Supplementary Materials

Expanded Methods

Characterization of BM-MSC

BM-MSC were identified using flow cytometric analysis (FACS). After being washed with PBS, BM-MSC were fixed and permeabilized (Invitrogen, Fixation & Permeabilization Buffer, 00-5123-43, 00-5223-56), then stained with antibodies. More than 90% of the cells expressed the BM-MSC surface markers CD90 and CD29 instead of the hematopoietic stem cell markers CD34 and CD45.

BM-MSC multi-differentiation identification

Osteogenic differentiation was carried out using an osteogenic medium consisting of ascorbic acid (50μM, sigma, 2043003), dexamethasone (10nM, Sigma, D4902), and β-glycerophosphate (10mM, Sigma, G9422). The medium was refreshed every 3 days for 21 days after which differentiation was identified using Alizarin Red S (Solarbio, G8550) staining. For adipogenic differentiation, the medium composed of dexamethasone (1μM, Sigma, D4902), indomethacin (200μM, Sigma, I7378), 3-isobutyl-1-methylxanthine (0.5mM, Sigma, I5879) and insulin (5μg/ml, Sigma, I2643) was used. Oil Red O staining was used to identify differentiation after 14 days.

Neurological function assessment

Neurological function was examined by using the neurological deficit score and the modified Garcia score system and were scoring by the investigator unaware of groupings. In short, a

neurological deficit was assessed using a scale ranging from 0 to 4. 0 = no apparent deficit, 1 = Ipsilateral forelimb weakness, 2 = Ipsilateral rotation, 3 = Body imbalance, torso tilted to the ipsilateral side, and 4 = no spontaneous motor activity or death. The evaluation of sensory-motor performance following tMCAO involved the modified Garcia score, which measures scores across five domains (body awareness, whisker sensation, limb balance, side movement, and forelimb gait), assigning a range of 0-3 points for each assessment.

Sensory-motor function assessment

To further assess the sensory-motor function of mice, rotarod test, foot-fault test, and adhesive removal test were performed as we described previously.[1] Mice were pre-trained for 3 consecutive days before tMCAO. Baseline data was obtained after the last training test, and tests were performed at 3d, 5d, 7d, 10d and 14d after ischemia. *Rotarod test* During a five-minute period, the speed of a rotating drum was accelerated from 5-50 rpm within five minutes and maintained at the speed. The latency of falling or spinning around on the rung was measured. *Foot-fault test* Mice were placed on a stainless-steel grid (20cm × 40cm with mesh size of 4cm^2) one meter elevated from the ground for the test. The number of total steps and forelimb foot-fault were recorded. *Adhesive removal test* The lesioned forepaws were taped with a 2x3mm adhesive tape. The time it took to remove the adhesive tape was used to measure tactile responses. Maximum observation time is 2 minutes.

Infarct volume analysis

Infarct volume of stroke mice was evaluated with 2,3,5-triphenyltetrazolium chloride (TTC, Sigma, T8877) staining or immunostaining of NeuN (Abcam, ab177487). In TTC staining, brain was isolated and cut into sections in mold (1 mm thick). The brain slices were then stained with 2% TTC dissolved in PBS. For immunologic staining of NeuN, six equally spaced coronal brain

sections encompassing the MCA territory were stained with NeuN antibodies. Infarct volume was analyzed with NIH ImageJ software. The infarct area was determined as the difference between the TTC stained area (red) or NeuN-positive area of contralateral hemispheres and ipsilateral hemispheres. Brain infarct volume was determined by multiplying the mean area of tissue loss by the distances between the two adjacent stained brain slices.

Intestinal permeability analysis

Functional integrity of the gut barrier assessed by intestinal permeability assay. In short, the mice were deprived of food for a period of 6h prior to administering 440mg/kg FITC-dextran (Sigma, FD4) through oral gavage. After 4 hours of gavage, the serum was obtained and examined with excitation at 485nm and emission at 528nm (Bio-Tek Synergy H1MF, Agilent).

Histologic analysis of mouse colon

A segment (1cm) of distal colon was isolated, luminal content was gently removed, and the tissue was fixed in 4% paraformaldehyde at room temperature for 48 hours. Fixed tissue was embedded in paraffin and sections prepared for histologic staining with hematoxylin-eosin (HE) and Alcian blue (AB). As for the Swiss roll, the colon tissue was separated, and the intestinal contents were gently removed. After fixation and embedded with paraffin, a longitudinal section of the entire colon was performed. Crypt length was measured in HE- or AB- stained sections in well-oriented crypts that extended the full length of the mucosa. Correspondingly, the crypt width was also measured. Number of goblet cells were measured by counting AB positive granules in well-defined crypts. In most cases, crypt length, width and length and number of goblet cells per crypt were averaged over at least 10 well-defined crypts. Histomorphological evaluation was measured according to the established scoring system.[2]

Evaluation of the mucus layer in the colon

To preserve the colon mucus layer for analysis, a section of distal colon containing fecal pellets at both edges was gently excised and fixed in Carnoy's solution (Servicebio, G1120) immediately. Following a 48-hour period, the stable tissue underwent two rinses with 100% methanol, succeeded by two rinses with 100% ethanol. Subsequently, it was embedded in paraffin and cross-sectioned through fecal pellets. To maintain the integrity of the mucus layer, water was abstained from during all fixation and processing procedures. Slides were stained with AB. In images of intact transverse sections of the colon, the area of AB staining around the fecal mass adjacent to the epithelium is measured to determine the average thickness of the mucus layer.

16S ribosomal RNA gene amplicon sequencing

The human fecal samples obtained from recruited patients with AIS or HCs. For collecting mice fecal samples, mice were placed individually in empty autoclaved cages and allowed to defecate freely in the morning at 3d after tMCAO. Fecal microbial genomic DNA was extracted, and 16S ribosomal RNA gene hypervariable regions V3-V4 have been amplified as described previously.[3]

Bioinformatics analysis of microbiota

QIIME (1.9.1) and R (4.2.2) were utilized for the analysis of 16S ribosomal RNA gene amplicon sequencing data. After normalizing the sequencing depth of all samples, UniFrac distances were applied to analyze β-diversity.To consider the distance matrix-based relationships of the various groupings, the data underwent downsizing through principal co-ordinate analysis (PCoA).To determine the similarity between samples, the UPGMA algorithm, which does not consider weights, was employed. Linear discriminant analysis (LDA) effect sizes (LEfSe) were used to determine the different taxa between groups, and LDA scores > 2 was set as the threshold.

Stroke dysbiosis index was calculated according to previous described.[4] The flora of patients and healthy individuals in the study were compared at the genus level using the matrix of relative abundance, and the differential genera were compared by R using welch *t* test. To alleviate the uncertainty introduced by small sample sizes, the obtained differential genus were further compared to another AIS cohort.[4] The intersection was presented in **Supplementary Figure 1A** using a Venn diagram. There were 6 genera finally selected: *Coprococcus*, *Roseburia*, *Un Erysipelotrichaceae*, *Un Clostridiaceae*, *Parabacteroides* and *Bilophila*. The SDI index was calculated by the formula: SDI = {average [∑ abundance (stroke enriched genera)]- average [∑ abundance (control enriched genera)]} ×100. In the mouse stroke model, the calculation of SDI calculated by the same methodology with the genera as follows: *Terrimonas*, *Dyadobacter*, *Pseudoalteromonas*, *FFCH7168*, *Edaphobaculum*, *A2*, and *Verrucomicrobium*.

Bulk RNA sequencing

Bulk RNA sequencing (RNA-seq) was conducted using the RNA extracted from colon and liver tissue. Total RNA in tissue was extracted with the TRIzol Reagent (Sangon Biotech, B511311-0100). RNA-seq libraries were generated using the NEBNext® Ultra™ RNA Library Prep Kit for Illumina. After qualification, the different libraries were sequenced on Illumina NovaSeq 6000 platform. Differential expression genes (DEGs) analysis of two groups was performed by R using the “DESeq2” package. To evaluate the functional enrichment of DEGs after BM-MSC transferred, Kyoto Encyclopedia of Genes and Genomes (KEGG) pathway enrichment, Gene Set Enrichment Analysis (GSEA) and Reactome analysis were conducted, with “clusterProfiler” package by R. Transcriptional level of tight junction related genes or mucin related genes were visualized by heatmap with the “pheatmap” package by R.

BM-MSC labeling and tracing

BM-MSC stained with DIR (Meilunbio, MB12482-1) was transplanted through the inner canthal orbital vein at 2h after reperfusion. Mice were sacrificed at 2h, 24h and 72h after transplantation. AniView600 (BLT) was used to measure the fluorescence intensity. The results were further validated by immunofluorescence after intravenous injection of WGA-labeled (Invitrogen W7024, 1μg/ml) BM-MSC in tMCAO mice.

Real time polymerase chain reaction (RT-PCR)

TRIzol (Sangon Biotech, B511311-0100) or the commercial kit (ESscience, RN001) was utilized to extract total RNAs from tissues or cells. Then, cDNA was synthesized using Fast Reverse Transcription kit (ESscience, RT001). Real time polymerase chain reaction (RT-PCR) was performed using SYBR Green qPCR Mix (Dongshengbio, P2092a). Primers were listed in **Supplementary Table 2**. *Gapdh* or *Actb* were used to normalize relative expression.

Western blot

Samples were lysed with RIPA (Beyotime, P0013) containing phosphatase and protease inhibitor (Meilunbio, MB12707). The Western blot analysis was conducted using the standard SDS-PAGE technique and chemiluminescence detection reagents (Meis, MF074-01) for enhanced visualization. Primary antibodies used were indicated in **Supplementary Table 3**. Image J (NIH) was used to evaluate immunoreactivity.

Enzyme linked immunosorbent assay (ELISA)

The ELISA was conducted using plasma or serum samples as per the instructions provided by the manufacturer. To limit the variation in plasma levels due to daily rhythms, blood samples from mice used to assay GC levels were collected during a 2-hour window between 6:00 PM and 8:00 PM. ELISA Kit used in the study was listed in **Supplementary Table 4**.

Immunofluorescence staining

Mice were sacrificed at indicated time points. Following adequate perfusion with 20ml of PBS and 20ml of 4% paraformaldehyde, the brains were sliced into coronal sections measuring 25μm, while the distal colon sections and liver sections were both sliced into 8μm serial sections using a frozen microtome. The MC38 or hepatocytes were cultured on poly-L-lysine (Sigma, P1274) coated coverslips and were fixed with 4% paraformaldehyde after treatment. Tissue sections or fixed cells were washed and incubated with primary antibodies overnight. After washed, sections or cells were incubated with secondary antibodies for 1h at room temperature. The primary and secondary antibodies used were listed in **Supplementary Table 3**. Images were captured with a confocal microscopy (Leica, TSC SP8) or TissueFAXS 7.0 (TissueGnostics, Austria) and were processing used Image J (NIH).

Lentiviral infection of murine primary hepatocyte

To knockdown the expression of PPARγ in murine primary hepatocyte. Four shRNA were designed and two efficiently knocked down the expression of PPARγ, whose sequences were: *Pparg* KD1: 5'-CCGGGCTCCACACTATGAAGACATTCTCGAGAATGTCTTCATAGTGTGGAGCTTTTT T-3'; *Pparg* KD2: 5'-CCGGGCCTCCCTGATGAATAAAGATCTCGAGATCTTTATTCATCAGGGAGGCTTTTT T-3'; *Pparg* KD3: 5'-CCGGGCCCTGGCAAAGCATTTGTATCTCGAGATACAAATGCTTTGCCAGGGCTTTTT T-3'; *Pparg* KD4: 5'-CCGGGCCCTTTACCACAGTTGATTTCTCGAGAAATCAACTGTGGTAAAGGGCTTTTT T-3'. After transforming the engineered transfer vectors into DH5α E. coli, they were subsequently

isolated using the Endo-free Plasmid Mini Kit (Omega, D6950). The plasmid combination consisting of pSPAX2, pMD2.G, and the vector was diluted in OPTI-MEM (GBICO, 31985070), and PEI MAX 25K (Polysciences, 23966) was utilized as the transfection agent. OPTI-MEM-containing plasmids were introduced into 293T cells and left to incubate for 8h before being replaced with a new medium. After 48h, the cell debris was eliminated by centrifuging the supernatant at 800g for 10min. The lentiviral medium was applied to primary mouse liver cells for 48h to achieve shRNA-induced *Pparg* knockdown, which was assessed using RT-PCR or western blotting for efficiency.

Bisulfite sequencing PCR analysis (BSP)

Liver of 3 days after tMCAO mice was isolated and subjected to BSP analysis. Briefly, CpG islands were predicted and BSP primer was confirmed used EMBOSS CpG Plot tool (http://www.ebi.ac.uk/Tools/emboss/cpgplot/) and MethPrimer (http://www.urogene.org/methprimer/). And bisulfite conversion of genomic DNA was performed using EZ DNA Methylation-Gold^{TM} kit (ZYMO Research, D5005). BSP primers were designed to amplify the target fragment and the production was subjected to clone sequencing.

Targeted Metabolomics for bile acid

Bile acid of 3 days after tMCAO mice was collected. Then ultra-high performance liquid chromatography coupled to tandem mass spectrometry (UHPLC-MS/MS) system (ExionLC™ AD UHPLC-QTRAP 6500+, AB SCIEX Corp., Boston, MA, USA) was used to quantitate bile acids.

178 # References

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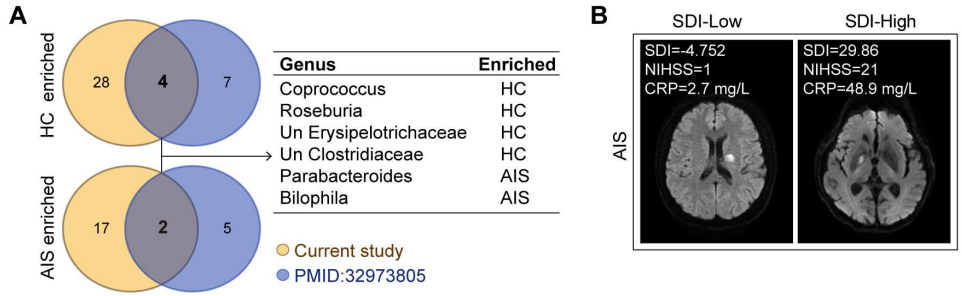
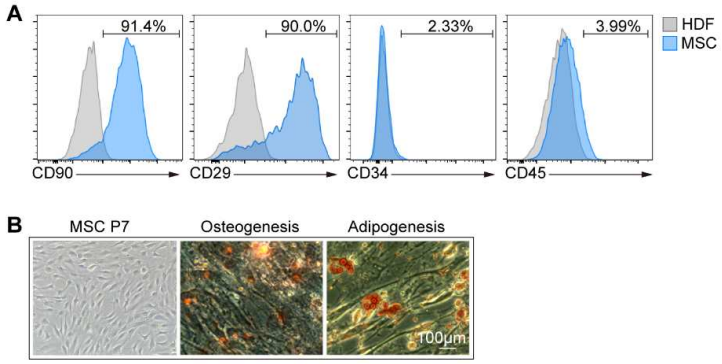
Supplementary Figures and legends

Supplementary Figure 1



Supplementary Figure 1. Stroke dysbiosis index (SDI) in AIS patients and its correlation with clinical severity. (**A**) Venn diagram showed the genus enriched in healthy control (HC) and AIS patients of two studies. The six enriched genus were further used to calculate the stroke dysbiosis index (SDI) of patients. (**B**) Representative images of the magnetic resonance diffusion weighted imaging (MR-DWI) and its clinical indicators of AIS severity of patients in groups between SDI-low and SDI-high groups. SDI was divided into SDI-low (SDI ≤ 1.102, median of the cohort) and SDI-high (SDI > 1.102, median of the cohort) groups.

199 *Supplementary Figure 2*



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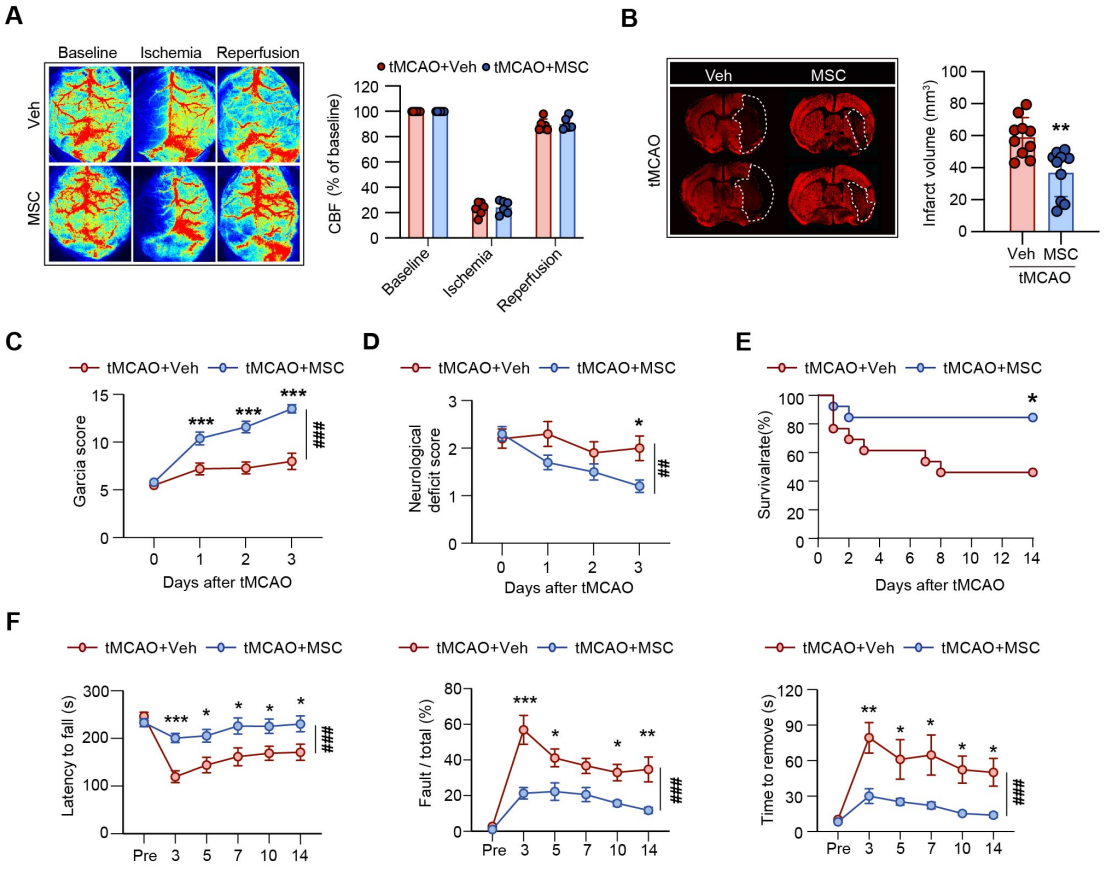
201 **Supplementary Figure 2. Identification of BM-MSC and multi-**
202 **lineage differentiation induction. (A)** Surface molecules of BM-MSC identified by flow
203 cytometry. Human dermal fibroblast (HDF) cells were used as negative control. Experiments were
204 repeated for three times. (**B**) Representative images of BM-MSC in bright field, adipogenesis
205 (stained with Oil Red O), and osteogenesis (stained with Alizarin Red S). BM-MSC (Passage
206 number 7, P7) was used in the experiments and repeated for three times.

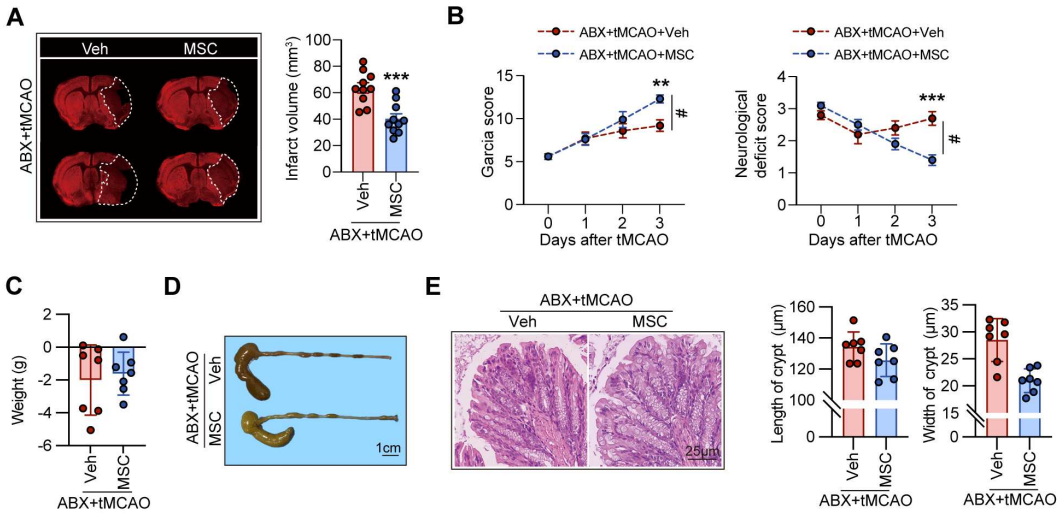
Supplementary Figure 3



Supplementary Figure 3. BM-MSC transfer ameliorated stroke severity. WT male C57/BL6 mice were subjected to 60min of tMCAO, then treated with a single dose of BM-MSC ($2x10^6$ cells per mice, i.v.) or equal volume of vehicle (Veh, i.v.) right after reperfusion. Animals were sacrificed at 3- or 14-d after tMCAO. **(A)** Regional cerebral blood flow (CBF) was not significantly different between the two groups at baseline, ischemia and 15 minutes after reperfusion. $N = 6$ in each group. **(B)** Infarct volume of stroke mice was quantified with 2,3,5-triphenyltetrazolium chloride (TTC) staining or immunostaining of NeuN with coronal sections collected at 3d after tMCAO. Representative images were displayed with dashed lines outlined

the infarct area. $N = 10$ in each group, $^{**}P < 0.01$, compared with Veh group by Student's t test (mean ± SD). (**C**) Garcia score of mice were evaluated at 0-3 d after tMCAO. $N = 10$ in each group. $^{***}P < 0.001$ compared to Veh group at indicated time points; $^{###}P < 0.001$ compared to Veh group in the whole experiment; by two-way ANOVA (standard error of the mean). (**D**) Neurological deficit score of mice were evaluated at 0–3 d after tMCAO. $N = 10$ in each group. $^{*}P < 0.05$ compared to Veh group at indicated time point; $^{##}P < 0.01$ compared to Veh group in the whole experiment; by two-way ANOVA (mean ± SD). (**E**) Survival analysis of mice in each group was recorded at 0–14 days after tMCAO. $N = 13$ in each group. $^{*}P < 0.05$, compared with Veh group by log-rank test. (**F**) Sensorimotor functions of the stroke models were accessed by rotarod test (left), foot-fault test (middle) and adhesive-removal test (right) at 3-14d after tMCAO. $N = 8$ in each group, $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$ compared to Veh group at indicated time points; $^{###}P < 0.001$ compared to Veh group in the whole experiment; by two-way ANOVA (mean ± SD).

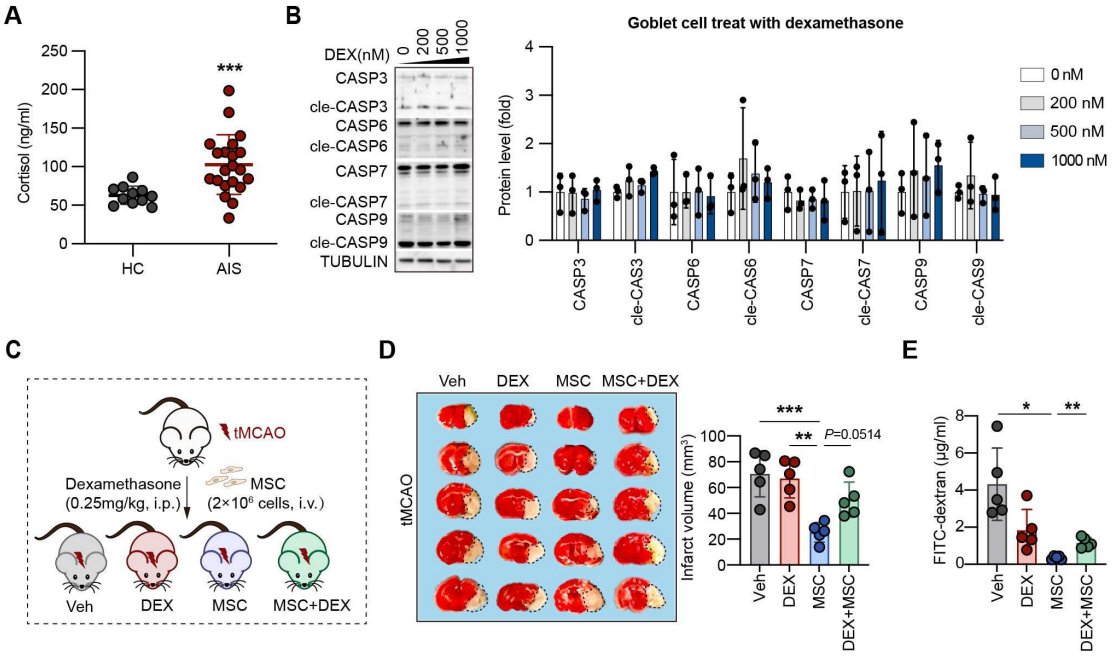
Supplementary Figure 4



Supplementary Figure 4. The protection of BM-MSC transfer in stroke was un-abolished by ABX. Gut microbiota was depleted with pre-treatment of broad-spectrum antibiotics (ABX) for 28 days before tMCAO (ABX+tMCAO), then treated with a single dose of BM-MSC ($2x10^6$ cells per mice, i.v.) or equal volume of vehicle (Veh, i.v.) right after reperfusion. **(A)** Infarct volume of ABX+tMCAO mice was quantified with TTC staining or immunostaining of NeuN with coronal sections collected at 3d after tMCAO. Representative images were displayed with dashed lines outlined the infarct area. $N = 10$ in each group, $^{***}P < 0.001$, compared with Veh group by Student's t test (mean ± SD). (**B**) Left: Garcia score of ABX+tMCAO mice were evaluated at 0-3 d after stroke. $N = 10$ in each group. $^{**}P < 0.01$ compared to Veh group at indicated time points; $\#P < 0.05$ compared to Veh group in the whole experiment; by two-way ANOVA (mean ± SD). Right: Neurological deficit score of ABX+tMCAO mice were evaluated at 0–3 d after stroke. $N = 10$ in each group. $^{***}P < 0.001$ compared to Veh group at indicated time point; $\#P < 0.05$ compared to Veh group in the whole experiment; by two-way ANOVA (standard error of the mean). (**C**) Weight loss of Veh- and MSC-treated group after ABX+tMCAO calculated by the

weight of d3-d1. $N = 7$ mice in each group, ns, no significant difference between the two groups, by Student's t test (mean ± SD). (**D**) Representative of colon picture of Veh- and MSC-treated group after ABX+tMCAO. (**E**) Representative colon section stained by HE of Veh- and MSC-treated group after ABX+tMCAO, the crypt length and width were also quantified. $N = 7$ mice in each group, ***$P < 0.001$, compared to Veh group, by Student's t test (mean ± SD).

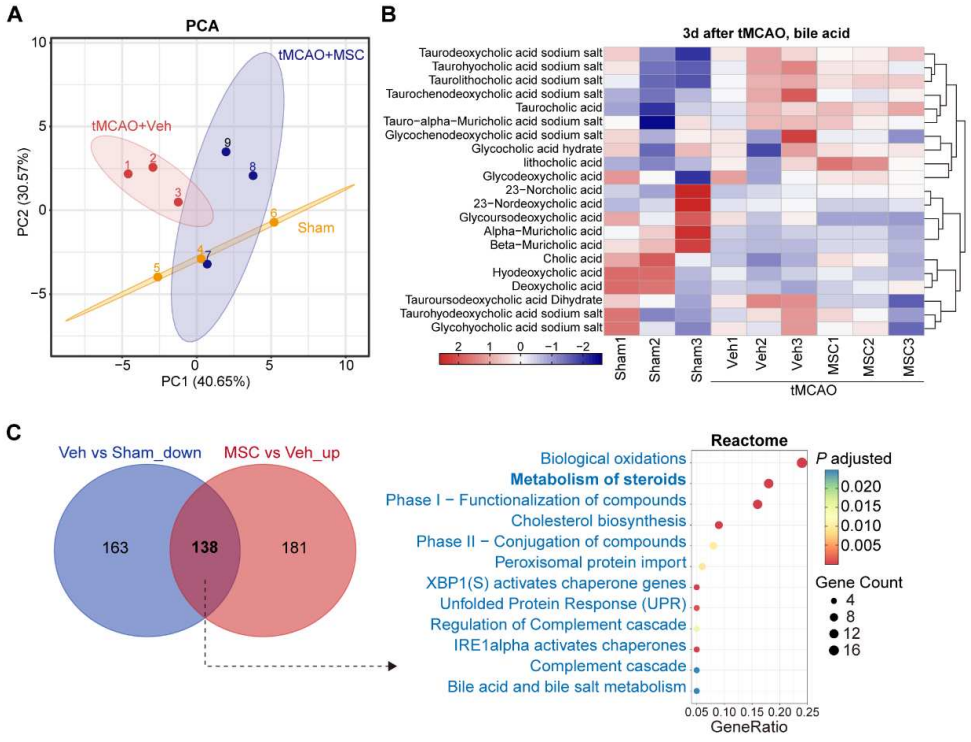
Supplementary Figure 5



Supplementary Figure 5. Impacts of dexamethasone on goblet cell survival and therapeutic effects of BM-MSC transfer. (**A**) Plasma cortisol concentrations in AIS patients ($N = 21$) and HC ($N = 11$). ***$P < 0.001$; by Welch's t test (mean ± SD). (**B**) Goblet cell was treated with 0, 200, 500, 1000nM dexamethasone (DEX) for 18h and the apoptosis was assessed by western blot. There is not significantly change before and after DEX treated. Experiments were repeated for three times, by one-way ANOVA (mean ± SD). (**C**) The experiment designed. WT male C57/BL6 mice were subjected to 60min of tMCAO, then treated with a single dose of BM-MSC ($2x10^6$ cells per mice, i.v.) or equal volume of vehicle (Veh, i.v.) right after reperfusion. Mice in the DEX group were treated with dexamethasone (0.25 mg/kg, i.p) at 2 hours after reperfusion, while mice in the DEX+MSC group received dexamethasone (0.25 mg/kg, i.p) in addition to BM-MSC transplantation. (**D**) Infarct volume of stroke mice were quantified with 2,3,5-triphenyltetrazolium

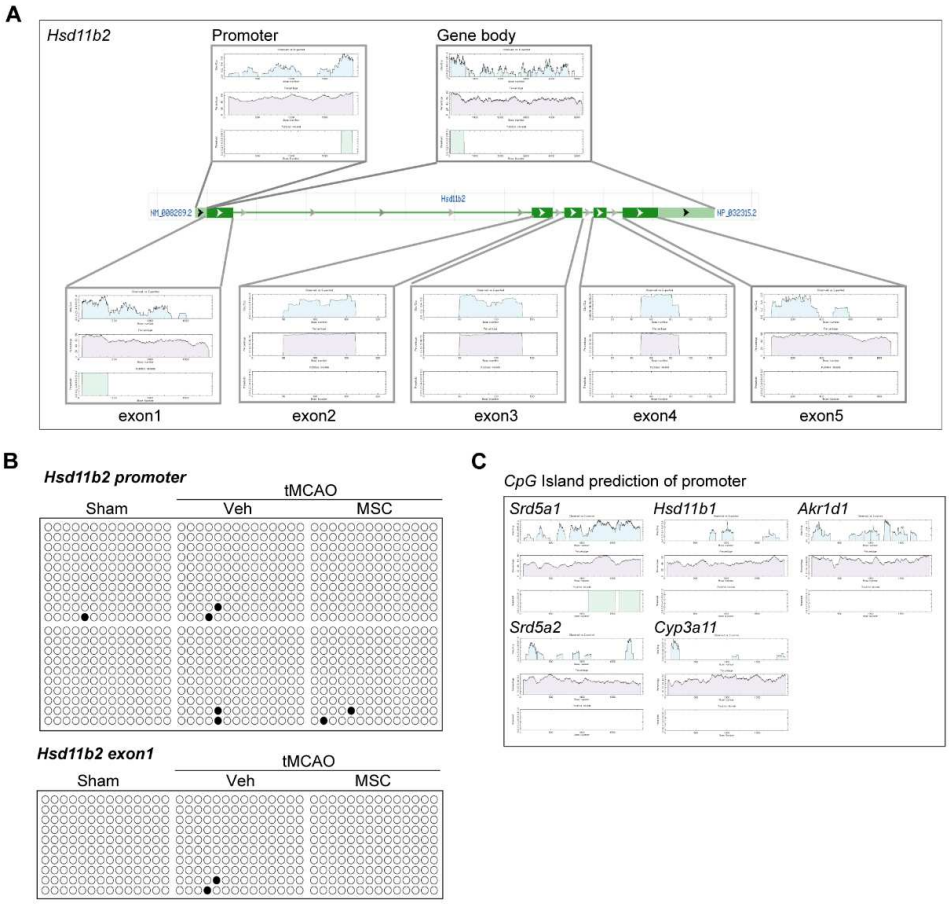
chloride (TTC) staining or immunostaining of NeuN with coronal sections collected at 3d after tMCAO. Representative images were displayed with dashed lines outlined the infarct area. $N = 5$ in each group, $^{**}P < 0.01$, $^{***}P < 0.001$, by one-way ANOVA (mean ± SD). (**E**) Intestinal permeability was assessed using the FITC-dextran permeability. $N = 5$ in each group, $^{*}P < 0.05$, $^{**}P < 0.01$, by one-way ANOVA (mean ± SD).

Supplementary Figure 6



Supplementary Figure 6. Changes in liver metabolism after BM-MSC treatment. (**A**) Principal component analysis (PCA) by targeted metabolomics of bile acid in the Sham, Veh and MSC treated groups. $N = 3$ in each group. (**B**) Heatmap showing the expression patterns of bile acid by targeted metabolomics in Sham, Veh and MSC treated groups. $N = 3$ in each group. (**C**) Liver tissue of mice was isolated at 3d after tMCAO and subjected to bulk RNA sequencing (RNA-seq). $N = 3$ in each group. Left: The venn diagram showed the intersection of the down-regulated DEGs in Veh versus Sham groups (blue circle) and up-regulated DEGs in MSC- versus Veh-treated groups (red circle). Right: Reactome pathway analysis of the DEGs in the intersection of Sham, Veh- and MSC-treated groups.

Supplementary Figure 7



Supplementary Figure 7. BM-MSC did not significantly alter the methylation level of hepatic *Hsd11b2* after stroke. (**A**) Prediction of the CpG island in the promoter region and gene body of *Hsd11b2.* (**B**) Bisulfite sequencing PCR analysis of *Hsd11b2* promoter region and *exon1* region showed the lacked methylated CpGs in liver of Sham and tMCAO (both Veh- and MSC-treated). (**C**) Prediction of the CpG island in promoter region of other glucocorticoid metabolism related genes, included *Srd5a1*, *Hsd11b1, Akr1d1, Srd5a2, and Cyp3a11.*

Supplementary Tables

Supplementary Table 1. Demographic characteristics of the AIS patients and healthy controls.

Demographics	Healthy control (*N*=40)	AIS patient (*N*=74)	*P*
Age, y (mean ± SD)	68.45±1.147	65.50±1.383	0.6637
Females sex, *N* (%)	17 (42.50%)	29 (39.19%)	0.7309
Diabetic mellitus, *N* (%)	3 (7.50%)	18 (24.32%)	0.0270
Hypertension, *N* (%)	28 (70.00%)	48 (64.86%)	0.0001
Hyperlipidemia, *N* (%)	21 (52.50%)	22(29.73%)	0.0167

P values were obtained by *Mann Whitney test* and *chi-square test.*

293 **Supplementary Table 2. QPCR primers used in the study.**

Gene	Forward primer	Reverse primer
Hsd11b1	CAGAAATGCTCCAGGGAAAGAA	GCAGTCAATACCACATGGGC
Hsd11b2	GGTTGTGACACTGGTTTTGGC	AGAACACGGCTGATGTCCTCT
Cyp3a11	GTGCTCCTAGCAATCAGCTT	CAGTGCCTAAAAATGGCAGAGG
Srd5a1	GAGTTGGATGAGTTGCGCCTA	GGACCACTGCGAGGAGTAG
Srd5a2	GATCCTGTGCTTTGGGAAACC	GCATCCCTACCGACACCAC
Akr1d1	TGCACACCACCAAATATCCCT	CTTCACTGCCACATAGGTCTTC
Actb	AGGGAAATCGTGCGTGACAT	GAACCGCTCATTGCCGATAG
Gapdh	CCCTTAAGAGGGATGCTGCC	TACGGCCAAATCCGTTCACA

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295 **Supplementary Table 3. Antibodies used in this study.**

Antigen	Source	Identifier
anti-human CD45-PerCPCy5.5	Biolegend	368504
anti-human CD90-FITC	Biolegend	328108
anti-human CD29-PE	Biolegend	303004
anti-human CD34-APC	Biolegend	343509
rabbit anti-HSD11B1	Affinity	DF3972
rabbit anti-HSD11B2	Affinity	DF9418
rabbit anti-SRD5A1	Proteintech	26001-1-AP
rabbit anti-PPARγ	Proteintech	16643-1-AP
mouse anti-HSP90	Proteintech	60318-1-Ig
rabbit anti-Caspase 3	Proteintech	19677-1-AP
rabbit anti-Caspase 7	Proteintech	27155-1-AP
rabbit anti-Caspase 9	Proteintech	10380-1-AP
mouse anti-ACTB	Proteintech	66009-1-Ig
rabbit anti-TUBULIN	Proteintech	11224-1-AP
rabbit anti-MUC2	Abcam	ab272692
rabbit anti-ZO1	Invitrogen	617300
rabbit anti-CLDN4	Affinity	AF5350
rabbit anti-NeuN	Abcam	ab177487
anti-rabbit secondary antibody conjugated with Cy3	Jackson	115-165-003
anti-rabbit secondary antibody conjugated with Alexa Fluor 488	Jackson	111-545-003
anti-rabbit secondary antibody conjugated with Alexa Fluor 647	Jackson	111-605-003
DAPI Fluoromount-G	Abcam	ab104139

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297 **Supplementary Table 4. The ELISA kits used in this study.**

Name	Source	Identifier
human LPS ELISA Kit	FineTest	EU3126
human LBP ELISA Kit	CUSABIO	CSB-E09629h
human cortisol ELISA Kit	Elabscience	E-EL-0157c
mouse LPS ELISA Kit	MEIMIAN	MM-0634M1
mouse LBP ELISA Kit	MEIMIAN	MM-44515M2
mouse cortisol competitive ELISA Kit	multi sciences	EK8100
mouse MUC2 ELISA Kit	MEIMIAN	MM-44508M1

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