Medicinal Chemistry
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Miscellaneous Targets

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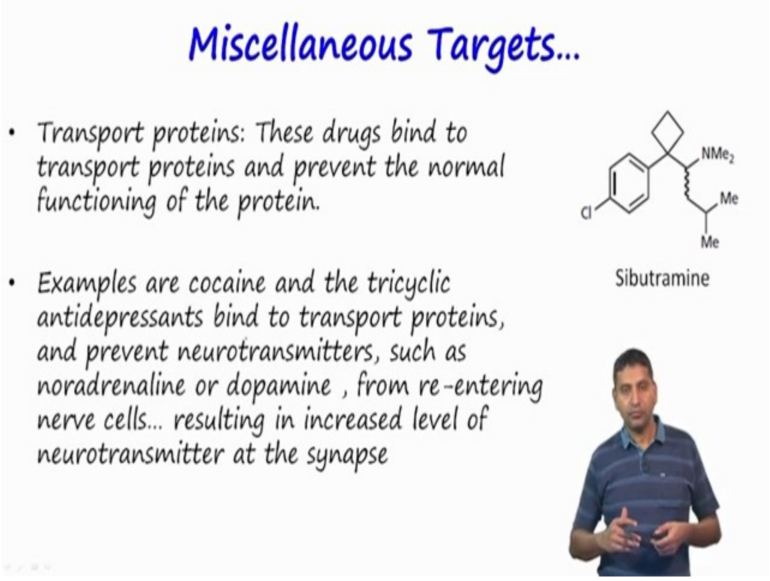

Miscellaneous Targets...

- Transport proteins: These drugs bind to transport proteins and prevent the normal functioning of the protein.
- Examples are cocaine and the tricyclic antidepressants bind to transport proteins, and prevent neurotransmitters, such as noradrenaline or dopamine , from re-entering nerve cells... resulting in increased level of neurotransmitter at the synapse

Sibutramine

Now let us look at some other miscellaneous targets, so we have looked at earlier what transport proteins are and so these are proteins which are which sort of help in transporting important metabolites. So for example we can look at amino acid transporters or transporters of neurotransmitters and so on, okay. (So if we can) there are drugs which can bind to these transport proteins and prevent the normal functioning of the protein.

So some examples are cocaine and some tricyclic antidepressants and these go and prevent neurotransmitters from getting back into the cell. So once it prevents this now because it is present in the synaptic area it results in increase level of neurotransmitter activity, okay.

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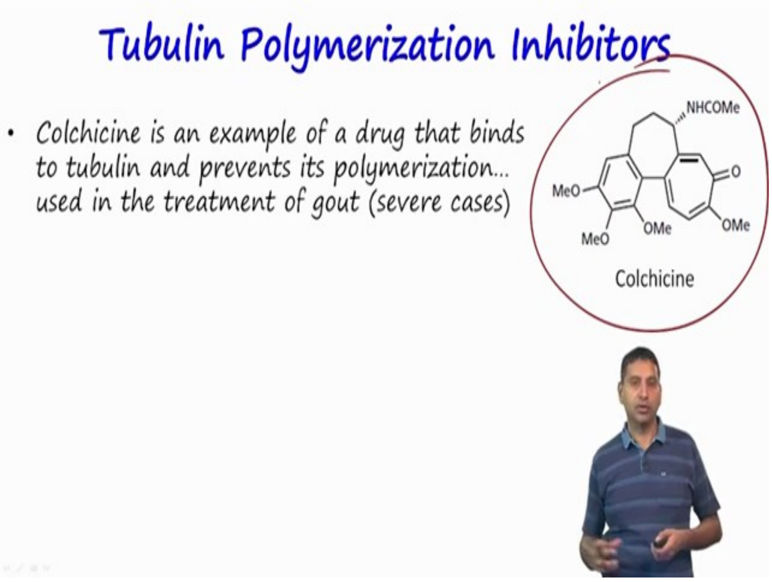

Tubulin Polymerization Inhibitors

- Colchicine is an example of a drug that binds to tubulin and prevents its polymerization… used in the treatment of gout (severe cases)

So next example is inhibitors of tubulin polymerization, we have already looked at previously that polymerization of tubulin is an important process during a cell division. So an example of an inhibitor of polymerization so what will happen is if we inhibit the polymerization then you are able to prevent the cell from dividing and this is extremely useful in the treatment of cancer for example.

So Colchicine whose structure is shown here is one such example of inhibitor of tubulin polymerization and this compound is actually used in the treatment of gout so where there is quite a bit of swelling in certain areas of the body.

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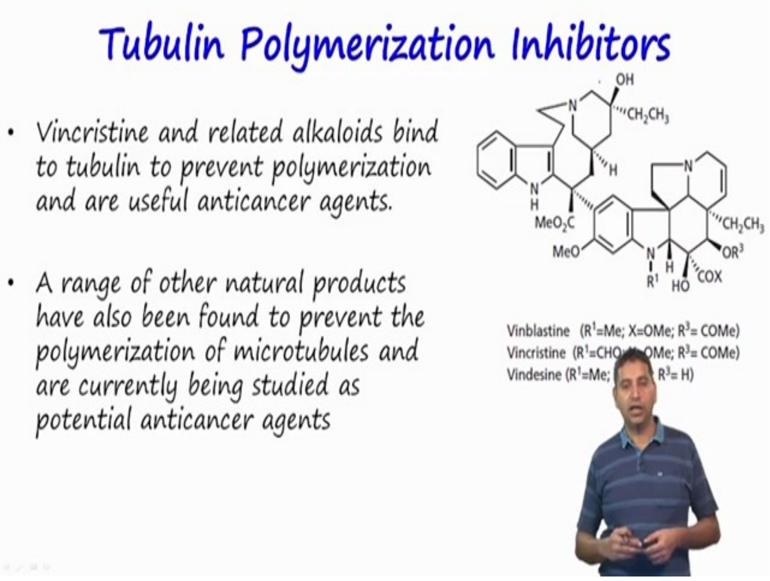

Tubulin Polymerization Inhibitors

- Vincristine and related alkaloids bind to tubulin to prevent polymerization and are useful anticancer agents.
- A range of other natural products have also been found to prevent the polymerization of microtubules and are currently being studied as potential anticancer agents

Vinblastine (R^1=Me; X=OMe; R^3= COMe)
Vincristine (R^1=CHO; OMe; R^3= COMe)
Vindesine (R^1=Me; R^3= H)

There are other natural products and natural product like molecules which are also known to have tubulin polymerization activity. An example is Vincristine, Vinblastine and so on and these are alkaloids which are extracted from natural sources and these bind to tubulin and prevent the polymerization and these complex molecules have very useful anticancer activity, okay and some of the analogues of these compounds are being examined or being studied as potential anticancer drugs.

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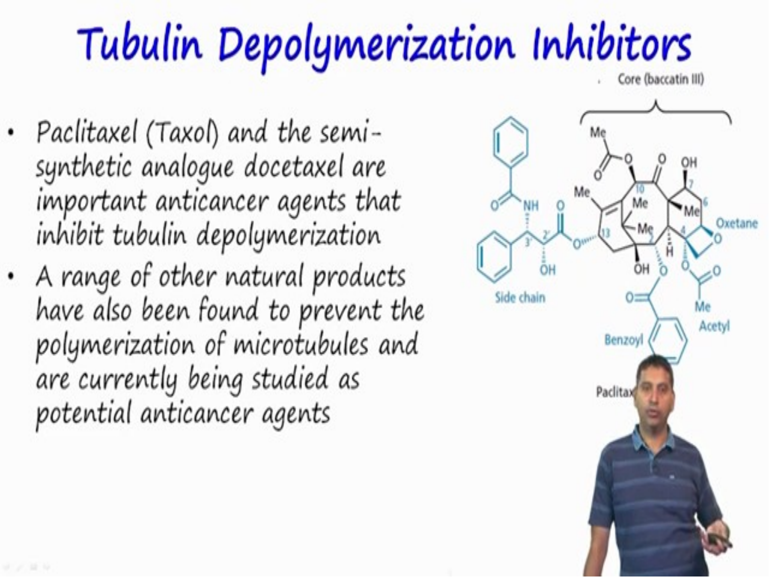

Tubulin Depolymerization Inhibitors

- Paclitaxel (Taxol) and the semi-synthetic analogue docetaxel are important anticancer agents that inhibit tubulin depolymerization
- A range of other natural products have also been found to prevent the polymerization of microtubules and are currently being studied as potential anticancer agents

The counter view or the opposite example is in tubulin depolymerization, so tubulin depolymerization also needs to occur because this is a process by which the normal cell I mean the cell functions normally and so inhibition of depolymerization also becomes an important way to inhibit cancers. So the natural product Taxol, I mean Paclitaxel which is commercially known as Taxol and semi-synthetic analogue, so semi-synthetic analogue means something that has been it has a structure which is similar to it but some of it is actually synthesized and these are known to inhibit depolymerization, okay and there are range of other natural products which have also been found to prevent the polymerization of microtubules and so on.

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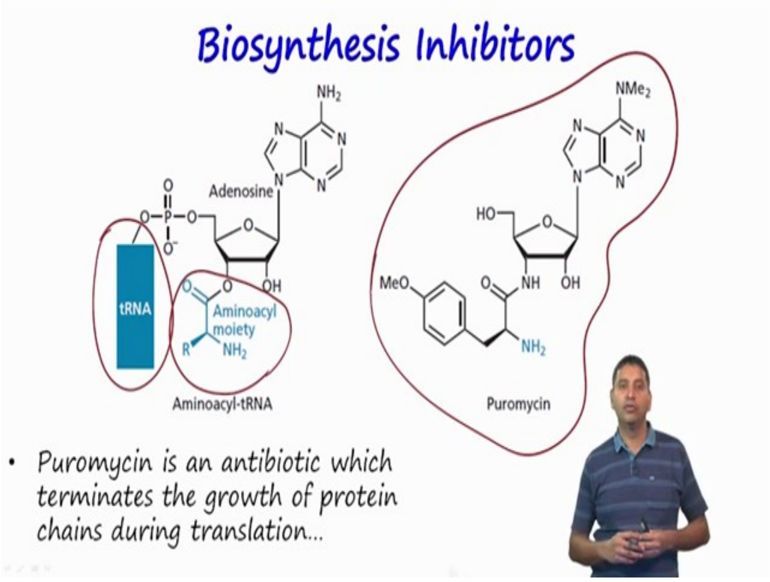

Biosynthesis Inhibitors

- Puromycin is an antibiotic which terminates the growth of protein chains during translation...

Now the other way in which one can design new drugs or one can look at drug targets is to inhibit Biosynthesis. So we have already looked at in detail the Biosynthesis of proteins, so proteins are synthesized by you know by using the machinery of RNA and RNA has this aminoacyl-tRNA which has the which is shown here which is then bound to the amino acid over here as shown here and this forms this is the charged or the armed tRNA and then it goes and binds in the ribosome and once it binds then its go into transfer this amino acid to the growing peptide chain. So Puromycin whose structure is shown here is a very good example of an inhibitor of this process.

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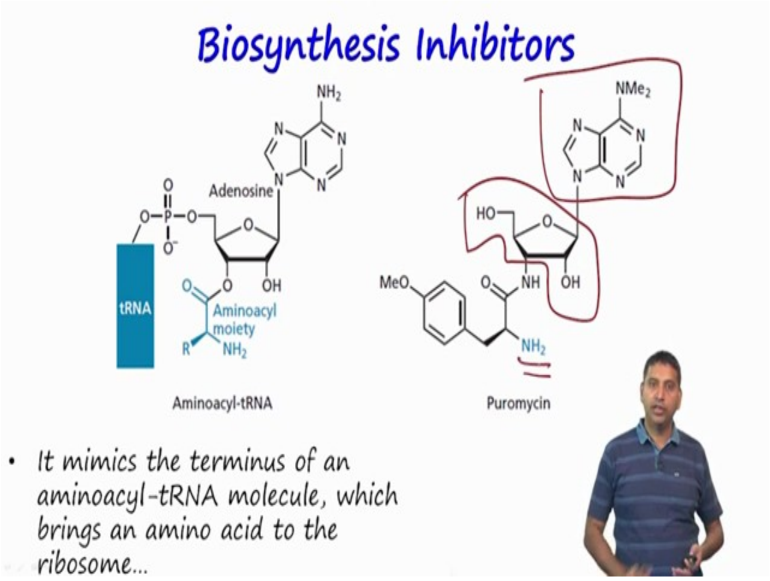

Biosynthesis Inhibitors

- It mimics the terminus of an aminoacyl-tRNA molecule, which brings an amino acid to the ribosome...

So what happens is that this actually mimics the terminus of the aminoacyl-tRNA molecule, so you see here the structure, here you have the adenosine part remains the same, the sugar part has the very similar structure as you can see here and there is an amino acid here, but the amino acid is now bound to an amide instead of a ester. So once it goes and binds to the ribosome, it acts as a way in which you can inhibit the biosynthesis.

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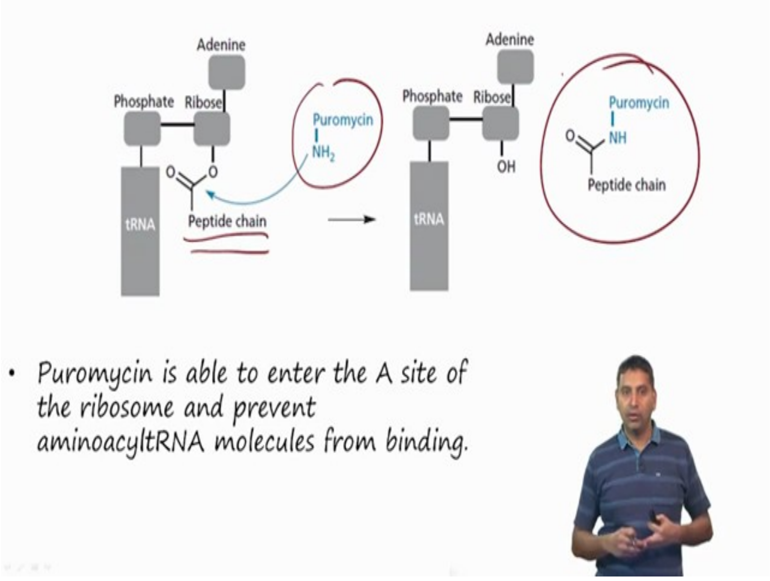

- Puromycin is able to enter the A site of the ribosome and prevent aminoacyltRNA molecules from binding.

So let us look at the example here, so what puromycin does is that it comes here and it can react with the growing peptide chain and once puromycin is bound here there is no further reaction possible and so it dissociates and it prevents the protein from growing.

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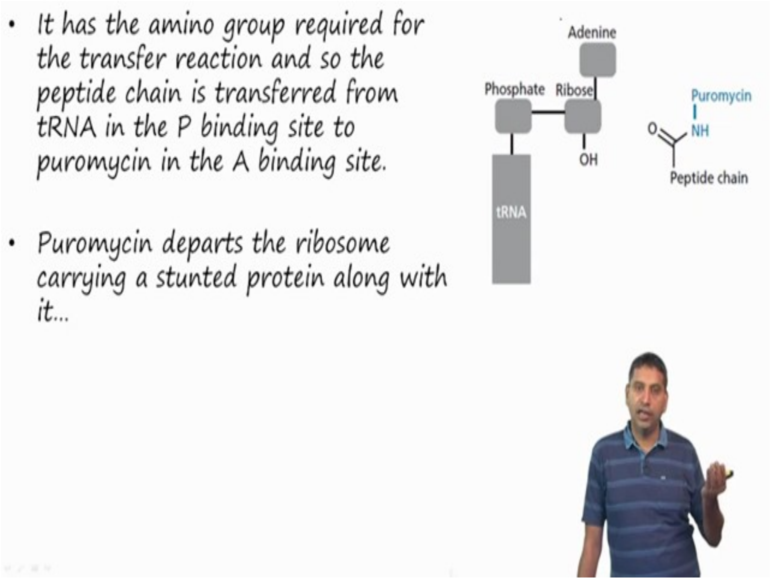

- It has the amino group required for the transfer reaction and so the peptide chain is transferred from tRNA in the P binding site to puromycin in the A binding site.
- Puromycin departs the ribosome carrying a stunted protein along with it...

(So once the protein) so it has the amino group required for the transfer reaction and so the peptide chain is transferred from tRNA in the P binding site to puromycin in the A binding site and then puromycin departs and carrying the protein, but the protein now is stunted, okay so let us say it supposed to have two hundred and fifty amino acids but let us say this process occurs at around 85, 87 then it stops at that process and then the entire protein is not synthesized.

So in this manner what happens is that puromycin can inhibit the biosynthesis of proteins and because the protein is now stunted the function of the protein is not the same and once you have the function of protein inhibited the cell can it can be fatal for the cell. So puromycin is a very important example of drug that inhibits biosynthesis.

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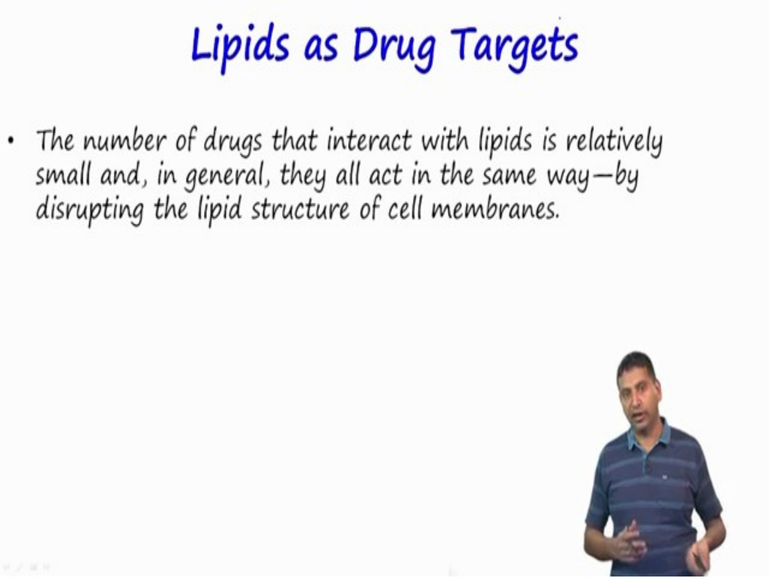

Lipids as Drug Targets

- The number of drugs that interact with lipids is relatively small and, in general, they all act in the same way—by disrupting the lipid structure of cell membranes.

Now in a cell we have many components, so we looked at proteins which are made up of amino acids and these proteins can be part of enzymes or it can be part of receptors and so on, and you also have importantly DNA and which is nucleic acids and we have already looked at how to target nucleic acids. The third major component inside the cell is lipids, so lipids are basically hydrophobic molecules they are typically long chain molecules which have lot of carbons in it or they can also be cyclic molecules such as cholesterol, but they are extremely hydrophobic in nature.

And we have already looked at previously that lipids are you know important components of the cell membrane, okay and so if lipids are considered as drug targets then what would happen is that they would disrupt the lipid structure of the cell membrane and once this is disrupted the protective layer around the cell is now you know become less efficient and the cell can rupture and die, okay.

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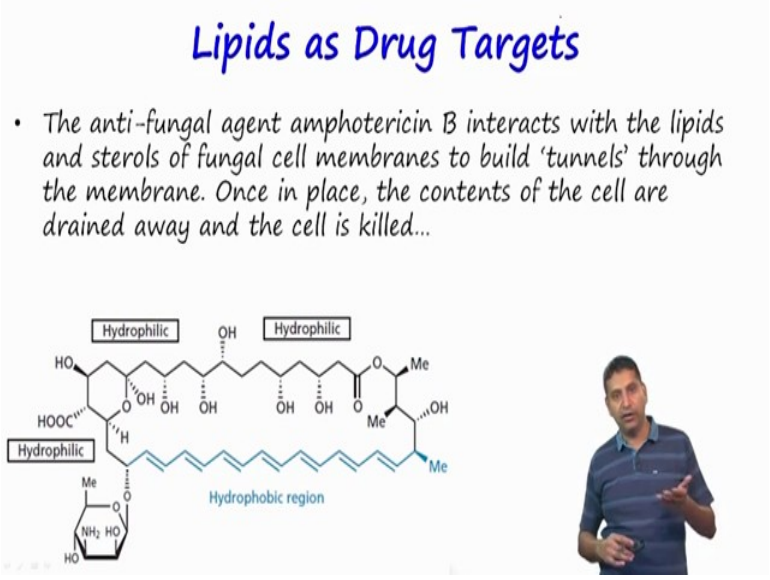

Lipids as Drug Targets

- *The anti-fungal agent amphotericin B interacts with the lipids and sterols of fungal cell membranes to build 'tunnels' through the membrane. Once in place, the contents of the cell are drained away and the cell is killed...*

So the first example that we looked at is the anti-fungal agent amphotericin B, so this is known to interact with lipids and sterols on the fungal cell membrane and what it does is it helps in building tunnels. So we will look at how the process happens, but once this tunnel is made then the contents of the cell are now going to be drained away there is going to be imbalanced in the inside the cell and the cell dies.

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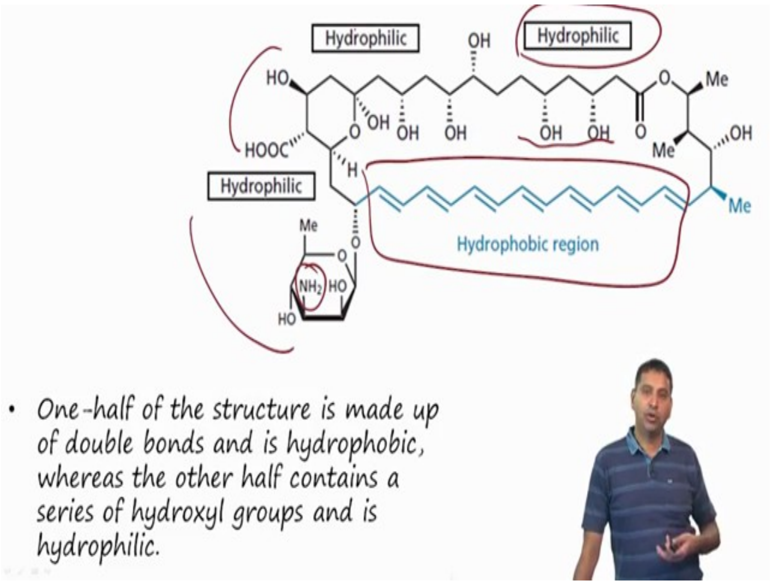

- *One-half of the structure is made up of double bonds and is hydrophobic, whereas the other half contains a series of hydroxyl groups and is hydrophilic.*

There are four major regions amphotericin B and so the first one that we look at is the hydrophobic region which is imparted by this long chain olefin rich lipid region and there is also hydrophilic region here you have a number of hydroxyl groups over here and then there are two hydrophilic regions one is composed of an amino sugar which is shown here and the other one is regular sugar with a carboxylic acid on it.

So essentially one half of the structure is made of hydrophobic regions and contains a number of double bonds and the other half contains hydroxyl groups and is actually hydrophilic. So this makes the molecule very unique.

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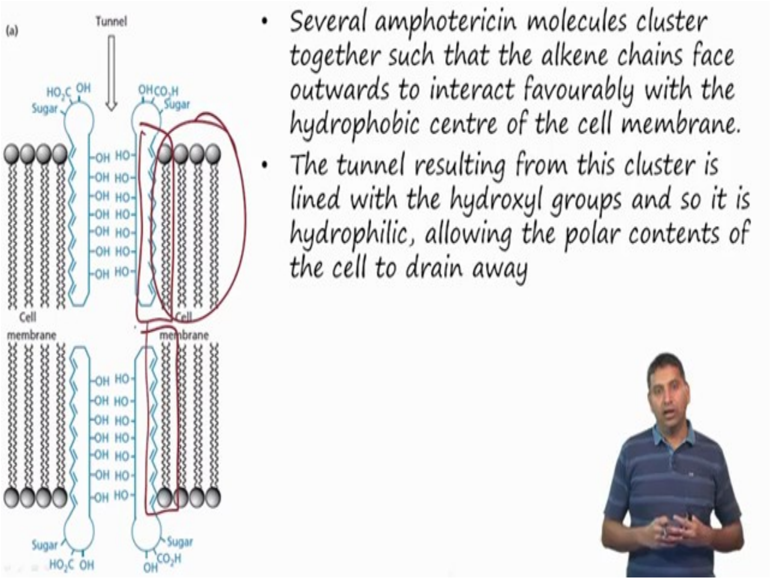

- Several amphotericin molecules cluster together such that the alkene chains face outwards to interact favourably with the hydrophobic centre of the cell membrane.
- The tunnel resulting from this cluster is lined with the hydroxyl groups and so it is hydrophilic, allowing the polar contents of the cell to drain away

Now because of this very unique structure what happens is that several amphotericin molecules can actually cluster together, the way they cluster is that you will have the hydrophobic regions actually interacting with other hydrophobic regions and whereas the hydrophilic regions will interact with the hydrophilic regions. So as we already know that in a cell membrane the area is merely made of lipids and lipids are the centre of the internal part of the membrane is quite hydrophobic and the external part of the membrane is quite hydrophilic.

So once amphotericin B the hydrophobic part now interacts with the membrane then you can think about the hydrophobic regions actually coming close to each other and forming favourable interactions and this exposes the hydrophilic regions to the other side. So you can imagine a number of such molecules which are going to sort of self-assemble this manner and in the process what happens is that it can form some sort of a tunnel and this tunnel resulting from this cluster is lined with hydroxyl groups and so what can happen is that you have it allows for the polar contents in the cell to drain away and so this is drained away it causes ionic imbalance and which can lead to cell death.

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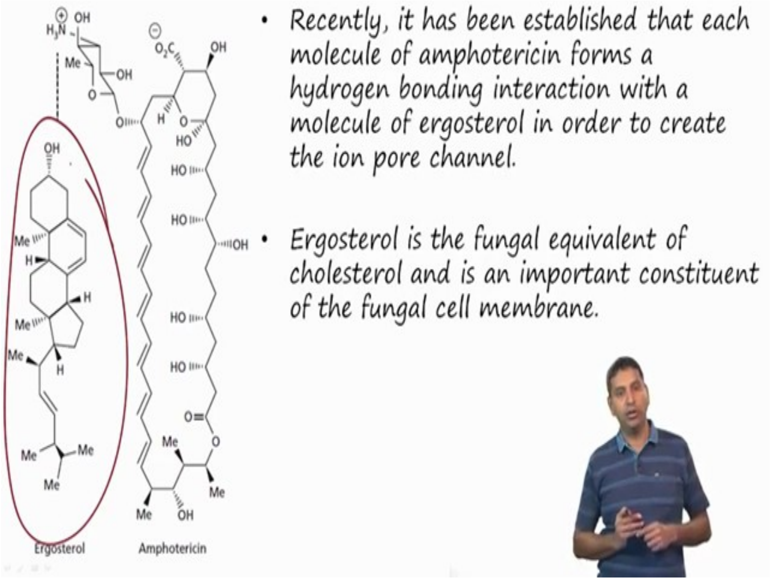

- Recently, it has been established that each molecule of amphotericin forms a hydrogen bonding interaction with a molecule of ergosterol in order to create the ion pore channel.
- Ergosterol is the fungal equivalent of cholesterol and is an important constituent of the fungal cell membrane.

There are some recent studies which have shown that amphotericin B actually forms a very important favourable hydrogen bonding interaction with Ergosterol. Ergosterol is the fungal equivalent of cholesterol and so this is the structure of Ergosterol over here and it is quite hydrophobic in nature, but there is a hydroxyl group at the terminus at one of the ends which can interact favourably with amphotericin B.

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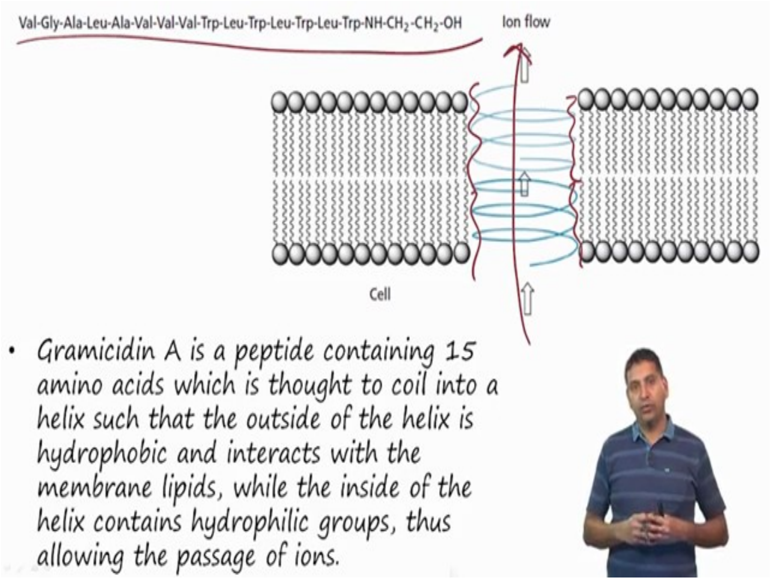

Val-Gly-Ala-Leu-Ala-Val-Val-Val-Trp-Leu-Trp-Leu-Trp-Leu-Trp-NH-CH_2-CH_2-OH

- Gramicidin A is a peptide containing 15 amino acids which is thought to coil into a helix such that the outside of the helix is hydrophobic and interacts with the membrane lipids, while the inside of the helix contains hydrophilic groups, thus allowing the passage of ions.

There are other peptides which are also known to form these kinds of channels or pores. So Gramicidin A which is whose structure is primary structure is shown here is a 15 amino acid containing peptide and what it can do is that because the it contains both hydrophobic as well as hydrophilic regions in its peptide structure it can interact favourably with the lipid membranes.

So what can happen is that you have the hydrophobic regions of the peptide (which are going to) it is going to form an alpha helix like structure and they are going to interact with the lipids and you can imagine that these are going to be stacked up and once it stacks it stacks up, it allows for ions to flow through it because the hydrophilic regions are going to be inside. So again this is a process by which ions can then escape from the cell and it leads to ionic imbalance and cell death.

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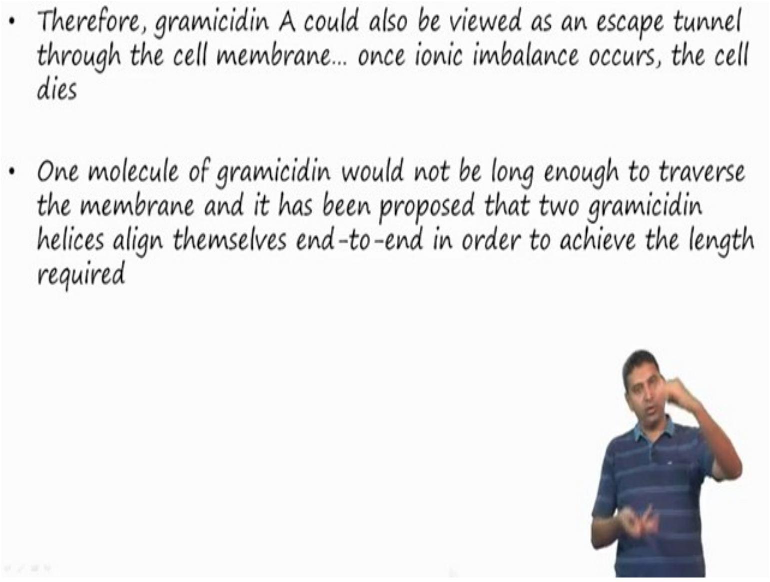

- Therefore, gramicidin A could also be viewed as an escape tunnel through the cell membrane... once ionic imbalance occurs, the cell dies
- One molecule of gramicidin would not be long enough to traverse the membrane and it has been proposed that two gramicidin helices align themselves end-to-end in order to achieve the length required

So Gramicidin A can also be viewed as an escape tunnel through the cell membrane. So once this ionic imbalance occurs the cell dies. One molecule of Gramicidin itself would not be long enough to traverse the membrane and so it appears that there will be two Gramicidin helices align themselves from end to end to achieve the length required.

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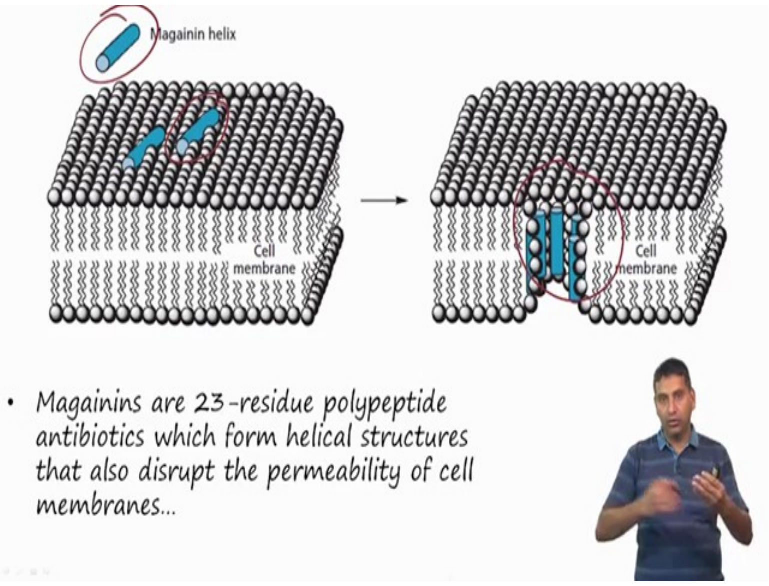

- Magainins are 23-residue polypeptide antibiotics which form helical structures that also disrupt the permeability of cell membranes...

So there is another class of polypeptides which is called Magainins and this is again a helix and it contains 23 residue polypeptides and these are antibiotics and they form helical structures which disrupts the permeability of cell membranes. So here is the an example of the cell membrane, so once it comes it binds to the this helix binds to the upper portion of the lipid membrane and then it actually aligns itself through process of self-assembly and it forms a pore.

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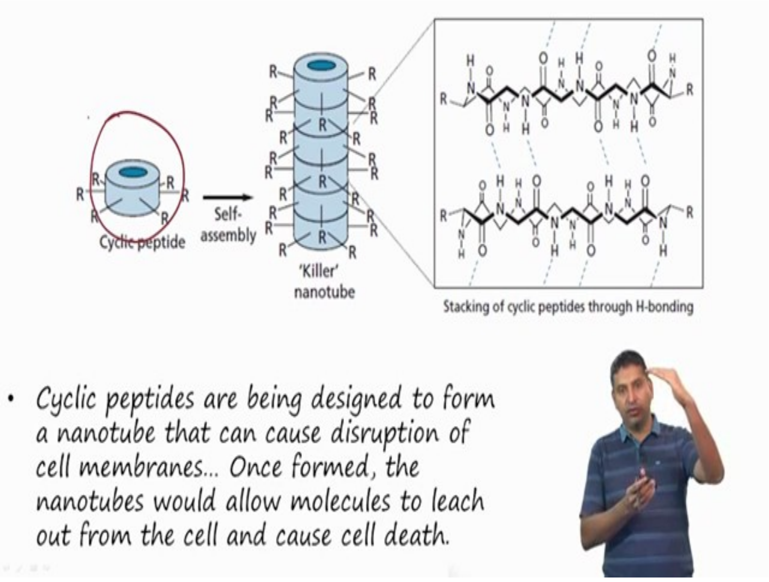

- Cyclic peptides are being designed to form a nanotube that can cause disruption of cell membranes... Once formed, the nanotubes would allow molecules to leach out from the cell and cause cell death.

So this cyclic peptide is shown by this cylinder over here and this cylinder can actually stack itself and it forms what is known as the nanotube and this nanotube can actually disrupt cell membranes, okay and once it is formed the nanotube would allow molecules leach out form the cell and cause cell death.

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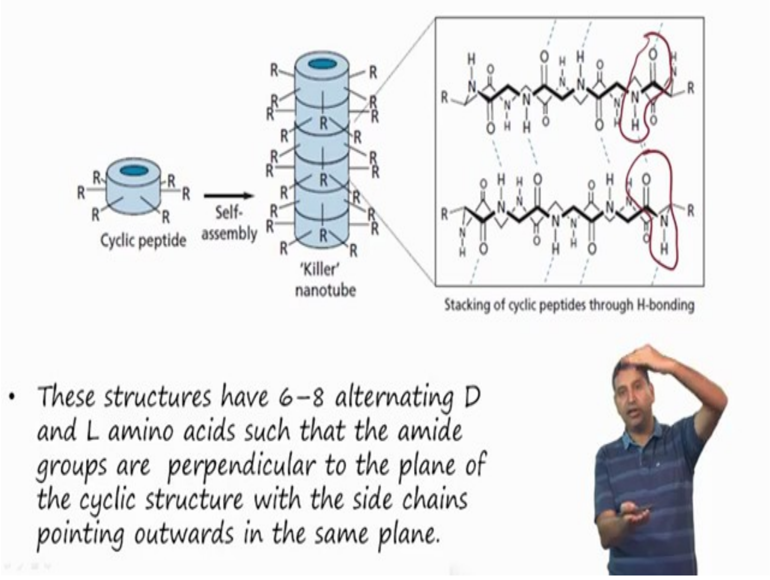

- These structures have 6–8 alternating D and L amino acids such that the amide groups are perpendicular to the plane of the cyclic structure with the side chains pointing outwards in the same plane.

If you look at closely at this nanotube, these structures have 6 to 8 alternating D and L amino acids, okay such that the amino acids the amide groups are perpendicular to the plane of the cyclic structure. So you have the amide group here and amide group over here and these are actually going to be perpendicular to the chain of the structure and once this forms the hydrogen bonded network then it can form multiples of these cyclic peptides can align themselves one on top of the other.

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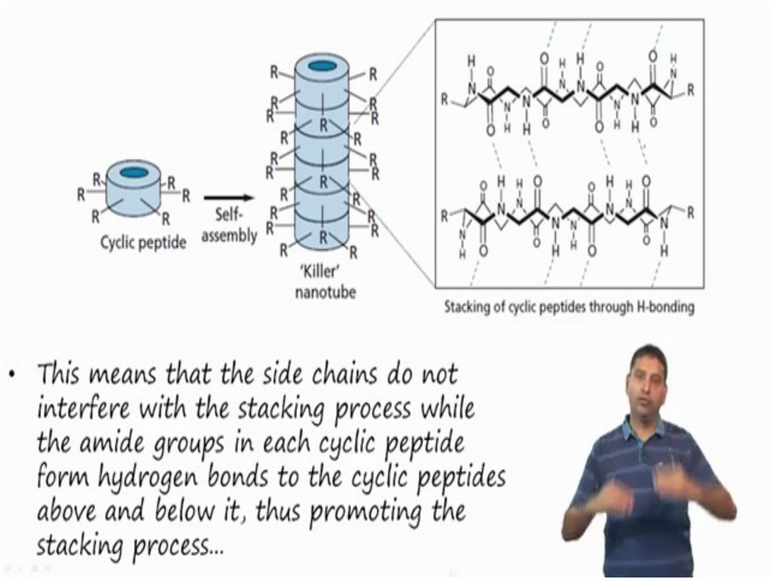

- This means that the side chains do not interfere with the stacking process while the amide groups in each cyclic peptide form hydrogen bonds to the cyclic peptides above and below it, thus promoting the stacking process...

And the side chains do not interfere with the stacking process and the side chains are actually exposed to the outside and each cyclic peptide forms hydrogen bonds to the cyclic peptide above and below it and once this forms this can form a nice self-assembled stack which can form sort of a nanotube.

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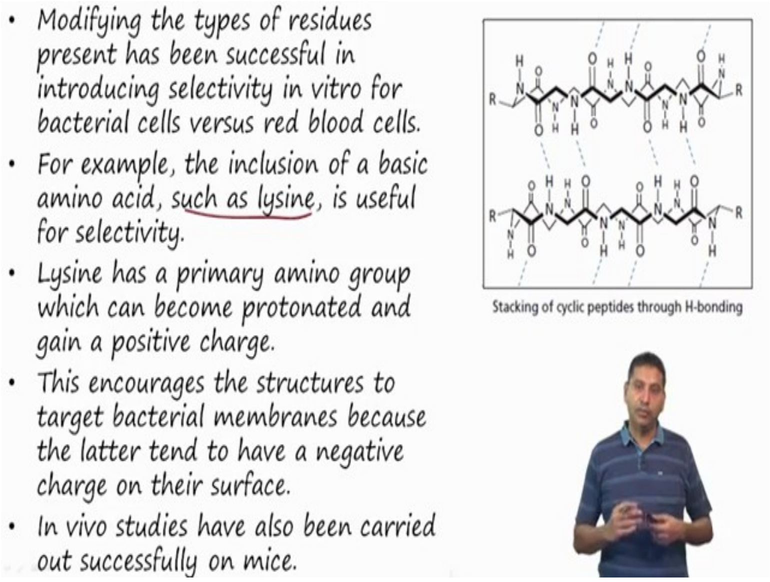

- Modifying the types of residues present has been successful in introducing selectivity in vitro for bacterial cells versus red blood cells.
- For example, the inclusion of a basic amino acid, such as lysine, is useful for selectivity.
- Lysine has a primary amino group which can become protonated and gain a positive charge.
- This encourages the structures to target bacterial membranes because the latter tend to have a negative charge on their surface.
- In vivo studies have also been carried out successfully on mice.

Stacking of cyclic peptides through H-bonding

So what it allows us to do is if we can modify the side chains then we could think about imparting selectivity in vitro for bacterial cells versus RBC’s. So otherwise what happens is that the cyclic peptides are known to to be toxic to red blood cells as well and they can cause what is known as haemolysis. So for example you can include the basic amino acid lysine which can be useful for such selectivity.

Lysine has a primary amino group which we have already looked at earlier and this can allow for protonation and once it gains a positive charge it can make it ionic. So this entire process enables the structures to target bacterial membranes because bacterial membranes tend to have a negative charge on the surface and a number of animal studies have been carried out and these molecules have been found to be having very promising activity and further work needs to be done on this at this point but they become good starting points for further drug discovery.

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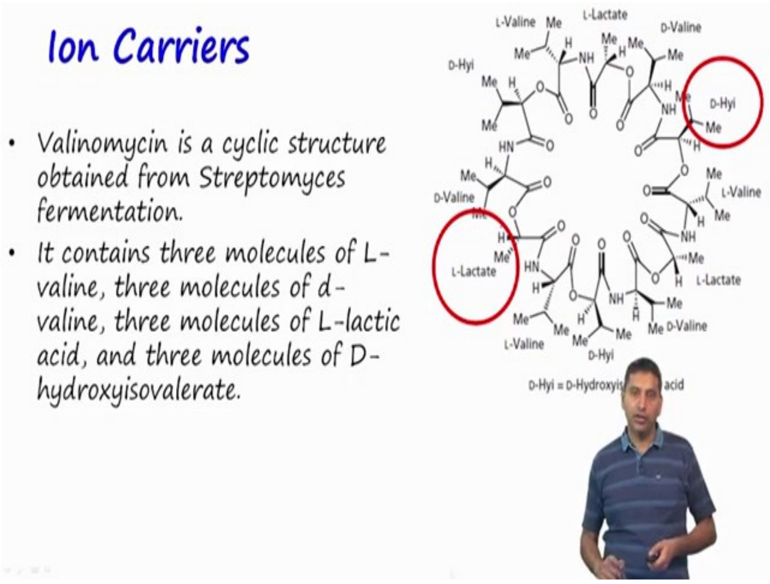

Ion Carriers

- Valinomycin is a cyclic structure obtained from Streptomyces fermentation.
- It contains three molecules of L-valine, three molecules of d-valine, three molecules of L-lactic acid, and three molecules of D-hydroxyisovalerate.

The next class of molecules are known as ion carriers, so the example that we are going to look at is Valinomycin. So Valinomycin is again a cyclic structure as shown here and it contains three molecules of L-valine and three molecules of D-valine and it also contains lactic acid and hydroxyisovalerate. So this is the structure of lactic acid lactate as shown here and this is the structure of hydroxyisovalerate, so together this forms a cyclic structure.

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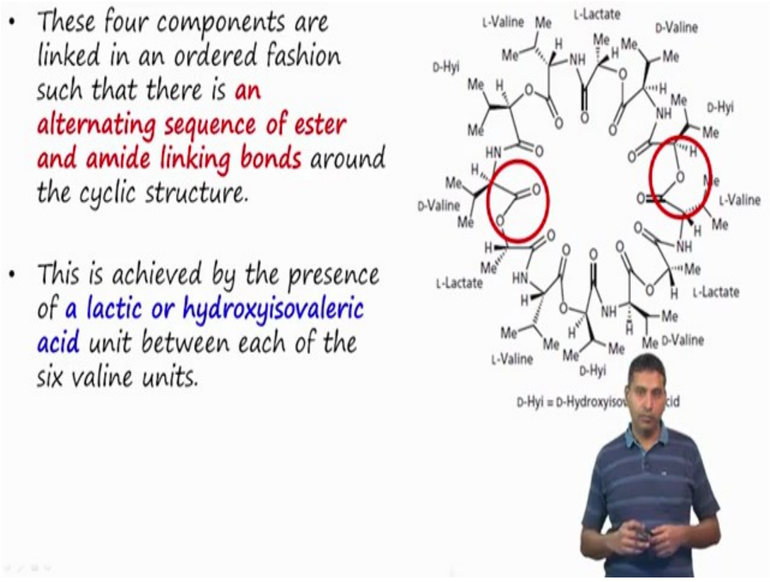

- These four components are linked in an ordered fashion such that there is an alternating sequence of ester and amide linking bonds around the cyclic structure.
- This is achieved by the presence of a lactic or hydroxyisovaleric acid unit between each of the six valine units.

And these four components are there is an alternating sequence of ester and amide linkages around the cyclic structure (so this can) this is achieved because we have an important molecule such as lactic acid or hydroxyisovaleric acid between the sub units of each of the six valine units. So as shown here, so you can see here the ester linkages which are present between the neighbouring amide units.

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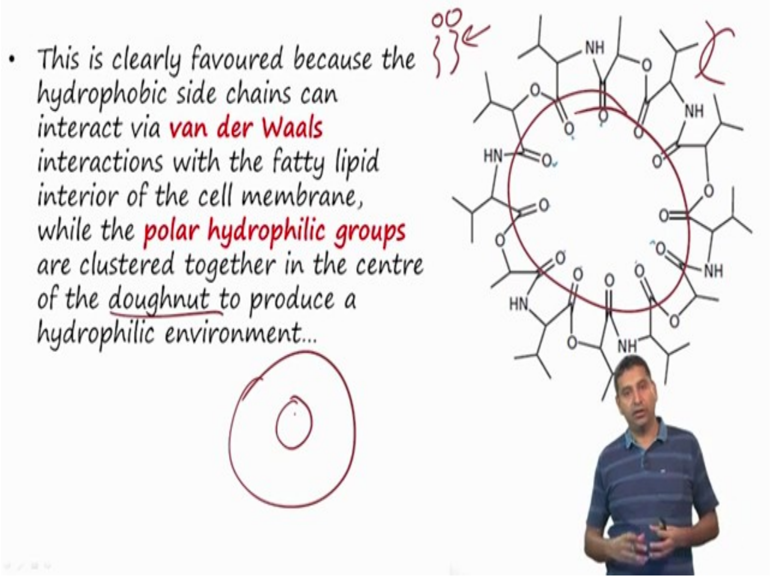

- This is clearly favoured because the hydrophobic side chains can interact via **van der Waals** interactions with the fatty lipid interior of the cell membrane, while the **polar hydrophilic groups** are clustered together in the centre of the doughnut to produce a hydrophilic environment...

So what happens is that this is actually clearly this structure is favoured because the hydrophobic chains can interact via Van der Waals interactions, so you have here this hydrophobic chains which can interact with each other and these can actually interact with the fatty lipid interior of the cell membrane. So we have already looked at, so the cell membrane has the inner part of the cell membrane actually is hydrophobic, whereas the hydrophilic groups are clustered together in the centre as shown here.

So what it can do is that it provides hydrophilic environment inside the molecule, whereas the hydrophobic environment is outside and this allows for the structure to actually go and bind to lipids and this resembles what is known as a doughnut, so doughnut is basically a food item which has a structure such as this, so where there is a whole in the centre and it has a sweet exterior.

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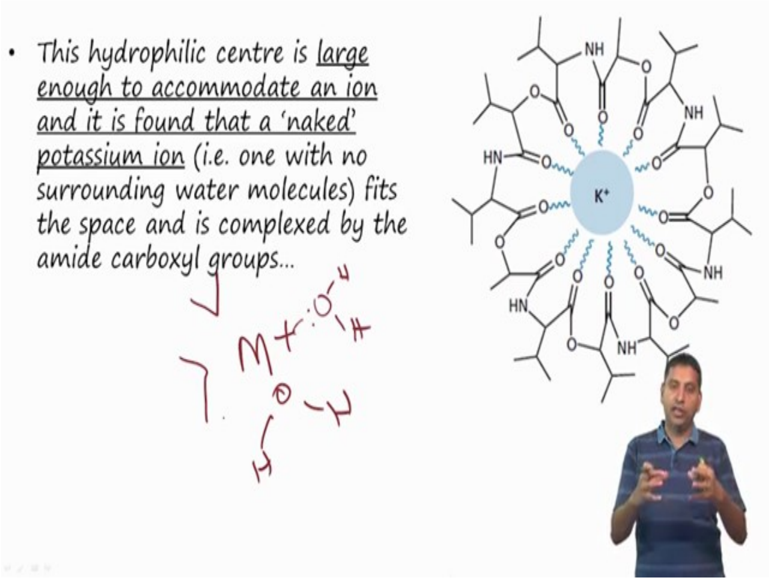

- This hydrophilic centre is large enough to accommodate an ion and it is found that a 'naked' potassium ion (i.e. one with no surrounding water molecules) fits the space and is complexed by the amide carboxyl groups...

So the hydrophilic centre is large enough to accommodate an ion and it is found that a naked potassium ion can actually pass through this. So what I mean by naked potassium ion is that it does not have water molecules around it, as we know any ion that is present inside the cell or inside solution is going to have let us say M plus it is going to have water molecules around it. So this is called you have interactions over here and you have a number of water molecules surrounding the molecule.

So this is actually called hydration and hydration is important because it stabilises the positive charge on the species. However, if the metal ion is actually very well hydrated, then what it does is that it effectively increases the size of the metal ion and because of hydration we have to deal with what is known as a hydrated ionic radius. So hydrated ionic radius is nothing but the radius of which is or the size of the molecule or like we assume that it is a sphere and this sphere which contains the metal ion as well as the water molecules and together they behave like a single unit, okay.

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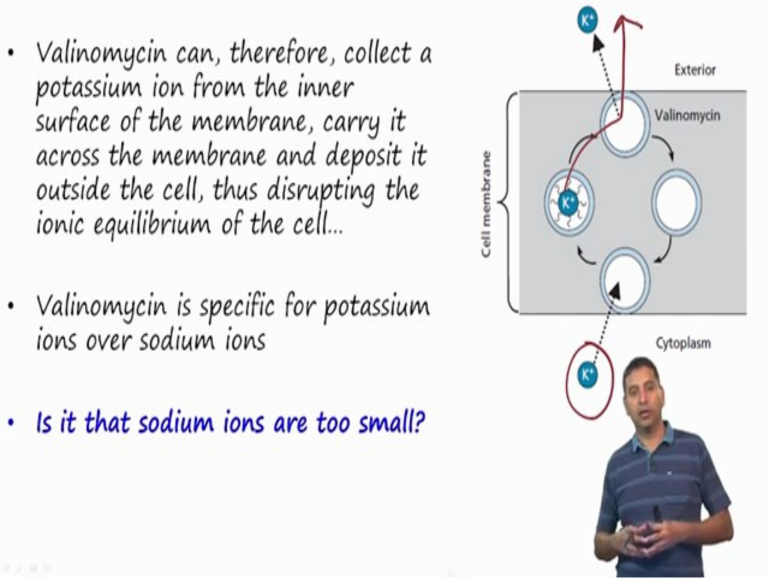

- Valinomycin can, therefore, collect a potassium ion from the inner surface of the membrane, carry it across the membrane and deposit it outside the cell, thus disrupting the ionic equilibrium of the cell...
- Valinomycin is specific for potassium ions over sodium ions
- Is it that sodium ions are too small?

So Valinomycin actually collects potassium ion, not a hydrated potassium ion but a naked potassium ion from the inner surface of the membrane and then it carries it across the membrane and takes it to the outside part. So this is the mechanism, so potassium ion becomes associated with the Valinomycin and Valinomycin as we know has a very hydrophobic exterior and then once it binds then it goes in and then the potassium ion is delivered outside the cell and then Valinomycin can do this again.

So we just looked at example of potassium ion and we have data to show that Valinomycin is actually specific for potassium ions and it does not work with sodium ions. Is it that sodium ions are too small? We already know that sodium ions and potassium you know sodium has a smaller radius compared to potassium ion.

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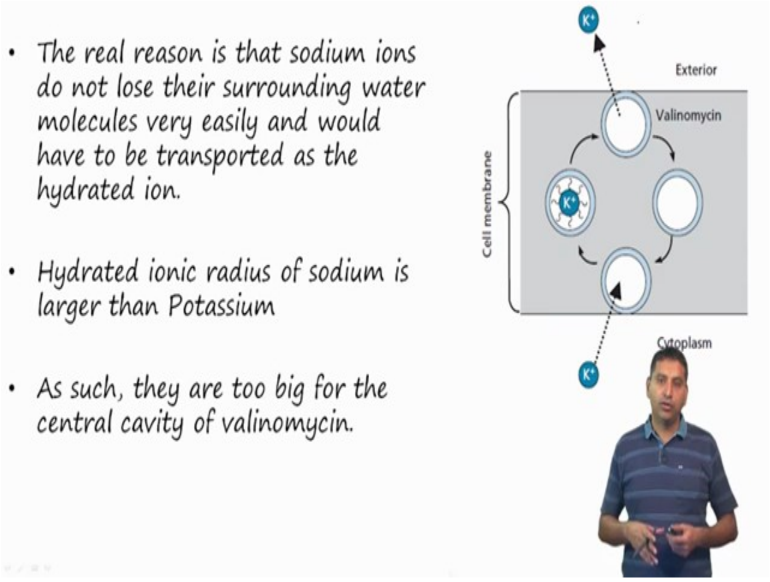

- The real reason is that sodium ions do not lose their surrounding water molecules very easily and would have to be transported as the hydrated ion.
- Hydrated ionic radius of sodium is larger than Potassium
- As such, they are too big for the central cavity of valinomycin.

But what happens is that sodium ion actually has a larger hydrated ionic radius compared to potassium. So as such sodium ion is too big the hydrated sodium ion is too big for the central cavity of Valinomycin (so) and sodium ions do not lose their surrounding water as easily as potassium ion. So potassium ion being larger has a (larger) greater tendency to lose the water molecules and become naked, whereas sodium ion does not do this. So together this imparts selectivity towards potassium ions over sodium ions.

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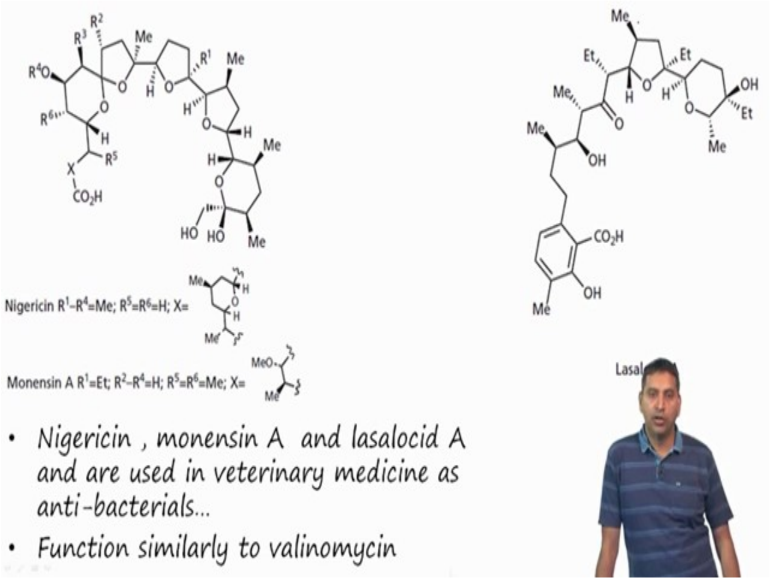

- Nigericin , monensin A and lasalocid A and are used in veterinary medicine as anti-bacterials...
- Function similarly to valinomycin

The next set of molecules which we are going to look at are Nigericin and related molecules whose structure is shown here and these have been used in animal medicine that is veterinary medicine as anti-bacterials and they functions quite similarly to Valinomycin.

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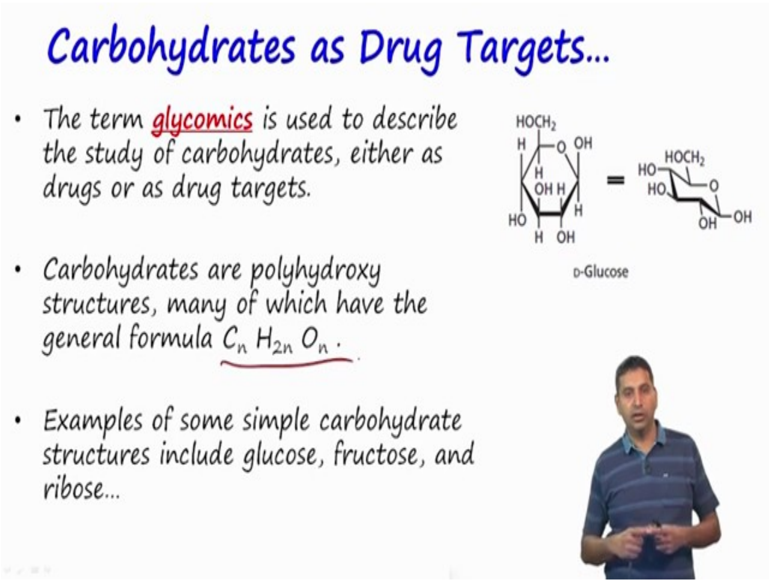

Carbohydrates as Drug Targets...

- The term glycomics is used to describe the study of carbohydrates, either as drugs or as drug targets.
- Carbohydrates are polyhydroxy structures, many of which have the general formula $C_n H_{2n} O_n$.
- Examples of some simple carbohydrate structures include glucose, fructose, and ribose...

So we have looked at again proteins, we have looked at nucleic acids and we just covered the topics of lipids. The next major class of molecules that are present inside the cell are carbohydrates. So the term glycomics is used to describe the study of carbohydrates and these studies are carried out using carbohydrates as drugs or drug targets and just to recap carbohydrates are polyhydroxy structures which have the general formula $C_nH_{2n}O_n$ and these are simple examples of simple carbohydrates include glucose, fructose, ribose and so on and we have already looked at ribose as an important component of nucleic acid and glucose is an important from the energetic stand point.

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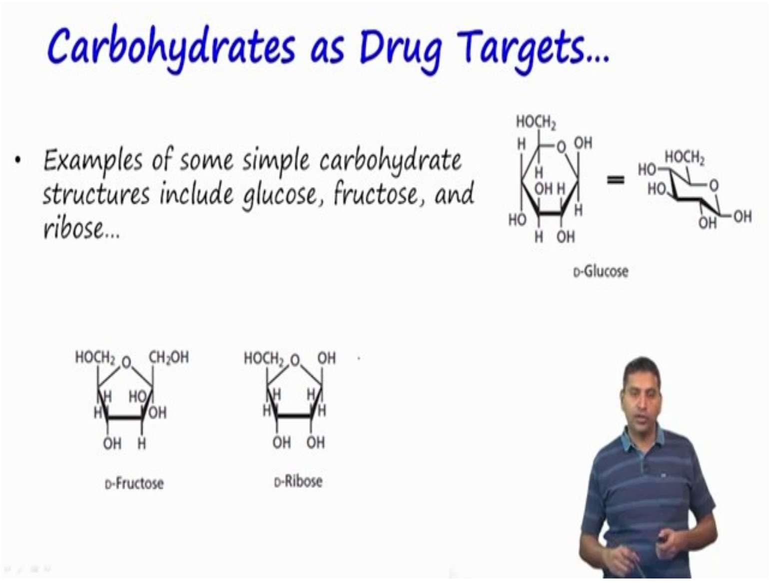

Carbohydrates as Drug Targets...

- Examples of some simple carbohydrate structures include glucose, fructose, and ribose...

Now the structure of fructose and ribose is shown here.

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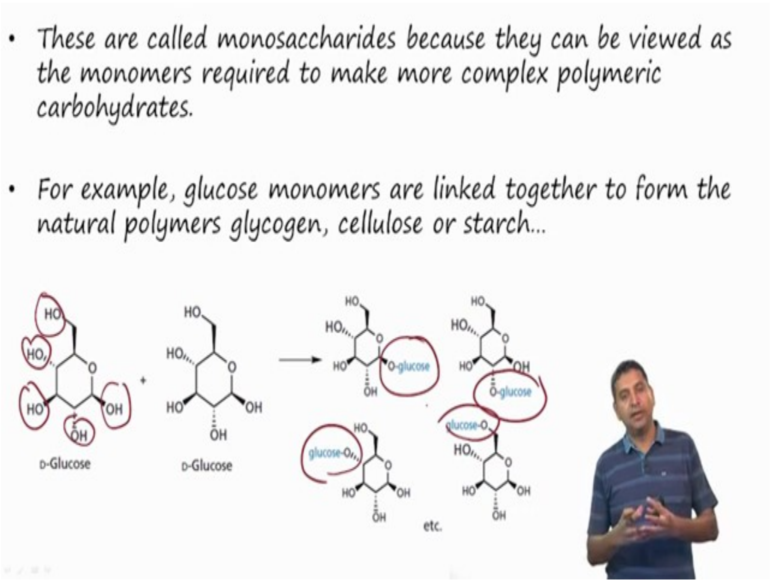

- These are called monosaccharides because they can be viewed as the monomers required to make more complex polymeric carbohydrates.
- For example, glucose monomers are linked together to form the natural polymers glycogen, cellulose or starch...

Now these are known as monosaccharides and these are essentially monomers and these can combine and form oligomers or polymers and because sugars have multiple hydroxyl groups on (the) it the combination can be fairly complex. So an example here is with glucose you have multiple hydroxyl groups which can interact with the next molecule and so you can have a O glucose combining 2 glucose molecules combining through one of these hydroxyl groups and gives you one structure and you can see here another combination here third combination, fourth combination and so on.

So if these molecules can combine to form incredibly complex structures and there are a number of natural polymers such as glycogen, cellulose or starch which are basically combinations of the sugars.

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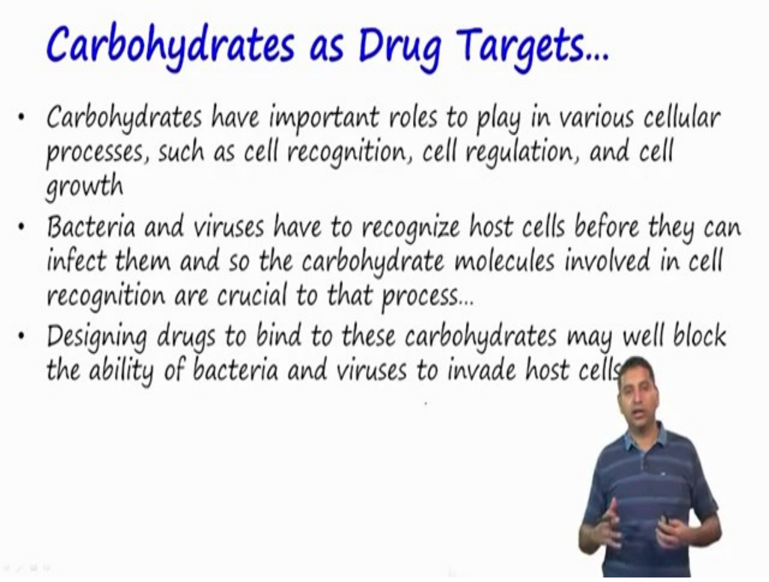

Carbohydrates as Drug Targets...

- Carbohydrates have important roles to play in various cellular processes, such as cell recognition, cell regulation, and cell growth
- Bacteria and viruses have to recognize host cells before they can infect them and so the carbohydrate molecules involved in cell recognition are crucial to that process...
- Designing drugs to bind to these carbohydrates may well block the ability of bacteria and viruses to invade host cells

Now for carbohydrates to be considered as drug targets the reason why they are interesting is because they have important roles to play in various cellular processes, very key role that carbohydrates play is in cell recognition. So the surface of the cell has a number of sugars which enable recognition of like cells. For example humans have present certain sugars on the surface which are which help it help the human cells to recognize itself and it also helps in understanding what are the sort of pathogenic cells there and get rid of it. So they are very crucial for cell recognition also for cell regulation and cell growth.

Now bacteria and viruses have to recognize host cells before they can infect them and these carbohydrate molecules are involved in the cell recognition process. So designing drugs that bind to these carbohydrates the hypothesis is that they may block these interactions of bacteria and viruses and prevent invasion of host cells. So therefore carbohydrates are important drug targets.

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Antigens and Antibodies

- The molecular tags that act as cell recognition molecules commonly act as antigens if that cell is introduced into a different individual.
- They identify that cell as being foreign.
- Bacteria have their own cell recognition molecules which are different from our own.

When we suffer a bacterial infection, the immune system recognizes foreign molecular tags and produces antibodies which bind to them and trigger an immune response aimed at destroying the invader.

Lastly we are going to look at antigens and antibodies. So we are not going to spend the whole lot of time on this but these are basically tags that help with cell recognition and antigens and antibodies help us understand if a cell is the same or it is a foreign cell. So bacteria have their own cell recognition molecules which are different from our own. So we need we have a very complicated set of antigen, antibody system which can help with immune response.

So we are not like I said we are not going to spend a huge amount of time on this but an immune response is triggered when there is a recognition that there is a foreign body that is or an invader that has come and once this immune response is activated it destroys the invading species.

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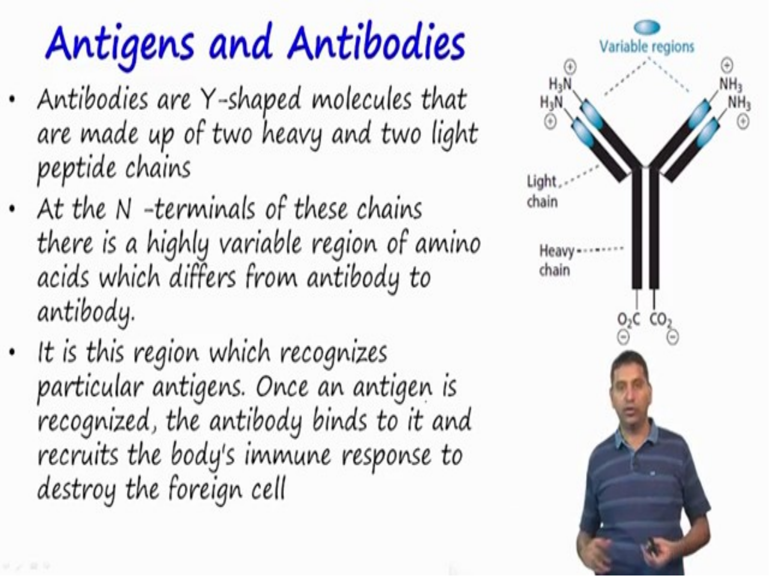

Antigens and Antibodies

- Antibodies are Y-shaped molecules that are made up of two heavy and two light peptide chains
- At the N -terminals of these chains there is a highly variable region of amino acids which differs from antibody to antibody.
- It is this region which recognizes particular antigens. Once an antigen is recognized, the antibody binds to it and recruits the body's immune response to destroy the foreign cell

Antibodies are Y-shaped molecules that are made up of two heavy and light chains, okay. So here is a structure of that and the N terminus of these chains has a highly variable region of amino acids and these can differ from antibody to antibody. It is this region which recognizes the antigen, okay. Once an antigen is recognized the antibody binds to it and recruits the body's immune response to destroy the foreign cell.

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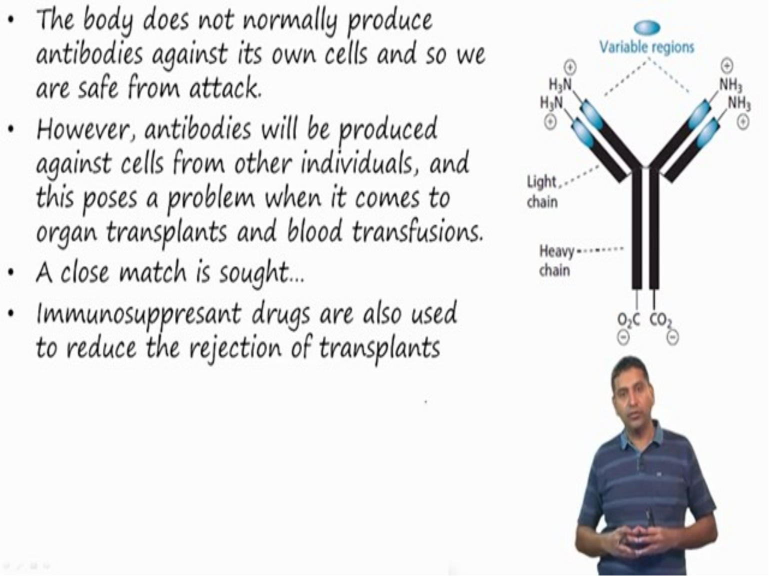

- The body does not normally produce antibodies against its own cells and so we are safe from attack.
- However, antibodies will be produced against cells from other individuals, and this poses a problem when it comes to organ transplants and blood transfusions.
- A close match is sought...
- Immunosuppresant drugs are also used to reduce the rejection of transplants

So the body does not normally produce antibodies against its own cells, so we are typically safe from attack. However, when there is an invading cell such as a bacterium or a virus then these antibodies will be produced and these are going to help with getting rid of this of the foreigner. Now there are also cases where people have organ transplants, where very an organ from one individual is transplanted to the other individual.

Now since the receiver of this transplant is going to recognize this new organ as something that is different from itself there could be rejection of the transplant. So what one way to address this problem is to use a close match (which are) which is basically genetically very close so that the recognition may be similar the recognition is not a major problem. However, immunosuppressant drugs are also used and these reduce the rates of rejection of transplants.

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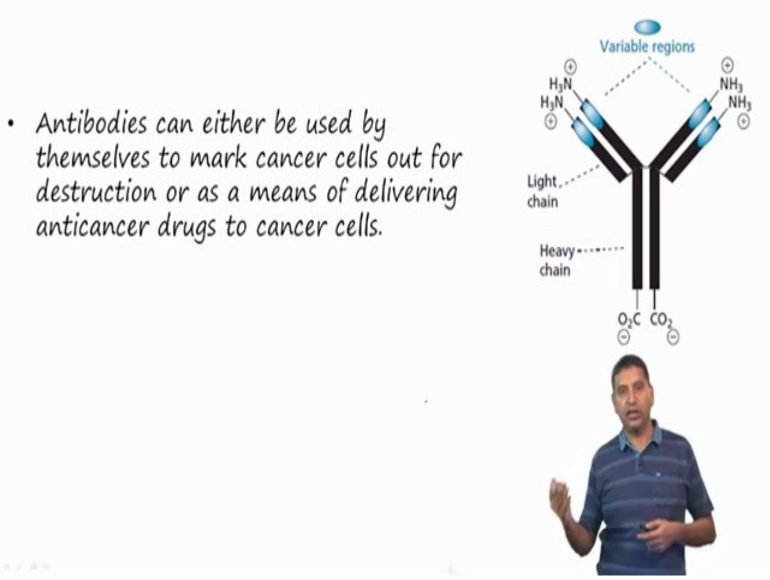

- Antibodies can either be used by themselves to mark cancer cells out for destruction or as a means of delivering anticancer drugs to cancer cells.

Antibodies can be either used by themselves to mark cancer cells or as a means of delivering anticancer drugs. So the new field of antibody drug conjugates has emerged in the past decade or so, wherein the antibodies are used because of their highly specific nature they are used to mark cancer cells and along with this you can deliver a drug and that will help in highly selective killing of cancers.