Introduction

Prostate cancer is one of the most common malignancies which harm men’s health in United States and Europe; it is also the second largest tumor causing male deaths. It is estimated by American Cancer Society that the new prostate cancer patients were 192,000 cases in 2009 in U.S, and 27,000 cases died of prostate cancer (Jemal et al., 2009). In Europe, new cases of prostate cancer were about 260 million every year, the patients with prostate cancer accounted for 11% of total male patients with cancer, and the patients died of prostate cancer accounted for 9% of total male patients died of any cancer. With the aging of our population and the improvement of diagnostic techniques, the morbidity and mortality of prostate cancer showed an increasing trend. The morbidity of prostate cancer has exceeded lung cancer, and ranks first; the mortality of prostate cancer ranks second after lung cancer (Ming et al., 2009). The pathogenesis of prostate cancer is complex, it is widely recognized that age, race and family history of cancer are the risk factors (Zhang et al., 2012). With the development of molecular biological techniques, it is recognized that the pathogenesis of prostate cancer may be the result of role of multiple genes (Schaid et al., 2004).

The human vitamin D receptor (VDR) is a nuclear receptor gene with 75 kb, located in the long arm of chromosome 12, and consists of 11 exons and 11 introns (Zmuda et al., 2000). Schwartz, et al believed that low dose ultraviolet radiation was a risk factor for prostate cancer (Schwartz et al., 1990; Moon et al., 2005). The skin produced vitamin D after exposure to UV, then the vitamin D was hydroxylated to D3[25(OH)D3] in liver, next, the D3[25(OH)D3] was transformed to biologically active D3[1,25(OH2)D3] in kidney; in addition, VDR is not only expressed in kidney and bone, it is also detected in prostate cells(Hidalgo et al.,2007). It has been reported that 1,25(OH)2D3 could promote differentiation, enhance immune regulation, and inhibit cell necrosis, tumor invasion and metastasis in prostate (Krishnan et al., 2003; Bao et al., 2006), all these biological effects were mediated by VDR (Schwartz 2012). Corder et al. (1993) believed that 1,25(OH)2D3 in peripheral blood was decreased significantly in prostate cancer susceptible population. Lots of gene polymorphism of VDR3’ end have been discovered so far, such as ApaI, BsmI, TaqI, Tru9I, EcoRV, in which TaqI (rs731236) has been widely studied. TaqI polymorphism located at codon 352, the wild-type T allele was transformed into the mutant C allele (ATT-ATC) (Vieira et al., 2006).

There was controversy in the researches of VDR and susceptibility to prostate cancer; in this study, we collected all literatures about VDR gene TaqI polymorphism and Asian men with prostate cancer, and analyzed them with Meta-analysis to evaluate comprehensively the relationship of VDR gene TaqI polymorphism and genetic susceptibility to prostate cancer in Asian men.

Materials and Methods

Literature collection

Search the key words “VDR” or “Vitamin D receptor”, “polymorphism” and “prostate cancer” in...
Pubmed database; at the same time, search the Chinese key words in VIP, WanFang, CBM and CNKI database until September, 2010. Collect the potentially relevant literatures through manual literature retrospective way.

Inclusion and exclusion criteria
The inclusion criteria are as followings: 1) the study is based on analysis of the relationship of VDR gene TaqI polymorphism and genetic susceptibility to prostate cancer; 2) case-control study; 3) genotype frequency distribution; 4) age matching between treatment group and control group; the literature must be full-text. The exclusion criteria are as followings: 1) no control group; 2) no genotype frequency distribution; 3) duplicated research; 4) there is a serious bias.

Data extraction and statistical analysis
Each article was evaluated independently by two reviews; the relevant information was extracted and inputted to computer to create the database. The relevant information included the first author, publication year, countries, the source of controls, genotype frequency and the total number of treatment group and control group.

Three models were chosen to analyze the VDR gene TaqI polymorphism and genetic susceptibility to prostate cancer: allele model (C vs. T), heterozygous model (CT vs. TT) and dominant model (CC+CT vs. TT). In this study, the included studies were analyzed with heterogeneity test (q test), then select the appropriate combination method according to q test results: if $P_{\text{heterogeneity}} > 0.05$, use fixed effect model to combine the studies. The mutant allele C of TaqI might be related to reduce prostate cancer risk compared to allele T ($OR=0.81$, 95%CI: 0.70-0.94, $P_{\text{heterogeneity}}=0.578$, Figure 1). There was no correlation between wild-type TT genotype and mutant CT genotype of TaqI ($OR=0.86$, 95%CI: 0.74-1.01, $P_{\text{heterogeneity}}=0.820$), however, TaqI carried the dominant CC+CT genotype might be associated with decrease of prostate cancer risk compared to wild-type TT genotype ($OR=0.84$, 95%CI: 0.73-0.97, $P_{\text{heterogeneity}}=0.702$, Figure 2).

### Table 1. General Data of Study

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<tr>
<th>author</th>
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<th>Country</th>
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<th>control</th>
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Figure 1. A Meta Analysis of Relation Between Taq I Polymorphism and Prostatic Cancer Susceptibility. (C vs T)
VDR gene TaqⅠ polymorphism and genetic susceptibility to prostate cancer in Asian men. We combined analyzed 10 case-control studies through a rigorous screening with Meta-analysis (1141 cases of prostate cancer, 1685 cases of control). The results showed that C vs. T (OR=0.81, 95%CI: 0.70-0.94, \(P_{\text{heterogeneity}}=0.578, P=0.006\)); CC+CT vs. TT (OR=0.84, 95%CI: 0.73-0.97, \(P_{\text{heterogeneity}}=0.702, P=0.020\)), suggesting individuals carried the mutant allele C have less risk of suffering from prostate cancer, allele C is a protective mutant.

This study has some limitations: 1) the number of cases and control is not much enough; 2) the confounding factors failed to control, such as occupation, diet, smoking and mental, which will affect the results; 3) the interaction between gene and gene, gene and environment, VDR and other locus can adjust the risk of prostate cancer; 4) the study included a small-sample study, its representative is not strong (Chaimuangraj et al., 2006); 5) the pathogenesis of sporadic and hereditary prostate is different, and we didn’t classify according to it; 6) there are four literatures which included group prostate care, group prostatic hyperplasia and control group, we combined the latter two groups as the control group, it is not the normal control group in strict terms. However, this study has three advantages: 1) the included studies are from different samples, which have a widely representation; 2) all case-control studies met the inclusion criteria; 3) there was no significant publication bias through qualitative funnel plot and quantitative Egger linear regression.

In summary, we analyzed the VDR gene TaqⅠpolymorphism and genetic susceptibility to prostate cancer in Asian men with Meta-analysis, and confirmed that VDR gene Taq allele C is a protective mutant, which could reduce the risk of suffering from prostate cancer, and might be a new marker of prostate cancer screening. However, the sample size is small in our study, it needs further multi-center and large sample studies to confirm the results. In addition, we only analyzed the VDR gene TaqⅠ locus, without considering the interaction between genes, genes and environment. In future study, we will further explore the other interaction, to facilitate the discovery of the pathogenesis of prostate cancer.

**References**


Chaimuangraj S, Thammachoti R, Ongphiphadhanakul B, et al


