ISHITA KANODIA

vitamins

Vitamin A

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- collective term for 3 compounds
  retinal, retinol, retinoic acid
  Retinoids

- cyclohexenyl/β-ionone ring with a polyisoprene side chain

- Retinal = CHO Retinol = CH_2OH
  Retinoic acid = COOH

Sources: Fish liver oils, green leafy vegetables, yellow veg. & fruits (mango, carrot)

RDA:

Adult	= 600 µg retinol	= 2400 µg β-Carotene
Pregnant Woman	= 600 µg "	= 2400 " "
Lactation	= 950 µg "	= 3800 " "
Infants	= 350 µg "	= 1400 " "
Children	= 400-600 µg	= 1600-2400 " "

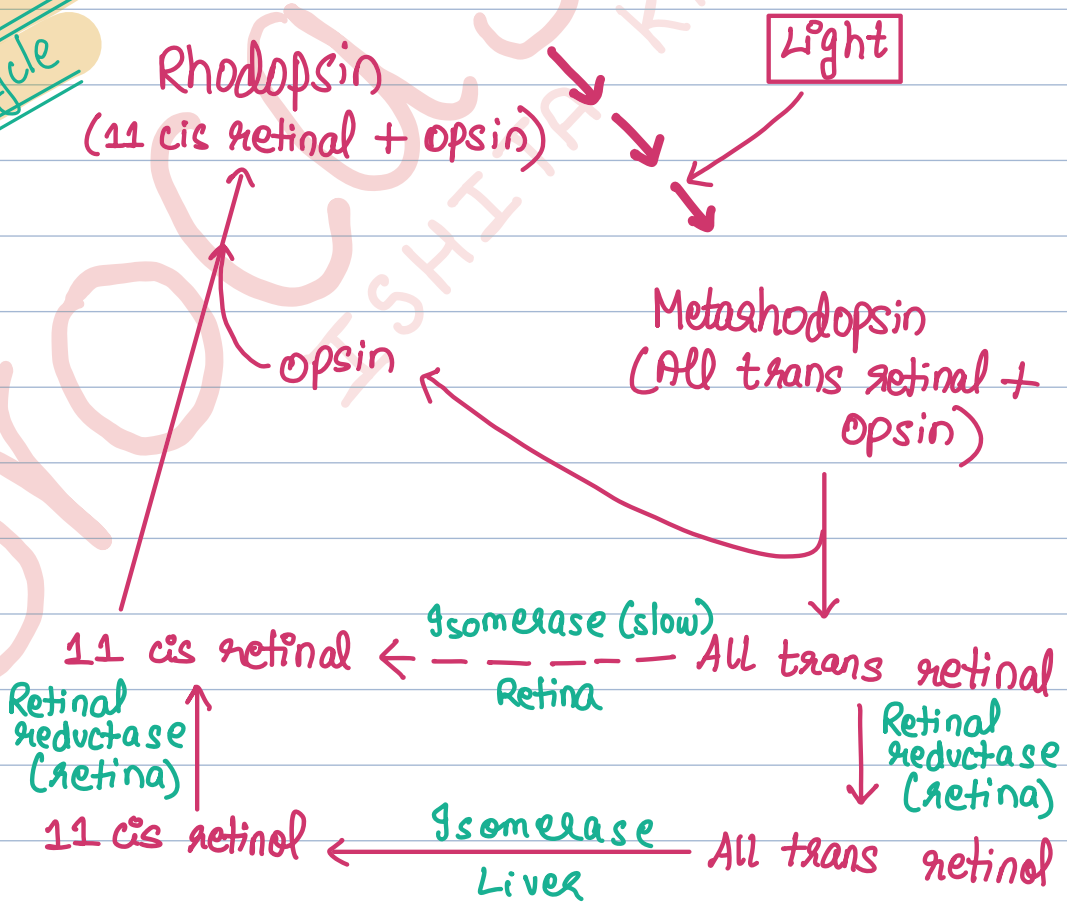
Provitamins: Carotenes (Plants contain different carotenes (α, β, γ...)) β-carotene is most imp.
- converted to vit. A in the liver
- one β-carotene molecule consists of 2 molecules of vit. A

→ digested by pancreatic hydrolases
→ absorbed in small intestine along with fats with the help of bile salts
→ in intestine, incorporated into chylomicron & transported to liver through lymph
→ stored here as retinol palmitate
→ transported to other tissues by retinol binding protein (RBP)

Functions: Retinal for vision
Retinol for reproduction
Retinoic acid for maintaing Normal epithelium.

- Rhodopsin in rod cells (dim light vision) & photopsin in cone cells (bright light & colour vision) both contain vitamin A (11 cis retinal)
- Retinol promotes the synthesis of transferrin & thus, helps in iron transport & hence, Hb synthesis
- Retinoic acid for normal differentiation & mucous secretion & for preventing keratinization of epithelial tissues
- Retinoic acid for growth (esp. skeletal)
- β-carotene has anti-carcinogenic effect by functioning as anti-oxidants to reduce the risk of cancers caused by free radicals.

Wald visual cycle / Rhodopsin cycle / Vitamin A cycle



Deficiency: Night blindness / Nyctalopia

- Xerophthalmia (dryness of eye) due to keratinisation in cornea & conjuctiva
- Corneal dryness (corneal xerosis) & conjunctival dryness (conjunctival xerosis)
- white triangular patches in conjuctiva called Bitot's spots
- Keratomalacia – if untreated ⇒ corneal ulceration & degeneration
- Renal failure due to keratinization in renal tract
- Respiratory infections due to " in respiratory "
- Reproductive failure; growth retardation
- Microcytic anemia since vit. A is required for iron transport & Hb synthesis.

Hypervitaminosis A: excess of vit. A
(excess of β-karotene is not toxic)

Symptoms:
- hepatomegaly
- Skeletal deformities
- Anorexia
- Irritability
- Dermatitis
- Headache
- Drowsiness
- Peeling of Skin

(SHIP ADHD)

Vitamin D
(sunshine vitamin)

- Two forms — D_2 (ergocalciferol)
  D_3 - cholecalciferol

Sources: D_2 → plants
D_3 → fish liver oils

Endogenously:
7-dehydrocholesterol → (UV rays → skin) → Cholecalciferol
Ergosterol → Ergocalciferol

RDA: Adults = 10 µg (400 I.U.) of D_3
Pregnancy, lactation = 15 µg

Provitamin: 7-dehydrocholesterol
Ergosterol

→ absorbed in small intestine along with fats with the help of bile salts
→ in intestine, incorporated into chylomicron & transported to liver through lymph
→ stored in liver & other tissues
→ transported to other tissues by vit. D binding protein (DBP - a plasma α_2 globulin)

- Vit. D is not itself biologically active
- has to be converted to Calcitriol (1,25-dihydroxy cholecalciferol)

Cholecalciferol
↓ 25 hydroxylase (liver)
25-hydroxy cholecalciferol
↓ 1-hydroxylase (kidney, bone, etc.)
1,25-dihydroxy cholecalciferol
(calcitriol)

Functions: (of calcitriol)

→ proper mineralisation of bone
→ increases the number & activity of osteoblasts
→ in osteoblasts, it stimulates Ca uptake & deposition
→ Bone matrix & collagen has Ca & phosphate
→ hypercalcemic hormone
→ promotes the reabsorption of Ca & phosphate by renal tubules, thus reducing excretion of Ca & phosphate.
→ increases the intestinal absorption of Ca & PO_4^{3-} & thus, ↑ blood Ca & PO_4^{3-} level.
→ Action: binds to a receptor in the cytosol & this complex is then transported to the nucleus, where it acts on DNA to stimulate the synthesis of calcium binding protein (CBP) which increases calcium absorption
→ promotes bone resorption & calcium mobilization to raise serum Ca & phosphate levels (during Ca deficiency state)

Deficiency: Rickets in children:
- disease of growing bone
- insufficient mineralization of new bones
- bones become soft & pliable (easily bent)
- bowlegs
- knock-knees
- Pigeon chest
- Rickety rosary (beaded appearance of ribs)

Osteomalacia in Adults:

- Insufficient mineralisation of bones
- Softness of bones
- Bone pain & aches
- Easy fracture of bones

Renal Rickets (Vit. D Resistant Rickets):

→ seen in patients with chronic renal failure
→ kidney is required for formation of calcitriol
→ in renal failure, non-production of calcitriol leads to poor bone mineralisation & rickets
→ does not respond to vit. D supplementation
∴ vit. D resistant rickets
→ responds to provision of calcitriol.

Hypervitaminosis D: → demineralisation of bones

→ hypercalcemia
→ calcification of soft tissues (especially renal tissues leading to renal stones)
→ loss of weight, weakness
→ polyuria, polydipsia

Vitamin E

Chemical Name: tocopherol (8 tocopherols have been identified)
α-tocopherol is the most active ; presence of tocol ring system

Sources: vegetable oils
[fish liver oils are deficient in Vit. E]
- vit. E requirement is proportional to PUFA intake as vit. E protects PUFA from oxidative damage
So, more PUFA intake requires more vit. E.

RDA: 8 mg (12 IU) to 12 mg (18 IU)

→ absorbed in small intestine along with fats with the help of bile salts
→ in intestine, incorporated into chylomicron & transported to adipose tissues & muscles
→ stored in adipose tissues, muscle & liver

Functions: → prevents peroxidative damage of PUFA of cell membranes caused by free radicals which destabilize the integrity of cell membranes (mainly in RBCs); vit. E is a powerful antioxidant which destroys the free radicals.
→ Selenium also has antioxidant property since Selenium is an integral part of glutathione peroxidase enzyme which destroys free radicals & protects RBC;
→ Selenium & Vit. E work synergistically to decrease each other's requirement. Thus, vit. E has selenium sparing action & vice versa.

Deficiency: Rare
→ may lead to anemia due to increased peroxidative damage of RBCs (hemolytic anemia)

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Vitamin K

Anti-hemorrhagic Vit.

Chemistry: Naphthoquinone derivative compound

Source: Green leafy vegetables; also produced by intestinal bacteria

RDA: 50 - 100 µg (if synthesized by intestinal bacteria)
1-2 mg (if not synthesized by intestinal bacteria)

- absorbed in small intestine along with fats with the help of bile salts
- transported to liver by chylomicrons
- mainly stored in liver
- transported to other tissues by LDL

Function: Helps in coagulation process
→ required for synthesis of active form of some blood clotting factors (2, 7, 9, 10)
→ all these factors are synthesized in inactive forms in the liver
→ converted to active forms by γ-carboxylase enzyme (by γ-carboxylation of some glutamic acid residues); vit. K is a co-factor of this enzyme.

Dicumarol & Warfarin are antivitamins of vit. K
- they are structural analogues of vit. K & competetively inhibit γ-carboxylation

∴ Warfarin is used in the treatment of thrombotic diseases.

Deficiency: Hemorrhage
→ internal bleeding
→ prolonged prothrombin time
→ delayed clotting time & bleeding time

- also seen in cholestasis (due to block of bile salts transport to intestine which is required for vit. K absorption) & prolonged antibiotic therapy (as it kills the intestinal flora which produces vit. K)

Vitamin C

Ascorbic acid → has reducing property

Sources: Citrus fruits, green leafy vegetables

RDA: Adults = 40 mg
Pregnancy & lactation = 80 mg

Functions: → required for collagen formation by conversion of inactive protocollagen to active collagen; vit. C is a cofactor of lysyl hydroxylase & prolyl hydroxylase enzymes which convert certain lysine & proline residues of protocollagen to hydroxylysine & hydroxyproline which are essential for cross-linking & tensile strength of mature collagen fibres

∴ vit. C is supplemented along with proteins in post-operative patients to facilitate wound healing & tissue repair.

→ found in large amounts in adrenal cortex ∵ required for synthesis of steroid hormones

→ Norepinephrine synthesis

Dopamine $\xrightarrow[Cu^{+2},\ \text{vitamin C}]{\text{Dopamine } \beta\text{-hydroxylase}}$ Norepinephrine

→ Bile acid synthesis

Cholesterol $\xrightarrow[\text{vit. C}]{7\alpha \text{ hydroxylase}}$ 7α-hydroxy cholesterol → → → Bile salts

→ Degradation of tyrosine

p-Hydroxyphenyl pyruvate $\xrightarrow[\text{oxidase}]{\text{p-hydroxyphenyl pyruvate}\quad Cu^{2+},\ \text{vit. C}}$ Homogentisate

→ vit. C is a natural antioxidant which reduces the formation of nitrosamine (a carcinogenic oxidant) in the intestine

→ reduces the risk of cataract formation

→ facilitates the reconversion of Methemoglobin to Hb for Hb regeneration.

→ facilitates iron absorption by reducing Fe^{+3} to Fe^{+2}

∴ vit. C is required for iron metabolism & erythropoiesis.

→ in high doses, appears to decrease the severity & duration of common cold.

Deficiency: Scurvy → due to defective collagen synthesis

- sore, spongy, swollen & bleeding gums; loose painful teeth
- fragile capillaries ∴ tendency to bleed under slight pressure
- hemorrhage (due to excessive bleeding)

→ microcytic anemia
→ delayed wound healing
→ Aching muscles
→ weakness
→ Swollen joints
→ Bone fragility, easy fractures

Vitamin B1
Thiamine

Chemistry: substituted thiazole ring & substituted pyrimidine ring joined by methylene bridge.
Sulphur containing vitamin

Sources: Dried yeast, wheat germ, unpolished whole grains of rice

→ Thiamine is present in the aleurone Layer (bran) of cereals
→ so, refining of cereal grains in mills destroys thiamine during polishing; but in parboiled (boiling of paddy with husk), the thiamine is not lost
→ It is lost during washing of cereals
→ Boiling the rice in excess water & discarding the water kanjee also results in loss of B1.
→ Destroyed during baking (cooking with baking soda).

RDA: Adult male = 1 - 1.6 mg
Adult female = 0.9 - 1.2 mg
Pregnancy & lactation = upto 2 mg
→ Thiamine requirement is dependent on calorie (carbs.) intake
∵ TPP, a coenzyme of thiamine, plays a central role in carb. metabolism (coenzyme for PDH, αKG dehydrogenase, transketolase)
→ 0.5 mg thiamine is required for every 1000 Kcal.

Functions: coenzyme = TPP (thiamine pyrophosphate)

TPP as a coenzyme:

(i) α-keto-glutarate $\xrightarrow[\text{TPP, FAD, NAD, CoASH, Lipoic acid}]{\text{α-ketoglutarate dehydrogenase}}$ succinyl CoA + CO_2

(ii) PDH complex

(iii) Xylulose-5-phosphate + Ribose-5-phosphate $\xrightarrow[\text{TPP}]{\text{transketolase}}$ Sedoheptulose-7-phosphate + Glyceraldehyde-3-phosphate

(iv) Nerve impulse transmission

→ Brain derives all its energy from carbs. & utilization of carbs requires thiamine.

→ Thiamine is essential for normal functioning of heart muscles

→ for normal maintenance of GIT; maintains good appetite & normal digestion.

Deficiency: Beri-beri (4 types)

(I) Dry Beriberi:

→ neurological manifestations

→ peripheral neuritis with burning & tingling sensation in legs & feet

→ Anorexia (loss of appetite)

→ Muscle wasting; loss of body weight

→ No edema

(II) Wet Beriberi:

→ all symptoms of dry beri-beri

+

edema & cardiovascular manifestations

(enlargement of heart, palpitation, tachycardia, death)

(III) Infantile Beriberi:

→ seen in children (b/w 2-5 months) born to thiamine deficient mothers [Breast milk of mothers has low thiamine content & mother's milk is the only source of thiamine for infants]

→ Restlessness; sleeplessness; convulsions; vomiting

→ Loud piercing cry that changes into a thin weak & almost inaudible voice

→ Cyanosis, dyspnoea, tachycardia

(IV) Wernicke - Korsakoff Syndrome / Wernicke's Encephalopathy / Cerebral Beriberi:

→ seen in chronic alcoholics since they generally consume less food & derive energy from alcohol which leads to insufficient provision of thiamine

→ Alcohol also inhibits thiamine absorption.

→ Peripheral & central neurological defects – severe loss of memory, nystagmus, insomnia, cerebellar ataxia, abnormal gait, mental confusion, psychosis, depression, etc.

Vitamin B2
Riboflavin

Chemistry: isoalloxazine ring attached to ribitol

Sources: Dried yeast, milk, meat, eggs

→ even though cereals are relatively poor sources of riboflavin, they provide 75% of riboflavin requirement of the body because they are consumed in large amounts

RDA: Adults = 1.5mg

Pregnancy & lactation = 1.9 mg

Functions: → 2 coenzyme forms
- FMN (flavin mono nucleotide)
- FAD (flavin adenine dinucleotide)

FMN Dependent Enzymes:

(i) L α-ā·ā $\xrightarrow[\text{FMN} \rightarrow \text{FMNH}_2]{\text{L-ā·ā oxidase}}$ α-keto acid + NH_3

(ii) NADH dehydrogenase (complex I of ETC)

(iii) Cytochrome C reductase of ETC

FAD Dependent Enzymes:

(i) D-α ā·ā $\xrightarrow[\text{FAD} \rightarrow \text{FADH}_2]{\text{D-ā·ā oxidase}}$ α-keto acid + NH_3

(ii) Succinate $\xrightarrow[\text{FAD} \rightarrow \text{FADH}_2]{\text{succinate dehydrogenase}}$ fumarate

(iii) α-Ketoglutarate $\xrightarrow[\text{CoASH, FAD, TPP, Lipoic acid, NAD} \rightarrow \text{NADH} + \text{H}^+,\ \text{CO}_2]{\text{α-Ketoglutarate dehydrogenase}}$ Succinyl CoA

(iv) Acyl co A dehydrogenase (of beta oxidation of fatty acids)

(v) PDH.

Deficiency: Ariboflavinosis

Symptoms:

→ Glossitis (soreness of tongue & magenta coloured tongue)
→ Cheilosis (fissuring of the lips)
→ Angular stomatitis (fissuring at the corners of the mouth)
→ Seborrhic dermatitis (inflammation of skin)
→ Corneal vascularization (reddening of eyes)
→ Photophobia (redness & burning sensation in eyes)

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Vitamin B3
Niacin

Chemistry: Niacin represents nicotinic acid & nicotinamide
↳ consists of pyridine ring

Sources: Peanuts, liver, legumes
→ a.a tryptophan can produce niacin in the body
1 mg niacin ⟹ 60 mg tryptophan.

RDA: Adults = 20 mg
Pregnancy & Lactation = 25 mg
(6.6 mg niacin is required for every 1000 Kcal energy).
→ Total niacin available from food –

$$\text{Niacin equivalent} = \text{Niacin content (mg)} + \frac{\text{tryptophan content (mg)}}{60}$$

Functions: Two coenzymes – NAD^+, $NADP^+$.

NAD^+ Dependent Enzymes:

(i) Glyceraldehyde-3-phosphate $\xrightarrow[NAD^+ \quad NADH + H^+]{\text{glyceraldehyde-3-P dehydrogenase}}$ 1,3-bisphosphoglycerate

(ii) Malate dehydrogenase, α-KG dehydrogenase, PDH

$NADP^+$ Dependent Enzymes:

Glucose-6-phosphate $\xrightarrow[NADP^+ \quad NADPH + H^+]{\text{Glucose-6-phosphate dehydrogenase}}$ 6-phosphogluconate

- $NADH + H^+$ ⟹ 3 ATP in ETC
- $NADPH + H^+$ ⟹ required for biosynthesis of fatty acids, cholesterol, etc.

Deficiency: Pellagra (3Ds: Dermatitis, Dementia, Diarrhea)
If untreated ⟹ Death (4th D)

- **Dermatitis:** erythema of skin is exposed to light
  ↳ Increased pigmentation around the neck ⟹ Casal's necklace.
- **Diarrhea:** inflammation of mucous membrane of GIT
  ↳ Diarrhea may often contain blood & mucous
  → Prolonged diarrhea ⟹ weight loss
- **Dementia:** Neurological symptoms – depression, delirium, irritability, memory loss ataxia, spasticity.

→ Pellagra is common among maize eaters ∵ in maize, niacin is present in bound form & is unavailable for absorption; Also, tryptophan content is very low in maize proteins (zein) & leucine content is high, which depresses the synthesis of niacin.

→ Pellagra like symptoms are seen in –

- hartnups disease
- carcinoid syndrome (due to less availability of tryptophan for niacin coenzyme formation)
- vit. B6 deficiency
- Isoniazid (INH) treatment in tuberculosis (due to failure of PLP formation which is required to convert Tryptophan to niacin coenzymes)

Vitamin B5
Pantothenic acid

Chemistry: consists of pantoic acid & β-alanine held together by amide bond.

Sources: Liver, meat, milk, dried yeast, whole cereals, legumes

RDA: 10 mg

Functions: Coenzyme A (CoASH) - involved in most metabolisms

(i) α-KG $\xrightarrow[\text{TPP, FAD, NAD, CoASH, Lipoic acid}]{\alpha\text{-KG dehydrogenase complex}}$ succinyl CoA + CO_2

(ii) Glycine + Succinyl CoA $\xrightarrow{\text{ALA synthase}}$ ALA $\rightarrow\rightarrow\rightarrow$ Heme

(iii) Fatty acyl CoA $\xrightarrow[\text{FAD} \rightarrow \text{FADH}_2]{\text{Acyl CoA dehydrogenase}}$ α-β-unsaturated acyl CoA

(iv) Acetyl CoA + Acetyl CoA $\xrightarrow{\text{Thiolase}}$ Acetoacetyl CoA + CoASH

(v) Acetyl CoA + CO_2 $\xrightarrow[\text{ATP} \rightarrow \text{ADP} + P_i^\circ]{\text{Acetyl CoA carboxylase; } Mn^{+2}, \text{Biotin}}$ Malonyl CoA

Acyl Carrier Protein (ACP): - component of fatty acid synthase complex

Both CoASH & ACP contain 4-phosphopantetheine (formed from pantothenic acid)

Deficiency: rare (∵ it is widely distributed in foods)

→ Burning foot syndrome in prisoners of war is ascribed to deficiency of pantothenic acid
[There is decreased synthesis of fatty acids.]

Vitamin B6
Pyridine derivatives

Chemistry: Pyridine derivatives – Pyridoxine, Pyridoxal, Pyridoxamine

Sources: dried yeast, rice polishing, wheat germs, legumes, oilseeds

RDA: 2mg (increases in pregnancy & lactation)

→ vit. B6 requirement is dependent on protein intake (∵ PLP is required for transamination, deamination, decarboxylation, in a.a metabolism)

Functions: coenzyme = Pyridoxal phosphate (PLP)

(i) Transamination reactions of a.a

Alanine + α-KG $\xrightarrow[PLP]{\text{Alanine transaminase (ALT)}}$ Pyruvate + Glutamate

(ii) Decarboxylation of a.a

Histidine $\xrightarrow[PLP]{\text{Decarboxylase}}$ Histamine

(iii) Non-oxidative deamination

Cysteine $\xrightarrow[PLP]{\text{cysteine desulphydratase}}$ Pyruvate + NH_3 + H_2S

(iv) Glycine synthase

$CO_2 + NH_4^+ + NADH + H^+ \xrightarrow{\text{Glycine synthase}}$ Glycine + NAD^+

(N^5N^{10} methylene FH_4 —PLP→ FH_4)

(v) ALA synthase

Glycine + Succinyl CoA $\xrightarrow[PLP]{\text{ALA synthase}}$ ALA → → → → Heme

(vi) Muscle glycogen phosphorylase requires PLP

(vii) Required for synthesis of sphingolipids & formation of myelin.

Deficiency: Neurological symptoms – depression, irritability, peripheral neuritis (due to decreased neurotransmitter synthesis like GABA, serotonin, catecholamines, & myelin formation failure)

→ Microcytic anemia due to failure of heme synthesis

→ Pellagra like symptoms ∵ PLP is needed for niacin coenzyme synthesis from tryptophan

→ Homocysteinuria ∵ conversion of homocysteine requires PLA

Isoniazid (INH), an antituberculosis drug, inhibits pyruvate kinase required for PLP formation from vit. B6 & causes vit. B6 deficiency (pellagra like symptoms).

Vitamin B7
Biotin

Chemistry: Imidazole derivative
↳ sulphur containing vitamin

Sources: Yeast, liver, legumes, egg, milk

RDA: 20-30 µg

Functions: Coenzyme form of biotin is biotin itself

$$\text{Acetyl CoA} + CO_2 \xrightarrow[\text{ATP}]{\text{Acetyl CoA carboxylase; } Mn^{+2}, \text{Biotin}} \text{Malonyl CoA} + ADP + P_i$$

Deficiency: Rare (∵ widely distributed in foods & synthesized by intestinal flora)
→ Deficiency symptoms – anorexia, depression, insomnia muscle pain, dermatitis, etc.
[High consumption of raw eggs can also lead to biotin deficiency]

Egg White Injury: eggs contain a glycoprotein called avidin (antivitamin of biotin)
→ it combines very tightly with biotin & prevents its absorption, thus inducing biotin deficiency.
→ caused due to consumption of raw eggs.
→ Heating denatures avidin (as avidin is a protein), eliminating its biotin binding capacity.
→ About 20 raw eggs per day would be required to induce biotin deficiency.
(Inclusion of an occasional raw egg in diet does not cause biotin deficiency).

Vitamin B9
Folic acid

Chemistry: consists of 3 components – Pteridine ring, PABA & glutamic acid

Sources: Green leafy vegetables, yeast, liver, eggs

RDA: 200 µg (increases during pregnancy & lactation)

Functions: Coenzyme – tetrahydrofolate (THF or FH_4)

→ **Required for one-carbon metabolism:**

FH_4 acts as a carrier of one-carbon compounds (eg- methyl, methylene, methenyl, formyl, formimino) & serves as acceptor or donor of these 1-carbon compounds in various reactions.

a) $CO_2 + NH_3$ ⟶ glycine

b) Glycine ⟶ Serine

c) Glycine ⟶ $CO_2 + NH_3$

d) Methionine ⟶ Formyl methionine

e) N^5 methyl FH_4 → Cobalamin → Methionine

FH_4 ← Methyl cobalamin ← Homocysteine

Deficiency: Megaloblastic anemia

→ ∵ folic acid is required for synthesis of DNA required during cell division, deficiency causes impaired cell division

→ Cells which undergo rapid cell division (RBCs, intestinal cells) are particularly more sensitive to folate deficiency.

→ folate deficiency delays DNA synthesis, but Hb synthesis is continued in RBC precursors. Cytoplasm is well developed but not the nucleus, resulting in formation of large & immature RBC called macrocytes/megaloblasts. Abnormal megaloblasts are released into the circulation & are rapidly destroyed in spleen resulting in megaloblastic anemia.

Antagonists: Aminopterin & amethopterin (Methotrexates) are structural analogues of folic acid. They inhibit dihydrofolate reductase enzyme & formation of tetrahydrofolate
∴ they are used as anti-cancer drugs, especially leukemia.

Vitamin B12
cobalamin

Chemistry: corrin ring with a central cobalt atom
→ found as methylcobalamin, adenosylcobalamin, hydroxocobalamin

Sources: not present in vegetables; found only in foods of animal origin; Curd is a good source since lactobacillus can synthesize B12.

RDA: Adults = 1 - 1.5 µg
Pregnancy & lactation = 2 µg

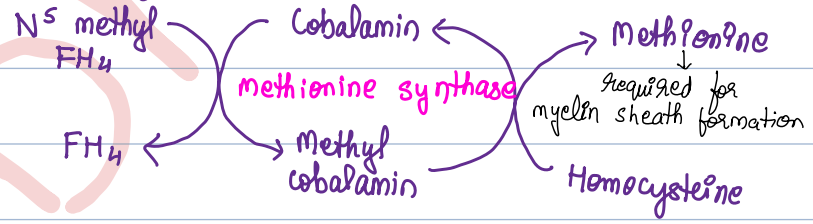
→ **Absorption** of vit. B12 requires mediation of intrinsic factor (IF) secreted by gastric parietal cells. One molecule of IF can bind to 2 molecules of vit. B12
→ The IF-B12 complex gets attached to specific receptors on intestinal mucosal cells & is then internalized; IF is digested here & B_{12} is converted to methylcobalamin
→ **Transported** in the blood bound to transcobalamin-II.

Functions: 2 coenzymes — Deoxyadenosylcobalamin, methyl-cobalamin.

Deoxyadenosylcobalamin:

L-methyl-malonyl CoA —(methylmalonyl CoA mutase / deoxyadenosylcobalamin)→ Succinyl CoA → TCA cycle

Methylcobalamin: Link b/w functions of folic acid & cobalamin.



Deficiency: Pernicious Anemia (megaloblastic anemia with neurological symptoms)
→ mostly caused due to failure of absorption of vit. B12 rather than dietary deficiency.

- Macrocytic Anemia: folate trap → secondary folate deficiency → affects RBC formation
- Neurological Symptoms: due to accumulation of methyl malonyl coA which interferes with myelin formation to cause demyelination of nervous system.

Methylmalonyl aciduria

Homocysteinuria

Cobalamin causes functional folate deficiency (folate trap):

→ cobalamin is required for the conversion N^5-methyl tetrahydrofolate to tetrahydrofolate by methionine synthase enzyme.

→ In cobalamin deficiency, there is failure of conversion of N^5-methyl tetrahydrofolate to tetrahydrofolate & entire body folate is trapped as N^5-methyl tetrahydrofolate. Tetrahydrofolate pool is reduced leading to functional folate deficiency.

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Pregnant Woman = 600 µg " = 2400 " "
Lactation = 950 µg " = 3800 " "
Infants = 350 µg " = 1400 " "
Children = 400 - 600 µg = 1600 - 2400 " "

vitamin - D RDA: Adults = 10 µg (400 I.U.) of D_3
Pregnancy, Lactation = 15 µg

vitamin - E RDA: 8 mg (12 IU) to 12 mg (18 IU)

vitamin - K RDA: 50 - 100 µg (if synthesized by intestinal bacteria)
1 - 2 mg (if not synthesized by intestinal bacteria)

vitamin - C RDA: Adults = 40 mg
Pregnancy & lactation = 80 mg

B_1 RDA: Adult male = 1 - 1.6 mg
Adult female = 0.9 - 1.2 mg

B_2 RDA: Adults = 1.5 mg
Pregnancy & lactation = 1.9 mg

B_3 RDA: Adults = 20 mg
Pregnancy & Lactation = 25 mg

B_5 RDA: 10 mg

B_6 RDA: 2 mg

B_7 RDA: 20 - 30 µg

B_9 RDH: 200 µg

B_{12} RDA: Adults = 1 - 1.5 µg
Pregnancy & lactation = 2 µg