## Lecture title: Vitamins: Part 2

## Lecturer Affiliation: Chapter two

## Summary:

## Therapeutic uses (Thiamine)

1. Prophylactically (2–10 mg daily) in infants, pregnant women, chronic diarrhoeas, patients on parenteral alimentation. Glucose infusion unmasks marginal thiamine deficiency.

2. Beriberi—100 mg/day i.m. or i.v. till symptom sregress—then maintenance doses orally

3. Acute alcoholic intoxication: thiamine 100 mg is added to each vac of glucose solution infused. Most neurological symptoms in chronic alcoholics are due to thiamine deficiency—peripheralneuritis, Wernick's encephalopathy, Korsakoff' spsychosis: give 100 mg/day parenterally.

4. In neurological and cardiovascular disorders, hyperemesis gravidarum, chronic anorexia and obstinate constipation—thiamine has been used even without definite proof of its deficiency—symptoms improve dramatically if thiamine deficiency has been causative.

## 2-Riboflavin (vit B2)

**Absorption and fate:** Well absorbed by active transport and phosphorylated in the intestine. Riboflavin phosphate(Flavin mononucleotide: FMN) is formed in other tissues as well. Body does not significantly store riboflavin; larger doses are excreted unchanged in urine. Thiamine and riboflavin are both synthesized by colonic bacteria but this does not become available to the host.

**Actions and physiological role**: Flavin adenine dinucleotide (FAD) and flavin mononucleotide(FMN) are coenzymes for flavoproteins involved in many oxidation-reduction reactions. Thiamine and riboflavin are devoid of pharmacological actions.

**Deficiency symptoms:** Riboflavin deficiency generally occurs in association with other deficiencies. Characteristic lesions are angular stomatitis; sore and raw tongue, lips, throat, ulcers in mouth; vascularization of cornea. Dry scaly skin, loss of hair; anaemia and neuropathy develop later.

**Therapeutic uses:** to prevent and treat aribo-flavinosis (2–20 mg/day oral or parenteral), generally along with other B complex members. There is no proof of benefit in any other condition.

## 3-Niacin (vit B3)

**Absorption and fate:** Niacin is completely absorbed from gastrointestinal tract. Physiological amounts are metabolized in the body, while large doses are excreted unchanged in urine. Modest amounts are stored in liver.

**Physiological role and actions:** Nicotinic acid is readily converted to its amide which is a component of the coenzyme Nicotinamide-adenine-dinucleotide (NAD) and its phosphate(NADP) involved in oxidation-reduction reactions. These pyridine nucleotides act as hydrogen acceptors in the electron transport chain in tissue respiration, glycolysis and fat synthesis. Flavoproteins regenerate them by oxidizing NADH and NADPH. Nicotinic acid (but not nicotinamide) in large doses is a vasodilator, particularly of cutaneous vessels. It also lowers plasma lipids.

**Deficiency symptoms:** Niacin deficiency produces 'Pellagra', cardinal manifestations of which are:

-Dermatitis—sunburn like dermal rash on hands, legs and face which later turn black, crack and peal.

-Diarrhoea—with enteritis, stomatitis, glossitis, salivation, nausea and vomiting.

-Dementia—with hallucinations preceded by headache, insomnia, poor memory, motor and sensory disturbances.

Anaemia and hypo proteinaemia are common in pellagra. Chronic alcoholics are particularly at risk of developing pellagra, because in addition to dietary deficiency, niacin absorption is impaired in them. Other B vitamin deficiencies are often associated.