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THE RELATIONSHIP BETWEEN THE VITAMIN D RECEPTOR GENE POLYMORPHISM AND OSTEOPOROSIS: A REVIEW ARTICLE

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Abstract

Osteoporosis, characterized by a decline in bone mineral density (BMD), significantly heightens the risk of bone fractures. The majority of osteoporosis cases are diagnosed in the aged middle-aged, and above. Due to its associations with morbidity and mortality, diminished quality of life, and the substantial costs associated with its treatment, osteoporosis has emerged as a prominent public health concern. The vitamin D receptor gene (VDR) is extensively expressed across various human tissues. Furthermore, four single nucleotide polymorphism (SNP) sites within the VDR gene, namely FokI, BsmI, ApaI, and TaqI, have been associated with osteoporosis. The correlation between osteoporosis and VDR gene polymorphism exhibits significant variability in different racial and ethnic groups. Therefore, using larger samples in collaborative studies involving multiple countries, regions, races, and ethnicities could contribute to a better understanding of the association between VDR gene polymorphism and osteoporosis. This review aims to synthesize current evidence on the relationship between VDR gene polymorphisms (FokI, BsmI, ApaI, and TaqI) and osteoporosis susceptibility, with a focus on elucidating the role of racial and ethnic heterogeneity in these associations.

Keywords: osteoporosis; polymorphism; vitamin D receptor gene

1. Introduction

Osteoporosis, as a bone disease, is characterized by compromised bone strength, making individuals more susceptible to fractures(1). It has become a major public health problem due to the associated morbidity, mortality, reduced quality of life, and high treatment costs(2-4) .

The main clinical symptoms of osteoporosis in the middle and late stages are height decrease, hunched posture, pain in the back or neck, and fractures of the bone. These symptoms worsen as bone mass deteriorates(5-6). In severe cases, osteoporosis can result in significant pain, impaired mobility, and one or more fractures(7). Osteoporosis is often described as a "silent" disease, and in the initial phases of bone loss, there are no typically discernible symptoms; however, early signs of osteoporosis may include weaker grip strength, receding gums, and more brittle fingernails(7).

Osteoporosis is a result of a number of risk factors such as aging, low BMD, diet habits, physical inactivity, medication use, smoking, alcohol abuse, and concomitant diseases(8-9). Additionally, genetic factors have been found to play a crucial role in causing osteoporosis(10-12). One of these genes is VDR, which has been identified as a crucial factor in the modulation and progression of BMD and osteoporosis(13-14). This review aims to highlight the relationship between VDR polymorphism and osteoporosis.

2.Definition and diagnosis of osteoporosis

The WHO defines osteoporosis as a systemic bone disease marked by bone microstructure degradation, decreased bone mass, decreased bone strength, increased fragility of the bones, and an increased risk of bone fractures(15). The WHO operational definition of osteoporosis as a BMD at the hip or lumbar spine is 2.5 standard deviations (SD) lower than the mean value observed in young, healthy women, which is indicated by a T-score of -2.5 SD or lower(16-17). A more stringent criterion establishes "low bone mass" or "osteopenia" as a T-score between 1 and 2.5 SD below the average. The phrase "severe" or "established" osteoporosis pertains to osteoporosis diagnosed based on the occurrence of one or more confirmed fragility fractures(16-17). According to this definition, Dual-energy X-ray absorptiometry (DXA) fracture scans are used to assess BMD(18). DXA utilizes radiation to measure the quantity of calcium and other minerals in a particular region of the bone, usually the hip and spine, which are the most frequently fractured bones(18).

In fact, most osteoporotic fractures occur in people who have negative tests (19). As a result, the potential influence of broad BMD tests on fracture burden is insufficient, and numerous organizations do not recommend BMD population screening(19). Furthermore, despite the fact that it is frequently used by these organizations, the WHO definition of osteoporosis is not universally recognized (20)

3.1Vitamin D

There are two types of vitamin D, which are fat-soluble: vitamin D_2 and vitamin D_3(21-22). Vitamin D_3 is produced by UVB irradiation of 7-dehydrocholesterol in the skin(23). Vitamin D_2 is obtained from plants and is created externally by ergosterol irradiation. It is subsequently ingested into the body through dietary means (24). In the liver, vitamin D originated from sun radiation and dietary sources undergo a procedure that yields 25-hydroxyvitamin D [25-(OH)D], also known as calcidiol. This particular kind of vitamin D predominates in the bloodstream and is employed to estimate A person's vitamin D level(23; 25). To achieve the biological activity, 25-(OH)D must be further hydroxylated in the kidneys, resulting in active 1,25-(OH)2D3(26) (Figure 1 shown in the appendix). The physiological actions of calcitriol are facilitated through its binding to VDR(27). Calcium and phosphate concentrations are regulated by the hormone calcitriol, which circulates in the blood and

encourages normal bone development and remodeling(23; 25).

3.2 VDR

VDR is a protein that results from the VDR gene's expression. It is a member of the nuclear receptor family of transcription factors and acts as a receptor for the active form of 1, 25-(OH) $_2D_3$(28). The highly conserved NH2-terminal DNA-binding domain (DBD) and the variable COOH-terminal ligand-binding domain (LBD) make up VDR's two fundamental functional domains(29). The DBD is a zinc-rich domain that is rich in cysteines(30-31). The LBD contains at least 12 alpha helices and 3 beta folds(32-33). Nuclear vitamin D receptors and membrane vitamin D receptors are the two main subtypes of VDR(34-35). The VDR can be divided into six functional areas from the N-terminal to the C-terminal (31-33). These functional areas have different functions but cooperate (31-33). The six functional zones between the terminals N and C of VDR are A/B, C, D, E, F, and H(31-33). They each have different functions, such as transcriptional activity, coactivating protein binding, DNA binding, etc (31-33). The A/B region is the shortest functional region in the VDR and contains sites where transcriptional activation and coactivation proteins bind(31-33). Region C is a rich transcriptional activation region that can bind to coactivating proteins(32-33). Region D is the core area and DNA-binding domain of VDR, and it is responsible for attaching VDR's DNA to the target gene's promoter region(31-33). The E region is a variable region that has a significant impact in regulating the transcriptional activation activity of VDR and the binding ability of coactivating proteins(32-33). Region F is involved in the composition and operation of the LBD of VDR and is the conformationally stable region of VDR(31-33). Region H is the LBD terminal region of VDR and an important site for VDR to bind to coactivator proteins(31-33). When vitamin D3 is converted into its active form in the body, it binds to VDR and controls the expression of multiple genes involved in calcium and phosphorus metabolism as well as other physiological processes(36).

3.3 Correlation between osteoporosis and the VDR gene

The VDR gene (Figure 2 shown in the appendix) is the effector gene for vitamin D. The VDR gene is situated on chromosome 12 and comprises 9 exons and 8 introns(37). It is about 100kb in length and has more than 100 SNP sites(38). VDR gene polymorphism is linked to osteoporosis risk, but there are differences between races and genders(39). Four polymorphic loci of VDR gene may be closely related to osteoporosis, namely ApaI, FokI, TaqI, and BsmI (40). However, the effects of these sites may vary in different populations(40).

3.3.1VDR gene ApaI and osteoporosis

ApaI is situated within the 3 '- regulatory region of the VDR gene (intron 8)(41-42). This polymorphism leads to cytosine substitution by adenine (C→A), which affects VDR mRNA stability and protein translation(41-42). In a study examining the relationship between VDR polymorphism and osteoporosis susceptibility in postmenopausal Saudi women, it was found that the heterozygous AC of ApaI was significantly more common in the osteoporosis group than in the control group ($p < 0.05$), the heterozygous AC of ApaIwas significantly associated with an increased risk of osteoporosis (43). The homozygous ApaI genotype was significantly associated with an increased risk of osteoporosis, according to research about the genetic susceptibility of the VDR gene ApaI to osteoporosis in Egyptian women(44). Individuals who were homozygous had lower BMD levels, whereas those who were heterozygous showed greater BMD levels(44-45). Zhanwen et al. determined VDR gene ApaI polymorphism in 155 osteoporosis patients and 113 controls in Shandong Peninsula region of China and found that VDR gene ApaI polymorphism is correlated with primary osteoporosis in the Han people in Shandong Peninsula region(45). The ‘a’ allele is the susceptible gene in women 65 years and older, and the ‘aa’ genotype is susceptible(45). Zhao et al. found that postmenopausal Chinese Han women with type aa had considerably less lumbar vertebral bone density than type Aa women(46). Xie et al.

studied changes in SNP of the VDRI, VDRII-1, and VDRII-2 genes between osteoporosis patients and normal people from different regions in China, including 417 people with primary osteoporosis from Beijing (236), Wuhan (123), and Fujian (61), as well as 60 normal adults as controls, and the study found significant variations in SNP in the VDRI, VDRII-1, and VDRII-2 genes between normal people and osteoporosis patients, mainly between Beijing and Wuhan(47).

Dundar investigated 136 postmenopausal women and concluded that the ApaI gene polymorphism in the VDR may be an important factor that affects post-menopausal women's lumbar BMD(48). The ApaI polymorphism of the VDR gene is linked to osteoporosis in post-menopausal women, according to Marozik's study of 54 post-menopausal women in Belarus who had the condition and 77 women who were controls(49). However, Linyan et al. analyzed 179 cases of ApaI polymorphism of the VDR gene in Mongolian women in Inner Mongolia, and the analysis showed that the VDR ApaI genotype did not correlate with arm BMD in all populations tested(50). There weren't any statistically obvious disparities in distributions of genotypes and alleles between the bone mass reduction group and the normal group, suggesting that ApaI polymorphism of the VDR gene may not be correlated with osteoporosis(50). Dabirnia et al. conducted a case-control study on Iranian menopausal women and found that the polymorphism of ApaI was unrelated to the occurrence of osteoporosis in Iranian menopausal women(51). Jirong et al. measured the BMD of L4-2, Ward triangle, and greater trochanter of the femoral neck in 592 post-menopausal women, and they found that there were no statistically significant disparities in BMD of three genotypes 'AA', 'Aa', and 'aa' in these regions (52). Through meta-analysis, Shen et al. likewise came to the conclusion that there was no significant correlation between post-menopausal women's fracture risk and ApaI polymorphism (53).

3.3.2 VDR gene FokI and osteoporosis

The VDR gene's FokI site is at the start of transcription at exon 2 of the gene initiation region's 5'end, resulting in the replacement of thymine in the VDR gene's second exon by cytosine at the first ATG site (T→C) to form three shorter and more active proteins(42; 54-55). This site is the only SNP site known to date to have an effect on the protein structure of VDR(54; 56). Some scholars have found that the FokI polymorphism is related to calcium absorption rate, body BMC, and body BMD, and the FF genotype is significantly greater than the ff genotype, suggesting that the VDR gene affects BMD accumulation by acting on calcium absorption from the small intestine calcium absorption(57-58). Mohammadi et al found that the VDR gene's Fok I may influence bone mass and foretell osteoporosis in the Iranian population after a study of 1032 cases over 20 years of age, and the ff genotype is a protective factor for postmenopausal women (59). Xin et al. concluded that the VDR gene's FokI polymorphism may be related to BMD in Beijing, China, by studying 230 Han men aged 20-80 years (60). A controlled study of 105 Thai individuals with postmenopausal osteoporosis and 132 normal postmenopausal participants showed that the polymorphisms of the FokI loci and a significant connection between osteoporosis, T appears as susceptibility alleles in Thai women, FokI may be the occurrence of potential molecular biomarkers(61). Ioannidis et al. conducted a meta-analysis about 3243 Asian women and found that FokI polymorphism is related with decreased lumbar bone density(62). Yasovanthi et al. confirmed that FokI polymorphism is related to low bone mass and BMD by studying Indian women and found that FokI gene polymorphism is a significant risk factor for osteoporosis(63). Changxin et al. studied 118 elderly patients with osteoporosis and a 140-healthy control population of Li nationality in Hainan. They found that the risk of osteoporosis with the 'Ff' genotype was 1.13 times greater than that with the 'FF' genotype, and the risk of osteoporosis with the 'ff' genotype was 1.64 times greater than that with the 'FF' genotype(64). However, Cusack et al. conducted a survey on 242 Danish girls who were 11.42 years old on average in 2006 and found that there is no connection between BMD and the FokI polymorphism(65).

3.3.3 VDR gene TaqI and osteoporosis

The TaqI locus of the VDR gene is situated in exon 9, and this polymorphism leads to a mutation of ATC-ATT at codon 352 of the VDR gene. This SNP site has been demonstrated to impact mRNA stability and modify the biological function of VDR, but since the synonymous mutation does not alter the sequence of amino acids in VDR, this is a T/C substitution in which the T allele creates a restriction endonuclease recognition site for TaqI and the C allele does not(41-42; 66). Melissa Kow et al. reported that the CC genotype frequency of the TaqI polymorphism was significantly lower in the control group compared to the osteoporosis cohort ($p < 0.05$)(38). Notably, this variant demonstrated a significant association with lumbar spine BMD in White British men, suggesting a potential protective role of the C allele against bone loss(38). A study of 611 postmenopausal Polish women (median age: 65.82 ± 6.29 years) demonstrated that TaqI genotypes exhibited significant differences in BMD, with AG heterozygotes showing the lowest median BMD values compared to homozygous groups ($p < 0.05$)(67). Wang et al studied 1212 cases of post-menopausal women with osteoporosis and 404 cases of post-menopausal women without osteoporosis, and found the proportion of tag SNP of TaqI in the observation group exhibited a statistically significant increase compared to the control group(68). Dehghan & Pourahmad-Jaktaji investigated post-menopausal Iranian women over 45 years old and found that the femoral neck BMD was substantially correlated with TaqI polymorphism(69). The femoral neck BMD was lowest in patients carrying the 'TT' gene, indicating that the TaqI polymorphism might potentially indicate the risk of osteoporosis in Iranian women(69). Remes et al. found that the femoral neck BMD of middle-aged Finnish men with the TT genotype of TaqI was lower than that of the Tt and tt(70).

In Jeddah, Saudi Arabia, a case-control investigation comprising 73 osteoporosis individuals and 73 normal controls disclosed a notable increase in the frequency of the homozygous (tt) TaqI genotype among individuals with osteoporosis compared to the control group (41). Xiaodan et al. studied the connection between the BMD and VDR gene polymorphism in Han Chinese children aged 0-6 and showed that different alleles at the TaqI site had no connection with BMD of Han children between the ages of 0 and 6(71). Dan et al. founded no statistical significance between the polymorphism of TaqI and osteoporotic vertebral fracture(72).

3.3.4 VDR gene BsmI and osteoporosis

The BsmI is situated proximate to the ApaI locus in the 3' untranslated region (intron 8). The nucleotide adenine is replaced by guanine (A→G) in these polymorphisms(73-74). This SNP locus plays a role in modulating mRNA stability(73-74). Sainz et al. earlier reported the distribution of polymorphic sites of Bsm I in 100 healthy preadolescent girls in the Americas in 1997 and found that the femoral neck's bone density in girls with "aa" and "bb" genotypes exhibited a 2% to 3% elevation compared to girls with "AA" and "BB" genotypes($P<0.05$), additionally 8%-10% higher lumbar bone density ($P<0.05$), so the VDR allele can predict the BMD of the femoral region and the lumbar vertebral column(75). Changxin et al. studied 118 elderly patients with osteoporosis of the Li ethnic group in Hainan, China, and 140 healthy control population, and discovered that individuals with the Bb genotype were 1.87 times more likely to have osteoporosis than people with the BB genotype, while those with the bb genotype were 4.21 times more likely to develop osteoporosis than people with the BB genotype(64). It is suggested that the polymorphism of the Bsm I enzyme restriction site is related to osteoporosis susceptibility in the elderly(64). Ahmad et al. conducted a case-control investigation including 254 postmenopausal osteoporosis individuals and 254 normal individuals in Northern India and revealed that the individuals with the bb genotype of BsmI exhibited the lowest bone mineral density in both the lumbar spine and hip joint(76). Langdahl et al. revealed an increased frequency of BB + Bb genotypes in BsmI among individuals with osteoporotic fractures, and those who possess the bb genotype exhibited elevated BMD throughout the whole hip as well as the

intertrochanteric area(77). The BsmI polymorphism was correlated with reduced BMD at the hip and showed a tendency to be linked with osteoporotic fractures(77). The BsmI genotype showed a significant variance in BMD among young Japanese women, according to research by Yuri Sakamoto et al. on 499 women between the ages of 20 and 24. The bone sono-assessment score of the BsmI AA genotype was also found to be lower than that of the other genotypes (78). However, Wang Qi et al. studied 323 cases of Tibetan women in China and found that the BsmI site's polymorphism in Tibetan women was not correlated with bone mass(79). Li Shengqiang et al. revealed that there were no statistical differences in BMD across various genotypes at the BsmI polymorphism loci in the lumbar spine, greater trochanter and Ward's area among elderly Han Chinese men in Fuzhou, China(37). A case-control study of Polish women (N=442: 197 osteoporosis, 98 osteopenia, 147 controls) found no significant association between the VDR rs1544410 (BsmI) polymorphism and osteoporosis risk via real-time PCR genotyping ($p > 0.05$)(80). A meta-analysis encompassing 2969 people, comprising 1580 osteoporosis individuals and 1389 control subjects, indicated that the presence of the BsmI polymorphism did not exhibit any significant association with the risk of osteoporosis within the Han Chinese people(81). Another meta-analysis of 4485 osteoporosis cases and 5490 controls found that BsmI polymorphism was obviously related to osteoporosis susceptibility in Caucasians, without any significant association in Asians, and that the BsmI genotype exhibited an elevated risk for postmenopausal osteoporosis among Caucasians, while such an association was not observed within the Asian population(82).

4. Conclusion

Current studies on the relationship between osteoporosis and VDR gene polymorphism have shown contradictory results. These discrepancies may be due to the ethnic differences among the participants from different geographical areas. Moreover, the variations of the osteoporosis risk factors such as age, gender, postmenopausal duration, dietary habits, lifestyle, and levels of physical activity among participants. Furthermore, variations in the definition of osteoporosis, and methods used to measure BMD, inclusion criteria, sample size, and other aspects of the studies may also cause variances in the analysis results.

Therefore, to clarify the actual relationship between VDR gene polymorphisms and osteoporosis, future research must prioritize large-scale, multinational collaborative studies with longitudinal designs, encompassing diverse populations across regions and ethnicities. Standardizing protocols for participant inclusion/exclusion criteria, phenotyping, BMD assessment, and covariate reporting is critical to minimize methodological biases. Simultaneously, emerging genomic and computational technologies offer unprecedented opportunities to address current limitations: genome-wide association studies (GWAS) can extend beyond candidate-gene approaches to identify polygenic interactions and population-specific risk loci, while machine learning and Bayesian meta-analyses disentangle gene-environment interactions and harmonize heterogeneous datasets. Next-generation sequencing (NGS) further resolves gaps in SNP-centric research by characterizing rare VDR variants and epigenetic modifications. To maximize translational impact, consortia should adopt federated learning platforms to securely integrate multi-center data, ensuring privacy while enhancing statistical power for developing ethnicity-specific prevention strategies. Integrating multi-omics approaches—combining genomics, epigenomics, and metabolomics—will systematically elucidate the mechanistic links between VDR polymorphisms and bone metabolism, transforming fragmented genetic associations into actionable targets for personalized interventions. By synergizing standardized global collaboration with cutting-edge technologies, this dual approach holds promise for reducing the global burden of osteoporotic fractures and advancing precision medicine in aging populations.

Conflicts of Interest: The authors declare that they have no conflict of interest.

Appendix:

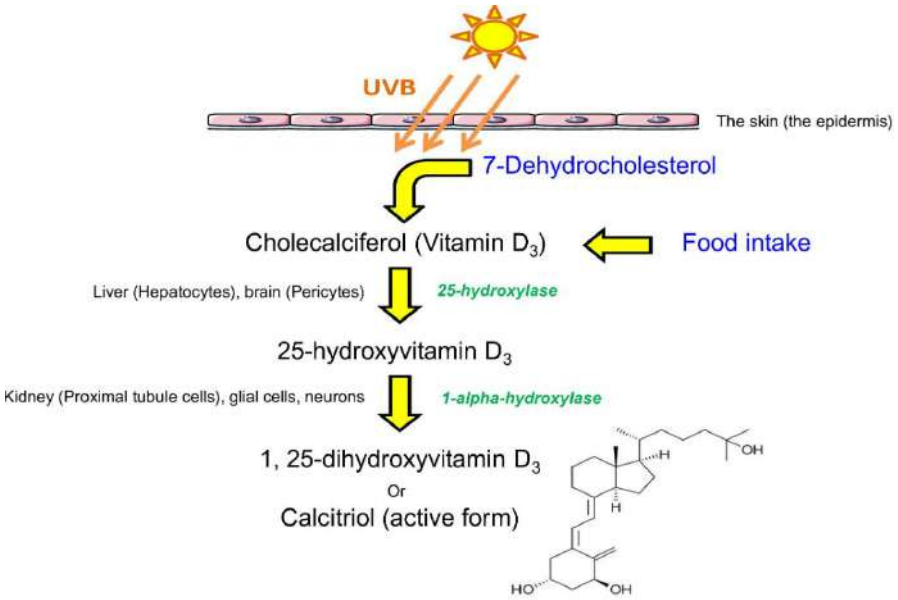

Figure 1. The generation path Synthesis of 1,25 -hydroxyvitamin D3 or calcitriol (active form) (83). reference under the terms of the Creative Commons Attribution License (CC BY), which allows the use, distribution, or reproduction in other forums as long as the source and original work are properly acknowledged.

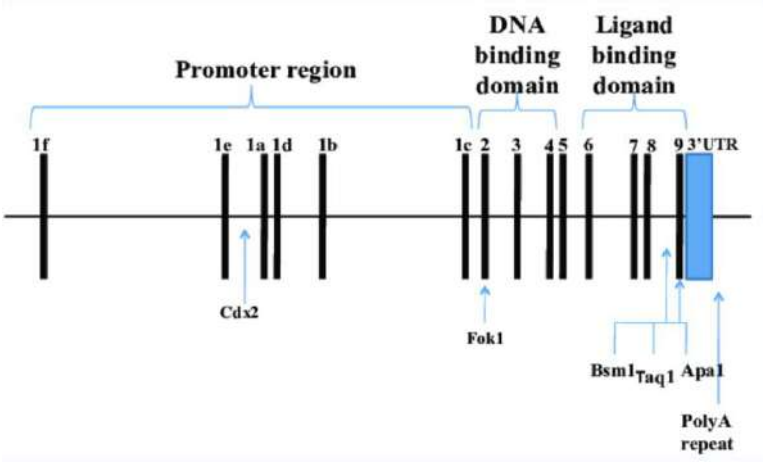

Figure 2. Structure of Vitamin D Receptor (VDR) Gene(84). reference under the Creative Commons Attribution 4.0 License, which allows for the copying, distribution, transmission, and adaptation of the published work as long as the original work and source are properly cited.

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