

ONLINE METHODS

Drugs. 2-AG was from Cayman Chemical. [^3H]-2-AG (radiolabeled on the glycerol moiety) was from American Radiolabeled Chemicals and the National Institute on Drug Abuse drug supply system. WWL70 and JZL184 were synthesized in the laboratory of B. Cravatt. SR144528 was a gift from Sanofi Research.

Cell culture. BV-2 and COS-7 cells were expanded in DMEM (HyClone Cat. # SH30243.01) supplemented with HEPES (10 mM), NaHCO_3 (10 mM), penicillin (100 U ml^{-1}), streptomycin (100 $\mu\text{g ml}^{-1}$) and FBS (10%, HyClone Cat. # SH30071.03). Mouse neurons and microglia in primary culture were prepared as described^{23,37} and according to the guidelines of the Institutional Animal Care and Use Committee of the University of Washington. Briefly, for neurons, one day-old mouse brains (C57BL/6) were collected, and their meninges and cerebellum removed. The remaining brain tissue was chopped and the cells dissociated and plated in 10-cm dishes (BD Falcon) coated with poly-D-lysine (0.1 mg ml^{-1}) at 5.0×10^5 cells per ml of Neurobasal Medium (Gibco Cat. # 21103-049) supplemented with B-27 (2%), Glutamax (1%), penicillin (100 U ml^{-1}), and streptomycin (100 $\mu\text{g ml}^{-1}$). Neurons were tested after 7–8 d in culture. For microglia, dissociated cells resulting from two brains were added to DMEM (10 ml) supplemented with NaHCO_3 (10 mM), penicillin (100 U ml^{-1}), streptomycin (100 $\mu\text{g ml}^{-1}$) and FBS (10%), and plated in 75-ml flasks coated with poly-ornithine (0.001%). The resulting floating microglia were plated in uncoated 10-cm dishes at 5.0×10^4 cells per ml of MEM (Gibco Cat. # 51200-038) supplemented with CellGro (10%, Cat. # 40-101-CV). Microglia were tested after 24–48 h in culture.

ABPP-MudPIT analysis of BV-2 cytosolic and mitochondrial proteomes. BV-2 cells in culture were detached, rinsed by centrifugation, resuspended in Tris buffer (300 μl , 50 mM, pH 7.5) and lysed by sonication. Cytosolic and mitochondrial fractions were prepared as described¹⁸, and shipped to the Cravatt laboratory for ABPP-MudPIT analysis. Specifically, BV-2 cytosolic and mitochondrial proteomes (1 mg in 1 ml Tris buffer) were incubated with FP-biotin (5 μM , 1 h, room temperature). Enrichment of the FP-labeled proteome was performed as described¹⁹, except that the Lys-C digestion step was omitted. MudPIT analysis was performed as described on an LTQ ion trap mass spectrometer (ThermoFisher) coupled to an Agilent 1100 series HPLC¹⁹. The tandem MS data were searched against the mouse IPI database with the SEQUEST algorithm, and results were filtered and grouped with DATASELECT³⁸. Peptides with cross-correlation scores greater than 1.8 (+1), 2.5 (+2), 3.5 (+3) and δCN scores >0.08 were included in subsequent spectral counting analysis. The average spectral counts of two cytosolic and three mitochondrial samples are reported for serine hydrolases with average spectral counts >15 and greater than tenfold enrichment in mitochondrial versus cytosolic samples.

qPCR. RNA was extracted using PerfectPure RNA Cultured Cell Kit (5 Prime). Reaction mixtures were prepared using Brilliant II 1-Step QRT-PCR Master Mix (Stratagene). Primer and probe combinations were designed using the Roche Universal Probe Library Assay Design Center (<http://www.universalprobelibrary.com>). Primers were from Operon and universal probes were from Roche. Reactions were run using a Stratagene Mx3000P QPCR system and consisted of 30 min incubation at 45 °C, followed by a 10-min denaturation at 95 °C and 40 cycles of 1 min at 95 °C and 30 s at 60 °C.

shRNA. Plasmids expressing shRNAs were purchased from the Sigma MISSION shRNA library or constructed in our laboratory (targeting sequences were inserted into pLKO.1 (Addgene plasmid 10878), according to the manufacturer's instructions³⁹). Each set of plasmid carrying a specific shRNA construct was transfected into AD293 cells using Lipofectamine 2000 (Invitrogen, according to the manufacturer's instructions). After 72 h, the supernatant of AD293 containing the lentiviruses was applied directly to BV-2 cells. After 16 h, the medium of BV-2 cells was replaced by DMEM supplemented with FBS (10%). After 24 h, the medium of BV-2 cells was replaced by DMEM supplemented with FBS (10%) and puromycin (2 $\mu\text{g ml}^{-1}$), and cells were kept for 5–9 d in culture for selection. qPCR was systematically performed on each batch to assess for % knockdown. Selected clones were then split and expanded for specific experiments.

Cell migration. Cell migration was performed and calculated as described²⁴. Briefly, BV-2 cells were incubated for 30 min in MEM containing the nuclear

fluorescent dye DRAQ5. Cells were then resuspended in MEM supplemented with Cellgro (10%) and loaded in upper chamber of a 96-well chemotaxis apparatus (Neuroprobe, 7×10^4 cells per 390 μl , filters with 10- μm pores). Lower wells contained the same medium supplemented with 2-AG. Migration occurred over 3 h (37 °C, 5% CO_2), after which the filter was removed and the cells that had not migrated were thoroughly wiped off the top side of the filter. The fluorescence emitted by the cells that had migrated toward the bottom surface of the filter was measured using an Odyssey Infrared Imaging System (LI-COR).

Transfections. COS-7 cells (~90% confluent in 10-cm dishes) were transfected with expression vectors (OpenBioSystems, 3 μg per dish) by using Lipofectamine 2000. After 4–6 h, the medium was changed and the cells were incubated for an additional 30–36 h in DMEM + FBS (10%). For ICC, transfections were performed similarly, but using cells grown on 12-mm coverslips placed in 24-well plates (~60% confluent). Here, GFP-expressing vectors (40 ng) were also added to identify the cells that had been transfected.

[^3H]-2-AG hydrolysis in cell homogenates and intact cells. Cell homogenates were prepared as described¹⁸. Homogenates were added to silanized glass tubes that contained 100 μl of Tris-HCl buffer (100 mM, pH 7.4) supplemented with fatty acid-free BSA (0.1%), [^3H]-2-AG (~1 nM) and inhibitors or vehicle (DMSO, 0.1%). Tubes containing this solution, but without cell homogenate, were used as control for non-enzymatic [^3H]-2-AG hydrolysis (blank) and this value was systematically subtracted from values obtained with homogenates. Tubes were incubated for 10 min in a shaking water bath at 37 °C. Reactions were stopped by adding ice-cold MeOH- CHCl_3 (1:1, 2 ml) and vortexing. Linear enzymatic activity with a set amount of protein was systematically verified and chosen for each homogenate. The hydrophobic and hydrophilic phases were separated by centrifugation (800g, 10 min). One milliliter of upper phase was recovered and mixed with Ecocint (4 ml) for radioactivity determination by liquid scintillation. For intact cells, the following '[^3H]-2-AG solution' was prepared in a silanized glass vial and allowed to equilibrate at room temperature for 75 min: MEM supplemented with BSA (0.15%) and [^3H]-2-AG (~1 nM). Cells grown in 12-well plates were pre-treated with inhibitors or vehicle for 30 min by adding 0.1 ml to each well. Cells were then rinsed twice with MEM supplemented with fatty acid-free BSA (0.15%) and incubated with [^3H]-2-AG solution for 20 min (with gentle shaking in a water bath at 37 °C). Non-specific hydrolysis was determined by adding 2-AG (100 μM), and this value was systematically subtracted from each data point. The entire medium (~1 ml) was recovered in a silanized glass tube, ice-cold MeOH (2 \times 1 ml) was added to the cells, and the resulting lysate pooled with the media. CHCl_3 was added such that there was a final ratio of 1:2:2 for MEM:MeOH: CHCl_3 . The amount of [^3H]-glycerol present in the hydrophilic phase was quantified by liquid scintillation as described above.

ICC and IHC. The following primary and secondary antibodies were used: affinity-purified ABHD6 (rabbit polyclonal, 1:2000 (IHC), 1:10,000 (ICC)), CB1 (guinea pig polyclonal, raised against the full carboxyl terminus of rat CB1, 1:2,000 (ICC and IHC)⁴⁰), MAP2 (mouse monoclonal, 1:500, Chemicon), SMI32 (mouse monoclonal, 1:1,000, AbCAM), GFAP (mouse monoclonal, 1:400, Millipore), GFP (mouse monoclonal, 1:400, Invitrogen), goat anti-rabbit conjugated with Texas Red (1:250, Invitrogen) or conjugated with Alexa555 for triple staining experiments (1:500, Invitrogen), goat anti-guinea pig conjugated with Alexa488 (1:500, Invitrogen), goat anti-mouse conjugated with Alexa647 (1:500, Invitrogen). Antibodies were diluted in PBS supplemented with donkey serum (2.5%) and Triton X-100 (0.5%) with or without 5 $\mu\text{g ml}^{-1}$ of the immunizing peptide and shaken at 4 °C for 18 h. For ICC, cells were fixed with PFA (4% in PBS) for 20 min (warming from 4 °C to room temperature), permeabilized with PBS supplemented with goat serum (5%) and saponin (0.1%) at room temperature for 30 min, incubated for 18 h at 4 °C in PBS supplemented with goat serum (2.5%), and incubated in PBS supplemented with Fc block (0.5 μg per cover slip; BD Bioscience) for 5 min at room temperature. Cells were then incubated with primary antibodies for 90 min at room temperature, washed 5 \times with PBS at room temperature and incubated for 1 h at room temperature with secondary antibodies. Cells were then rinsed 7 \times in PBS and once with de-ionized water. Cover slips were mounted with Vectashield and sealed with nail polish. For IHC, mice (WT C57BL/6, GAD67-GFP C57BL/6 or Iba1-GFP C57BL/6; 8 wks old) were perfused and fixed with PFA (4% in PBS), and whole brains cryoprotected in 15%

sucrose (24 h), followed by 30% sucrose (48 h). Coronal sections that included the prefrontal cortex (30 μ m) were prepared using a microtome and stored in PBS at 4 °C. Sections were rinsed 3 \times with PBS and incubated for 90 min at room temperature with PBS supplemented with donkey serum (5%) and Triton X-100 (1%). Primary antibodies, which had been incubated for 16–18 h with or without 5 μ g ml⁻¹ of the inoculation peptide in PBS supplemented with donkey serum (2.5%) and Triton X-100 (0.5%), were applied to each section for 16–18 h at 4 °C with gentle agitation. Sections were then rinsed 6 \times with PBS supplemented with Tween-20 (0.05%, at room temperature). Sections were then incubated with secondary antibodies diluted in PBS with supplemented with donkey serum (2.5%) and Triton X-100 (0.5%) for 1 h at room temperature with gentle agitation, followed by seven rinses with PBS and one rinse with deionized water. Sections were mounted onto charged slides and allowed to dry for ~18 h, after which coverslips were mounted with Vectashield and sealed with nail polish. All fluorescent images were collected on a Zeiss Axio Observer Z1 equipped with a Pan-Apochromatic 20 \times /0.8DicII (single plane) or Pan-Apochromatic 63 \times /1.4 oil lens (Z-stack images at 0.26 μ m that were deconvolved using an Apotome). The same exposure settings were used for the experimental, immunizing peptide, and secondary only controls. Images were processed in Photoshop by gating the background to the secondary-only control. The intensity was gated to the experimental image and was the same setting used for the immunizing peptide control.

Electron microscopy. Two C57BL/6 mice were perfused by one of two methods (similar results were obtained independent of the perfusion method). Both mice were perfused through the heart by first washing with saline followed by either (i) 4% paraformaldehyde with 0.1% glutaraldehyde (TAAB) in acetate buffer (pH 6.0; 5 min) followed by 4% paraformaldehyde with 0.1% glutaraldehyde in borate buffer (pH 8.5; 50 min)⁴¹; or (ii) 4% paraformaldehyde with 0.1% glutaraldehyde for 30 min. Immediately after perfusion, the brain of each mouse was dissected and coronal sections (50 μ m) were cut using a vibratome. All incubations and washes were carried out on a horizontal shaker. The sections were washed in phosphate buffer (PB; 5 \times ; 10 min) and incubated in 30% sucrose in PB overnight at 4 °C. Slices were freeze-thawed over liquid nitrogen three times to ensure penetration and then washed in PB (3 \times ; 10 min) before being treated with 0.5% sodium borohydride in PB (15 min). After washes in PB (3 \times ; 10 min) the slices were transferred to 0.05 M Tris-buffered saline (TBS; pH 7.4) before being blocked for 40 min in BSA (3%) in TBS at room temperature. ABHD6 polyclonal antibody (1:1,500, rabbit) was diluted in TBS and incubated with the slices overnight at room temperature. After 18 h, the slices were washed with TBS (4 \times ; 10 min) before being incubated with ultra small gold-conjugated rabbit secondary (1:50; Aurion) diluted in TBS with 0.8% BSA, 0.1% gelatin and 0.05% sodium azide overnight at 4 °C. After washes in the same solution (1 \times ; 30 min) and TBS (3 \times ; 10 min) the slices were postfixed with 2% glutaraldehyde and silver intensified with Aurion R-Gent intensification kit for 15 min.

After intensification, sections were dehydrated by treating with OsO₄ (1% for 1 min then 0.5% for 20 min on ice water), then dehydrated in ethanol and propylene oxide and finally embedded in Durcupan (Fluka). During the 70% ethanol step, the sections were treated with 1% uranyl acetate in 70% ethanol for 40 min. Selected blocks containing different cortical areas within layer 4–5 were selected to be cut into 60-nm ultrathin sections with an Ultramicrotome (Reichert) and mounted on copper grids. Contrast staining was then done with lead citrate for 3 min.

We collected 60,000 \times magnified images using a camera (Morada; Olympus) mounted to an electron microscope (JEM 1200 EXII; Jeol). Specifically, synaptic contacts (which had both an obvious postsynaptic density within a reasonably small postsynaptic site and vesicle accumulation in the presynaptic site, as well as

a synaptic gap between the two sites) containing at least 1 gold particle on either the pre- or postsynaptic side were collected for analysis. Images were analyzed using ImageJ software (NIH) by manually counting gold particles within both pre- and postsynaptic elements of previously specified synapses, and the area of each element was also measured. The number of gold particles was normalized to the area of each element for comparison.

eCB levels. Cells grown in 10-cm dishes (one dish per condition) were pretreated for 30 min with inhibitors or vehicle (DMSO, 0.1%) by adding 1 ml directly to the medium (37 °C, shaking water bath). To stimulate the neurons, glutamate (100 μ M) and carbachol (1 mM) were added in 1 ml for an additional 2.5 min²⁷. The reaction was stopped by collecting the medium and adding 5 ml of ice-cold methanol. 2-AG and anandamide were extracted, purified and their levels determined as described⁴². Briefly, the cell medium and homogenate were added to CHCl₃ containing d₅-2-AG (150 pmol) and d₄-anandamide (50 pmol) for Folch extraction. The organic phase was recovered and dried under N₂, and anandamide and 2-AG partially purified by solid-phase chromatography columns (silica), eluting them with ethylacetate/acetone (1:1, 2 ml). The eluate was then dried under N₂, derivitized with BSTFA (SupelCo) and analyzed by CI-GC-MS (Varian CP-8400 Autosampler, CP3800 Varian GC, Varian Saturn 2000 mass spectrometer).

Electrophysiology. Electrophysiology experiments were performed in Bordeaux, France according to the criteria of the European Communities Council Directive (86/609/EEC) and the United States National Institutes of Health *Guide for the Care and Use of Laboratory Animals*. Mouse brain slices (C57BL/6, ~8 wks old, 300 μ m) were prepared as described³². In brief, immediately after cutting, slices containing the prefrontal cortex were stored for ~30 min at 32–35 °C in artificial cerebrospinal fluid (ACSF) containing (in mM): NaCl (126), KCl (2.5), MgCl₂ (2.4), CaCl₂ (1.2), NaHCO₃ (18), NaH₂PO₄ (1.2) and glucose (11), and equilibrated with 95% O₂/5% CO₂. Slices were then placed in the recording chamber at room temperature and superfused with ACSF (2 ml min⁻¹, 32–35 °C). The superfusion medium contained picrotoxin (100 μ M) to block GABA_A receptors. Inhibitors were added at the final concentration to the superfusion medium. To evoke synaptic currents, stimuli (100–150 μ s duration) were delivered at 0.1 Hz through a glass electrode filled with ACSF placed in layer 2/3. The recording pipette was placed in layer 5/6 and was also filled with ACSF. Both the field excitatory postsynaptic potential (fEPSP) area and amplitude were measured (graphs depict area).

Data analysis. Values are expressed as mean \pm s.e.m. Statistical analysis and dose-response curves were generated using GraphPad PRISM (version 4).

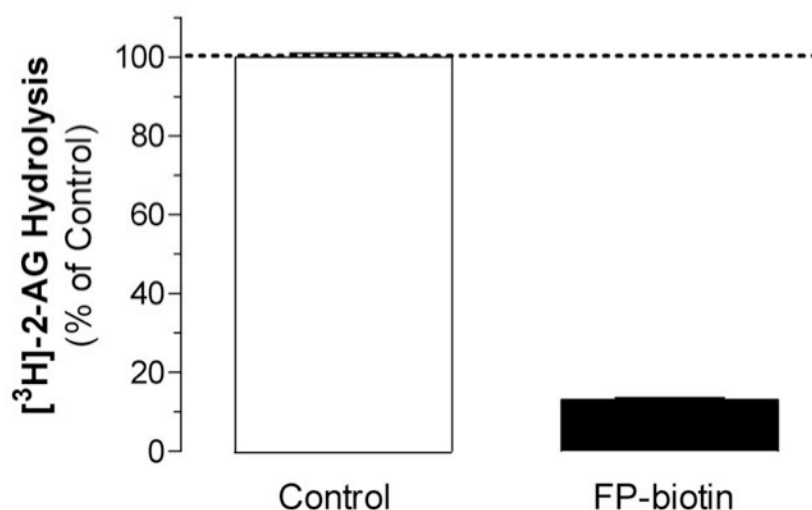
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ABHD6 controls 2-AG accumulation and efficacy at cannabinoid receptors

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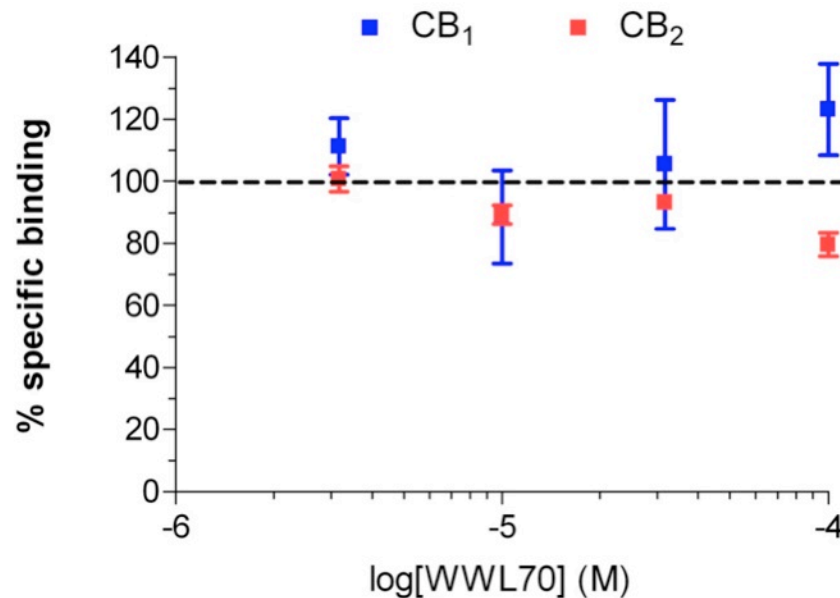
Supplementary Figure 1: The ABPP probe (FP-biotin) inhibits ~90% of the 2-AG hydrolysis in the mitochondrial fraction of BV-2 cells.

Mitochondrial fractions of BV-2 cells were pre-incubated with or without the FP-biotin probe, and [^3H]-2-AG hydrolysis was quantified by measuring [^3H]-glycerol production after a 10 min reaction at 37°C. The FP-biotin probe inhibited ~90% of the total [^3H]-2-AG hydrolysis in this subcellular fraction of BV-2 cells (n=6).



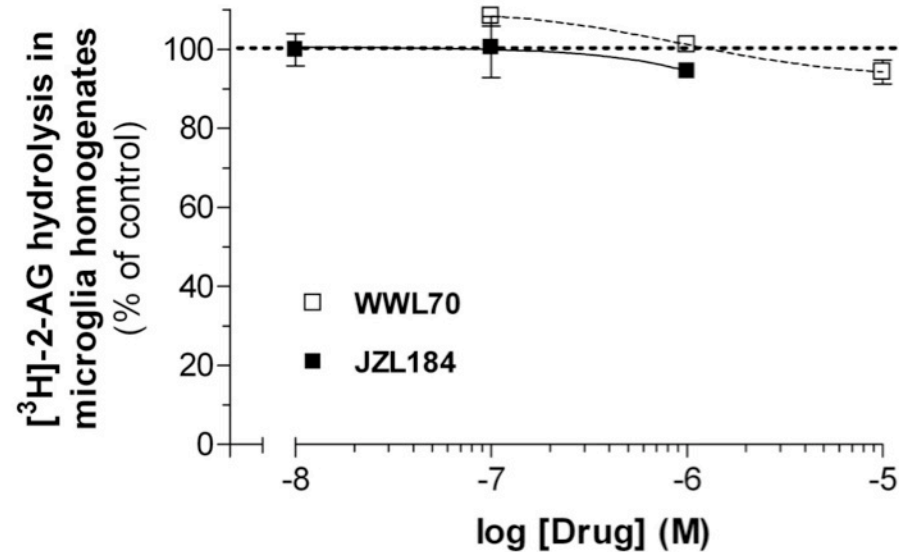
Supplementary Figure 2: WWL70 does not displace the specific binding of CP55940 to CB₁ or CB₂ receptors.

Increasing concentrations of WWL70 were tested on membrane homogenates prepared from cells expressing either mouse CB₁ or mouse CB₂ to determine whether this compound displaces the specific binding of the CB₁/CB₂ agonist [³H]-CP55940 (~1 nM). Data points represent the mean of two independent experiments performed in triplicate.



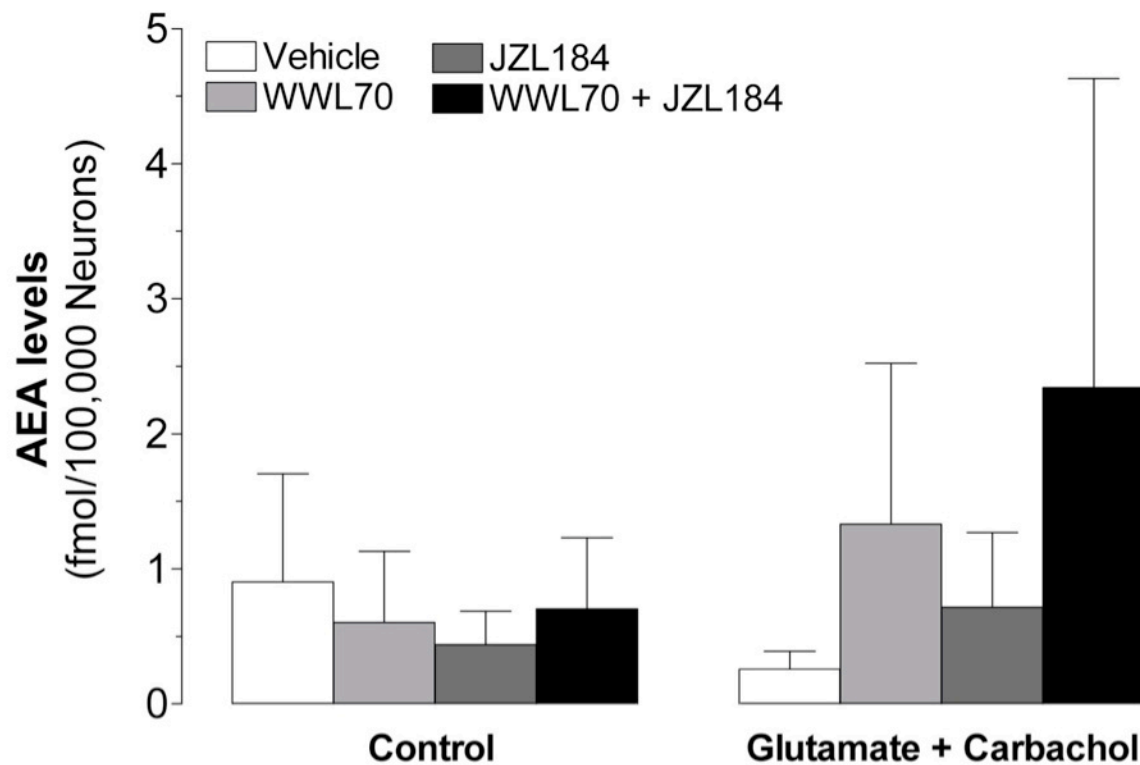
Supplementary Figure 3: 2-AG hydrolysis in microglia does not involve ABHD6 or MAGL.

2-AG hydrolysis in homogenates prepared from microglia in primary culture is not sensitive to the ABHD6 inhibitor WWL70 or the MAGL inhibitor JZL184. [3 H]-2-AG hydrolysis was quantified by measuring [3 H]-glycerol production after a 10 min incubation at 37°C (N = 3, *i.e.* three independent experiments performed in triplicate).



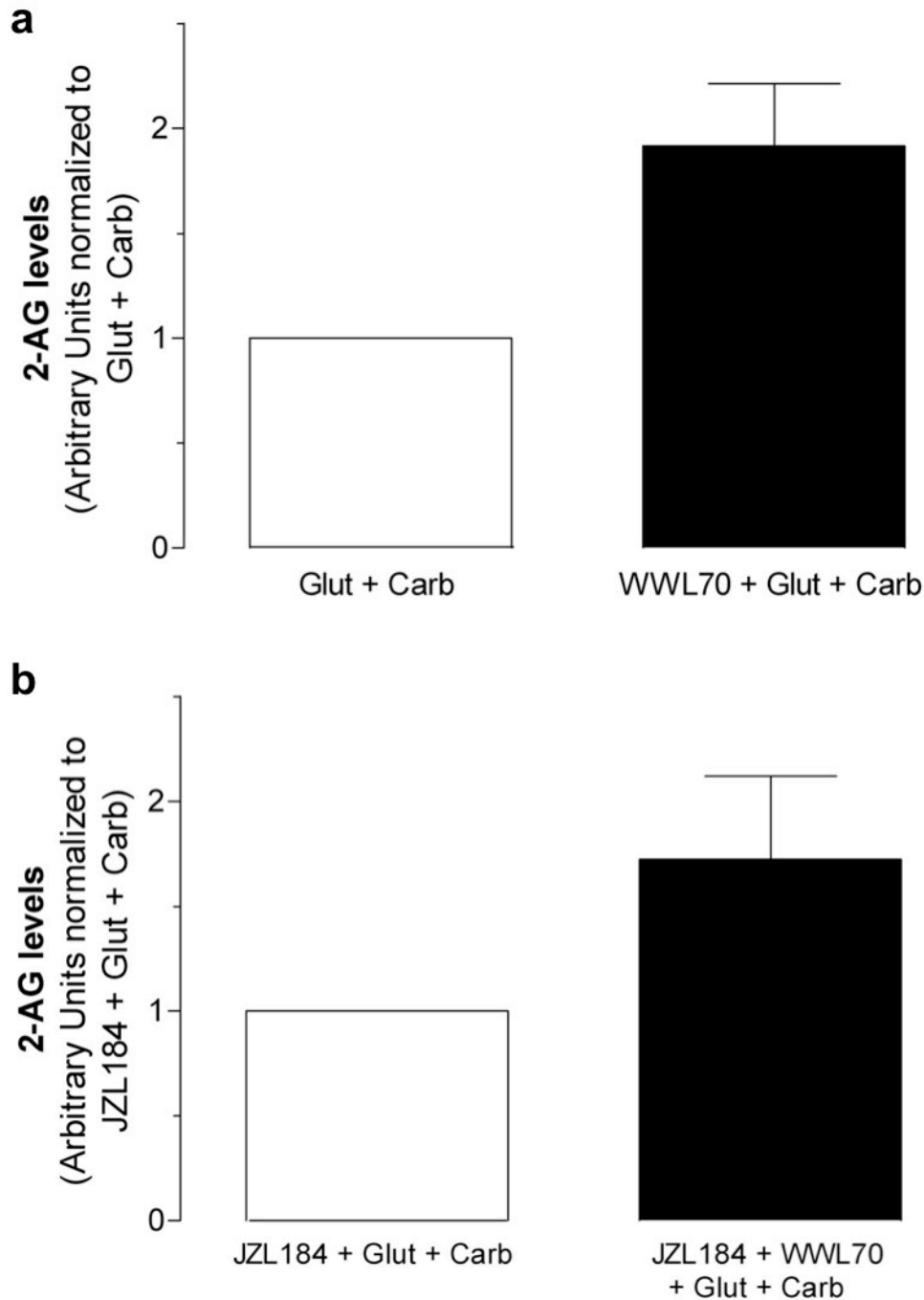
Supplementary Figure 4: Inhibition of ABHD6 and MAGL in intact neurons does not significantly affect AEA levels.

Intact neurons in culture were preincubated with WWL70 (10 μ M) and/or JZL184 (1 μ M) or vehicle (0.1% DMSO) for 30 min, and then stimulated with or without glutamate (100 μ M) + carbachol (1 mM) for 2.5 min. Lipids were then extracted and AEA levels quantified by GC-MS. Data represents mean \pm s.e.m. for three independent experiments, each performed in duplicate.



Supplementary Figure 5: ABHD6 inhibition increases the stimulated accumulation of 2-AG proportionally in both the presence and absence of MAGL inhibition.

Primary neurons in culture were pretreated with WWL70 (10 μ M), JZL184 (1 μ M), or both for 30 min, and then stimulated with a combination of glutamate (100 μ M) and carbachol (1 mM). 2-AG levels were then quantified by GC-MS. Note that WWL70 treatment increased 2-AG accumulation by \sim 2 fold in both the absence (a; n=4) and presence (b; n=4) of JZL184.



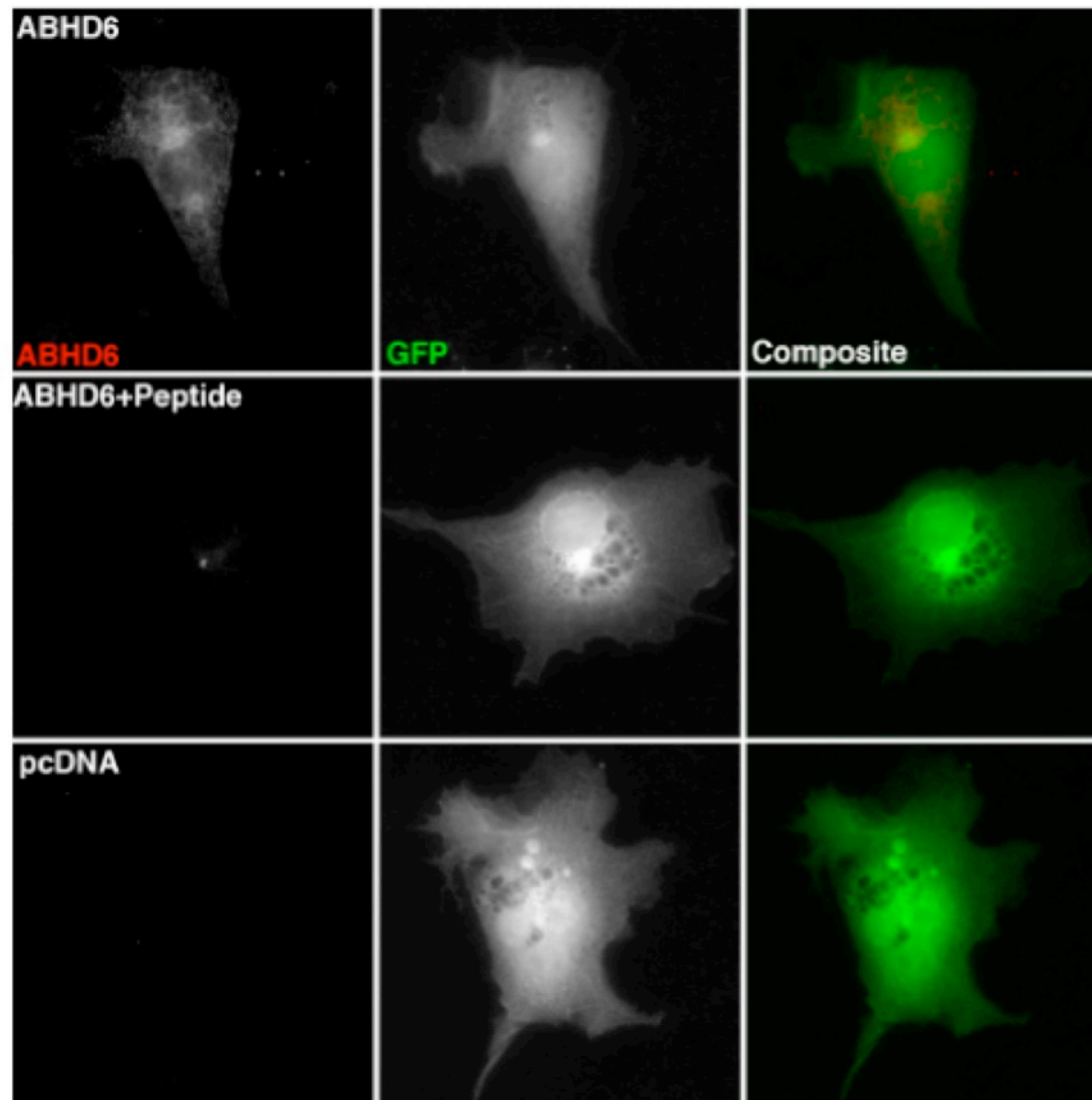
Supplementary Figure 6: ABHD6 Epitope

The rabbit polyclonal antibody generated to visualize ABHD6 recognizes a middle epitope (38 amino acids shown here in red).

Amino Acid Sequence for Mouse ABHD6
MDLDVVNMFVIAGGTLAIPILAFVASFLLWPSALIRIYYWYWRRTLGMQVRYAHH EDYQFCYSFRGRPGHKPSILMLHGFSAHKDMWLSVVKFLPKNLHLVCV DMPGHEG TTRSSLDDL SIVGQVKRIHQFVECLKLNKKP FHLIGTSMGGHVAGVYAAYYPSDV CSLSLVCPAGLQYSTDNPFVQRLKELEESAAIQKIPLIPSTPEEMSEMLQLCSYV RFKVPQQILQGLVDVRIPHNSFYRKLFLEIVNEKSRYSLHENMDKIKVPTQIIWG KQDQVLDVSGADILAKSISNSQVEVLENCGHSVVMERPRKTAKLIVDFLASVHNT DNKKLN

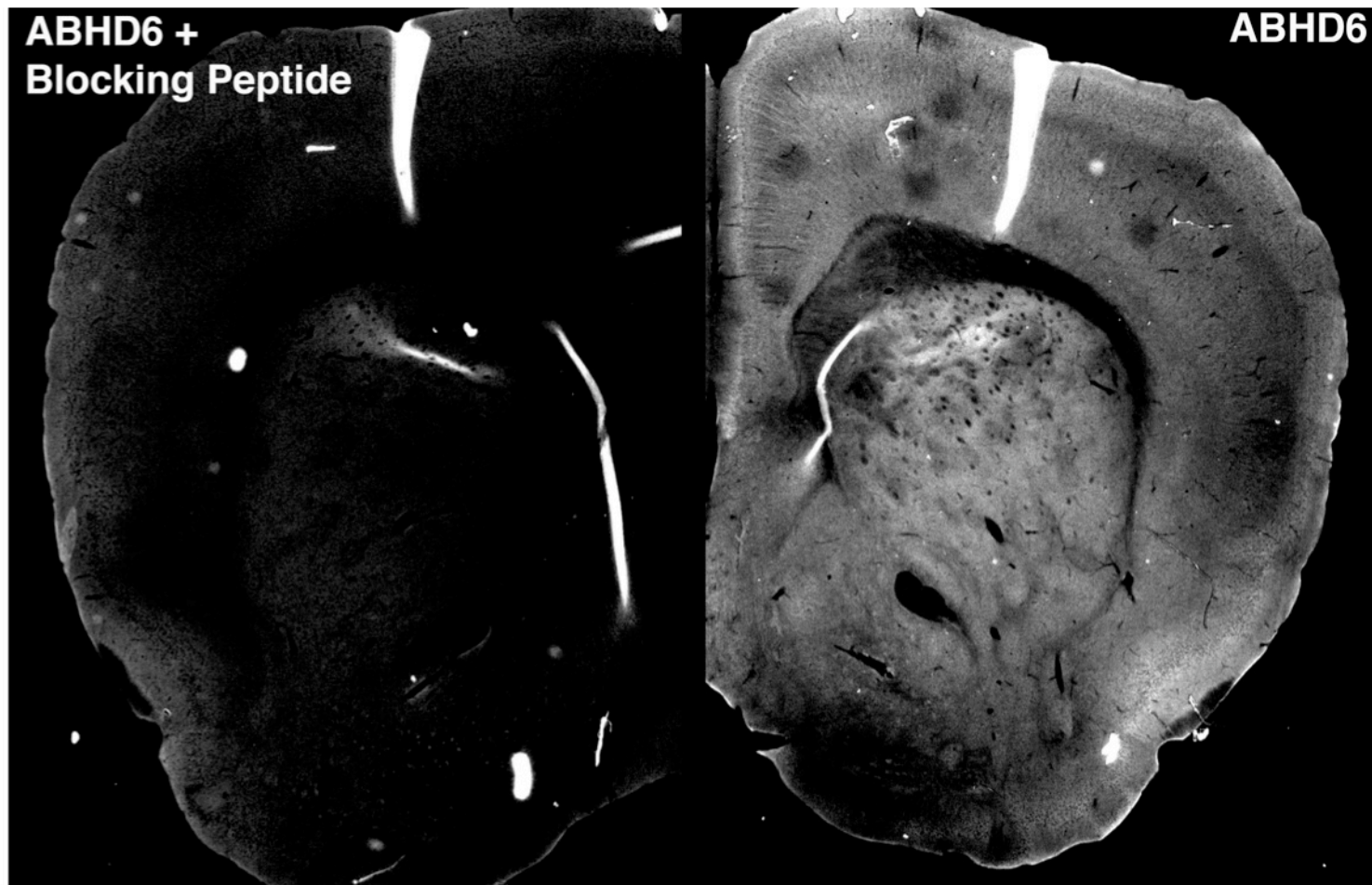
Supplementary Figure 7: ABHD6 antibody labels mouse ABHD6 transfected in COS-7 cells.

COS-7 cells were transfected with a plasmid that expresses both ABHD6 and GFP or a control plasmid that only expresses GFP (pcDNA). Incubation with the ABHD6 antibody (1:1000) resulted in clear labeling of cells expressing this protein (top row). However when the antibody is preincubated with the immunizing peptide (5 μ g; middle row) or when ABHD6 is not expressed (bottom row) then the labeling for ABHD6 is negative.

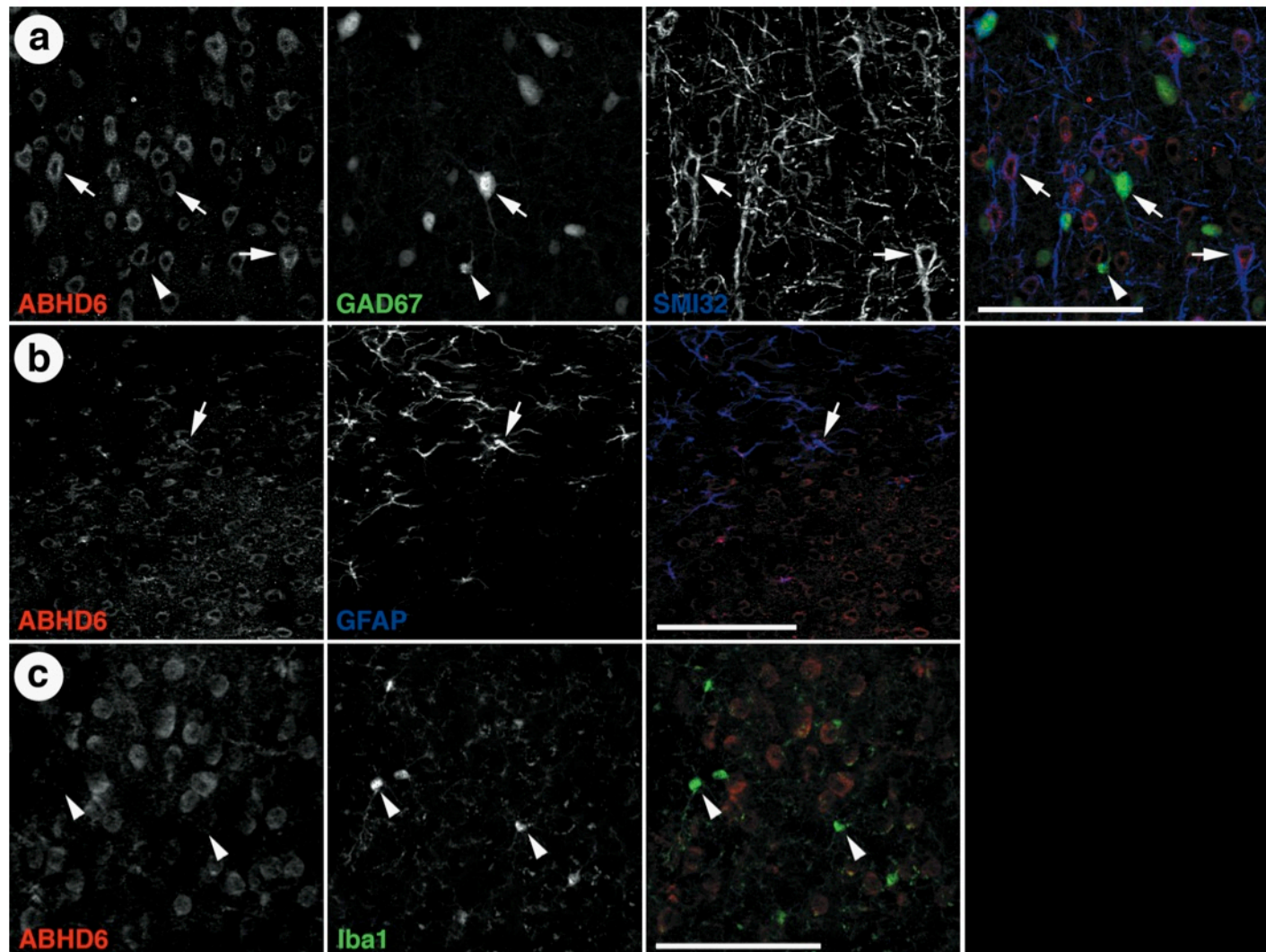


Supplementary Figure 8: ABHD6 expression in mouse brain

Coronal section of a 2 month old mouse brain at 2x magnification showing ABHD6 immunofluorescence +/- blocking peptide.

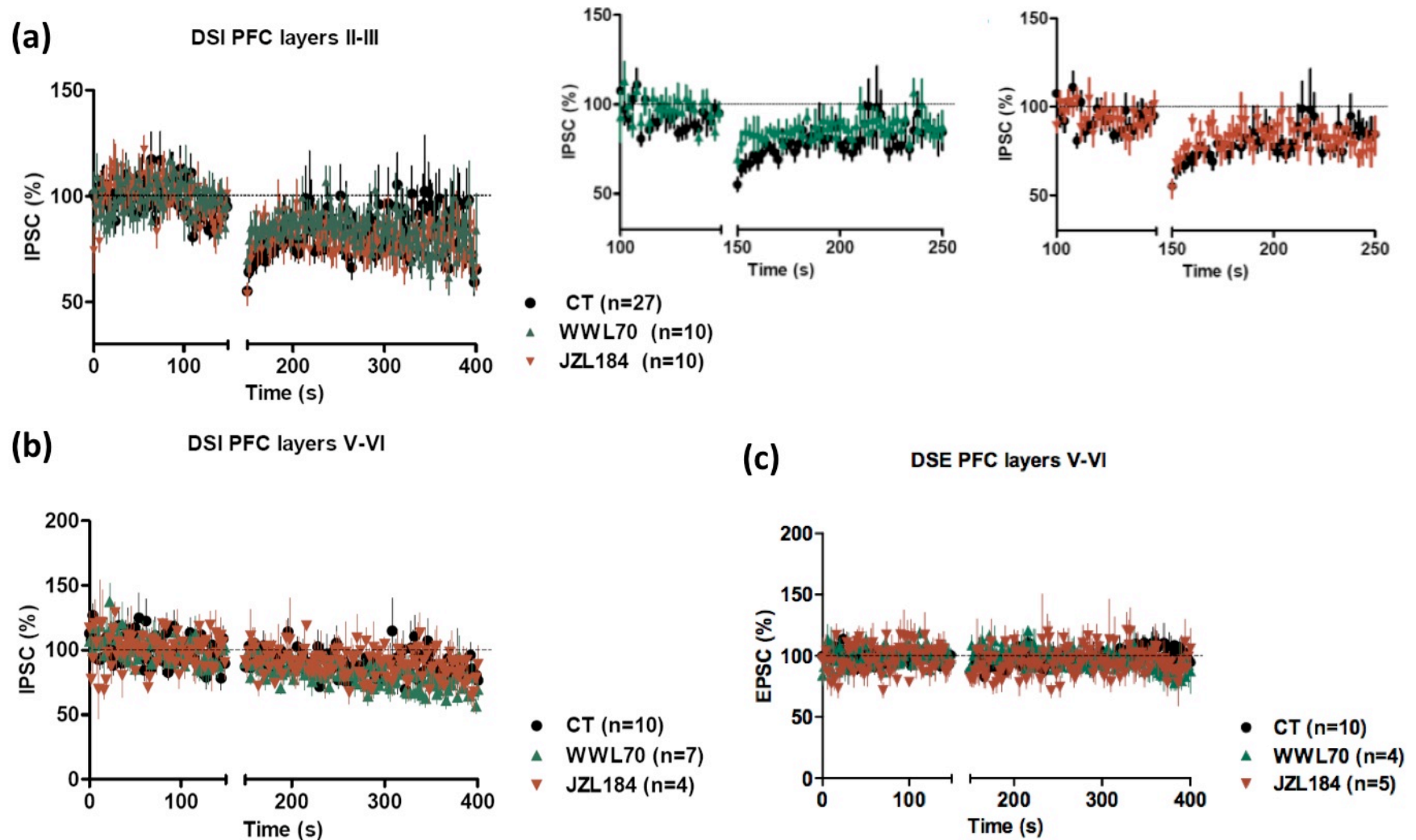


Supplementary Figure 9: ABHD6 is expressed in excitatory neurons, inhibitory neurons, and astrocytes, but not in microglia. **a)** Mouse cortical section expressing GFP in GABAergic interneurons (GAD67; green) were co-stained for ABHD6 (red) and a marker of glutamatergic neurons (SMI32; blue) revealing that ABHD6 is expressed in many glutamatergic neurons and some GABAergic neurons. **b)** Coronal section co-stained for ABHD6 (red) and a marker of astrocytes (GFAP; blue) shows that ABHD6 is also expressed in astrocytes. **c)** Microglia expressing GFP under the Iba1 promoter in the cortex lack staining for ABHD6. Arrows indicate cells positive for ABHD6, whereas arrowheads indicate ABHD6 negative cells. Scale bar = 100 μ m.



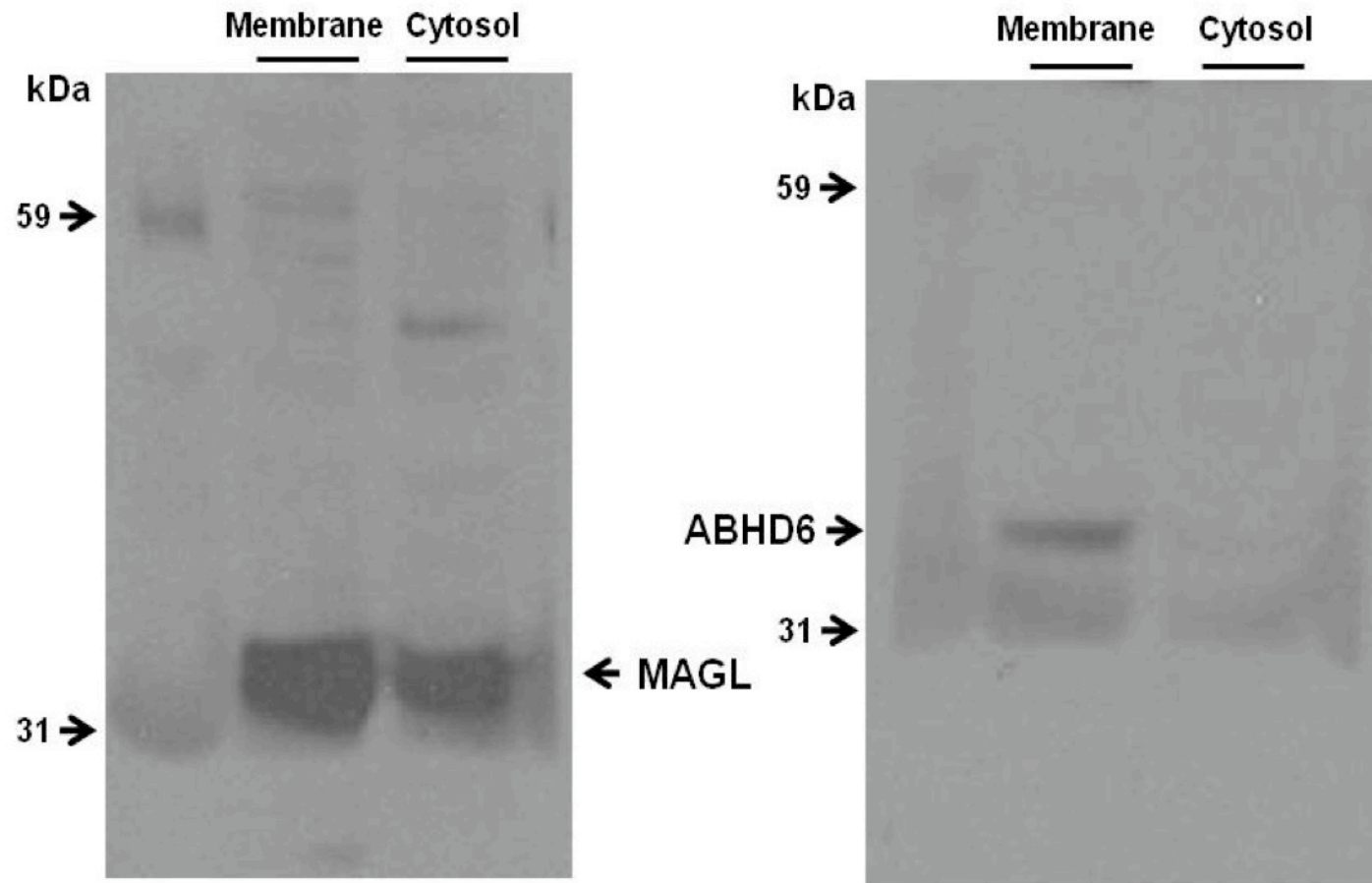
Supplementary Figure 10: Depolarization induced Suppression of Inhibition (DSI) and Excitation (DSE) in mouse cortex.

(a) A 10 second voltage step from -70mV to 0mV induces DSI in cortical layer 2/3. The rate of decay of this effect is not altered by WWL70 (10 μ M) or JZL184 (1 μ M). Insets: Detailed views for each inhibitor. The depolarization protocol did not induce (b) DSI or (c) DSE in cortical layer 5/6.



Supplementary Figure 11: Sub-cellular distribution of MAGL and ABHD6 in mouse brain

Western blots showing that MAGL immunoreactivity is found in both sub-cellular fractions of mouse brain (left blot), whereas ABHD6 immunoreactivity is only found in the membrane fraction (right blot).



Supplementary Table 1: Percent knockdown of each candidate enzyme by different shRNA constructs.

BV-2 cells were infected with the shRNA constructs listed here. The resulting BV-2 knock-down clones were harvested and the corresponding mRNA levels were measured by qPCR. These values were normalized to the respective mRNA levels in BV-2 cells transfected with scrambled shRNA. Results are expressed as the percentage decrease in mRNA levels compared to scrambled. Constructs highlighted in yellow were used for our experiments. n.a. = not applicable (i.e. colonies did not grow).

Scrambled	Knock-Down (%)
CAACAAGATGAAGAGCACCAACTCGAGTTGGTGCTCTTCATCTTGTTG	0
FAAH	
GCTCTTTACCTACCTGGGAACTCGAGTTTCCAGGTAGGTAAAGAGC	62
GCATTGTGCATGAAAGCCCTACTCGAGTAGGGCTTTCATGCACAATGC	52
GCCCAGATGGAACACTACAACTCGAGTTTGTAGTGTTCCATCTGGGC	83
CCCTTCTTACCAAACAACATACTCGAGTATGTTGTTTGGTAAGAAGGG	50
GCAGATTTATTTCTAGCGAATCTCGAGATTCGCTAGAAATAAATCTGC	47
NTE	
CCTGTTCCCTAGACTGGGTTATCTCGAGATAACCCAGTCTAGGAACAGG	78
CCTATGAACGTGGACGGATATCTCGAGATATCCGTCCACGTTCATAGG	66
CCTGTATTGGACCTCACATATCTCGAGATATGTGAGGTCCAATACAGG	55
GCCTGTATTGGACCTCACATACTCGAGTATGTGAGGTCCAATACAGGC	25
CCCGCCTTATTCATCTGCTAACTCGAGTTAGCAGATGAATAAGGCGGG	54
ABHD12	
GCCCTTTCATCTCGGTAGAACTCGAGTTTCTACCGAGATGAAAGGGC	49
CCCTTATATTGGAGTCTCCATCTCGAGATGGAGACTCCAATATAAGGG	-81
GCAGTGGAATTAATTTGCAACTCGAGTTGCAATTTAATTCCACTGC	50
GTGGTGATAATCCTGTGTATACTCGAGTATACACAGGATTATCACCAC	62
CCGAGACTTCAAAGTCCAGTTCTCGAGAACTGGACTTTGAAGTCTCGG	n.a.
ABHD6	
GCATGAGAATATGGACAAGATCTCGAGATCTTGTCATATTCTCATGC	-3
CAGACATATTAGCCAAGTCAACTCGAGTTGACTTGGCTAATATGTCTG	-3
CCTGCAGTACTCAACTGACAACCTCGAGTTGTAGTTGAGTACTGCAGG	n.a.
GCCATTCCAATCCTGGCATTCTCGAGAAATGCCAGGATTGGAATGGC	37
CAGCACTGATAAGAATCTATTCTCGAGAAATAGATTCTTATCAGTGCTG	51
CCTGGCATTGTGTCGTCTTCTCGAGAAAGACGCAACAAATGCCAGG	4
TGAGGACTATCAGTTCTGTTACTCGAGTAACAGAACTGATAGTCCTCA	18
CGTGGTCAAGTTCCTTCCGAACCTCGAGTTCCGAAGGAACCTGACCACG	-34
CCATGGATTCTCCGCACACAACCTCGAGTTGTGTGCGGAGAATCCATGG	19
ATCAGTTTGTAGAATGCCCTACTCGAGTAAGGCATTCTACAACTGAT	24
GCTACTGACTGAGAAGAGAACTCGAGTTTCTCTTCTCAGTCAGTAGC	49