

# VITAMIN D RECEPTOR POLYMORPHISM INTERACTS WITH SUNLIGHT EXPOSURE IN PARKINSON'S DISEASE IN THE CENTRAL VALLEY OF CALIFORNIA

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**Background:** Vitamin D is a critically important vitamin/hormone that influences numerous biological processes. Evidence from studies including our own suggests that sunlight exposure leading to vitamin D production may be inversely associated with Parkinson's disease (PD) possibly by protecting against oxidative damage.

**Methods:** We are examining whether variations in genes in the vitamin D biologic pathway coupled with long-term sunlight exposure decreases PD risk among 288 incident Caucasian PD cases and 472 Caucasian population controls enrolled in the Parkinson's Environment and Genes study. Patients were diagnosed between 1998 - 2007 and clinically confirmed as probable or possible idiopathic PD. Controls >65 years were recruited from Medicare or randomly selected residential parcels. We developed a model using geographic information systems (GIS) tools linking geocoded participant addresses with solar radiation data to estimate historical ultraviolet radiation (UV) exposure. We genotyped single nucleotide polymorphisms (SNPs) rs1544410 (BsmI), rs2228570 (FokI) and rs4334089 in the vitamin D receptor (*VDR*) based on previous studies, potential functional relevance or amino acid change.

**Results:** High lifetime UV ( $\geq 90^{\text{th}}$  percentile) weakly protected against PD (OR = 0.78, 95% CI = 0.46, 1.31), compared to levels  $< 90^{\text{th}}$  percentile. Compared to *FokI* GG genotype, GA (OR = 0.84, 95% CI = 0.56, 1.24) or AA genotypes (OR = 0.95, 95% CI = 0.56, 1.63) were not individually associated with PD. Among subjects with high lifetime UV, carriers of AA genotype had a lower odds of PD (OR = 0.04, 95% CI = 0.01, 0.84) compared to GG genotype; GA heterozygotes experienced some protection (OR = 0.28, 95% CI = 0.05, 1.65). Subjects with both high lifetime UV exposure and AA genotype were less likely to have PD (OR = 0.14, 95% CI = 0.02, 1.29) compared to subjects with combined lower UV exposure and GG genotype.

**Conclusions:** Results suggest that *VDR* variations may interact with UV exposure, a surrogate for vitamin D, in PD.