Biochemistry

MASTERS

Amino Acid Metabolism

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24 Last

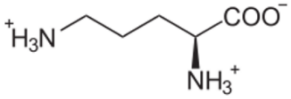
Nitrogen & Amino Acid Metabolism

Introduction

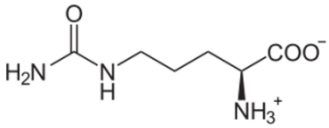
- Nitrogen balance is critical for our body, both high or low levels of nitrogen results in problems (not good situation for our body)
- High levels of nitrogen results in toxicity due to accumulation of ammonia (NH_3) or production of reactive nitrogen species (RNS)
- Excess nitrogen (in form of ammonia) can be gotten rid in form of urea by urea cycle
- Nitrogen (ammonia) is used in our body to make Amino acids, and also is produced from breaking down amino acids
- Nitrogen also is needed for the synthesis of:

1- Nucleotides (ATP, GTP, CTP, UTP, dATP, dCTP, dGTP, dTTP)

2- Non-Protein Amino Acids (which means that those amino acids isn't found in proteins)
   - ➔ Ornithine
   - ➔ Citrulline
   - ➔ Sarcosine



Ornithine



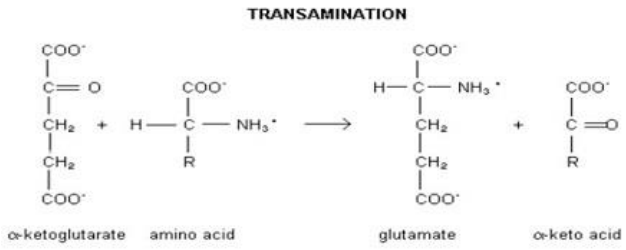
Citrulline

3- Other Nitrogen-Containing Compounds
   - ➔ Choline
   - ➔ Vitamin
   - ➔ Carnitine

Disposal of amino acid nitrogen *(how to remove amino group from amino acids)*

1- Transamination

- A transport of amino group from amino acid to α-keto acid (α-ketoglutarate)
- It begins with the removal of the amino nitrogen.
- Amino group is transferred to α-ketoglutarate by transamination. This results in the formation of the keto acid corresponding to the amino acid + glutamic acid.



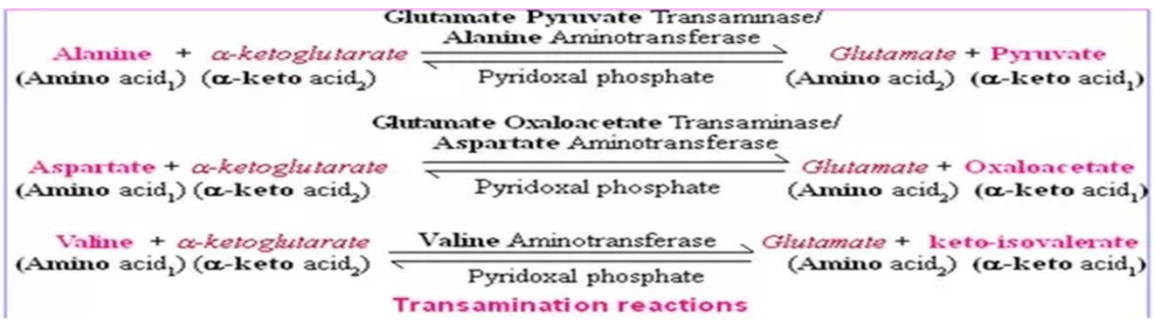
- Then Glutamate is the subjected to <u>oxidative deamination</u> by **Glu DH**

- This reaction is catalyzed by **transaminases**, in which are named in terms of the amino acids and need pyridoxal phosphate as coenzyme which is derive from vitamin B6 (pyridoxine)
- Transferases involves:

a- **Alanine aminotransferase**(glu-pyruvate transaminase): transfers amino group from alanine (ala) to α-ketoglutarate forming pyruvate (α-keto acid of ala) & glutamate

b- **Aspartate aminotransferase**(glu-OAA transaminase): transfers amino group from Aspartate (asp) to α-ketoglutarate forming OAA (α-keto acid of ala) & glutamate

c- **Valine aminotransferase:** transfers amino group from valine (Val) to α-ketoglutarate forming keto-isovalerate (α-keto acid of ala) & glutamate



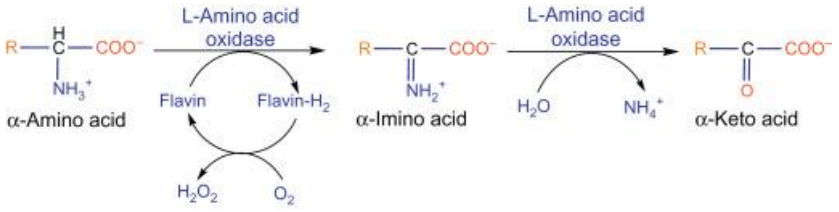
Important notes

- All amino acids with the exception of **threonine(thr)** and **lysine(lys)** can participate in transamination reactions.
- Most of the nitrogen of the amino acids eventually appears in the form of glutamate.
- The transamination reaction is reversible

2- Oxidative deamination

- Catalyzed by **L-amino acid oxidase**; for all amino acids except **serine (ser)** and **threonine**
- This enzyme needs flavin (derived from vitamin B2 –riboflavin–) as coenzyme (prosthetic group)
- steps (all are catalyzed by L-AA oxidase):

> **Remember**: all amino acids in our body are in L configuration not in D



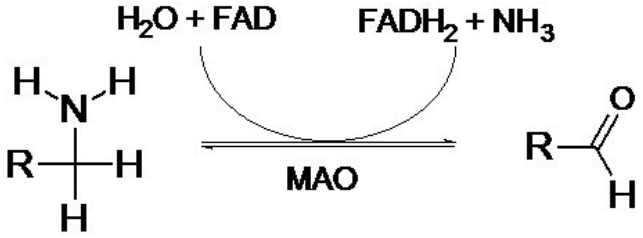
a- Formation of α-imino acid: A double bond is formed between N and αC (oxidation) which results in reduction of flavin prosthetic group (flavin-H_2)

➔ To replenish the flavin (in oxidized form) it donates the 2 electrons to O_2 forming hydrogen peroxide.

b- Replacement of imino group by adding water, thus removing the amino group in form of ammonia and forming α-Keto acid.

3- monoamine oxidase:

- presents in the liver
- needs FAD as coenzyme (prosthetic group)
- catalyzes the aerobic oxidation of wide variety of physiologically important amines such as epinephrine, norepinephrine, dopamine, and serotonin (derived from tyrosine).



- This enzyme removes the amino group in form of ammonia and adds water and produces the amines + $FADH_2$

4- Non-oxidative deamination:

- A group of **pyridoxal phosphate dependent dehydratases** catalyze the removal of the amino groups of serine, threonine, and cysteine.
- Ser gives pyruvate when deaminated
- Thr gives α-Ketobutyrate when deaminated

serine - threonine dehydratase

serine -----> pyruvate + NH4+

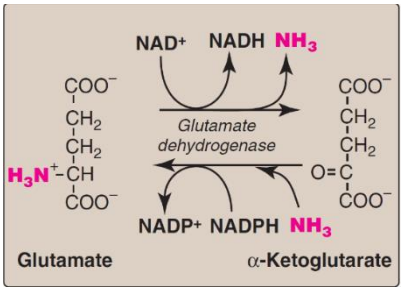
threonine --> alpha ketobutyrate + NH4+

Ammonia fixation

- This means how make ammonia in a non-toxic form in our body & this via:

1- Glu DH:

➔ Catalyzes a reversible reaction, in which ammonia is add to α-ketoglutarate to form Glu & vice versa.



➔ Glu to α-ketoglutarate (Degradation) direction, NAD^+ is reduced to NADH

➔ α-ketoglutarate to Glu (synthesis) direction, NADPH is used

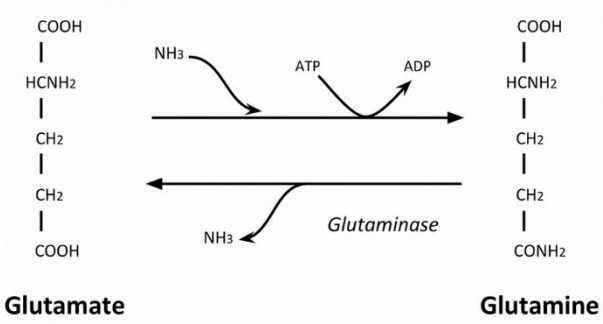
Direction of reaction	Allosteric regulators
like any enzyme, it depends on the relative concentrations of substrates; glutamate, a-ketoglutarate, ammonia, and the ratio of oxidized to reduced coenzymes	ATP and GTP are allosteric **inhibitors** ADP and GDP are allosteric **activators**

2- Glutamine (Gln) synthetase

➔ ammonia also can be trapped in form of Gln by adding it to Glu to form Gln. This process needs ATP consumption

➔ Gln acts as non-toxic transporter of ammonia from peripheral tissues to liver

➔ In liver ammonia is released from Gln by glutaminase and used in urea cycle to form urea to be excreted from the body



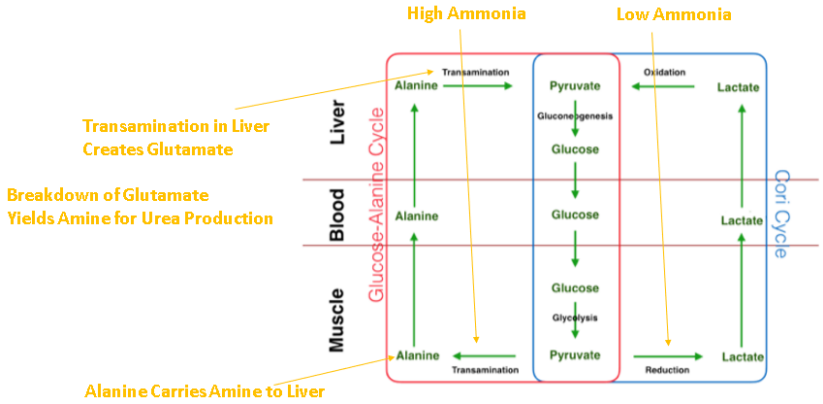
How ammonia is transported from extrahepatic tissue (peripheral tissue)

1- In form of glutamine (already discussed how)

2- From Skeletal muscles in form of alanine

- In exercising muscle, pyruvate has 2 fates:

a- When there are **low levels of ammonia**, pyruvate is reduced to lactate and then lactate go to liver through bloodstream and converted back to glucose which can be consumed again by this muscle (Cori cycle)



b- When there are **high levels of ammonia,** it is trapped as glutamate then the amino group is transferred to pyruvate forming alanine in which it travels to liver through bloodstream

➔ In liver another transamination reaction between Ala and α-ketoglutarate forming pyruvate (used to synthesize glucose) & glutamate

➔ This is called alanine-glucose cycle, important for regenerating glucose also in transportation of ammonia from skeletal muscles

Note: pyruvate can be used to synthesize alanine, leucine, isoleucine & valine

Central role of glutamate

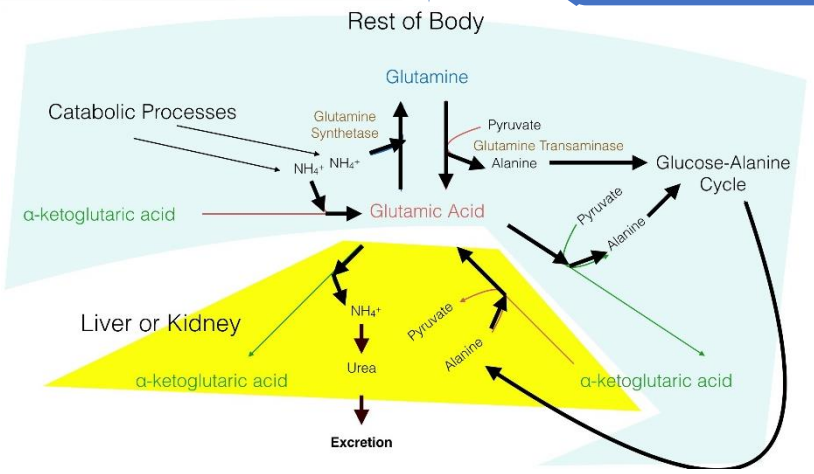
- Because of the participation of 2-oxoglutarate (α-ketoglutarate) in numerous transaminations, Glu is a prominent intermediate in nitrogen elimination in addition to anabolic pathways.

- Glu, formed during nitrogen elimination, is either oxidatively deaminated by liver **glu DH** forming ammonia or converted to glutamine by gln synthetase and transported to kidney

- There (in kidney) the Gln is sequentially deaminated by **glutaminase** and then deaminated by kidney **Glu DH**

- this figure summarized the central role of glu in nitrogen disposal & anabolic pathways (will be explained in video)

- An important thing to remember that the ammonia that is released from glu in liver and kidney is used to form urea to be excreted in urine.

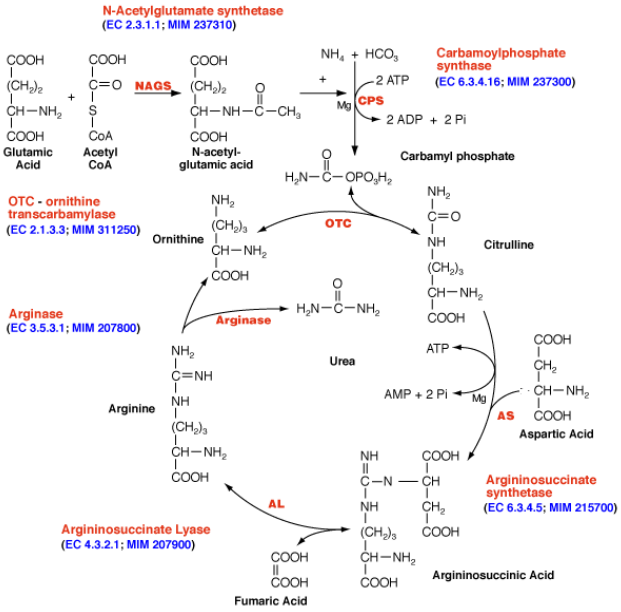


Urea Cycle

- Ammonia is toxic compound if its concentration goes above 4 x 10^{-5} M, how it produces its toxicity? Via reductive amination of α-ketoglutarate which results in depletion of TCA intermediates.
- Synthesis of urea results in a nontoxic which is easily excreted in the urine. In simple words, urea is a non-toxic excretion method of ammonia
- Very low level of urea synthesis may occur in kidney and brain, but the major site is liver
- Regarding steps of urea cycle, the first two steps in the mitochondrial matrix; the remaining three steps in cytosol.

1- Carbamoyl phosphate synthesis – feeder reaction –

- by adding up bicarbonate HCO^{-3} (or CO_2) to free ammonia (from glutamate or alanine).
- Catalyzed by **Carbamoyl phosphate synthetase -1(CPS-1)** – found in mitochondria –
- Requires 2 ATP (one of the Pi remains in the skeleton the other will be used later).
- CPS-1 is activated by N-acetylglutamate in which is synthesized from acetyl coA + glutamate by **N-acetyl glutamate synthetase** which is activated by arginine
- CPS-2 is involved in the pyrimidine metabolism in cytosol. Both 1&2 give carbamoyl phosphate.
- This compartmentalization is important, why? It is simple, if carbomyl phosphate is produces in mitochondrial matrix then it is used for urea cycle, and if it is produced in cytosol then it is used for pyrimidine biosynthesis



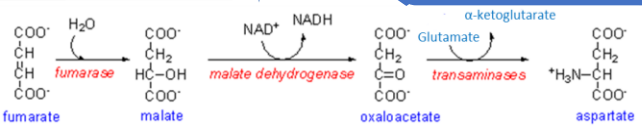
2- Ornithine–carbomyl phosphate condensation

- Ornithine molecule enters the mitochondria and receives the carbamoyl group (without inorganic phosphate) to form Citrulline.
- Catalyzed by **Ornithine transcarbamylase (OTC)** (most common to be defective in case of hyperammonemia)
- This reaction goes forward by using the inorganic phosphate that mentioned before.
- Ornithine & Citrulline are non-genetically coded & non-protein Amino acid
- a counter-transport (antiporter) system that allows ornithine to enter the mitochondria and citrulline to go out.

3- Aspartate – citrulline condensation

- After citrulline is transported to the cytosol, it condenses with asp forming Argininosuccinate
- Catalyzed by **Arginosuccinate synthetase (ASS)**

- Requires 1ATP hydrolysis to AMP (equals using 2 ATP)
- How can we get aspartate? From transamination of OAA with Glu as in figure



4- Arginosuccinate lysis
- The release of fumarate from Arginosuccinate to form Arginine.
- Catalyzed by **Arginosuccinate lyase (ASL)**
- Fumarate is converted again into Aspartate in TCA cycle to keep proper aspartate level to use it in the urea cycle: (Fumarate→ Malate→Oxaloacetate→Aspartate)

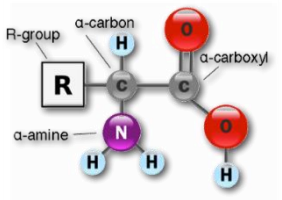
5- Arginine hydrolysis
- Arg is hydrolyzed (by adding water) to orthenine and UREA.
- Catalyzed by **Arginase I** –manganese containing– , unlike previous enzymes, it is found in liver only, so urea cycle occurs only in the liver
- Other isozyme found in kidney (arginase II)
- Ornithine enters the mitochondria to participate in urea cycle again

Fate of urea
- Some goes to the bile
- The most amount diffuses into the blood then the kidney to get urinated
- another small amount travels to small intestine where certain bacteria, by enzyme urase, break down Urea to CO_2 and Ammonia which returns to the liver through blood to be used again in urea cycle

Overall Stoichiometry of Urea Cycle
Aspartate + free NH_3 + HCO^{-3} (carbon) + 3ATP + H_2O (oxygen) >> Urea + fumarate + 2ADP + 2Pi + AMP + PPi

Disorders of urea cycle
- The primary metabolic disturbance is in the liver, since it is the major site.
- The principal clinical abnormalities is damage to the central nervous system, severe mental retardation.
- The neurological damage is mainly caused by toxic effects of elevated blood ammonia particularly after heavy protein meals.
- Five diseases, each representing a defect in the biosynthesis of one of normally expressed enzymes of the urea cycle.
- In four of these diseases: CPSD, OTCD, ASSD & ASLD. (D: deficiency)
- Arginase deficiency disease of progressive spastic tetraplegia (paralysis in 4 limbs) and mental retardation

Amino Acids Metabolism



- as a reminder, AAs composed of alpha C that is bound to: Amino group, Carboxyl group, Side chain "R & hydrogen atom.
- There are 20 common amino acids and one rare amino acid (selenocysteine)
- There is no Single Pathway for Amino Acid Metabolism.
- Synthesis Pathways are Grouped **According to Common Anabolic Precursors**

1- α-ketoglutarate (GLN)
2- Serine
3- Aspartate
4- Aromatic
5- Pyruvate
6- Histidine

Classification of AAs:

1- **Depending on their R group (already discussed in mid)**
2- **Glucogenic/ketogenic (already discussed)**
3- **In term of essentiality:**

a- **Essential:** our body can't synthesize them (we take them from diet)
b- **Non-essential:** our body can synthesize them
c- **Semi-essential:** required for children growth such as histidine
d- **Sparing amino acids:**

- Tyrosine synthesis requires the availability of phenylalanine
- Cysteine requires methionine.
- If these essential aa (Phe & Met) are only available at or below the minimal requirement, then tyrosine and cysteine become essential amino acids.

Essential AA summarized as **PVT.TIM.HALL**

P: henylalanin	**T: hreonine**	**H: istidine**	**A: rginine**
V: aline	**I: soleucine**	**L: ysine**	
T: ryptophan	**M: ethionine**	**L: eucine**	

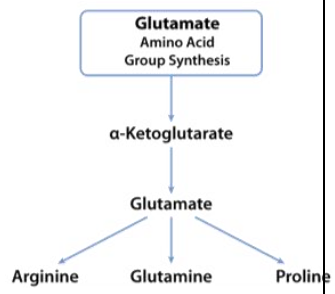
Nitrogen balance in the body

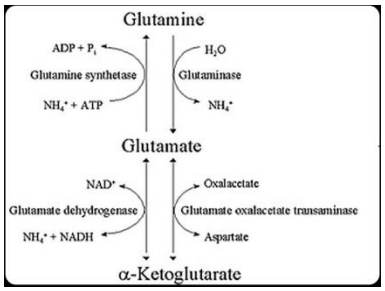
1- **Negative nitrogen balance:** deficiency of just one amino acid prevents the synthesis of proteins, thus, the other amino acids cannot be utilized. That is more nitrogen is excreted than is taken into the body. (food deprivation, illness, and aging)
2- **Nitrogen equilibrium:** is a characteristic of adult where nitrogen loss just balance nitrogen intake.
3- **Positive nitrogen intake:** is a characteristic of growing children and of the convalescent adult where the nitrogen intake is greater than nitrogen loss.

ملاحظة مهمة، في تفاصيل معينة الدكتور طلبها منكم، وهاي التفاصيل رح تلاقي بجنبها imp خلال الشرح لتميزها عن غيرها، ولكن للحيطة ادرس كل شيء، واذا انزنقت ركز وادرس الي بده إياه الدكتور

α ketoglutarate group

- α-ketoglutarate is the precursor molecule (imp)
- it starts with the synthesis of Glutamate (imp), and already discussed how can we get glutamate, via transamination with α-ketoglutarate, or reductive amination via Glu DH, or from deamination of glutamine by glutaminase
- (imp) includes, Glu, Arg, Gln & Pro



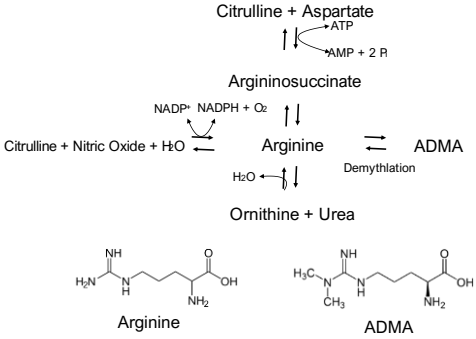


Glutamine synthesis: (imp)

➔ Glutamine synthetase is highly regulated by allosteric effectors, for example alanine, glycine, AMP, tryptophan inhibits it
➔ It is scavenger (cleaner) for ammonia
➔ This figure summarizes the whole process

Arginine synthesis (imp):

➔ from lysis of arginosuccinate
➔ from condensation of Citrulline, nitric oxide (NO) & water
➔ From condensation of ornithine & urea



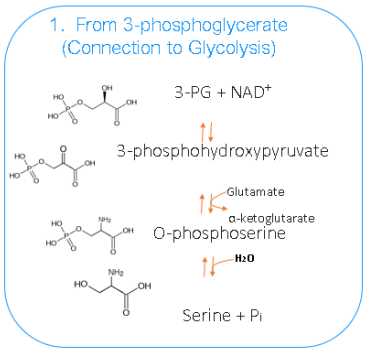
➔ From demethylation of asymmetric dimethyl arginine (ADMA)

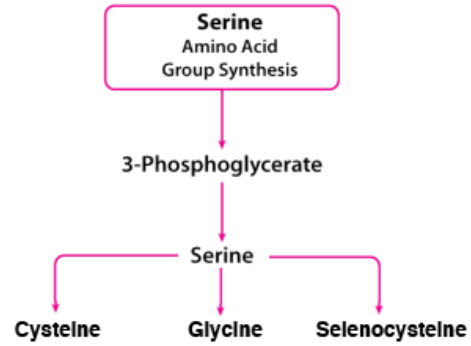
Serine Family (imp)

- Serine is a parent compound for cys, gly & selenocysteine

Serine can be synthesized via 2 pathways:

1- **From 3-phosphoglycerate** as in figure





➔ Steps:

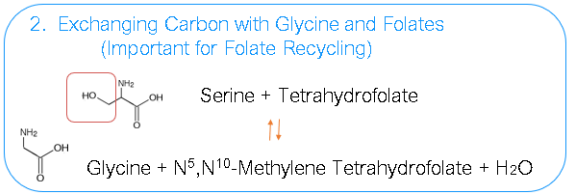
a- 3-phosphoglycerate is reduced to 3-phosophohydroxypyruvate

b- Then a transamination reaction with glu, forming O-phosphoserine

c- Release of Pi by adding water to form serine

➔ Detailed step is not required according to doctor

2- **Exchanging Carbon with Glycine and Folates**



➔ The difference between glycine & serine is methylene group + OH

➔ So, when this methylene is transferred to tetrahydrofolate (THF) – derived from folic acid (B9) – to form glycine + N^5,N^{10}-methylene THF + water

➔ This reaction is reversible, so we can synthesize serine from glycine as in figure

Cysteine Metabolism

- Ser provides backbone for Cys. (imp)
- Methionine Catabolism provides SH group of cysteine. (imp)
- NH_4^+ is released
- Cysteine is synthesized through homocysteine formation from methionine cycle as follows:

1- methionine is converted to SAM (S–Adenosyl Methionine)

➔ catalyzed by **SAM synthetase**

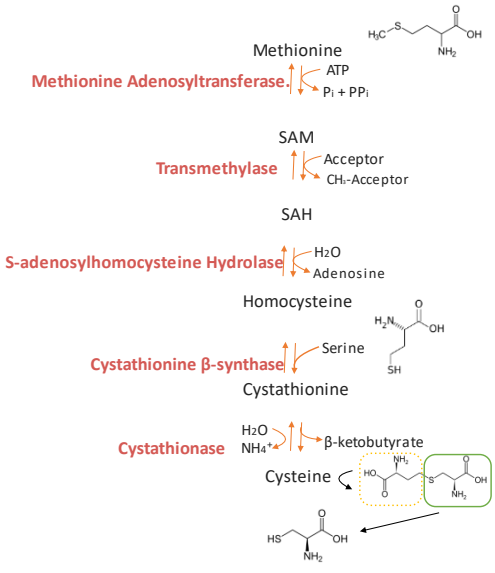
➔ SAM is a **methyl donor** for numerous reactions (Norepinephrine ➔ epinephrine)

➔ methionine' S-atom becomes activated by ATP with the addition of adenosine to the sulfur of methionine, adjacent to the methyl group to form S–Adenosyl Methionine (SAM).

➔ the methyl group is usually transferred to a carbon, oxygen or nitrogen atom.

2- SAM becomes SAH (S–Adenosyl Homocysteine), by losing its methyl group. Catalyzed by **methyl transferase**

3- SAH is converted to homocysteine by losing Adenosine .



4- Homocysteine fates:

a- Converted back to meth (not required)

b- Converted to cysteine through:

➔ **serine** condenses with **homocysteine** forming **cystathionine by cystathionine β-synthase**.

➔ **cystathionine** is hydrolyzed by **cystathionase** producing **cysteine** & **α-ketobutarate**.

➔ **Both require vitamin B6**

➔ α-Ketobutyrate eventually converts to succinyl-coA (an intermediate of the TCA cycle),which means that methionine is glucogenic amino acid.

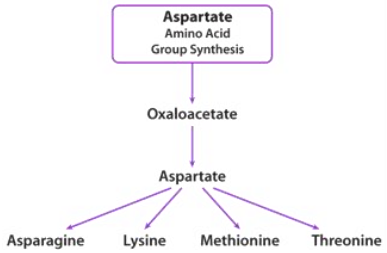
- What is required from you according to dr that: (imp)
  - ➔ formation of cysteine through formation of homocysteine
  - ➔ increase homocysteine levels increase the risk of having cardiovascular diseases, and it is increased in such diseases
  - ➔ cystathionine β-synthase reaction
  - ➔ cystathionine β-synthase deficiency results in accumulation of homocysteine & homocystinuria → increase the risk of cardiovascular diseases

Selenocysteine synthesis

- Sometimes Called 21st Amino Acid
- Contains selenium, which is rare element & excellent antioxidant, instead of sulfur.
- Not Specified Directly in Genetic Code
- Uses Stop Codon with Unusual Structure
- Synthesized from Serine on tRNA (transfer RNA):
  - ➔ Ser is bound to a specific tRNA which is specific for a stop codon with a specific sequence on mRNA
  - ➔ Then ^{Ser}tRNA is transformed to ^{Cys}tRNA then to ^{Sel}tRNA by replacing the SH on cys with SeH to form selenocysteine

Aspartate family

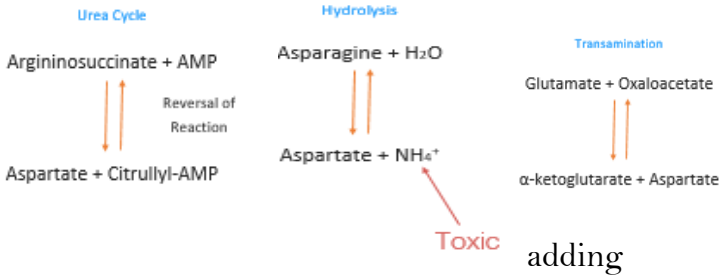
- Remember that asp came from the oxaloacetate by transamination reaction.
- aspartate is a **parent compound** for the aspartate family that belongs the following : methionine and asparagine lysin and threonine.



Aspartate synthesis

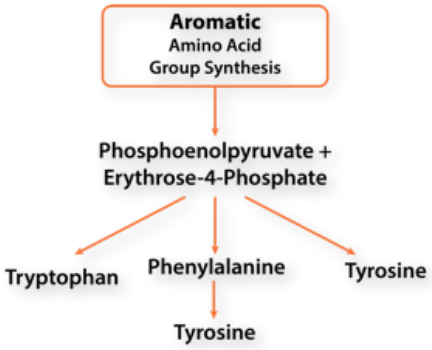
1. Transamination of the amino acid.
2. Hydrolysis of the asparagine by asparaginase into aspartate and ammonia which is toxic.
3. Hydrolysis of the Argininosuccinate that come from the urea cycle.

- asparagine can be synthesized from asp by adding ammonia to asp



Aromatic Family

- it originates from the **phosphoenolpyruvate** which is a glycolytic intermediate and **erythrose-4-phosphate** which is intermediate of the hexose monophosphate shunt.

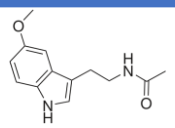


What is important to know about these amino acids that:

1- Tryptophan (Trp):

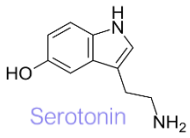
- give the neurotransmitters and other important compounds like:

a- Melatonin:



- ➔ the hormone that is important – or affects – for sleep, Mood and blood pressure
- ➔ Circadian Rhythm (Day-Night cycle) Sensing
- ➔ Production Affected by Blue Light

b- Serotonin

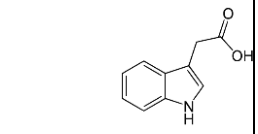


- ➔ Neurotransmitter that Causes Vasoconstriction
- ➔ Enhances Memory/Learning, Contributor to Happy Feelings

c- Niacin

- ➔ Vitamin B_3
- ➔ Nicotinamide, which is Part of NAD^+/NADH & $NADP^+$/NADPH, is derived From it
- ➔ Deficiency Leads to **Pellagra**

d- Auxins



- ➔ Stimulate Cell Division and Rooting in Plants (important in plant growth)
- ➔ Indole-3-Acetic Acid Most Important

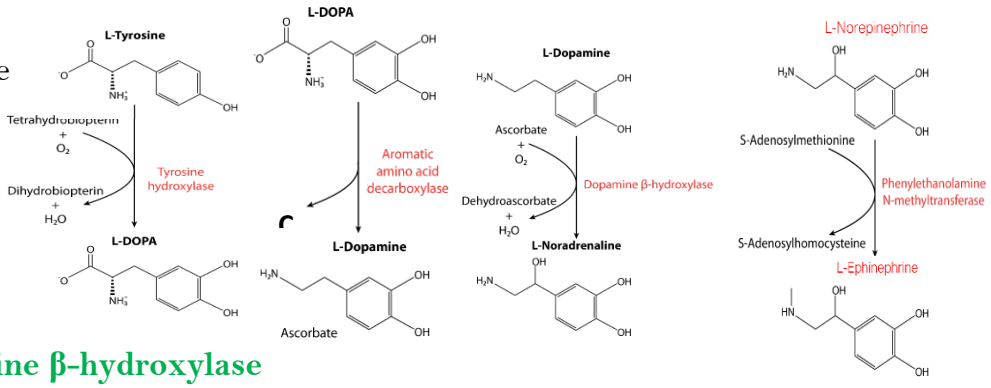
2- Phenylalanine (Phe)

- can convert into tyrosine by an enzyme called **Phenylalanine hydroxylase** (PAH)
- Mutations in this enzyme cause **phenylketonuria** (PKU), a genetic disorder that leads to brain damage and mental retardation if untreated due increase of Phenylalanine
- **NutraSweet:**
  - ➔ the sweetener that use instead of using sucrose to sweet tea or coffee, so some people have diabetes use it and adding it to the coffee for example instead of the sucrose.
  - ➔ Contains Phenylalanine so that, the people who have deficiency in the Phenylalanine Hydroxylase Enzyme must avoid it because it contains Phenylalanine.
  - ➔ **So, if they take it the** Phenylalanine concentration will be high and this will cause problems

3- Tyrosine (tyr):

- tyr is Not Essential if Phe is present, in which Phe is converted to Tyr **via Phenylalanine Hydroxylase**
- is the precursor molecule for the important neurotransmitters
  - ➔ Catecholamines **belong the following** (L-Dopa, L-Dopamine, Norepinephrine, and Epinephrine)
  - ➔ Thyroid Hormones
  - ➔ Melanin

Synthesis of catecholamines



- by the enzyme **hydroxylase** the tyrosine will converted into L-DOPA
- then L-DOPA will convert into L-dopamine by **decarboxylase**
- then L-dopamine will convert into norepinephrine by **dopamine β-hydroxylase**
- then norepinephrine will convert into epinephrine by **phenylethanolamine N-methyltransferase**

Physiological aspects of the Catecholamines

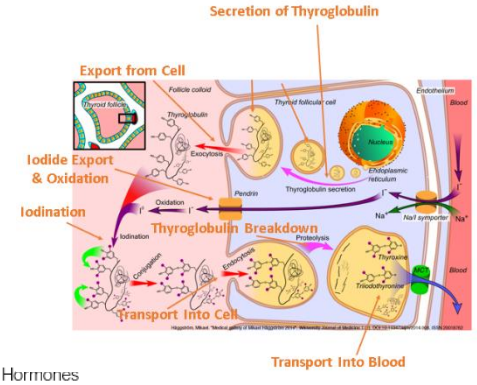
الدكتور حكى مش مهمات بس اعرفوا الأشياء الي فوق ما رح يتطرق لهاي الأشياء ان شاء الله

1- L-Dopa
- Precursor to Dopamine
- Crosses Blood-Brain Barrier
- Used to Treat Parkinson's Disease

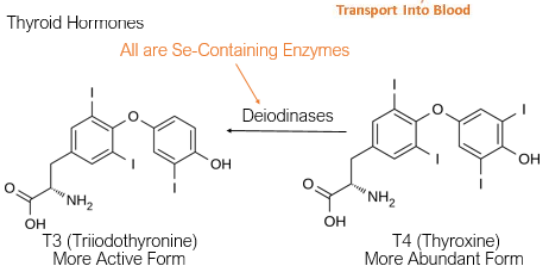
2- Dopamine
- Neurotransmitter
- Inhibits Norepinephrine Release in Blood Vessels - Acts as Vasodilator
- Reduces Insulin Production in Pancreas
- Deficiency Causes Parkinson's Disease
- Links to Schizophrenia and ADHD

3- Norepinephrine
- Hormone and Neurotransmitter
- Works Through Noradrenergic Receptors
- Fight or Flight Response
- Increases Heart Rate and Blood Pressure

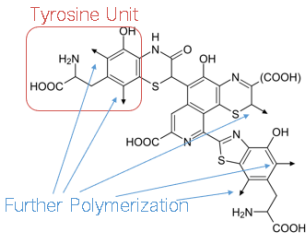
4- Epinephrine (Adrenalin)
- Hormone
- Actions Similar to Norepinephrine
- Fight or Flight Response
- Increases Heart Rate and Blood Pressure



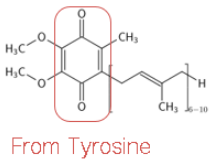
Thyroid hormones
- tyrosine is precursor of the thyroid hormone.
- The iodine is added to the tyrosine which is T4 (thyroxine) it contains 4 iodine.
- **Deiodinases** (which is selenium-containing enzymes) removes one of the iodine will be removed to be converted to T3 that has 3 iodine.



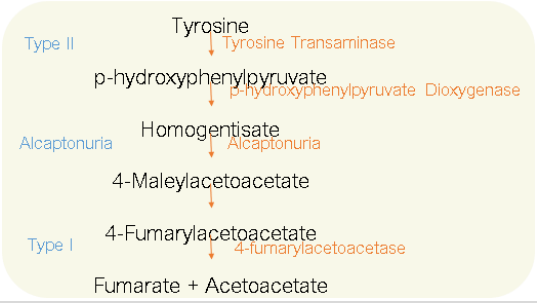
Melanin
- For skin pigmentation
- come from the Oxidized and Polymerized of oxidized Tyrosine.



- Benzoquinone Portion of Coenzyme Q come from tyrosine

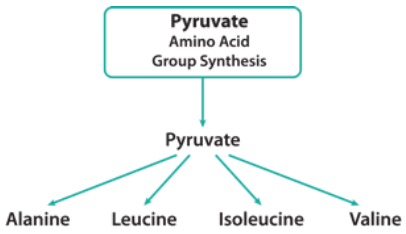


Tyrosine Catabolism diseases
- in the tyrosine catabolism there will be enzyme deficiencies that will cause different types of amino acid inborn diseases.
- Results in Tyrosinemia (accumulation of tyrosine in blood), types:
  - Type I, deficiency in 4-fumarylacetoacetase
  - Type II, deficiency in tyrosine transaminase
  - Type III



➔ Alcaptonuria (Black Urine Disease), deficiency in alkaptonuria)

- Treatments
  ➔ Restricted TYR/PHE Diet
  ➔ Liver Transplant



Pyruvate Family

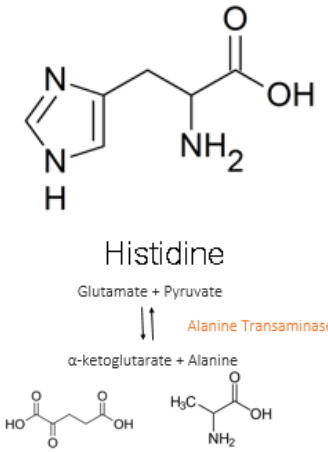
- Includes alanine and other 3 amino acids which are called branched chain amino acids that are (leucine, isoleucine, valine).

Alanine Metabolism

- Alanine is Produced from Pyruvate via Transamination reaction, so pyruvate react with glutamate to produce the α-ketoglutarate and alanine.
- Also it is a by product of Catabolism of Valine, Leucine, and Isoleucine

Histidine (His) Metabolism:

- its metabolism overlaps with Nucleotide Metabolism forming the Ribose-5-Phosphate & PRPP (phosphoribosyl pyrophosphate)
- Ribose-5-Phosphate is a common intermediate in the histidine metabolism and Nucleotide Metabolism.
- It is the most complex of All the Amino Acids
- 10 Steps in its synthesis pathway
- The Second Enzyme of this Pathway **(ATP-phosphoribosyltransferase)** inhibited by Histidine (Feedback Inhibittion)



By end of this lecture you should remember the following:

1- The six classes of the biosynthetic pathways of amino acid.
2- The precursor of each group
3- know how some amino acids that are mentioned are synthesized from the parent compound
4- know about some general features that some amino acids

Details about glucogenic vs ketogenic categories

1- Glucogenic

- Broken Down to Glycolysis/Gluconeogenesis Intermediates
- Includes (again) Alanine, Cysteine, Glycine, Serine Asparagine, Aspartate, Arginine Histidine, Proline, Glutamine Glutamate, Methionine, Valine

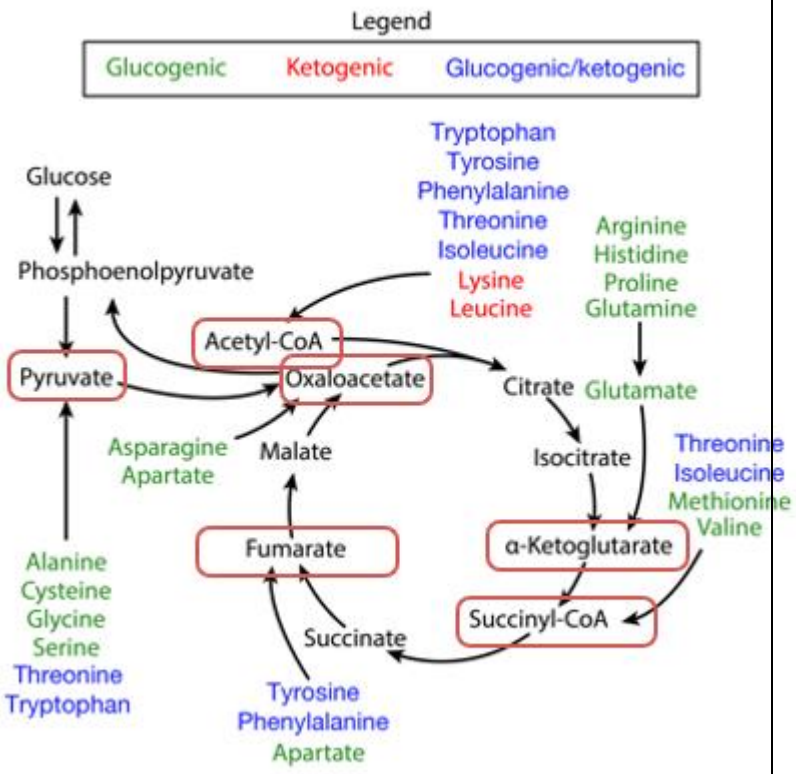
2- Ketogenic

- Broken Down to Acetyl-CoA
- Includes: Lysine, Leucine

3- Both

- Makes Intermediates in Both Pathways
- Includes: Threonine, Tryptophan, Tyrosine, Phenylalanine, Isoleucine

- This figure represents the breakdown of amino acids and which they enter the TCA cycle.
  - ➔ So, they could feed the TCA cycle when it is required.
  - ➔ Some amino acids feed α-ketoglutarate via glutamate these amino acids are Arginine, histidine, proline, and glutamine.
  - ➔ Threonine, isoleucine, methionine, and valine will convert into succinyl-CoA to feed the TCA cycle.
  - ➔ Tyrosine, phenylalanine, and Aspartate will convert into fumarate to feed the TCA cycle.



- Those amino acids that feed into pyruvate or glucose may be called **glucogenic amino acids**.

- So that the amino acids in the green will feed into glucose or intermediate to synthesis of glucose (so that we called them glucogenic).

- The amino acids that labeled in red are called ketogenic amino acids because they will provide intermediate that cannot be used for synthesis of glucose like Acetyl-CoA.

- So that the breakdown of the lysin and leucine will lead to Acetyl-CoA that cannot be used for synthesis of glucose so that it called ketogenic.

- And the amino acids that labeled with blue are considered as glucogenic and ketogenic.
- It is important to memorize the amino acids in each category.

Done by: Qutaibah Essam & Mohammad alomari

The END