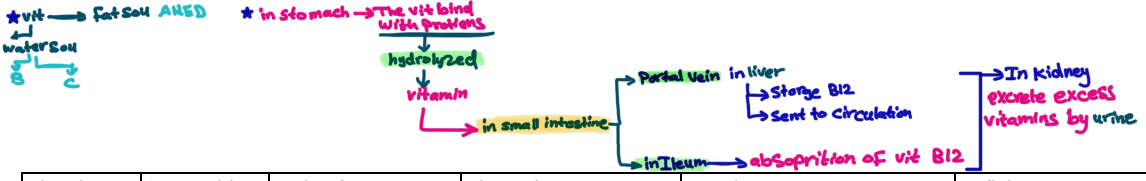
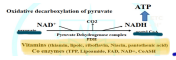


Vitamin	Composition	Active form	Absorption	Function	Deficiency
Vitamin B1 (Thiamin) Anti-beri beri	Pyrimidine + thiazole with methylene bridge → thia: sulfur zole: nitrogen → The tri form of this vit inactive → Requirement → adult 1-1.5 → Pregancy + high carb diet need ↑	Thiamin --> Thiamin pyrophosphate (TPP) using ATP and ATP-dependent thiamin diphosphotransferase → active form Can't absorption 	Low conc. = carrier mediated High conc. = simple diffusion Inhibited by alcohol Serosal side of intestine: Na+ dependent ATPase In serum bound to proteins, mainly albumin 90% in RBCs in blood In cells uptake by Na+ dependent thiamin transporter 1 & 2 (THTR 1 & 2) Basolateral membrane ↓ cell	Activated aldehyde unit in: • Oxidative decarboxylation of α-keto acids • Transketolase reaction (PPP) → ribose sugar, NADP • ACh synthesis Important in: • Producing energy from carbs • Nerve and muscle function • Appetite and growth • Therapy for heart failure and Alzheimer's disease	Caused by: • Low intake, malabsorption, defective phosphorylation to TPP • Antihistamine factors → cleave • Caffeic and tannic acids • Alcoholism • Excessive loss in urine × Storage in: muscle, heart, brain liver, kidneys × Sources: Plant: beans, peas nuts, bran; animals: liver, heard, kidney, milk Leads to: • Mild: gastrointestinal complaints and weakness • Moderate: Wernike korsacoff syndrome, Peripheral neuropathy, Mental abnormalities • Severe: Wernike korsacoff syndrome + Beri beri (dry): atrophy and weakness of muscles, peripheral neuropathy, memory loss, (wet): dry + oedema
Vitamin B2 (Riboflavin)	Flavin ring + D-ribitol (sugar alcohol) × light sensitive × heat stable	FMN and FAD coenzymes FMN: flavin mononucleotide	RFT-1 and RFT-2 in intestine RFT-3 in brain Rf in blood associated with albumin/globulin Source: liver, beef, milk, fish, egg, nuts + Yeast + Green leafy cereals	Involved in ATP production: • Oxidative decarboxylation • Citric acid cycle • Beta-oxidation of fatty acids • Electron transport Associated with anti-oxidant glutathione reductase, reducing GSSG to 2 GSH using FAD and NADPH	Symptoms: • Glossitis and angular stomatitis • Keratitis and dermatitis • Chielosis (cracked red lips) • Ocular manifestations (vascularization of cornea) Deficiency occurs in newborn infants with hyperbilirubinemia who are treated with phototherapy
Vitamin B3 (Niacin) * vit PP * Source → Same B1	Carboxylic acid derivative of pyridine; Formed from tryptophan and vit. B6 Tryptophan → Niacin	NAD+ (for oxidative pathways) and NADP+ (for reductive pathways) coenzymes	---- In intracellular Proteintyrosine kinase-mediated Pathway	NAD+ redox reactions: • Oxidative decarboxylation of keto acids • Beta-oxidation of fatty acids • Citric acid cycle NADP+ redox reactions: • Glucose-6-phosphate dehydrogenase • Folate reductase	Caused by: • Elderly w/ restricted diets • Malabsorption • Maize-dependent population • Vit. B6 deficiency • Hartnup disease (less tryptophan production) • INH (anti-TB) (decreased B6) Clinical use: treatment of hyperlipidemia Deficiency causes pellagra, characterized by four D's: dermatitis, diarrhoea, dementia and death → Symtric!!
Vitamin B5 Pantothenic acid	Pantoic acid + beta-alanine → inactive form	4-phosphopantotheine (gets -SH (thiol) active group) → active form × The source → Same B1	Absorbed through SMVT (sodium-dependent multivitamin transporter) along with biotin → transport B5, B6 × As intake (B5) increase 10-fold ↓ absorption rate decrease to 10%	Formation of CoASH for: • Oxidative decarboxylation of keto acids • Oxidation of fatty acids • Acetylating reactions as acetyl choline Formation of ACP for: • Fatty acid synthesis	Deficiency is rare as it is widespread in food Leads to: • Burning foot syndrome → Severe burning and excessive sweating
Vitamin B6: Pyrodixine	Exists as three: → Alcohol Pyridoxine Pyridoxal → aldehyde Pyridoxamine → amine	Active form is pyrodixal phosphate Pyrodixal + ATP using pyrodixal kinase × Requirements adult → men 2.2 mg/day → women 2 mg/day → Pregancy (2.5) + Lac	---- × The absorption inactive form × all form pyridoxine is converted into Pyridoxal → in mucosal cells × in blood → albumen!! × storage: coenzyme form PLP (active form) liver, brain, kidney, brain Serine PLP dehydratase → Pyruvate + NH3 homocysteine + serein → cysteine glycine PLP → serein glycogen PP → glucose 1-phosphate Glycogen Phosphorylase	Transamination (amino → keto) • Converts amino acids into keto acids • Keto acids enter Krebs cycle and get oxidized to release energy • During transamination, PLP interacts with amino acids to form Schiff base • Amino group is handed PLP forming pyridoxamine phosphate and ketone is liberated Decarboxylation: × vasodilator × allergic reaction • Histidine to histamine • Glutamate to GABA Deamination → another ex: Tryptophan → Serotonin Transsulfation Condensation	Caused by: • Decreased dietary intake • Impaired absorption • Alcoholism • Antivitamins: isoniazid and penicillamine Leads to: • Serotonin, epinephrine, norepinephrine and GABA not produced properly • Deficiency of vit. B3 • Decreased Hb levels Hypervitaminosis (2.5g/day): • Sensory neuropathy: imbalance, numbess, muscle weakness and nerve damage
Vitamin B7 Biotin Anti-egg white factor Vitamin H × Sources: * Rich egg, liver, kidney, yeast pulses, nuts vegetables * Poor cereals, dairy products	Biotin is imidazole derivative formed from imidazole + thiphene ring with valeric side chain	Biotin covalently bonds to ε-amino group of lysine to form biocytin coenzyme → active form × the biocytin bind with lysine → Covalent bon	----	Prosthetic group for carboxylase: • Pyruvate carboxylase (Pyruvate → oxaloacetate) • Acetyl CoA carboxylase → Acetyl CoA → Malonyl CoA • Propionyl carboxylase • β-Methyl crotonyl CoA carboxylase Carboxylation reactions that don't require biotin: (few)	• Deficiency is rare due to wide presence in food • Biotin is synthesized by bacterial flora in gut • Deficiency leads to dermatitis, weakness and nausea, loss hair around the eye • Antagonists: Each molecule of avidin in egg whites binds to 4 molecules of biotin preventing absorption

				• Formation of carbamoyl phosphate in urea cycle • Incorporation of CO_2 in purine synthesis	
Vitamin B9 1. Folic acid 2. Pterorylglutamic acid × Source: + green veg + fruits + yeast + liver × requirements adult 400 mcg/d Pregnant 400–1000 mcg/d	---- Pteridine ring + glutamic acid + Para-amino benzoic acid	Folic acid is reduced by dihydrofolate reductase to tetrahydrofolic acid activating it	----	Coenzyme to: • Utilize amino acids • Produce nucleic acids • Form blood cells in bone marrow • Ensure rapid growth in infancy, adolescence and preganancy • B6,B9,B12 vits. Control homocysteine blood level Tetrahydrofolate (FH4) and one-carbon metabolism): • Sources of one-carbon are serine, glycine, histidine, formaldehyde and formate • Carried by FH4 on its N at positions 5 and/or 10 Use of one-carbon group: • Transfer methyl group to dUMP to dTMP, and thus is required for cell division, thus inhibitors used for cancer chemotherapy • Forming serine from glycine	Deficiency causes: • Decreased supply • Increased requirements • Vigorous iron therapy during pregnancy • Some antimalarials • Methotrexate (folate antagonist) • Malabsorption in jejunum due to intestinal wall diseases • Alcoholism • Deficiency of enzymes required for metabolism Deficiency leads to: • Megaloblastic anemia • Heart disease and some other chronic conditions • Impaired cognitive status • Neural tube defect such as spinal bifida
Vitamin B12 Cobalamin × Source: → liver → fish → seafood → egg → dairy product	Synthesized by micro-organisms in animals and incorporated into its tissues	5-Deoxydenosylcobalamin (active) Methylcobalamin (active) Hydroxycobalamin (inactive) Cyanocobalamin (inactive)	1. Free B12 binds directly to transcobalamin I of salivary and gastric mucosal cells within stomach and will remain bound till reaching duodenum in the small intestine 2. Gastric and intestinal proteases release B12 3. B12 binds to intrinisc factor forming intrinsic factor-B12 complex that binds to ileum receptors to facilitate absorption. 4. Transcobalamin II in blood transports B12; 50% liver, 50% other tissues storage	Cofactor in: • Methylation of homocysteine to methionine for DNA & myelin & neurotransmitters synthesis, as well as brain metabolism and growth • Conversion of L-methylmalonyl CoA to succinyl CoA to join citric acid cycle for producing energy → to tissue	Causes of deficiency: • Dietary deficiency (amount of B12 stored in liver enough for 3-6 years • Using certain medications such as metformin, proton pump inhibitors, histamine H2 blockers • High levels of homocysteine in blood Leads to: • Tingling, numbness in extremeties, nerve damage and memory loss • Dementia, heart disease and stroke
Vitamin C (L-ascorbic acid)		Ascorbate Labile form	Not synthesized or stored in human body	1. Reducing agent in many reactions 2. Synthesis of collagen (hydroxylysine and hydroxyproline) 3. Iron absorption 4. Regeneration of reduced form of vit. E 5. Antioxidant	Deficiency leads to: • Improper formation of connective tissue • Wound healing defects • Scurvy: sore and spongy gums, loose teeth, fragile blood vessels, swollen joints and anemia