EFFECT OF DIET VITAMIN C AND D ON THE TOOTH MOVEMENT ACCELERATION IN ORTHODONTIC TREATMENT:

SYSTEMATIC REVIEW

THESIS

Submitted for Completion the Requirements to Receive Bachelor Degree in Dentistry

MAUDY ANGGI ARIZKI

J011191058

DEPARTMENT OF ORTHODONTIC

FACULTY OF DENTISTRY

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Dengan ini menyatakan bahwa skripsi yang berjudul "Effect of Diet Vitamin C and D on The Tooth Movement Acceleration in Orthodontic Treatment" adalah benar merupakan karya sendiri dan tidak melakukan tindakan plagiat dalam penyusunannya. Adapun kutipan yang ada dalam penyusunan karya ini telah saya cantumkan sumber kutipannya dalam skripsi. Saya bersedia melakukan proses yang semestinya sesuai dengan peraturan perundangan yang berlaku jika ternyata skripsi ini sebagian atau keseluruhannya merupakan plagiat dari orang lain. Demikian pernyataan ini dibuat untuk dipergunakan seperlunya.

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Makassar, 31 Oktober 2022

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ABSTRACT

EFFECT OF DIET VITAMIN C AND D ON THE TOOTH MOVEMENT ACCELERATION IN ORTHODONTIC TREATMENT

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Background: The principle of orthodontic tooth movement stated that when given pressure, the tooth would release a chemical signal which in turn affect cell differentiation in bone cellular structure which resulted in tooth movement. The pressure given would travel along the length of the tooth, such as when a pressure is applied to the crown it would be distributed to the root, and the periodontal ligament and then to the alveolar bone structure. The movement of the tooth itself needs to be maintained properly in the sense that the movement occurred is a form of stress responses, which would, if left unchecked, would cause uncontrolled movement, therefore proper risk mitigation actions needs to be taken. Previously it was found that bone metabolism that affect by various drugs and chemical can also affect tooth movement in orthodontic treatment. Vitamins are are one of them. Due to COVID-19 people are taking vitamins at an increased rate, one of which is vitamin C and D People who use orthodontic appliances are generally concerned about their dental aesthetics. In fact, the vitamins they consume, one of which is in the form of vitamins C and D, affect the movement of teeth in orthodontic treatment. Therefore, this systematic review was made to find out wether dietary vitamins C and D affect the acceleration of tooth movement in orthodontic treatment. **Purpose:** To determine the mechanism of orthodontic tooth movement (OTM) in orthodontic treatment as well as the effect of dietary vitamin C and D on the acceleration of OTM. **Method:** This research is a systematic review using the PRISMA (*Preferred Reporting Items for Systematic Reviews and Meta-analyses*) method which is carried out systematically by following the correct stages or research protocol and collect literature from search engines such as PubMed and Science Direct which discuss the effect of diet vitamin C and D on the tooth movement acceleration in orthodontic treatment. **Result:** There are six kinds of literature relevant to the topic. **Conclusion:** From all literature, it can be concluded that vitamin C and D can be an effective agent to increase and accelerate orthodontic tooth movement.

Keywords: orthodontic tooth movement (OTM), vitamin C, vitamin D, acceleration in orthodontic tooth movement

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CHAPTER 1

INTRODUCTION

1.1 Background

Oral health along with holistic health and nutritional status are corelating factors which corresponds with each other in the grand scheme of things. The interest of both to maintain and increase oral health, nutrition is a paramount factor. In order to reach a balance of nutrition, a balanced amount of vitamins, minerals and other supporting substances are needed in order to preserve a healthy organ and tissue function. Therefore, an inability to preserve this balance would result in function and maintenance failure which in turn would adversely impact oral health.[1]

Riskesdas 2018 data stated that there are around 57,6% of Indonesia's population suffers from oral health problems in the last 12 months.[2] The most common problem being found is malocclusion. Malocclusion prevalence in Indonesia is 80% which is a relatively high percentage.[3] Malocclusion is defined as a condition in which there are irregularities or abnormal relationship during the occlusion process, outside of the range that can be called an "ideal" occlusion. Malocclusion is not a life-threatening condition however it is high prevalence and the preventable nature and present treatment access, makes it a public health problem in the dental world.[4]

Orthodontic appliances both fixed and removable has been the treatment of choice and gold standard in both preventive actions and treating the malocclusion conditions. The main goal of Orthodontic treatment is to correct the dental abnormalities which lies in tooth and jaw relations, one of the other purposes being esthetic purpose which in turn would have a positive impact on one's psychosocial aspect.[5]

Principally speaking, orthodontic treatment relies on the fact that when given pressure, the tooth would release a chemical signal which in turn affect cell

differentiation in bone cellular structure which resulted in tooth movement.[5]. The pressure given would travel along the length of the tooth, such as when a pressure is applied to the crown it would be distributed to the root, and the periodontal ligament and then to the alveolar bone structure. The movement of the tooth itself needs to be maintained properly in the sense that the movement occurred is a form of stress responses, which would, if left unchecked, would cause uncontrolled movement, therefore proper risk mitigation actions needs to be taken. The needed movement being the attachment mechanism moves along with the tooth.[6]

Previously it was found that bone metabolism that affect by various drugs and chemical can also affect tooth movement in orthodontic treatment. Vitamins are are one of them. Vitamins work as free radical inhibitors or act as an antioxidant.[7]

Vitamins are organic compounds, small amount can be found in food and considered essential for basic biological function of the body. Factually, 3 out of 12 vitamins are divided in 2 classes in accordance with its solubility in water and fat.[8]

Vitamin C is a type of vitamin which is soluble in water. It usually called ascorbic acid, it acts as an antioxidant and co-factor in the synthesis of collagen, metabolism of both carnitine and catecholamines, moreover it also helps in the absorption of iron in food. Since humans cannot naturally synthesize vitamin C, humans fulfill their intake through food.[9] Several researches show that vitamin C is essential in stimulating osteoclasts in cell culture. If a vitamin C deficiency occurs there will be hampering in the osteogenesis and regeneration and reorganization of periodontal ligament, which in turn would reduce tooth movement due to it hampering tissue repair time. The definitive impact of vitamin deficiency on orthodontic treatment is in the maintenance of periodontal ligament. Ascorbic acid works by increasing the proliferation of osteoclast and bone progenitor cells, a deficiency would cause reduced regeneration rate and collagen fiber degradation, which in turn would impair orthodontic tooth movement. [10]

One type of vitamins is fat soluble vitamin, which is used by the body to increase the absorption of calcium, magnesium and phosphate. [11] Bone remodeling is a process which is triggered by orthodontic forces. Its phase is resorptive phase dan bone formation phase of the alveolar process. There exists relationship between vitamin D receptors, periodontitis and bone metabolism. Along with Vitamin C, Vitamin D also plays a role in calcium and phosphor absorption rate, other capability of vitamin D is to reduce osteoclast differentiations and enhancing the activity of existing osteoclast. [12]

Due to COVID-19 people are taking vitamins at an increased rate, one of which is vitamin C and D.[13] People who use orthodontic appliances are generally concerned about their dental aesthectics. [13] In fact, the vitamins they consume, one of which is in the form of vitamins C and D, affect the movement of teeth in orthodontic treatment.[14,15] Therefore, this systematic review was made to find out wether dietary vitamins C and D affect the acceleration of tooth movement in orthodontic treatment.

1.2 Study Problem

To understand whether dietary intake of vitamin C and D would have an effect on tooth movement.

1.3 Study Purposes

1.3.1 General Purposes

This paper aims to understand the effect of dietary vitamin C and D on acceleration tooth movement in orthodontic treatment.

1.3.2 Special Purposes

1. To determine the mechanism of orthodontic tooth movement (OTM) in orthodontic treatment.
2. To determine the effect of dietary vitamin C and D on the acceleration of OTM.

1.4 Study Benefits

1.4.1 Scientific Benefits (Academic)

This review would add new insight and knowledge to medical and dentistry students regarding the effect of dietary vitamin C and D on the acceleration of orthodontic tooth movement.

1.4.2 Practitioner Benefits

This review would use as a reference point in prescribing and education regarding the benefits in taking Vitamin C and D in regards with orthodontic treatment.

CHAPTER II

LITERATURE REVIEW

2.1 Tooth Movement in Orthodontics

2.1.1 Tooth movement Definition

According to Lie et al, orthodontic movement is determined by coordinated tissue formation and resorption in both the bone and periodontal ligament (PDL) surrounding the tooth. Pressure or occlusal loads in the tooth would cause local cellular hypoxia and causing fluid flow which then causes an aseptic inflammation which in turn causing osteoclastic resorption and osteoblastic bone deposition in the pressure bearing areas.[5]

Mechanical Stimulus and PDL remodeling in the alveolar bone is the main cause of Orthodontic tooth movement (OTM). Bone remodeling consists of resorption on the pressured sites. OTM is managed by the given force and also the biological response of the PDL. Changes in blood flow caused applied forces causes alteration in the micro level environment of the PDL which in turn leads to the secretion of inflammatory mediators' secretions such as cytokines, GF, neurotransmitters, colony stimulate factors and arachidonic metabolites. All these secretions in turn work as the main etiological factor of bone remodeling.[6]

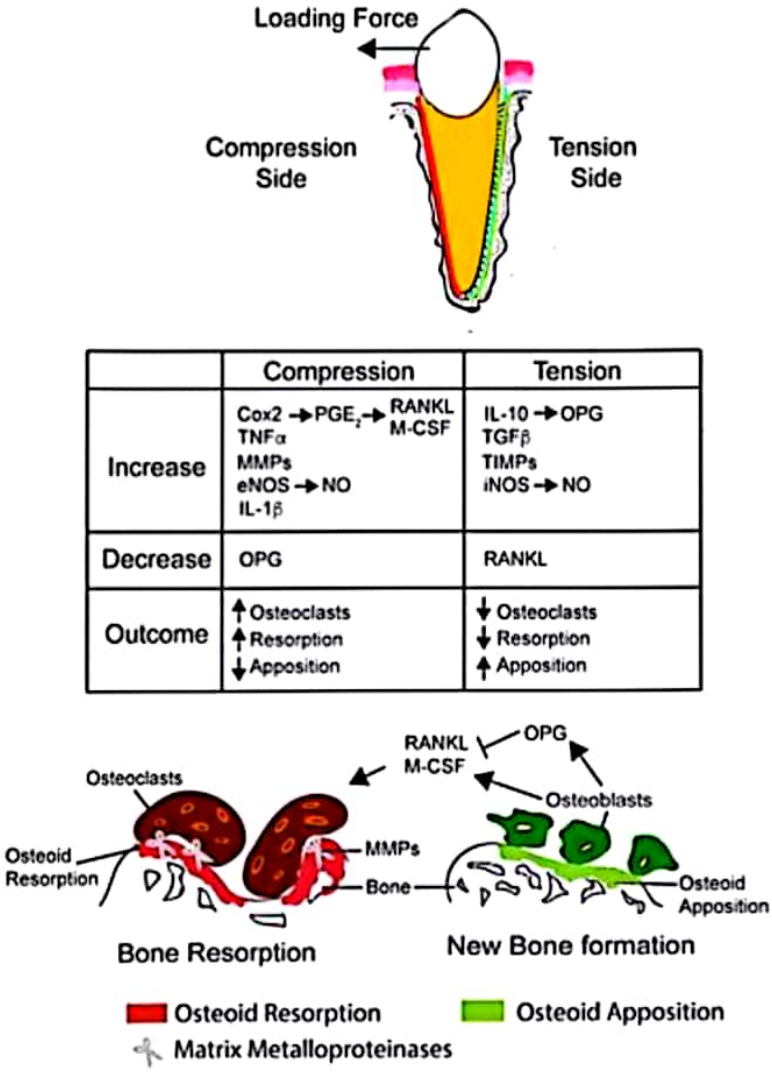

Figure 2.1.1 Signaling pathways associated with compression and tension due to orthodontic loading
Source: Li Y, Jacox L, Little S, Ko C. Orthodontic tooth movement: The biology and clinical implications. Kaohsiung J Med Sci. 2018;34(4):207–14

2.1.2 Tooth Movement Theory

It's postulated by Rahardjo, there are two mechanisms affecting teeth movement in orthodontics. There is a possibility that the second theory together plays a role in tooth movement.

1. **Electricity biological theory (piezoelectric)**

   According to this theory, bone metabolism is affected by electric signals that occurred when the alveolar bone is deformed by pressure. Cell

membrane receptors or membrane permeability (or possibly both) is affect by these electrical signals. This situation will affect cell activity. Bone is a mass or piezoelectric material, meaning that the rate surface electrical discharges when subjected to pressure. It is piezoelectric process that bridges the remodeling of bone caused by orthodontic forces. In one study it was found that bone has a piezoelectric effect of approximately eight times that of dentin and cementum. This piezoelectric strength is related to the tissue's ability to create remodeling. Because bone has a large piezoelectric effect, it is the bone that is most easily remodeled.[16,17]

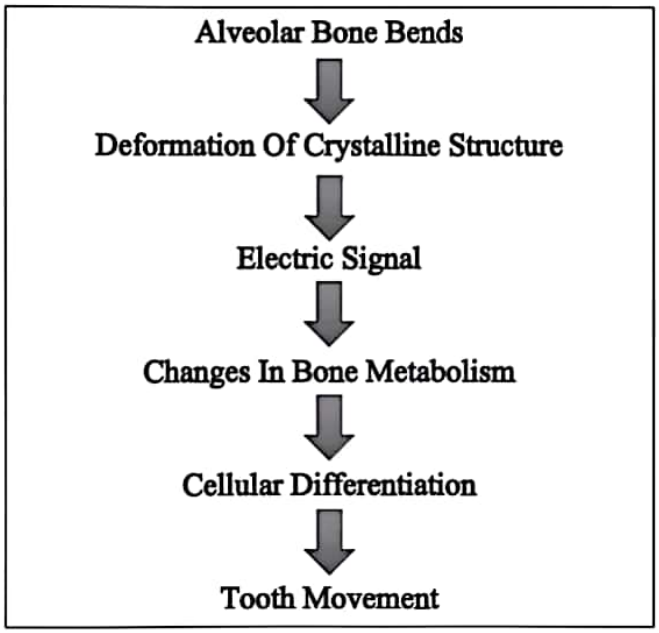

Figure 2.1.2 Bio-electric theory of tooth movement
Source: Asiry M. Biological aspects of orthodontic tooth movement: A review of literature. Saudi J Biol Sci. 2018;25(6):1027–32

2. Pressure-tension theory

The existence of tension and pressure on the periodontal ligament results in chemical changes. This is ultimately acting as a stimulus for cellular changes in tooth movement. Changes in blood flow to the periodontal ligament occur due to long-term pressure. This causes the tooth to shift in the tooth socket and eventually affects the periodontal ligament. In areas of

the periodontal ligament that are under pressure, blood flow and oxygen supply are reduced. Meanwhile, in the area of the periodontal ligament that gets a pull the blood supply will remain or increase. Metabolites also undergo the same changes. This happens in a matter of minutes. Chemical changes that occur will affect the release of biologically active agents that will stimulate cell differentiation and activity. Broadly speaking, the incident can be described as follows:

1) Blood flow changes, due to pressure on the periodontal ligament.
2) Formation or release signal chemistry.
3) Activity cells are triggered by chemical changes. [16,17]

Factors affecting tooth movement according to Pressure-Tension theory.

Factors affecting tooth movement	Pressure side	Tension side
Blood flow	Decreases	Increases
Oxygen level	Decreases	Increases
Carbon dioxide level	Increases	Decreases
Cell replication	Decreases	Increases
Fiber production	Decreases	Increases

Figure 2.1.2 Factors affecting tooth movement according to Pressure-Tension theory
Source: Asiry M. Biological aspects of orthodontic tooth movement: A review of literature. Saudi J Biol Sci. 2018;25(6):1027–32

2.1.3 Tooth Movement Phases

According to Asiry, Charles J Burstone is the first one who organize tooth movement into three phases, namely initial, lag, and postlag phases. Initial phase occurred after force application, which occurred rather quickly as the tooth is displaced in the periodontal space. This phase lasts from 24-48 hours. PDL is then pressurized and then stretched resulting vascular extravasation which in turn facilitate inflammatory cell chemoattraction and osteoblast and osteoclasts progenitor response to the PDL space.

The movement in the lag phase is, if any, only minimal. The reason for this is the hyalinization of the pressed PDL. There will be no movement

until some cells eliminate the necrosed tissue. The tooth stops moving for 20 to 30 days in the lag phase, and total elimination of the necrotic tissue occurs during this period along with the bone marrow resorption by its side. The necrotic tissues from the pressed bone and PDL sites are eliminated by macrophages, foreign body, giant cells, and osteoclasts.

A forty-day period usually marks the start of the postlag phase, in which there would be a sudden or gradual movement of the tissue. It is even hypothesized that during this period's displacement, necrotic tissues are developed and removed at a constant rate.[17]

2.1.4 Involved Cells in Tooth Movement

Tooth movement induced by orthodontic treatment can cause sequential reactions involving the periodontal tissue and alveolar bone, resulting in the release of numerous substances from the dental tissues and surrounding structures. Zaina et al. stated that osteoblasts and osteoclasts are importantly involved in OTM. Therefore, these two cells have become foci in many OTM studies. Osteoblasts are involved in bone formation 40-48 hours after orthodontic force is applied.[18] Osteoblasts begin to differentiate with the bone marrow-derived stem cells which moved into the blood vessels. The migration of mesenchymal stem cells from the blood vessel wall or the activation of mesenchymal stem cell progenitor cells and the formation of anterior osteoblasts occur approximately 10 hours after the applied force. This sequence of cellular activities during the stem cells' development into osteoblasts and osteoclasts can be used to determine the potential markers for OTM. Osteoclast is a multinucleated cell that degrade and resorb bone. It is the osteoclasts derived from hematopoietic stem cells that work together with the osteoblasts to remodel the bones.[16]

2.1.5 Factor Influencing Orthodontic Tooth Movement

1. **Level of Force**

It is assumed that an optimal force system is important for an adequate biological response in the periodontal ligament.1 It also has been suggested for a long time that the optimal force is related to the surface area of the root.2–4 In the past 70 years, the concept of optimal force has changed considerably. Schwarz5 proposed the classic concept of the optimal force. He defined optimal continuous force as ''the force leading to a change in tissue pressure that approximated the capillary vessels' blood pressure, thus preventing their occlusion in the compressed periodontal ligament.'' According to Schwarz, forces well below the optimal level cause no reaction in the periodontal ligament. Forces exceeding the optimal level would lead to areas of tissue necrosis, preventing frontal bone resorption. Tooth movement would thus be delayed until undermining resorption had eliminated the necrotic tissue obstacle.[34]

2. **Medicine**

Topical administration of synthetic analogues of eicosanoids increase the rate of OTM, whereas their inhibitors might decrease it. The most important inhibitors are the NSAIDs, which have both analgesic and anti-inflammatory effects. Although they all show a similar action, their effect on the rate of OTM is not uniform. The studies on the effects of NSAIDs on OTM were all performed over relatively short experimental periods. The effects found in these studies, therefore, might underestimate the effects of prolonged administration— eg, in rheumatoid arthritis patients.[35]

Of the other analgesics, only paracetamol has been studied in relation to orthodontics, and no effect on the rates of OTM could be established. No experimental data are available on the effect of opioid analgesics in this respect.[35]

Corticosteroids, and especially glucocorticoids, stimulate OTM, but this depends on the relative anti-inflammatory activity of the corticosteroid and

the administration protocol. Local or systemic application of PTH also increases the rate of OTM. The same effect is seen when endogenous PTH synthesis is stimulated by, for example, a low-calcium diet. Intermittent short administration of PTH or its active fragment (1-36) (teriparatide), on the other hand, has an anabolic effect on bone. However, no data are available to show that such an administration regimen inhibits OTM.[35]

Administration of exogenous thyroxine increases the rate of OTM in a dose-dependent manner. Likewise, calcitonin is involved in bone remodeling and calcium homeostasis, although no experimental data on its effect on the rate of OTM are available. The same applies to estrogen supplementation, specific estrogen receptor modulators (such as raloxifene), and oral contraceptives. Although an inverse relationship between estrogens and OTM was suggested, direct evidence for this effect is not available from the literature.[35]

Administration of vitamin D3 increases the rate of OTM in a dose-dependent manner, whereas bisphosphonate administration decreases the rate of OTM in a dose-dependent manner. The use of bisphosphonates is complicated by serious osteonecrosis in the maxilla and the mandible. This threat is greatest in patients with prolonged bisphosphonate use, and, because of the extremely long half-life of these drugs, patients can experience problems years after they discontinue therapy. In orthodontic patients, bisphosphonates can be used to prevent relapse, but they should be used with great caution.[35]

2.2 Vitamin C

2.2.1 Definition of Vitamin C

Activating process of enzyme prolyl hydroxylase is needed role from Ascorbate Acid or vitamin C, which supports the formation of hydroxyproline in the stage of hydroxylation. Without ascorbate acid, fiber collagen formed in

all network bodies becomes handicapped and weak. Because of that, this vitamin is vital in subcutaneous tissue, bone, teeth, and cartilage. [19]

Lack of vitamin C can cause bone growth to stop. There is no new collagen is found between the cells even the cells of the growing epiphysis proliferate. Bone becomes easy to fracture at point growth because of failure bone for ossifying. Other things that can occur, i.e., in a fractured bone, which has to experience ossification in patients with vitamin C deficiency, osteoblasts could not shape matrix new bone. As a result, the fractured bone could not grow. [19]

Vitamin C or ascorbic acid was first isolated in 1923 by Hungarian biochemist and Nobel laureate Szent-Gyorgyi and synthesized by Howarth and Hirst. It exists in reduced [ascorbate] and oxidized forms as dehydroascorbic acid which are easily inter-convertible and biologically active thus it acts as important antioxidant. Vitamin C is easily oxidized acid and destroyed by oxygen, alkali and high temperature. Most of the plant and animal species have the ability to synthesize vitamin C from glucose and galactose through uronic acid pathway but man and other primates cannot do so because of deficiency of enzyme gulonolactone oxidase [EC 1.1.3.8] required for it's biosynthesis.[20]

2.2.2 Sources of Vitamin C

Chambial et al. stated that vitamin C can find in fruit, i.e., strawberries, tomatoes, broccoli, cabbage, brussels, radishes, Indian gooseberries, oranges, green peppers, red peppers, and other leafy vegetables, becomes source tree for vitamin C because of their high vitamin C content, ranging from 5,000 mg/100 g.[20]

2.2.3 Benefits of Vitamin C on Bone

According to Aghajanian et al. from the analysis result, several studies show a positive impact on bone health, epidemiology and genetic models of the effect of vitamin C. [21] Vitamin C, which is natural and traditionally linked with the disease scabies, is important for guarding bone health. This is important for

matrix bone in the production of collagen. Vitamin C also works to remove free radical which is harmful to bone health. [22] By overall, vitamin C can influence the expression of bone matrix genes in osteoblasts by providing a positive effect on the formation of bone trabecular. Recent studies on the molecular pathways for vitamin C action that include direct vitamin C pre-regulation of target gene transcription by affecting the activity of transcription factors and epigenetic modification of key genes involved in skeletal development and maintenance have been in recent studies. This has been discussed clearly. [21]

2.2.4 Benefits of Vitamin C on Periodontal Tissue

It's postulated by Pussinen et al. that vitamin C has been a candidate for a long time for modulating periodontal disease. Additional vitamin C has known that needed during regenerating network and disease infectious. Avitaminosis-C generally relates to the synthesis of damaged collagen, which results in dysfunctional tissue, such as disturbance in healing wounds and capillaries being broken because of a lack of support from wall capillaries by connective tissue. Regeneration collagen which working for guard quality element attachment is essential for healthy periodontal tissue. It is logical to hypothesize that low vitamin C concentrations are the cause of concern and risk factors for periodontal disease since vitamin C has an immunomodulating function that affects the host`s susceptibility to infectious diseases and is involved in the synthesis of intercellular substances such as collagen fibers found in various forms of connective tissue and bone and tooth matrix. [23]

2.2.5 Mechanism of Action of Vitamin C

According to Aghajanian et al., vitamin C in the previous study has shown that it can act as a stimulus in differentiation osteoblasts on the side effect antioxidants in osteoclast genesis. Several factors of growth and hormones are the responsible in the proliferation and differentiation of cells, chondrogenic and osteogenic. Action one or more factor growth osteogenic this regulated by vitamin C in cell bone. For example, treated osteoblasts with vitamin C have

been upgraded the expression transformation growth factor (TGF)-β, OPN, and estrogen receptor (ER)-α. All of those factors are a crucial factor in bone cells differentiation. Vitamin C treatment increases sonic hedgehog (Shh) signaling by advancing expression resulting tc1, Shh pathway target genes in MC3T3-E1 osteoblasts. Similarly, AA modulates MapK/ERK signaling and Indian hedgehog (Ihh), bone morphogenetic protein 2 (BMP-2), and SOX9 expression via collagen matrix induction. However, whether the change in growth factor expression or action is due to a direct effect or an indirect effect of vitamin C on bone cells remains to be inspected further. [21]

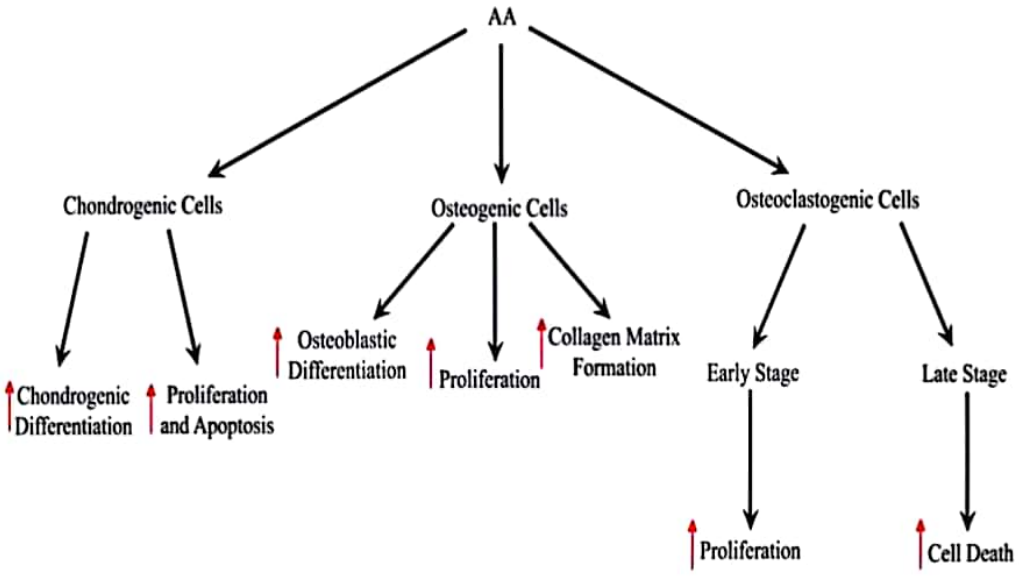

Figure 2.2.5 Differential effects of vitamin C on different cell types
Source: Aghajanian P, Hall S, Wongworawat M, Mohan S. The Roles and Mechanisms of Actions of Vitamin C in Bone: New Developments. J Bone Miner Res. 2015;30(11):1945–55

Vitamin C that act as an agent-reducing antioxidant in the formation and function of several cells, such as myoblasts, chondroblasts, adipocytes, odontoblasts and osteoblasts is strongly involved. Vitamin C can be ingested through food because humans cannot synthesize it. Vitamin C deficiency can change in the tissue, especially in a tissue that is rich in collagen, such as bone, skin and bone cartilage. These vitamins are involved in extracellular synthesis matrix, proliferation cells and differentiation. Vitamin C, in previous

study has found that can stimulate proliferation and differentiation of cell bone and increase collagen synthesis to push bone formation. Besides that, vitamin C in some line cell osteoblasts can also induce expression marker differentiation and mineralization of osteoblasts. Vitamin C also promotes the proliferation of different cells and stimulates DNA synthesis in cell culture. However, a very high concentration of vitamin C could become cytotoxic and causes inhibition of proliferation of cells and increased apoptosis. Several studies report increased proliferation of osteoclasts and their progenitor cells, stimulating more rapid tooth movement although results regarding the effect of vitamin C on tooth movement are found to be conflicting at best.[24]

2.3 Vitamin D

2.3.1 Definition of Vitamin D

Singh stated that the term vitamin D is used for various compounds that naturally prevent rickets. They include ergocalciferol (calciferol vitamin D), cholecalciferol (vitamin D), dihydrotachysterol, alfacalcidol (1a-hydroxycholecalciferol) and calcitriol (1,25-dihydroxy cholecalciferol). This vitamin plays an important role in calcium metabolism, regulates calcium homeostasis, and maintains a normal level of plasma calcium and phosphate. [25]

Vitamin D also helps control calcium and bone storage and also increases calcium absorption from the gastrointestinal tract. Vitamin D can enhance calcium absorption mechanisms, primarily by promoting active calcium transport through the ileal epithelium. Vitamin D also primarily plays a role in increasing the formation of calcium-binding proteins in the intestinal epithelium which helps in the absorption of calcium. The specific function of vitamin D is to help metabolize calcium in the body as a whole and in bone formation.[19]

2.3.2 Sources of Vitamin D

It's postulated by Nair et al. that the main source of vitamin D is synthesized from skin which exposed to sunlight. This can generally be obtained between 1000 hours and 1500 hours in the summer, spring, and fall.

Vitamin D is also produced by the skin itself. The amount of vitamin D produced is equivalent to between 10,000 and 25,000 IU. Various factors that decrease the production of vitamin D3 in the skin include increased skin pigmentation, aging, and application of topical sunscreens. Changes in the sun's zenith angle caused by changes in season of year, latitude or time of year can also affect the skin's production of vitamin D3 dramatically.[26]

Source of vitamin D can also obtain from milk, cow's milk and breast milk (ASI), oil cod, vitamin D fortified foods and drinks, and supplements. [15]

2.3.3 Benefits of Vitamin D on Bone

a. Prevent Bone Demineralization

In a research done by Laird et al, inadequacy of vitamin D over a prolonged period of time would result in bone demineralization. The lack of vitamin D would result in the body taking calcium from the bone in order to even out the circulating calcium level in the body. This in turn would cause a weakened bone structure over time through secondary hyperparathyroidism, ultimately leading to osteoporosis and osteomalacia. What constitutes as osteoporosis is standard deviation of BMD of 2,5 below the average for healthy young individuals.[27]

b. Prevent Fractures in Bones

Three trials of vitamin D supplementation and intervention has reported a statistically significant reduction rate for fractures incidence. There has also been reported that combination therapy using vitamin D has resulted in a reduced fracture incidence rate. [27]

c. Support Health Bone

A study has shown that calcitriol 20 IU/day for 6 months can decrease IL-1 and TNF- concentrations and increase BMD in postmenopausal

women with osteoporosis. This shows that vitamin D has a potential to alter cytokine production, which in turn can reduce fracture risk.[27]

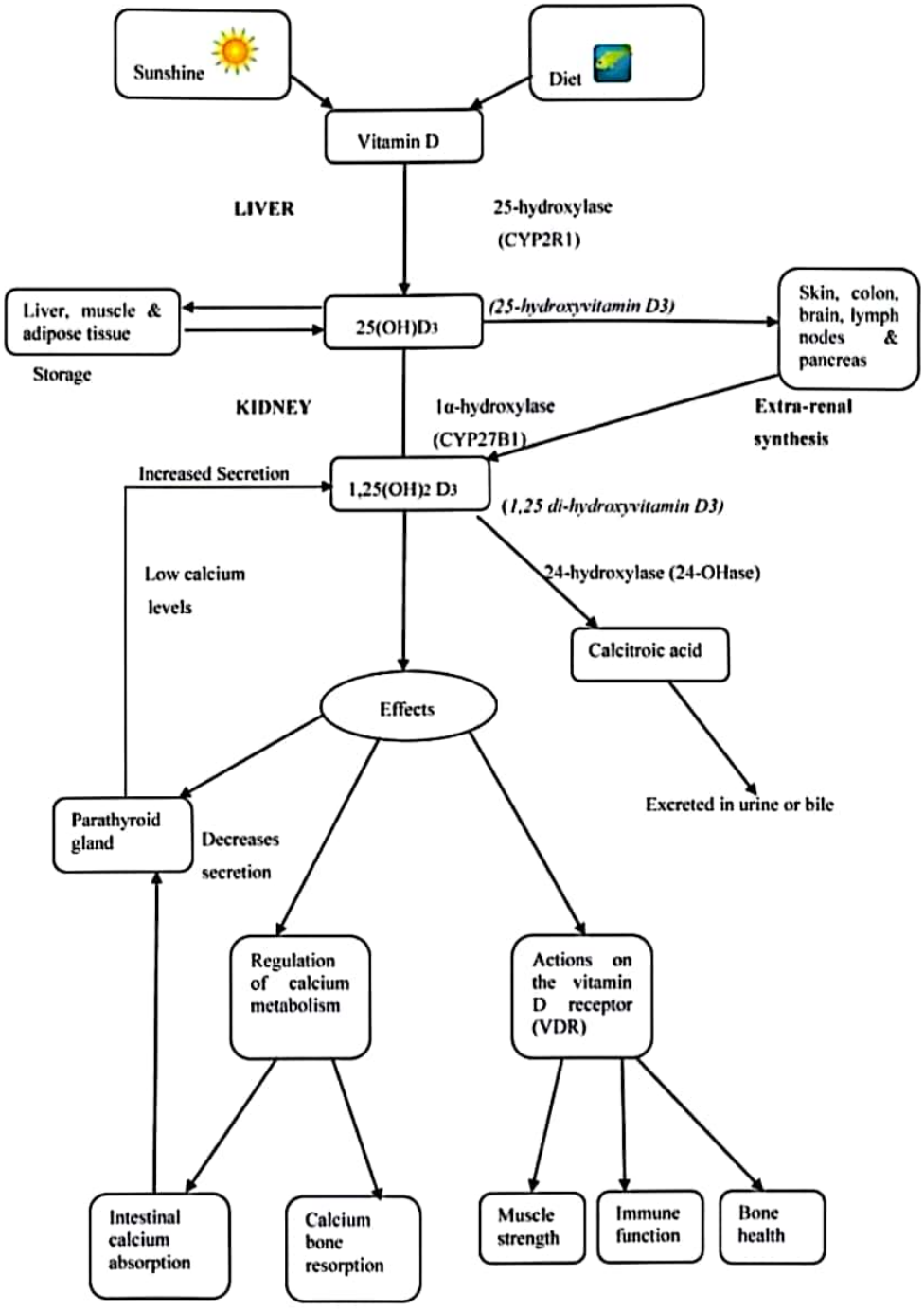

Figure 2.3.3 Summary diagram, of vitamin D metabolism and function
Source: Laird E, Ward M, McSorley E, Strains J, Wallace J. Vitamin D and bone health: potential mechanisms. Nutrients. 2010;2(7):693–724

2.3.4 Benefits of Vitamin D on Periodontal Tissue

A research by Anand et al. showed a significant correlation between vitamin D and calcium intake with periodontal health. Supplementation of both of these factors has an enhancing effect to one's periodontal health, which also increases mandibular bone density and hamper alveolar bone resorption. Vitamin D and calcium supplementation at daily doses higher than 800-1000 IU has found to decrease the periodontal disease severity, supporting the argument to conduct a randomized clinical trial regarding the potential benefits of vitamin D in periodontal health. Other capacity of vitamin D is an anti-inflammatory capacity in which it inhibits the cytokine cell expression and causing antibiotic molecule secretion by macrophages. Increased risk of infectious disease has been tied in with vitamin D deficiency, therefore adding to its role in treating periodontal diseases. not only attributed to its direct effect on bone metabolism, but also to its antibiotic effect on periodontophatogens and its inhibitory effect on inflammatory mediators contributing to periodontal breakdown.[28]

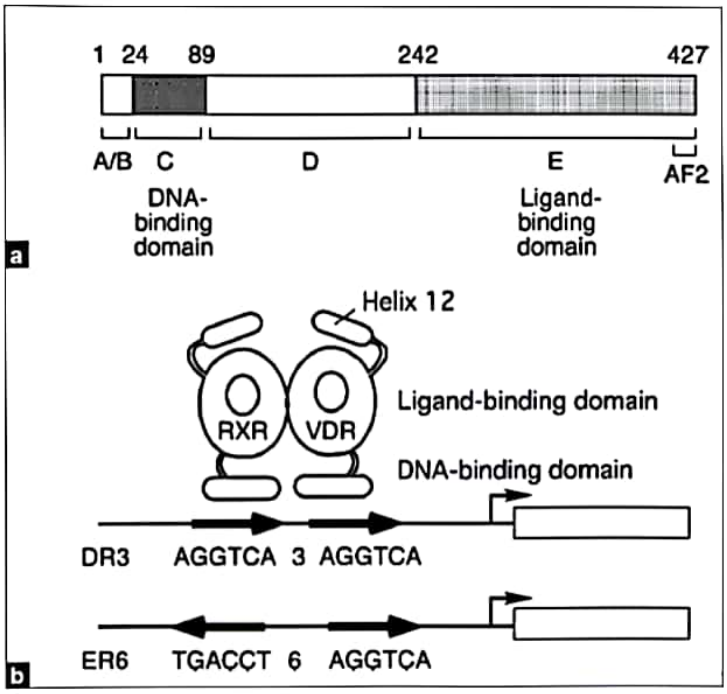

Figure 2.3.4 Vitamin D receptor polymorphism
Source: Anand N, Chandrasekaran S, Rajput N. Vitamin D and periodontal health: Current concepts. J Indian Soc Periodontal. 2013;17(3):302–8

2.3.5 Mechanism of Action of Vitamin D

According to Laird et al, bone remodeling—which includes bone resorption by osteoclasts and bone matrix synthesis by osteoblasts—is required for tooth mobility. In vitro studies using a variety of stimuli, including vitamin D, have examined osteogenic differentiation and matrix mineralization.

According to Laird et al., vitamin D increases bone resorption via boosting osteoclast quantity and activity. In the presence of receptor activator nuclear factor ligand B (RANKL) and macrophage colony-stimulating factor (m-CSF), osteoclasts carrying VDR, CYP27B1, and 25OHD showed increased development. However, this same study showed that their ability to absorb was inhibited. To activate RANK on osteoclasts and their hematopoietic progenitors, osteoblasts produce membrane-associated RANKL. Osteoclast precursors are stimulated to differentiate into osteoclasts and are actively supported by this cell-to-cell contact and m-CSF, which is also produced by osteoblasts. [27]

2.4 Tissue Changes on Tooth Movements on Orthodontics

2.4.1 Periodontal Ligament

According to Chambial et al., the periodontal ligament is a type I collagen fiber-connected connective tissue that connects the cementum with the alveolar bone. It shares some characteristics with tendons and other ligaments in the appendicular skeleton (Sharpey fibers). The periodontal ligament measures 0.15 to 0.38 mm wide when it is in homeostasis (depending on the type of tooth). The two primary purposes of the periodontal ligament are to deliver blood vessels and nutrients to the cementum, alveolar bone, and the periodontal ligament itself, as well as to transmit and absorb mechanical stress. [20]

The periodontal ligament consists of several cells, namely fibroblasts, osteoblasts, osteoclasts, and cementoblasts. In addition, there are still other cells, such as macrophages, and usually there are also remnants of Malassez cells. The periodontal ligament also contains capillary blood vessels in the form

of a plexus. Each cell that makes up the periodontal ligament has its role, namely:

a. Fibroblasts: are responsible for changes in the extracellular matrix and have a relatively high metabolic activity.

b. Osteoblasts: cells located on the surface of bones are responsible for the formation of the organic matrix of bone which then undergoes mineralization and eventually becomes bone. These cells also play a role in activating osteoclasts to bypass the formation of various cytokines. These cells are regulators of bone homeostasis. When bone minerals surround an osteoblast, it becomes an osteocyte.

c. Osteocytes: one osteocyte and another osteocyte are connected through cytoplasmic extensions located in the bone canaliculi. They are thought to be responsible for detecting the presence or absence of forces affecting the bone.

d. Osteoclasts: these cells have many nuclei. These cells are derived from blood monocytes and are responsible for bone resorption.

e. Cementoblasts: these cells function to form cement.

f. Cementoclasts: these cells function to absorb cement.[16]

2.4.2 Alveolar Bone

Alveolar bone, according to Jiang et al., is a mineralized connective tissue made up of organic matrix, minerals, and water. The composition of alveolar bone is made of 40% water, 37% organic matrix, primarily collagen, and 23% mineralized tissue. Loss of alveolar bone can result from tooth loss. Trabecular bone constitutes the bulk of the alveolar bone. It also has an inner (lingual) and outer (labial) cortical plate, as well as a compact bone plate known as the lamina dura that is situated next to the periodontal ligament. The periodontal

ligament enters the lamina dura and attaches to the alveolar bone, with the cementum serving as its other endpoint.[29]

2.4.3 Tissue Change Mechanism on Tooth Movements on Orthodontics

According to Rahardjo, the periodontal ligament plays a very important role in the process of orthodontic tooth movement because the ability of this tissue to respond to the mechanical forces it receives will cause remodeling of the alveolar bone so that the teeth can move. Without the periodontal ligament (e.g., in the case of ankylosis), the tooth will not be able to move. Broadly and concisely, it can be said that if an adequate force is applied to the teeth, the pulled periodontal ligament will experience bone apposition and, in the area of pressure, will experience bone resorption. In fact, what is happening is a complex event involving both cellular and molecular reactions in the periodontal ligament. [16]