



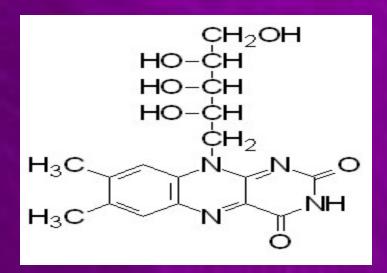


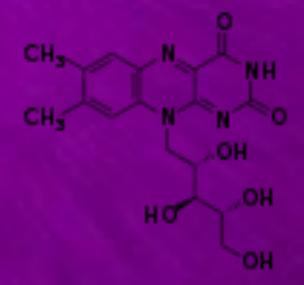


Vitamin B2 (riboflavin)

• <u>Chemistry:</u>

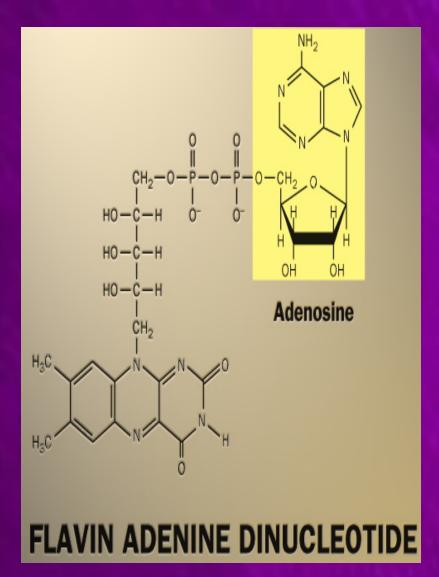
- It is an organic yellow compound formed from:
- Flavin pigment or dimethyle isoalloxazine.
- D-ribitol derived from D-ribose

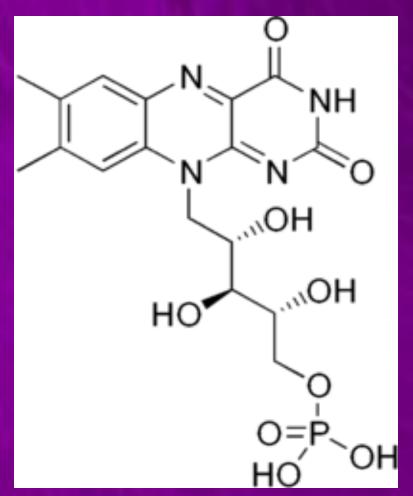




<u>Properties:</u>

- 1. Destroyed by light.
- 2. Alcoholic in nature due to ribitole.
- 3. It undergoses reversible reduction to a colourless substance called lecucoriboflvin.
- 4. Heat stable in neutral or acid solution but not in alkaline solution.
- 5. On exposure to light, the ribityle residue is split off with the formation of a yellow pigment soluble in chloroform which is called :
 - Lumiflavine (in alkaline medium).
 - Lumichrome (in acid or neutral medium).
- 6. riboflavin is present in tissues in two biologically active forms:
- 7. Flavin mononucleotide (FMN).
- 8. Flavin adenine dinucleotide (FAD).





• Flavin mononucleotide

• <u>Sources:</u>

- Plants: yeast, dry beans and nuts. Grean leafy vegetables and fruits and germinating seeds.

– Animals<u>:</u>

• Liver, kidney, eggs and milk.

• <u>Excretion:</u>

• In milk, in sweat, in stool and in urine.

<u>Function</u>

1-It enter in the formation of 2 important coenzymes which are FMN and FAD.

- FMN and FAD are coenzymes for flavoprotein enzymes acting as hydrogen carriers in oxidation and reduction reactions.
- Their active centers include N1 and N10 of flavin ring.

2-It is a growth promoting factor in rats.

Riboflavin (vitamin B2) works with other B vitamins to promote healthy growth and tissue repair, and helps release energy from carbohydrates RDA: 1.7 mg Healthy skin Water-soluble Healthy red blood cell production @ADAM, Inc.

<u>Deficiency</u>

Cheilosis is a dry fissured lips.
 Angular stomatitis is fissuring at the angle of the mouth.
 Inflammation of tongue.
 In the skin: seborrhoic dermatitis.
 Vascularization of cornea, dryness and photophobia.
 Synthesis of protein is impaired.



- <u>Requirements:</u>
 - Adults 1.5-1.8mg/day.
 - Pregnant and lactating female 1.7-2mg/day.Children 0.6mg/day.

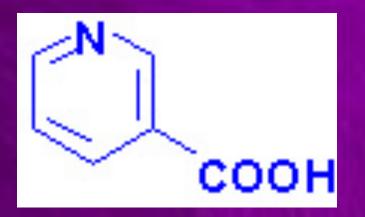




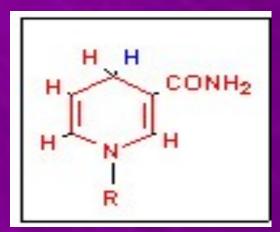
Vitamin B3 (niacin or niacinamide)

• <u>Chemistry:</u>

It is also called Pellagra preventing factor (PP).
It is a pyridine 3-carboxylic acid.



Nicotinic Acid



Nicotinamide

• <u>Properties</u>:

- It is a white crystalline substance.
- It is stable in acid solution but not in alkalies.
- Not destroyed by light.
- It is the most stable member of vitamin B and thermostable.
- It is present in tissues in the form of nicotinamide.
- Nicotinic acid and nicotinamides are slightly soluble in water.

• <u>Sources:</u>

- Plants: yeast, Cereal grains, Some nuts and Legumes.
- Animals: Liver, kidney and meats.

• <u>Absorption:</u>

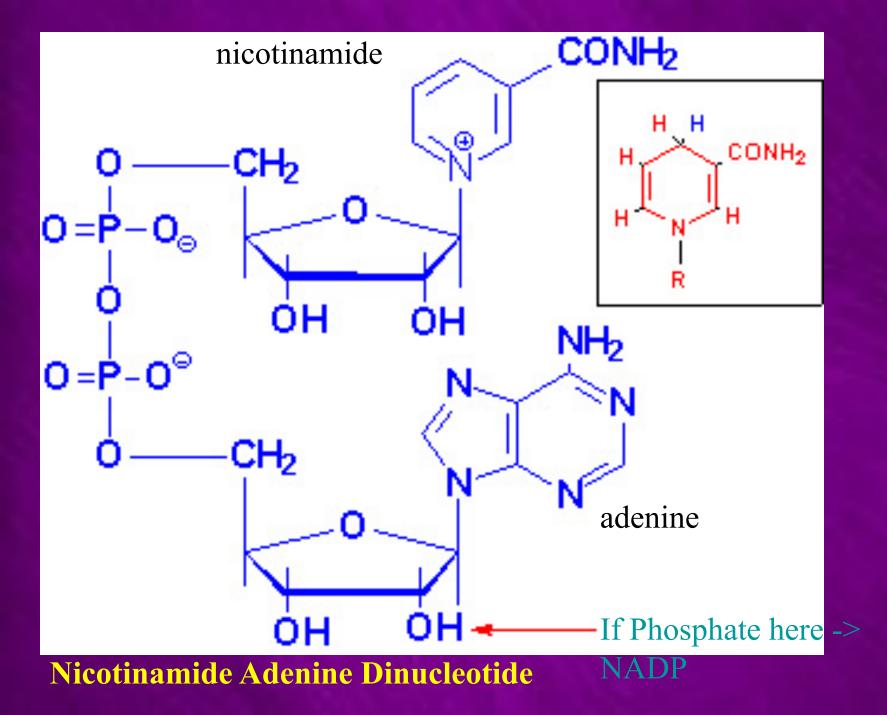
- Small intestine as such or as nicotinamide.

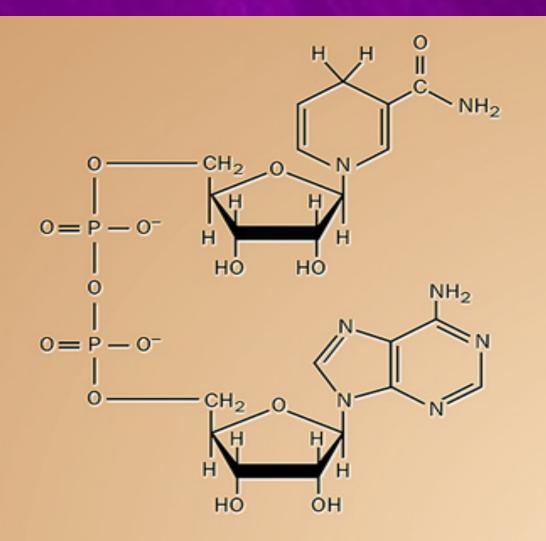
• <u>Excretion:</u>

- In urine: as nicotinic acid or nicotinamide or N1methyl nicotinamide.
- In sweat: as nicotinamide(traces).
- In milk: as nicotinamide (small amounts).

<u>Function:</u>

- Nicotinic acid in the form of nicotinamide enters in the formation of coenzyme I, II and III.
- These coenzymes acts as electron and hydrogen carriers:
 - Coenzyme I (NAD): nicotinamide adenine dinucleotide.
 - Coenzyme II (NADP): nicotinamide adenine dinucleotide phosphate.
 - Coenzyme III: acts as a coenzyme for synthesis of taurine from cysteine.
- NAD and NADP are coenzymes for reductases, dehydrogenases and hydroxylases, acting as hydrogen carriers in oxidation-reduction reactions.
- The piridine ring is the active part of the 2 coenzymes, C4 accepts one hydrogen atom while N1 accepts one electron from another H atom converting to hydrogen ion.
- NAD acts as coenzyme for lactate dehydrogenas, malate dehydrogenase, glutamate dehydrogenase and isocitric dehydrogenase.
- NADP acts as coenzyme for glucose-6-phosphate dehydrogenase, enoylenzyme reductase, phenylalanine hydroxylase and tryptophane hydroxylase.





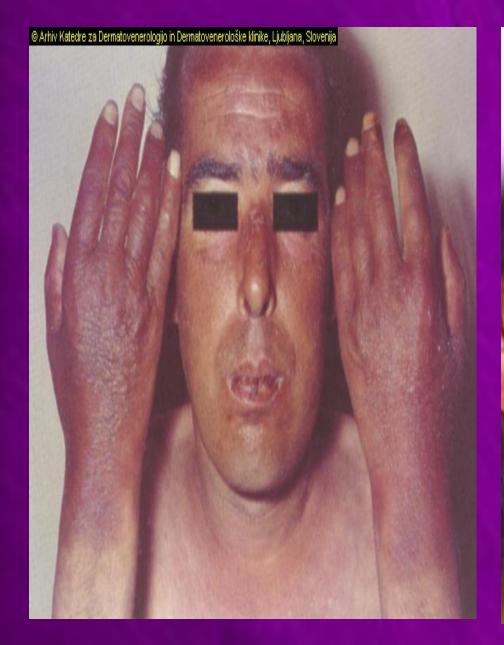
Nicotinamide adenine dinucleotide (NADH)

• <u>Relation between nicotinic acid and tryptophane:</u>

- Nicotinic acid synthesized in the body from tryptophan will result in deficiency of nicotinic acid e.g. zein of maize.
- Vitamin B6is necessary for synthesis of nicotinic acid so deficiency of vitamin B6 results also in deficiency of niacin.

<u>Deficiency</u>

- Deficiency manifestations in mane:
- Niacin deficiency causes Pellagera (Pella= skine, agra= rough) which occurs among the poor population consuming mazin as the chief constituent of diet (as maize is deficient in tryptophan).
- Pellagra symptoms are 3D's:
 - 1-Dermatitis: dry, rough, scaly with rough brown discolouration to exposed skin, glossitis and stomatitis.
 - 2-Diarrhoea.
 - 3-Dementia: mental power is lost.
- Deficiency manifestations in dog:
- A condition is called canine black tongue is produced it is characterized by dark red areas with necrotic lesions in the mucosa of the mouth.
- Pellagra manifestation may result from nicotinic acid, tryptophan or vitamin B6 deiciency.



An inability to absorb niacin (vitamin B3) or the amino acid tryptophan may cause pellagra, a disease characterized by scaly sores, mucosal changes and mental symptoms

vitamin

@ADAM, Inc.

• <u>Requirements:</u>

– Children: 16mg/day.

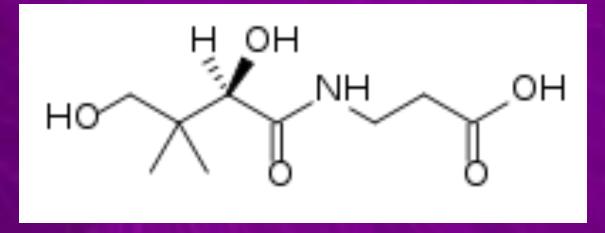
– Adult: 15mg/day.

– Pregnant and lactating female: 20mg/day.

Vitamin B5 (Pantothenic acid)

<u>Chemistry:</u>

- It is also called chick antidermatitis factor.
- It consists of pantoic acid and β-alanine.



• <u>Roperties</u>:

- It is soluble in water.
- It is destroyed by alkali, acid and heat (thermolabile) unstable.
- In tissues the vitamine is present almost in the form of CoA.
- It is usually written as CoA-SH because the activity of CoA is due to the free –SH group at its end.

• <u>Sources:</u>

- Plants: yeast, wheat and rice.
- Animals: it is present in all living tissues e.g. liver, eggs, kidney but thr richest known source of pantothenic acid is the royal jelly of bees.

• <u>Excretion:</u>

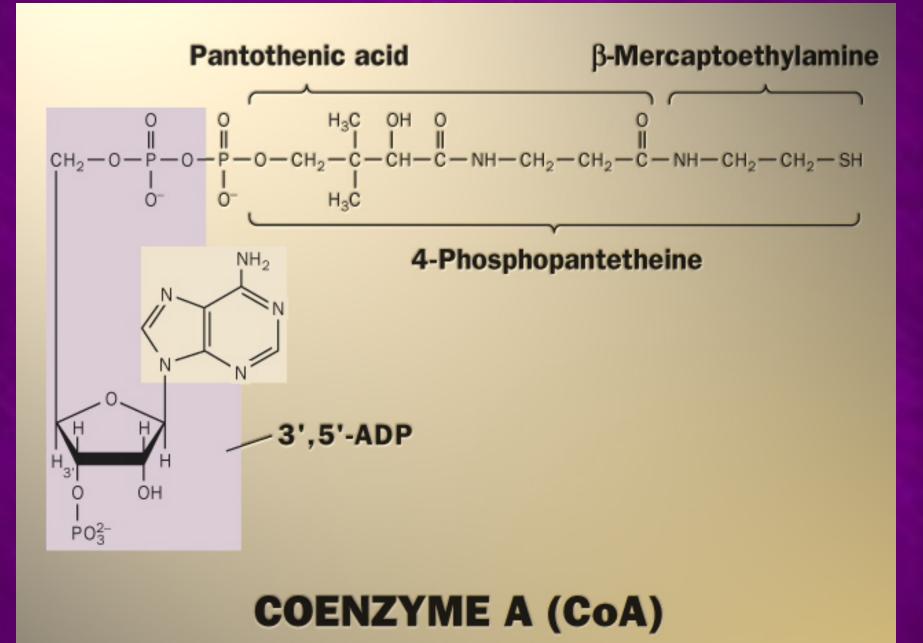
- Mainly by urine.
- Small amounts are excreted by milk and sweat.
- <u>Requirements:</u>
 - Adults: 10 mg/day.

<u>Function:</u>

1-It enter in the formation of coenzyme A (CoA-SH) which has the following metabolic function:

- It combine with acetic acid to for active acetate which may be oxidized to give energy or enter in the synthesis of cholesterol, steroid hormones and acetylecholine.
- It combines with succinic acid to give active succinate enters in the synthesis of heme.
- CoA is imortant in fatty acid oxidation.

2- pantothenic acid enters in acyl carrier protein structure (ACP). It is a coenzyme necessary for synthesis of fatty acid.

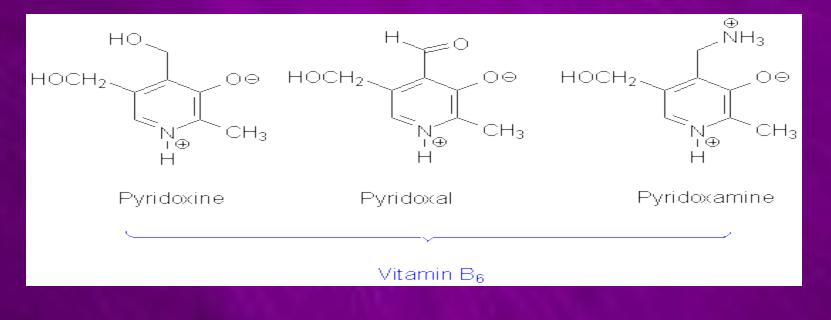


<u>Deficiency</u>:

- Deficiency of pantothenic acid is shown in the following symptoms: 1-Inadequate growth. 2-Lesions of the skin and its appendages. 3-haemorrhage below the skin, kidneys and adrenal cortex. 4-Normocytic anemia. 5-Impairment of adrenal function. 6-GIT disturbances. 7-Fatty liver. 8-haemorrhage below the skin, kidneys and adrenal cortex. 9-nervous manifestations e.g. myelen degeneration of peripheral nerves.
- In rats: it causes special dermatitis especially around the eye "spectacle eye" and depigmentation of hair "antigrey hair vitamins".

Vitamin B6 (pyridoxine, pyridoxal, or pyridoxamine, or pyridoxine hydrochloride)

- It is called rat antidermatitis factor.
- It is methyle-3-hydroxy4,5-dihydroxy methyl pyridine derivative.
- There are 3 active forms of pyridoxine in nature which are:



• <u>Properties:</u>

- All forms of vit. B6 are soluble in waterand alcohol.
- All are destroyed by light.
- It is colorless crystalline substance.
- Alcoholic in nature.
- Pyridoxal can be phosphorylated to give pyridoxal phosphate which acts as a coenzyme.

• <u>Sources:</u>

- Animals:
 - Egg yolk.
 - Royal jelly of bees (very rich in vitamin B6).
- Plants:
 - Yeast.
 - Rice polishings.
 - Germinal portion of various seeds.



•Small intestine.

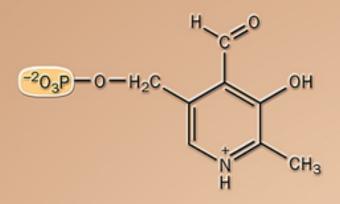
• <u>Excretion:</u>

•In urine:

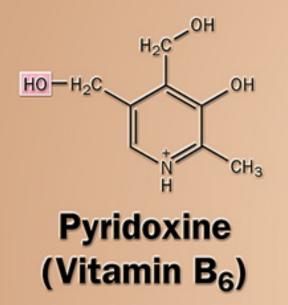
As pyridoxal and pyridoxamine (small amounts).
The major urinary excretion is 4-pyridoxic acid.
In sweat.
In milk.

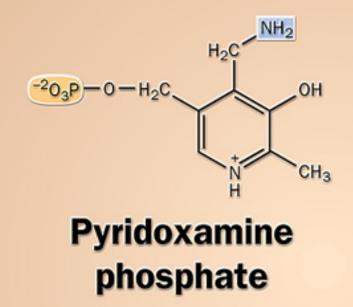
•<u>Function</u>

- 1-Vitamin B6is converted in thebody into pyridoxal phosphate(PLP) which is an important coenzyme in aminoacid metabolism, as it catalyzes the following reaction:
 - a) Transaminases: "transfere of aminogroup".
 - It is one of the most important functions of pyridoxal phosphate.
 - It trnsfer amino group from an aminoacid to α -keto acid to form a new aminoacid and new α -keto acid "cotransaminase".
 - Examples of transaminases which use PLP as a coenzyme are glutamic oxaloacetate transaminase GOT and glutamic pyruvic transaminase GPT.
 - *b) Decarboxylation: codecarboxylation.*
 - *Removal of CO2 of certain amino acids* "codecarboxylase" *e.g.decarboxylation of histidine, tyrosine and 5hydroxytryptophane.*
 - Dopamine produced is used for synthesis of adrenaline hormone.
 - Deficiency of PLP leads to convulsions due to defeciency in the amount of γ-amino butyric acid (GABA) which acts as an inhibitory neurochemical transmitter in the brain.



Pyridoxal phosphate





- c) It is important in the mewtabolism of tryptophane converting it into nicotinic acid by activating kynureninase enzyme.
- d) Trans-sulfuration (transfer of sulfur):
 - In metabolism of cysteine, PLP is concerned with the transfere of sulfur from methionine to serine to form cysteine.
- e) It acts as a coenzyme in dehydration of serine and threonine amino acids.
- *f)* It is essential coenzyme in haemoglobin synthesis.
 - It helps absorption of amino acids from intestine.
 - Pyridoxine is involved in amino acids transport into the cells.

2-Vitamin B6 in CNS is initimately concerned with the metabolism of γ amino butyric acid and glutamic decarboxylase are found the gray matter. It acts as a regulator of neural activity.

<u>Deficiency:</u>

- 1. Impaired growth due to disturbed amino acid metabolism.
- 2. Microcytic hypochronic anemia (hemoglobin synthesis).
- 3. Interference with tryptophan metabolism thus may cause pellagra.
- 4. Skin lesions.
- 5. Epileptiform convulsions and neuritis.
- 6. Demyelination of peripheral nerves and axons.
- 7. In rats: dermatitis is the most characteristic. Causes of deficiency:
 - 1. Low vitamin intake.
 - 2. Pregnancy due to increased demand of vitamin for embryo.
 - 3. Tuberculous patients taking high doses of isonicotinic acid hydrazide (INH) as it combines with pyridoxine forming hydrazone which inactivates the vitamin.

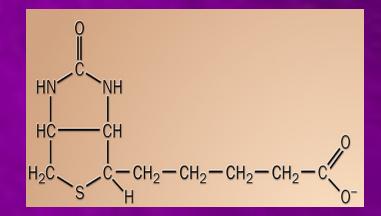
• **Daily requirements:**

- Adults: 2mg/day which must be increased in high protein diet.
- Pregnant and lactating female: 2.5mg/day.

Vitamin B7 (Biotin, vitamin H or coenzyme R)

• <u>Chemistry:</u>

- It is formed of:
- Imidazole ring.
- Thiophene ring.
- Valeric acid.



Properties:

- It is a white crystalline compound.
- It is soluble in hot water and dilute alkali.
- Biotin is present in nature in combination with lysine forming biocytin which is active and acts as a coenzyme.
- Avidin, a basic protein present in raw egg white, form a very stable biologically inactive complex with biotin.

• <u>Sources:</u>

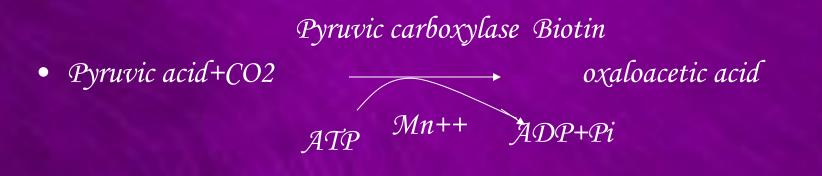
- Egg yolk, liver, kidney, milk and yeast.
- Large amounts are present in royal jelly of bees.
- In man much of the biotin requirement is supplied by its synthesis from intestinal bacteria.

• <u>Excretion:</u>

- In urine and stools.
- <u>Requirements:</u>
 - Adults: 150-300ug/day.

<u>Function</u>

- Biotin is a coenzyme helping CO2 fixation. So it is a coenzyme helping different (carboxylases). It is found in the form of biocytin or "CoR"
- Example of CO2 fixation:
- 1-fixation of CO2 into pyruvic acid to form oxaloacetic acid.



2-Fixation of CO2 to form C6 of purines. 3-Fixation of CO2 into acetyle CoA to form malonyl CoA which is used for

synthesis of fatty acids.

acetyle CoA carboxylase acetyle CoA +CO2 ______ malonyl CoA Mn++ Biotin

4-Fixation of CO2 into propionyl CO-A converting it into methyl malonyl CO-A this reaction needs propionyl COA carboxylase.

5-carbamoyl phosphate synthetase:

• Catalyseing the formation of carbamoyl phosphate from CO2 and ammonia. This carbamoyl phosphate is important for formation of urea and pyrimidines.

<u>Deficiency</u>

- Biotin deficiency in man is rare as it is synthesized by intestinal flora, but if deficient, it causes:
- 1. dermatitis of extremities.
- 2. Pallor of the skin and loss of hair.
- 3. Anorexia (loss of appetite), anemia, fatigue in muscles and vomiting. Causes of deficiency:
- Intake of antibiotics and sulfa drugs which inhibit the bacteria of the intestin.
- Intake of raw egg white which contains avidin.
- A basic protein in raw eggs known as avidin was found to cause deficiency of biotin because itcombines with it forming avidin-biotin complex and this prevent absorption of biotin this property impairs the value of biotin and causes what is called egg white injury.

