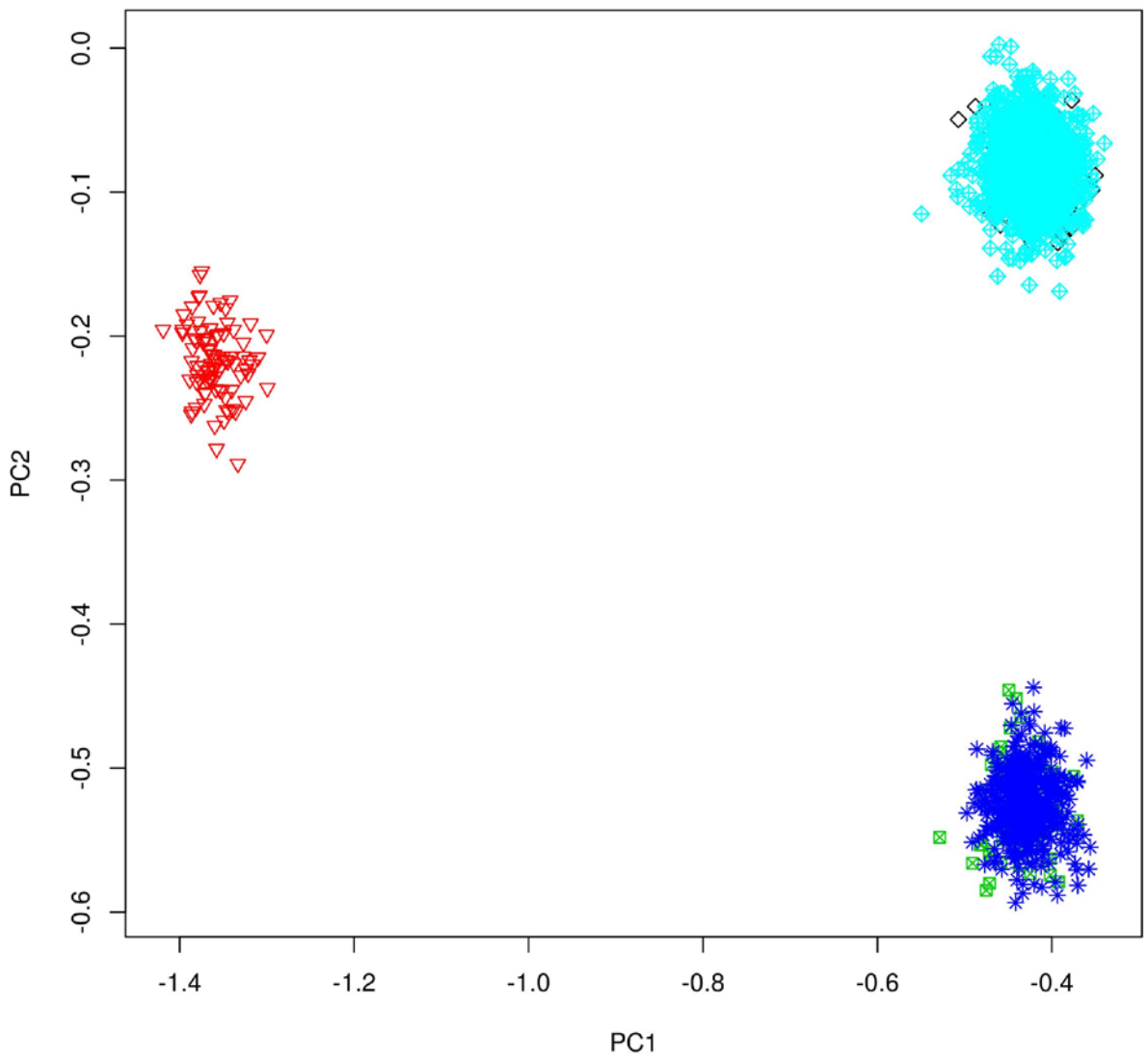
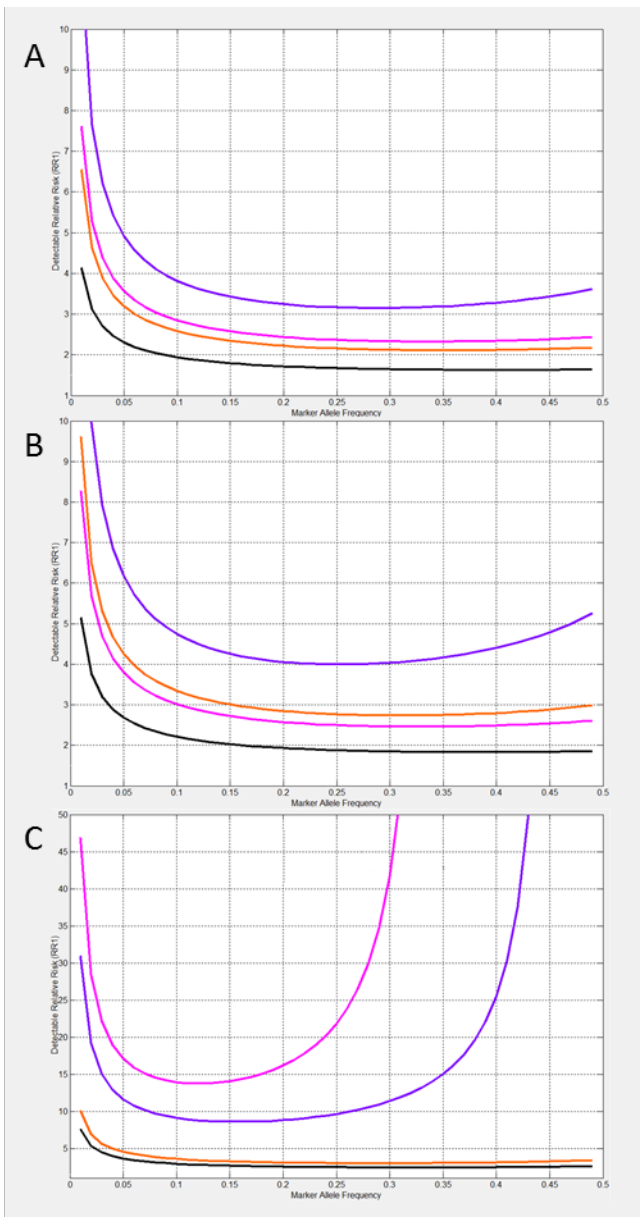
Supplementary Figure e-1: Principal components analysis.



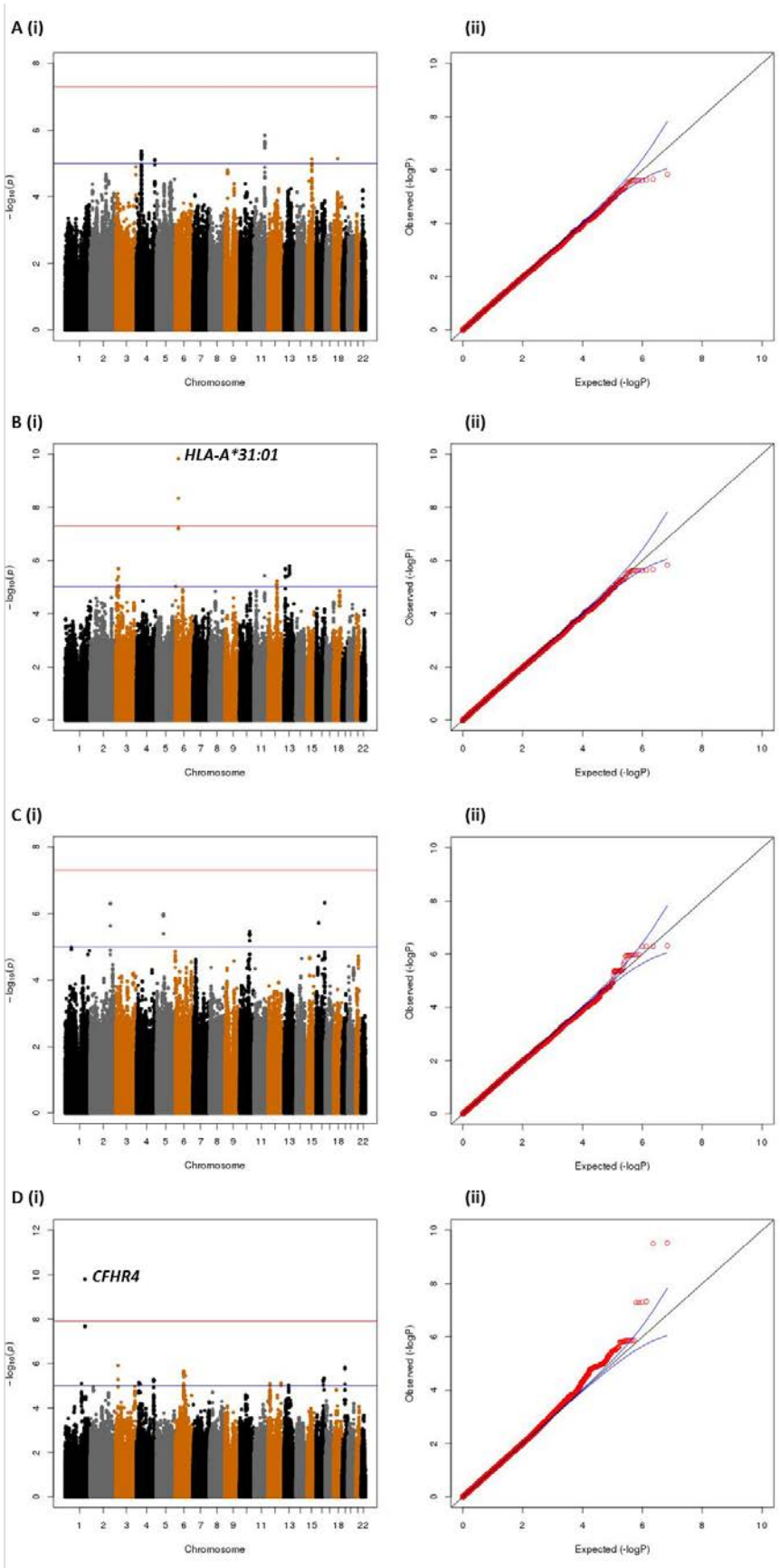
Principal components analysis of all subjects from Hong Kong (HK - dark blue) and European sites (Euro - light blue) overlaid with HapMap broad ancestral groups from Nigeria (YRI - red), Utah (CEU - diamond) and Beijing (CHB - green).

Supplementary Figure e-2: Power curves for GWAS.



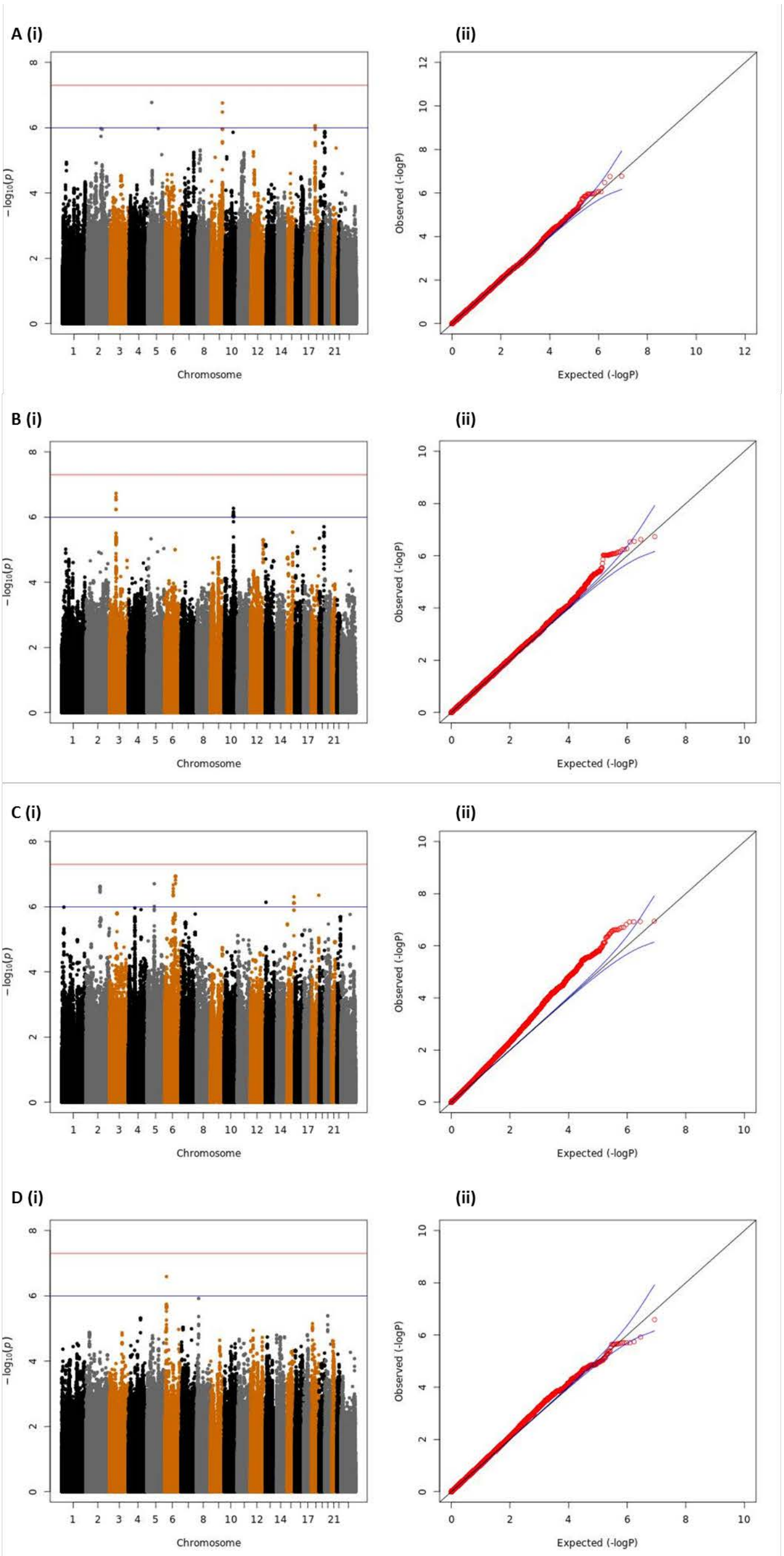
We estimated 80% power for genetic association for all MPE (black), carbamazepine-MPE (orange), lamotrigine-MPE (pink) and phenytoin-MPE (purple) in our (A) meta-analyses, (B) European cohort and (C) Han Chinese cohort.

Supplementary Figure e-3: European-ancestral cohort GWAS results.



Manhattan (i) and quantile-quantile (ii) plots for MPE vs tolerant controls, for (A) any AED (Genomic inflation factor (λ) =1.01), (B) carbamazepine (λ =1.02), (C) lamotrigine (λ = 0.99), and (D) phenytoin (λ = 1.03). *HLA-A*3101* was significantly associated with carbamazepine-induced MPE while intronic variants in *CFHR4* were significantly associated with phenytoin-induced MPE.

Supplementary Figure e-4: Han Chinese cohort GWAS results.



Manhattan (i) and quantile-quantile (ii) plots for MPE vs tolerant controls, for (A) any AED ($\lambda = 0.99$), (B) carbamazepine ($\lambda = 1.02$), (C) lamotrigine ($\lambda = 1.14$), and (D) phenytoin ($\lambda = 1.06$).