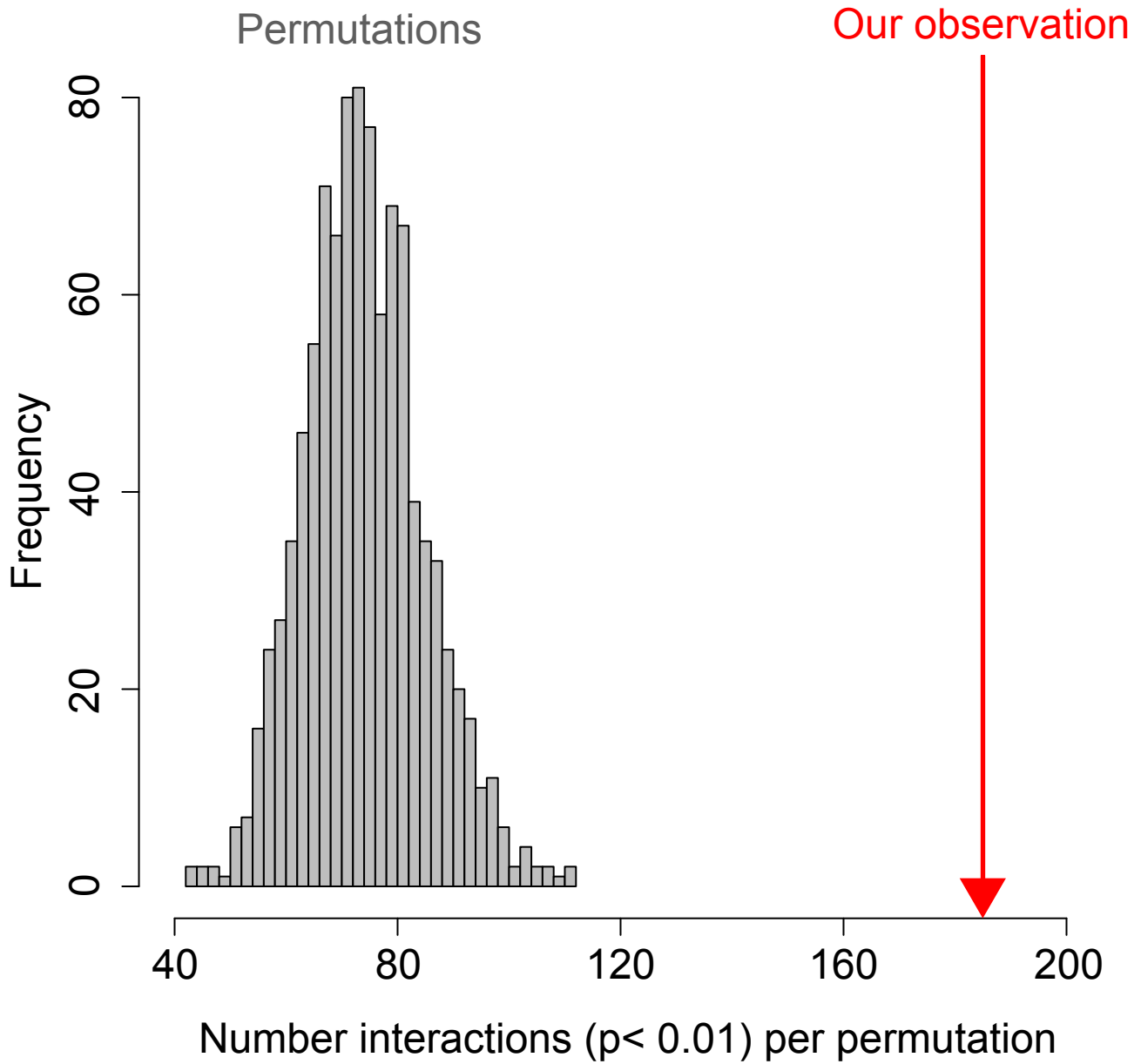
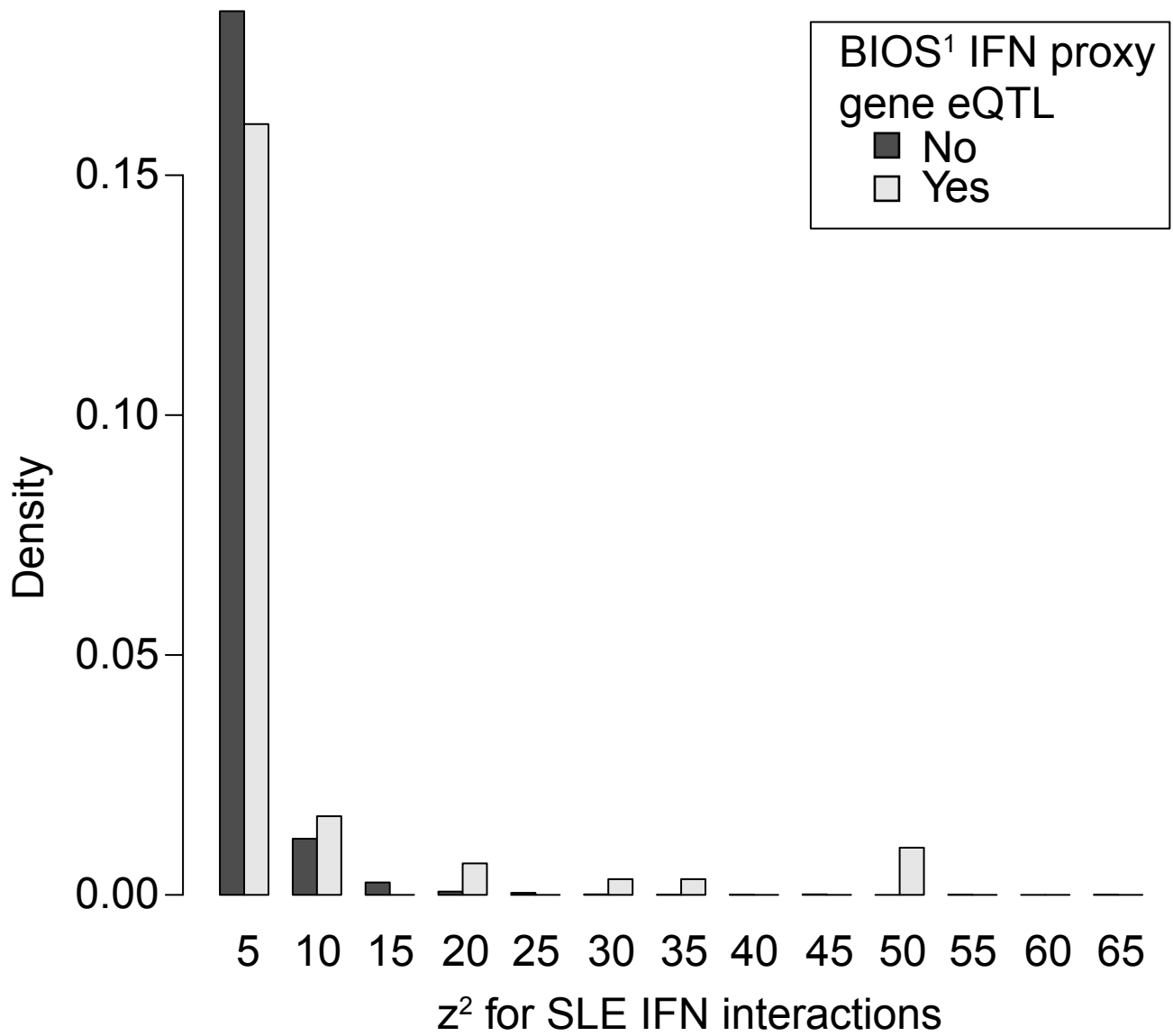


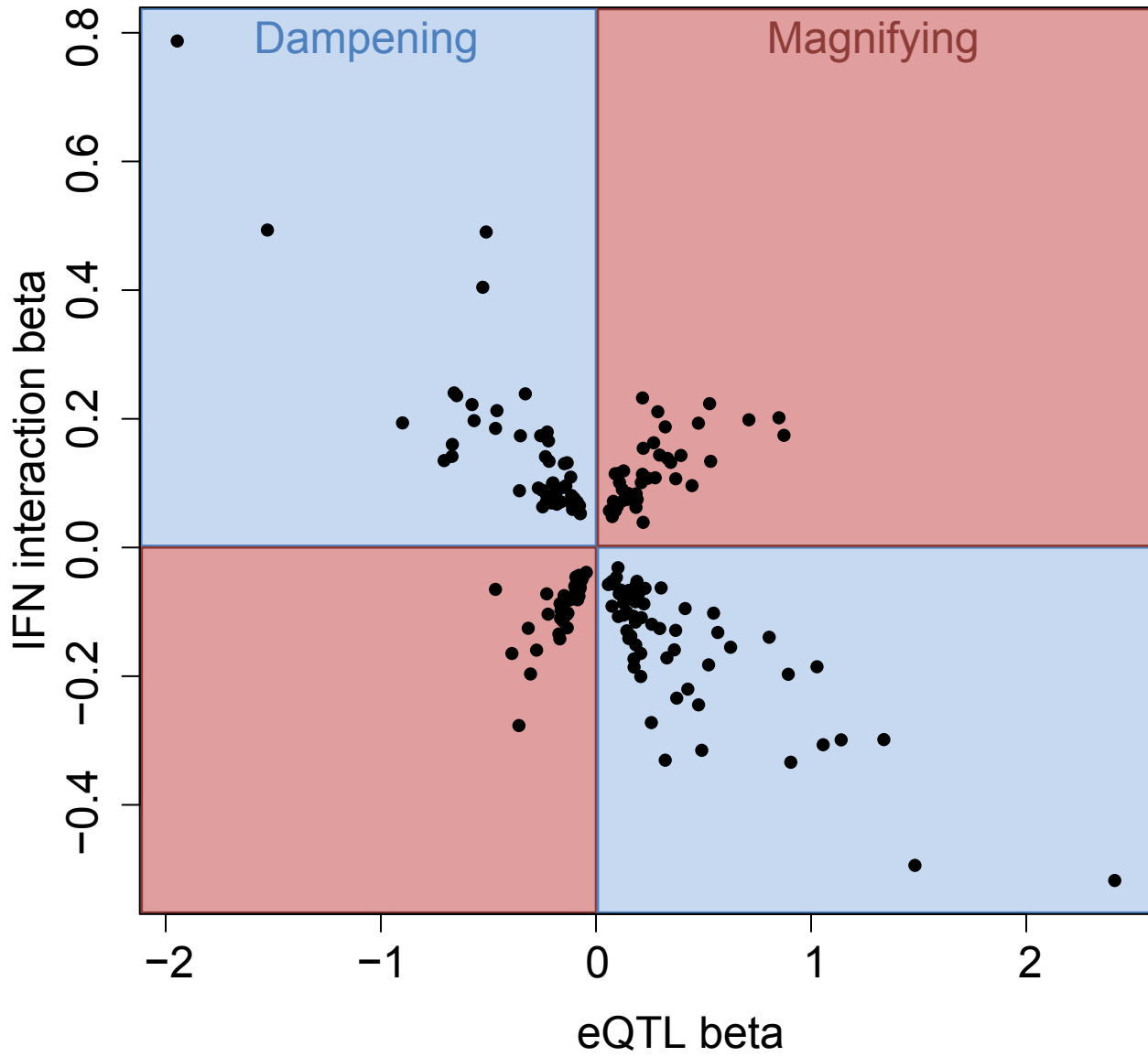
Supplementary Figure 1. eQTL interactions can be divided into magnifiers, where environmental exposure and the SNP eQTL effects are in the same direction, and dampeners where environmental exposure and the SNP eQTL effects work in the opposite direction.



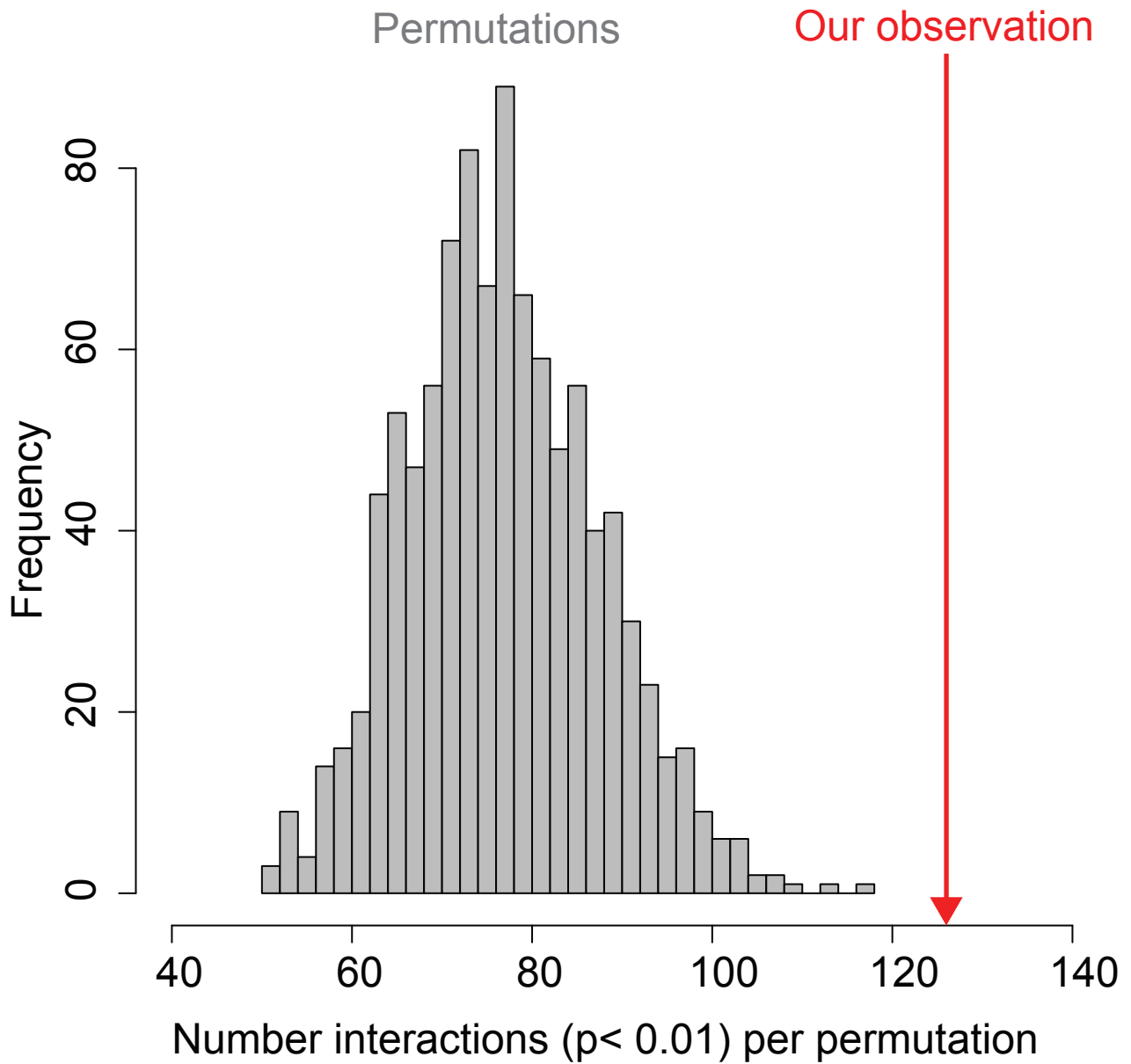
Supplementary Figure 2. Number of significant interactions ($p < 0.01$) from 1,000 permutations of IFN status. The median number of interactions observed from the permutations was 74.



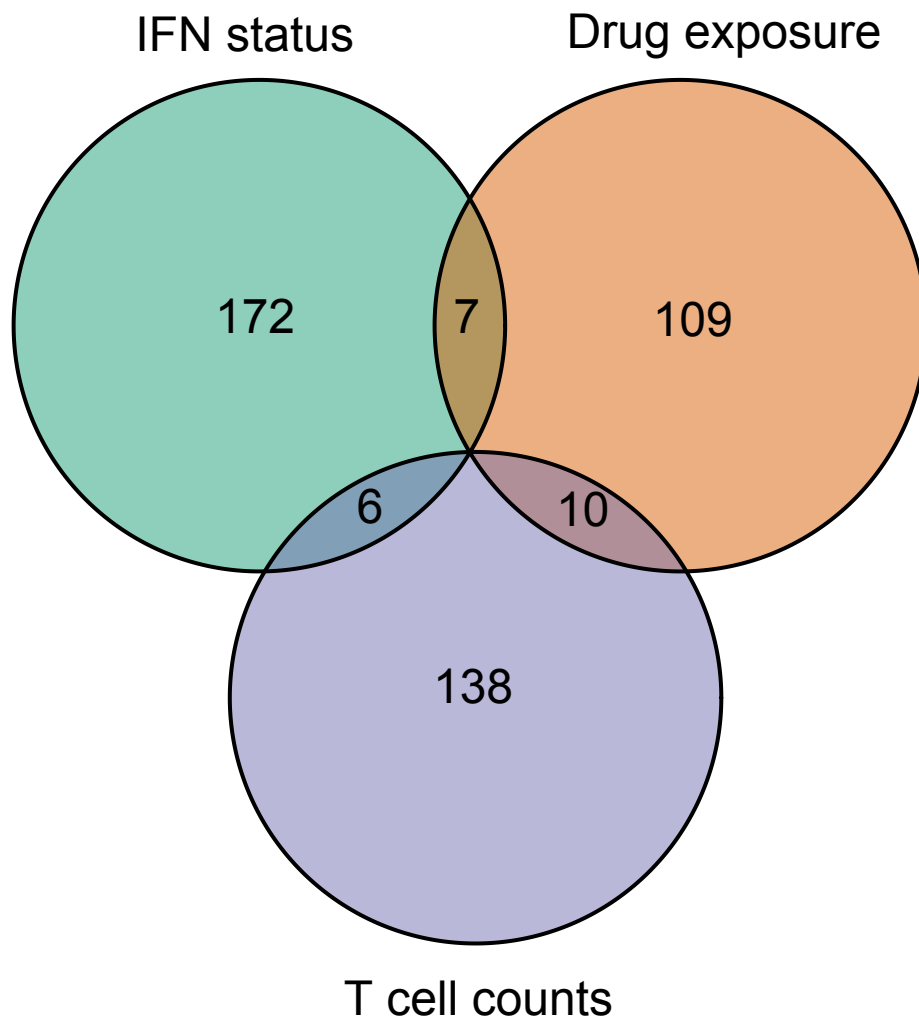
Supplementary Figure 3. Overlap of SLE IFN interaction genes with previously reported IFN proxy interactions. The z^2 for each gene tested for an IFN interaction in the SLE cohort is plotted.



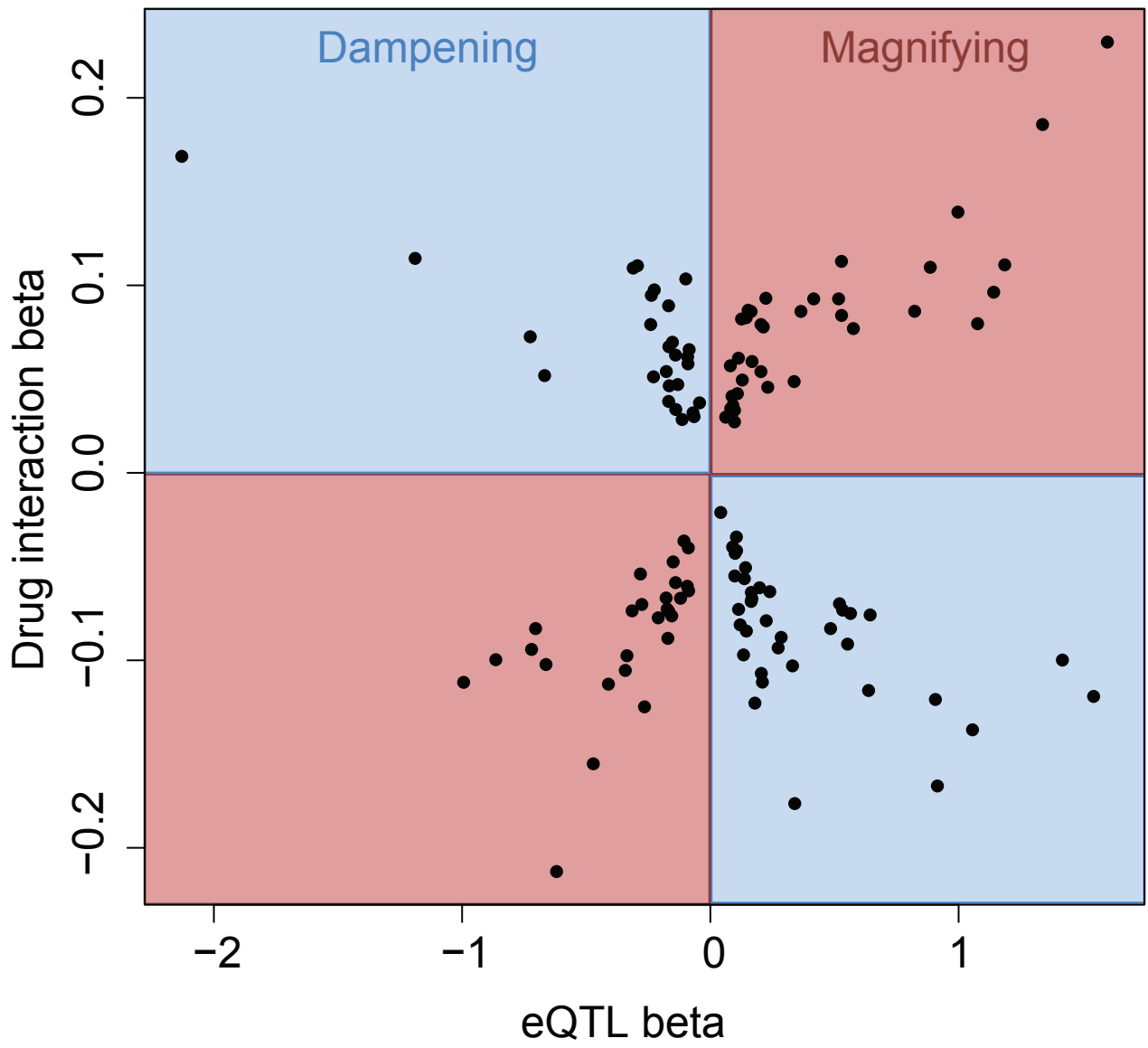
Supplementary Figure 4. Magnifying or dampening effect of the IFN interaction on the original eQTL effect.



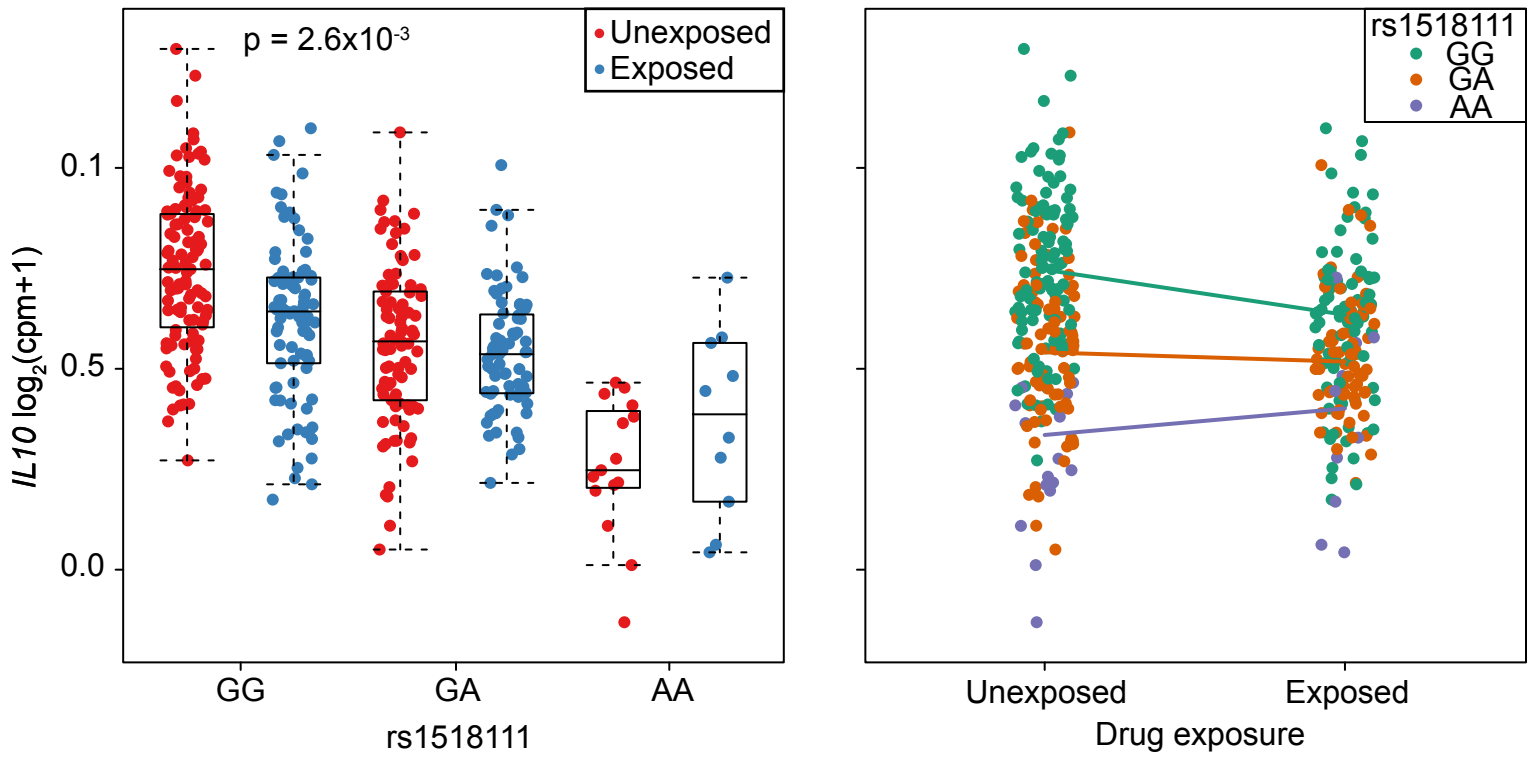
Supplementary Figure 5. Number of significant interactions ($p < 0.01$) from 1,000 permutations of drug exposure. The median number of interactions observed from the permutations was 77.



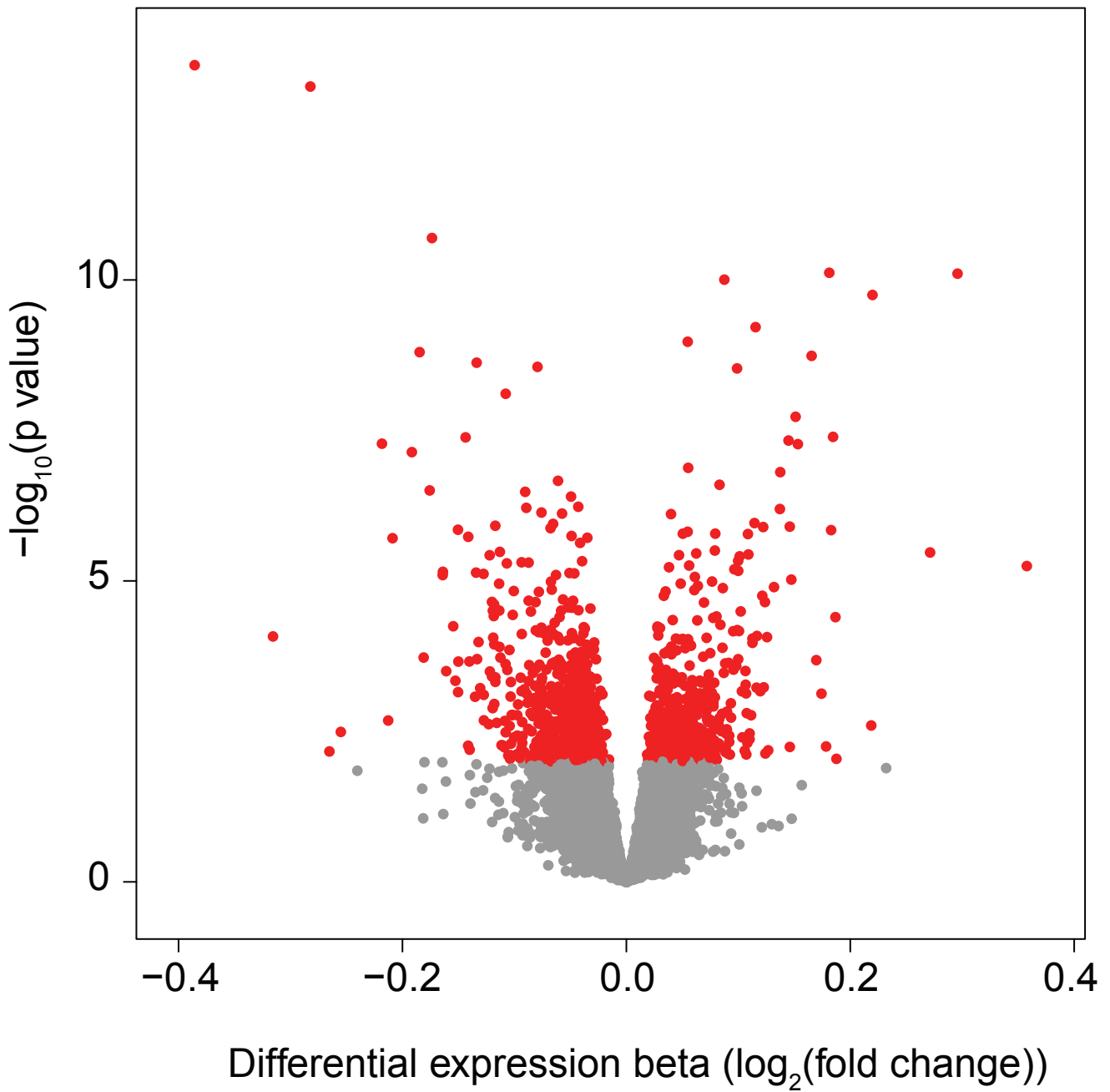
Supplementary Figure 6. Overlap of eQTL interactions with T cell counts, IFN status and drug exposure.



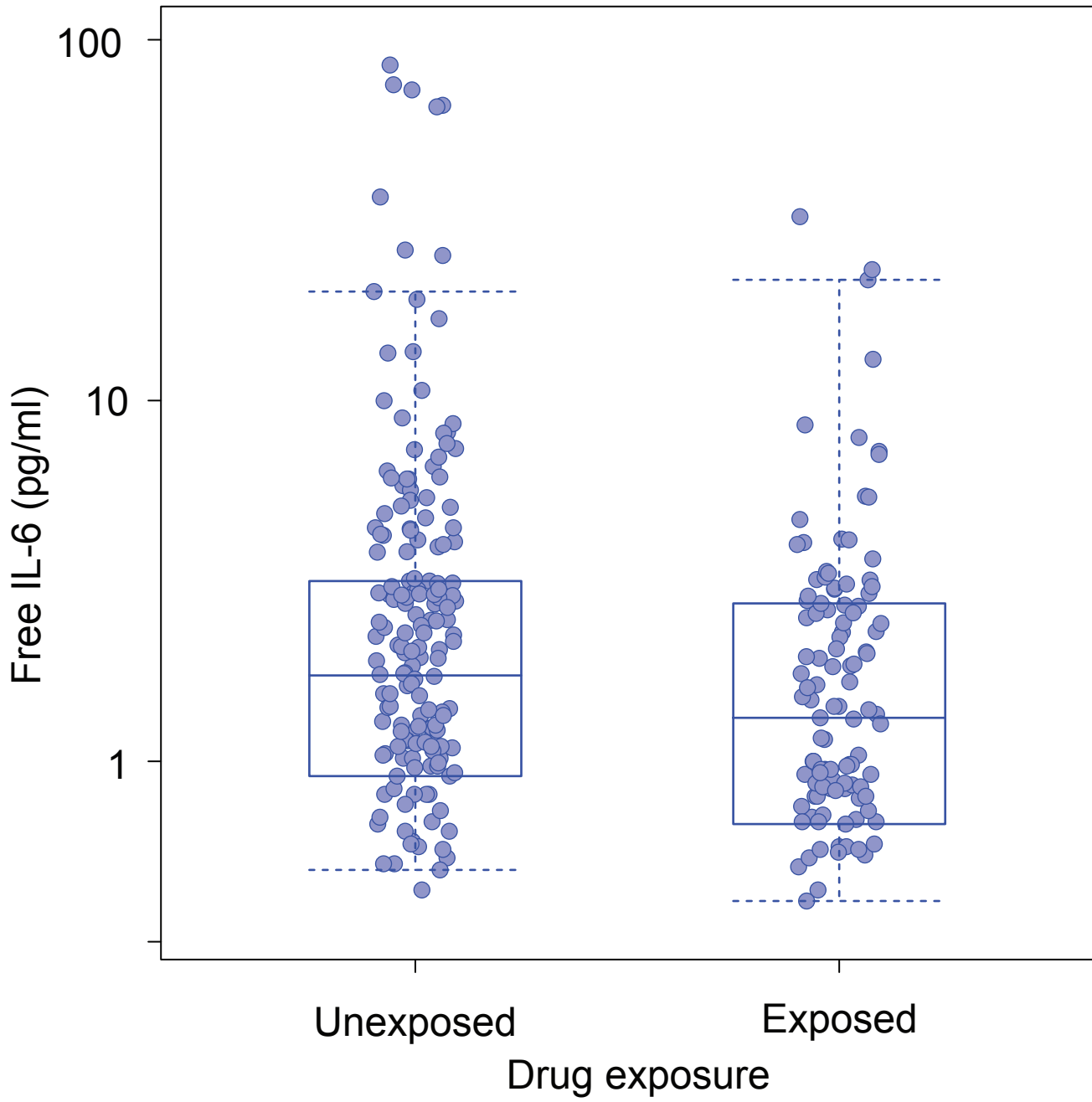
Supplementary Figure 7. Magnifying or dampening effect of the drug exposure interaction on the original eQTL effect.



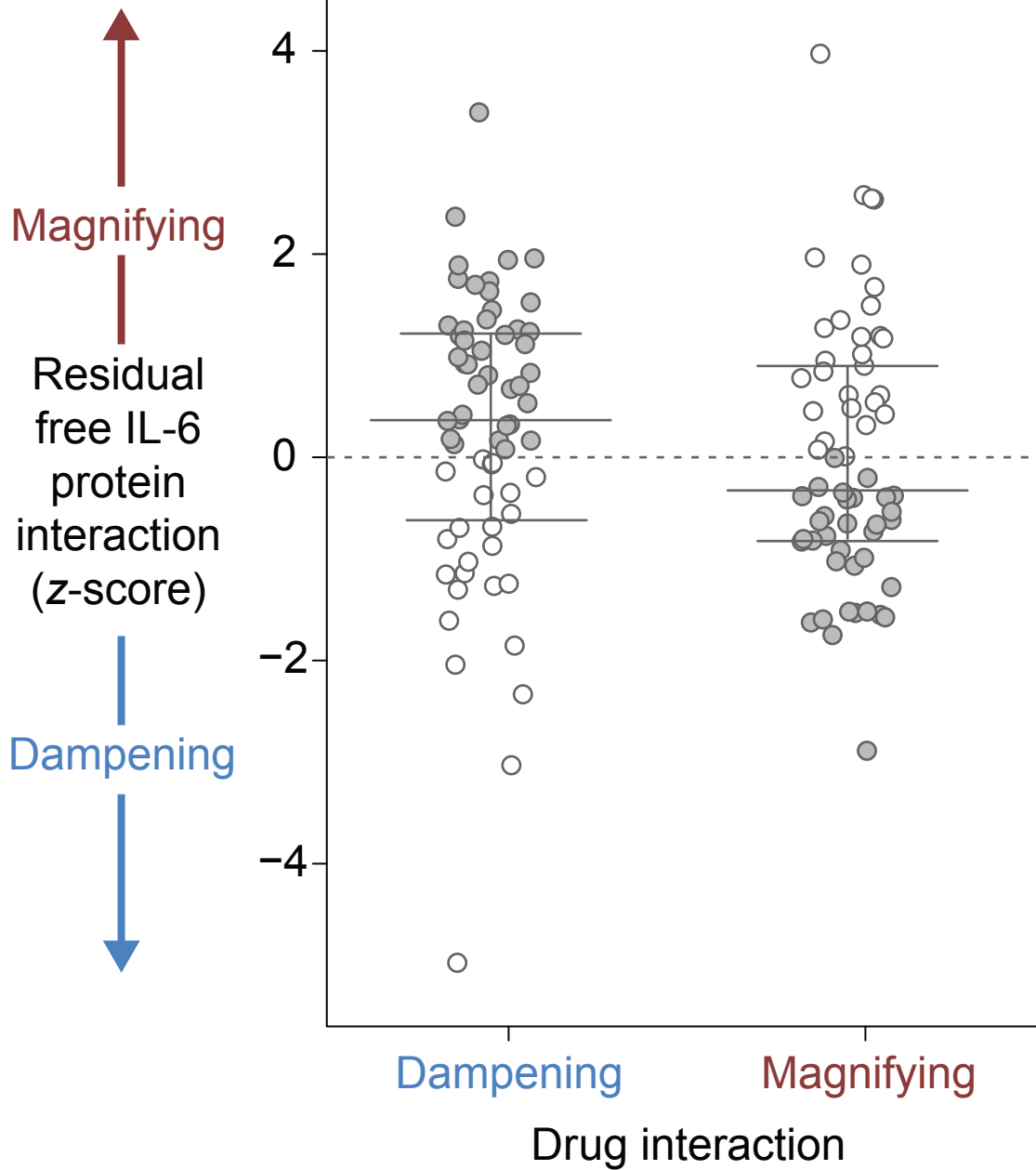
Supplementary Figure 8. Drug exposure interaction with the *IL10* eQTL plotted with respect to *rs1518111* genotype (left) and drug exposure (right).



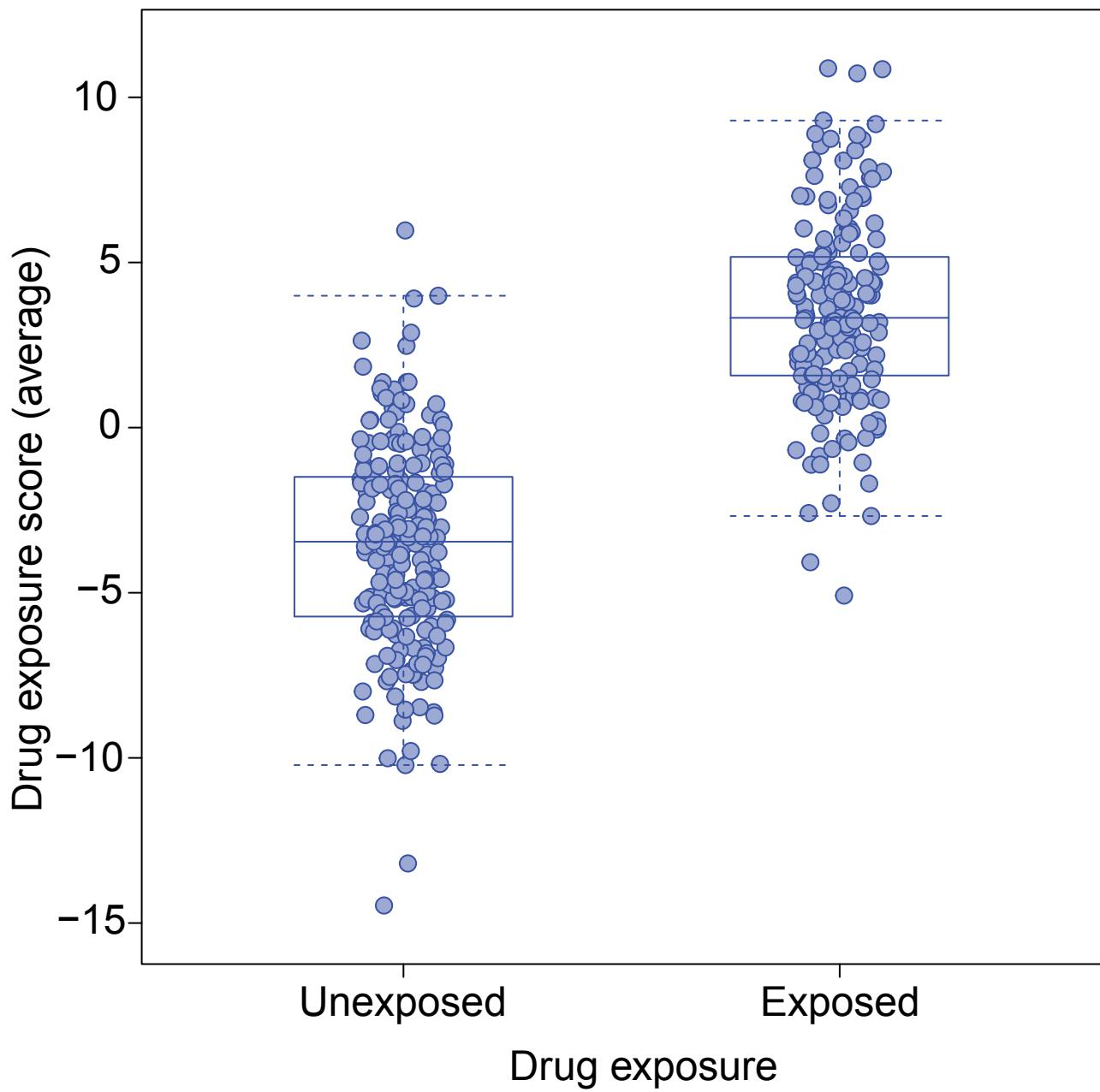
Supplementary Figure 9. Differential expression following drug exposure. Red points represent genes with $p < 0.01$.



Supplementary Figure 10. Free IL-6 protein levels in the unexposed and drug exposed samples.



Supplementary Figure 11. Concordance of residual free IL-6 protein interaction effects (after modeling drug exposure) with drug exposure interaction effects (grey indicates consistent direction).



Supplementary Figure 12. Drug exposure score calculated from 126 drug-eQTL interactions.