Online methods

Human lymphocyte isolation and culture. Naive $CD4^+CD45RA^+$ T lymphocytes from the peripheral blood of healthy donors were isolated by negative selection using MACS isolation columns (Miltenyi). 5.0 $x10^5$ lymphocytes/mL were cultured with plate-bound anti-CD3 (eBioscience; clone OKT3, 2.5 µg/ml for coating) and soluble anti-CD28 (BD Biosciences; 2 µg/ml) in X-VIVO 15 medium (Lonza) and supplemented with 2mM GlutaMAX, 100 U/mL penicillin and 100 mg/mL streptomycin (Invitrogen). For T_H differentiation, lymphocytes were cultured in the presence of recombinant human IL-12 (10 ng/mL) and mouse anti-human IL-4 (clone 3007; 5 µg/mL) for T_H1 differentiation; IL-4 (200 ng/mL), mouse anti-human IFN-γ (clone K3.53; 5 µg/mL) and mouse anti-human IL-12 (clone 24910; 5 µg/mL) for T_H2 differentiation; and recombinant human IL-1β (25 ng/mL), IL-6 (25 ng/mL), IL-23 (25 ng/mL), TGF-β1 (25 ng/mL), mouse anti-human IL-4 (clone 3007; 5 µg/mL) and mouse anti-human IFN-γ (clone K3.53; 5 µg/mL; all from R&D Systems) for T_H17 differentiation. Lymphocytes were collected directly after isolation and after 1, 3 and 6 days of culture for analysis of *IL17* and *IL26* mRNA. For analysis of IL-26 regulation, naive T lymphocytes were cultured in the presence of above-mentioned cytokines for T_H17 differentiation (alone or in all possible combinations), which were added to the cultures daily. Lymphocytes were collected after 6 days of culture for analysis of *IL26* mRNA.

For T_H lymphocyte polarization, memory $CD4^+CD45RO^+$ T lymphocytes from healthy donors or MS patients were isolated by negative selection using MACS isolation columns (Miltenyi), and were cultured as previously published (Kebir et al., 2007). Briefly, 1.0 $x10^6$ lymphocytes/mL were cultured with plate-bound anti-CD3 (eBioscience; clone OKT3, 2.5 µg/mL for coating) and soluble

anti-CD28 (BD Biosciences; 2 µg/mL) in X-VIVO 15 medium (Lonza) and supplemented with 2mM GlutaMAX, 100 U/mL penicillin and 100 mg/mL streptomycin (all from Thermofisher Scientific). Lymphocytes were cultured in the presence of recombinant human IL-12 (10 ng/mL) and mouse anti-human IL-4 (clone 3007; 5 µg/mL) for T_H1 differentiation; IL-4 (200 ng/mL), mouse anti-human IFN-γ (clone K3.53; 5 µg/mL) and mouse anti-human IL-12 (clone 24910; 5 µg/mL) for T_H2 differentiation; and IL-23 (25 ng/mL), mouse anti-human IL-4 (clone 3007; 5 µg/mL) and mouse anti-human IFN-γ (clone K3.53; 5 µg/mL) for T_H17 differentiation (all the reagents were from R&D Systems). Lymphocytes were harvested on day 6 and analyzed for intracellular cytokines by flow cytometry as previously described (Kebir et al., 2009; Larochelle et al., 2012). For M1 and M2 macrophages, and immature and mature dendritic cells (iDC and mDC, respectively), CD14 positive cells were isolated from PBMC of healthy controls by positive isolation using MACS CD14 beads and MACS magnetic columns (Miltenyi). For the macrophages, $CD14^+$ cells are plates in 1640 RPMI (Wisent) with 10% FBS supplemented with 2mM GlutaMAX, 100 U/mL penicillin and 100 mg/mL streptomycin (all from Thermofisher Scientific). Cells were treated with 25 ng/mL M-CSF (R&D Systems). After 5 days, cells were stimulated with 20 ng/mL rhIFN-γ (Thermofisher Scientific) for 1h followed by 100 ng/mL LPS (Sigma-Aldrich) to generate M1 macrophages and with 20 ng/mL rhIL-4 and 20 ng/mL rhIL-13 (both R&D systems) for M2 macrophages. Two days later, cells were harvested. For dendritic cells, $CD14^+$ cells were plated in 1640 RPMI (Wisent) with 10% FBS supplemented with 2mM GlutaMAX, 100 U/mL penicillin and 100 mg/mL streptomycin (all from Thermofisher Scientific), 10 µM HEPES (Wisent), 1 mM Sodium Pyruvate (Thermofisher Scientific), 1x non-essential amino acids (Sigma-Aldrich), 50 µM β-Mercaptoethanol (Thermofisher Scientific). Cells were treated with 50 ng/mL GM-CSF (BD Biosciences) and 10 ng/mL rhIL-4 (R&D systems). After 3

days, half of the medium was refreshed, and cells were again treated with 50 ng/mL GM-CSF and 10 ng/mL rhIL-4. At day 5, immature DC were harvested. For mature DC, half of the medium was again refreshed, and they were stimulated with 50 ng/mL GM-CSF, 10 ng/mL IL-4 and 100 ng/mL LPS. Mature DC were harvested at day 7.

Human primary brain-derived endothelial cells. Primary human brain-derived endothelial cells were isolated and cultured as previously described (Alvarez et al., 2011). Optimal barrier properties were obtained by treating BBB-EC monolayers with astrocyte-conditioned media (ACM). Human fetal astrocytes were isolated and cultured as previously described (Alvarez et al., 2011; Saikali et al., 2007). ACM was harvested from confluent astrocyte monolayers and filtered before adding it to BBB-EC cultures.

Mouse primary brain-derived endothelial cells. Primary cultures of mouse brain endothelial cells were prepared from 8-10-week-old C57BL/6 mice (Jackson Laboratory) as previously described (Podjaski et al., 2015).

Protein isolation and western blot. Human memory $CD4^+$ T lymphocytes (isolated *ex vivo*), differentiated T_H1 and T_H17 lymphocytes, and resting, inflamed (100 U/mL IFN-γ and 100 U/mL TNF-α) and IL-26 treated (100 ng/mL) BBB-ECs were washed and lysed with RIPA buffer containing protease inhibitors (both ThermoFisher Scientific). The suspension was sonicated and after centrifugation, supernatants were collected and protein concentration was determined with the Pierce BCA Protein Assay Kit (ThermoFisher Scientific). SDS-PAGE was performed as previously described (Podjaski et al., 2015). Blots were incubated overnight (4°C) with the

following primary antibodies: rabbit anti-human IL-26 (1/100; Abcam), rabbit anti-human IL-10RB (1/1,000; Abcam), rabbit anti-human IL-20RA (1/500; Abcam), mouse anti-human JAM-1 (1/300; BD Biosciences), rabbit anti-human ZO-1 (1/125; Invitrogen) and mouse anti-actin B (1/100,000; Sigma-Aldrich). HRP-labelled species-specific secondary antibodies were added for 1h at room temperature, after which ECL substrate (Amersham) was added to detect labelled protein bands.

RNA isolation, cDNA conversion and quantitative PCR. RNA was isolated from T lymphocytes, monocytes, B cells, macrophages, dendritic cells and human BBB-ECs with MinElute columns (Qiagen). To obtain cDNA, 1 µg of RNA was converted with the QuantiTect Reverse Transcription Kit (Qiagen). cDNA samples were prepared in triplicate with TaqMan FAM-labelled probes for *IL17*, *IL26, IL22*, *RORC*, *MCAM*, *Csf2* (all Life Technologies Genomic). VIC-labelled probes for *18S* (Life Technologies Genomic) were added as an endogenous reference control. Samples were amplified and measured with the QuantStudio 7 Flex Real-Time PCR System (Applied Biosystems). The comparative Ct method was used to compare levels of mRNA.

mRNA Sequencing and analysis. Human primary BBB-ECs were cultured until confluency. Cells were then stimulated with either 0, 10 or 100 ng/mL rhIL-26 or 10 ng/mL for 24h, in triplicate. Cells where lysed with Trizol™ Reagent (Invitrogen) and stored at -80℃ until RNA was isolated (as described above). RNA quantity was assessed with the NanoDrop Spectrophotometer ND-1000 (NanoDrop Technologies, Inc.) and RNA integrity was assessed with the 2100 Bioanalyzer (Agilent Technologies). The RIN value was on average 9.2 (min-max: 8.8-9.6). Libraries were generated using 250 ng RNA. mRNA was enriched using the NEBNext

Poly(A) Magnetic Isolation Module (New England BioLabs). Libraries were further prepared using the NEBNext RNA First Strand Synthesis, NEBNext Ultra Directional RNA Second Strand Synthesis Modules and the NEBNext Ultra II DNA Library Prep Kit for Illumina (all New England BioLabs). Libraries were quantified using the Quant-iT™ PicoGreen® dsDNA Assay Kit (Life Technologies) and the Kapa Illumina GA with Revised Primers-SYBR Fast Universal kit (Kapa Biosystems). Average size fragment was determined using a LabChip GX (PerkinElmer) Analyzer. Sequencing was performed using the NovaSeq6000 S2 PE100, with 50M reads generated per sample. Quality checks, library preparation and sequencing were all performed at Genome Québec (Montreal).

RNA Seq analysis. Raw sequencing files (FASTQ) were trimmed for adapter content using Trim Galore v0.6.0. The resulting reads were then processed with kallisto v0.46.0 to perform a pseudo-alignment and to quantify gene expression. The gene counts were obtained through the lengthScaledTPM method and the table was pooled using the R package tximport v1.13.10. Differential expression analysis was performed using the R package limma v3.41.6. The gene counts were first normalized with the voom method from limma and then differential expression between groups was assessed using a linear modelling approach. The limma pipeline uses a false discovery rate approach to correct the effect of multiple testing on nominal p-values. Pathway analysis was done with the fgsea R package (v1.11.0) by ranking genes according to the t-statistic of a given comparison (CTL vs IL26 [10ngmL & 100ng/mL]) to determine enrichment scores (Figure e-3). The database used was the curated hallmark gene sets, which can be obtained at http://software.broadinstitute.org/gsea/msigdb/collections.jsp.

Immunocytofluorescence. Differentiated human T_H17 lymphocytes were stimulated for 4 hours with 1 µg/mL ionomycin and 20 ng/mL phorbol 12-myristate 13-acetate (PMA) in the presence of

2 µg/mL brefeldin A (all from Sigma-Aldrich). Lymphocytes were then transferred to glass microscope slides via cytospin (in 25% (v/v) glycerol) and stained as previously described (Kebir et al., 2009) using the following primary antibodies: mouse anti-human IL-17 (1/30; R&D Systems), rat anti-human RORγt (1/10; eBioscience), rabbit anti-human IL-26 (1/20; Abcam). Donkey anti-rat Alexa Fluor (AF)488 (Thermofisher Scientific, 1/400), donkey anti-mouse AF488 (Thermofisher Scientific, 1/400) and donkey anti-rabbit RRX-conjugated secondary antibodies (Jackson ImmunoResearch, 1/400) were used. Isotype controls were used in the negative control slides. Nuclei were stained with TOPRO-3 (1:300; Thermofisher Scientific).

Mouse BBB-ECs were grown onto Ibidi µ-Slide $VI^{0.4}$ slides coated with collagen IV. When confluent, cells were either left untreated or treated with 100 U/mL IFN-γ and 100 U/mL TNF-α (Thermofisher Scientific) or 100 ng/mL rhIL-26 (R&D Systems). After 24h, cells were fixed with 70% ethanol and stained with the following primary antibodies: rat anti-mouse JAM-A (1/50; Santa Cruz) and rabbit anti-mouse claudin-5 (1/75; Thermofisher Scientific). Donkey anti-rat Cy3 (Jackson ImmunoResearch, 1/300) and donkey anti-rabbit AF488-conjugated secondary antibodies (Thermofisher Scientific, 1/400) were used. Isotype controls were used in the negative control slides. Nuclei were stained with TOPRO-3 (1:300; Thermofisher Scientific). Qualitative microscopic evaluation was performed using a Leica SP5 confocal microscope as previously described (Alvarez et al., 2011). Quantification of the fluorescent signal was analysed using ImageJ software.

Immunohistofluorescence. Human brain tissue was obtained from six patients with clinical and neuropathological MS diagnosis. Autopsy samples were preserved, and lesions selected as previously described (Podjaski et al., 2015). Control brain tissue was obtained from normal

appearing CNS tissue resected during surgery for intractable epilepsy, as described above. EAE mice (treated with rhIL-26 or control) were anaesthetized and perfused with ice-cold PBS, after which the brain and spinal cord were dissected. Samples were preserved as previously described (Podjaski et al., 2015).

All cryosections (7 µm) were treated with heparinase prior to staining for T lymphocytes and IL-26. Briefly, unfixed sections were incubated at 37°C for 1h with 1mIU heparinase I (Sigma-Aldrich) followed by fixation in ice-cold acetone. Sections were co-stained as previously described (Podjaski et al., 2015) with mouse anti-human CD4 (1/20, Thermofisher Scientific) and rabbit anti-human IL-26 (1/20, Abcam), and detected with goat anti-mouse AF488 (1/300, Thermofisher Scientific), goat-anti rabbit biotin (1/500, Dako/Agilent) and streptavidin-Cy3 (1/1500, Jackon ImmunoResearch). For the IL-26 receptor, primary antibodies were incubated and detected sequentially. First, sections were incubated with rabbit anti-human IL-20R1 (1/33, Miltenyi) followed by donkey anti-rabbit AF488 (1/400, Thermofisher Scientific) for detection. Sections were blocked with 10% rabbit serum prior to incubation with rabbit anti-human IL-10R2 biotinylated (1/8, Cloudclone) and streptavidin-Cy3 (1/1500, Jackon ImmunoResearch). Appropriate isotype controls were used, whenever these were not available primary antibodies were omitted to control for non-specific binding. Nuclei were stained with TOPRO-3 (1:300; Thermofisher Scientific). Qualitative microscopic evaluation was performed using a Leica SP5 confocal microscope and ImageJ, as previously described (Alvarez et al., 2011).

Immunohistochemistry. Human MS brain cryosections were stained with Luxol Fast blue and haematoxylin/eosin, as previously described (Alvarez et al., 2011), to evaluate the degree of leukocyte infiltration and demyelination (using Open Lab software), in order to classify the lesions.

Enzyme-linked immunosorbent assay. To quantify levels of IL-26 in serum and CSF samples of MS patients and healthy or OND controls, respectively, samples were analysed by ELISA (Cusabio), according to the manufacturer's guidelines.

Experimental autoimmune encephalomyelitis. EAE was induced in 6-8-week-old female C57BL/6 mice (Jackson Laboratory) by subcutaneous injections of 200 µg MOG_{35-55} (Alpha Diagnostic International Inc.) in a total of 100 µL emulsion of complete Freund's adjuvant (supplemented with 4 mg/mL of *Mycobacterium tuberculosis*; Thermofisher Scientific) per mouse on day 0. Pertussis toxin (400 ng/mouse, diluted in sterile HBSS; List Biological Labs, Inc.) was injected intraperitoneally (i.p.) on days 0 and 2. For rhIL-26 treatment, we injected mice i.p. with 200 ng rhIL-26 (R&D Systems) in 200 µL sterile HBSS (or 200 µL HBSS alone as control treatment) daily, starting at day 5 post-immunization and up to day 24. Mice were scored using a 5-point scoring system (0 = normal; 1 = limp tail; 2 = slow righting-reflex; 3 = paralysis of one hindlimb; 4 = paralysis of both hindlimbs; 5 = moribund) up to day 40, as previously described (Kebir et al., 2007; Larochelle et al., 2012; Podjaski et al., 2015).

Flow cytometry. Mouse immune cells were isolated from the CNS, lymph nodes and spleen of EAE mice at onset (day 7), peak (day 15) and chronic (day 27) phases of the disease, as previously published (Larochelle et al., 2012). Cells were stimulated with 20 ng/mL PMA, 1 µg/mL ionomycin and 2 µg/mL brefeldin A (all Sigma-Aldrich) for 4 hours or immediately stained. All cells were blocked using purified rat anti-mouse CD16/CD32 (BD Biosciences) before staining and a Live/Dead stain (Aqua Dead Lymphocyte Stain kit; Thermofisher Scientific) was added. Cell surface was stained with anti-mouse CD45 PerCP-Cy5.5, CD3 FITC, CD11b BV786, CD4

PE-Cy7, CD8 Pacific blue (all BD Biosciences) and/or CD25 APC (eBioscience). After surface staining, the stimulated cells were permeabilized and fixed using the Cytofix/Cytoperm kit (BD Biosciences). Cells were then stained with anti-mouse IL-17 AF700, GM-CSF PE, IFN-γ APC and IL-10 BV605 (all BD Biosciences). The cells that were not stimulated were permeabilized and fixed using a FoxP3/Transcription factor Staining Buffer set (Ebioscience). Cells were then stained with anti-mouse FoxP3 PE (Ebioscience).

All samples were acquired with the BD LSRII flow cytometer and analyzed using the FACSDiva software (BD biosciences).

Blocking assay. Confluent BBB-EC monolayers were pre-treated with or without 5 µg/mL goat anti-human IL-10R2 or 5 µg/mL mouse anti-human IL-20R1 (both R&D Systems). After 1h 100 ng/mL rhIL-26 or control buffer solution (2.8 mM NaH_2PO_4, 98 mM NaCl, 1.4% Glycerol) was added. Samples were collected for western blot after 24h.

In vitro permeability assay. Human BBB-ECs were plated on 0.5% gelatin-coated Boyden chambers (3 µm pore size; BD Falcon). When confluent, monolayers were treated with 100 ng/mL IL-26 (R&D Systems). Control wells were left untreated. After 24h, 50 µg/mL 3kDa dextran-Cascade Blue, 10kDa dextran-AF647 and 70 kDa dextran-FITC (Thermofisher Scientific) were added to the upper chambers. Both the upper and lower chambers were sampled (50 µl) after 30min, 1h, 2h, 4h and 6h. Fluorescence was quantified with a plate reader (Biotek, Synergy 4). Experimental conditions were prepared in triplicates. Permeability coefficients were calculated as previously described (Alvarez et al., 2011.

Transendothelial electrical resistance measurement. The electrical properties of human BBB-ECs were measured with the ECIS Zθ instrument and 8W10E+ electrode arrays (Applied Biophysics) as previously described (Alvarez et al., 2011). Impedance (4000Hz) was measured starting at the time of seeding up to 92h after seeding. When confluent, monolayers were treated with either 40% (v/v) ACM or 100 ng/ml IL-26 (R&D Systems). Control wells were left untreated.

Statistical analysis. Graphpad Prism software was used for statistical analyses. Results are presented as mean ± standard error of the mean (SEM). For experiments with two conditions, Student's t-test was performed, with Welch's correction where necessary. For other experiments, one-way ANOVA and Tukey's or Dunnett's post-test were used. Data were tested for outliers with the Grubbs' test. For correlation analyses, Pearson correlation coefficients were calculated. For analysis of EAE clinical scores and weights, the area under the curve was calculated for both groups and differences were tested with Student's t-test. Values were considered statistically significant when $p<0.05$.

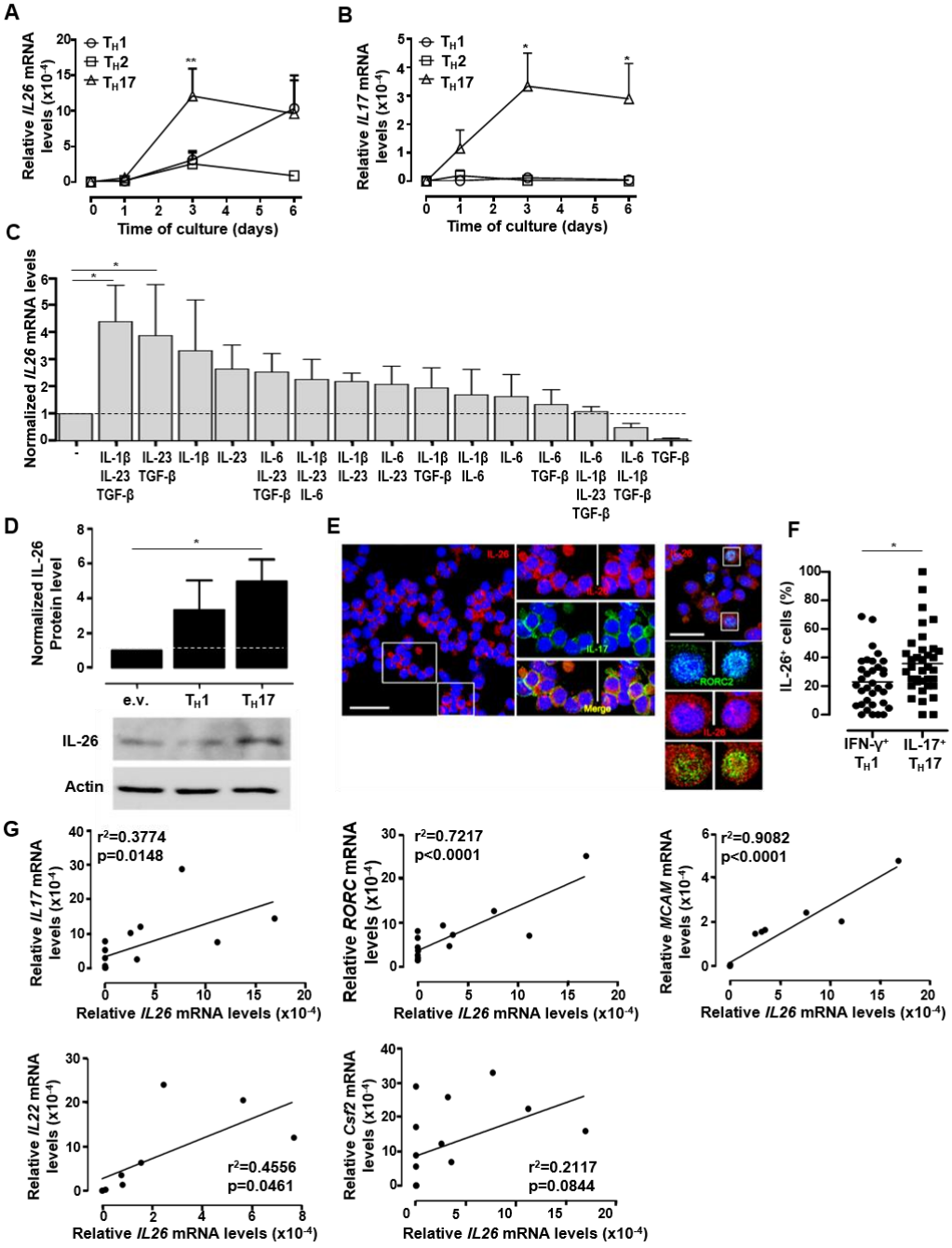

Figure e-1. *IL26* mRNA expression is primarily expressed by T_H17 lymphocytes.

(A) Expression of *IL26* and (B) *IL17* mRNA in naive $CD4^+$ T lymphocytes from 6-9 HD directly *ex vivo* (day 0) and after T_H1, T_H2 and T_H17 differentiation (after 1, 3 and 6 days of culture) by qPCR. (C) Expression of *IL26* mRNA by qPCR in naive $CD4^+$ T lymphocytes from 5 HD, stimulated daily with different cytokine combinations for 6 days. Data shown are normalized to the condition without cytokines (-). (D) Expression of IL-26 protein in *ex vivo*

(e.v.) lymphocytes and in T_H1 and T_H17 polarized lymphocytes (after 6 days in culture) from 3 HD by western blot. Protein expression, relative to beta-actin, was calculated and normalized to T_H0 (e.v.; upper panel, white dotted line). A representative blot is shown (bottom panel). (E) Immunocytochemistry of T_H17 polarized lymphocytes (left panel). Lymphocytes were stained for IL-26 (red), IL-17 or RORC (green). Nuclei were stained with TOPRO-3 (blue). Scale bars: 20µm. (F) Percentage IL-26^+cells within IL-$17^+CD4^+T_H17$ polarized lymphocytes or IFN-$\gamma^+CD4^+T_H1$ lymphocytes are quantified on at least 5 fields of view from immunohistochemistry derived images from 6 HD (right panel). (G) Relative expression of *IL26* mRNA plotted against the relative expression of other T_H17-associated genes (*IL17*, *IL22*, *CSF2*, *RORC*, *MCAM*) in *ex vivo* $CD4^+CD45RO^+$ T lymphocytes, T_H1 and T_H17 polarized lymphocytes (after 6 days in culture) from 5 HD. All mRNA levels are relative to *18S* and were assessed by qPCR. Data are presented as mean ± SEM (A-D). *$p<0.05$; ** $p<0.01$. Statistical tests: repeated measures one-way ANOVA followed by the Dunnett's multiple comparison test (A, B and D), one-way ANOVA followed by the uncorrected Fisher's LSD test (C), Student's two-tailed t-test (E), Pearson correlation (F). HD = healthy donors; SEM = standard error of the mean.

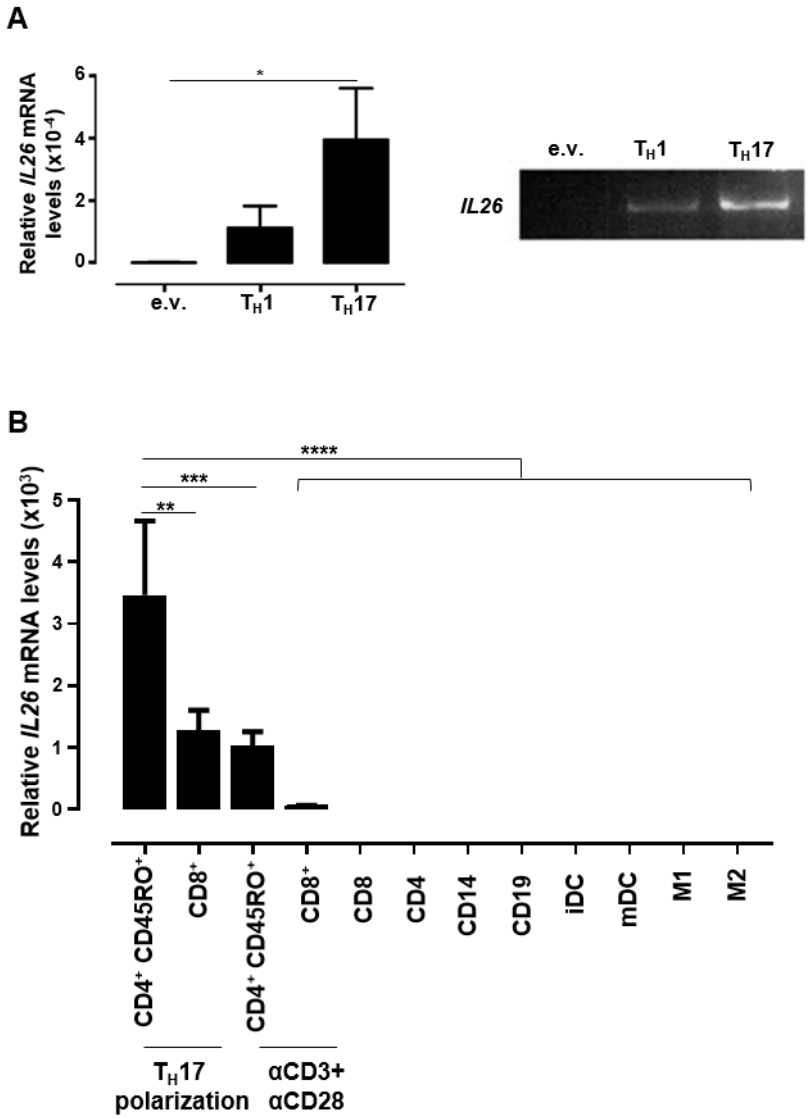

Figure e-2. *IL26* mRNA is mainly expressed by activated memory cells CD4 T lymphocytes.

(A) Expression of *IL26* mRNA in *ex vivo* (e.v.) $CD4^+CD45RO^+$ T lymphocytes, T_H1 and T_H17 polarized lymphocytes (after 6 days in culture) from 3-5 HD by qPCR (left panel) and RT-PCR (right panel). (B) Expression of *IL26* mRNA in *ex vivo* $CD4^+$ and $CD8^+$ T lymphocytes; $CD14^+$ and $CD19^+$ cells; anti-CD3/anti-CD28 stimulated $CD4^+$ $CD45RO^+$ and $CD8^+$ T lymphocytes with or without IL-23, anti-human IFN-γ and anti-human IL-4 (T_H17 polarization); M1 and M2 differentiated macrophages; and immature and mature dendritic cells (iDC and mDC, respectively) from 3 HD. All mRNA levels are relative to *18S* and were assessed by qPCR. Data are presented as mean ± SEM. ** $p<0.01$, *** $p<0.001$, **** $p<0.0001$. Statistical tests:

repeated measures one-way ANOVA followed by the Dunnett's multiple comparison test. HD = healthy donors; SEM = standard error of the mean.

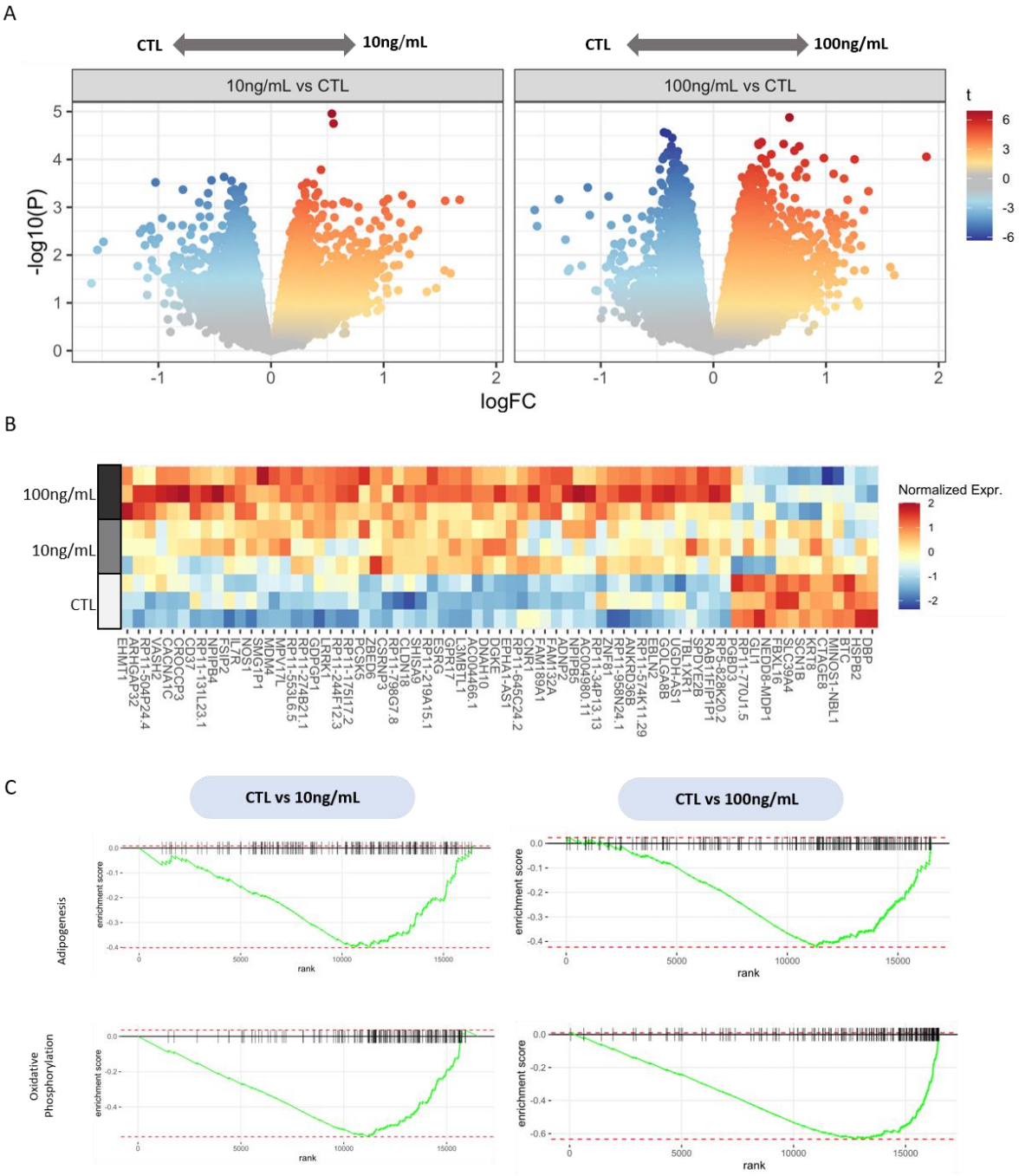

Figure e-3. Differentially expressed genes by IL-26 treated BBB-ECs.

(A) Volcano plot showing the global results of the differential expression analysis. The 2 conditions are separated, showing a more significant effect in 100ng/mL treatment. Points are colored with the t-statistic, which was used for the ranking of genes in the following GSEA. Positive fold-changes are associated with increased expression in the IL-26 condition and

negative fold-changes in associated in higher expression in the control condition. (B) Heatmap of the top DEGs (66; FDR<0.1 & |logFC|>0.5). Genes and samples are ordered with a hierarchical clustering, which cluster triplicates together. The values plotted are the scaled voomed expression of the DEGs. (C) Enrichment plot showing the ranking for 2 examples mentioned in the text. The green curves represent the random-walk approach that allows to assign an enrichment score which is then normalized (NES). Genes associated with these terms tend to be at the bottom of the list and are therefore associated with the negative t-statistics. CTL = control.

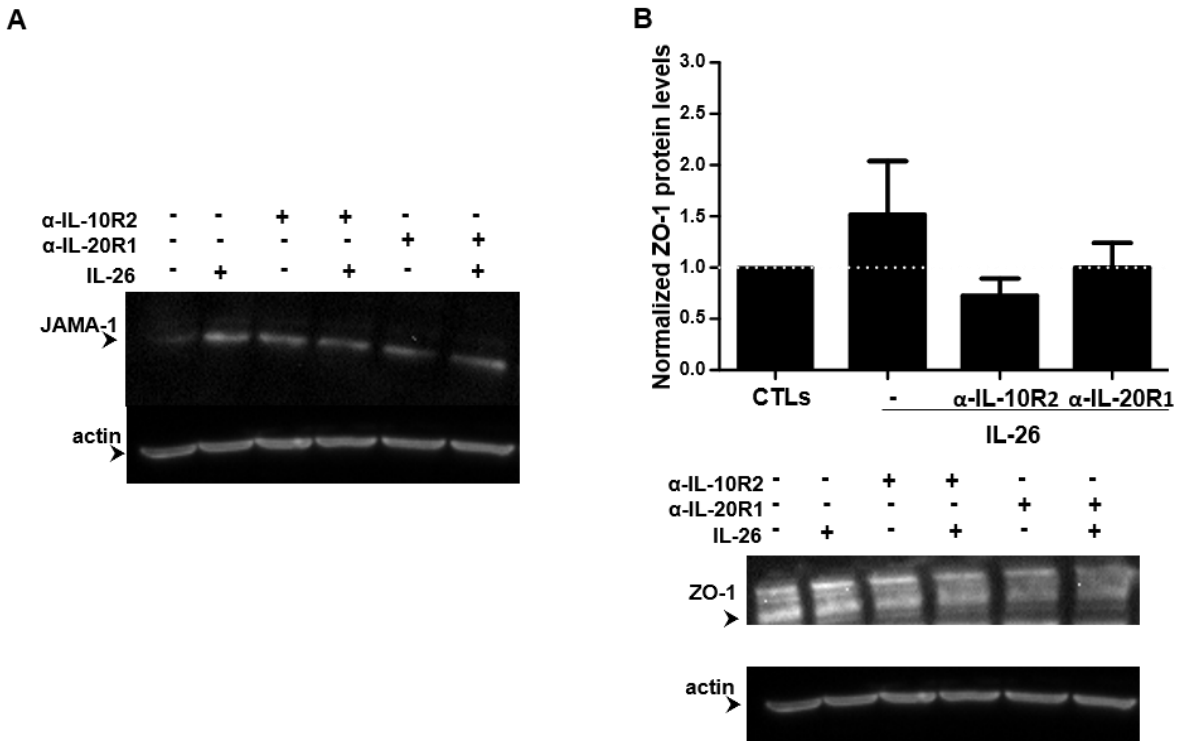

Figure e-4. IL-26 does not affect ZO-1 protein expression.

(A) JAM-1 and (B) ZO-1 protein expression on BBB-ECs pre-treated with or without 5 µg/mL anti-IL-10R2 or 5 µg/mL anti-IL-20R1. After 1h, 100 ng/mL rhIL-26 or control was added for 24h. Samples were analysed by western blot (n=3). (A) A representative blot is shown. (B) Protein expression, relative to beta-actin, was calculated and normalized to the respective controls (upper panels, white dotted line). A representative blot is shown (bottom panels). Data are represented as mean ± SEM (B). Statistical tests: a repeated measures one-way ANOVA followed by the Dunnet's multiple comparison test. BBB-ECs = blood brain barrier endothelial cells; SEM = standard error of the mean.

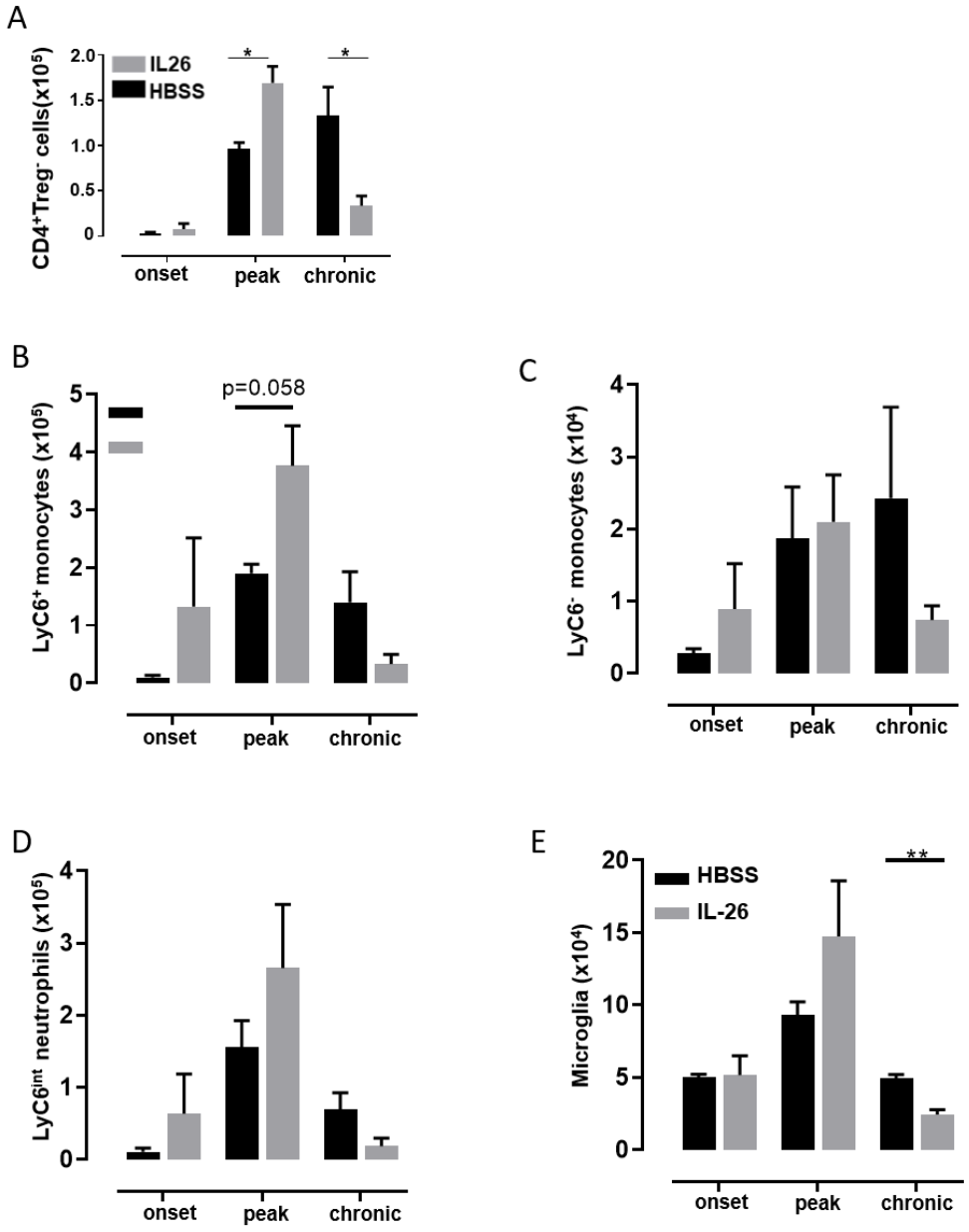

Figure e-5. Myeloid cells, microglia and non-regulatory T cells in EAE.

MOG_{35-55} immunized C57BL/6 mice injected i.p. with either 200 µl HBSS or 200 µl containing 200 ng rhIL-26 in HBSS. IL-26 treatment was started at day 5 after disease induction and was given daily until day 24. (A-E) CNS of perfused mice was collected and immune cells were isolated, counted and analyzed by flow cytometry at onset (day 7), peak (day 15) and chronic (day 27) phases of disease. Bars represents total number of cells. Data are represented as mean ± SEM. *$p<0.05$. Statistical analyses performed: Student's two-tailed t-test. n=3 animals per

group. CNS = central nervous system; EAE = experimental autoimmune encephalomyelitis; HBSS = Hank's balanced salt solution; MOG = myelin oligodendrocyte glycoprotein.

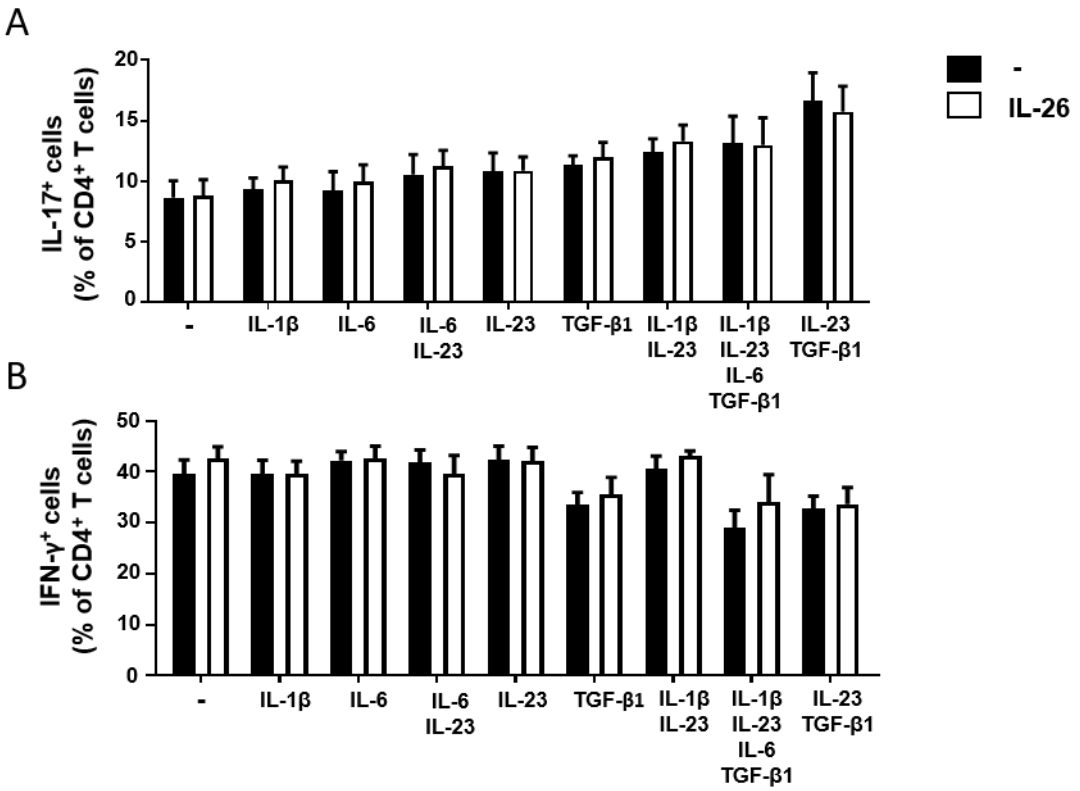

Figure e-6. IL-26 does not affect IL-17 and IFN-γ expressing TH lymphocytes.

$CD4^+$ memory cells were stimulated with αCD3/αCD28 in the presence of different cytokine combination (IL-6, IL-23, IL-1β and/or TGF-β1) with or without IL-26. (A) IL-17 positive cell percentages and (B) IFN-γ positive cell percentages.