Vitamin D Receptor Gene Polymorphisms and Peak Bone Mass In Southern Chinese Women Annie WC Kung, Shirley SC Yeung, KS Lau.

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Controversial results were reported on the association of vitamin D receptor polymorphisms and bone mineral density (BMD). We studied allelic frequencies of the BsmI. Apa I and Taq I restriction fragment length polymorphisms (RFLPs) in 144 normal healthy southern Chinese premenopausal women aged between 30 to 40 and correlated their peak bone mass with the VDR genotypes. In comparison to Western populations, the B allele of the BsmI site is only found in 5% of the Chinese population. BBAAtt genotype is virtually non-existent in Chinese. Except for the slightly higher BMD values at the mid-lateral L3 vertebra (13.8%, p=0.045) and at the Ward's triangle (13.3%, p=0.08) in the bb subjects, no difference could be detected at other sites between the Bb and bb subjects. The same findings were observed when comparing the Tt to tt subjects. Analysis of the VDR genotype revealed that subjects with BbAaTt and BbAATt haplotypes had the lowest peak bone mass. Their L2-4 lumbar spine, midlateral L3 vertebra, and Ward's triangle BMD was 1.04SD, 0.90SD and 0.75SD respectively lower than the bbAATT counterparts, but none of the comparisons were statistically significant. However with the low frequency of B allele, our study had limited power to detect a small difference in the BMD of the In conclusion, although VDR polymorphism is believed to affect calcium absorption, this study failed to confirm a strong relationship between VDR genotype and peak bone mass in our population with low dietary calcium intake.

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Favourable effects of cholesterol-lowering therapy on blood coagulation and fibrinolysis in non-insulin-dependent diabetic patients with dyslipidaemia

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The effects of cholesterol-lowering therapy on parameters of coagulation and fibrinolysis were assessed in a group of non-insulin-dependent diabetic patients with dyslipidaemia in a randomised doubleblind placebo-controlled trial using an HMG-CoA reductase inhibitor. The treatment group received fluvastatin 20mg daily for 6 weeks followed by 40mg daily in the following 6 weeks. Plasma total cholesterol and LDL cholesterol both decreased in the fluvastatin group (n = 37) at week 6 (p < 0.01) and week 12 (p < 0.001) compared with the placebo group (n = 20) whereas plasma triglyceride remained unchanged. In the fluvastatin group, a small but significant increase in HDL-C (p < 0.05) was observed at week 12 compared to baseline. There were also significant reductions in factor VII coagulant (FVIIc) activity (p < 0.05) and von Willebrand factor antigen (p < 0.05) at week 12 but no changes were seen in plasma fibringen concentration. Both plasmingen activator inhibitor-1 antigen and tissue plasmingen activator antigen decreased after fluvastatin treatment (p < 0.05). None of the changes in haemostatic parameters correlated with changes in fasting lipid profile after treatment except for FVIIc. The reduction in FVIIc correlated with the improvement in plasma cholesterol (r=0.43, p=0.01) and with LDL cholesterol (r=0.40, p=0.02). Treatment of dyslipidaemia with fluvastatin in patients with non-insulindependent diabetes mellitus was associated with reductions in coagulation factors and improvement in fibrinolysis in addition to its cholesterol lowering effect. The changes in the haemostatic system were potentially beneficial for these patients who were at high risk of developing cardiovascular disease.