

Figure S1. Soft thresholding power. Soft thresholding results for parental and reciprocal species intercross (A) and backcross (B) RNAseq datasets. These differ for each dataset, with the line indicating the threshold used in WGCNA cluster generation.

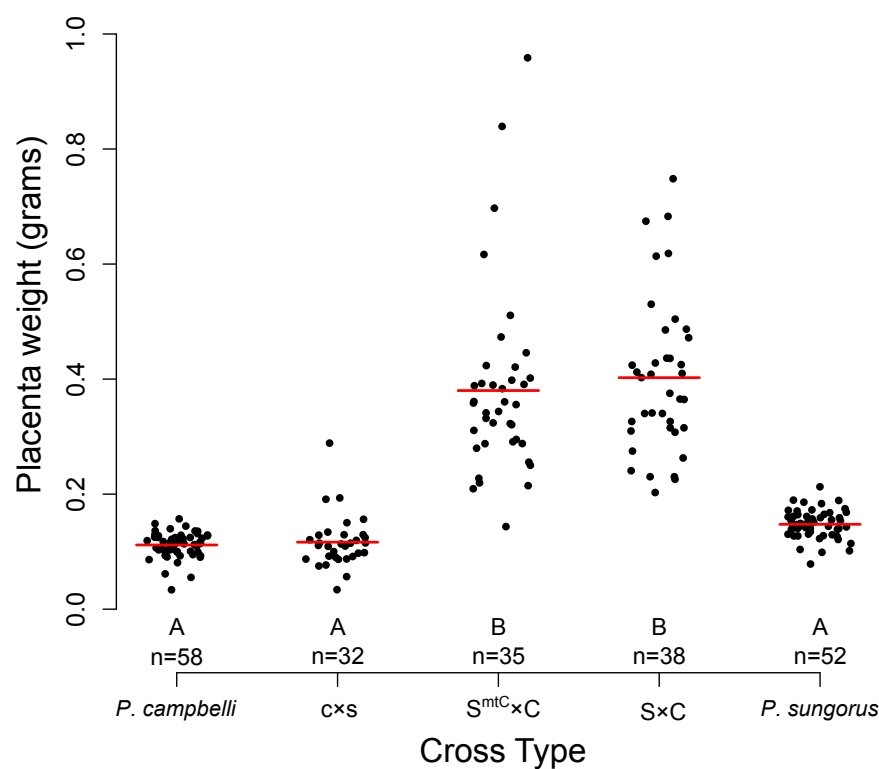


Figure S2. Mitochondrial interactions had no effect on placenta size. Placentas from the *S^{mtC}×C* cross were indistinguishable from *S×C* hybrids ($F_{4,213} = 106$, $P < 0.001$, full ANOVA model, Tukey's HSD test significance groups indicated by letter). Data for *P. campbelli*, *cxs*, *S×C*, and *P. sungorus* from (Brekke and Good 2014).

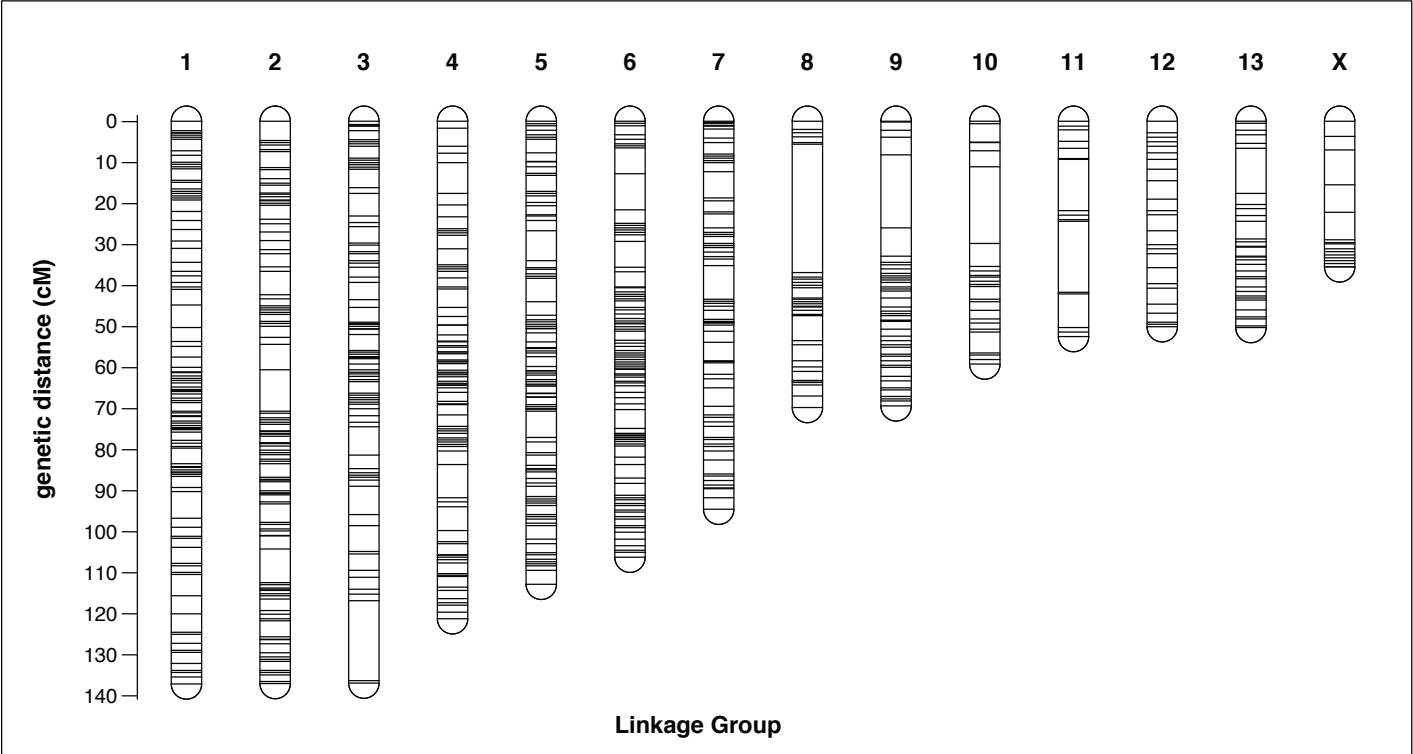


Figure S3. Genetic map of Phodopus dwarf hamsters. Map based on 1,215 RAD markers spanning 1,213.7 cM across 14 linkage groups numbered by decreasing length. The sequence of each marker and their exact locations in centiMorgans can be found in Supplemental Table 1. Specific locations of genes can be found in Table S2. Visualized with R/LinkageMapView (Ouellette et al. 2018).

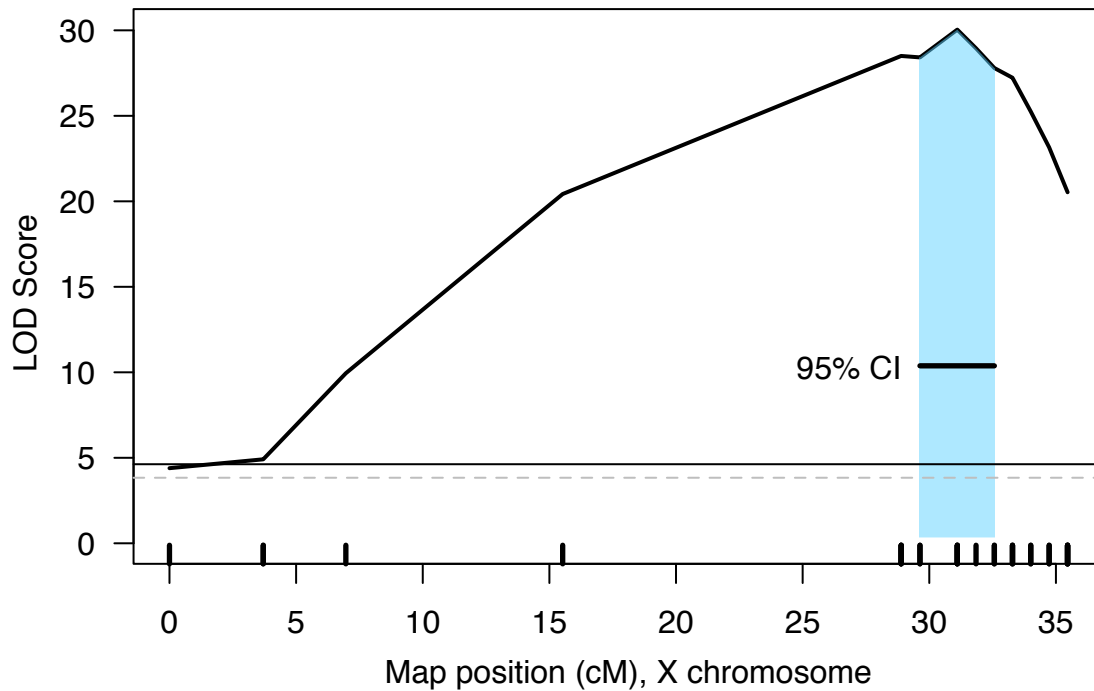


Figure S4. QTL interval on the X chromosome overlaps with increased marker density. Placental weight QTL likely corresponds to region of reduced recombination on the map. Solid line indicates permutation-based $P = 0.01$ significance threshold, dashed line indicates permutation-based $P = 0.05$ significance threshold.

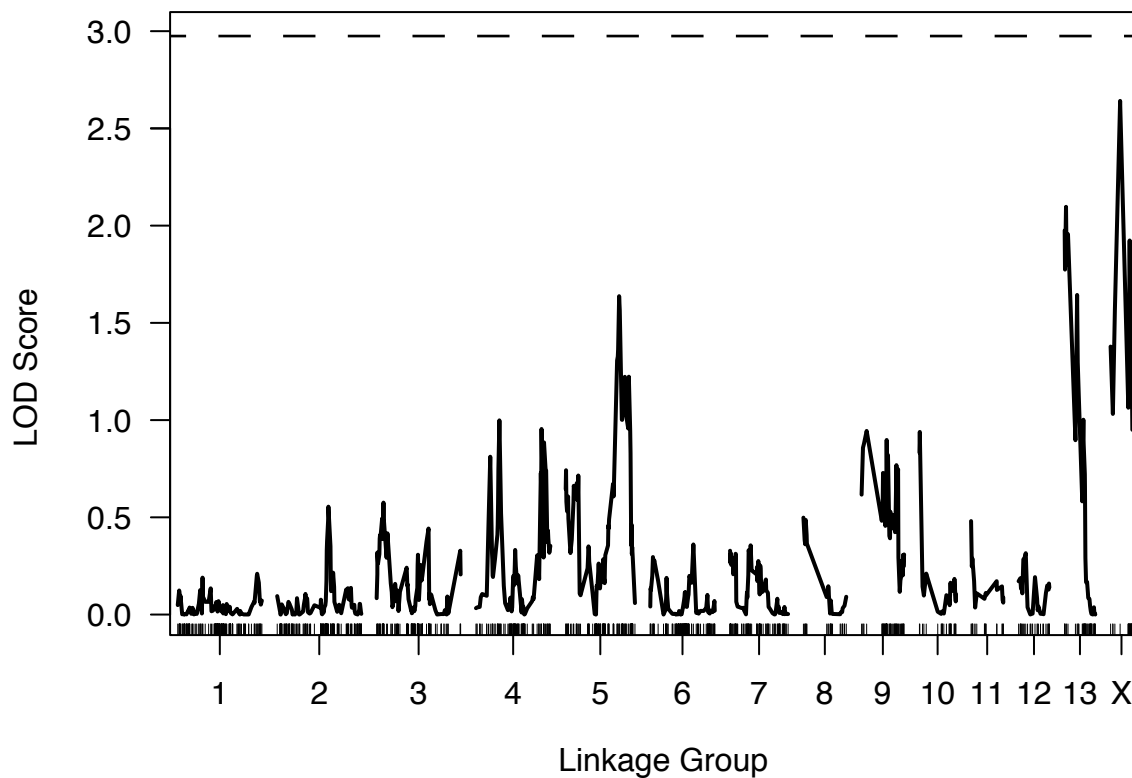


Figure S5. No QTL for embryo weight were detected in the BC mapping experiment. No peak passes the permutation threshold when controlling for Theiler stage and edema. $P = 0.05$ permutation threshold indicated with dashed line.

Supplemental Information

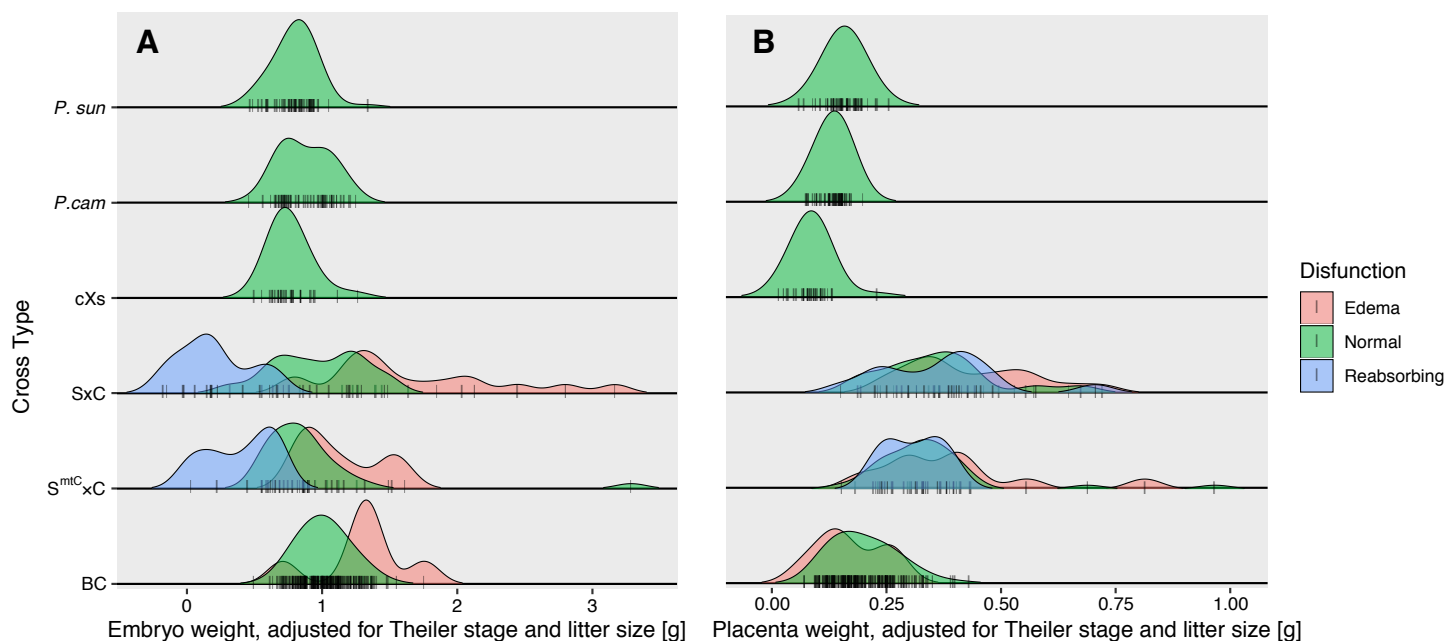


Figure S6. Edema and reabsorption explained much of the variance in embryo size, but not placenta size, in interspecies hybrid hamsters. Reabsorption (blue) and edema (red) shifted embryo size away from the mean in *P. sungorus* x *P. campbelli* (SxC) F₁, S^{mtC}xC F₁, and BC hybrid hamsters (A), but had little effect on placenta size (B).

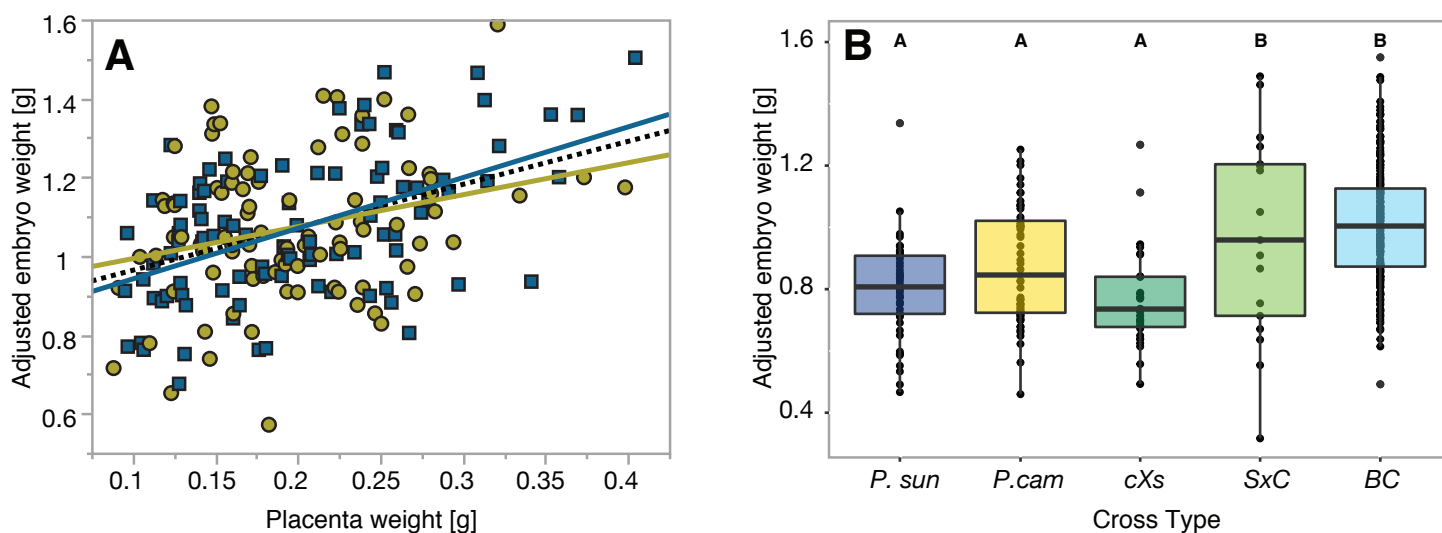


Figure S7. Embryo weight in the BC. (A) Embryo weight was positively associated with placental weight in the BC, and more strongly so in males (blue, adjusted $r^2 = 0.257$, $F_{1,95} = 33.8$, $P < 0.0001$) than females (yellow, adjusted $r^2 = 0.065$, $F_{1,88} = 7.15$, $P = 0.0090$). (B) When BC embryo weights were analyzed along with F₁ hybrids, the overgrown SxC F₁ and BC hybrids showed a slight but significant increase in size controlling for stage and edema (adjusted $r^2 = 0.159$, $F_{1,184} = 36.0$, $P < 0.0001$, ANOVA).

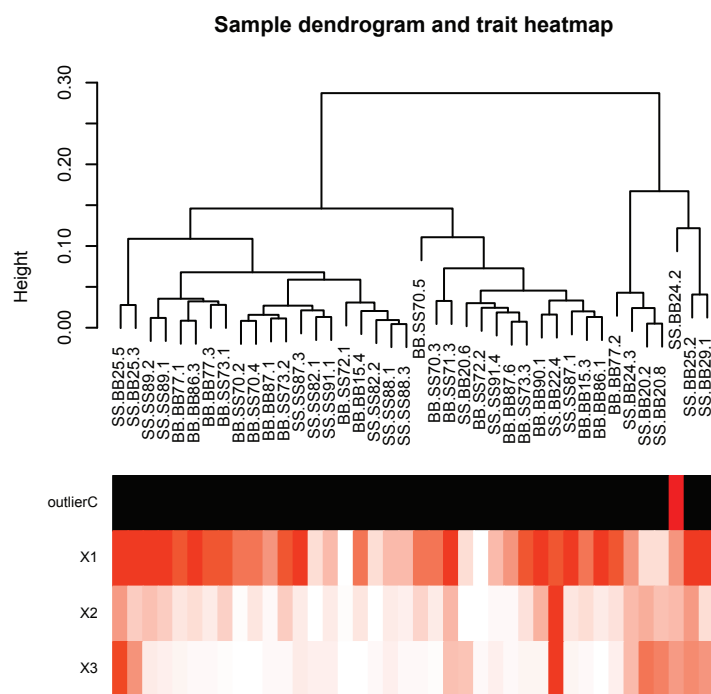
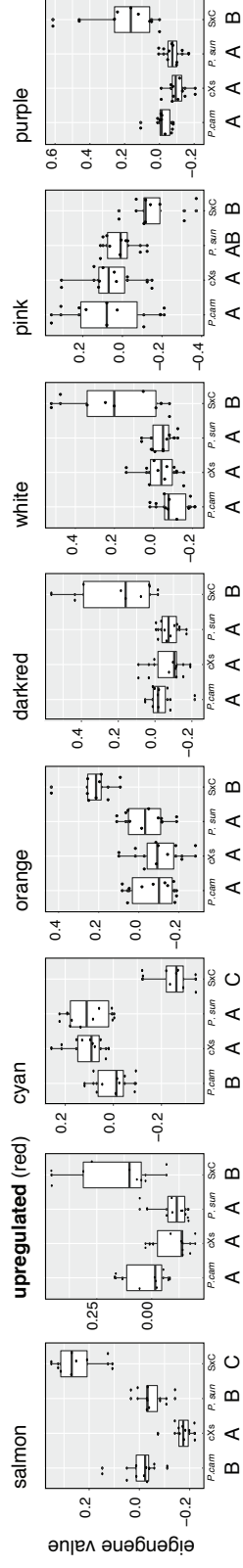
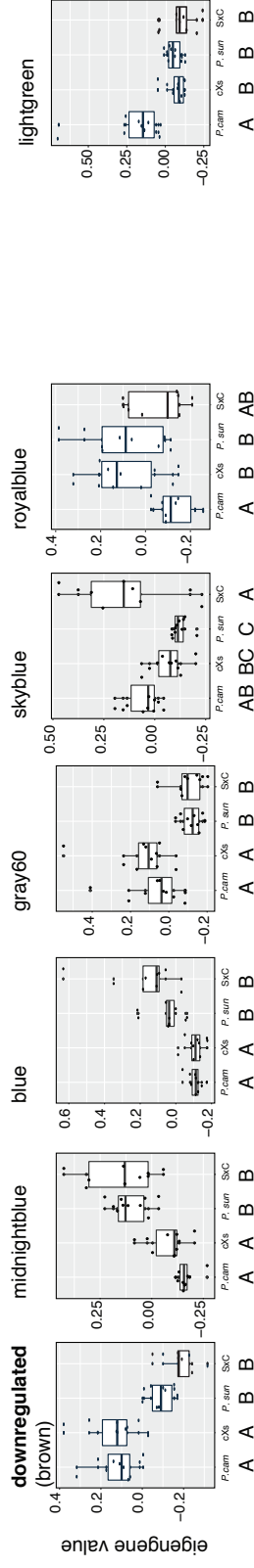


Figure S8. WGCNA output for F_1 data. Outlier identification of sample to be removed in red.

Transgressive, SxC is outlier



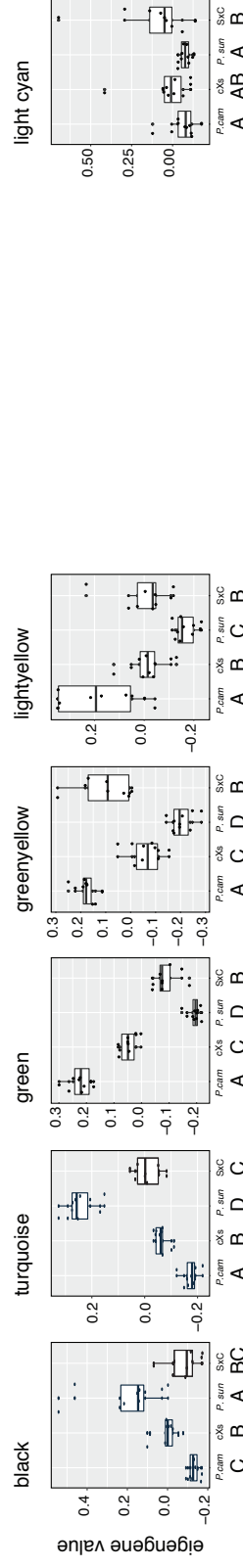
Maternal



Paternal

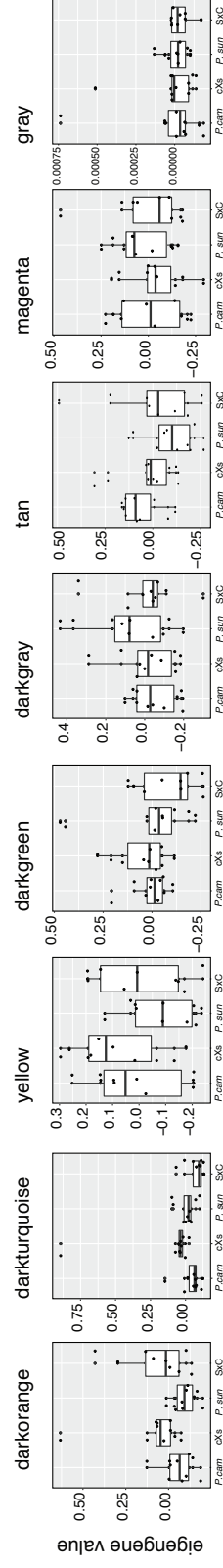
One Parent Dominant

Hybrids Intermediate



Both Hybrids Transgressive, same direction

No Relationship



cross type

Figure S9. Assignment of inheritance patterns of modules in network. Summary values of module expression (module eigengene) tested with an ANOVA to identify parent of origin and transgressive expression. Color names are arbitrarily and randomly generated by the program, and have no additional meaning. Letters indicate Tukey's HSD test assigned significance groups at $P < 0.05$.

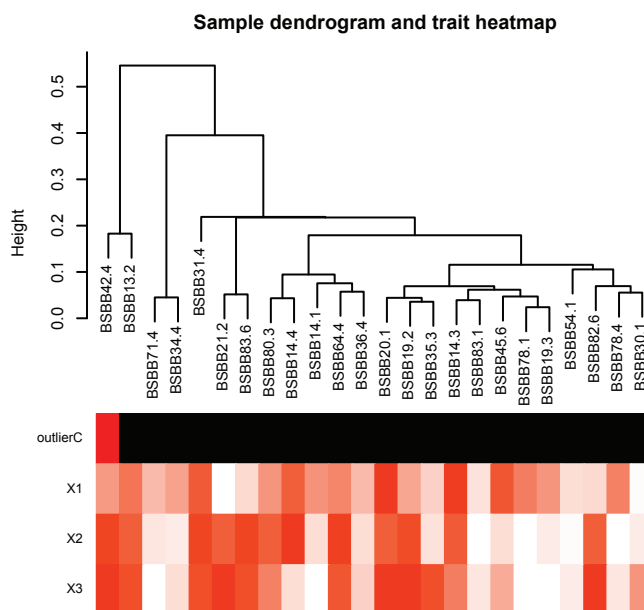


Figure S10. WGCNA output for BC data. Outlier identification of sample to be removed in red.

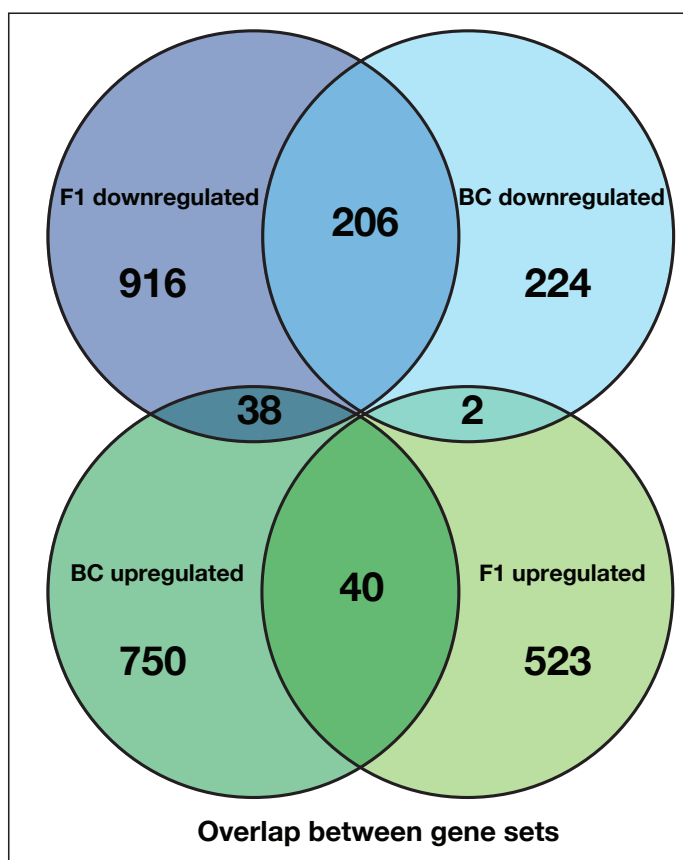


Figure S11. Count overlap between key F1 and BC modules. Venn diagram showing counts of genes shared between F₁ and BC downregulated and upregulated placenta modules. The downregulated modules shared the most genes (206).

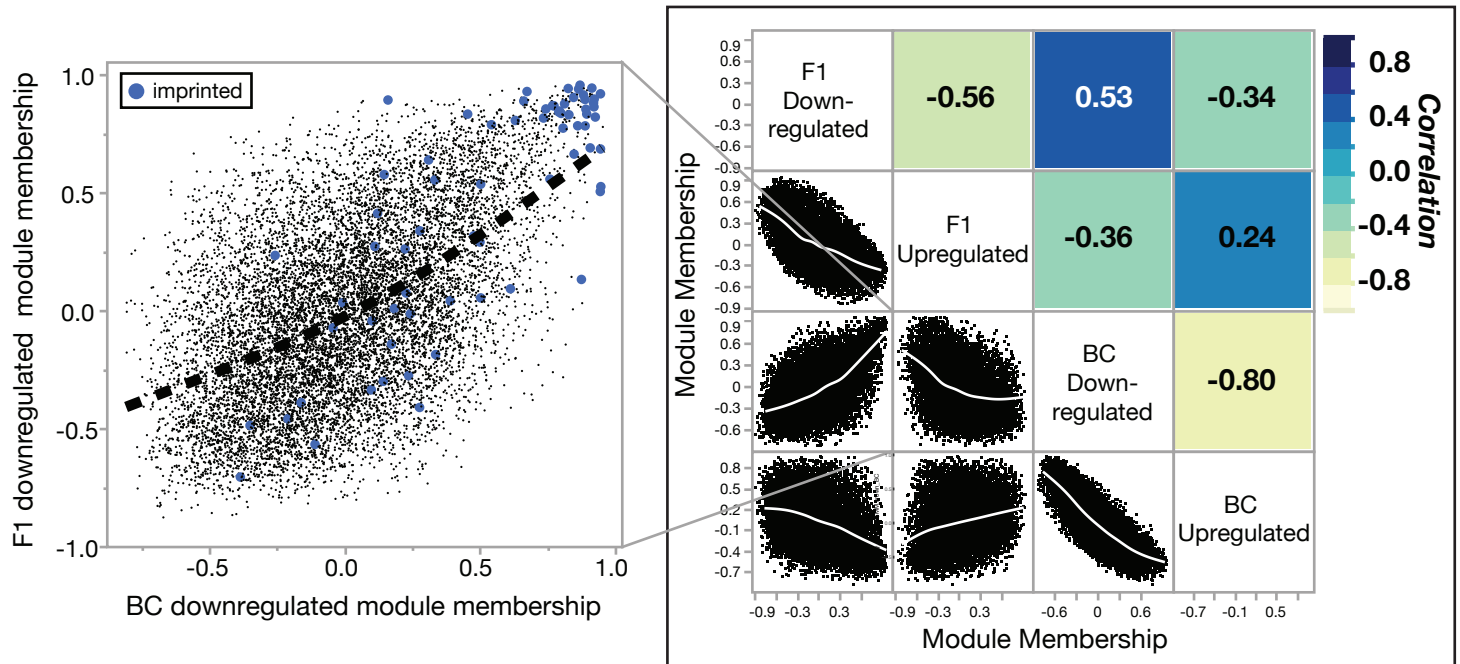


Figure S12. Correlation between key F1 and BC modules. Each gene in the network has a correlation to each module eigengene, whether that gene is placed in the module or not. We can assess how similar two modules are by asking whether genes are generally showing the same bivariate correlation to each module. Not only were the downregulated and upregulated modules within each data set negatively correlated with each other (F_1 , Pearson's $R = -0.56$, $P < 0.0001$, BC, Pearson's $R = -0.80$, $P < 0.0001$), the F_1 and BC downregulated modules across experiments were positively correlated with each other (Pearson's $R = 0.53$, $P < 0.0001$). Notably, the same candidate imprinted genes shared high connectivity/module membership with the network in both data sets (blue dots, inset).

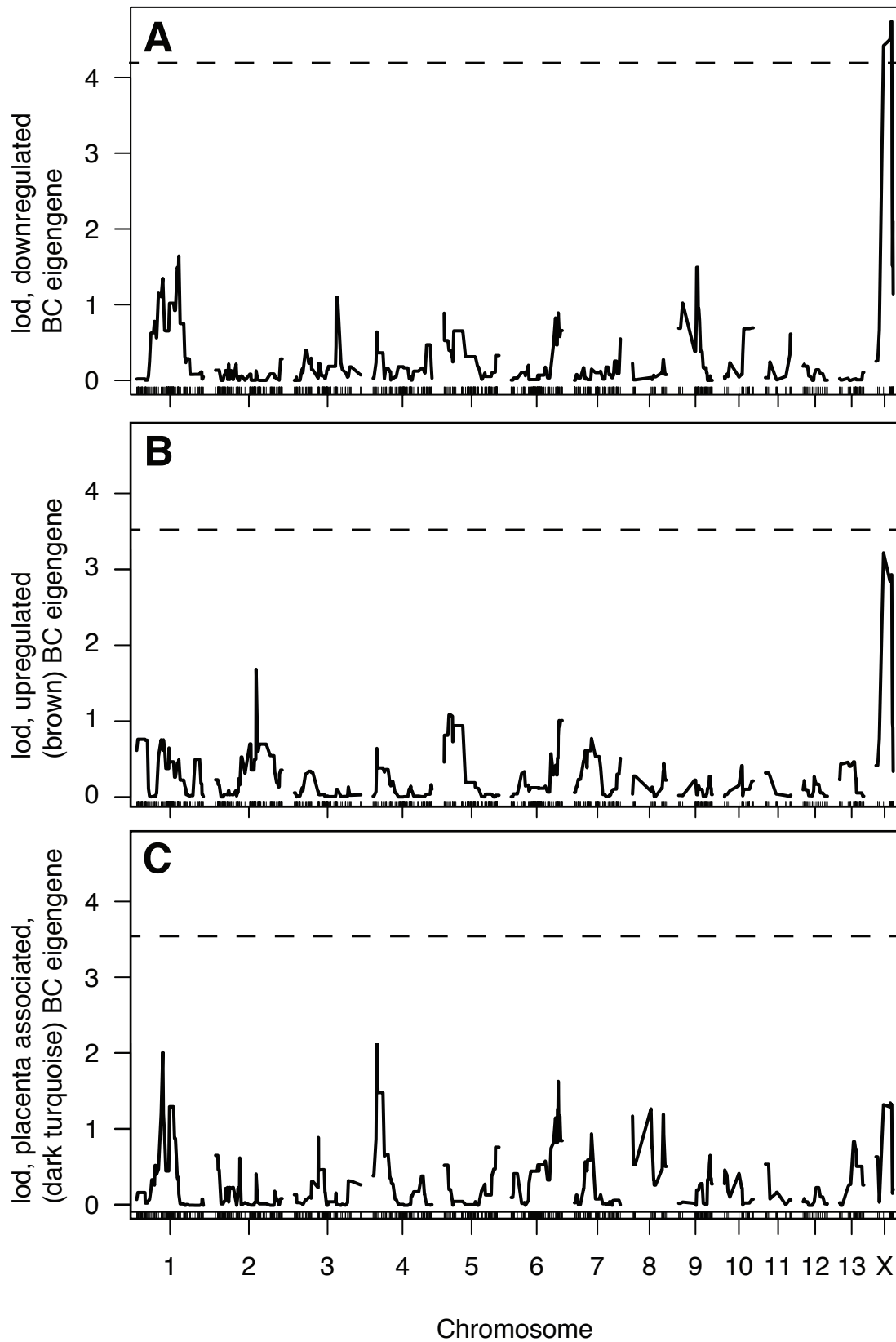


Figure S13. EigenQTL in BC hybrids. Using module eigengenes as phenotypes summarizing gene expression patterns in 23 BC hybrids, (A) we found that the downregulated module had a single X-linked QTL that passed a $P = 0.05$ permutation threshold (QTL peak at 31.1cM, LOD=4.739). (B,C) No QTL were detected for the other tested modules. Dashed line indicates permutation-based significance threshold ($P = 0.05$).

Supplemental Information

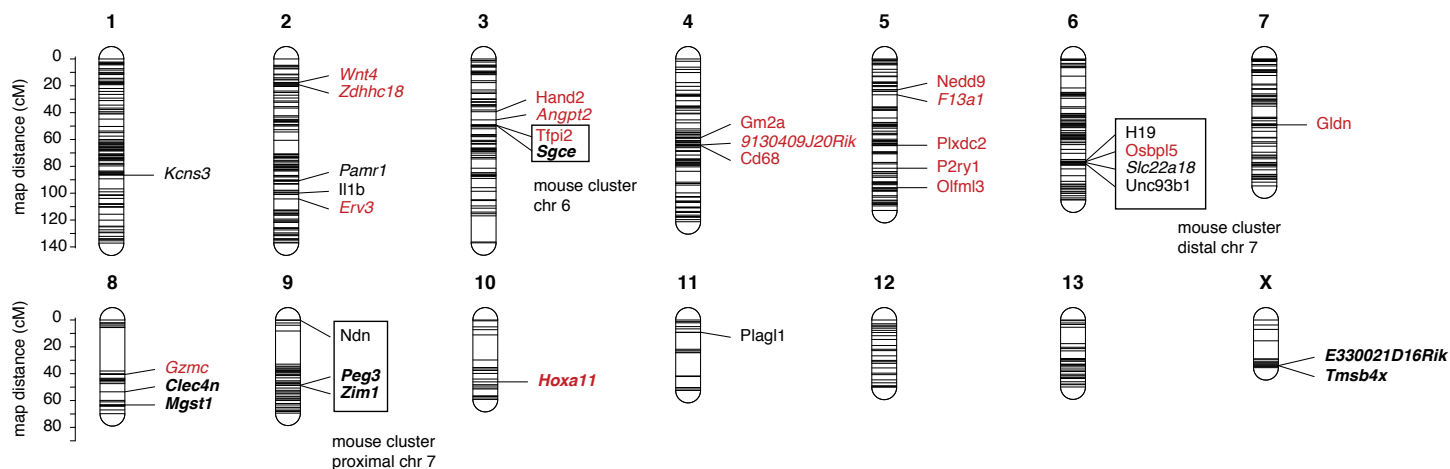


Figure S14. Candidate imprinted genes placed on the genetic map. Candidate imprinted genes in the BC downregulated module are indicated in red, and clusters with potential homology to imprinted clusters in *Mus* are indicated with boxes. Visualized with R/LinkageMapView (Ouellette et al. 2018).

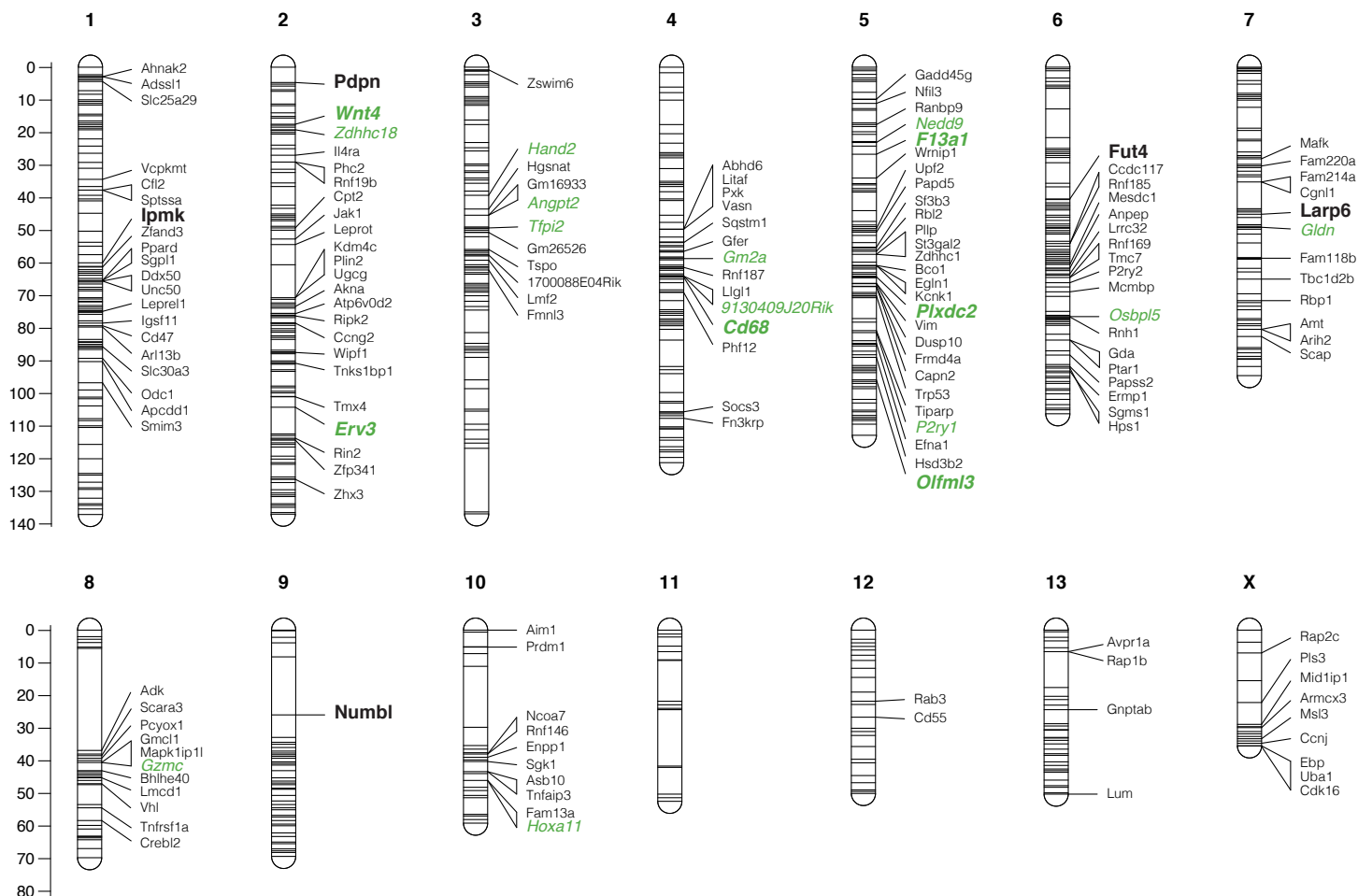


Figure S15. BC downregulated module placed on the genetic map. BC downregulated network hub genes are in bold, and candidate imprinted are indicated in green. Visualized with R/LinkageMapView (Ouellette et al. 2018)

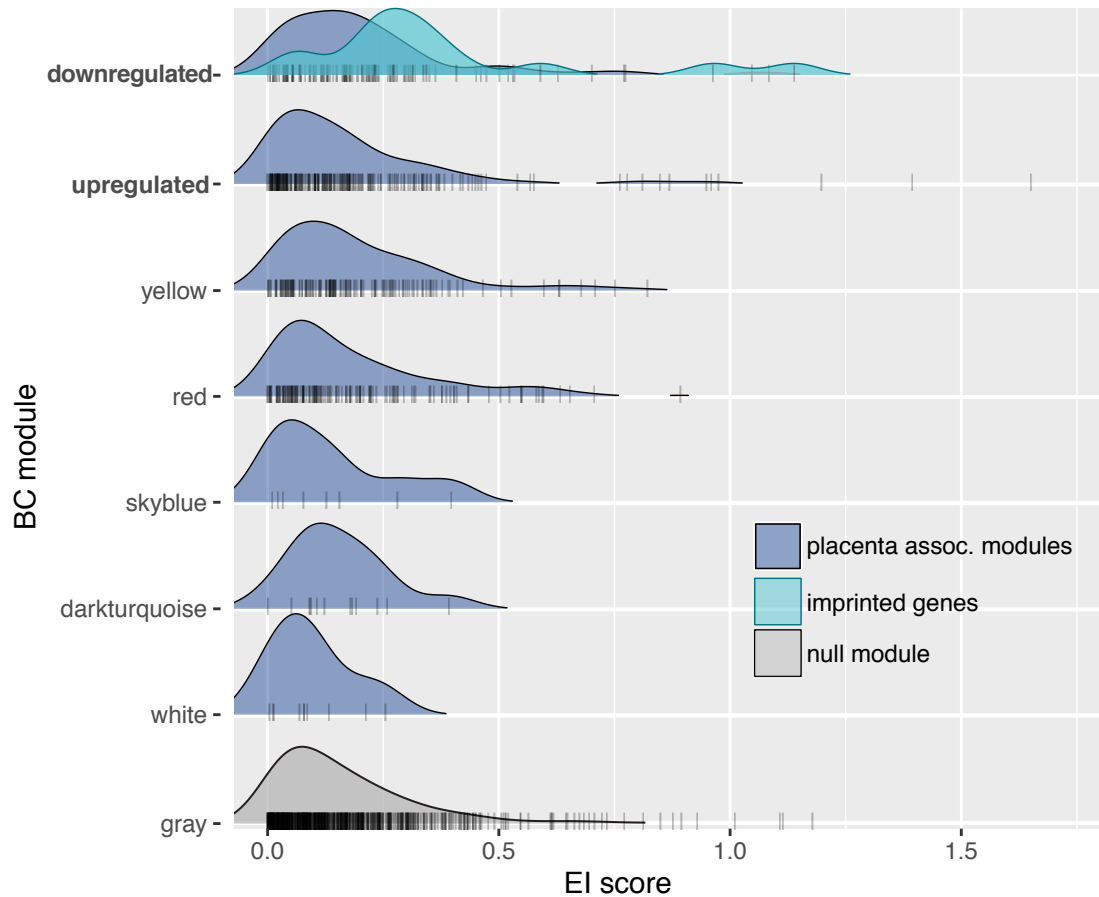


Figure S16. EI score distributions by module and imprinting status. The gray module includes all genes that were not placed in any module in the network analysis, and serves as the null expectation of the distribution of the score. All BC modules associated with placenta size are shown. Candidate imprinted genes in the downregulated module showed a shift towards increased EI values, while other placenta associated modules did not.

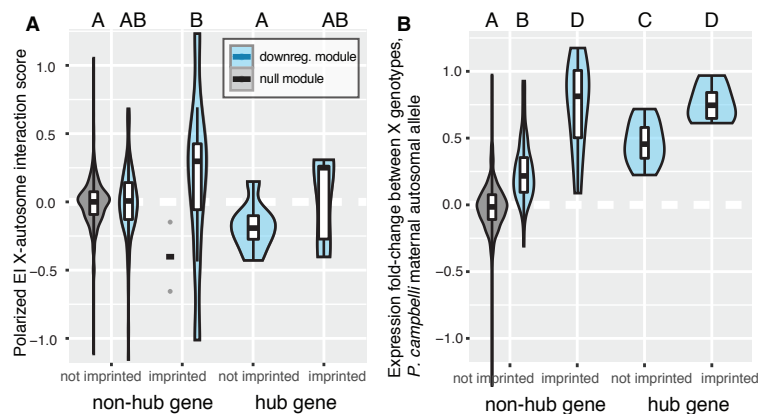


Figure S17. Polarized EI score distributions by hub and imprinting status. (A) Polarized expression interaction scores indicated that imprinted genes were more likely to have a larger fold change when maternal alleles were mismatched with the maternal X chromosome (a positive value). (B) Positive values for gene expression fold change between X genotypes for individuals with a (mismatched) homozygous *P. campbelli* autosomal genotype indicated that expression was higher for individuals with a *P. campbelli* X chromosome than for those with a *P. sungorus* X chromosome. All letter groups indicate significance based on a Tukey's HSD test, $P < 0.05$, and imprinting status. The grey module includes all genes that were not placed in any module in the network analysis, and serves as the null expectation of the distribution of the score. All BC modules associated with placenta size are included here, with shift of increased EI values for the candidate imprinted genes in the downregulated module, but not the other placenta associated modules.

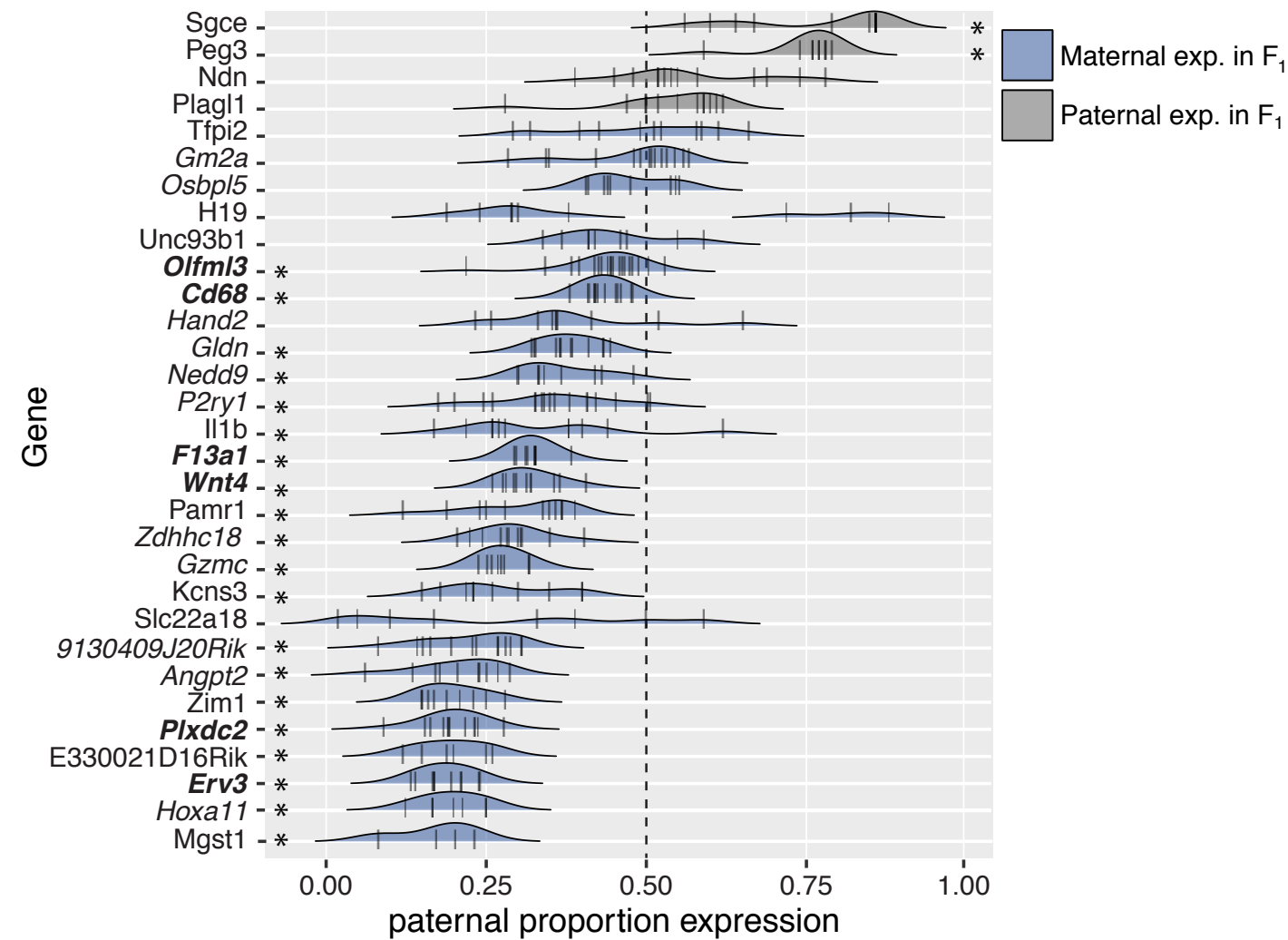


Figure S18. Allelic bias in candidate imprinted genes in BC. Proportion of paternal expression for all heterozygous BC hybrids for each of the 31 candidate imprinted genes that were placed on the map. To calculate allele-specific expression, the reference (*P. sungorus*) allele was designated as the maternal allele and the proportion of maternal reads was averaged over variable sites for heterozygous BC individuals, excluding all homozygous individuals at each gene. Generally, we recover qualitatively consistent signals for allelic bias at genes that displayed maternal (blue) or paternal bias (gray) in F_1 hybrids. Bolded genes are hub genes in the BC downregulated module. Stars indicate that mean paternal expression was significantly different from 0.5 (one-sample T-test, Bonferroni corrected $P < 0.05$).

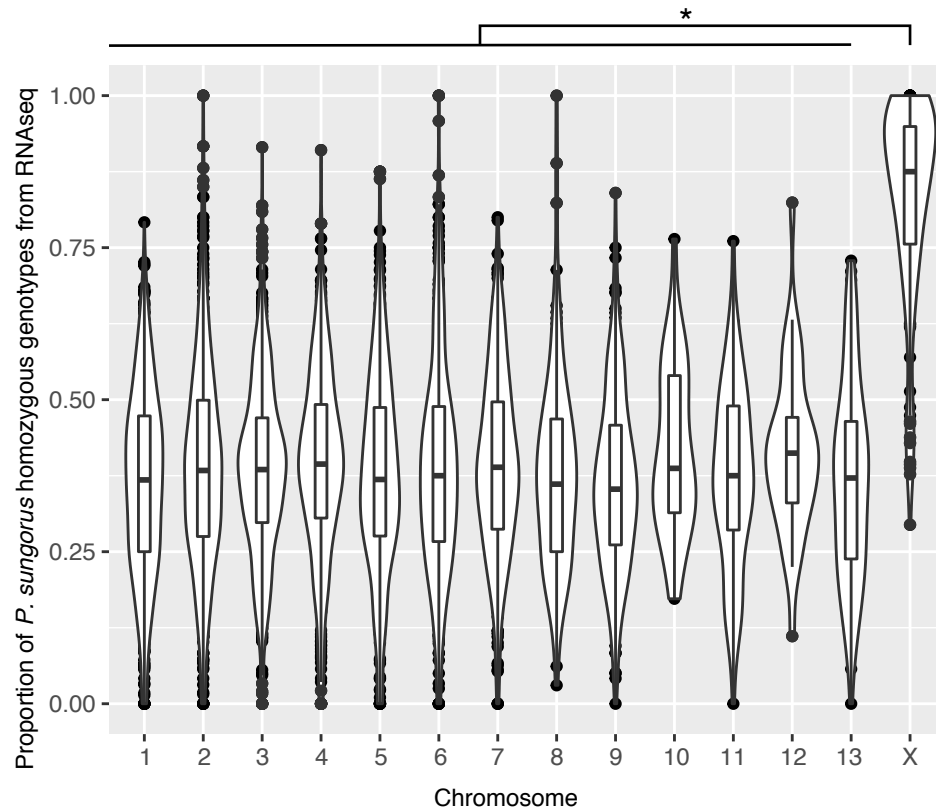


Figure S19. Allelic bias on the X chromosome in heterozygous BC females. The average proportion of genotypes for each mapped gene called as homozygous for the maternal *P. sungorus* allele is shown for each chromosome. Estimates for each gene were based on RNAseq reads from females inferred to be heterozygous based on their local genetic map genotype. No backcross individuals should be homozygous for the *P. sungorus* allele based on standard diploid expectations and unbiased gene expression, while paternally imprinted XCI should generate only homozygous *P. sungorus* genotypes in heterozygous females (proportion = 1.0). The autosomes showed a skew towards expression of *P. sungorus* alleles relative to these predictions, likely reflecting errors in the genetic map genotyping, calling heterozygous genotypes from using RNAseq data, and uncertainty in species origin of individual variants. Overall, the X chromosome showed strong allelic bias towards the *P. sungorus* maternal allele, consistent with intact paternal XCI. Star indicates a significant difference (adjusted $r^2 = 0.17$, $F_{1,3560} = 930.3$, $P < 0.0001$, ANOVA).

Table S1. A full description of all RAD markers including their ID, the linkage group they are found on, genetic position in centiMorgans, the position and polarization of the diagnostic SNV between *P. campbelli* and *P. sungorus*, and the sequence of the marker which always begin with TGCAGG (the restriction-enzyme cut-site of SbfI, i.e.: CC_TGCA^GG). SNVs in the sequence are denoted with standard IUPAC ambiguity codes.

Table S2. WGCNA modules generated from F₁ and pure species placental gene expression data. Color names are arbitrarily and randomly generated by the program, and have no additional meaning. The upregulated and downregulated modules are discussed in the manuscript are indicated as such. Counts of genes in each module, correspondence with previous pairwise analysis (Brekke *et al.* 2016), association with inheritance pattern and phenotypes, and enrichment for candidate imprinted genes indicated.

Table S3. WGCNA modules generated from BC placental gene expression data. As before, color names are arbitrarily and randomly generated by the program, and do not correspond whatsoever with the arbitrarily assigned names given to the F₁ data. The upregulated and downregulated modules are discussed in the manuscript are indicated as such. Counts of genes in each module, correspondence with F₁ network analysis, association with phenotypes, and enrichment for candidate imprinted genes indicated.

Table S4. A full description of the genetic locations of each gene from that was captured and associated with the map. Columns are: Linkage group (LG), position in centiMorgans (cM), gene name from the *P. sungorus* transcriptome (Trinity_Component), the exon that the SNP appears in (exon), the position of the SNP in the exon (snp_pos_in_exon), the gene name (Associated_Gene_Name), and the mouse ensemble gene ID of that gene (Ensembl_Gene_ID).

Table S5. SRA sequence accession numbers for each individual by sequence type.

