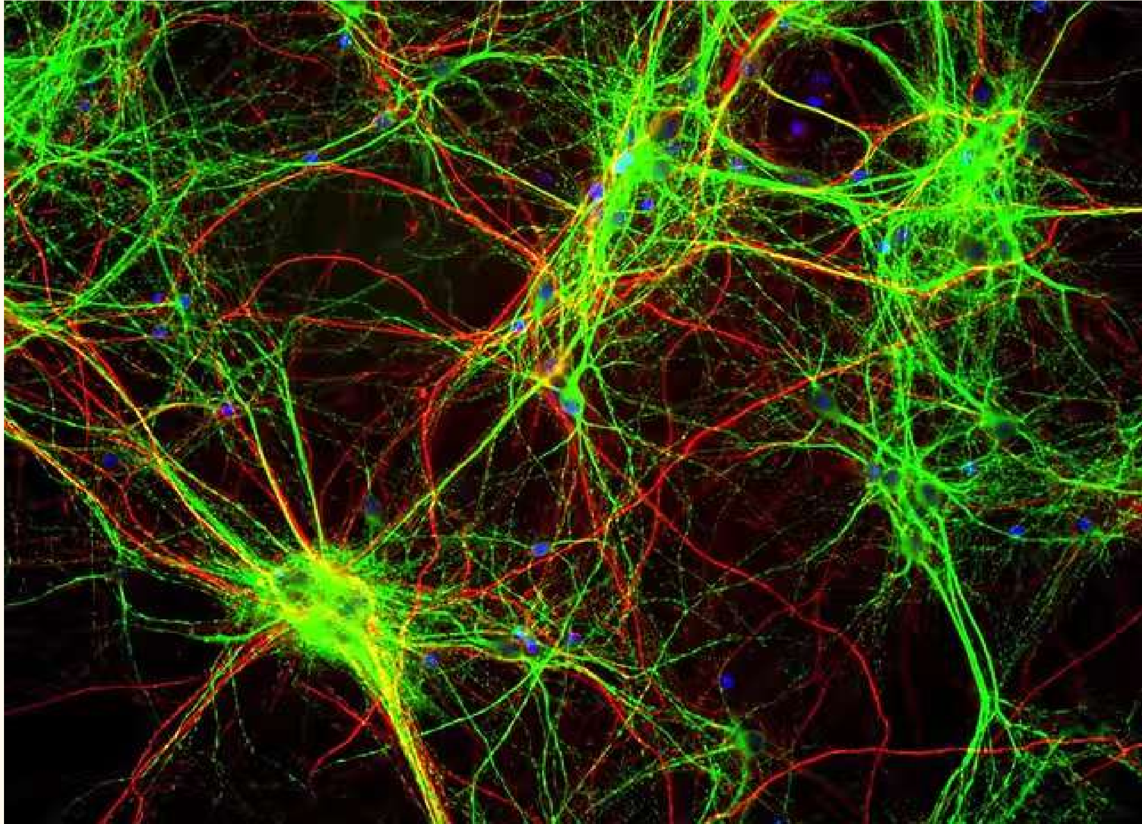
JULY 2022

# BIOLOGY NEWSLETTER



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Hey everyone, we're all waiting for that clock to tick into the final seconds of school, so here's one final newsletter to give you some things to think about over those 6 weeks! It's been a pleasure writing these articles and getting so many incredible people involved, and I look forward to continuing that trend into the new academic year!

I hope you all have a wonderful break, and thank you again to everyone who has contributed to my (suppressed) dream of being a science communicator,

Lucia

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## P1 Transduction

Why is it so, so warm these days… I was very lucky to get some work experience at the biosciences labs at the University of Birmingham a few weeks back, so I will be recounting some of my tales here today!

Living things tend to be quite complicated. If we want to find out how something works, often we'll remove a part and observe the change. On a large scale however, this can be a little impractical - like cutting off your leg and making the astute observation that you can no longer walk might not cut it. Instead, microbiologists like to zoom in to certain genes and delete them (termed "gene knockout") or reduce their expression ("gene knockdown"), and then observe the changes in the phenotype. In a highly imaginative example, take a bacterium with a gene you would like to investigate. If it is expressed normally, the bacterium might appear a lovely dark blue. Do gene knockdown, and it'll be a lighter blue. Do gene knockout, and it might be translucent. In this way you could deduce that the gene codes for a pigment, where knockdown reduces the amount of genes expressed that would code the pigment.

In this article, I'm going to focus on a type of gene knockout, P1 transduction. Now, while this phrase probably seems a little daunting, let's break it down. First of all, P1 refers to a **bacteriophage**, which uses the process of transduction to transfer genetic information from one bacterium to another of the same species (as phages tend to be specific). This means that it is a relatively fast way of transferring a specific gene knock-out to many bacterial strains, providing they have sufficient similarity in the regions on either side of the deleted gene. P1 are used as they are generalised transducers (Moore), meaning that they will occasionally package random sections of the hosts DNA as well as their own. The phages replicate in a solution of donor bacteria which contain a genome segment with your gene of interest (I'll refer to this as GoI in future!) The bacteria will **lyse**, and release the phages, producing what is called a P1 lysate. The phage population in the lysate is now let loose on a second, recipient strain of E.coli bacteria. Some of the phages will inject both their own genome and the sections from the donor strain into the recipient. Homologous recombination then means that the incoming genome sequence of the donor bacterium replaces the corresponding section in the recipient. The phages have now done their job, so we add sodium citrate to stop them continuously infecting the bacteria, and we are left with some bacteria that hopefully contain our GoI.

In the labs, the investigation was on the effect of not having a certain gene, so we need to come up with a way to not only remove this gene, but also verify that the bacteria we are testing have actually lost that and not something totally different.

To do this, we will use small, moveable fragments of DNA called cassettes. In this case, they are antibiotic resistance cassettes as they contain a gene that expresses a protein that provides resistance against an antibiotic - this gene is also called a selectable marker ("Gene Cassette").

The cassette is attached to a clone of the gene that we would like to delete. This clone is then transported to the genome (often via plasmids) and then the bacteria will most likely substitute the original gene with the one containing the cassette. Sometimes, either side of the cassette are “signposts” for an enzyme that can cut out this section of DNA (the part with the cassette in place of the original gene), which makes for a cleaner gene-knock-out. (Saragliadis et al.)

Only some of the phages will have successfully transduced the section of the genome that now contains the cassette instead of the GoI, which means that the bacteria without the cassette are redundant. Going forward, to make sure that the only bacteria in our cultures are the ones without the GoI but with the cassette, we grow them on an agar plate that contains an antibiotic. The selectable markers provide the bacterium with resistance to the antibiotic. Thus, those bacterial colonies that grow on the plate are only able to do so as they have expressed the selectable marker from the cassette, which then should also mean that the cassette successfully replaced the GoI.

Ok, quick summary because that is a lot: The whole point of all of this fuss, is to take a bacterial strain that has been genetically modified to remove a GoI, and to then transfer this mutation to a different bacterial strain. Doing it this way is faster than individually inserting cassettes into each new bacterial strain that you want to play around with.

P1 infects donors. P1 multiplies in donors and produces progenies (new phages). Some progenies receive sections of the donor’s genome where the cassette has replaced the GoI. Donor cell lyses and releases phages into the medium, producing a lysate. A handful of recipient cells are infected by a P1 that is carrying the antibiotic cassette, which is incorporated into the recipient genome. This results in antibiotic resistant clones that are selected for on a selective plate. (All good? If not, drop me an email and I’ll try to explain it a little better!)

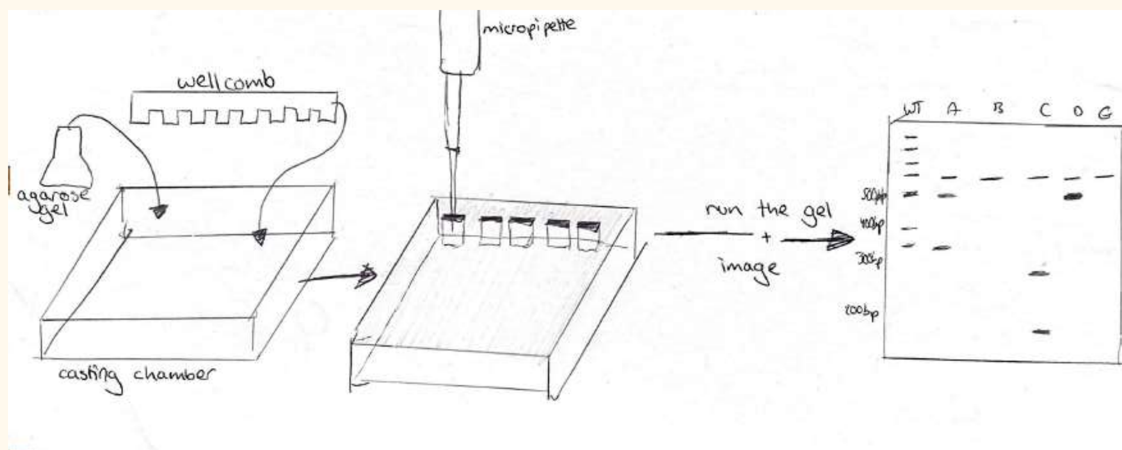
Using selectable markers and selective plates is one way to verify if you have been able to successfully transduce your GoI, however another, perhaps more reproducible method is by using PCR and electrophoresis. Both of these are techniques covered at A-level, but I’ll do us all a favour with a quick summary.

Polymerase Chain Reaction involves replicating DNA over and over again in a machine that controls the temperature. We can add primers to either end of the section of DNA that we are interested in, so that only this section is amplified. This is done to increase the amount of DNA of that section, so that it can be detected in the next stage.

Electrophoresis relies on the fact that DNA is negatively charged to seperate it with an electric current (Rogers). Similarly to chromatography, we’ll be comparing the resulting diagram to that

of a known sample/ control, which is called the "wild type". Sadly, by that microbiology is not referring to a certain Maverick in an F-14 (I highly recommend Top Gun by the way, if only for the shades and the adrenaline), but is instead talking about the original/ unedited set of bacterial genes as they would appear in nature.

For electrophoresis, we need to prepare a gel for the amplified DNA to separate on. This is made of agarose and a buffer, which are mixed and then heated in a microwave until melted. (You might laugh but honestly I got the feeling that the microwave was the most used piece of equipment in the lab…) This is then poured into a casting chamber (a glorified box really) with some DNA staining dye and well combs. These are like place holders, so that once the gel is set and the combs are removed, there is a line of slots into which you can pipette a minuscule amount of the DNA from the PCR. Connect up the power, and you're ready to run the gel! To see the results, we pop the gel into a blue-light imager, which takes a picture for us. As we had some foresight we ran both the wild type and our mutant bacterium, so that we can cross reference to see if we managed to remove the GoI.




And there you have it - a long list of some cool techniques! I've also summarised the **protocol** below (albeit with some extra steps), so that you can join up those techniques and see where they fit into a few days' worth of work in a biosciences lab. It tends to be a lot of waiting: 20 min for the agar plates to dry, 2.5 hrs for PCR, 24hrs for bacteria incubation… but you can spend the rest of that time chatting to the students and researchers in that lab. Every researcher I've spoken to is more than happy to spend hours talking about their niche topic that maybe 3 other people in the whole world could talk about at length. Moral of the story? If you ever get the opportunity to do work experience in a lab - go for it!

Protocol

- Inoculate LB (lysogeny/ luria broth) with mutant *E.coli* donor strain
- Add phage
- Infected cells lyse + release phages
- Centrifuge and remove supernatant → P1 lysate

- Inoculate more LB broth with *E.coli* recipient strain
- Add P1 lysate to recipient strain and allow to adsorb
- Add sodium citrate - is a chelator of calcium ions - aka binds to free calcium so phages can't use - phages need calcium ions to adsorb to host (chelation = bonding of molecule to transition metal ion)
- Purify the cells via several cycles of centrifugation, then spread resuspended cells onto a selective plate and incubate
- Select single colonies of bacteria, streak out onto plate - basically 2 rounds of purification to ensure absolutely no phages are in the bacterial colonies
- Again, select single colonies and suspend in medium
- Perform colony PCR
- Run a gel to confirm absence of GoI

## Glossary:

**Bacteriophages**: “bacteria-eaters” as a literal translation! They are viruses that infect bacteria, so are really useful for genetic engineering

**Protocol**: a fancy name for a set of labs instruction - aka the sheet of paper we get given for required practicals

**Lytic cycle**: one half of the viral reproductive cycle. Involves the destruction of an infected cell (the cell is lysed). Viruses that only use this method of reproduction are called virulent phages

**Lysogenic cycle**: the other half of viral reproductive cycles. Involves the integration of viral nucleic acid into host genome, where it remains dormant for some time (Wikipedia Contributors, “Lysogenic Cycle”)

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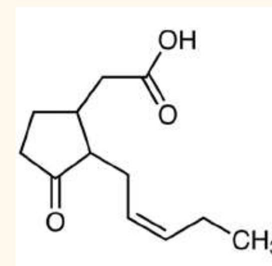
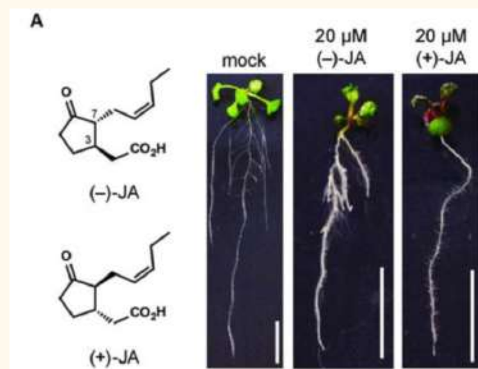
## Jasmonate




In a plant, there are many hormones that are synthesised within the plant itself. These allow the organism to respond to biotic (living) and abiotic (non-living) stresses, e.g. herbivores and temperature respectively. Jasmonates are a group of lipid-derived compounds, including jasmonic acid (JA), which interact with other plant hormones such as auxin and allow the plant to thrive despite the different challenges of its environment.

Originally found in jasmine oil[1], jasmonates are very important in many processes in the plant, but it's role in wound response is the most understood[2]. When JA accumulates in wounded plants, it responds in multiple ways to mitigate the threat. Jasmonic acid activates the expression of the gene encoding antifungal proteins, meaning that it uses the plant's DNA to synthesise molecules such as thionin and osmotin in order to prevent fungal infection of the plant, which may jeopardise the organism. In addition to stimulating protective chemicals against insects and infection, the JA also modulates the expression of cell wall proteins such as PRP, which could construct the barrier to infection. Hence jasmonic acid enhances both chemical and physical defences[3] once the plant has been wounded, and the plant utilises this by increasing the amount of linolenic acid released from phospholipids for JA biosynthesis[4].

We always talk about how plants need enough sunshine to produce enough food for themselves, but have you ever thought about when plants are subjected to *too* much light? Whilst JA protects from biotic factors such as fungi and insects, it also protects plants from the biotic factors of sunlight. The ability of JA to inhibit the expression of **endogenous** genes involved in photosynthesis suggests that jasmonate could help reduce the plant's capacity for carbon assimilation under conditions of excess light or carbon.




This occurs through a jasmonate-induced loss of chlorophyll that decreases the amount of energy absorbed by the photosynthetic apparatus of the plant, which works in conjunction with the JA-stimulated accumulation of anthocyanins[5], protecting against excess radiation through the absorption of these light **photons**.

However, JA's effect on the pigments in leaves also hints at another of its roles in the plant. When applied to leaves in a research setting, JA decreases the expression

[1] https://www.sciencedirect.com/science/article/abs/pii/S0079660302720709
[2] https://en.wikipedia.org/wiki/Jasmonate
[3] https://www.bbc.co.uk/bitesize/guides/z29trwx/revision/3
[4] BIOSYNTHESIS AND ACTION OF JASMONATES IN PLANTS | Annual Reviews
[5] https://academic.oup.com/aob/article/111/6/1021/151869?login=true#83973710

of nuclear and chloroplast genes involved in photosynthesis. These treatments also cause a loss of chlorophyll from leaves and cell cultures. Jasmonate's ability to cause this **chlorosis** led to the suggestion that this compound plays a role in plant **senescence**. JA may inhibit the synthesis of chloroplast proteins during an early phase of leaf formation where lots of cell division and nutrient absorption occurs. This is consistent with the fact that there are higher levels of JA in young leaves compared with older leaves of soybean. Furthermore, increased JA levels in roots, caused by mutants, reduce the length of the root and stunt growth of the plant due to the over-expression of genes which respond to JA, implying that JA is related to the ageing and maturation of the plant.

Jasmonate might be also expected to play a role in formation of flowers, fruit, and seed because research has indicated the relatively high levels of this compound in developing plant reproductive tissues. Using thale cress, JA was shown to not be essential for production of viable ovules. However, the mutants of the A. thaliana that lacked JA and the JA-insensitive mutant failed to produce viable pollen unless supplied with JA, suggesting that JA is important in the creation of plant reproductive organs as well as protein storage in plants, suggested due to high levels of jasmonate in vegetative sinks e.g. forming tubers.

Shown to be involved in multiple processes of the plant, like many compounds present in flora, humans have harnessed this chemical to treat disease. As mentioned above, its growth-inhibitory effects have been known for quite a while, but only recently has it been able to show its surprisingly high selectivity on cancer cells. Some plant extracts have been used as therapeutic agents in cancer therapy for a long time - it will be interesting to see whether such plants contain high levels of jasmonates.

**Endogenous** - Originating from within an organism (i.e. not externally produced)
**Photons** - Packets of light
**Chlorosis** - When the the plant produces an inadequate amount of chlorophyll leading to the yellowing of its leaves
**Senescence** - Senescence is the last stage in plant development i.e. ageing. Consequently, genes expressed during leaf senescence (senescence-associated genes, SAGs) code for proteins with functions in sink/source relationships, photosynthesis and plant defence[6]

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[6] https://academic.oup.com/aob/article/100/4/681/146613?login=true#1188581

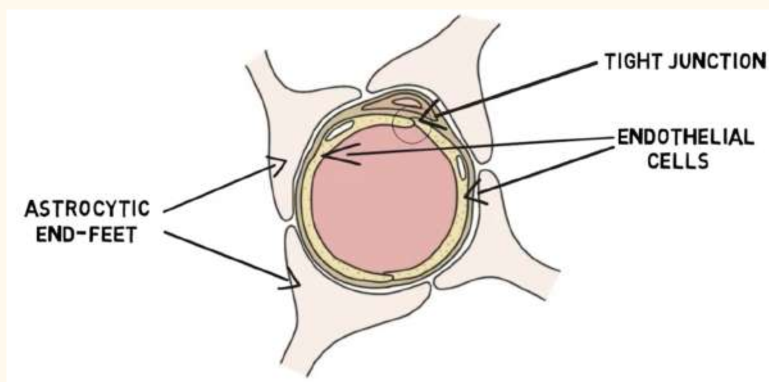
## The Blood-Brain Barrier

The brain is an organ composed of tissues and cells that respire to function. Of course, these cells require a method of delivering respiration reactants (oxygen and glucose) and removing any waste products produced. The vascular system of blood vessels supplies the brain with oxygen and removes CO2 from respiring cells. But how are these blood vessels specialised in the brain?

Many of you might have heard of the blood brain barrier. This is the term for a unique complex that surrounds the brain's blood vessels. In the capillaries surrounding other tissues, there are small fenestrations (gaps) in the endothelial cells (inner lining cells) of the capillaries to allow for movement of substances between the vessels and tissue fluid. However, in the brain, these endothelial cells are fused together to form tight junctions that restrict diffusion.[1] The fused-together endothelial cells are surrounded by astrocytes - a type of star shaped glial cell.

Glial cells are non-neuronal cells that appear in the nervous system to provide physical and metabolic support to neurons. Its greek name neuroglial literally translates into "nerve glue", which explains the purpose of these axon-lacking and dendrite-lacking cells pretty well. There are more glial cells in the brain than neurons[2], and they help neural development, insulation and recovery from injury as well as having roles in propagation of signals and nutrient and waste transport.

Astrocytes are the biggest type of glial cell. As well as releasing neurotransmitters for synaptic transmission and controlling ion concentration, they help filter anything diffusing out of the blood vessel into the brain[3]. Astrocytes have projections called end-feet that extend to the blood vessel wall. They play critical roles in the blood-brain barrier as they are involved in the stimulation of the fusing of the endothelial cells into tight junctions as well as maintaining and regulating the blood-brain barrier.




The blood-brain barrier acts as a barrier (obviously) between the bloodstream and the extracellular space in the brain to avoid the potentially dangerous substances such as pathogens and toxins from passing into the brain. Only water, oxygen and some small lipid-soluble substances can be easily transported between the blood-brain barrier.

However, there are some parts of the brain that do not contain these specialised cells as they require direct access to the bloodstream. These are called circumventricular organs and they include the posterior pituitary gland that has to release hormones into the blood.

In medical scenarios, the restrictive nature of the blood brain barrier poses an issue when there is a need to deliver drugs into the brain[4]. The blood-cerebrospinal fluid barrier is also an issue here, with the purpose of this barrier being to produce cerebrospinal fluid as opposed to the blood brain barrier which allows ion, nutrient and waste exchange during neurotransmission[5].

Modern medicine uses intracerebral or intrathecal (into the spinal fluid) injections to deliver drugs to the brain. The only non-invasive method of breaching the blood-brain barrier is through intranasal administration, as the intracerebral injection requires a craniotomy and the intrathecal injection requires a pump to be installed at the base of the spine.

However, there is current research looking into the ability to bypass these barriers in order to deliver chemicals straight into the brain.[6] Nanocarriers such as polymersomes or nanoparticles can also be utilised through the temporary disruption of the blood-brain barrier via bradykinin. Should the release of nanocarriers intravenously prove successful, it could be used as a less invasive treatment than intracerebral/thecal injections with a more optimal delivery to the brain cells than the intranasal method.

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6. https://www.ingentaconnect.com/content/ben/ctmc/2014/00000014/00000009/art00007 (N.B. If you would like to read the whole paper, please feel free to email me: 16jains562@kechg.org.uk)

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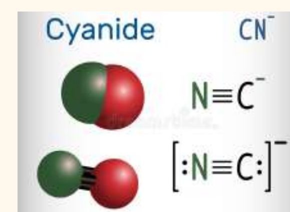
## How does cyanide affect the body?




Chemically, a cyanide compound is a compound that includes a cyano group, which consists of a carbon atom triple bonded to a nitrogen atom. Highly toxic cyanide compounds include the soluble salts of NaCN and KCN. The most toxic cyanide compound is hydrogen cyanide - a gas that is fatal when inhaled. This can be produced via the combustion of polyurethanes.

The reason cyanide is poisonous is because its ion (CN-) is an inhibitor of the electron transport chain during mitochondrial respiration, so no more ATP is produced in aerobic respiration. More specifically, cyanide binds to the iron in an enzyme (cytochrome oxidase) used in the mitochondrial respiration. Cyanide stops sufficient oxygen being utilised for respiration, killing off cells and organs that require a lot of oxygen, such as the CNS and the heart. Early symptoms include headaches and dizziness, while prolonged exposure can lead to seizures, loss of consciousness and cardiac arrest. These symptoms can set in within minutes of breathing in highly concentrated air.

Treatment of cyanide poisoning must be administered as soon as possible - testing takes a significant amount of time so if poisoning is suspected, the person affected should be treated as soon as possible, even before test results come back.[1]

In the past, we used amyl nitrite or sodium nitrite to treat cyanide poisoning. These nitrites oxidise Fe2+ into Fe3+ in the haemoglobin molecules to form a molecule called methaemoglobin (metHb). This metHb can now bind with cyanide, so it competes with the cytochrome oxidase. This means that the cyanide binds with the metHb instead of the cytochrome oxidase, leaving the enzyme free for respiration. However, this metHb cannot carry oxygen anymore, so it restricts transport of oxygen to respiring cells. High concentrations of methaemoglobin in the blood can cause dyspnea (shortness of breath) and weakness.[2]

That's why currently we use vitamin B12a (aka hydroxocobalamin)[3] to treat cyanide poisoning as it reacts with the cyanide to form cyanocobalamin - a compound that can be safely filtered by the kidneys - which also has minimal side effects on the body.

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## Antibiotic Resistance

Antibiotics have been a lifesaver (quite literally) for the treatment of bacterial infections. They are effective when used properly and easy to prescribe. But there is a massive issue occurring currently surrounding antibiotics - antibiotic resistance.

So how do they work? Antibiotics support the natural immune system in fighting the bacterial infection by killing the bacteria through blocking some of the processes that occur within the bacteria. They are specific to the bacteria they are fighting, so cannot be used in a general manner. There are two main types of antibiotics - broad spectrum and narrow spectrum which refers to the amount of bacteria it can affect. The way they destroy the bacteria can vary and a doctor will choose the most effective and relevant antibiotic regime to use, by doing detailed analysis on the bacteria if necessary to ensure the right antibiotic is being used.

However bacteria randomly mutate which can lead to a decrease in the effectiveness of the antibiotic. When the bacteria survives it can increase its multiplying and mutating, making it even more difficult. Contact with other bacteria can also lead to resistance and this resistance can be passed on to other forms of bacteria causing a resistant strain all together. The World Health Organisation has called antibiotic resistance one of the biggest public health disasters today. Superbugs are one step further and are resistant to several different antibiotics which makes the infection all the more difficult to treat. The bacteria that causes tuberculosis is incredibly difficult to treat due to its antibiotic resistance, and is being found in hospitals even more commonly.

How does antibiotic resistance even happen? Well there are several reasons, including livestock being dosed with the antibiotics leading to drug resistance, the antibiotic cycle not being followed to the level it should be, and overprescribing. A lot of people believe viral infections can be cured with antibiotics, when they only work on bacterial infections. If you develop a resistant infection it could take longer to cure and there could be more complications. Antibiotic resistance can also be passed on through using antibiotics for viral infections and a general negligence of good hygiene.

So, what’s the solution? There are a couple of different ways the resistance can start to be dealt with. First of all, a general education in what an antibiotic does and can treat is needed so people realise that they aren’t a golden solution for everything. That will stop the overprescribing of them which allows them to only be used in the necessary circumstances. This will also stop the development of resistant bacteria so they are less likely to be passed through the generations and mutate further. There is also a new push for more drugs and therapies to be created to allow for more treatments being available for common bacterial mutations. More research is also being done, to look into the resistance in greater detail and understand it further, which can allow better diagnosis and specific antibiotic usage.

If we all become more educated on antibiotics and adopt simple lifestyle changes, maybe we can change the way resistance has been growing and try to limit the mutations of common bacterial infections.

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## Rat dissection

Whilst all us eager year 12 students were busy putting our gloves on, the rat was being taken out of its encasement by Dr Newman. We were all overcome by the strong smell of formaldehyde which is used to keep the body intact and avoid decomposition.

The first thing we did was to begin dissecting the rat's abdominal region. As we all expected, the first organ we saw in the dissection was the rat's stomach; which was miniscule compared to the size of a human stomach. Connected to the stomach, we observed the link between the small intestine and the stomach, similar to that of a human. The small intestine was held in place and coiled up tightly by lots of fat linkages, which meant that the aid of a scalpel was required to dislodge the small intestine and allow for us eager students to unravel it and assess its length. As in a human, the rat's intestines were extremely long, and it took us about 10 minutes to fully take out the full length of the intestines. Once unravelled, it was a moment of realisation for all of us to fully comprehend the length of the intestines in relation to the rat itself. There was a stark difference.

Once we had dismembered the rat's intestines, we moved on to look at the rat's liver. After discussing with Dr Newman as to whether it was the liver, we decided that our initial suspicions were right despite the fact that its size was a lot larger than we had expected. Generally speaking, the rat's liver was about twice the size of the rat's stomach, which led to our doubts over whether it was the liver as compared to a human, the liver was a lot larger in proportion compared to some of the other organs. Adjacent to the liver, we recognised the rat's duodenum,

(part of the small intestine) as well as the pancreas which was immediately underneath followed by the spleen and the kidney. Another interesting part of the rat's body was its caecum, a pouch connected to the junction of the small and large intestines which is common in rodents. We had briefly heard about the caecum due to an exam question that quite a few of us had seen. The purpose of the caecum is to allow for complete digestion of the fat ingested by any rodent. By ingesting its excretment, the animal can absorb any nutrients which have been excreted in the form of droppings, and as the material passes through the digestive system again, the rodent is able to absorb the majority of the nutrition in its food as the caecum absorbs any excess nutrients from the partially digested food.

Subsequently, we moved on to dissect the thoracic cavity of the rat, which contained some more vital organs that we had the opportunity to observe. After cracking a few ribs, we were able to open up the cavity of the rat to observe the heart and lungs. Once we had opened the cavity, we realised that there was a blue tint in the parts where there were blood vessels. After discussion with Dr Newman, we understood that this was blue latex, and it is commonly used to help with the study of anatomy to allow for the veins and arteries to be seen.

After dissecting the thoracic cavity, we quickly moved on to examining the rat's skull, in which our main target was the brain. The skull was very difficult to dismember, yet we still managed to prise it open enough so that we could observe the brain. Although we couldn't take it out of its skull, we still managed to see its size in relation to the rat's body, and that was our 45 minute dissection complete!

We all had a lot of enjoyment in dissecting and observing the inner workings of a rat, and I'm sure we can all agree that it was a very intriguing experience which we learnt a lot from. I hope you readers learnt something from this article about the dissection, and are a little more interested in biology in general :)

Special thanks to Dr Newman for helping us with this practical and making it possible for us to conduct!

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**Just a quick warning - there will be some pictures of our rat opened up on the following pages, so if you don't feel comfortable with that - don't read on!**