not reversible. Overall, I think that euthanasia is acceptable when an innocent person chooses to be relieved from extreme suffering in later life. I believe that in these cases the means of killing someone can be justified as they are making this person happier, rather than allowing them to suffer.

References:

Oxford Paperback Dictionary. (1988). Oxford, Oxford University Press.
Doughty, S. 2012. The bill for dying. The Daily Mail, 16th Oct. Available from: http://www.dailymail.co.uk/news/article-2218343/One-dying-patients-hospital-treatment-costs-NHS-7k-final-year-life.html [Accessed: October 6th 2016].
NHS. (2014). Euthanasia and assisted suicide arguments. [Online] NHS. Available from: http://www.nhs.uk/Conditions/Euthanasiaandassistedsuicide/Pages/Arguments.aspx Amnesty International. (n.d.) The Death Penalty and Deterrence. [Online] Amnesty International. Available from: http://www.amnestyusa.org/our-work/issues/death-penalty/us-death-penalty-facts/the-death-penalty-and-deterrence [Accessed: October 6th 2016].
Death Penalty Information Centre. (2015) Financial Facts about the Death Penalty. [Online] DPIC. Available from: http://www.deathpenaltyinfo.org/costs-death-penaltyLAYWERS.COM (n.d.) Death sentence appeals take time for a reason. [Online] criminal.lawyers.com [Available from: http://criminal.lawyers.com/criminal-law-basics/death-sentence-appeals-take-time-for-a-reason.html [Accessed: October 6th 2016].

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PhD Tutor's note
I really enjoyed my time teaching at Friesland School. The staff and pupils were welcoming and incredibly enthusiastic about the programme. L.'s essay showed a level of sophisticated thinking and an in-depth understanding of the complexity of the issues we studied. The essay is a great example of how an issue like euthanasia is not at all straight-forward and clear. She dealt with the complex philosophical theories very well, and used good examples to substantiate her argument.

Activities Increase Life Satisfaction and Happiness in Adolescents?

Year 10, Key Stage 4

Bucklers Mead Academy, Somerset
A. Kahn, supervised by A.L. Mottershaw

Studying psychology to develop our understanding of positive psychological strengths is important because we can help people feel happier within themselves, enable them to achieve their full potential and allow them to improve in various aspects of their lives. The aim of our experiment was to see if gratitude activities affect happiness and life satisfaction. To test this, we had a total of three groups: group 1 listed five things they were grateful for every day, group 2 wrote a gratitude letter once a week and the control group had no assignments to complete – the experiment took place over a course of two weeks and every participant filled out questionnaires every week for three weeks. Our test demonstrated that wellbeing activities increase overall life satisfaction and happiness in adolescents. Our research could be used to encourage psychologists or support figures in schools to use wellbeing activities to increase positive affect in students.

Introduction

Studying wellbeing is important because the research it produces can help people improve their mental health considerably, increase longevity (Diener and Chan, 2011), and become more successful in life in general (Boehm and Lyubomirsky, 2008). Happiness is closely related to wellbeing and being happy can increase your motivation and responses to challenging tasks and it simply makes your life, and others', much more enjoyable. Lyubomirsky, King and Diener, (2005) have found that happier people are not just successful in their careers but are often more successful in multiple sections in their life including family relations, marriage, friendship and productivity (work performance). There is a clear absence of gratitude studies regarding adolescents in psychology (Froh, Miller and Snyder, 2007), which makes it appear as if adolescents are insignificant; that further research on adolescents would be pointless and have little value. However, I believe learning more about wellbeing in adolescents can help create a healthier adult population in future and it could even be argued that adolescents are the most important age group to observe because this is an essential period of life where people go through several development changes and are vulnerable to many variables which could impact their happiness and wellbeing and could affect their lives later on (Richter, 2006).

From previous studies, for example Lyubomirsky, Dickerhoof, Boehm and Sheldon (2011), it has been established that actively participating in happiness interventions (which usually consist of expressing gratitude and optimism) can significantly increase PA (positive affect), especially in children and adolescents, both immediately post-intervention and even six months later. Numerous factors that could affect the impact these interventions have on participants are things such as amount of effort, level of participation and optimism – their ability to imagine their best possible future (Lyubomirsky, Dickerhoof, Boehm and Sheldon, 2011). Research has also found that wellbeing in adolescents can be improved by counting blessings daily (Froh, Sefick and Emmons, 2008).

The current literature in wellbeing research of adolescents has mainly been accumulated in the United States and there is an inadequate amount of research done in the UK. Also, the majority of published research mainly focuses on adults and comments little on children or adolescents. The aim of our study was to see how reminding yourself of what you are grateful for in life impacts overall life satisfaction and happiness for adolescents. We hypothesised that the wellbeing activities will increase happiness and life satisfaction. The independent variable in our experiment was the type of wellbeing activity - life satisfaction and happiness were the dependent variables, which were measured across several domains of participants' lives. Our research will add to the body of literature out their already and helps fill the gap in adolescent research in wellbeing because our test concentrates on a specific age group (only adolescents). In conjunction to this, our experiment is taking part on a smaller scale at a small town as opposed to the city schools where the majority of research is produced.

Method

Participants

In total, 18 students (five boys and 13 girls) within the age range of 14–15 years old participated in our experiment to explore the effect gratitude has on happiness and life satisfaction. These students all attended the same secondary school in South West England and were studying in Year 10.

Materials

To measure the dependent variable, all participants used the BMSLSS – Brief Multidimensional Students' Life Satisfaction Scale (Seligson, Huebner and Valois, 2003) to assess satisfaction in various significant aspects of their lives including: family life, friendship, school experience, living conditions, themselves and their overall life satisfaction. The scale is responded to using seven options: 1 = terrible, 2 = unhappy, 3 = mostly dissatisfied, 4 = mixed satisfaction, 5 = mostly satisfied, 6 = pleased and 7 = delighted. The scale should be able to measure various items on the questionnaire and produce similar results (strong internal consistency). Happiness was assessed using the Subjective Happiness Scale (Lyubomirsky and Lepper, 1999) and again included 7 response options but had four items, which were either a statement or a question. The scoring of the scale uses reverse coding for the fourth item (the 7 in the scale translates to a 1, 6 = 2, 5 = 3, 4 = 4, 3 = 5, 2 = 6 and 1 = 7).

Procedure

For the experiment, participants were split into three equal groups (n per group = 6). Participants in group 1 were instructed to remind themselves and preferably write five things that they were grateful for everyday for two weeks. Group 2 wrote a letter of gratitude to a person whom had deserved a thank you for either showing great levels of kindness towards the participant or someone who had done a favour but had not been properly thanked.

The letter was written in a 30-minute period once a week for two weeks; a total of two letters were produced per participant. The contents of the letter were not shared within the group but all participants discussed how they felt when writing the letter, how difficult they found it and how well they managed to complete the letter within the time limit. Finally, the control group had no activity or task to perform; they simply filled out the life satisfaction questionnaires once a week, every week for three weeks alongside every participant from both groups.

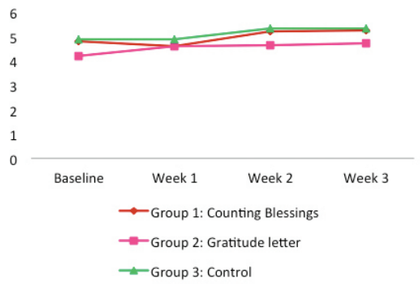

Fig. 1:
Mean happiness scores for the 3 groups from baseline-week 3

Table 1.
The mean happiness and life satisfaction score of the three groups across each time point.

	Happiness			Life Satisfaction		
	Group 1: Counting Blessings	Group 2: Gratitude Letter	Group 3: Control	Group 1: Counting Blessings	Group 2: Grattitude Letter	Group 3: Control
Baseline	4.81	4.20	4.88	5.19	4.95	4.95
Week 1	4.60	4.60	4.88	5.40	5.44	5.13
Week 2	5.19	4.65	5.31	5.59	5.59	5.25
Week 3	5.25	4.70	5.31	5.63	5.79	5.25

As shown in Table 1, our results show that all groups increased in both happiness and life satisfaction as the study progressed. In group 1 the mean happiness changed across the study by 0.44 (displayed by Figure 1) and the mean life satisfaction (looking at Figure 2) also increased by 0.44. Figure 1 shows us that the mean happiness for group 2 increased by 0.50. This small increase may be because the participants already had high levels of wellbeing, also known as an 'emotional ceiling', so it would be more difficult for the activities to further increase their happiness compared to a group who had low levels of wellbeing (Froh, Kashban, Ozimkowski & Miller, 2008). However, Figure 2 informs us that the life satisfaction had a much bigger increase of 0.84 for group 2, resulting in an overall substantial increase of wellbeing. Finally, group 3's (the control group) mean happiness rose by 0.43 and experienced the smallest difference with the life satisfaction only being increased by 0.30.

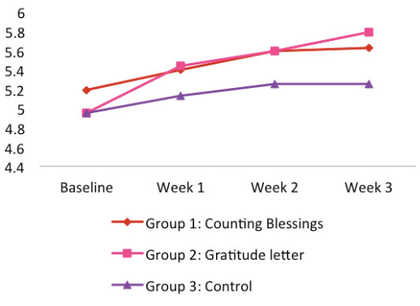

Fig. 2:
Mean Life Satisfaction scores for the 3 groups from baseline-week 3

Group 1 had the biggest impact on happiness because looking at Figure 1 we can see that there was the biggest difference between each interval and group 2 had the most impact on life satisfaction as it has a steeper gradient. Although the control group began with a high happiness and life satisfaction score it either maintains the same score or only increases slightly. However, the increase was only 0.01 lower than group 1 which suggests that counting blessings is not the most effective activity of improving wellbeing. Overall, there was a bigger increase of life satisfaction and wellbeing for the gratitude activities compared to the control group.

Discussion

The results that our experiment produced demonstrated that life satisfaction and happiness can be increased by participating in wellbeing activities. This supports our hypothesis that counting blessings everyday can improve your wellbeing and increase happiness and life satisfaction. Our hypothesis was supported as there was an overall increase for group 1 (gratitude group) in both wellbeing domains.

Our results support previous literature that found that wellbeing activities can improve wellbeing in adolescence. For example, Froh, Sefick and Emmons (2008) also found that wellbeing activities such as counting blessings increased life satisfaction and happiness much more than the control group and the hassle group (refers to a group who wrote down five things which they found annoying/irritating). Moreover, Seligman, Steen, Park and Peterson (2005) discovered that a gratitude visit created a substantial increase in positive changes for the following month; further increase depended on the participants' dedication to their activity (how frequently they performed their activity).

Although our research supports previous findings, our experiment did not mirror the results of some past studies. For example, Layous, Nelson, Orberle, Schonert and Reich (2012) found a distinctive pattern in their results and had a larger increase of happiness and life satisfaction than our results as we only found a small increase in our dependent variable (happiness and life satisfaction). This could be because the location of our study was different: their tests were done in the US and ours took place in the UK. Another reason could be because the activities we used were different – our activities focused on gratitude and in Layous et al. (2012), the experimental group were instructed to perform three acts of kindness and the control group visited three places. This activity was chosen because they wanted the participants to do something without attempting to increase wellbeing.

Our findings have implications for education – teachers could use wellbeing activities at school to improve the students' wellbeing (life satisfaction and happiness), which would enable them to become more successful at school. Additionally, our findings also have implications for parenting as parents could use the wellbeing activities at home to make their children happier and participation in these activities could improve the mood and relationships within the family home.

Our study is a considerable addition to the gratitude literature and has several strengths. Firstly, all participants were of a similar age - this means that it is easier to compare results and more accurate results can be produced. Secondly, in our experiment we included a control group and more than one activity that allowed us to identify which activity was most effective at improving wellbeing and had the biggest impact on life satisfaction and happiness. Finally, all participants reunited once a week, every week to track progress and discuss any issues with the activities, which ensured that the wellbeing activities were the best they could possibly be and that no participant had any problems completing the task.

However, our experiment also had a number of limitations. Firstly, our study was conducted over three weeks so time constraints meant that we were unable to study the long-term effects of the wellbeing activities. Secondly, the generalisation of our results could be questioned because it does not successfully represent a large population as only a small sample of people were studied and there was very little diversity amongst the participants. Thirdly, we do not know if we are measuring what we claim to be because we are unable to know for definite if all participants completed their tasks to the same standard – this poses a threat to the internal validity of our study. Fourthly, the tasks were quite different and required different amounts of time and effort; the letter, in total, took less time than writing out a list everyday but it needed additional effort and more thought; this might explain the difference in effectiveness of each activity and that thought-provoking activities are more effective than ones that are time consuming. Finally, there are some individual differences that could have affected the outcome of the experiment such as personality and maturity.

To improve this research, we could perform the experiment over a longer period of time to observe long term impacts. Also, to make the results more suitable to be generalised we could test a larger sample of people with a mix of nationalities, religions and genders and study how the larger diversity of people affects the outcome. Thirdly, using different means of measuring the dependent variable such as a gratitude questionnaire (similar to McCullough, Emmons and Tsang, 2012), could improve the accuracy of our results. Finally, using a personality questionnaire would make the test fairer as we could consider their personality when analysing the result.

To build upon our findings that wellbeing activities increase happiness and life satisfaction, future researchers should test different age groups to observe the difference, hold the study at a different location – not only in different cities, villages and abroad but also at colleges and primary schools. Furthermore, future researchers should use a wider variety of activities so the most effective activity can be identified and participants have a choice and will not become bored continuously doing the same activity (Lyubomirsky, 2008).

In conclusion, we found that to increase happiness and life satisfaction in adolescents we can use wellbeing activities. This is important because with this evidence councillors and psychiatrists can use this as another technique to help people with depression to acknowledge the good in their lives and aid them in becoming happier, healthier people.

Bibliography

Boehm, J. K., & Lyubomirsky, S. (2008). Does happiness promote career success? Journal of Career Assessment, 16(1), 101–116.
Diener, E., & Chan, M. Y. (2011). Happy people live longer: Subjective well-being contributes to health and longevity. Applied Psychology: Health and Well-Being, 3(1), 1–43.
Froh, J. J., Kashdan, T. B., Ozimkowski, K. M., & Miller, N. (2009). Who benefits the most from a gratitude intervention in children and adolescents? Examining positive affect as a moderator. The Journal of Positive Psychology, 4(5), 408–422.
Froh, Miller & Snyder (2007). Gratitude in children and adolescents: Development, assessment, and school-based intervention.
Froh, J. J., Sefick, W. J., & Emmons, R. A. (2008). Counting blessings in early adolescents: An experimental study of gratitude and subjective well-being. Journal of School Psychology, 46(2),
Layous, K., Nelson, S. K., Oberle, E., Schonert-Reichl, K. A., & Lyubomirsky, S. (2012). Kindness counts: Prompting prosocial behavior in preadolescents boosts peer acceptance and well-being. PloS One, 7(12), e51380.
Lyubomirsky, S. (2008). The how of happiness: A scientific approach to getting the life you want, New York: The Penguin Press
Lyubomirsky, S., Dickerhoof, R., Boehm, J. K., & Sheldon, K. M. (2011). Becoming happier takes both a will and a proper way: an experimental longitudinal intervention to boost well-being. Emotion, 11(2),
Lyubomirsky, S., King, L., & Diener, E. (2005). The benefits of frequent positive affect: does happiness lead to success? Psychological Bulletin, 131(6), 803–855.
Lyubomirsky, S., & Lepper, H. S. (1999). A measure of subjective happiness: Preliminary reliability and construct validation. Social Indicators Research, 46(2), 137–155.
McCullough, M. E., Emmons, R. A., & Tsang, J.-A. (2002). The grateful disposition: a conceptual and empirical topography. Journal of Personality and Social Psychology, 82(1), 112–127. https://doi.org/10.1037//0022-3514.82.1.112
Richter, L. M. (2006). Studying Adolescence. Science (New York, N. Y.), 312(5782), 1902–1905. http://doi.org/10.1126/science.1127489
Seligman, M. E., Steen, T. A., Park, N., & Peterson, C. (2005). Positive psychology progress: empirical validation of interventions. American Psychologist, 60(5), 410–421.
Seligson, J. L., Huebner, E. S., & Valois, R. F. (2003). Preliminary validation of the brief multidimensional students' life satisfaction scale (BMSLSS). Social Indicators Research, 61(2), 121–145.

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A. Kahn is a Year 9 pupil at Buckler's Mead Academy. A. Mottershaw is a PhD student studying the genetic and environmental influence on wellbeing in adolescence at the University of Bristol.

PhD Tutor's note
A. has presented a clear and coherent scientific report, showing that she engaged well with relevant psychological literature. The report shows a critical appreciation of the advantages and disadvantages of the literature, data analyses, and the implications of the findings. There is excellent critical thinking and evaluation throughout the report, especially in the discussion. It was a pleasure to work with A., who was committed throughout the course and always produced work of a very high standard. In general, I was very impressed with the students at Buckler's Mead Academy. They put so much effort into the course, including collecting their own data, engaging with scientific literature, and participating in insightful discussions.

Mechanism Design and Matching Markets: How Should We Allocate School Places?

Year 10, Key Stage 4

William Edwards School, Essex
T. Bal, supervised by A. Johnson

The matching market that I have chosen to evaluate is the market for school places that decides which students get selected for which schools. A market would include buyers (consumers) and sellers (producers) and enables exchange between two or more groups of agents.[8] In economics, agents refer to people and how they are able to choose their actions and make decisions. Here, the students would effectively be the consumers and the schools would act as the producers (although in the UK, the government provides funds for this). This is a two-sided matching market, which means that both groups of agents have preferences over whom they would like to be matched with on the other side of the market.[3] This means there are two sides that need to be taken into consideration when matching these agents.

Model

There are only two groups of agents: the students and the schools. Each of these agents would have preferences, where a preference is just a way of telling us what somebody likes and if they like it more or less than

something else. Each student would have a preference over which school they would like to go to and each school would have a preference over which students they would like to accept. Preferences are written in the order of what is liked the most to the least. A discrete set of agents is presented in these brackets { }, for example, N = {Lucy, Rose, Linda} and this shows the names of the agents.

A mechanism is a way to determine who gets what based on their preferences. The deferred acceptance algorithm (introduced by Gale and Shapley in 1962) is a mechanism that has been implemented in schools in Boston and New York City.[6] [7] The deferred acceptance algorithm is a type of mechanism where the match cannot be certain until the algorithm has ended:

- Firstly, each student will list their preferences over schools and each school will list their preferences over students.
- Then, each student will apply to the school that is their first preference and if a school receives over a certain number of applications from students, the school will select the students they like the best, rejecting the others.
- Any rejected students will have to apply to their second preference and each school can choose their favourite students from the new set of applicants, including those kept from the last round.
- This process will simply continue until every student has been accepted by a school or they have applied to and been rejected from all schools.[8]

If a student is accepted by a school, their place is not permanent or guaranteed, as the school may choose to replace that student with another 'better' student from one of the other rounds. In 2005, the Boston Public School Committee voted to use the new mechanism of deferred acceptance to establish school places and it was implemented in the public schools of New York City in 2003. [6] When deferred acceptance was used in high schools in New York City during 2003, it decided the placement of approximately 100,000 9th and 10th graders.[6]

An Example

I have created a simple example to show how matching students to schools will work using deferred acceptance. There are three schools and six pupils. For each school, q=2 (where q represents quantity and this is the maximum intake of pupils for each school).

Two Sets of Agents:
Pupils: N = {Ben, Kylie, Jerry, Gigi, Orlando, Bella}
Schools: H = {William Edwards, Harris Academy, Gable Hall}

Pupils' Preferences:
Ben: William Edwards, Harris Academy, Gable Hall
Kylie: Harris Academy, Gable Hall, William Edwards
Jerry: William Edwards, Harris Academy, Gable Hall
Gigi: Harris Academy, Gable Hall, William Edwards
Orlando: Harris Academy, Gable Hall, William Edwards
Bella: Gable Hall, Harris Academy, WIlliam Edwards

Schools' Preferences:
William Edwards: Ben, Gigi, Jerry, Orlando, Kyle, Bella
Harris Academy: Gigi, Bella, Orlando, Jerry, Ben, Kyle
Gable Hall: Gigi, Jerry, Bella, Kylie, Orlando, Ben

Since Ben's first preference was William Edwards and this school's first preference was Ben, Ben is guaranteed a place here. This is the same situation for Gigi, so she gets a place at Harris Academy. William Edwards' second preference was Gigi; however, she has already got a place at Harris Academy, so William Edwards offer a place to Jerry (their third preference). Jerry accepts this offer, as his first preference was William Edwards. Gable Hall's first and second preferences (Gigi and Jerry) have already been taken, so this school moves onto their third preference (Bella). Bella gets a place here because Gable Hall was her first preference. Kylie is Gable Hall's next preference after Bella and since Gable Hall is Kylie's first preference, she is bound to get a place here. There is still one place left at Harris Academy and Orlando is the next available preference so he gets accepted.

Final Matches (see Fig. 1):

μ (Ben) = William Edwards
μ (Kylie) = Gable Hall
μ (Jerry) = William Edwards
μ (Gigi) = Harris Academy
μ (Orlando) = Harris Academy
μ (Bella) = Gable Hall

Analysis

An advantage of deferred acceptance is that it is Pareto optimal, meaning that it is not possible to make one agent better off without making at least one other agent worse off. If I gave Kylie her first preference of Harris Academy, Kylie would be made better off but this school would be made worse off, as it would have received its last preference. This ensures that no resources are wasted, so deferred acceptance is Pareto efficient. In New York City, only 3000 students did not receive one of their school choices, as opposed to 30,000, preventing students from improving their situation by lying, so no agents have an advantage over others. It is also individually rational; no schools/ students will become worse off by taking part. Each agent should gain something, even if it is not their first preference.

Deferred acceptance gives a stable matching, which means that there is no 'blocking'.[1] [10] The agents would have been matched in such a way that all individuals form a blocking pair since they would rather be matched together but have instead been matched apart. No student will lose their place at a school to another student with a lower priority, which makes this mechanism fair.[1]

A disadvantage is that it is not perfect. It is not possible for every agent to get what they would have preferred in the end. To improve this, the schools could increase their intake of pupils, so that more students have a higher chance of getting into the school they want. This is not always feasible and realistic, as schools can only take so many pupils. Another disadvantage is that deferred acceptance involves waiting for the best candidate's application, which can be a time-consuming process. There are deadlines put in place to stop a certain agent waiting longer than others, meaning each agent would receive the same amount of time to find their favourite applicants.

Before the deferred acceptance algorithm was put into place in BPS (Boston Public Schools) the Boston Mechanism was used. [6] The Boston Mechanism matched approximately 57,000 students to 125 schools but it soon failed. [5] The Boston Mechanism was a way of matching students to schools by assigning students to their first preference, according to their priority [10]:

- The priorities of students were: students at the school, students with a sibling at the school and in the walk zone, students with a sibling at the school and students in the walk zone.[10]
- Students would have strict preferences over schools and the schools would have a list of priorities for their applicants.
- Next, each school considers the students who have put that school as their first preference and assigns seats to these students one at a time, using the priority order.[9]
- Each school assigns seats in this way until they reach their maximum capacity or until there are no more students who have put that school as their first preference.[9]

This process is repeated with the remaining students but their second and third preferences are looked at instead.

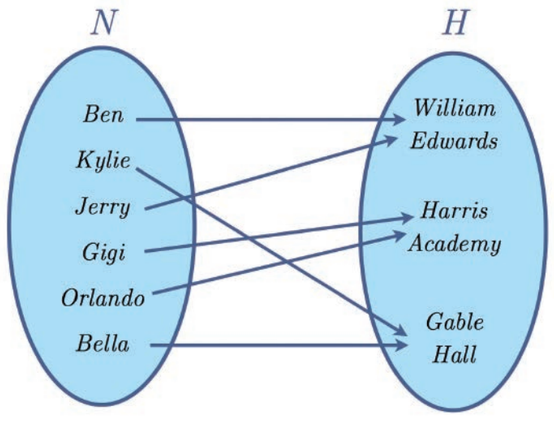

Figure 1: The Final Match using Deferred Acceptance

This mechanism relates to Roth's 'Priority Matching' and involves the same process.[1] Unlike deferred acceptance, priority matching is unstable because schools essentially choose students who apply first for that school until they reach their constraint (maximum spaces). Schools do not wait for their favourite students in each round. If there are any students who have put the same school as their first preference and have the same priorities for that school, then those ties are broken randomly.[2] A computer randomly generates a number for each student and the student with the lowest number will receive a place at the school, and so it all comes down to luck.[4]

One of the reasons why the Boston mechanism failed was because it was not strategy-proof, so some students were able to become better off by lying. The Boston mechanism is not fair - there will be disadvantaged families who do not know how to cheat this system.[10] If some families attempt to manipulate their preferences, they will not know how other students have ranked the schools, so there will be nothing to compare to. [2]

Conclusion

I believe that deferred acceptance is an effective algorithm and a fair way of matching agents, since all the agents can have preferences, which are considered before the final judgement is made. Deferred acceptance has been used to determine school places in Boston and New York City for over a decade, meaning it must be useful to their education system.[6] Personally, I do not believe that it is possible to improve the functioning of deferred acceptance because on a large scale, there will never be a mechanism that suits each agent's first preference. Deferred acceptance works so effectively, that one adjustment could make it inefficient.

About the authors
T. Bal is a Year 10 pupil at William Edwards School in Essex. A. Johnson is a PhD Candidate in Microeconomic Theory at Royal Holloway, University of London.

PhD Tutor's note
All the pupils at William Edwards demonstrated great resilience throughout this course. They were quickly introduced to a new subject, new terminology, new mathematics and a new way of approaching problems. T. has produced a particularly outstanding piece of work that demonstrates an understanding of all these areas. However, this essay goes above and beyond as it incorporates substantial independent research not only into one of Deferred Acceptance's applications but also the theory behind the algorithm itself. After only a few hours of tutorials, I can only commend her on this work.

Image Attributions

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