- Vitamin D is considered as steroid hormone.
- Cholecalciferol (vitamin D3) is synthesized in the skin by the sunlight (UV) (-7-Dehydrocholesterol is converted to vitamin D3), and it can be found in animal tissue.
- Ergocalciferol (vitamin D2) is derived from ergosterol in plants.
- The biologically active form is 1,25dihydroxycholecalciferol (calcitriol).

## Metabolism and actions of vitamin D:

- In liver: Cholecalciferol is converted to 25hydroxycholecalciferol (calcidiol) by the enzyme 25-hydroxylase.
- In kidneys: the 1-a-hydroxylase enzyme converts 25-hydroxycholecalciferol to 1,25-dihydroxycholecalciferol (biologically active)
  Active vitamin D is transported in blood by gc-globulin protein.
- Vitamin D synthesis is tightly regulated by plasma levels of phosphate and calcium.
- Plasma phosphate: Direct effect
- PTH: Indirect effect.
- Calcium homeostasis is maintained by parathyroid hormone (PTH) and calcitonin.

- Circulating level of >75 nmol/L is required for beneficial health effects.
- Nutritional rickets:
  - -demineralization of bone in children.
  - -continued formation of collagen matrix of bone.
  - -soft and pliable bones.
  - -skeletal deformities including bowed legs.
  - -low serum levels of vitamin D.
- Osteomalacia: demineralization of bones in adults.

## • Inherited rickets:

- -Vitamin D-dependent rickets (types 1 and 2)
- -Causing vitamin D deficiency mainly because of genetic defects in:
  - Vitamin D synthesis or receptor.
- -Diagnosis is done by Measuring serum levels of: 25-hydroxycholecalciferol – PTH – Calcium – Phosphate – Alkaline phosphatase

## • Osteoporosis:

- Reduction in bone mass per unit volume.
- primary osteoporosis: Post-menopause.
- Increases fragility of bones and susceptibility to fractures.
- Diagnosis: WHO standard: Serial measurement of bone mineral density.
- Bone formation markers: osteocalcin,
  Bone-specific alkaline phosphatase and
  P1NP.
- Bone resorption markers: CTX-1.