SUPPLEMENTAL MATERIAL

SAH model establishment

Male, wild type C57BL/6, average weighing 25–30g, were obtained from the Laboratory Animal Services Centre of the Chinese University of Hong Kong. Transgenic mice used in this study including B6.129S1-Stat3^{tm1Xyfu}/J (homozygous *STAT3$^{flox/flox}$*) and B6J.B6N(Cg)-Cx3Cr1$^{tm1.1(cre)Jung}$/J (Homozygous *Cx3Cr1$^{Cre/Cre}$*) were purchased from the Jackson Laboratory (JAX Stock #016923 and #25524, JAX®, ME, USA). All of the animals were housed in a temperature-controlled environment, with a 12-hour light-dark cycle and free access to food and water. The mice expressing *STAT3$^{flox/flox}$ Cx3Cr1$^{cre/-}$* were selected for subsequent experiments. The littermate carrying *STAT3$^{flox/flox}$ Cx3Cr1$^{-/-}$* was used as control; the sham model was operated in the same procedures of SAH except without filament perforation of intracranial vascular. The procedures involving animals and their care were conducted under the approval of the Ethics Committee of the Chinese University of Hong Kong.

Mice were anesthetized and fixed in a supine position in advance, and all the surgical operations were through a surgical microscope. Firstly, make a 1-cm incision in the midline of the neck, dissecting the left common carotid artery (CCA), left external carotid artery (ECA) and left internal carotid artery (ICA). The ECA was ligated as far cranially as possible, the occipital artery was exposed and coagulated to avoid bleeding. Two 1.5-cm-length 5–0 silk sutures were used for filament fixation. Temporarily block the blood of ECA with a microclip, and make an incision on the ECA for filament insertion then a 20-mm-long blunted 5–0 monofilament nylon suture was inserted from ECA to the lumen of ICA continue to the intracranial vessels. The vessel was perforated at the bifurcation of the middle cerebral artery (MCA) where the resistance was encountered. Then immediately withdrawn the filament to introduce the bleeding into

subarachnoid space. The sham model was operated in the same procedures except without filament perforation [21]. The mice were maintained at 37 °C throughout the operation and recovery. Ointment protecting vision was applied to their eyes. Buprenorphine was injected intraperitoneally (i.p.) twice a day for consecutive 3 days for analgesia. Bodyweight was evaluated on day 1, 3, 5, and 10 after SAH induction for the wellbeing of mice.

Establishment of STAT3 conditional knock-out mice model

The Cre-LoxP system was utilized. Mice with a STAT3 deletion in microglia were generated by crossing mice with the floxed STAT3 alleles with mice expressing Cre under the control of the Cx3Cr1 promoter. Homozygous $STAT3^{flox/flox}$ mice were mated to homozygous $Cx3Cr1^{Cre/Cre}$ mice to generate the hybrid mice of the first generation carrying Cx3Cr1-Cre and heterozygous STAT3 (F1: $STAT3^{flox/+}Cx3Cr1^{Cre/-}$) The F1 mice were then crossed with the homozygous $STAT3^{flox/flox}$. The hybrids of the F2 generation was expected to disperse the genotypes into four types, which were $STAT3^{flox/flox}$ $Cx3Cr1^{cre/-}$, $STAT3^{flox/flox}$ $Cx3Cr1^{-/-}$, $STAT3^{flox/+}$ $Cx3Cr1^{cre/-}$, and $STAT3^{flox/+}$ $Cx3Cr1^{-/-}$. The F2 mice expressing $STAT3^{flox/flox}$ $Cx3Cr1^{cre/-}$ ($Cx3Cr1^{cre/-}$ as a heterozygote, means Cx3Cr1 positive cell have dominant Cre thus can work in the Cre-LoxP system and can cooperate with LoxP to specific deplete the STAT3 in Cx3Cr1-Cre positive cells.) were selected for subsequent experiments. The littermate carrying $STAT3^{flox/flox}$ $Cx3Cr1^{-/-}$ was used as control. Genotype assay was described in the online-only Data Supplement.

Generation of microglia specific STAT3 knockout mice

To investigate the role of STAT3 in microglia-dependent neuroinflammation in SAH, we generated transgenic mice in which STAT3 was deficient in a microglia-specific manner. The genotypes of F1 and F2 generation ($STAT3^{flox/flox}$ $Cx3Cr1^{cre/-}$, $STAT3^{flox/flox}$ $Cx3Cr1^{-/-}$, $STAT3^{flox/+}$

Cx3Cr1$^{cre/-}$, and *STAT3*$^{flox/+}$ *Cx3Cr1*$^{-/-}$) were determined by PCR. Theoretically, each of the four phenotypes of F2 generation accounted for one-fourth of the total number of the F2 generation. Mice carrying *STAT3*$^{flox/flox}$ *Cx3Cr1*$^{cre/-}$ indicated the complete Cre-mediated deletion of STAT3 occurred in microglia at the DNA level, which was employed for the subsequent SAH induction. The littermates carrying *STAT3*$^{flox/flox}$ *Cx3Cr1*$^{-/-}$ presenting two alleles of *STAT3*^{flox} however without the expression of Cre recombinase were used as the control group (Figure 2A). PCR determination of genotypes demonstrated that transgenic mice of F1 generation carrying *STAT3*$^{flox/+}$*Cx3Cr1* $^{Cre/-}$ demonstrated the heterozygous expression of one 187-bp STAT3^{flox} allele and a 146-bp wild-type STAT3 allele, as well as one allele of 380-bp *Cx3Cr1*^{cre} and a 302-bp *Cx3Cr1*$^{-}$. The genotyping assay of F2 hybrids was generally consistent with the theoretical hypothesis (Figure 2B). Total of 548 transgenic mice of F2 mice were bred. *STAT3*$^{flox/flox}$ *Cx3Cr1*$^{cre/-}$, *STAT3*$^{flox/flox}$ *Cx3Cr1*$^{-/-}$, *STAT3*$^{flox/+}$ *Cx3Cr1*$^{cre/-}$, *STAT3*$^{flox/+}$ *Cx3Cr1*$^{-/-}$ and, *Stat3*$^{+/+}$*Cx3Cr1*$^{-/-}$(wild-type) were presented in F2 generation, accounting for 20.26%, 27.01%, 25.73%, 26.64%, and 0.36% of the total number respectively. Both F1 and F2 offspring were born alive and capable of normal fertility. It was noted that 2 out of the total F2 hybrids were found with cataracts, which was excluded from the subsequent experiments. Otherwise, mostly transgenic mice grew healthy and behaved normally through the close monitor. There was no evidential preponderance of gender distribution among F2 hybrids (Supplementary Table VI).

Statistical analysis

All the data were expressed as mean ± SEM. Statistical analyses were performed by IBM SPSS 23.0 software. For the cross-sectional assessment cohort, statistical analyses are done by two-way ANOVA to assess the treatment effect across 4 time points. The independent t test was used to determine the significance of the between-group comparison. The equality of error

variance was tested as appropriate. P<0.05 after Bonferroni adjustment for multiple comparisons was considered statistically significant.

Real-time reverse transcription polymerase chain reaction

Mice were cardinally perfused with sterile saline followed by RNALater Solution at each time point. The RNA was extracted via using Trizol reagent (Invitrogen, Carlsbad, CA, USA) and PureLink™ RNA Mini Kit (Thermo Scientific. Waltham, MA, USA) according to the manufacturer's instructions. For mRNA expression, 1μg of total RNA was reverse-transcribed into cDNA by using high capacity reverse transcriptase (Applied Biosystems, Carlsbad, California, USA).

Real-time reverse transcription polymerase chain reaction (PCR) was performed to determine the activation status of the STAT3 signaling pathway, biochemical characterization of STAT3 deletion the expression of M1/M2 related microglial markers and inflammatory cytokines. The primer were designed by using AlleleID 6.0 software (AlleleID®, PREMIER Biosoft. USA). The primers sequences were listed in Supplementary Table I-II. A 10-μL total PCR reaction mixture with SYBR Green master premix Ex Taq (Takara, Japan) was utilized to perform the amplification reaction by QuantStudio 12 Flex Real-Time PCR System according to the manufacturer's instruction (ABI 7500, Thermo Fisher Scientific, Waltham, MA, USA). RT-PCR was performed with the cycling conditions as follow: 50 °C, 2 min, 95°C, 10 min, 40 cycles of 95°C, 15 s; 60°C, 1 min. Differential gene expressions were calculated using the 2-ΔΔCT method with Glyceraldehyde 3-phosphate dehydrogenase (GAPDH) as an endogenous control. The expression levels of the genes were reported as fold changes compared with the sham group.

Western blotting

Western blotting was performed to examine the activation status of STAT3 and JAK2 at the protein level. Mice were cardinally perfused with sterile saline at each time point. Brain tissue lysed and extracted by Radioimmunoprecipitation assay (RIPA) buffer (Beyotime, # P0013B) with Protease Inhibitor (Roche, # 5892970001), Phosphatase Inhibitor (Thermo Scientific, #78420) and Phenylmethylsulfonyl fluoride (PMSF) (Beyotime, # ST506). 20 μg total proteins from each sample were separated on Tris-polyacrylamide gel by electrophoresis and blotted onto nitrocellulose membranes (GE Healthcare). Membranes were blocked by 5% non-fat milk for 1 hour at room temperature. Then the membranes were incubated with primary antibodies p-STAT3 (1:1000; Cell Signaling Technology, #9145S), STAT3 (1:5000; Cell Signaling Technology, #8768S), p-JAK2 (1:1000; Cell Signaling Technology, #3771S), JAK2 (1:2000; Cell Signaling Technology, #3230S), and GAPDH (1:5000; Cell Signaling Technology, #5174S) overnight at 4°C. After the thorough wash, the membranes were incubated with the secondary antibody (1:5000; Cell Signaling Technology, #7074) for 1 hour at room temperature. The blotting was visualized by the enhanced chemiluminescence (ECL) plus detecting reagent and exposed onto x-ray films. Image J was used to quantitatively analyze the western blot results, and Grapad Prism 7.00 was used for the statistical analysis.

Genotype assay

The 2 mm tip of the tail was collected for genotyping. Genomic DNA was extracted by QuickExtract™ DNA Extraction Solution (Lucigen, #QE09050) according to the manual. PCR was employed for the genotyping. Two different pairs of primers were routinely used to detect the genotypes of every litter of transgenic mice. The primer sequences were listed in Supplementary Table III. 2 μl DNA templates were used for PCR examination. The amplification was performed in 20 μl volume system by using 2×Power Taq PCR MasterMix (BioTeke, # PR1702) at the

following conditions: 4 minutes at 94°C followed by 35 cycles of 45 seconds at 94°C (denaturation), 45 seconds at 63°C (annealing), 30 seconds at 72°C (elongation), and 5 minutes at 72°C (reading). PCR products were separated on a 1.5% agarose gel, stained with Ethidium bromide (EtBR), and photographed for analysis. $STAT3^{flox}$ was amplified in a 187-bp fragment. $STAT3^{+}$ was amplified in a 146-bp fragment. $Cx3Cr1^{cre}$ was amplified in a 380-bp fragment. $Cx3Cr1^{-}$ was amplified in a 302-bp fragment.

Neurobehavioral tests

The Mouse Motor and Sensory Scale (mMSS) was employed to evaluate the sensorimotor deficits of SAH mice before the operation and at day 1, 3, 5 and 10 after SAH induction as published previously [35]. The sensorimotor function was graded on a scale of 5-27 (27 as normal score and 5 as maximal deficits score). The scale was a composite of the motor (0-12) (spontaneous activity, the symmetry of limb movements, climbing, balance) and sensory (5-15) (proprioception, vibrissae, visual, olfactory and tactile responses) (Supplementary Table IV). In terms of the severity of the injury, the lower score indicated the more serious brain injury symptoms and vice versa.

Gait analysis was performed by computer-assisted Catwalk XT (Noldus Information Technology, Wageningen, Netherlands) test (Datto et al., 2016). The catwalk system consisted of a glass walkway with an internal light source, an inverted camera, and computer software. The light through the glass plate could be reflected when the animal's paws were in contact with it. The images of footprints were captured by the inverted camera and then converted into digital signals by the software. Mice were trained for 7 days before the experiment to ensure the mice reaching the same criteria walking unforcedly through the walkway without interruptions or

hesitation. Gait assessments were conducted at days 1, 3, 5 and 10 after SAH. In a darkened environment, the footprints along 1.3-meter-long glass plate were recorded simultaneously when mice walked through the walkway. A large amount of spatial and temporal gait parameters related to individual paws were generated and analyzed (Supplementary Table V).

Mice were then trained to learn to escape on hidden platform conditions, depending on the spatial cues. The platform was placed in the center of one quadrant of the tank and submerged 2 cm beneath the water surface. The platform remained in the same position throughout the training trials. The Intertrial interval (ITI) is 30 seconds to diminish the quadrant preference. Briefly, mice were placed facing the tank wall on a hidden condition from pseudo-randomly selected 4 quadrants Northwest (NW), Southwest (SW), Southeast (SE), and Northeast (NE). Mice were allowed to swim freely for maximal 60 seconds to find the platform, followed a 5 seconds rest on the platform as a trial. On D1 post- SAH, mice were trained for 5 days with 4 trials per day. Escape latency, moved distance and swimming velocity of each training trial were recorded and analyzed. The shorter latency indicated better spatial learning ability.

To detect the memory function, a probe test was conducted 24 h after the last training trial. The platform was removed from the tank during the probe test, and the mouse was allowed to swim freely for 60 seconds. The time mice spent in platform quadrant and platform area and the distance in platform quadrant was recorded of probe trial. After removal from the tank, mice were manually dried with paper towels and warmed under an infrared light before placed back to home cages. All tests were performed at roughly the same time every day to minimize the variability in performance due to time of day.

Immunohistochemistry

Immunohistochemistry (IHC) was performed to examine neurons and microglia. The 5 μm paraffin brain sections were used. After a xylene/ethanol dewax-rehydration series, the microwave antigen retrieval was performed in citrate buffer for 20 minutes. Endogenous peroxidize activity was quenched with 0.3 % Hydrogen peroxide (H_2O_2) after primary antibodies incubation. The brain sections were then incubated for 1 hour with blocking buffer comprising 2.5% goat serum, 1% Bovine serum albumin (BSA) and 0.1% Triton-100. The primary antibody NeuN (1:400; Millipore, clone A60, #MAB337) or Iba1 (1:200; Abcam, #ab5076) was applied subsequently at 4 °C overnight. Envision+System-Horseradish peroxidase (HRP) secondary antibody was applied for 1 hour at room temperature. Diaminobenzidine (DAB) was utilized for visualization of colorimetric reaction. Three random fields were examined on CAPS, Hippocampus (The CA1 region of the hippocampus was selected for analysis), and M1 cortex respectively of each mouse under Microscope (Leica) at 40X magnification. The define of neuronal loss was used cell counting methods, NeuroN+ cells in 3 continue filed of one location were recorded. The survived neurons were quantified by Image-Pro software.

Immunofluorescence

Immunofluorescence was utilized to define the microglial polarization after SAH. Mice were cardinally perfused with saline followed by 10% buffered formalin at each time point. The brain samples were immersed in the gradient concentration of sucrose solution from 15% to 30% for dehydration, followed by embedding in the Optimal cutting temperature (OCT) compound for cryosection. The frozen sections were immunolabeled with primary antibodies including CD 68 (1:400; Biorad, clone FA-11, #MCA1957), CD16/32 (1:200; BD Biosciences, #553141), and

CD206 (1:500; R&D, MMR, #AF2535), at 4 °C overnight. The sections were subsequently stained with fluorescence-conjugated secondary antibodies, including Donkey anti Rat Donkey DyLight 680 IgG H+L (1:200; Invitrogen, #SA5-10030), anti-Goat Alexa Fluor® 647 IgG H+L (1:200; Invitrogen, #A21447), and Donkey anti-Rat Alexa Fluor® 488 IgG H+L (1:200; Invitrogen, #A21208), at room temperature for 2 hours. After washed thoroughly with Phosphate-buffered saline (PBS) buffer, slides were then mounted with 4′,6-diamidino-2-phenylindole (DAPI) (Abcam, #ab104139). Immunofluorescent images were acquired by using confocal microscope (Zeiss, # LSM880). Quantification of M1/M2 microglial phenotype were performed on three randomly selected high power microscopic fields across three sections.

Supplemental tables

Primer sequences for the factors of STAT3 signaling pathway

Gene	Primer sequences (5' to 3')
STAT3	Forward: GAACCTCCAGGACGACTTTGA
	Reverse: GCTCACTCACAATGCTTCTCC
SOCS3	Forward: ACCAGCGCCACTTCTTCACG
	Reverse: GTGGAGCATCATACTGATCC
JAK2	Forward: GCAGCAAGCATGATGAGTC
	Reverse: CAACTGCTTAGCCACTCCA
NF-Kb	Forward: AACACTGGAAGCACGGATGA
	Reverse: CTGGCGGATGATCTCCTTCTC
Src	Forward: CGGTTACATCCCCAGCAACTA
	Reverse: TGTGGTCTCACTCTCCCTCA
GAPDH	Forward: GAGAGTGTTTCCTCGTCCCG
	Reverse: ACTGTGCCGTTGAATTTGCC

Supplementary Table I. Primer sequences for the factors of STAT3 signaling pathway.

Primer sequences for M1/M2 microglial markers and cytokines

Gene	Primer sequences (5' to 3')
M1 markers	
CD16	Forward: TTCTGCTGCTGTTTGCTTTTGC
	Reverse: GGGTTGTGGGTCCCTTCGC
CD32	Forward: GCCGTGCTAAATCTTGCTGCTG
	Reverse: TGTCAGTGTCACCGTGTCTTCC
CD86	Forward: GCAGCACGGACTTGAACAAC
	Reverse: TTGTAAATGGGCACGGCAGA
M2 markers	
CD206	Forward: GTCAGAACAGACTGCGTGGA
	Reverse: AGGGATCGCCTGTTTTCCAG
CD163	Forward: CACTGGCTCTGCTTACTTCGG
	Reverse: TTCTTTGTGGGCTTCGTTGGTC
TREM2	Forward: TGGTGGAGGTGCTGGAGGAC
	Reverse: AGGTGGGTGGGAAGGAGGTC
Pro-inflammatory cytokines	
IL-6	Forward: GCTGGAGTCACAGAAGGAGTGGC
	Reverse: GGCATAACGCACTAGGTTTGCCGA
TNF-α	Forward: GACGTGGAAGTGGCAGAAGAG
	Reverse: TGCCACAAGCAGGAATGAGA
Anti-inflammatory cytokines	
IL-4	Forward: GAGACTCTTTCGGGCTTTTCG
	Reverse: TGCTCTTTAGGCTTTCCAGGA
TGF-β	Forward: CGAGGCGAGATTTGCAGGTA
	Reverse: CGGCTGGACTGTTGTGACT
Internal control	
GAPDH	Forward: GAGAGTGTTTCCTCGTCCCG
	Reverse: ACTGTGCCGTTGAATTTGCC

Supplementary Table II. Primer sequences for M1/M2 microglial markers and cytokines.

Primer sequences for transgenic mice genotyping

Gene	Primer sequences (5' to 3')
$STAT3^{flox}$ or $STAT3^{+}$	Forward: TTG ACC TGT GCT CCT ACA AAA A
	Reverse: CCC TAG ATT AGG CCA GCA CA
$Cx3Cr1^{cre}$ or $Cx3Cr1^{-}$	Cx3cr1 forward: CCT CAG TGT GAC GGA GAC AG
	Cre forward: GAC ATT TGC CTT GCT GGA C
	Common reverse: GCA GGG AAA TCT GAT GCA AG

Supplementary Table III. Primer sequences for transgenic mice genotyping.

Mouse Motor and Sensory Scale

Function		0	1	2	3
Motor	**Activity** (5 minutes open field)	No movement	Moves, no walls approached	1-2 walls approached	3-4 walls approached
	Limb symmetry (suspended by tail)	Left forelimb, no movement	Minimal movement	Abnormal forelimb walk	Symmetrical extension
	Climbing (on inverted metal mesh)	Fails to hold	Hold < 4 seconds	Holds, no displacement	Displaces across mesh
	Balance	Falls < 2 seconds	Falls > 2 seconds	Holds, no displacement	Displaces across rod
Sensory	**Proprioception** (cotton tip to both sides of neck)		No reaction	Asymmetrical head turning	Symmetric head turning
	Vibrissae (cotton tip to vibrissae)		No reaction	Asymmetrical head turning	Symmetric head turning
	Visual (tip toward each eye)		No reaction	Unilateral blink	Bilateral blink
	Olfactory (lemon juice on tip)		No sniffing	Brief sniff	Sniff > 2 seconds
	Tactile (needle stick to palm)		No reaction	Delayed withdrawal	Immediate withdrawal

* Mouse Motor and Sensory Scale is was combined from 2 prior scales: Garcia (1995) and Crawley (1999&2000)

Supplementary Table IV. Mouse Motor and Sensory Scale (mMSS). mMSS was employed to evaluate the sensorimotor function of SAH mice. Sensorimotor function was graded on a scale of 5-27 (27 as normal score and 5 as maximal deficits score). The scale was a composite of motor (0-12) (spontaneous activity, symmetry of limb movements, climbing, balance) and sensory (5-15) (proprioception, vibrissae, visual, olfactory and tactile responses) (Du et al., 2016)

Definitions of gait parameters

Gait parameters	Definition
Static gait parameters	
Max Contact Area (cm^2)	The maximum area of the left hind paw that comes into contact with the glass plate
Max Intensity	The maximum intensity of a paw
Mean Intensity	The mean intensity of the complete paw
Max Contact Max Intensity	The maximum intensity of the paw at Max Contact
Max Contact Mean Intensity	The mean intensity of the paw at Max Contact
Static gait parameters	
Run Duration (s)	The duration in seconds of the recorded run
Stride Length (cm)	The distance between successive placements of the same paw
Stand (s)	The duration in seconds of contact of a paw with the glass plate
Swing (s)	The duration in seconds of no contact of a paw with the glass plate
Swing Speed (mm/s)	The speed of a paw during Swing phase. Swing speed = Stride length / Swing
Step Cycle (s)	The time in seconds between two consecutive initial contacts of the same paw. Step Cycle = Stand +Swing

* The mean values of each parameters during a complete trial are used for the analysis

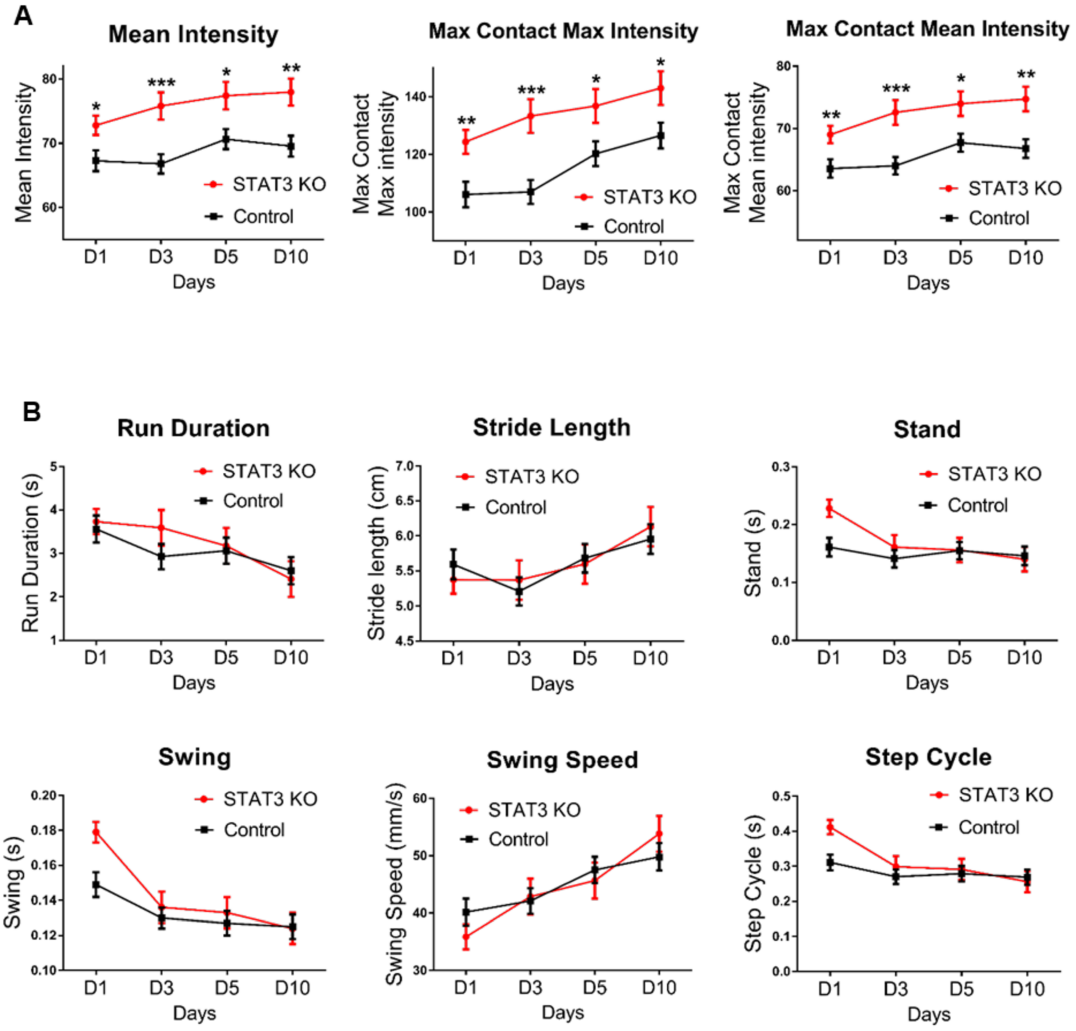
Supplementary Table V. The definition of gait parameters in Catwalk gait analysis.

Genotype analysis and sex distribution of F2 transgenic hybrids

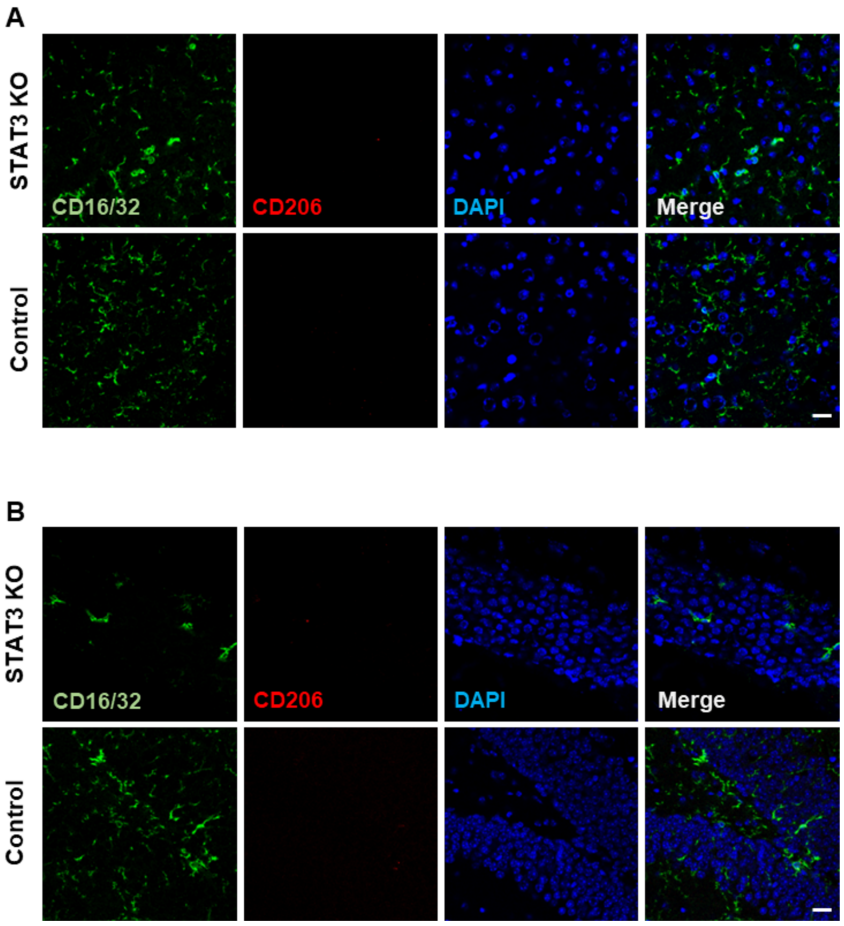
	Quantity	Percentage
Total bred mice	548	
Male	286	52.16%
Female	262	47.81%
Genotype analysis		
$STAT3^{flox/flox}$ $Cx3Cr1^{cre/-}$	111	20.26%
Male	50	45.05%
Female	61	54.95%
$STAT3^{flox/flox}$ $Cx3Cr1^{-/-}$	148	27.01%
Male	82	55.41%
Female	66	44.59%
$STAT3^{flox/+}$ $Cx3cr1^{cre/-}$	141	25.73%
Male	75	53.96%
Female	64	46.04%
$STAT3^{flox/+}$ $Cx3cr1^{-/-}$	146	26.64%
Male	78	53.06%
Female	69	46.94%
wild-type	2	0.36%
Male	0	0.00%
Female	2	100.00%

Supplementary Table VI. Genotype analysis and sex distribution of F2 transgenic hybrids crossed by $STAT3^{flox/flox}$ $Cx3Cr1^{-/-}$ and $STAT3^{flox/+}$ $Cx3cr1^{cre/-}$ mice.

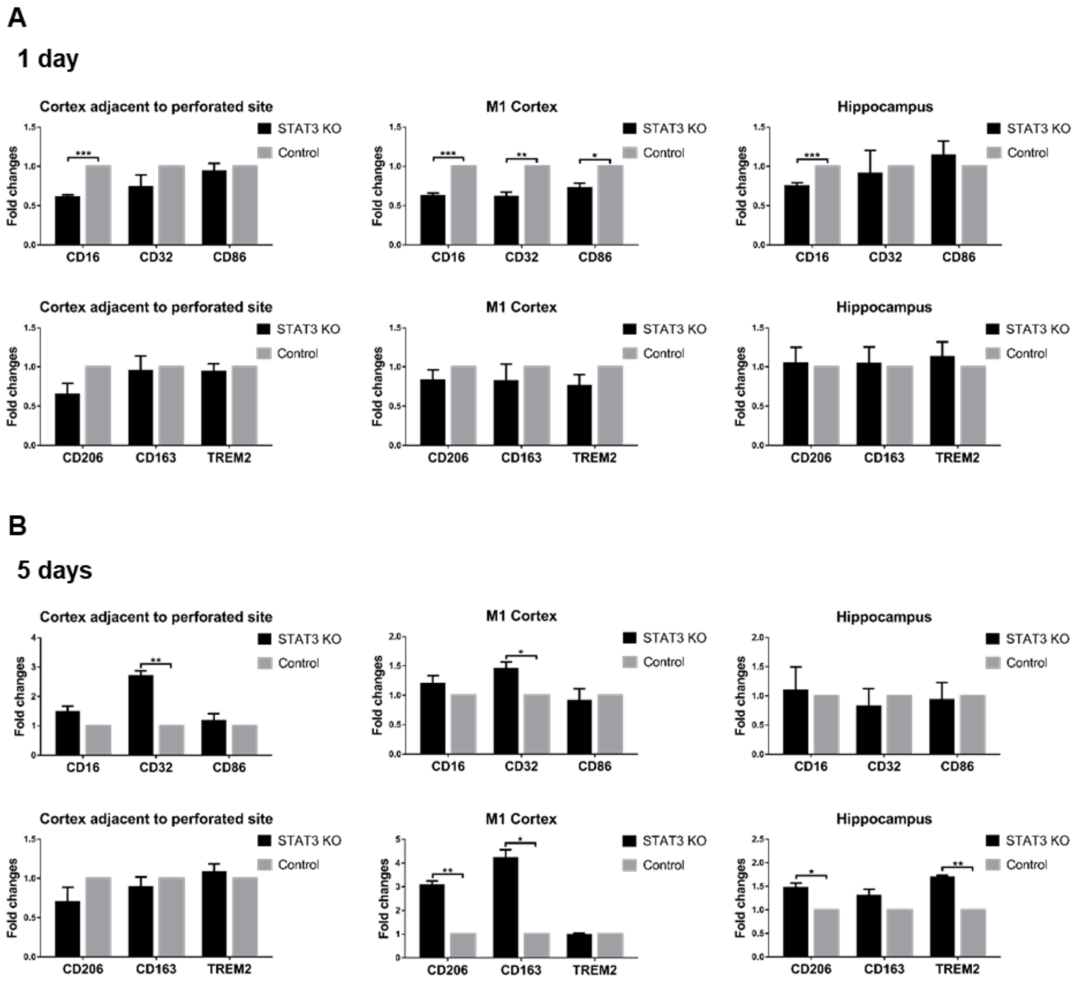
Supplementary Figures



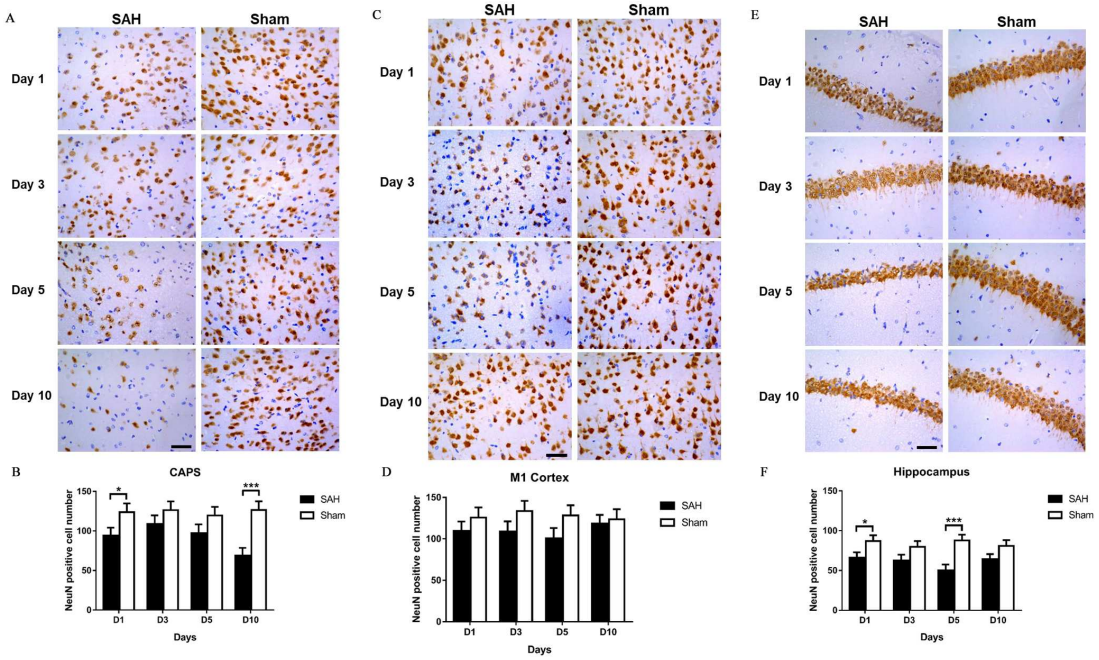
Supplementary Figure I. The time-course gait analysis of STAT3 mice after SAH. **A**, The static gait parameters included the Mean intensity, Max contact max intensity, and Max contact mean intensity of paws were assessed. **B**, The dynamic gait parameters included Run duration, Stride length, Stand, Swing, Swing speed, and Step cycle. Microglial STAT3 deletion significantly improved the static rather than dynamic gait function in SAH. n=8-16 per group. Values were the mean ± SEM. *P≤0.05, **P≤0.01, ***P≤0.001.



Supplementary Figure II. Immunofluorescent examination of M1/M2 microglial polarization in STAT3 KO mice after SAH. The representative confocal microscopic images showed the visualization of CD16/32 (M1, Green), CD206 (M2, red) and DAPI (Nuclei, blue) co-expression in M1 Cortex and hippocampus of STAT3 KO and control groups of mice at 1 day after SAH. n=5-6 per group. Bar=20μm.



Supplementary Figure III. Real time PCR analysis of microglial polarization in STAT3 KO mice after SAH. **A-B**, The M1 microglial markers including CD 16, CD32, and CD86, and the M2 microglial markers including CD 206, CD163, and TREM2 were detected at 1 and 5 days after SAH. The mRNA expression of M1/M2 microglial markers was shown in the fold change compared to the control. n=3 per group. Values were the mean ± SEM. *P≤0.05, **P≤0.01, ***P≤0.001.



Supplementary Figure IV. The NeuN IHC assessment of neuronal loss in the CAPS, M1 cortex and hippocampus after experimental SAH. **(A, C, E)-** Time course representative NeuN staining of coronal brain sections in the CAPS, M1 cortex and Hippocampus of SAH and Sham mice. Bar=50μm. **(B, D, F)-** Quantification of neuronal loss in CAPS, M1 cortex and Hippocampus of SAH and Sham mice (n=6-7/group/time point). Values were the mean ± SEM. *P⩽0.05, **P⩽0.01, ***P⩽0.001.