Genome-wide association studies (GWAS) of alcohol use disorders (AUD) have identified genes that influence pharmacokinetic factors (e.g. ADH1B, ADH1C), but pharmacodynamic factors have not yet been identified. Obtaining carefully diagnosed cohorts remains challenging. Instead, we obtained quantitative measures using the Alcohol Use Disorder Identification Test (AUDIT) from two population-based cohorts of European ancestry: UK Biobank (UKB; N=121,630) and 23andMe (N=20,328) and performed a GWAS meta-analysis. We also performed GWAS for AUDIT items 1-3, which focus on consumption (AUDIT-C), and items 4-10, which focus on the problematic consequences of drinking (AUDIT-P). The GWAS meta-analysis of AUDIT identified 11 associated risk loci. Novel associations were localized to genes including JCAD and SLC39A13; we replicated previous signals in the genes ADH1B, ADH1C, KLB, and GCKR. The dimensions of AUDIT showed positive genetic correlations with alcohol consumption (rg=0.78-0.96) and DSM-IV alcohol dependence (rg=0.33-0.64). AUDIT-P and AUDIT-C showed different patterns of association across several traits: AUDIT-P was positively genetically correlated with schizophrenia (rg=0.22), major depressive disorder (rg=0.26), and ADHD (rg=0.23), whereas AUDIT-C was negatively genetically correlated with major depressive disorder (rg=-0.23) and ADHD (rg=-0.10). We also identified thresholds for dichotomizing AUDIT that optimize genetic correlations with DSM-IV alcohol dependence. Coding individuals with AUDIT total score of ≤4 as controls and ≥12 as cases produced a high genetic correlation with DSM-IV alcohol dependence (rg=0.82) while retaining most subjects. We conclude that AUDIT scores ascertained in population-based cohorts can be used to explore the genetic basis of alcohol consumption and AUD.