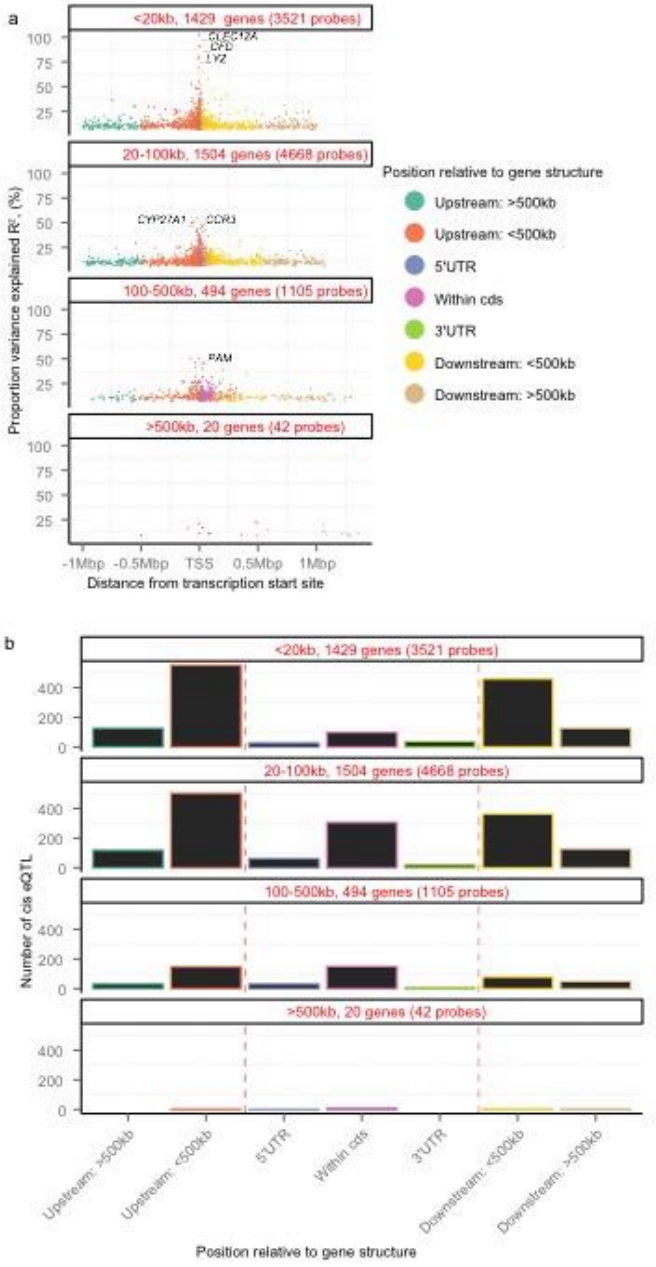
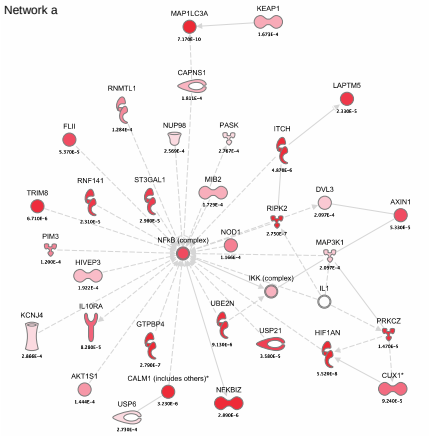
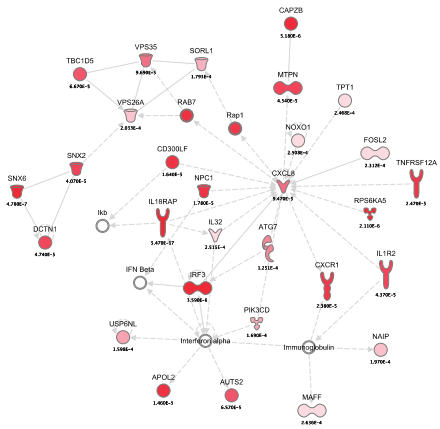


Supplementary Figure 1. Effect size and location of *cis* eQTL in neutrophils relative to transcription start site (TSS) (a) or gene structure (b), partitioned by gene size. Each point in (a) denotes the peak eQTL for a gene and is plotted according to the proportion of variance explained by the variant with strongest statistical association with gene expression and location of the variant relative to the TSS. Several genes with large effect sizes are annotated. (b) shows the number of *cis* eQTL identified in neutrophils according to the location of the peak eQTL relative to gene structures.

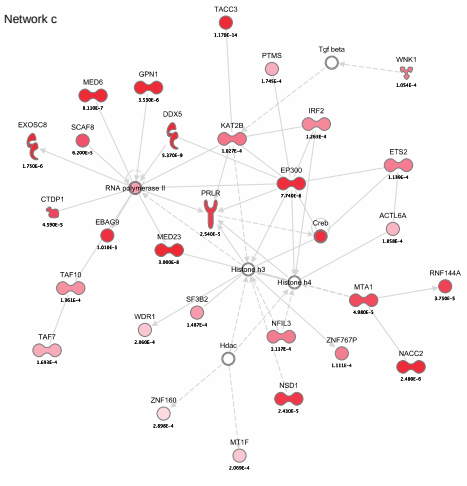
Network a



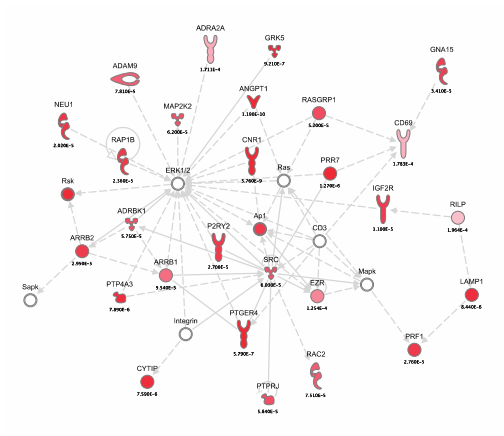
Network b



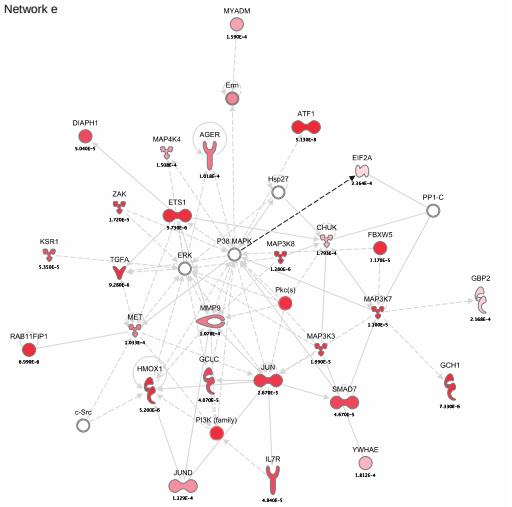
Network c



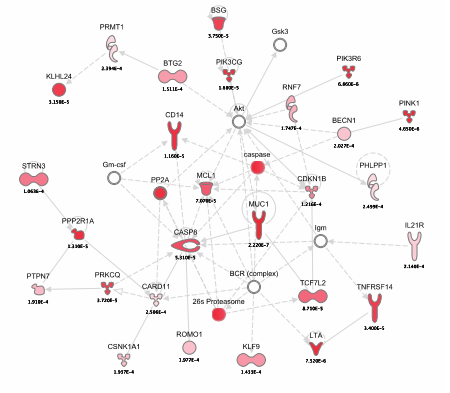
Network d

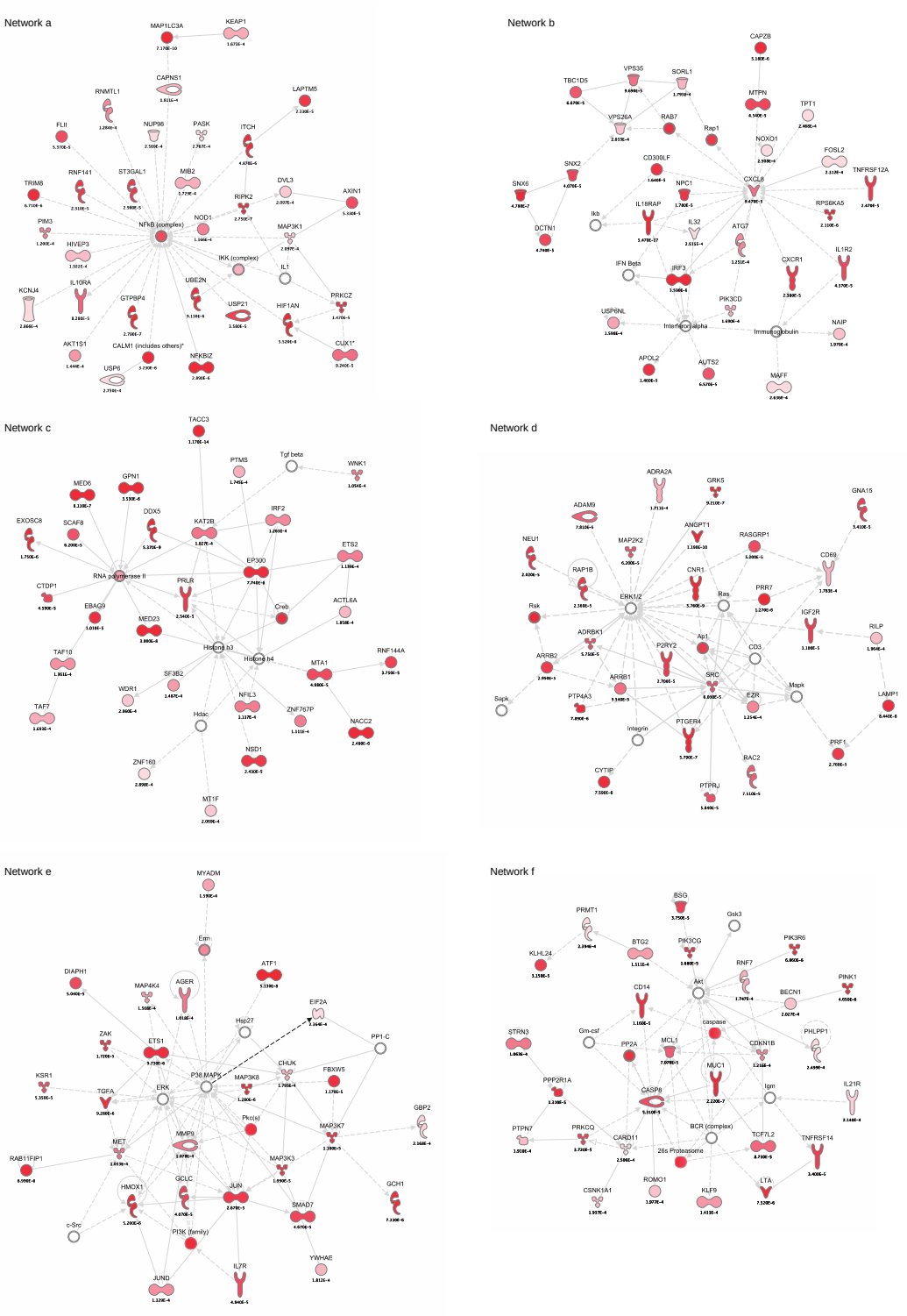


Network e



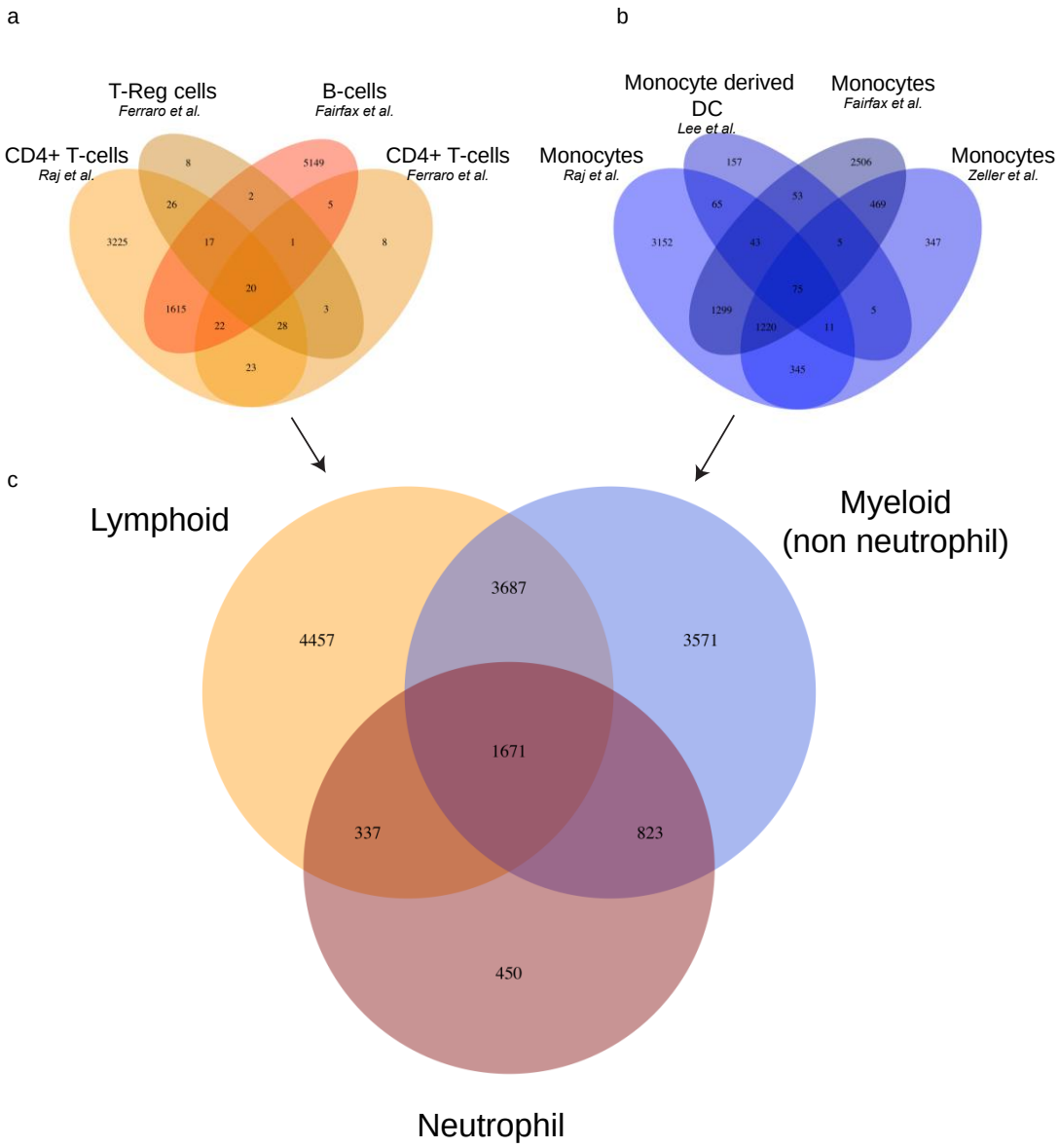
Network f



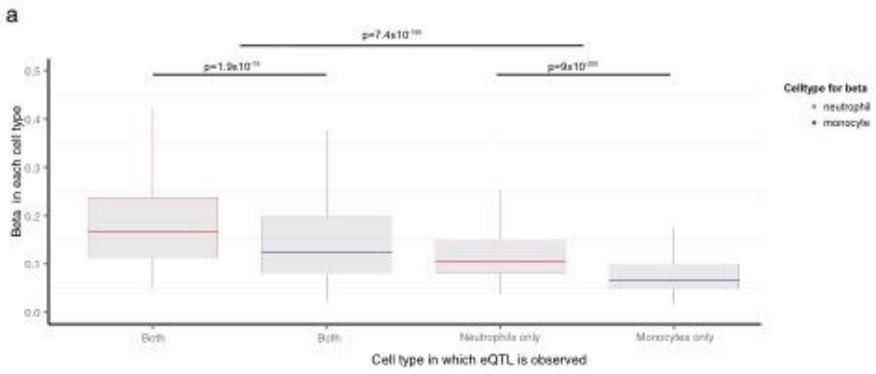


Supplementary Figure 2. Pathway analysis of genes with neutrophil specific *cis* eQTL. Amongst 975 genes with an eQTL in neutrophils only, a number of gene networks are identified as enriched for the genes showing *cis* eQTL specific to neutrophils (top 10 shown here with intensity of red shading related to p value of eQTL, gene names and p values shown above and below

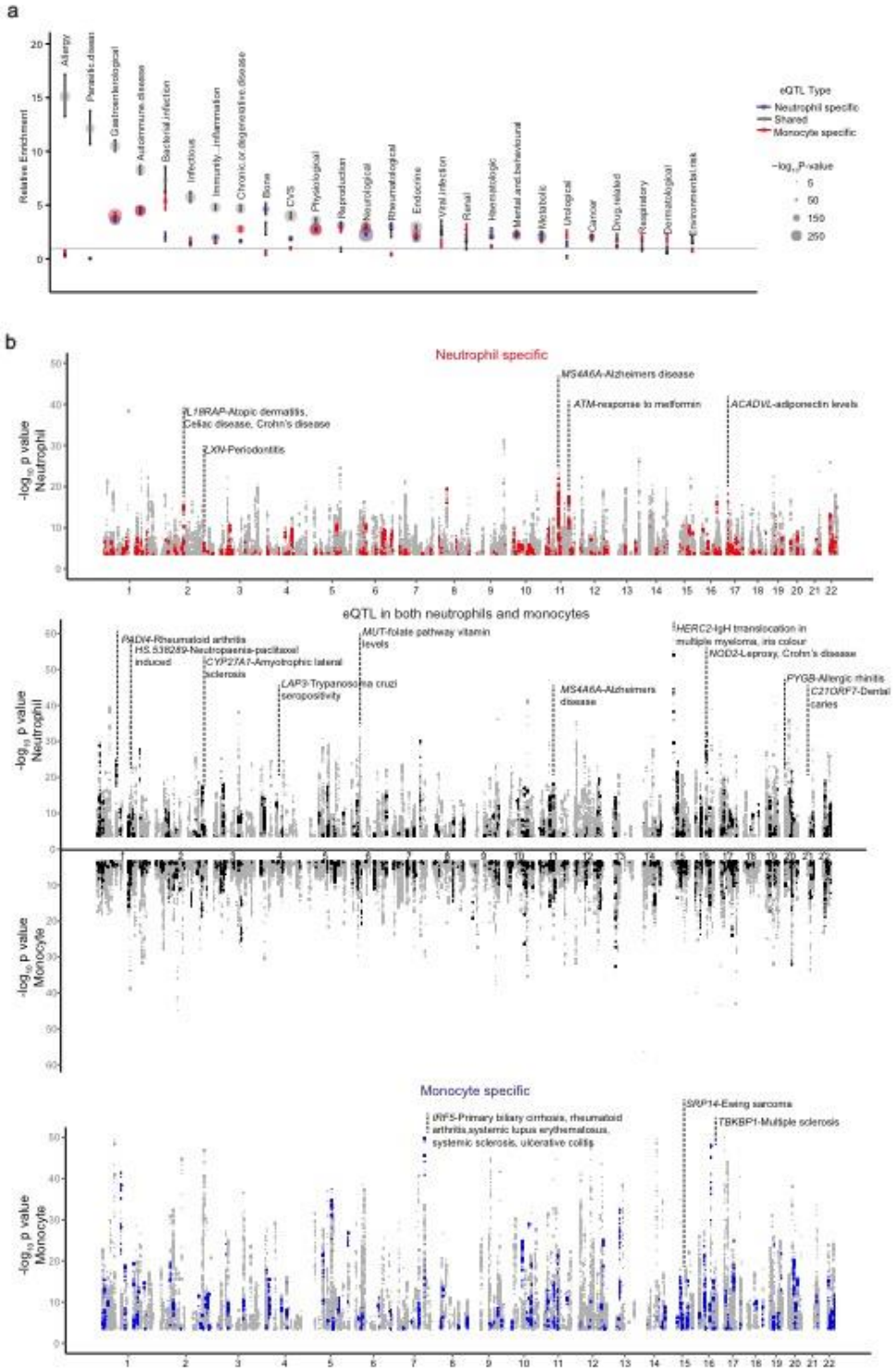
molecular symbol). Network a ($p=1x10^{-36}$, Fishers exact test) involves genes associated with NFkB such as *MAP1LC3A* encoding LC3 involed in autophagy ($p=7.1x10^{-10}$), *TRIM8* ($p= 6.7x10^{-6}$) which modulates p53 activity and *CALM1* (calmodulin 1) ($p=3.2x10^{-6}$); network b ($p=1x10^{-30}$) involving *CXCL8* (IL8) ($p= 9.5x10^{-5}$) a critical modulator of neutrophil recruitment and behaviour with associated genes showing eQTL including *IRF3* ($p=3.6x10^{-6}$), *IL18RAP* ($p=5.5x10^{-17}$) and *CXCR1* (IL8 receptor) ($p=2.4x10^{-5}$); network c ($p=1x10^{-30}$) centred on histones H3 and H4; network d ($p=1x10^{-26}$) involving genes linked with ERK1/2 MAP kinases; network e ($p=1x10^{-26}$) also focused on ERK and P38 MAPK and involving a network of genes including many transcription factors such as *ETS1* ($p=9.7x10^{-6}$), *ATF1* ($p=5.1x10^{-8}$) and *JUN* ($p=2.7x10^{-5}$); network f ($p=1x10^{-26}$) involving Akt and caspases; network g ($p=1x10^{-26}$) including eQTL involving *ATM* ($p=1.5x10^{-18}$), heat shock proteins *HSPA1A* and *HSPA1B* ($p=1x10^{-6}$) and *MCM5* ($p=3.4x10^{-11}$); network h ($p=1x10^{-23}$), network i ($p=1x10^{-19}$) involving genes linked to the nuclear transcriptional regulator NURP1 (also known as p8) showing eQTL such as the phopsholipid gene *PCTP* ($p=9.5x10^{-13}$); and network j ($p=1x10^{-18}$) involving IL12, PI3K and TCR which includes genes showing *cis*-eQTL such as *STAT5A* ($p=1.9x10^{-5}$) important in GM-CSF signaling and control of granulocyte homeostasis[69] and IDO1 ($p=4.8x10^{-6}$) which has a role in neutrophil self-regulation through tryptophan catabolism[70].



Supplementary Figure 3. Euler diagrams showing overlap of genes with an eQTL previously observed in lymphoid (a) or myeloid (b) cell types and their overlap with genes with eQTL in neutrophils (c). We extracted lists of genes reported to have a significant eQTL from several previous studies and show the number of overlapping or non-overlapping genes for the four studies reporting eQTL in lymphoid cells[14,15,17] (a) or myeloid cells[13-16] (b). Overlapping the lists of genes from A and B, with the list of 3281 genes with an eQTL in neutrophils yields panel with number of overlapping genes noted (c).



Supplementary Figure 4: Effect sizes of eQTL. These tend to be larger for eQTL that are observed in both cell types (labelled as both) or those seen in one cell type only and are larger in neutrophils than monocytes regardless of whether the eQTL is observed in both cell types or one only. Box lower and upper border denote 25th and 75th centiles respectively, central line denotes median and whiskers extend to 1.5*IQR



Supplementary Figure 5: Shared and cell type specific *cis* eQTL are enriched for variants associated with diseases or traits in GWAS studies. (a) Enrichment of shared and cell type specific eQTL for variants reported in the GWAS catalog as associated with a trait or disease by GWAS ontology category. For each category we compared the proportion of eQTL

variants associated with a trait in the relevant category to the proportion of all variants tested that had this property by a Fisher's exact test. Tails show 95% CI of the enrichment estimate (b) Manhattan plots demonstrating significant (all FDR<0.05) shared (black), neutrophil-specific (red) or monocyte-specific (blue) eQTL that are either associated with a trait in the GWAS catalog (color) or not (grey). An arbitrary selection of diseases for which the risk variant (or a variant in linkage disequilibrium r^2>0.8) is also an eQTL are highlighted.