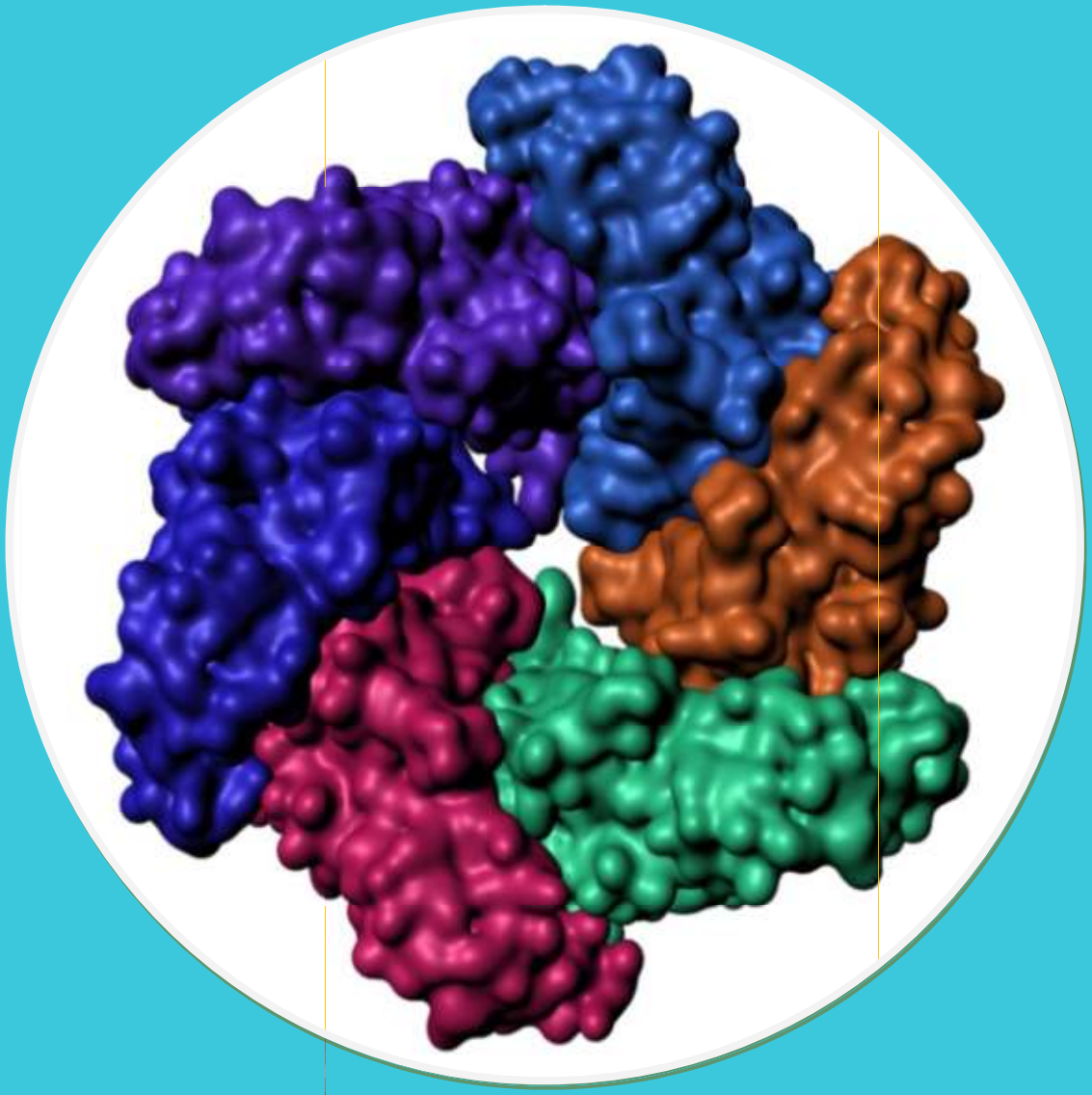

Proteins: it's all about the shape

Key Stage 4 Programme

Pupil Name	
Handbook Designed by	Eliza Ploskon-Arthur

Timetable and Assignment Submission

Timetable – Tutorials

1			
2			
3			
4			
5			
6 (Feedback)			

Timetable – Homework Assignments

Tutorial 1		
Tutorial 2		
Tutorial 3		
Tutorial 4		
Tutorial 5		

Assignment Submission – Lateness and Plagiarism

Lateness	
Submission after midnight on 9th August	10 marks deducted
Plagiarism	
Some plagiarism	10 marks deducted
Moderate plagiarism	20 marks deducted
Extreme plagiarism	Automatic fail

The Brilliant Club KS4 Programme – Pupil Feedback Report

Grade	Marks	What this means
1st	70+	Performing to an excellent standard at undergraduate level
2:1	60-69	Performing to a good standard at undergraduate level
2:2	50-59	Performing to an excellent standard at A-level
3rd	40-49	Performing to a good standard at A-level
Working towards a pass	0-39	Performing below a good standard at A-level
Did not submit	DNS	No assignment received by The Brilliant Club

Lateness	
Any lateness	10 marks deducted
Plagiarism	
Some plagiarism	10 marks deducted
Moderate plagiarism	20 marks deducted
Extreme plagiarism	Automatic fail

Name of PhD Tutor			
Title of Assignment			
Name of Pupil			
Name of School			
ORIGINAL MARK / 100		FINAL MARK / 100	
DEDUCTED MARKS		FINAL GRADE	

If marks have been deducted (e.g. late submission, plagiarism) the PhD tutor should give an explanation in this section:

Learning Feedback Comment 1 - *Enter Key Learning Priority Here*	
What you did in relation to this Key Learning Priority *Enter feedback here*	How you could improve in the future *Enter feedback here*
Learning Feedback Comment 2 – *Enter Key Learning Priority Here*	
What you did in relation to this Key Learning Priority *Enter feedback here*	How you could improve in the future *Enter feedback here*
Learning Feedback Comment 3 – *Enter Key Learning Priority Here*	
What you did in relation to this Key Learning Priority *Enter feedback here*	How you could improve in the future *Enter feedback here*
Resilience Comment	
How you showed learning resilience during the course *Enter feedback here*	How you could build learning resilience in the future *Enter feedback here*

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Course Rationale

Deep down inside every living organism there are billions of tiny particles, proteins, which control every process. Despite relatively simple composition, proteins can create a vast number of shapes specially adapted to suit their function. This course is designed to tell you about those tiny molecular machines. We will look inside the proteins using scientific software used every day by thousands of researchers and analyse how small changes in protein composition – mutations, can lead to disease.

You will first be introduced to key concepts of protein structure. We will then consider how small changes in protein composition can affect the function that the protein fulfils in the organism. These changes can be viewed as beneficial, when they drive evolution, but can also have a devastating impact, leading to disease.

This course, along with exploring the fascinating aspects of molecular biology, aims to develop skills such as researching sources of information, testing hypotheses and critically evaluating data. We will also learn how to deliver information in a visual way by preparing scientific figures. The final assignment is designed to put all those skills into practise by preparing and presenting the poster. The interdisciplinary nature of protein research with the involvement of biology, chemistry and physics gives an opportunity to find something interesting for everyone.

Mark Scheme Table

	Skills	1st (70-100)	2:1 (60-69)	2:2 (50-59)
Subject knowledge	Knowledge and Understanding	o All content included is relevant to the specific question o Good understanding of all the relevant topics. o Scientific terms are defined and used accurately throughout o Clear justification on how the content included is related to the specific issues that are the focus of the assignment	o Most of the content included is relevant to the specific question o Good understanding of most the relevant topics o Scientific terms are used accurately but not always clearly defined. o Adequate justification on how the content included is related to the specific issues that are the focus of the assignment	o Some of the content included is relevant to the specific question o Good understanding on some of the relevant topics but occasional confusion on others. o Scientific terms are used mostly accurately with occasional confusion and often not defined. o Some justification on how the content included is related to the specific issues that are the focus of the assignment
Critical thinking	Research and Evidence	o Use evidence/calculations to support claims consistently clearly and convincingly o Evidence of further reading beyond materials provided which were used in an appropriate context o Data is effectively analysed and appropriate conclusions are reached	o Use evidence/calculations to support claims mostly clearly and convincingly o Evidence of further reading beyond materials provided o Data is analysed and the conclusions that are reached are mostly appropriate	o Use evidence/calculations to support claims-at times clearly and convincingly o Limited evidence of further reading beyond materials provided o There is an attempt to analyse data ~~is~~ and draw conclusions
	Developing an Argument	o Argument/proof exceptionally well-developed and well-justified o Makes links effectively between subjects that have not previously been associated o Uses concepts from the tutorials in an unfamiliar context, and does so accurately and confidently. o Content is analysed effectively to support the argument	o Argument/proof clear and well-developed and position justified o Some evidence of linking subjects that have not previously been associated o Use some concepts from the tutorials in an unfamiliar context, but not always accurate o Analysis of content to support the argument	o Argument/proof clear but not well-developed o Limited evidence of linking subjects that have not previously been associated o Limited use of concepts from the tutorials in other contexts o Some analysis of content to support the argument

	Critical Evaluation	○ Moved beyond description to an assessment of the value or significance of what is described ○ Evaluative points are explicitly justified ○ Effective critiques on the reliability of sources provided	○ Mostly description but some assessment of the value or significance of what is described ○ Evaluative points are mostly justified ○ Some evidence of critiques on the reliability of sources provided	○ Only description with minimal assessment of the value or significance of what is described ○ Evaluative points are at times justified ○ Limited evidence of critiques on the reliability of sources provided
Written communication	Structure and Presentation	○ Ideas are arranged in a logical structure that is appropriate for the assignment ○ The introduction clearly outlines the aim of the report ○ The conclusion summarises all the main points clearly and concisely ○ Figures, tables and graphs are effectively constructed including appropriate headings, units and scales. ○ All sources are referenced correctly in an agreed format	○ Ideas are arranged in a structure that is mostly appropriate for the assignment ○ The introduction points to the aim of the report ○ The conclusion summarises most of the main points clearly ○ Most figures, tables and graphs are well constructed ○ Most sources are referenced correctly in an agreed format	○ Ideas are arranged in a structure ○ The introduction mentions the aim of the report ○ The conclusion summarises some of the main points clearly ○ Some figures, tables and graphs are well constructed but contains some errors ○ Some sources are referenced correctly in the agreed format with occasional errors
	Language and Style	○ No spelling, grammar or punctuation errors ○ Writing style consistently clear, appropriate for scientific documents and easy to follow ○ Accurate and consistent use of technical language and vocabulary	○ Minimal spelling, grammar or punctuation errors ○ Writing style mostly clear, appropriate for scientific documents and easy to follow ○ Some attempts of using technical language and vocabulary, but not always accurate	○ Some spelling, grammar or punctuation errors ○ Writing style moderately clear, appropriate for scientific documents and easy to follow ○ Use of simple language and vocabulary effectively but struggles to use technical language

Glossary of Keywords – to be filled in during the course

Word	Definition
Amino acids	
Chemical bond	
Protein	
Enzyme	
Mutation	
Ligand	

Tutorial 1: What are proteins?

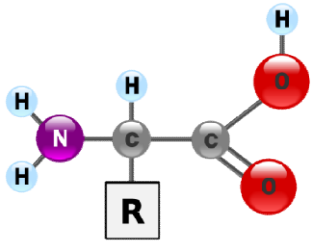
What is the Purpose of Tutorial 1?

- To learn what proteins are and what they are made off.
- What governs the structure of proteins?

This tutorial will contain information and concepts that you may not be familiar with. Do not worry if you do not understand everything immediately. We will go through it many times during the course.

Concepts:

Amino acids
Basic building blocks of proteins (have a look at Appendix 3 for a full list and structures)

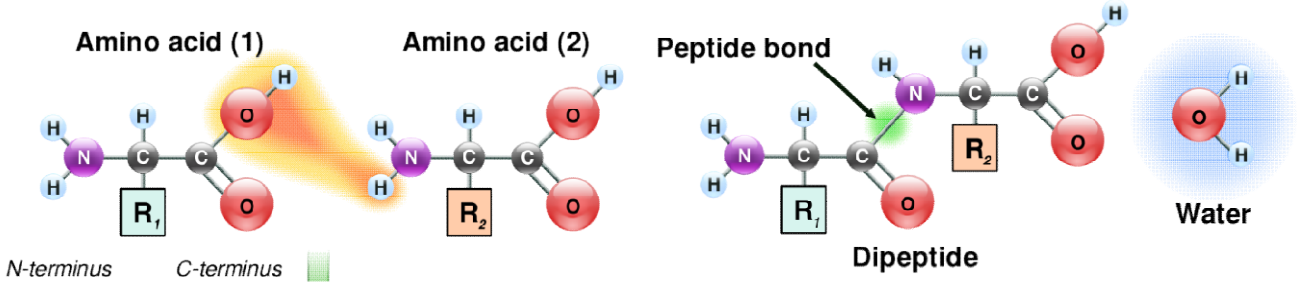


Activity - Build the amino acid alanine with Molymod

Bonding
What is a chemical bond?

Types of bonding:

Amide bond – "peptide bond"



How is a protein shape created ?

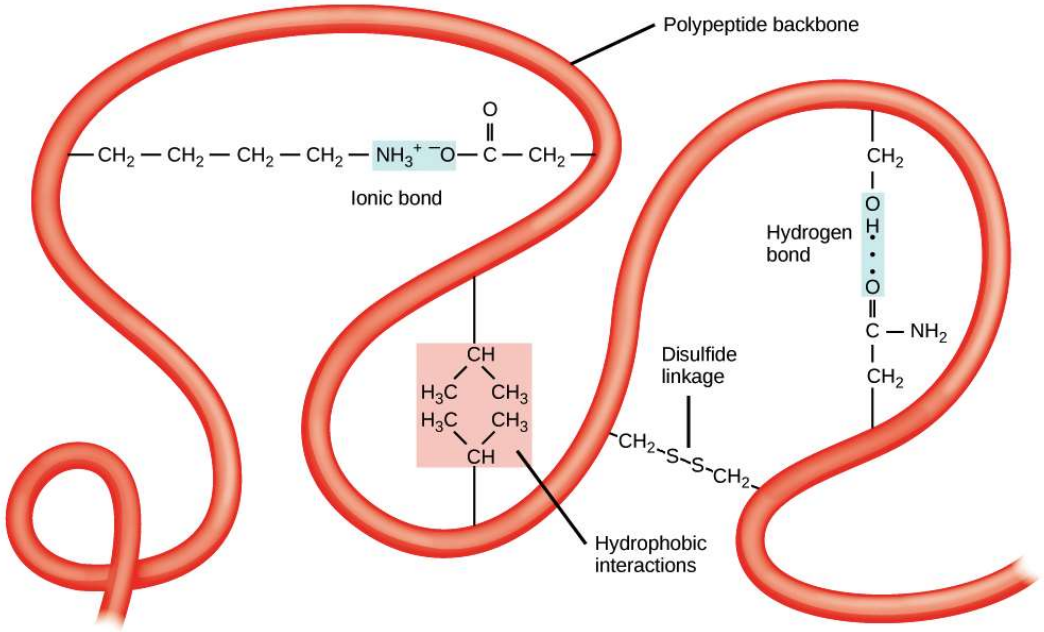
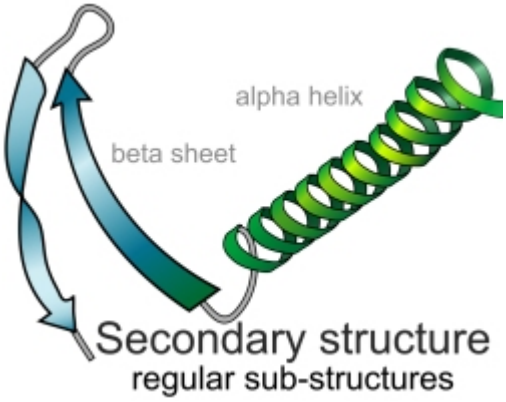


Image modified from OpenStax Biology (Khan Academy)

Specific amino acid sequence will result in characteristic secondary structures.



Activity - You will be given a pipe cleaner as a model of a protein backbone with regions of polar, hydrophobic and charged amino acids indicated. Bend it so that it fulfils the rules of protein folding. Draw your structure. And repeat it to find a different solution.

In the **final assignment** you will be required to prepare a **scientific poster**. You will need the skills of: researching, analysing the text, making a scientific figure and creating a graph. All those skills will be developed during this course.

Baseline Test

The homework assignment for the first tutorial is a baseline test to see your initial level of attainment in this subject area. In addition, you will be asked to answer short quiz related to the topics of the Uni Pathways course. The assignment will test for some or all of the subject specific skills that are required later in the final assignment. However, it is much shorter than the final assignment and it will be an introduction to the subject as well as a challenge!

Do not worry too much about doing 'well' or 'badly' on the baseline test, it takes into account the fact that you may not be familiar with the subject area. It is designed to help you and your tutor identify where you are at the start of the programme and to help you measure your progress along the way.

Today's Homework is:

Read the extract from the following article and answer the questions.

Enzyme regulation (from Khan Academy)

Introduction

The cells of your body are capable of making many different enzymes, and at first you might think: great, let's crank all of those enzymes up and metabolize as fast as possible! As it turns out, though, you really don't want to produce all of those enzymes at the same time, or in the same cell.
Needs and conditions vary from cell to cell and change in individual cells over time. For instance, stomach cells need different enzymes than fat storage cells, skin cells, blood cells, or nerve cells. Also, a digestive cell works much harder to process and break down nutrients during the time that follows a meal as compared with many hours after a meal. As these cellular demands and conditions changes, so do the amounts and functionality of different enzymes.
Because enzymes guide and regulate the metabolism of a cell, they tend to be carefully controlled. In this article, we'll take a look at factors that can affect or control enzyme activity. These include pH and temperature, as well as:

- Regulatory molecules. Enzyme activity may be turned "up" or "down" by activator and inhibitor molecules that bind specifically to the enzyme.
- Cofactors. Many enzymes are only active when bound to non-protein helper molecules known as cofactors.
- Compartmentalization. Storing enzymes in specific compartments can keep them from doing damage or provide the right conditions for activity.
- Feedback inhibition. Key metabolic enzymes are often inhibited by the end product of the pathway they control (feedback inhibition).

Regulatory molecules

Enzymes can be regulated by other molecules that either increase or reduce their activity. Molecules that increase the activity of an enzymes are called activators, while molecule that decrease activity of an enzyme are called inhibitors. There are many kinds of molecules that block or promote enzyme function, and that affect enzyme function by different routes.

Cofactors and coenzymes

Many enzymes don't work optimally, or even at all, unless bound to other non-protein helper molecules called cofactors. These may be attached temporarily to the enzyme through ionic or hydrogen bonds, or permanently through stronger covalent bonds. Common cofactors include inorganic ions such as iron and magnesium. For example, the enzyme that builds DNA molecules, DNA polymerase, requires magnesium ions to function.
Coenzymes are a subset of cofactors that are organic (carbon-based) molecules. The most common sources of coenzymes are dietary vitamins. Some vitamins are precursors to coenzymes and others act directly as coenzymes. For example, vitamin C is a coenzyme for several enzymes that take part in building the protein collagen, a key part of connective tissue.

Enzyme compartmentalization

Enzymes are often compartmentalized (stored in a specific part of the cell where they do their job) -- for instance, in a particular organelle. Compartmentalization means that enzymes needed for specific processes can be kept in the places where they act, ensuring they can find their substrates readily, don't damage the cell, and have the right microenvironment to work well.
For instance, digestive enzymes of the lysosome work best at a pH around 5.0, which is found in the acidic interior of the lysosome (but not in the cytosol, which has a pH of about 7.2). Lysosomal enzymes have low activity at the pH of the cytosol, which may serve as "insurance" for the cell: even if a lysosome bursts and spills its enzymes, the enzymes will not begin digesting the cell, because they will no longer have the right pH to function.

Feedback inhibition of metabolic pathways

In the process of feedback inhibition, the end product of a metabolic pathway acts on the key enzyme regulating entry to that pathway, keeping more of the end product from being produced.
This may seem odd – why would a molecule want to turn off its own pathway? But it's actually a clever way for the cell to make just the right amount of the product. When there's little of the product, the enzyme will not be inhibited, and the pathway will go full steam ahead to replenish the supply. When there's lots of the product sitting around, it will block the enzyme, preventing the production of new product until the existing supply has been used up.

1. Why is it important that cells control the enzymes inside them?

2. The text mentions the lysosome as a cellular compartment. Give 2 examples of other cellular compartments that know of.

3. Some drug molecules have structures that are very similar to those of enzyme cofactors. Why do you think this might be?

4. When blood sugar rises, your body releases insulin into the blood effectively lowering blood sugar levels. Once blood sugar levels have stabilised, the body stops releasing insulin. Why do you think some patients with diabetes are given insulin as a medication.

Tutorial 2: Protein science goldmine – Protein Data Bank

What is the Purpose of Tutorial 2?

- To learn how scientists work out the structure of a protein
- How we can view protein structure and draw information from it

www.rcsb.org as on the 22nd October 2017.

There are few techniques used by the scientific community to elucidate the structures of proteins. Once solved, all structures are published online so that researchers around the world can use the information freely. We will use the largest of these databases, the Protein Data Bank (PDB), to explore some disease related proteins and their structures.

Concepts:

Proteins fold to their lowest energy structure

Activity – let's play with FoldIT
(The game requires software that can be accessed from memory stick or alternatively downloaded and installed.)

Computers can handle complex maths easily and can help in determining the structure of proteins but sometimes proteins fold in ways that are non-intuitive for computers. Humans are however very good at these non-intuitive problems but are less so at the complex maths. FoldIT is a game developed to help researchers learn from humans how computers can be more intuitive. The aim of the game is to solve the structure of a protein with the lowest energy.

Consider how you might introduce the game in a 3 minute flash presentation/elevator pitch?

How do proteins do fulfil the function they are made for?

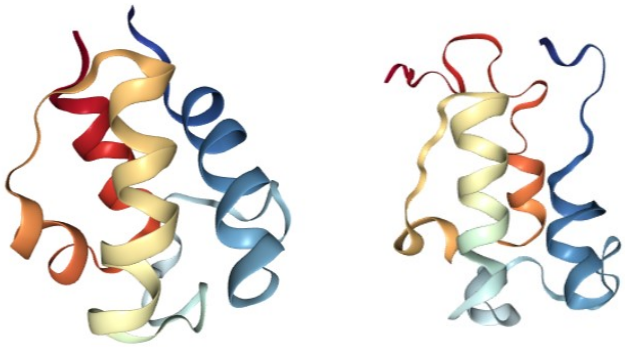
Activity – explore PDB and look at the protein in NGL viewer and export the image.
NGL (proteinformatics.charite.de/ngl) is web based software for visualising protein structures.
Detailed instructions for the activity will be provided during the tutorial.

Today's Homework is:

The protein serine threonine kinase 1 (SRPK1) is involved in creating blood vessels. In some cancers it goes wrong and scientists are trying to develop cancer therapies which can fix SRPK1.

Analyse the structure of SRPK1 (PDB code: 4WUA) bound to an experimental drug (SRPIN340). Prepare a figure presenting the chemical interactions that hold the inhibitor in particular place in the structure. Think how to present it in the most informative manner and annotate the figure! Send it to your tutor by email.

Tutorial 3: Mutation of proteins as the engine of evolution



What is the Purpose of Tutorial 3?

- What does evolution look like on molecular level?
- Changes in protein can lead to the changes in shape and function

Concepts:

<u>What is evolution?</u>

<u>What causes the changes in phenotype?</u>

Activity – change the Lego so it fits better in the box with minimum number of changes. Note the changes.

Remove = deletion

Insert = insertion

Exchange = substitution

Activity – how the insulin-like growth factor receptor (IGF2R) evolved – analyse proteins in NGL viewer.
IGF2R is involved in embryonic growth and development of cancer.
Detailed instructions for the activity will be provided during the tutorial.

Today's Homework is:

You will be given two texts about the same scientific topic. Read these texts and:
1. Highlight the buzz words that are characteristic for each piece
2. Write up short summary (up to 100 words) describing the difference about the style of the writing used in each text. You will need to reference both texts (please look at the Appendix 1 of this course handbook for guidance on referencing) and add their references in the form of a bibliography at the end of your analysis.
3. Write a scientific style summary (between 150-200 words) from the scientific text and make 3 bullet points briefly stating the most important findings.
Please send answers to the questions 2. and 3. by email.

theguardian

Genetic map of cancer reveals trails of mutation that lead to disease

Map shows how 20 patterns of mutation drive 30 cancer types, pointing the way to prevention and treatment strategies

Ian Sample, science correspondent

Wednesday 14 August 2013 18.00 BST

The first detailed map of genetic faults that cause cancers is published today, offering profound insights into the disease.

The map describes more than 20 "genetic signatures", or patterns of mutation, that alone or in combination drive 30 different types of cancer, including brain, lung, pancreas and breast tumours.

Independent cancer specialists who have seen the research said it was "extremely important" and was likely to lead to new strategies to prevent and ultimately treat the disease.

While scientists know, or have a good idea about, the causes of around half of the genetic signatures behind cancers, the rest are a mystery. They may be due to chemicals in the environment, or faults that arise during ageing.

Most cancers are thought to be caused by mutations in DNA, perhaps triggered by chemicals or radiation, which go unrepaired and build up over a person's lifetime. The mutations eventually lead to uncontrolled cell proliferation.

The causes of some cancers are well understood.

Too much ultraviolet (UV) light from the sun's rays causes mutations in skin cells, and if those mutations fall in certain genes in one cell, the cell becomes a cancer. Similarly, the chemicals in tobacco smoke cause mutations in mouth, throat and lung cells, and lead directly to tumours in those areas.

"What is remarkable is how little we know about the processes that are causing the mutations in other types of cancer," said Mike Stratton, the lead author on the study and director of the Sanger Institute in Cambridge. "That is the question we set out to address."

His team studied more than 7,000 genomes from cancer patients. By comparing the DNA of cancer cells with that of healthy cells in the same patients, they spotted patterns of mutations that arose in common forms of the disease.

In skin cancer, for example, UV light usually caused a mutation in which the letter "C" in the genetic code was changed into a "T". This switch could occur at various locations. Similarly, the most damaging tobacco carcinogens turned a "C" into an "A" to cause lung cancer.

All of the cancers were driven by two or more patterns of mutation, though some were more complex. Liver cancer was the result of six different mutation processes, according to the

study in Nature.

Some patterns of mutations caused only one type of cancer, for example neuroblastoma in young children, but others were common in many forms of the disease. In 25 of the 30 cancers, at least one mutational signature was due to ageing. Another pattern of mutations, caused by the faulty repair of damaged DNA, appeared in pancreatic, ovarian and breast cancers.

The work has shed light on how the body's natural defences might inadvertently drive a range of cancers. When cells are infected by viruses, they can switch on genes that produce a family of enzymes. These enzymes destroy viruses by mutating their DNA, but the onslaught may cause collateral damage. "It's speculation, but it may be that in killing the virus, many mutations are scattered in the genome of the cell itself, and that cell can then go on to become a cancer," said Stratton.

The findings are expected to drive research into the causes of mutations behind each cancer. One way to do this is to expose human cells to suspected carcinogens in the environment to see if they produce similar patterns of mutations. The work could also lead to the development of drugs that mutate cells even further and kill them off in the process.

Ray Waters, head of cancer studies at Cardiff School of Medicine, said the map was a step towards more personalised treatments for cancer. "Cancer medicine is moving away from general therapies that are used on everyone towards knowing what mutations are causing cancer in an individual and what then will be the best therapies to begin with."

Nic Jones, chief scientist at Cancer Research UK, said: "We know that environmental factors like smoking and overexposure to UV rays can cause faults in DNA which can lead to cancer, but for many cancers we don't know what triggers the faults ... The genetic fingerprints identified in this fascinating and important study identify several new processes driving the development of cancer. Understanding what's causing them could be an extremely important way to get to the bottom of how cancer develops in the first place, and this will lead to new ways to prevent and treat the disease."

22 AUGUST 2013 | VOL 500 | NATURE |

Signatures of mutational processes in human cancer

All cancers are caused by somatic mutations; however, understanding of the biological processes generating these mutations is limited. The catalogue of somatic mutations from a cancer genome bears the signatures of the mutational processes that have been operative. Here we analysed 4,938,362 mutations from 7,042 cancers and extracted more than 20 distinct mutational signatures. Some are present in many cancer types, notably a signature attributed to the APOBEC family of cytidine deaminases, whereas others are confined to a single cancer class. Certain signatures are associated with age of the patient at cancer diagnosis, known mutagenic exposures or defects in DNA maintenance, but many are of cryptic origin. In addition to these genome-wide mutational signatures, hypermutation localized to small genomic regions, 'kataegis', is found in many cancer types. The results reveal the diversity of mutational processes underlying the development of cancer, with potential implications for understanding of cancer aetiology, prevention and therapy.

The text below is taken from the above Nature article.

Somatic mutations found in cancer genomes may be the consequence of the intrinsic slight infidelity of the DNA replication machinery, exogenous or endogenous mutagen exposures, enzymatic modification of DNA, or defective DNA repair. In some cancer types, a substantial proportion of somatic mutations are known to be generated by exposures, for example, tobacco smoking in lung cancers and ultraviolet light in skin cancers, or by abnormalities of DNA maintenance, for example, defective DNA mismatch repair in some colorectal cancers. However, our understanding of the mutational processes that cause somatic mutations in most cancer classes is remarkably limited.

Different mutational processes often generate different combinations of mutation types, termed 'signatures'. Until recently, mutational signatures in human cancer have been explored through a small number of frequently mutated cancer genes. Although informative, these studies have limitations. To generate a mutational signature, a single mutation from each cancer sample is entered into a mutation set aggregated from several cases of a particular cancer type. A signature that contributes the large majority of somatic mutations in the tumour class is accurately reported. However, if multiple mutational processes are operative, a jumbled composite signature is generated.

We compiled 4,938,362 somatic substitutions and small insertions/deletions from the mutational catalogues of 7,042 primary cancers of 30 different classes (507 from whole genome and 6,535 from exome sequences). In all cases, normal DNA from the same individuals had been sequenced to establish the somatic origin of variants.

Certain childhood cancers (like neuroblastoma) carried fewest mutations whereas cancers related to chronic mutagenic exposures such as lung (tobacco smoking) and malignant melanoma (exposure to ultraviolet light) exhibited the highest prevalence. This variation in mutation prevalence is attributable to differences between cancers in the duration of the cellular lineage between the fertilized egg and the sequenced cancer cell and/or to differences in somatic mutation rates during the whole or parts of that cellular lineage.

In principle, all classes of mutation (such as substitutions, indels, rearrangements) and any accessory mutation characteristic, for example, the sequence context of the mutation or the transcriptional strand on which it occurs, can be incorporated into the set of features by which a mutational signature is defined.

Applying this approach to the 30 cancer types revealed 21 distinct validated mutational signatures. These show substantial diversity. There are signatures characterized by prominence of only one or two of the 96 possible substitution mutations, indicating remarkable specificity of mutation type and sequence context. By contrast, others exhibit a more-or-less equal representation of all 96 mutations. There are signatures characterized predominantly by C>T (6 signatures), C>A (3 signatures), T>C (6 signatures) and T>G mutations (2 signatures).

Each mutational signature is the imprint left on the cancer genome by a mutational process that may include one or more DNA damage and/or DNA maintenance mechanisms, with the latter either functioning normally or abnormally. Here we consider likely mechanisms or underlying causes by comparing signatures with mutation patterns of known causation in the scientific literature or by associating them with epidemiological and biological features of particular cancer types.

Abnormalities in DNA maintenance may be responsible for mutational signatures, and the roles of defective DNA mismatch repair and defective homologous-recombination-based DNA double-strand break repair have been discussed above. Other signatures may result from abnormal activity of enzymes that modify DNA or of error-prone polymerases. Signatures 2 and 13 have been attributed to the APOBEC family of cytidine deaminases. On the basis of similarities in the sequence context of cytosine mutations caused by APOBEC enzymes in experimental systems, a role for APOBEC1, APOBEC3A and/or APOBEC3B in human cancer seems more likely than for other members of the family. However, the reason for the extreme activation of this mutational process in some cancers is unknown. Because APOBEC activation constitutes part of the innate immune response to viruses and retrotransposons it may be that these mutational signatures represent collateral damage on the human genome from a response originally directed at exogenous viruses. Confirmation of this hypothesis would establish an important new mechanism for initiation of human carcinogenesis.

The diversity and complexity of somatic mutational processes underlying carcinogenesis in human beings is now being revealed through mutational patterns buried within cancer genomes. It is likely that more mutational signatures will be extracted, together with more precise definition of their features, as the number of whole-genome sequenced cancers increases and analytical methods are further refined.

The mechanistic basis of some signatures is, at least partially, understood but for many it remains speculative or unknown. Elucidating the underlying mutational processes will depend upon two major streams of investigation. First, compilation of mutational signatures from model systems exposed to known mutagens or perturbations of the DNA maintenance machinery and comparison with those found in human cancers. Second, correlation of the contributions of mutational signatures with other biological characteristics of each cancer through diverse approaches ranging from molecular profiling to epidemiology. Collectively, these studies will advance our understanding of cancer aetiology with potential implications for prevention and treatment.

Tutorial 4: Are you really different?

What is the Purpose of Tutorial 4?

- How can we establish whether a mutation causes any meaningful changes in protein?

Concepts:

Proteins have a range of physical properties

Activity – Analyse the changes in insulin-like growth factor receptor upon mutation (PDB code: 2cnj)
Detailed instructions for the activity will be provided during the tutorial.

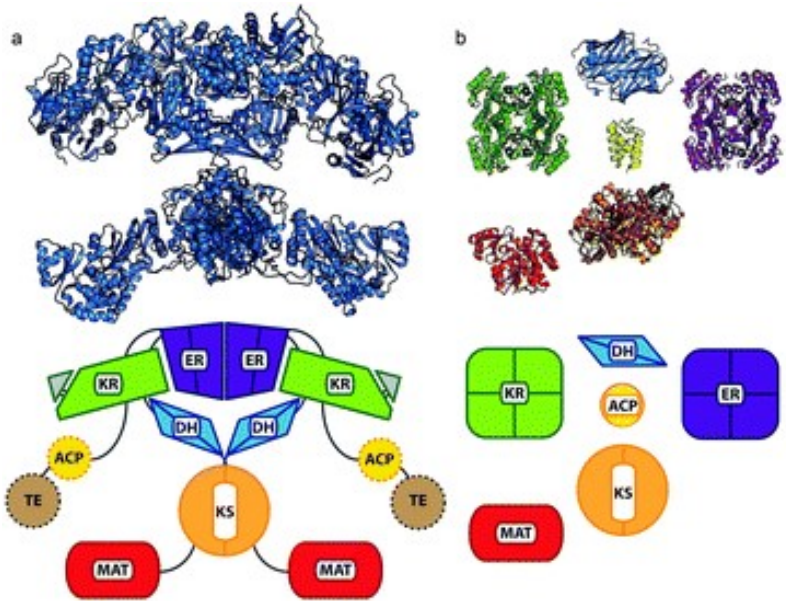
1. Use Expasy server to estimate pI and charge
2. Use STRUM (zhanglab.ccmb.med.umich.edu/STRUM/) to predicting the fold stability change

Activity – how to present the numeric data – preparing a graph.
We will use the data generated from the activity above.

Today's Homework is:

Prepare a graph presenting differences in physical properties of selected mammalian lysozymes (detailed instructions will be provided during the tutorial). Think how to present it and annotate it! Send it to your tutor by email.

Tutorial 5: Evolution on a large scale – fatty acid synthase



What is the Purpose of Tutorial 5?

- Learn about fatty acid sythase
- What kind of changes can be introduced to mega complex to make it more efficient?

Concepts:

<u>What is an enzyme?</u>

<u>Why and how do we measure enzyme efficiency?</u>

Activity – how to present the information in manner attracting attention.
We will analyze some examples of "good" and "bad" scientific posters.

Final assignment:

Prepare and present scientific poster:

Sickle-cell anaemia is caused by a single point mutation in haemoglobin that causes substantial changes in the physical properties of the protein.

You will be expected to:
- provide an introduction
- provide background information
- test whether the above hypothesis is correct
- explain your methods
- give a summary with your conclusion and possible future work
- your oral presentation should be no longer then 5 min.

Tutorial 6 – Feedback tutorial

What is the Purpose of Tutorial 6?

- To receive feedback on final assignments.
- To share examples of best practice with the other pupils in your group.
- To write targets for improvement in school lessons.
- To reflect on the programme including what was enjoyed and what was challenging.

Final assignment feedback

What I did well…	What I could have improved on…
•	•
•	•
•	•

My target for future work is…

Reflecting on Uni Pathways

What did you most enjoy about Uni Pathways?
•   •   •

What did you find challenging about the programme?	How did you overcome these challenges?
• • •	• • •

Appendix 1 – Referencing correctly

When you get to university, you will need to include references in the assignments that you write, so we would like you to start getting into the habit of referencing in your Brilliant Club assignment. This is really important, because it will help you to avoid plagiarism. Plagiarism is when you take someone else's work or ideas and pass them off as your own. Whether plagiarism is deliberate or accidental, the consequences can be severe. In order to avoid losing marks in your final assignment, or even failing, you must be careful to reference your sources correctly.

What is a reference?

A reference is just a note in your assignment which says if you have referred to or been influenced by another source such as book, website or article. For example, if you use the internet to research a particular subject, and you want to include a specific piece of information from this website, you will need to reference it.

Why should I reference?

Referencing is important in your work for the following reasons:

- It gives credit to the authors of any sources you have referred to or been influenced by.
- It supports the arguments you make in your assignments.
- It demonstrates the variety of sources you have used.
- It helps to prevent you losing marks, or failing, due to plagiarism.

When should I use a reference?

You should use a reference when you:

- Quote directly from another source.
- Summarise or rephrase another piece of work.
- Include a specific statistic or fact from a source.

How do I reference?

There are a number of different ways of referencing, and these often vary depending on what subject you are studying. The most important to thing is to be consistent. This means that you need to stick to the same system throughout your whole assignment. Here is a basic system of referencing that you can use, which consists of the following two parts:

1. A marker in your assignment : After you have used a reference in your assignment (you have read something and included it in your work as a quote, or re-written it your own words) you should mark this is in your text with a number, e.g. [1]. The next time you use a reference you should use the next number, e.g. [2].
2. Bibliography: This is just a list of the references you have used in your assignment. In the bibliography, you list your references by the numbers you have used, and include as much information as you have about the reference. The list below gives what should be included for different sources.

    a. Websites – Author (if possible), title of the web page, website address, [date you accessed it, in square brackets].

    E.g. Dan Snow, 'How did so many soldiers survive the trenches?', http://www.bbc.co.uk/guides/z3kgjxs#zg2dtfr [11 July 2014].

    b. Books – Author, date published, title of book (in italics), pages where the information came from.

    E.g. S. Dubner and S. Levitt, (2006) *Freakonomics*, 7-9.

    c. Articles – Author, 'title of the article' (with quotation marks), *where the article comes from* (newspaper, journal etc.), date of the article.

    E.g. Maev Kennedy, 'The lights to go out across the UK to mark First World War's centenary', Guardian, 10 July 2014.

Appendix 2 – Using the VLE

VLE username	
VLE password	

Please remember the following key details...

- You are able log into the VLE either through the link on our website (www.thebrilliantclub.org) or going directly to the VLE site at (https://portal.thebrilliantclub.org/sign-in).
- Please update your profile with your full name and email address- this will allow you to retrieve forgotten passwords or usernames
- If you forget your log-in details you can request them to be emailed to you by clicking the link on the VLE home page. (If you are still having problems you can email: schools@thebrilliantclub.org)

What is the VLE?

The VLE is a virtual learning environment for all pupils on Uni Pathways it is used for:

- messaging your tutor
- submitting homework
- submitting your final assignment
- accessing resources for your tutorials
- finding out more information about university and careers

How should I use the VLE?

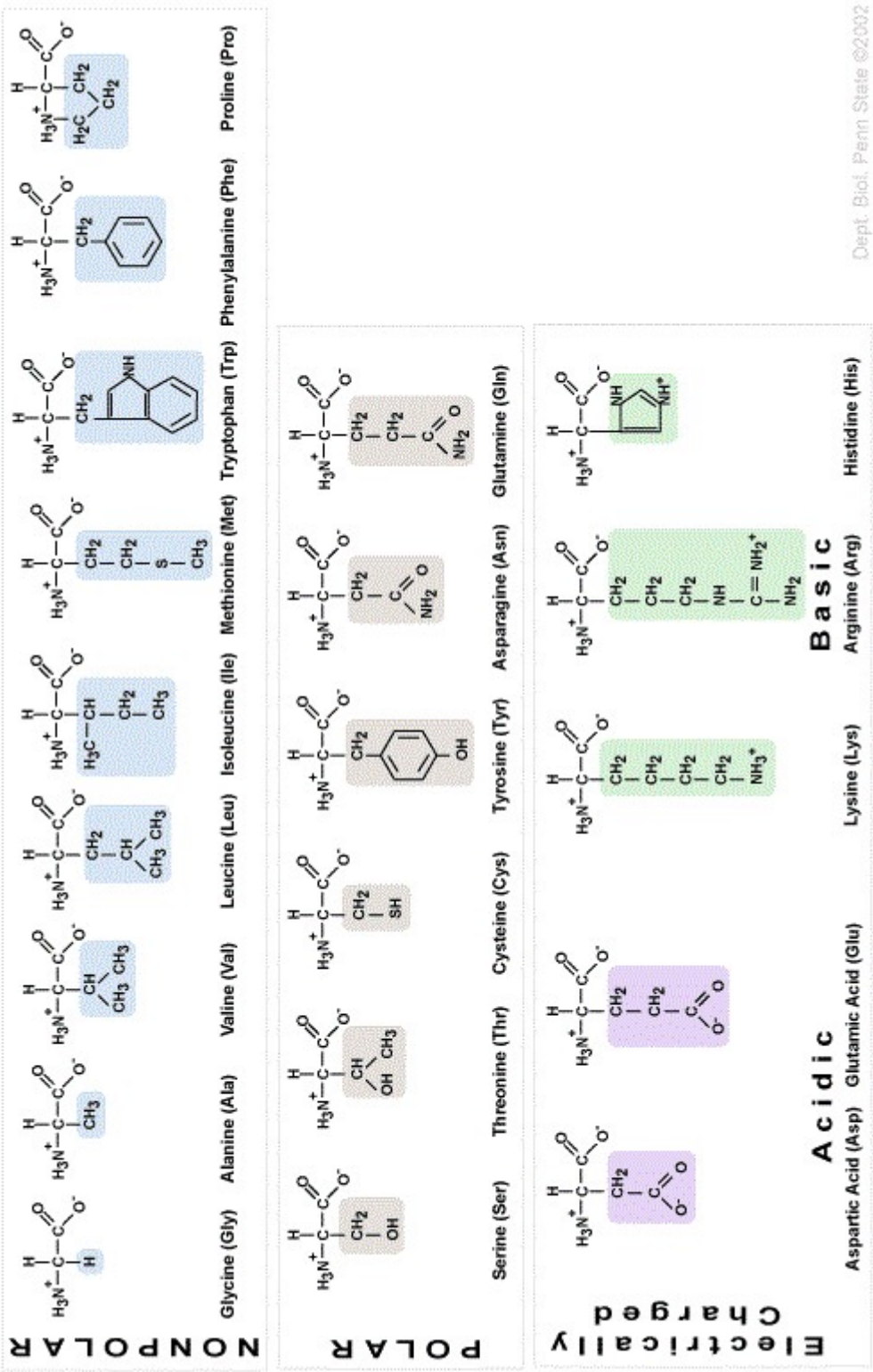
The VLE is a professional academic environment in which pupils are able to message their PhD Tutor. Here are a few things to consider:

- Ensure you keep a professional tone in the messages you send to your tutors.
- Ensure you always reply to your tutors in a timely manner.
- Thank your tutor for the effort they are putting in to give you your feedback etc.
- Submit all homework to your tutor on time.

IMPORTANT: Final assignment

- When you submit your final assignment, please remember that you need to do so through the 'My Activities' tab and not as an attachment to a message.

Appendix 3 – Amino Acids



Notes

Notes

Notes

Proteins: it's all about the shape

Key Stage 4 Programme

researchersinschools.org