inference on a posterior sample with 9,000 network structures. Inspection of the trace plots suggest good mixing of the Markov chain (Supplementary Figure S2).

We performed Bayesian model averaging and, for each of 91 possible pairs (Y_u, Y_v), we obtained the posterior probabilities of $Y_u \to Y_v$, $Y_u \leftarrow Y_v$ and of no direct causal connection. The results are shown in Supplementary Table S7. Figure 4 show a model-averaged network.

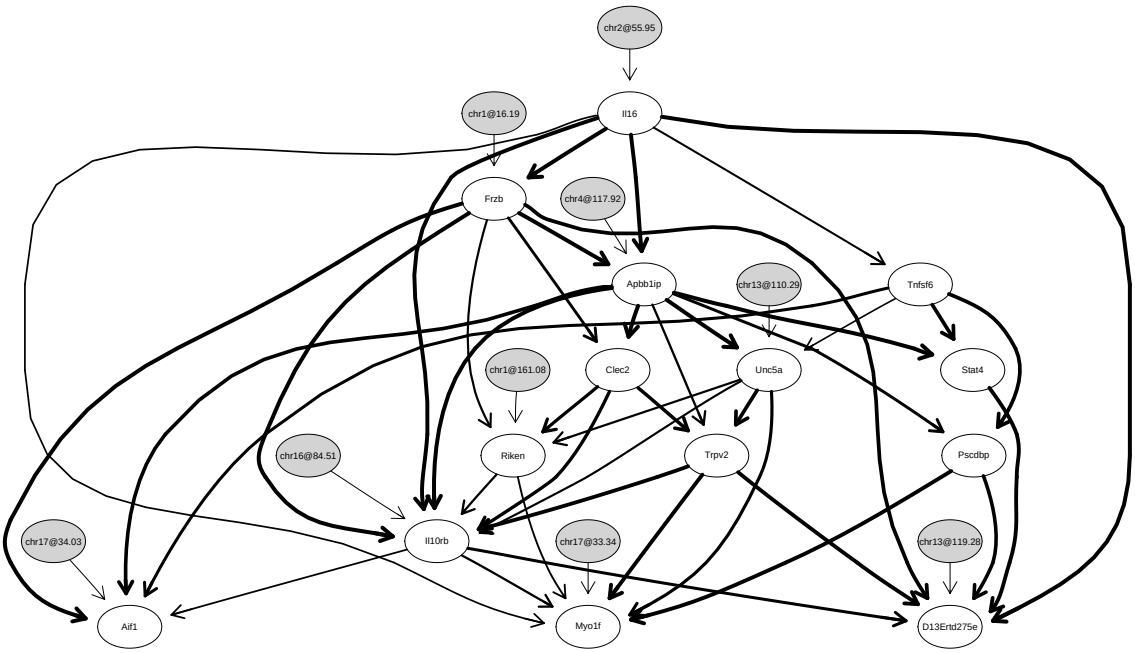

Figure 4: Model-averaged posterior network. Arrow thickness is proportional to the posterior probability of the causal relation computed via Bayesian model averaging. For each pair of phenotypes, the figure displays the causal relationship (presence or absence of an arrow) with highest posterior probability. Light grey nodes represent QTLs and show their chromosome number and position in centimorgans. Riken represents the riken gene $6530401C20Rik$.

This network suggests a key role of $Il16$ in the regulation of the other transcripts in the network. $Il16$ is upstream to all other transcripts, and is the only one directly mapping to the locus of chromosome 2. We would have expected the *cis* transcript, $Pscdbp$, to be the upstream phenotype in this network. However, the data suggests $Pscdbp$ is causal to only two other transcripts and that some other genetic factor on chromosome 2 may be driving this pathway. This estimated QTLnet causal network provides new hypotheses that could be tested in future mouse experiments.