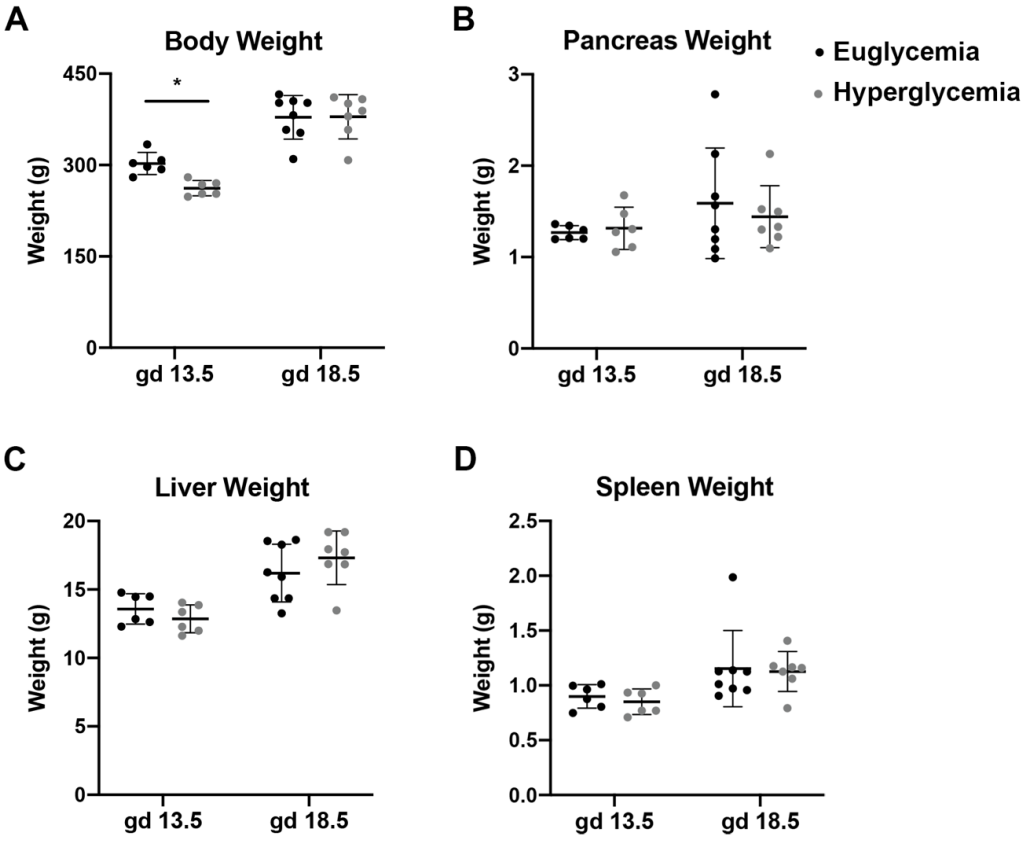
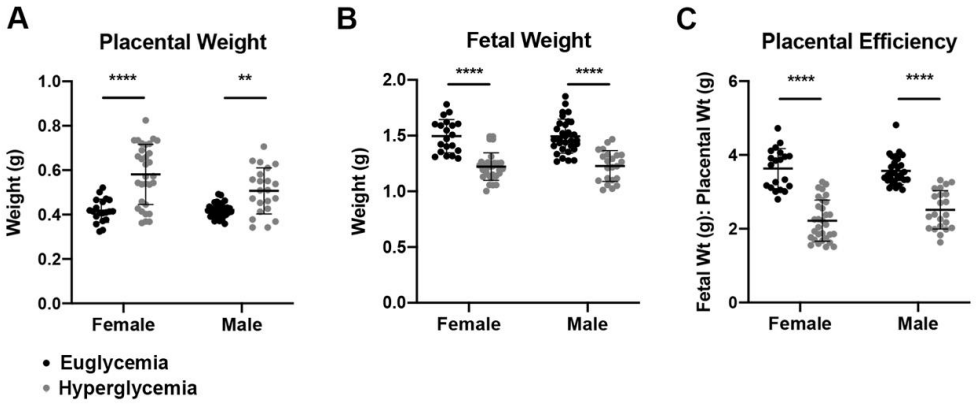
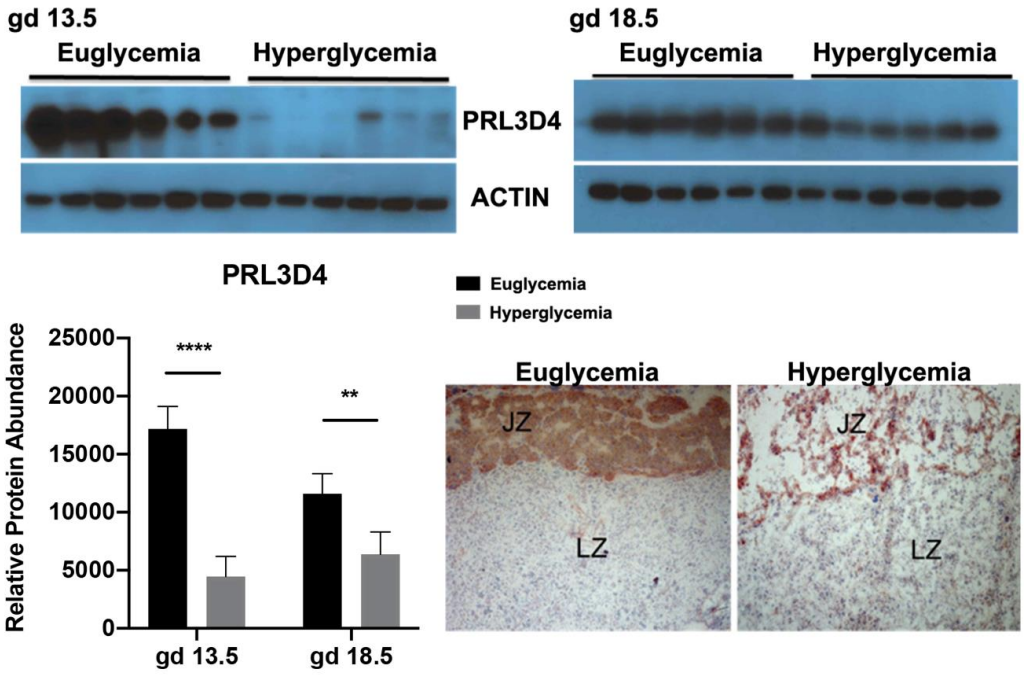


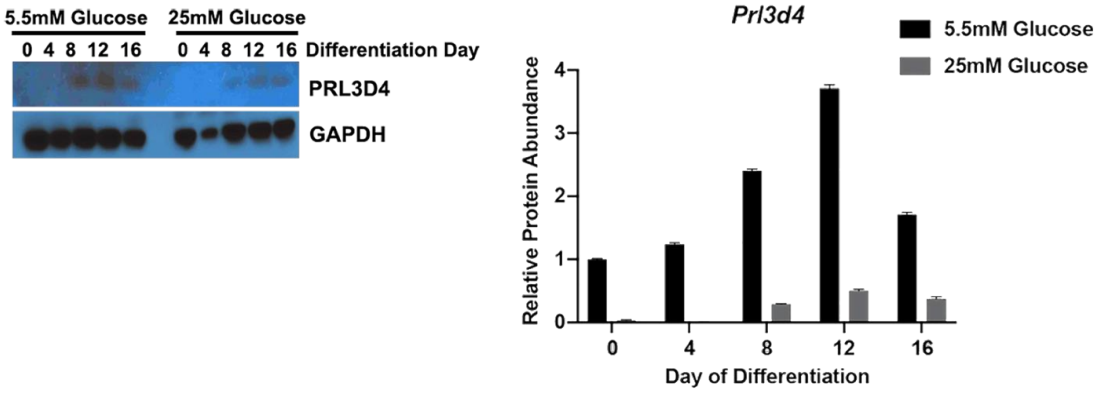
Supplemental Figure 1. Hyperglycemia has minimal effects on maternal weight. Total body, pancreas, liver, and spleen weights were measured in pregnant rats treated with vehicle control solution (black; 0.1 M citrate buffer, pH 4.2) or streptozotocin (gray) on gestation days (gd) 13.5 and 18.5. Maternal body weights were significantly less in rats with hyperglycemia on gd 13.5 ($n=6$ per group, $p<0.05$); however, maternal body weights of hyperglycemic and euglycemic rats were not significantly different on gd 18.5 ($n=8$ for Eug group, $n=7$ for Hyp group, $p>0.05$). Hyperglycemia did not affect pancreas, liver, or spleen weights on gd 13.5 or 18.5 ($n=8$ for Eug group, $n=7$ for Hyp group, $p>0.05$).



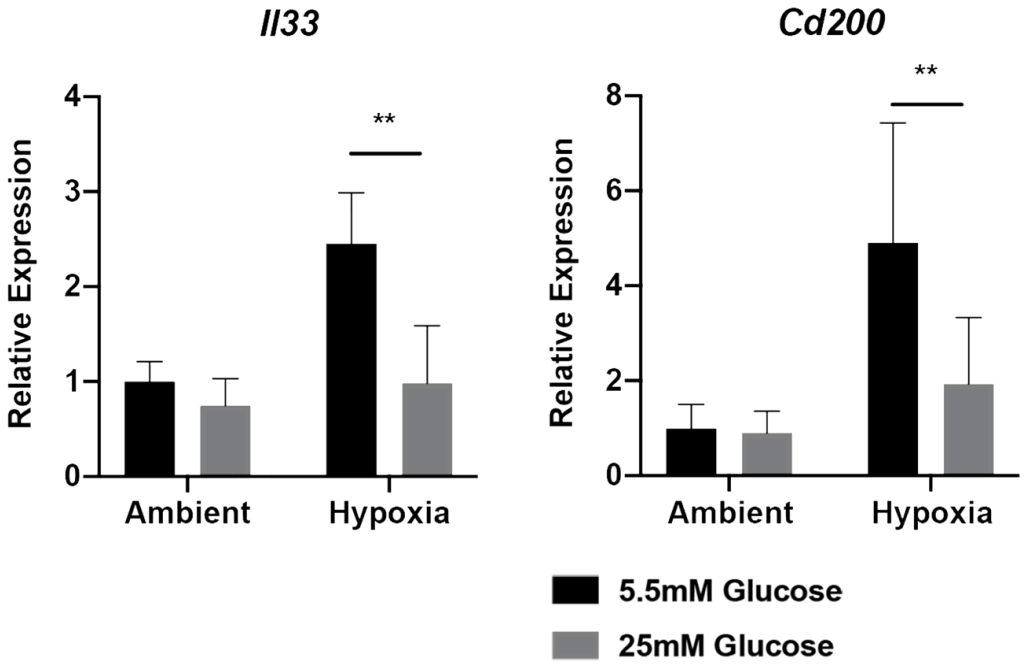
Supplemental Figure 2. Examination of sexual dimorphisms in hyperglycemia-induced placental malformation. Placental and fetal weights (g) were measured at time of sacrifice on gd 18.5 in rats with euglycemia (black) and hyperglycemia (gray). Hyperglycemia significantly increased placental weights of female (n=20; $p<0.001$) and male conceptuses (n=31; $p<0.01$) compared to euglycemia (female n=22, male n=31). Hyperglycemia significantly decreased weights of female (n=20, $p<0.0001$) and male fetuses (n=31, $p<0.0001$) compared to euglycemia (female n=22, male n=31). The ratio of fetal to placental weight (g), a measure of placental efficiency, was significantly decreased as a result of hyperglycemia in both female and male conceptus sites (Eug female n=22, Eug male n=31, Hyp female n=20, Hyp male n=31; $p<0.0001$).



Supplemental Figure 3. Hyperglycemia significantly reduced PRL3D4 protein expression in the junctional zone. Western blots of PRL3D4 protein levels in junctional zone tissue obtained from euglycemic or hyperglycemic pregnant dams on gestation days (gd) 13.5 and 18.5. PRL3D4 protein levels were quantified with ImageJ (NIH), normalized to beta actin (ACTIN), and graphed as 'Relative Protein Abundance' (y-axis). PRL3D4 protein levels are significantly decreased in hyperglycemic animals on gd 13.5 and 18.5 (n=6 per group, **p<0.01, ****p<0.0001). Representative photomicrographs depict immunohistochemical analysis of PRL3D4 (brown) expression in the junctional zones (JZ) and labyrinth zones (LZ) under conditions of euglycemia and hyperglycemia.



Supplemental Figure 4. High glucose decreases PRL3D4 in rat trophoblast stem (TS) cells. Western blot of PRL3D4 protein levels in rat TS cells cultured in differentiation medium containing 5 mM glucose (black) or 25 mM glucose (gray). Protein was collected from both treatment groups at five time points (days 0, 4, 8, 12, and 16). PRL3D4 protein levels were quantified with ImageJ (NIH), normalized to glyceraldehyde 3-phosphate dehydrogenase (GAPDH), and graphed as 'Relative Protein Abundance' (y-axis; n=5 per group).



Supplemental Figure 5. High glucose inhibits rat trophoblast stem (TS) cell hypoxia response. Rat TS cells were cultured in medium containing 5 mM glucose (black) or 25 mM glucose (gray) and were maintained in either ambient air or exposed to 24 h of low oxygen (0.5% O_2). RNA was isolated, and *Il33* and *Cd200* transcript levels were measured by RT-qPCR (n=4-6 per group, $p<0.01$).

Supplemental Table 1. Primer sequences for qRT-PCR analyses

Target	Forward	Reverse
18S	GCAATTATTCCCCATGAACG	GGCCTCACTAAACCATCCAA
Alox15	TGGATGGGATCAAGGCCAAT	CGAGGGCGTGAAAATAGGTG
Cd200	GTCTACTTGAATCTGATGTTA	TGTAAATACTGAGGACCC
Ceacam11	GCTTTACACCCTACGAGCCA	GCACTTGGTTCCTTTCCGTC
Cgm4	GTGTATCCGCAATGTCACCC	ATAGTGAGCTTGGCAGGGTT
Cts7	TGCTCCAAGGCCTGATTACA	TCTGCTTTTCCTCCTCTGGG
Gdp2	GAGAGTTACCAGGAGCACGT	CCCAGGTGATTGTCGTCTCT
Il33	CAACGACCAATCTGTTAG	CGGAGTAGCACCTTATCT
Mmp12	GCTGGTTCGGTTGTTAGG	GTAGTTACACCCTGAGCATAC
Pappa2	CGCTCTCTACTTCTGGGAGG	TCGAAAGTGGCTGTCAGGAT
Pcdh12	GAGCCTGGTTCGACTCTCTG	GGGCTTGGCCGAGTATTTAT
Phlda2	CGATGAGATTCTTTGCGAGGG	GTACTTGGAGGTGTGCTCCA
Plac1	TTGGCTGTCCTCCCAATCAT	AGCACAGGACACAGGAATCA
Prl3d4	AACCAACTGTGCTTGTGTCC	GAACGTGGATGGAAGCAGTG
Prl4a1	GCTCCTGGATGCCATAAAGA	TATTGGGCGATTGCAAGAAT
Prl5a1	ATGCGGCTGTCTAAGATTCAAC	CTTCCATGATACATCTGGGCAC
Prl7b1	AACAATGCCTCTGGCCACTGC	AGGCCATTGATGTGCTGAGACAGT
Prl8a3	AGGTGGGATGAGACTGTTGT	ACCTTCCATGGCACTCAGTT
Prl8a5	GAGAGCTGAAACCCTCCGTA	TGCACGATTTGAATGGCAGT
Prl8a7	GCCATCCATAAAGCTGAGACG	TGGGGTGTTTATGAGAGTTGAATG
Prl8a9	CCATTCGACTCTCTCAAACTC	GATCCAGGCACCCACAAAA
Taf7l	ACGGCATTACTCCACCACTT	ACAACTTCCCAGCGGACAC
Tpbpa	CAGCTGCCATACTCCCTGAT	TATGTCGAGCTCCTCCTCCT