Vitamins

- Water Soluble

8 B Complex & V_c

Active form

→ B_1 (Thiamine) ⟶ TPP (Thiamine Pyrophosphate)

→ B_2 (Riboflavin) ⟶ FAD, FMN (To memorise "FADy is FMNist" xD)

→ B_3 (niacin) ⟶ NAD, NADP

→ B_5 (Panthotenic acid) ⟶ CO-A

→ B_6 (Pyridoxine) ⟶ Pyrodoxal Phosphate

→ B_7 (Biotin) ⟶ Biotin lycine Complex

→ B_9 (Folic acid) ⟶ tetra hydropholate

→ B_{12} (Cobalamin)

→ V_c (ascorpic acid)

- Fat Soluble

→ A, D, E, K

⟹ All of these Vitamins are in need to be Converted into the active form, they will work as a Coenzymes.

■ Most of Vitamins are absorbed in the first part of intistines "Dudenum" exept of VB_c (Jujenum) VB_{12} (illum)

■ Water Soluble Vitamines aren't stored in the liver, exept of V_{B12}.

"It's rare to have a water Soluble Vitamin toxicity"

Thiamin (B_1)

□ Pyrimidine joined to a thiazole By a Methglene Bridge.

1.5 → mg/day for adults

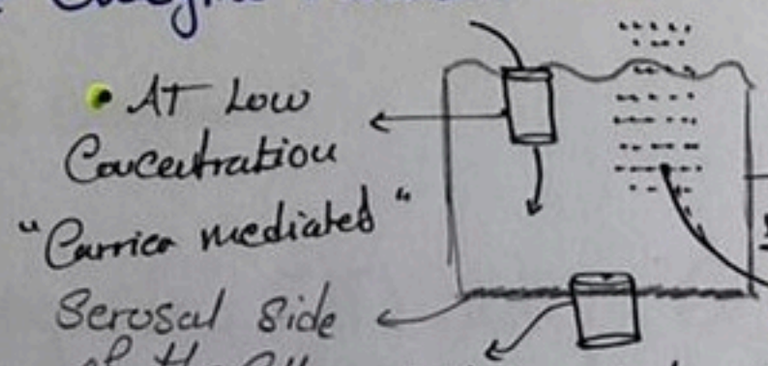
(For a pregnant women, or a person with high Carbohydrates Couteing diet it will be 2 → 2.5 mg/day)

→ Pyrimidine Participates in building the neucleotides (So, it's a part of DNA forming)

□ Thiamine is eaten as thiamin pyrophosphate

→ Pyrophosphatase enzyme releases the thiamine

• The active form is TPP (Thiamine Tyrophosplate)



• goes to the blood & binds to the albumin

The main protein in the blood plasma.

→ From the high Concentrated to the less.

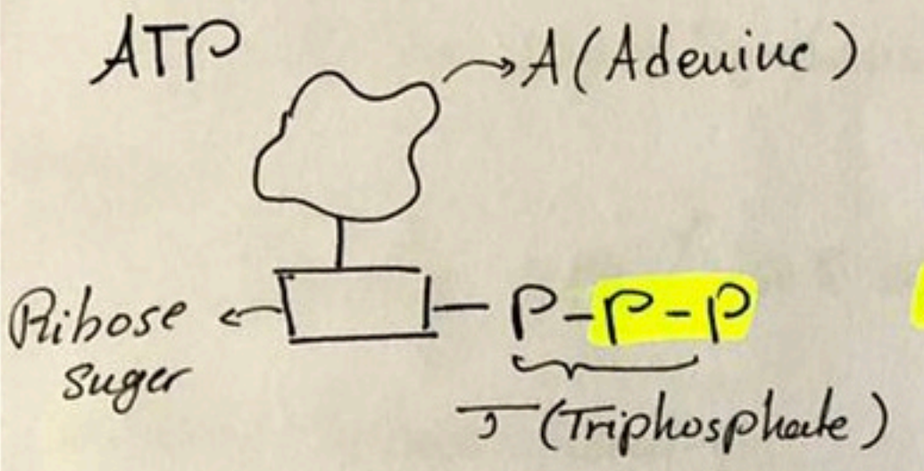
□ After binding of the thiamine to the albumin there should be a cellular uptake.

- It's uptaken by the cells using carriers. which is a thiamine transporter dependent on Na^+

(Human thiamine transporter 1 & 2)

Thiamine is found in muscles, heart, brain, liver & kidneys.

Excreted in urine.



diphosphotransferase enzyme

② (Thiamine Pyrophosphate)

So

Thiamine + ATP → TPP + AMP

These 2 phosphates are added to the thiamine, The ATP is then converted into AMP (Adenocine Monophosphate.)

□ note that the phosphate groups are going to be added to the thiazole ring

Suger → Pyruvate

Pyruvate dehydrogenase Complex enzyme

Acetyl CO-A

↓

Kerbs / Centric acid cycle

↓

ETC

"electric transport chain"

"oxidative phosphorelation"

"Adding 2 phosphate groups to the AMP to form ATP again"

-Energy-

□ This needs 5 Co enzymes

(TPP, lipoamide, FAD, NAD, Co_A SH)

(Thiamine) (lipoic acid) (Riboflavin) (Niacin) (Pantothenic acid)

B_1 B_2 B_3 B_5

- Functions
  - TPP (Serves as a Coenzyme that transfers an activated aldehyde C=O in the following reactions)
    - Oxidative decarboxylation
      α-Keto acids into a Co-A-enzyme containing Compounds
    - Trans Ketolase reaction (Pentose phosphate pathway (PPP))
      turns glucose into ribose & deoxyribose
      (Pentose sugar) → (Components of DNA)
    - Acetyl Choline Synthesis:
      → neurotransmitter
      & For mylein Synthesis also

(Thiamine) B1
- Can be used as a
  - Heart Failure Therapy
    increasing the ATP production
  - Alzheimer disease therapy
    Since it have a role in nerve function.

- Produces energy from Carbohydrates
  (The precourse reaction of Pyruvate)
- Nerve function
  (Acetylcholine & mylein)
- Muscle function
  (related to nerve impulses)
- Apetite & growth
  (Brain)

- There are 3 major - Causes of all Vitamins deficiency
  - Low intake
  - high need
  - Malabsorption

For thiamine - defective Phosphorylation - is added "not turned to the active form"

- Alcoholism: Thiamine deficiency

- Antithiamine factors:
  Enzymes present in some microorganisms & shell fish that cause cleavage of thiazole & Pyrimidine rings "They're called thiaminases"

- Plant thiamine antagonists:
  Caffeic acid, Tannic acid in Coffee & Tea are heat-stable (won't be effected by heat)
  they oxidise thiazole rings, thus, they will be unable for absorption

- Excessive loss (diuretics)

- Manifestation of Thiamine deficiency -

■ Mild

- Gastrointestinal Complaints
- Weakness, due to the reduced ATP production

■ Moderate

- Wernicke Korsakoff Syndrome
- Peripheral neuropathy (ataxia)
- Mental abnormalities (impaired memory)
- Vision Problems

(Related to Alcholism)

■ Severe

(Beri Beri)

Dry:	wet
• Weakness in the muscle +	• Dry Symp. +
• Peripheral Neuropathy +	• loss of Mylein sheath that Causes Peripheral edema
• Memory loss	

& Wernicke Korsakoff Syndrome.

Riboflavin (B_2)

- (Flavin ring + 1)-Ribotol)

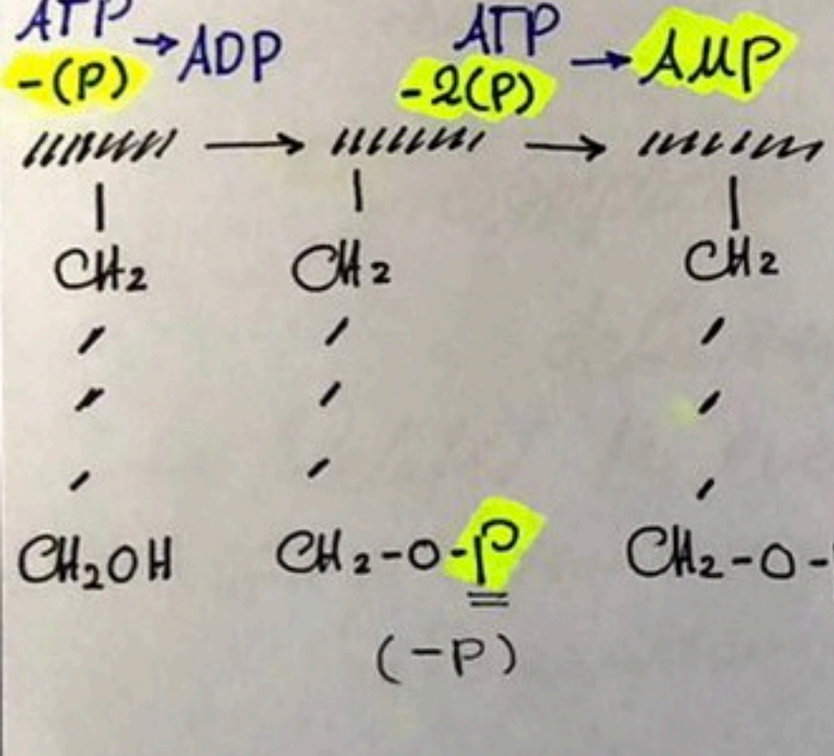
= Ribose + 2H

□ Coenzyme forms of B_2

- FAD & FMN

(Remember FADy is FMNist) XD

- How are FAD & FMN Formed?



FMN	FAD
→ Flavin mononucleotide	→ Flavin adenine dinucleotide
→ Formed by ATP Phosphorolation of "RF"	
- Since there is ATP Kinase enzyme is needed -	

□ Intestinal phosphatase

Breakes down the phosphate groups to give the free riboflavin (RF) form

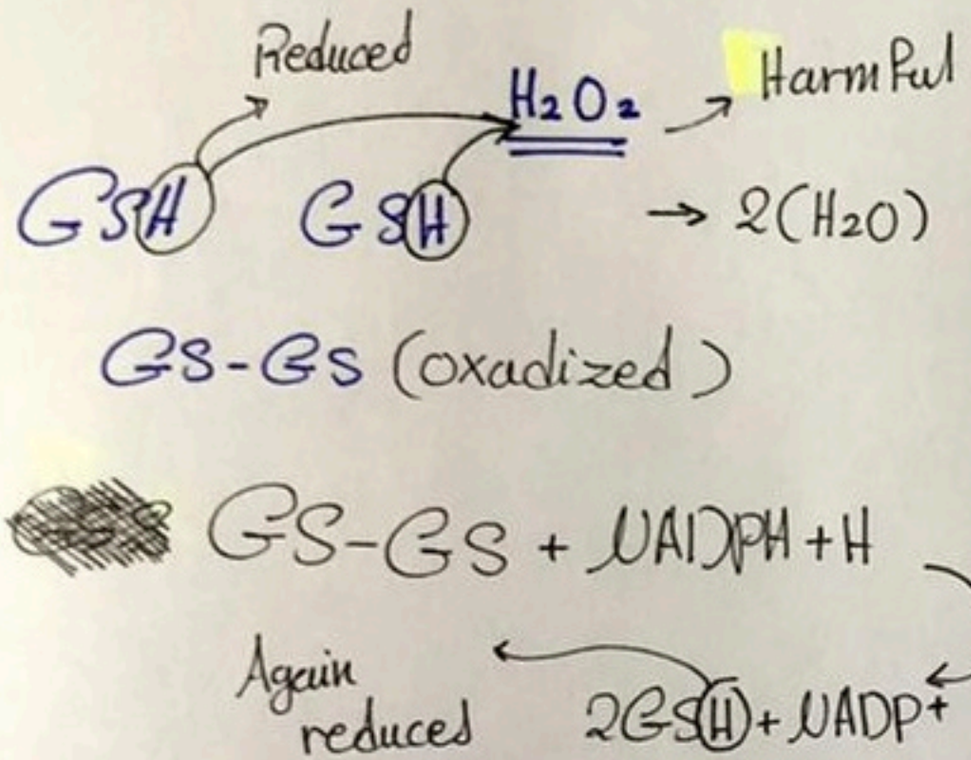
Absorption

- As we mentioned, phosphatase breaks down the Complex form of FMN & FAD by hydrolizing.

It goes to the blood stream through a specific Carrier mediated

- Riboflavin transporter 1&2 are in the intestines
- " " 3 is more brain specific

Then it binds to the albumin or globulin in the blood stream



Functions

- Involved in energy metabolism.
  - → oxidative decarboxilation
  - → Citric acid Cycle
  - → Electron transport

  } Together
  - → β oxidation of fatty acids
- Antioxidant glutathione reductase

Symptoms of deficiency

→ Related to energy production

- Skin & mucous membrane inflammation.
- Glossitis (severe) & angular stomatitis
- Keratitis, Dermatitis
- Cheilosis (Cracked & red lips)
- Ocular manifistation (Vascularization (Growig Blood vessels) of Cornea)

- The Chemical structure is fluorecent, light sensitive & heat stable.
- The active form is FAD, FMN
- Reactions that require FAD
  - → oxidative decarboxilation of Keto acids PDH → ATP
  - → C.A.C Citric acid Cycle → ATP
  - → β oxidation of fatty acids → ATP

$$\underset{FAD}{FMN} + 2H \longrightarrow \underset{FADH_2}{FMNH_2}$$