Overview and Integration of Cellular Metabolism

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Lecture 45: Metabolism of Histidine, Proline, Arginine and Lysine

Hello everyone, welcome back to your classes on Overview and Integration of Cellular Metabolism. We are continuing with amino acid metabolism, this is the last class of amino acid metabolism. We will be again we discussing amino acid metabolism, when we move to integration section and in border of metabolism. Today's topic is metabolism of histidine, proline, arginine and lysine, we have a lot to cover and these are the concepts that we will be discussing. Today we will be discussing the important product from each of these amino acid, we will be discussing the metabolism of each of these amino acid. For example, for arginine we will be discussing nitric oxide, for histidine we will be discussing histamine, we will be discussing how these all amino acids are connected, what is the interrelationship among them and their integrated metabolic fate.

So, let us start right way histidine alright. Histidine why it is special, if you look at the chemistry it has got an imidazole ring, this side chain this pentagon right 5 carbon chain which has got 2 nitrogen imidazole ring, it is a basic amino acid by its chemical nature and it is a same I told you the amino acids that are not fully required in diet, but their function can be essential when there is growth development right. So, histidine falls in that category alright. So, it is a semi essential basic amino acid right.

Now, one thing that histidine is known for is its buffering action, this will not be discussed in this class is a concept of acids and bases you all need to know what is the pKa that is the dissociation constant of the amino acid, how they dissociate in a acidic or basic medium. The pKa of histidine is actually 6.1 which imparts to its maximum buffering action because it is very near to the physiological pH and histidine as it is present in hemoglobin will be seeing when we discuss in synthesis in the next class, it helps in this physiological activity right. So, these are the few things about histidine the chemistry. So, regarding the metabolism of histidine what we need to know is how histidine is degraded mainly right.

So, histidine by the action of histidase is converted to urocanate or in some cases it is

also known as urocanic acid which upon action by urocanase enzyme is converted to 4 imidazolone 5 propionate right which undergoes a hydrolase enzyme action right. Hydrolase what hydrolase do they I mean breaking down of the molecule by incorporating water molecule right. On action of an hydrolase it forms 4 miminoglutamate or N 4 miminoglutamate the most important intermediate of histidine metabolism which is also abbreviated as FIGLU F I G L U 4 miminoglutamate right. This 4 miminoglutamate actually can be converted to glutamate involving a 1 carbon metabolism right tetrahydrofolate forms N 5 formimino tetrahydrofolate. I told you when we are discussing 1 carbon metabolism that this belongs to a 1 carbon pool.

1 carbon pool is basically those molecules which participate in 1 carbon reaction. And thus histidine actually histidine metabolism also contributes to the 1 carbon pool by formation of N 5 formimino tetrahydrofolate. And this glutamate can be converted to alpha ketoglutarate by transamination right. So, and that alpha ketoglutarate which is an intermediate of T c cycle can always be converted back to glucose by reversal of glycolysis and gluconeogenesis. So, histidine is glucogenic there is no doubt about it all right.

As I discussed histidine also contributes to the 1 carbon pool by formation of formimino tetrahydrofolate, but one important thing to remember is because of this reaction that is histidine is converted to glutamate and the intermediate is 4 miminoglutamate right. And conversion of 4 miminoglutamate to glutamate needs folic acid right. So, whenever there is a deficiency of folic acid, figlu will be accumulated and it will be excreted in urine. So, actually figlu excretion is a marker of folic acid deficiency a very important applied question for you. So, let us know how this figlu excretion, excretion test actually perform the exact procedure the what is the amount of histidine this is also known as histidine loading dose.

So, histidine is loaded I mean excess dose is given 3 times at 4 hourly interval 5 grams of each ok this is the dose. What happen urine is collected 24 hour after the initial dose. So, we will measure the total amount of figlu that is excreted in 24 hours of collected urine and generally it is less than 30 milligram per 24 hour and this is raised in folate deficiency alright. So, this is the cut off value beyond which it will deal with folate deficiency. Now, this test is not done routinely why because we can directly measure folic acid very accurately nowadays with the help of enzyme linked immunosorbent assay or ELISA.

So, know this this figlu excretion test is important was important, but nowadays it is mostly historically important because we have got ELISA in almost all even peripheral some peripheral centers also right. So, if we got ELISA figlu excretion test is historically

important right, but still for metabolic point of view it is very important. So, as I told you we will discuss all the important product from histidine right and one of them is histamine right. Regarding any competitive exam or any exam this is the reaction or this is the product that you need to remember right. The intermediates of the previous reaction the exact intermediates what was the sequence of reaction may not be so important for you to remember that is the histidine degrading pathway right, but this is important figlu that step of course, it is important right.

But here we should know that histidine by the action of a decarboxylase enzyme decarboxylase means it loses one COO carboxyl group in the form of carbon dioxide and forms histamine right and this is very very very important. In fact, it is so important that question often comes regarding histamine metabolism or degradation of histamine compared to degradation of its parent amine was a histidine. So, let us discuss while we are discussing this histamine just like catecholamine when we were studying tyrosine right you were taught how the catecholamines like adrenaline nor adrenaline were being synthesized right and catecholamine degradation is also equally important to know. If you recall catecholamines were degraded by two enzymes MAO monoamine oxidase and COMT catechol orthomethyltransferase the mechanism is almost similar over here. Here since it is a diamine so diamine oxidase and histamine n-methyltransferase simple.

So, methyl transfer and amine oxidation. So, it forms if it is acted upon by diamine oxidase it forms imidazole acetaldehyde and if it is undergoing a methyl transfer it forms n-methylhistamine. Now know this both of these products can actually feedbackly inhibit their formation right. So, this is a feedback loop where the action of DAO and HNMT can be inhibited by imidazole acetaldehyde and HN methyltransferase respectively right. So, what happens next by the action of aldehyde dehydrogenase both of them can be converted to their final excretory form that is imidazole acetate or n-methylimidazole acetate right fine.

So, these are the enzymes and even on this n-methylhistamine DAO diamine oxidase can act to form n-methylimidazole acetaldehyde. So, these are the final forms in which histamine is finally, converted to an when the action of histamine is over. So, these are the enzymes. So, histidine decarboxylase diamine oxidase methyltransferase the fourth is the feedback loop that is being shown again monoamine oxidase B very important MAO A was acting in catecholamines on catecholamines this is MAO B isoenzyme right and then again this reaction is catalyzed by diamine oxidase. So, why histamine is so important again discussion about histamine detail discussion about histamine is out of the purview of this metabolism lesson, but here is an overview of histamine what it can do.

Most of you may already know that histamine is the mediator of all allergic reaction all alafiraxis, arctic area, pruritus etcetera are done by histamine. Histamine acts by multiple class of sub class of receptor H 1, H 2, H 3 and H 4 and one of the example that is H 2 receptor helps in secretion of gastric acid. So, we often take medicines like ranitidine, famotidine right, ran-tac-fam-tac those are H 2 receptor blocker that will help in decreasing gastric acid secretion. So, that is one example again those antihistaminics are anti allergics how it is used. So, all the reactions that is pruritus, arctic area, hot flushes etcetera are there apart from skin it is also acting on cardiovascular system, it is acting on nerves, it is acting on bone marrow, leukocyte, respiratory tract, uterus, gastric epithelium everything right.

So, you can actually form an endless list of what are the actions of histamine it is that much important. So, you should actually diverge from the metabolic lessons and make a note of yourself or make a point wise note what are the functions of histamine. It will be discussed more or later when you are studying pharmacology because H all the histamine receptor blockers actually active pharmacological agents or medicines ok. There is an another dimension of histidine metabolism that is metabolism of dipeptides. This is not the normal way in which histidine is metabolism is the preferred way histidase and urokinet pathway.

Nevertheless histidine is also involved in synthesis of compounds like anserine and carnosine also 1 methyl histidine by action of multiple enzymes. For example, it is carnosine synthase which forms carnosine, carnosine n methyl transfers again play of 1 carbon it forms anserine, anserine nase degrades it into 1 methyl histidine. Again histidine can be converted to 1 methyl histidine by a methyl transferase. One very important thing to know is you are seeing role of an amino acid which we discussed as a variant of alanine when we are discussing alanine that is beta alanine right. So, beta alanine plays a role in histidine dipeptide metabolism.

So, why do we need to know the role of ancine and carnosine? Well they are very important antioxidants ok, theoretical are tackled or oxidative stress are tackled by these intermediates or these compounds right and they have been shown to reduce cognitive decline in research studies ok. So, you should know that at least you should know these are the products from histidine metabolism even if you do not remember the entire pathway. So, you can jolly well have an MCQ all of these compounds are products of histidine metabolism except you can get anserine, carnosine, eurocanate and sarcosine very close related name carnosine and sarcosine we read sarcosine in glycine and serine metabolism. So, be very careful regarding your questions and if you are mindful and remember your lesson well enough will be good to answer any questions ok. So, what are the disorders of histidine metabolism? The first disorder that we need to know is

histidinemia, autosomal recessive inheritance, absence of histidase enzyme.

So, normally what histidase does it helps in conversion or breaking of histidine to eurocanic acid. If that is not happening naturally histidine will be diverted to alternate routes of metabolism very similar to what was happening in phenylalanine metabolism over there phenylalanine was being converted to tyrosine. If phenylalanine hydroxyl is deficient to a getting converted to phenyl acetate, phenyl lactate, phenyl pyruvate same things are almost similar thing is happening over here. Histidine if it is not acted upon by histidase it is forming imidazole pyruvic acid, imidazole lactic acid and imidazole acetic acid right. And all these intermediates you are just required to remember the names and these intermediates will lead to features of amino acid metabolism defect disorders of amino acid metabolism which is mental retardation and delayed speech development all right.

How we can prevent this? If we diagnose this this is naturally we need to reduce the load of histidine in diet right and generally it may have some protective effect right, but know this this is the autosomal recessive disorder it is not always expressed unless both the genes are recessive and homozygous ok. You can if you have studied phenylketry well enough you can easily draw the analogy to this ok. Next urocanic aciduria, what is urocanic aciduria? Defect in the urocanase enzyme. So, urocanase was converting urocanate to 4 imidazolone 5 propionate if this is deficient all the previous intermediates will accumulate. So, it is very easy to remember urocanic acid and histidine are extra in urine right.

However, it is good compared to other diseases that clinical manifestations are minimal ok only the high level of these compounds are found. Now imidazole amino aciduria, what is imidazole amino aciduria? It is a defect in renal transport mechanism. So, these amino acids and its products in dipeptides are excreted in large amount that is histidine and 1 methyl histidine right. We already read this reaction a while back. So, these intermediates will be excreted in urine right.

This has this is very important because it leads to problem in eye cerebro macular degeneration and retinal degeneration which may lead to blindness right. And the defect in urinary transmission or renal defect is actually transmitted as a dominant trait right. So, excess amino acids may be excreted in urine that is present in majority, but the cerebro macular degeneration is actually recessive trait. So, it is good it is beneficial in a way that even if the disease is disease is present in most individual the symptom the dangerous symptom of blindness appears I mean is present very less right. So, remember imidazole amino aciduria selective receptor defect or selective transport mechanism defect in which histidine that is this imidazole amino acid is excreted in urine right.

So, after histidine we now focus on to proline right. Proline is a non essential glucogenic amino acid right. Whenever we are discussing non essential means we at least need to know how it is synthesized. Proline does not participate in transamination reaction. What was the another amino acid that was not participating in transamination right this is your homework.

I hope you have recalled your lessons well enough and you should be able to answer it right. So, this is the synthesis of proline ok. So, you can see proline is first acted upon by an enzyme delta 1 pyrrolein 5 carboxylate synthetase this is a big intermediate it is abbreviated as p 5 c s. And it is in the similar reaction step that this enzyme or this compound will be reduced as well. And this enzyme synthase, synthetase by the action of ATP reductase and dehydrogenase are all acting, but the only important thing for you to remember in this whole synthetic pathway is the source of synthesis is glutamate and ornithine most important source being glutamate and since ATP is used this is a synthetase enzyme right.

A very important intermediate of glutamate semi aldehyde is formed right and it undergoes a spontaneous cyclization to form proline. Now, you may think that this discussion is not in depth right well it is on purpose because when we will be studying degradation of proline you will see this is the exact reversal of this pathway right. So, ornithine is a minor source of proline glutamate is the most important source because when proline is degraded it has a tendency to form glutamate and also ornithine right. So, as I told you this is the exact reversal during degradation of proline. So, what happens the exact reverse steps are happening.

So, first of all a dehydrogenase enzyme proline dehydrogenase it was there it is also here it is converting it to pyrrolein 5 carboxylate this has not been shown over here right, but it is there. So, it is first formed into pyrrolein 5 carboxylate right which undergoes spontaneous conversion to this semi aldehyde right and the semi aldehyde is again acted upon by semi aldehyde dehydrogenase to form glutamate which is then finally, transaminated to alpha-tetoglutarate right. So, the most important thing to remember over here is the fate of proline and presence of this compound glutamate semi aldehyde ok this one important structure not important names very important. Proline in protein is converted to its hydroxyl form it is has got a tendency to be hydroxylated by the enzyme prolyl hydroxylase right. For example, proline in prolyl is present in collagen and it is hydroxylated.

So, there is presence of proline there is also presence of hydroxyproline right. So, it is hydroxylated it is known as hydroxyproline and for this hydroxylation this prolyl

oxidase it needs multiple coenzymes first of all it needs molecular oxygen right then it is vitamin C iron and alpha-ketoglutarate right. And if there is excess degradation of this compound it will again lead to production of hydroxyproline. So, hydroxyproline can be actually isolated from these proteins right, but it proline needs to be incorporated first and then it needs to be hydroxylated while it is inside the peptide chain all right a single proline generally is not hydroxylated by prolyl hydroxylase prolyl hydroxylase acts on the long peptide chain. So, since we read that this prolyl hydroxylase enzyme needs vitamin C to perform right and we also discussed that this hydroxyproline or formation of hydroxyproline is very important for the structure of collagen the deficiency of vitamin C leads to collagen defect and the clinical feature is actually scurvy characterized with breeding gum there are multiple plethora of clinical features of scurvy and breeding gum is one of them right.

So, basically what defect of collagen leads to collagen is a structural protein. So, it leads to decrease strength of fibres right. Now, excretion of hydroxyproline in urine is increased in infiltrating bone tumour and in diabetic patient due to enhanced rate of protein catabolism I told you hydroxyproline is normally present in structural protein. So, whenever excess protein is being degraded we can find hydroxyproline because hydroxyproline is directly a breakdown product of proteins and peptides and whenever there is increased catabolism we can find hydroxyproline in urine ok.

Now this disease immunoglycinuria right. So, proline is an amino acid right proline is an amino acid contains an amino group and likewise hydroxyproline is also an amino acid. So, this disease immunoglycinuria as you have guessed by now there is also glycine. So, whenever we are associating urea with an this it means excretion of these in term or these amino acids in urine. So, why does it happen it is due to defect in luminal reabsorption of these amino acids this amino acids and glycine that is proline hydroxyproline and glycine we have read glycine urea alright where we have also read a variant where there was defect in renal reabsorption of glycine this is a combined transporter which is sodium potassium dependent that is also ATP dependent active transporter and it acts as a co transporter. So, a combined co transporter of sodium potassium and these amino acids and glycine which is defective in brush bordered cells of gastrointestinal tract as well as renal proximal convoluted tubule.

So, the when it was supposed to be absorbed from gut it is not absorbed when it was supposed to be reabsorbed from urinary tubule it is not reabsorbed resulting in excretion of these amino acids and glycine in urine and stool this is the disease of immunoglycinuria you can have multiple choice questions. So, be very mindful right. We move to our next amino acid of discussion that is arginine highly basic semi essential amino acid if we consider the fate it is glucogenic what is so special about arginine it has

got a guanidinum group right these are special features regarding the chemistry of arginine. So, if we are discussing the fate of arginine we already know we have read urea cycle and over there you know how arginine was produced as an intermediate of urea cycle and the enzyme arginase was acting upon arginine to produce ornithine which was again repeating the urea cycle. If you have if you do not recall right now it is high time you pause this video have a look at urea cycle and then resume it right.

So, what happens ornithine then undergoes further reaction right in the urea cycle. If we are not concerned about urea cycle we just read in synthesis reaction just a few while back that ornithine can be transaminated during synthesis of proline it can be transaminated to semi aldehyde same semi aldehyde very important and that semi aldehyde is being converted to glutamate by a dehydrogenase and ultimately by transaminase to alpha keto glutarate right. And you already know from urea cycle disorder that hyper argininemia is an inbounder of arginine metabolism due to defect of the enzyme arginase alright. So, an important product of arginine metabolism is nitric oxide or NO this enzyme or this product is it is a gaseous product and it is synthesized by the enzyme nitric oxide synthase or NO synthase often abbreviated as NOSNOS right. And the number of cofactors that this enzyme needs are many NADPH, flavin mononucleotide or FMN, flavin adenine dinucleotide FAD, heme and tetrahydrobiopterin right.

This nitric oxide has got very short side effect it exerts the action quickly and it is then destroyed. So, this is the reaction that is where nitric oxide is synthesized the arginine is converted to another atypical amino acid that is citrulline right and oxygen is converted to nitric oxide right. Why it is ENOS? I will tell you very soon ok and these are the cofactors right oxygen NADPH, FMN, FAD and heme. So, this nitric oxide synthase has got three iso enzymes. So, let us discuss them one by one the first iso enzyme or NOS1 or neuronal variant is also known as NOS it is seen in central peripheral neurons right.

So, since it is present in neurons or nerve it acts on the nervous system. So, cerebrum as well as in neurons of gastrointestinal tract right nitric neurons right it is a cytoplasmic enzyme and it is activated by calcium all of these facts are multiple choice questions. Second variety macrophage NOS or INOS why it is called I because it is inducible right same thing INOS macrophage NOS inducible NOS or NOS2 it is mainly seen in macrophage is a neutrophil and it also present in liver cells. So, why do we call it INOS or inducible because it is induced by cytokines right. Cytokines are chemical compounds that are mediators of multiple reaction for example, inflammation.

So, few examples of cytokine is interleukin 1 tumor necrosis factor. So, these are the cytokines or chemical compound that can induce this enzyme to form nitric oxide right

again this is cytoplasmic enzyme one key difference from first isoform is it does not active it is not activated by calcium. So, calcium has got no effect on this enzyme right. The third variety is endothelial very important INOS right it is in endothelial cells platelets endocardium and myocardium right. So, in these sites blood is constantly flowing right these all these tissues mentioned are constantly in touch with blood vessels arteries and nitric oxide is actually constantly relaxing blood vessels.

Hence it is also known as endothelium derived relaxing factor or EDRF it is located in the plasma membrane and this variety third isoform is actually activated by calcium. There are some debates where many scientists have said that NO and EDRF are different, but multiple research papers have finally, shown that there is no structural difference and action of NO and EDRF. So, for all practical purposes these are considered to be one and the same. So, these are few roles of NO right nitric oxide it is and how the drugs that help in production of nitric oxide antagonist production of nitric oxide can be used for therapeutic manipulation. For example, in chest pain we need angina that is a disease where blood vessels are narrowed and due to lack of blood supply there occurs chest pain.

It can be treated when we can device a drug or medicine which can increase production of NO. For example, nitroglycerin or GTN or sorbitrate often we see chest pain one small tab hard shaped tablet is put under the tongue same mechanism using nitric oxide right. It acts as a vitro vasodilator it prevents platelet aggregation it regulates blood flow it lowers blood pressure. So, NO antagonist actually markly increases blood pressure. It functions as a messenger molecule and also it mediates bactericidal action of macrophages the action of INOS right during multiple infection those cytokines they induce this production of INOS and thus kills the bacteria right.

Since it is a vasodilator it has got role in erection of the penis because it dilates the vessels of corpus lutei ok . And very important this action of NO via CGMP all right. This is again a discussion of signaling and messenger, but you should know if you are studying nitric oxide for your own interest you can go to the biosignaling chapter of your any textbook biochemistry and over there you can read how NO acts right. So, finally, we look into the interrelationship of amino acids as we always do. So, we can see proline via formation of delta 1 pyridine 5 carboxylate is converted to glutamate gamma semi aldehyde right.

You already read that arginine via arginase is converted to ornithine and ornithine can be transaminated to form the same semi aldehyde. That semi aldehyde is ultimately converted to glutamate by a dehydrogenase enzyme. We have also read that this histidine by action of his multiple steps is converted to glutamate you know glutamine we have

read during ammonia metabolism can be converted to glutamate and ultimately by GDH reaction glutamate dehydrogenase it can form alpha ketoglutarate. So, you can see so many of these amino acids are forming alpha ketoglutarate all right. So, all of these via glutamate is getting converted to alpha ketoglutarate ok.

So, we are left with lysine the lysine is also basic amino acid. So, why we are discussing lysine separately because in spite of being an essential amino acid and basic it is a ketogenic amino acid. So, it is metabolic it is actually different. We should know serial proteins are deficient in lysine and it does not participate in transamination reaction right. So, if we consider lysine in protein it can be present in multiple forms that is lysine residues are often chemically treated hydroxyl lysine methyl lysine and acetyl lysine.

So, these derivatives can be hydrolyzed to form free lysine right. So, like hydroxyproline hydroxy lysine is also present in collagen. Now, when we are discussing methyl lysine the methylation is done by the common methyl donor that is S adenosyl methionine we already know that right and this when these proteins with methyl residues are degraded by proteolytic enzyme it will release methyl lysines as well ok. So, why do we need to know about methyl lysine because whenever because methyl lysine actually a cofactor I mean a precursor for carnitine synthesis right. So, this is are the steps by which carnitine is synthesized ok. The intermediate enzymes and the intermediate products are not necessary for you to remember for any competitive exam, but in case of an exam like national common entrance exam for medical exam there might be some image based questions where one of the intermediates might be removed and you will may be required to fill up using an image based answer.

So, one of the four answers will be given and you need to choose the right one which fits the puzzle right. So, but the most important thing for you to know is tri methyl lysine serves as a precursor and what is the biological important role of carnitine you have already read when we were learning beta oxidation and oxidation of fatty acid that carnitine is help is basically involved in transport of fatty acid to the mitochondria right. So, if we just look into the what are the steps the first step is actually an oxidation step by a dioxinase enzyme followed by a splitting in reaction by aldolase you already know aldolase always cleaves and it leads to formation of I mean glycine residue is given off thereafter by action of a dehydrogenase and another oxidation we get carnitine right. So, what are the functions of lysine? So, lysine and hydroxy lysine are present as important product in or important residues in collagen and elastin and they help in structural cross linking of protein right. Lysine has got an epsilon amino group if you look at the structure of lysine the amino group is present in the epsilon carbon right and it can form shift bases by linking with proteins and this lysine is also found in high quantities in histones histine is also found right and because these are basic amino acids this is closely

associated with nucleic acids.

Finally, we need to know the ketogenic fate of lysine you already discussed what is the fate of lysine right. So, in this reaction what we need to know is this intermediate alpha keto adipic acid. So, whenever we remember alpha keto adipic acid which you have also learned when you are being taught about tryptophan metabolism right because tryptophan the fate of tryptophan also lies in the same pathway. So, lysine and hydroxy lysine actually forms alpha amino adipic acid which is then transaminated to form alpha keto adipic acid it loses one carbon dioxide decarboxylase enzyme to form glutaryl CoA and once we get glutaryl CoA there are three more intermediates one is glutaconyl CoA, crotonyl CoA you have all you have read glutanyl from glutaconyl CoA to crotonyl CoA during metabolism of branched chain amino acid right.

If you do not remember you should revise your lessons properly. So, ultimately once we get glutaryl CoA you already know that crotonyl CoA dissociates to form acetoacetyl CoA which again further splits up to form two molecules of acetyl coenzyme A. So, if we look at the metabolic fate of the ketogenic amino acid you can see tryptophan right as well as phenylalanine and tyrosine forms acetoacetyl CoA. Leucine branched chain amino acid was also forming acetoacetyl CoA right and isoleucine is also forming both acetyl CoA and succinyl CoA right. So, we have magnified this thing if this is for you to just recall what was taught before right so that you can connect all the dots over here and finally, you can also join all the things all the amino acids to their common ketogenic fate which again lies over here which ends over here right and mind it isoleucine since it was both glucogenic and ketogenic it forms succinyl CoA and acetyl CoA which was already discussed during branched chain amino acid metabolism. And so, if you can actually recall all of the amino acids have now been discussed at this point you may think ok only one amino acid was left.

So, it is not basic it is not related to this, but it is very important to know that as per gene by the action of as per genease is converted to aspartate and you already know the action of AST by which it can be converted to oxaloacetate and thus it can also enter the final pathway of metabolism of carbon skeleton of amino acids. So, at this point you should be able to know and answer what are the sources and what is the metabolic fate of all amino acids that is shown in this diagram all right. So, to conclude this chapter has this we have discussed the metabolism of histidine how histamine is formed how histamine is degraded we have discussed about metabolism of proline we have discussed about metabolism of arginine nitric oxide role of nitric oxide we have discussed the metabolism of lysine what is the common metabolic fate what is the common fate of glucogenic amino acid what is the common fate of ketogenic amino acid and all the

amino acids are somehow connected in a common pathway these are my references and I thank you for your attention. .