Vitamin D has an array of impact on human body physiological processes such as bone and calcium metabolism, homeostasis. It augments bone mineralization. Vitamin D deficiency results into low bone mineral density, osteoporosis and osteopenia. There is a relationship between alveolar bone density and vitamin D intake. [1]

There is a relationship between vitamin D status and bone health. Low vitamin D status is associated with higher risk of fractures. [2] Vitamin D plays an important role in calcium and phosphate metabolism through paracellular diffusion of calcium. The 1,25(OH)2D3 has an ATP dependent mechanism. Passive transport also occurs across the cell. The calcium transport to the extracellular fluid is by active transport through membrane proteins. In the intestinal cell, 1,25(OH)2D3 binds to a vitamin D responsive element on a responsive gene, such as VDR (Vitamin D receptor). Then this complex forms a heterodimer with the retinoid receptor and binds to the vitamin D receptor. Next, the vitamin D receptor translocates to the nucleus and binds to a vitamin D responsive element on a responsive gene, such as osteocalcin, calcium binding protein or 24-hydroxylase. After this transcription and translation occurs and proteins are synthesized and this regulates the active transport through the cell. This active transport requires an enzymatic process because of oestrogen deficiency. Calcium and vitamin D supplementation improves bone mass in these women. [4] Calcium and vitamin D supplementation also improves bone mass in elderly persons. [3] There are many target genes for vitamin D receptor. There are also manifested in oral cavity. In leukemia there is uncontrolled proliferation of leukemic cells and retards the development and growth of tumours in animal models raising the possibility of its use as an anticanter agent. Calcitriol inhibits the proliferation of many malignant cells by inducing cell cycle arrest and the accumulation of cells in the G0/G1 phase of the cell cycle, Apoptosis, Inhibition of Invasion and Metastasis, Antiinflammatory Effects, Regulation of prostaglandin metabolism and signalling, Induction of apoptosis, inhibition of stress-activated kinase signalling. [7]

**Immunomodulatory effects**

Vitamin D has potent immunomodulatory effects. VDR (Vitamin D receptor) is widely expressed in various immune cells of both innate and adaptive immunity. VDR ligand decreases expression of various pro-inflammatory molecules like IL-2, IFN, IL-6,8 in T cells and proliferation of T lymphocytes and keratinocytes. It increases expression of anti-inflammatory cytokines IL-10. VDR ligands prevent differentiation, maturation, activation of antigen presenting cells and dendritic cells. Vitamin D deficiency results in to variety of autoimmune disorders like IBD, multiple sclerosis. VDR polymorphisms are associated with increased susceptibility to both Crohn’s disease and ulcerative colitis in human subjects. [8]

**Cancer and vitamin D**

VDR is present in a wide variety of other cells including malignant cells. Recently it has been recognized that calcitriol exerts antiproliferative and prodifferentiating effects in many malignant cells and retards the development and growth of tumours in animal models raising the possibility of its use as an anticancer agent. Mechanism of anticancer action is growth arrest and differentiation. Calcitriol inhibits the proliferation of many malignant cells by inducing cell cycle arrest and the accumulation of cells in the G0/G1 phase of the cell cycle, Apoptosis, Inhibition of Invasion and Metastasis, Antiinflammatory Effects, Regulation of prostaglandin metabolism and signalling, Induction of mitogen-activated protein kinase phosphatase-5 (MKP5) and inhibition of stress-activated kinase signalling. [9]

Vitamin D receptor (VDR) are present in keratinocytes and Vitamin D induces the differentiation and inhibits the proliferation of keratinocytes. Various studies have shown that vitamin D inhibits the growth of squamous cell carcinoma cells both in vivo & vitro. Vitamin D also inhibits leukemic cells growth. Leukemia is also manifested in oral cavity. In leukemia there is uncontrolled proliferation of haemopoietic cells occurs and these cells are for teeth loss and periodontal disease. [1,2]

Post menopausal women are at high risk for periodontal disease, tooth loss due to progressive bone loss and inflammatory processes because of oestrogen deficiency. Calcium and vitamin D therapy along with hormone replacement therapy increases bone mass in these women. [10] Calcium and vitamin D supplements prevents osteoporosis and enhance tooth retention in elderly persons. [11] There are many target genes for vitamin D in the ameloblasts and odontoblasts of developing tooth germ. There is alteration in enamel and dentin in vitamin deficient children. [9]

**Bioavailability and Transport**

The vitamin D metabolites are bound in the circulation to vitamin D binding protein. The active metabolite 1,25(OH)2 D3 enters the cell and binds to the vitamin D receptor. Then this complex forms a heterodimer with the retinoid receptor and binds to a vitamin D responsive element on a responsive gene, such as that of osteocalcin, calcium binding protein or 24-hydroxylase. After this transcription and translation occurs and proteins are formed such as the calcium binding protein or osteocalcin. The classic effect of 1,25(OH)2D3 on active calcium transport occurs in the intestinal cell. In intestine, Calcium enters the cell through membrane proteins. In the intestinal cell, 1,25(OH)2D3 binds to the vitamin D receptor and the calcium binding protein is synthesized and this regulates the active transport through the cell. The calcium transport to the extracellular fluid is by an ATP dependent mechanism. Passive transport also occurs through paracellular diffusion of calcium. The 1,25(OH)2D3 has its effect on the classic target organs which includes bone, intestine and kidney and stimulates calcium transport from these organs to the blood. The production of 1,25(OH)2D3 is stimulated by parathyroid hormone (PTH). There is a negative feedback through calcium which decreases parathyroid hormone (PTH) and a direct negative feedback from 1,25(OH)2D3 to PTH. The active metabolite 1,25(OH)2D3 also shows rapid actions through a membrane receptor. [1]

**Bone metabolism**

Vitamin D plays an important role in calcium and phosphate homeostasis. It augments bone mineralization. Vitamin D deficiency results into low bone mineral density, osteoporosis and osteopenia. There is a relationship between alveolar bone density, osteoporosis and tooth loss. Low bone mass is a risk factor
unable to differentiate into mature cells. Vitamin D induces differentiation of these myelomonocytic leukemic cells. Various studies have also shown that vitamin D also have an additive effect on other treatment modalities for cancer treatment like retinoids, vitamin K and chemotherapeutic agents, for differentiation of leukemic cells. [9]

**Antimicrobial effects**

Vitamin D has potent antimicrobial activity against bacteria, viruses, and fungi. Various mechanism of antimicrobial activity are activation of various immune cells like B lymphocytes, T lymphocytes, macrophages, etc. Vitamin D boosts immune system by modulating production of antimicrobial peptides (AMPs) and cytokine response. Vitamin D exerts its antibacterial activity by the production of various peptides like B-defensins and cathelicidin. These peptides have antibacterial action against various strains of streptococcus, staphylococcus, klebsella, pneumonia, E coli. Vitamin D also has antiviral and antifungal activity. As all these microbial diseases have oral manifestations therefore Vitamin D because of its antimicrobial action can be used as a therapeutic agent alone or as an adjunct to various antimicrobial agents. [9]

**Vitamin D and Oral Health**

Vitamin D can reduce risk of dental caries because it induces production of peptides like cathelicidin and defensins and these have both antimicrobial and anti-endotoxin activity. [8] Oral Candidiasis is most prevalent lesion of immunocompromised individuals. A strong association between vitamin D deficiency and oral Candidiasis has been observed. Vitamin D modulates immune system and down regulates expression of calprotectin (which is an immune regulatory protein complex which down regulates neutrophil recruitment and inhibits neutrophil oxidative function). Vitamin D deficiency results in increased expression of this immune-regulatory protein complex and this ultimately results in decreased neutrophil functions and hence increased chances of opportunistic infections like oral candidiasis. [10]

Vitamin D prevents acute and chronic gingivitis. There is negative association between serum concentration of vitamin D and prevalence of bleeding on probing. It reduces susceptibility to gingival inflammation through anti-inflammatory effects. [11] Vitamin D deficiency leads to disorder of immune system, promotes infection and inflammation resulting into periodontitis.

**Vitamin D deficiency**

There are various risk factors for vitamin D deficiency including old age, female sex, non-white race, malabsorption (inflammatory bowel disease and cystic fibrosis), obesity, low dietary intake, less outdoor physical activity, exclusive breast-feeding, sunscreen use, clothing, higher latitude season of year. [12] According to the United States Institute of Medicine, the recommended dietary allowances of vitamin D are:

- Infants 0–6 months: 400 IU/day*
- Infants 6–12 months: 400 IU/day*
- 1–70 years of age: 600 IU/day (15 μg/day)
- 71+ years of age: 800 IU/day (20 μg/day)
- Pregnant/lactating: 600 IU/day (15 μg/day)

Asterisk for infants indicates Adequate Intake (AI) for infants, as an RDA has yet to be established for infants. [13]

**Conclusion**

Vitamin D plays an important role in oral health by maintaining bone mass, preventing tooth loss, caries, gingivitis and periodontitis, prevention of malignancy, prevention against infection by boosting immunity and having antimicrobial properties.