difficulty in choosing one optimal threshold for all studies and regions.

Bayesian Model averaging Because of its computational burden for simulations, we only consider the BMA approach under a tagging genotyping scenario. This demonstrates good control of the type 1 error, even tending to be mildly conservative, as has previously been reported when posterior predictive p values are interpreted similarly to standard p values [Meng, Sep., 1994]. Despite the slightly more conservative type 1 error rates, the BMA approach appears more powerful than the PCs approach (Figure 2), which presumably reflects the greater degrees of freedom required for the PCs approach.

Sensitivity to the assumption of equal linkage disequilibrium

The proportional colocalisation test assumes identical patterns of LD in the two datasets so that the effect of a shared causal variant is proportional across any set of SNPs. To explore its sensitivity to this assumption, we considered sampling haplotypes for one dataset from a subset of European populations, and for the other dataset from a either a mixture of European populations or a mixture of European and African populations. As might be expected, for strongly admixed datasets, the control of type 1 error rate is lost, with type 1 error rates up to 8 fold that seen under the case of no mixing (Figure 3). However, it is perhaps surprising that the effect of mixing between two European populations, or mixing very small proportion of African haplotypes ($\sim 5\%$) into a mainly European population, is barely detectable at the sample sizes of 1,000 used, and indicates that the method is not very sensitive to small departures from the assumption of equal linkage disequilibrium. Of course, as with any genetic analysis, it remains sensible not to rely on this property, but to formally examine the evidence for population structure and exclude obviously outlying samples.

The case of multiple causal variants

So far, we have only considered the case of a single causal variant for each trait. But the proportional test makes no assumption about the number of causal variants, only that their effects are proportional. Figure 4 shows that in the case of eQTL data with two shared causal variants, having equal effects on each trait, type 1 error rates are still controlled. It has been reported that genes may exhibit a common cross-tissue eQTL, located proximal to the gene, as well as distinct