Introduction to Biomolecules
Prof. K. Subramaniam
Department of Biotechnology
International Institute of Technology-Madras

Lecture-27
Catabolism of Amino Acids

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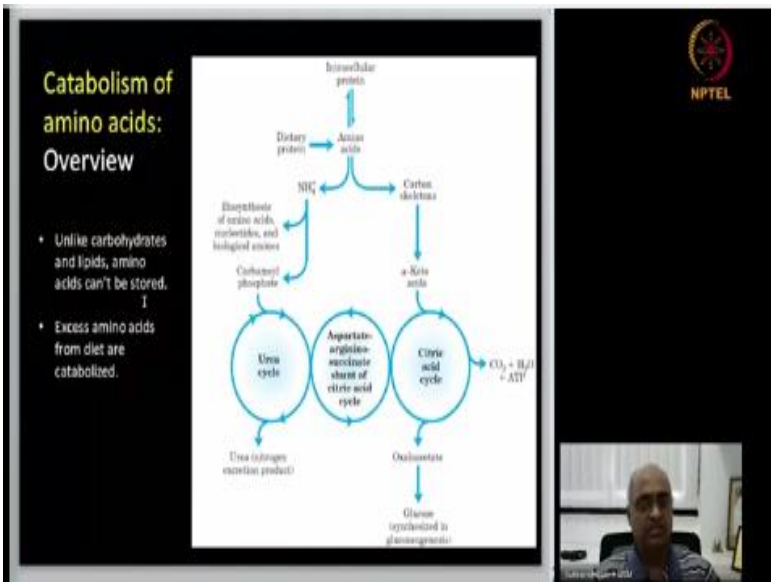

So, in the last class we discussed about how nitrogen enters into the biological system via the action of nitrogenous complex in the bacterium in root nodules**.** So, that is the main entry for bulk of the nitrogen. So, there are other free living bacteria which also do nitrogen fixation. So, then we also saw the primary root within the biological system is formation of glutamine**.** And then alpha-ketoglutarate through transamination becomes glutamate.

So, the glutamine and glutamate these two amino acids are the key entry points, and as a result their level is significantly higher compared to other amino acids. So, today what we are going to do is, in the interest of time and given that it is an introductory class, we are not going to go into the complete details of amino acid biosynthesis and amino acid degradation**.** So, we will not have back going by the previous couple of semester's experience I know that we will not be able to do that.

But you will get a general idea in any case from carbohydrate chemistry itself. So, here what we are actually going to focus is not about what happens to the carbohydrate skeleton part of the amino acids. That is the rest of the amino acid minus the amino group. So, here our focus is going to be what actually happens the amino group itself**,** catabolism of amino group is what we are going to focus.

So, before we get into that there are couple of important points here that is shown in the bullet on the left. So, unlike carbohydrates and lipids, amino acids cannot be stored**,** so there are no free amino acid storage like the way we have carbohydrate storage or lipid storage. And free amino acids in the cytoplasm will also perturbed the osmotic balance as well the excess amino acid means then you will get a lot of water getting into the cell, and the cell will swell up.

So, as a result the catabolism of amino acids need to be tightly regulated and any excess amino acid is promptly the deprived of the amino group and the rest of the carbon skeleton is recycled through carbohydrate and lipid metabolism. And the amino group gets excreted as urea**.** So, this amino group to urea is what today we are going to focus. So, the main point is that amino acids cannot be stored and they need to be right away catabolized.

So, if you have a protein rich diet the amino acids from it will be used to meet the requirements of your body and any excess gets excreted. So, it is not a good idea to have a diet that is exceptionally enriched for proteins**.** So, protein deficiency in general in Indian diet makes people to think that you have to have a lot of protein. But at the same time you cannot go to the other extreme it does not cause harm, it is just that it gets thrown away.

It does cause harm to some extent when you have excess amino acids to neutralize the acidic versions of the products from amino acid catabolism. Calcium gets taken away from the bone and as a result you get bone related problems. So, calcium intake has to be very high if your protein intake is very high. So, let us look at and what happens to the amino group in amino acids.

So, this summarizes the multiple roots of entry and exit. So, you have intracellular proteins that through normal wear and tear and when the time is up they get broken down into hydrolyzed into amino acids. And from the dietary protein also in the digestive tract the way which we will see in the next slide; get hydrolyzed into free amino acids. So, from these amino acids ammonia is removed, this is done in the liver, so this is what we are goanna focus today.

And the carbon skeleton are essentially keto acids, then they are recycled through the citric acid cycle. For example pyruvate is a keto acid produced by removing amino group from alanine. And oxaloacetate in citric acid cycle again is by removing the amino group from aspartic acid. So, that is how they get into the citric acid cycle and get oxidized. And excess of that the oxaloacetate will be used by gluconeogenesis pathways that we discussed earlier to make glucose**.**

So, ultimately if your food, diet is deprived of or deficient in carbohydrates and lipids, then energy will be derived from amino acids amino acids after all are a significantly reduced state of carbon compared to carbon dioxide. So, there is free energy available to be extracted from. And that does not happen directly from amino acids, they are used to make glucose**.** So, and this glucose is what brain is going to depend on.

So, this is what happens to the carbon skeleton. Then the amino group ammonia essentially liberated from amino acids is recycled by incorporation into purine, pyrimidine synthesis and other biological amines which we are not going to learn there are set of molecules called polyamines. So, their biosynthesis and amino acid biosynthesis it gets recycled and really excess more than what is needed is combined with the carbonic acid to form carbamoyl phosphate.

And this carbamoyl phosphate via urea cycle is converted into urea and this urea is excreted in the urine. So, this is what happens in our body, not all animals excrete ammonia only in the form of urea. For example animals in the ocean like marine animals they are in large excess of water; their release of free ammonia is not going to be a major issue, so they even excrete ammonia. Some excrete as uric acid, like birds do that and mammals excrete in the form of urea and the mammals ammonia catabolism is what has been well studied.

So, therefore our focus is going to be on this urea cycle. And as you can see here through a aspartate-argininosuccinate shunt, these 2 cycles are actually combined. Since both were discovered by Hans Krebs, this combination is often called the Krebs bicycle**.** So, he actually elucidated urea cycle before citric acid cycle, although he got the Noble price for the citric acid cycle. So, let us follow the nitrogen now**.**

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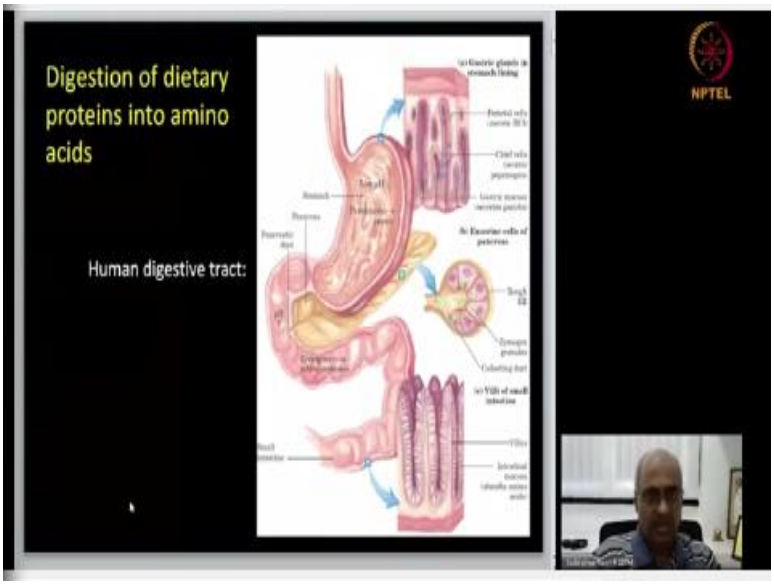

So, this is our digestive tract focusing only starting from stomach and downstream. So, the dietary amine protein that comes to the stomach stimulates the production of these hormones, gas stream which stimulates these cells, parietal cells and sieve cells**.** They are on the same stomach lining**,** so like small intestine here again you have folded structure to generate larger surface area.

And these glandular cells produce important things for protein digestion, when the parietal cells produce HCl and the sieve cells produce pepsinogen, so pepsinogen is an inactive form of protease called pepsin**.** So, I will just blow it up little bit, so that it is easier to see. So, this again we saw when we were learning about enzymes and enzyme regulation, yesterday for example we not yesterday, day before yesterday we recollected protein modification.

How covalent modification act an activate or inactivate enzymes? We saw how aminotransferases get uridylated or how aminotransferases also regulate the nitrogenase activity

itself. So, we saw covalent modification and then we have earlier seen phosphorylation as a covalent modification, how that regulates enzyme? We saw allosteric enzymes and we also saw proteolytic cleavage activating enzyme activity and one example is pepsin.

So, pepsin is produced as an inactive pepsinogen and from that a few amino acids are cleaved off to activate or to produce the active version pepsin, active means pepsin is a protease and as a protease it is going to be active. So, one of the reasons why it is produced in an inactive form similarly you are going to encounter more proteases when we go ahead and discuss about pancreas little down here, the blue arrow.

So, many of these digestive proteases are not produced in the active version. The reason is if they are produced in active version, they will digest those producing glandular cells themselves. So, this is one way of making them in an inactive form and sending in to the right place and activate it when they have to work on the digestive proteins. And this low pH ensures 2things, one it kills any pathogenic bacteria that may come from the food and second it denatures the proteins**.**

So, when you reduce pH or increase pH the 3 dimensional structure of proteins will be lost due to charged differences like protonation of many of the side chains that may be otherwise carrying negative charge. And it will disrupt hydrogen bonding as well, so due to that proteins get denatured and become better suited for digestion by pepsin, so this is what happens in the stomach.

And as the partially digested proteins enter into the small intestine, so that low pH stimulates pancreas to produce digestive enzymes**.** Pancreas has 2 parts, for example the islets of Langerhan, they produced the hormones glucagon, insulin and somatostatin and they are endocrine part of the pancreas and therefore pancreas is an endocrine plant also. At the same time the exocrine cells of pancreas shown here, they produce a multiple proteases, the main ones are trypsin chymotrypsin, carboxy peptidase, A carboxy peptidase, B these proteases meaning, protein digesting or protein hydrolyzing or peptide bond cleaving enzymes.

So, these enzymes are produced like pepsinogen in an inactive version, these inactive versions of proteins where the inactivity is due to presence of a few more amino acids, such structures are called zymogen**.** And zymo is a generic word referring to an enzyme, like the way glyco referring to any carbohydrate while gluco would refer only to glucose. So, here pepsinogen that it refers only to pepsin's inactive version.

Zymogen is a generic word to any such enzyme. So, it is produced as zymogen granules and they are secreted via the collecting a duct into the intestine. And here then the pH increases due to the intestinal duct secretion of bicarbonate, the carbonic acid buffer that we have. And that brings the pH to neutral level. And there these zymogens become proteases primarily through autocatalytic activity of trypsin.

So, trypsin becomes active then that cleaves the other zymogens into active proteases and then they digest the partially broken polypeptides. So, trypsin and chymotrypsin are endopeptidases, meaning they cleave in the middle somewhere in the polypeptide chain. They are specific to certain amino acids, they like at the carboxy terminus of the lysine**,** so that is where they cleave or originate.

While carboxy peptidase A and carboxy peptidase B are exoproteases. They start from the carboxy terminal end of peptides and hydrolyze the proteins into amino acids. So, the end of all these proteases activity is free amino acids**.** And these free amino acids produced by digestion in the small intestine are transported; see this is the microvilli of small intestine. In a very large surface area compacted into kind of a folded tube in your abdomen?

And here these intestinal mucosa cells, these linings, cells, these are the ones that absorb the amino acids. So, this is how the protein that you eat enters your body. As amino acids into intestinal mucosal cells and from there they are transported to the rest of the body, primarily first to liver. So, liver is actually the main biochemistry factory in our system. And that is why when you have liver infection you are not allowed to eat any complex food and the only food you are allowed to eat is pure and simple carbohydrate, no protein, no lipid.

Because they all have to be handled by the liver, while carbohydrate can be handled by all other tissues. So, that is why when you have jaundice, the doctor advises to eat only idli with sugar and nothing else, not even buttermilk or curd or bread or anything. And the reason is liver is where everything is metabolized, you cannot even take medicine, because the medicines are metabolized by the liver. And that is why serious liver disease normally becomes fatal, because it is difficult to treat**.** Now let us follow these amino acids.

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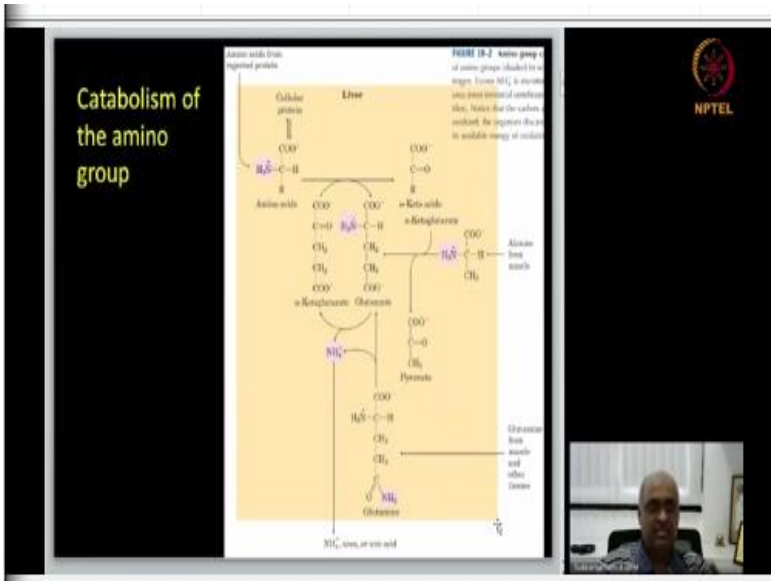

So, this is again a summary sort of slide what happens to the amino acids. So, the amino acids coming from the intestine, so here is a generic amino acid**,** so amino group, carboxyl group here you have the R group. And for cellular protein normal breakdown and synthesis also, you get the amino acids. So, these amino acids are combined with alpha-ketoglutarate in transamination reaction, so you follow this pink square.

Collecting the amino group various amino acids into glutamate form**.** And therefore each amino acid will become it is corresponding keto acid, after losing the amino group. And this keto acid alpha-ketoglutarate becomes glutamate, so this happens in liver**.** And from other tissues, it is primarily transported to liver in the form of glutamine**,** we will see this in a minute but you are already familiar with it.

When we were discussing about the nitrogen fixation in plants I told you about how the ammonia is combined with the glutamate to form glutamine. And glutamine with alpha-ketoglutarate produces 2 glutamate molecules and from there through transamination to other amino acids, that is what I told you. So, in that we discussed an enzyme called glutamine synthetase and then I said that exists in all animals**.**

So, that enzyme is responsible for collecting the amino group from various amino acids in the form of glutamine by the glutamine synthetase reaction that we already discussed. But we will look at the reaction once more in the next slide, and this glutamine through bloodstream come to liver**.** So, the amino group from variety of amino acids are collected in the form of the amide nitrogen in glutamine side chain.

And this glutamine through bloodstream comes to the liver. And here glutaminase is going to release ammonia and it is going to become glutamate. And this glutamate alpha-ketoglutarate this cycle like reaction gathers all the amino groups in the liver itself, primarily the amino acids metabolized in the liver coming from digestive tract for example. So, there also collected into glutamate and from glutamate to amino group is removed, regenerating the alpha-ketoglutarate.

So, in the liver itself you take the ammonia from glutamate and from the rest of the body glutamine comes to the liver and from there from glutamine ammonia is taken out. And this ammonia enters into urea cycle and gets excreted in urine as urea if (()) (21:51) uric acid. So, this is what happens to the nitrogen. And one exception is the muscle amino acids which we will see separately as a separate cycle**.**

Because it deserves any separate slide for itself, we will discuss that when we go there. So, next we need to learn some biochemistry that is taking us to our next B complex vitamin**.** So, remember we have learnt about the last B complex vitamin we learnt was biotin, which carries carbon dioxide**,** pyruvate carboxylase reaction for example. So, the next one we are going to see is a vitamin that plays an important role in these transamination reactions.

Where the keto group and the amino group of a keto acid and amino group amino acid exchanges, they swap this. Creating this keto acid into an amino acid and this amino acid into keto acid, this is called transamination reaction. And the enzymes are called aminotransferases or transaminases**.** And this is an important reaction, transamination particularly since we are not going to go into amino acid catabolism or amino acid biosynthesis a key concept like reaction in those kind of metabolism of amino acids is this transamination.

So, therefore we are going to have a close look at the mechanism of transamination, so that is going to come in the next couple of slides again, where are we? Here.

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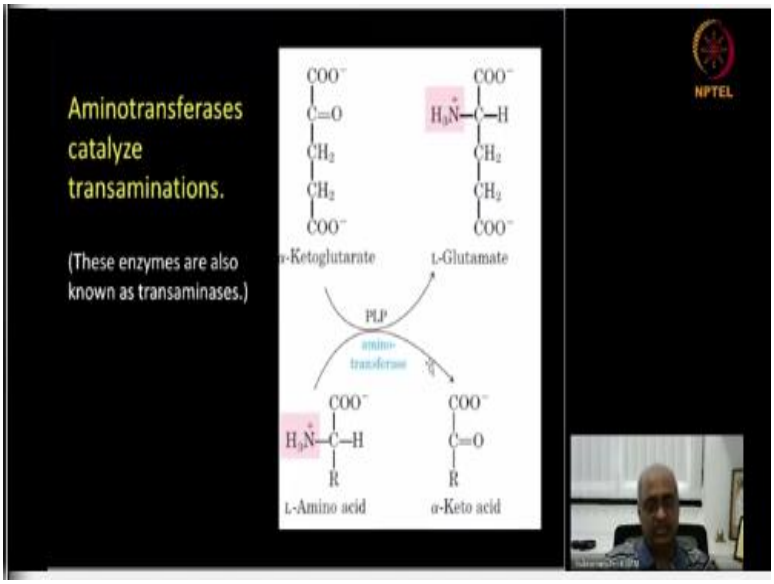

So aminotransferase, so here is an example**,** alpha-ketoglutarate, this is the same thing in the previous slide but it is just that it is shown big. So, you have this keto group and the amino group swapping to form amino acid and keto acid, this is called aminotransferases. So, they are also known as transaminations, because this reaction is called transamination**.** The crucial co-factor for these aminotransferases is pyridoxal phosphate. So, this is a B complex vitamin pyridoxine or pyridoxal phosphate.

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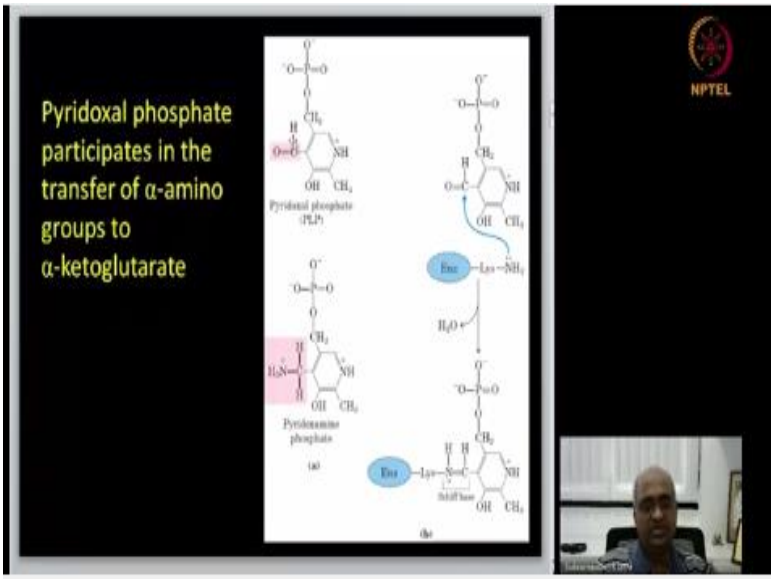

It exists in an amine form like this as well as in an aldehyde form. So, this is vitamin B6 really important reaction and a really important cofactor. So, here again you have this conjugated double bond in a ring structure which helps in delocalizing electrons temporarily stabilizing carbon ion kind of electron deficient atomic structure by delocalizing, essentially in very layman terms juggling the electrons for some time, stabilizing an electron deficient carbon.

So, such structures are called electron sinks, so we have already seen multiple examples and this is one more**.** So, it exists in this amine form pyridoxamine phosphate and aldehyde form pyridoxal phosphate, it swings between these 2 and that is what is critical for it is role as a cofactor. So, this is usually attached covalently by a aldimine**,** so this is an amino group lysine's epsilon.

The side chain amino group with this aldehyde structure forms aldimine double bond which is also known as Schiff's base**.** So, this is how it exists normally in the enzymes active structure and so that formation itself is shown here**.** So, this carbonyl carbon always important in biochemistry**,** so this partial negative charge and partial positive charge that makes it attractive for a nucleophilic attack by these sort of electrophiles and that is how you have the Schiff's base. And now let us look at the reaction itself.

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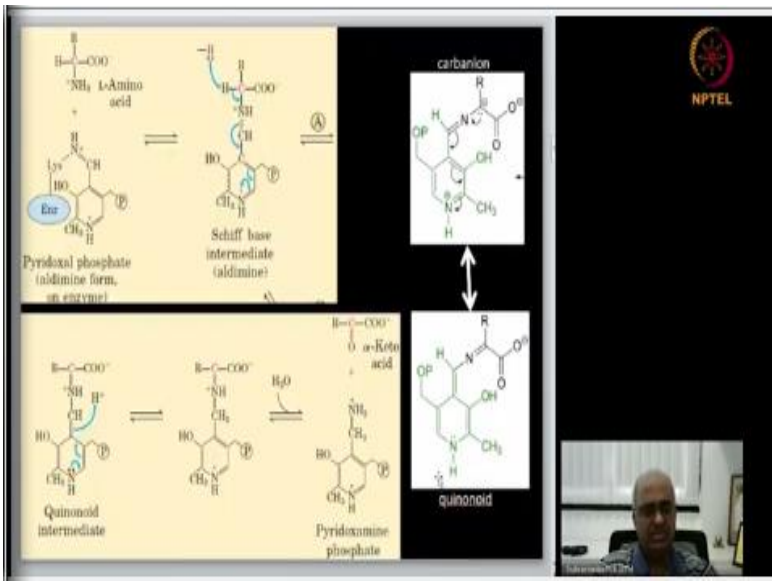

It is extremely simple mechanism, there is nothing complicated about it. The first step is this covalently bonded pyridoxal phosphate to the lysine's amino group swaps this Schiff's base with the amino group of an incoming amino acid into this structure. So, instead of this you have this attached in the same way, so that is what is this structure. And here a base group, base catalysis general base catalysis, remember general acid base catalysis that we have learnt and seen multiple examples so far.

So, abstracts are proton from this amino acid leading to this flow of electrons and making this nitrogen having an excess electron. And this leads to the double bond switching leads to a carbon ion structure here, because it lost one electron which is gained by this nitrogen**.** And this delocalization of electron within this ring structure can stabilize by alternating between these structures.

So, this is carbon ion and this is quinonoid structure, we saw this in ubiquinone also when we were in mitochondria oxidative phosphorylation. So, this keeps alternating and as a result it stabilizes this carbon ion structure. So, when it swings the alternates between these 2 structures, you pay attention to this double bond. So, when this electron is transferred then you get the double bond between this nitrogen and carbon here.

And in that quinonoid structure it can lose a proton and the electrons return in this direction leading to switching the double bond to single bond with the CH 2 formation**.** So, now this becomes labile for hydrolysis leading to the formation of the keto acid**.** So, this double bond is now exchanged with this oxygen and then you have this amino group generated. So, this is half of the reaction, all that we have done is we have taken this amino group on the pyridoxal phosphate in the form of pyridoxamine phosphate now and the keto acid is released.

So, remember if you remember the 2 substrate enzyme catalysis reactions, we had random ordered, sequential, double displacement or ping pong. So, this is an example of ping pong, where one substrate binds becomes one product, that product leaves. Now the group taken is on the enzyme, here it is on the enzyme's cofactor, then the second substrate binds and this group is transferred to that substrate and then that leaves.

So, S1, P1, P1 goes then S2, P2 then P2 goes, this kind of sequential 2 substrate binding and 2 product leaving is what we call double displacement or ping pong mechanism. So, this transamination is a perfect example of a ping pong mechanism. So, this is the role of pyridoxamine pyridoxal phosphate. Now the second half of the reaction will be exactly the reverse, a keto acid binds to the pyridoxamine phosphate version of the enzyme.

And it goes retraces the path and in the carbon ion form, it can switch to producing the amino acid version of the original keto acid that enter. So, therefore we are not going to have another slide telling the same thing in the reverse direction. So, I am going to just say that this is the reaction mechanism and the reverse is the rest of the reaction to complete the transamination process.

So, this is very important enzyme mechanisms you need to remember, it is as important as biotin or thiamine pyrophosphate in pyruvate dehydrogenase complex. So, I am not sure whether we are going to encounter any more vitamins, this is probably the last vitamin we are encountering. So, let us now move further from this transamination reaction, so this explained this reaction mechanism explained us, how the amino groups get collected on alpha-ketoglutarate into glutamate form**,** so just orienting you in the larger map.

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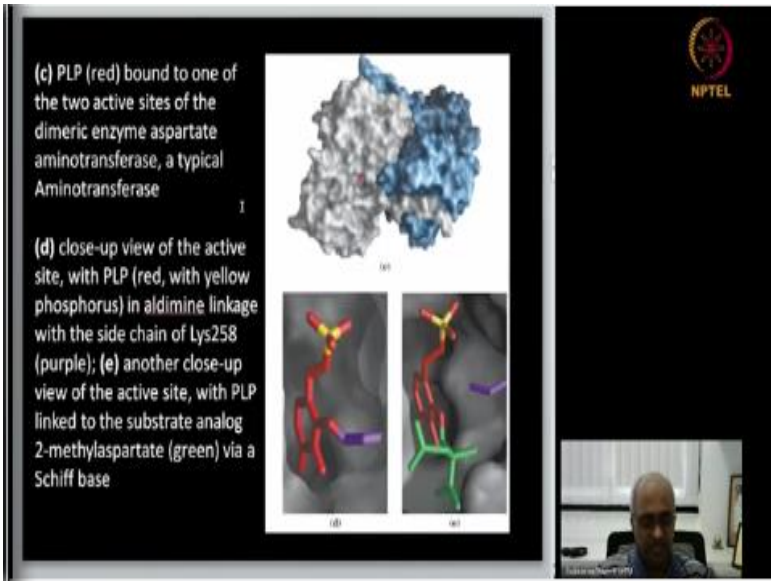

So, now let us get back to this path of the amino group but before that little bit closer look at appreciating beauty of this enzyme's active site. So, it is a dimer, so in one of them you see this tiny red thing that is this pyridoxal phosphate**.** So, this is a close-up view, this is the pyridoxal phosphate and so this yellow is the phosphorus to which you have these oxygens attached there. And this is the lysine side chain which is in the ship's base with the pyridoxal phosphate**.** So, this is another view of the same thing where you have the aspartic acid analog methyl aspartate is in Schiff's base with the pyridoxal phosphate.

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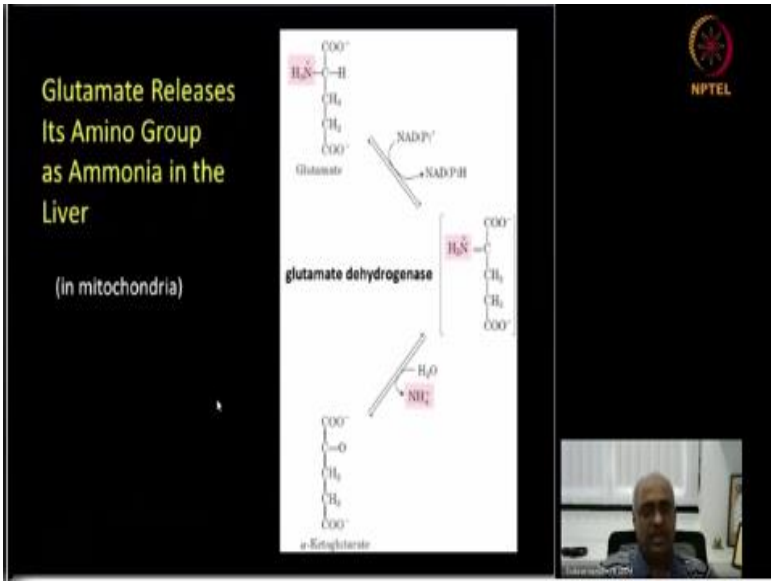

Now we already saw that the glutamate releases it is amino group as ammonia in the liver, regenerating alpha-ketoglutarate to go with that cycle. And that is catalyzed by glutamate dehydrogenase enzyme, so this is essentially an oxidation reaction. So, in the process NAD or NADP depending on what is available whose level is at the right optimum concentration, one of these oxidized versions gets reduced**.**

So, you have reducing equivalent because it is an oxidation reaction that is conserved here which could go to oxidative phosphorylation. So, remember this is actually happening in mitochondria of the liver. So, glutamate dehydrogenase therefore removes the amino group as ammonia from glutamate regenerating alpha-ketoglutarate. So, this could either go and participate in transamination reaction or it can go into TCA cycle depending on the flux through the metabolic network.

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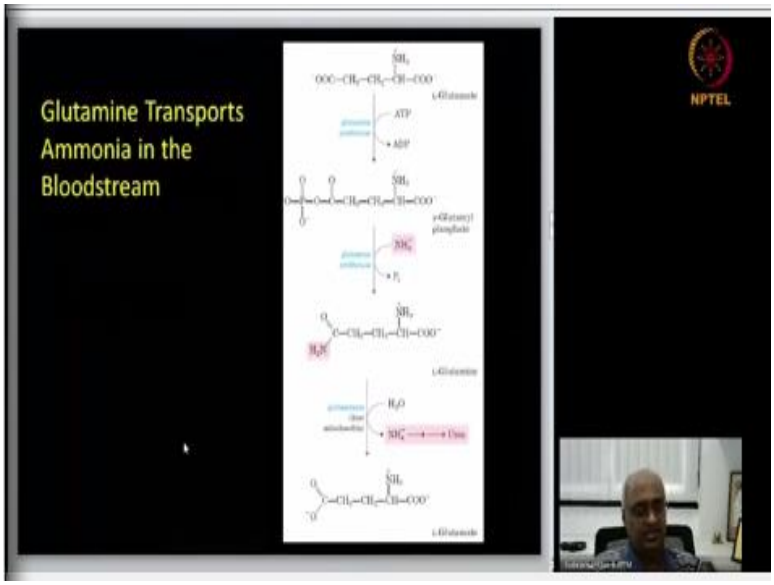

So, how from other tissues amino acid amino group is transferred to the liver I said it happens via the formation of glutamine, so we look at this. So, this we have already seen glutamine synthetase has this glutamyl phosphate intermediate. And then by combining with the ammonia liberated from other amino acids becomes glutamine. So, glutamine synthetase reaction at these 2 steps leads to the formation.

First activation of the reacting group by attaching a good leaving group and then you have glutamine. And this glutamine is transported via bloodstream into liver and in liver mitochondria glutaminase removes ammonia and forms glutamate**.** So, these are the 2 ways, this is one way from glutamine and the other one is from glutamate, these are the 2 amino acids from which ammonia group is removed in liver mitochondria**.** By 3 enzymes, here glutamate dehydrogenase, so here glutamine synthetase and glutaminase, you get the ammonia liberated.

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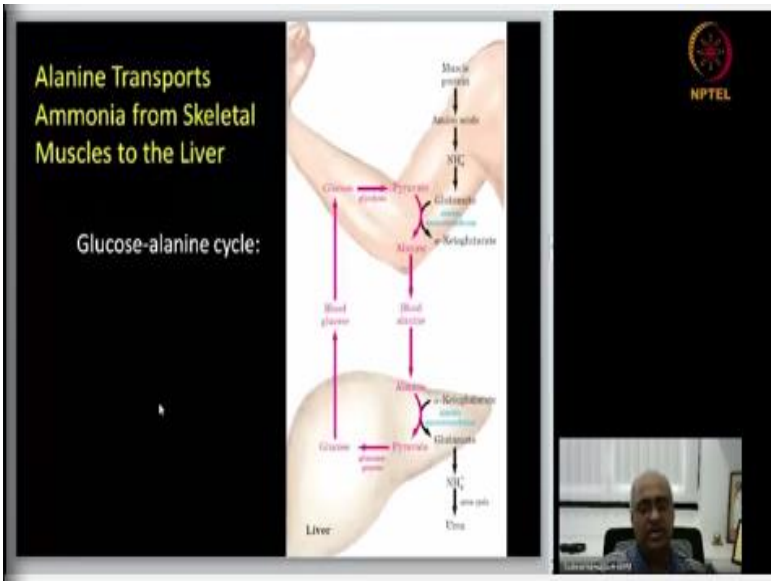

So, let us make the ammonia wait for some time, we will take a detour and consider another route by which ammonia is collected from amino acids to the liver. So, this is in our muscles**,** so in muscles it is slightly different. If you recall our discussion on glycolysis before the pyruvate dehydrogenase reaction, so, we learned under oxygen deficient conditions glycolysis can operate because glycolysis does not require oxygen.

You will get 2 ATPs at the end of it, starting from glucose, coming to pyruvate and then going into lactic acid. So, when you do that you end up getting 2 ATP molecules and that is what powers muscle cells when you are vigorously exercising. For example when you go for a short sprint, that time the oxygen transport will not be sufficient enough to generate the required energy in muscles.

So, muscles rapidly consume glucose via glycolysis generating ATP molecules. And we also remember that glyceraldehydes-3-phosphate dehydrogenase enzyme will consume NAD and reduce it to NADH. And that NAD needs to be replaced replenished for the glycolysis to operate continuously and that is done by the lactate dehydrogenase in the muscle, ending up converting pyruvate into lactate**.**

So, I am saying this primarily to focus on the pyruvate, so that one way of handling pyruvate is by converting into lactate, regenerating the NAD for the continuous operation of glucose to replenish the supply of oxidized NAD. And the other route is what are the focus now. So, this pyruvate via it is a keto acid CH 3 COOH and that keto group can participate in transamination reaction and when it does with the glutamate for example produces alanine.

And this alanine via bloodstream comes to the liver**.** So, from all other tissues it comes in the form of glutamine, from muscles it comes to the liver in the form of alanine. So, that is the primary focus in this slide to distinguish between the 2 modes of transport of the amino group. And this alanine here again through transamination reaction, the same in the reverse direction produces pyruvate and glutamate.

And from glutamate we just saw glutamate dehydrogenase will liberate ammonia. And this pyruvate as we have seen earlier undergoes gluconeogenesis to produce glucose in the liver. This glucose via blood goes back to the muscle, so that the muscle can continuously operate glycolysis**.** So, this is what we call as glucose alanine cycle, because glucose via pyruvate becomes alanine, then alanine becomes via pyruvate glucose.

So, this glucose alanine cycle helps continuous operation of glycolysis in the muscle and it also allows the muscle to get energy from amino acids. For example here glutamate is continuously getting consumed to produce alanine and this cycle operates**.** So, this is the glucose-alanine cycle and this is the operation of which transports the amino group from muscle to liver.

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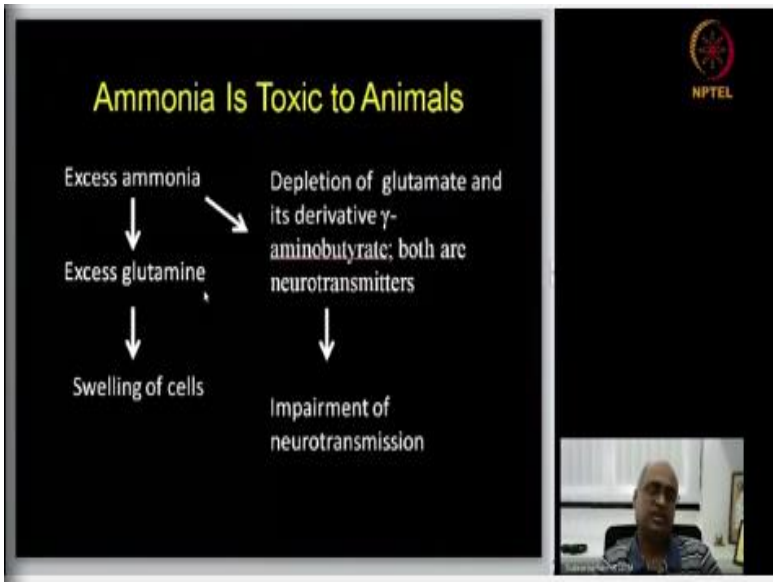

So, now what about ammonia? So, we made ammonia to wait for some time, now we catch up with ammonia. So, this excess ammonia is bad because it is toxic and excess ammonia availability via glutamine synthetase reaction will make excess glutamine. And glutaminase molecule there causing osmotic imbalance and excess glutamine means then water will flow from low concentration like low glutamine concentration to high glutamine concentration inside the cell, so the cell will swell up.

So, you do not want to have excess glutamine and to prevent that you need to get rid of ammonia. And similarly when you do this conversion you will use up glutamate and when you use the glutamate, then glutamate will not be available one, and second it will not be available for production of molecules for which glutamate is the precursor, one is this gamma aminobutyrate, these 2 are important neurotransmitters.

So, as a result neurotransmission will be impact and that will eventually lead to death**.** So, first you will get into coma and then it will lead to death. So, excess ammonia or inhaling lot of ammonia is really bad for health. So, the summary here is the ammonia produced in the liver from glutamate and glutamine must be excreted.

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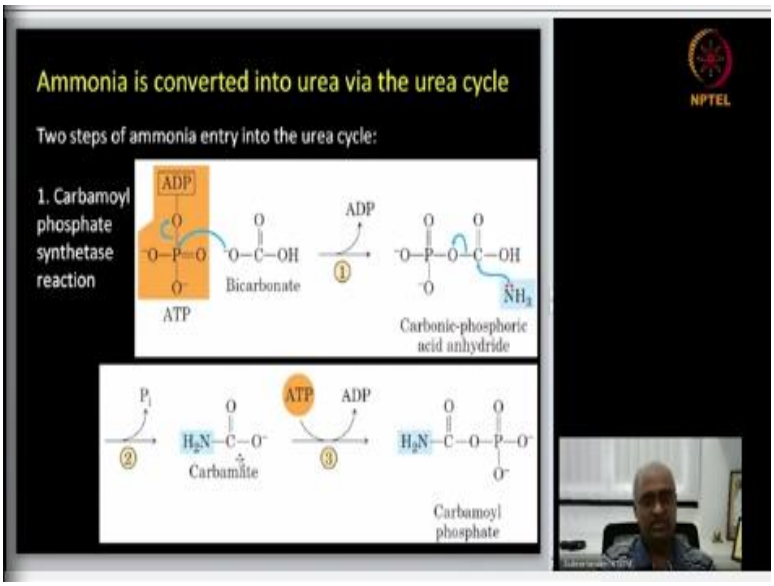

And that is done by urea cycle and I guess I do not want to do half of it or one reaction today, because we are going to get to 9:15 2 minutes. So, I will stop here, in the next class we will begin urea cycle fresh**.** So, if there are any questions feel free to ask, we have more than 3 minutes left, I guess there are is there any reason why amino acids are not stored in the body? I just told you one of them is, this very reason, amino acids are going to be osmolytes they will seriously cause osmotic imbalance.

So, that is why they are not stored and proteins are produced only to the extent the proteins are required. And most of if amino acid is not stored cannot be converted into proteins very, very quickly, regardless of where the instruction comes from, yes, you can convert amino acids into proteins. But proteins are again not storable form of molecules, they have activities and therefore their levels need to be strictly regulated as per requirement.

They are not bulk storing molecules like lipid or carbohydrates, proteins end up doing catalysis and proteins also cause osmotic issues. For example in our serum we have albumin and the albumin is present primarily to take care of the osmotic balance. So, protein again is not storable, is there any disease because of deficiency of an amino acids? See, there the reason to anything called amino acid deficiency; you have protein deficiency that is connected to amino acid deficiency**.**

So, if your diet is poor in amino acids, let us say you eat lot of corn, like every day you watch movie and every day for whatever reason you keep eating popcorn, I do not understand the connection between popcorn and movie. But anyways in cinema there does people eat always popcorns. So, popcorns do not have tryptophan and lysine and as a result you will have deficiency of 2 amino acids, meaning you cannot make proteins, out of the 20 amino acids even if 1 is not available where that amino acid is required the protein synthesis will stop there.

So, when you have diet like that you have problem of protein deficiency and this used to be a very common problem. It is still a major problem in very poor countries like India**,** where I told you for want of quarter carrot a day children go blind in some parts of India. Very similarly there are poor children who do not get enough proteins in their diet, like you think of lentils, like black gram, pigeon gram red gram, green gram all these lentils that we eat, either as dhal with your chapati or sambar with your rice.

This is a luxury it is a very expensive diet for very poor bulk of the Indians**.** And they usually have, you can see this in some of the poorly managed public eateries or where someone cuts corners, they will give you a very watery sambar or watery dhal; you can see in both sides of the country this phenomena. That is primarily because they do not want to use enough lentils. And if you do not have lentils in your food and if the rice that you eat is polished white, meaning the outer part of the rice is where you have really high quality rice albumin, readily digestible protein.

When that is removed out of rice and you eat very little lentil and if you are a vegetarian, then of course you suffer protein deficiency. And when you have protein deficiency, your serum will not have enough albumin. And when you do not have albumin, water accumulates in your abdomen and you get a pot belly. And your face swells into perfect round that is called moon face, this moon face, pot belly in younger developing children is the classical symptoms of malnutrition called kwashiorkor and marasmus. These 2 diseases can be treated by ramping up protein supply over a period of time, this is what world food program does in different parts of the world.