

Additional file

Title: Mechanism of piR-DQ590027/MIR17HG Regulation of Permeability across the glioma conditioned normal BBB

Additional file Summary

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Figure.S1 The permeability of glioma-conditioned normal BBB was increased. “ECs” represents co-cultured ECs (hCMEC/D3) with human astrocytes; “GECs” represents co-cultured ECs (hCMEC/D3) with glioma cells. (A) TEER values in the *in vitro* glioma-conditioned normal BBB model. Data represent the mean \pm SD ($n = 5$, each). $*P < 0.05$ vs. the ECs group. (B) HRP flux in the *in vitro* glioma-conditioned normal BBB model. Data represent the mean \pm SD ($n=5$, each). $*P<0.05$ vs. the ECs group.

Figure.S2 The prediction results of Bioinformatics software. (A) The predicted binding site for piR-DQ590027 in the base sequence of MIR17HG. (B) The predicted binding sites of MIR17HG and miR-153/miR-377. (C) The predicted binding sites of miR-153 and miR-377 in 3'UTR of FOXR2. (D) The predicted binding sites of FOXR2 and the promoter regions of ZO-1, claudin-5 and occludin respectively (“yellow” represents the binding sites; “red” represents transcription start site).

Figure.S3 The schematic cartoon underlying the mechanism of piR-DQ590027 /MIR17HG regulating the permeability of glioma conditioned normal BBB.

Figure S3

