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**Supplementary Fig. 1** Reduced proliferation and angiogenesis was observed in FGR. (***A***) Representative labelling of Ki67 (green) and endothelial cells (red; CD34) in cortex demonstrating an overall decrease in proliferating cells in the FGR brain. (***B***) Significant reductions in Ki67+ cell counts were found in the parenchyma and juxtavascular regions. (***C***) Analysis of Ki67+ vessels relative to the total number of vessels demonstrated a significant reduction in vessels undergoing proliferation in the FGR group. (***D***) Examination of angiogenic markers angiopoietin-1 (Ang 1; green) & angiopoietin-2 (Ang2; red) found similar labelling patterns in both NG and FGR. (***E*** *&* ***F***)Western blot analysis showed no significant overall difference in expression between NG and FGR. (***G***) Analysis of Ang2/Ang1 ratio demonstrated a mild but non-significant increase in FGR brains. All values are expressed as mean +/- SEM (minimum *n* = 6 for NG and FGR). Unpaired student’s t-test (◆*p* < 0.05 (perivascular); \*\**p* < 0.01 (parenchymal)) (Scale bars: 50m; ***D*** high magnification: 10m).

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**Supplementary Fig. 2** Glial interactions with transverse vessels demonstrate increased microglia and decreased astrocytic interaction in FGR.Representative labelling of microglia (grey) and astrocytes (green) interactions with vasculature (red) of the neonate brain. (***A***) NG demonstrated resting glial morphology, with long thin process extensions forming an organised network of tight interactions along the vasculature. (***a***) Ramified microglia are present in close proximity, while astrocytes showed end-feet interaction with vasculature (***a’***). (***B***) Representative labelling for FGR demonstrated an increase in activation of glia based on morphology, with retraction of extended processes and dense cellular bodies. FGR displayed close interaction of activated microglia with the vessel (higher magnification shown in ***b***). (***b’***) There was an evident decrease in GFAP labelling spanning the length of vessel. (***C***) In severe instances there was a high degree of activated juxtavascular microglia (***c***) and significant loss GFAP labelling along the vasculature with some regions completely void of astrocyte interaction (arrowheads; ***c’***). (***D*** & ***E***) Ibuprofen treated FGR and NG groups displayed glial morphology comparable with untreated NG, indicating a normalised resting state glial morphology and strong interaction with the vasculature (***d-d’*** & ***e-e’***). (Scale bars: 50m).

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**Supplementary Fig. 3** Elevated Claudin-1 labelling in FGR brain. (***A***) Robust labelling of tight junction protein claudin-1 (Cldn1; red) was observed at the pial surface, across cortical layers, and white matter of FGR brains at postnatal day 4. CD3+ cells were observed in regions with high degree of Cldn1 labelling (***Aa***). Cldn1 labelling showed strong co-localisation to astrocytes (GFAP; green) which displayed activation as characterized by disorganized and thickened process morphology at both parenchymal and perivascular regions (***Ab*** & ***Ac***). NG and FGR+Ibu brains showed limited Cldn1 labelling, predominantly restricted to astrocyte end-feet interacting with CD3+ cells and the vasculature (***B*** & ***C*** respectively). (***D***) Quantification of Cldn1 areal coverage demonstrated a significant elevation in FGR, which was partially ameliorated following ibuprofen administration. (***E***) Astrocytes in FGR brains displayed significantly elevated expression of Cldn1 compared with all groups. Ibuprofen administration significantly reduced the degree of Cldn1 labelling in FGR animals. All values are expressed as mean +/- SEM (minimum *n* = 6 for all groups). Two-way ANOVA with Tukey post-hoc test (\**p* < 0.05; \*\**p* < 0.01; \*\*\**p* < 0.001; \*\*\*\**p* < 0.0001) (Scale bars: 100m, ***Ab*** & ***c***: 50m, ***B*** & ***C*** inserts: 10m).