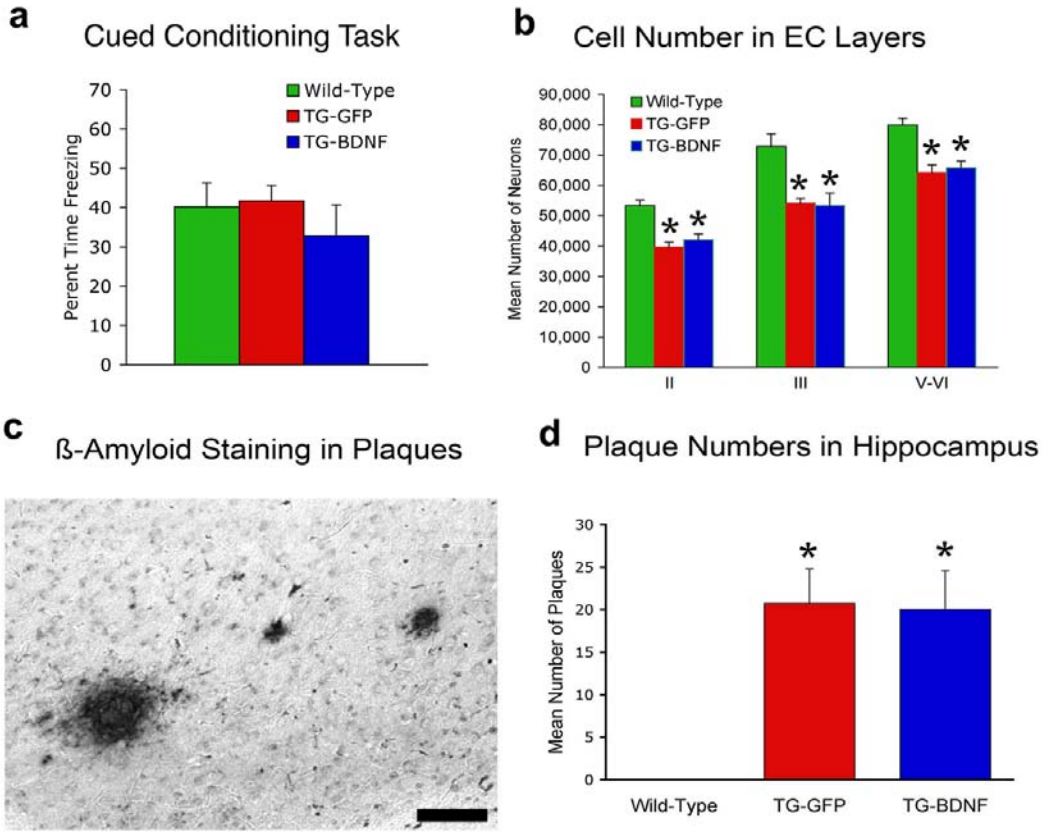
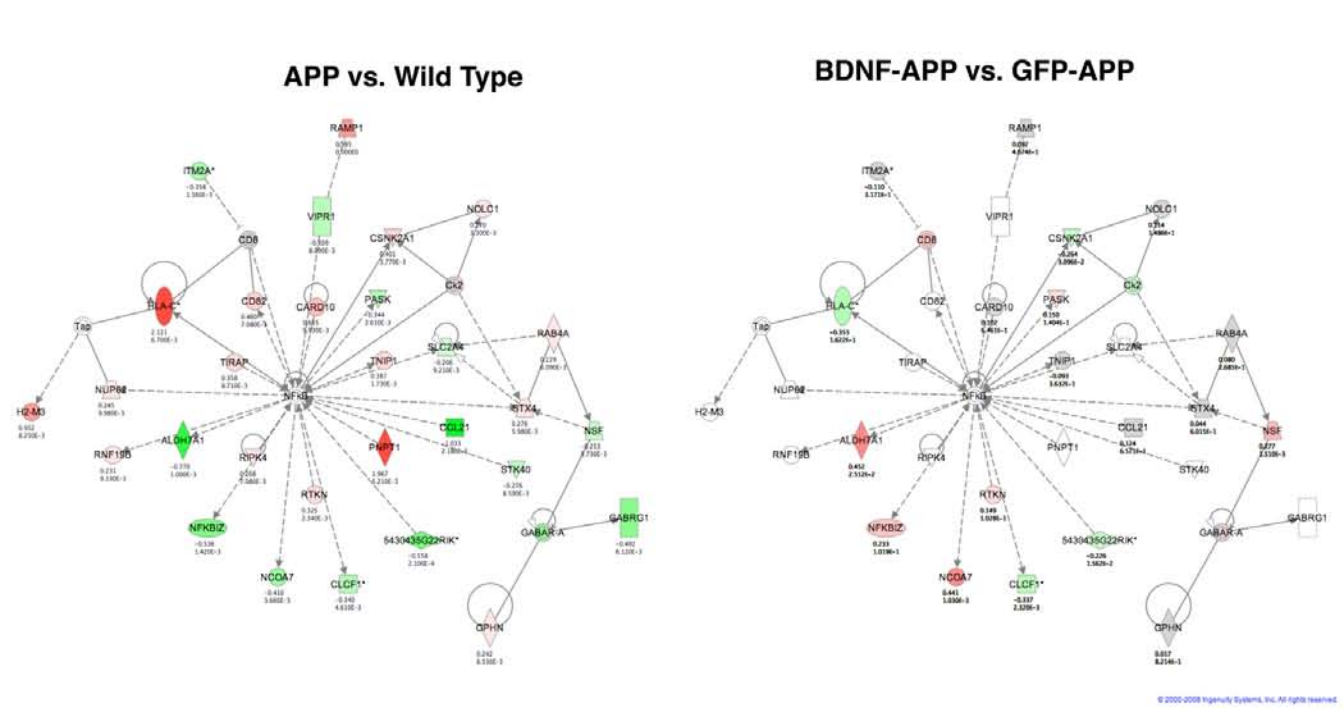
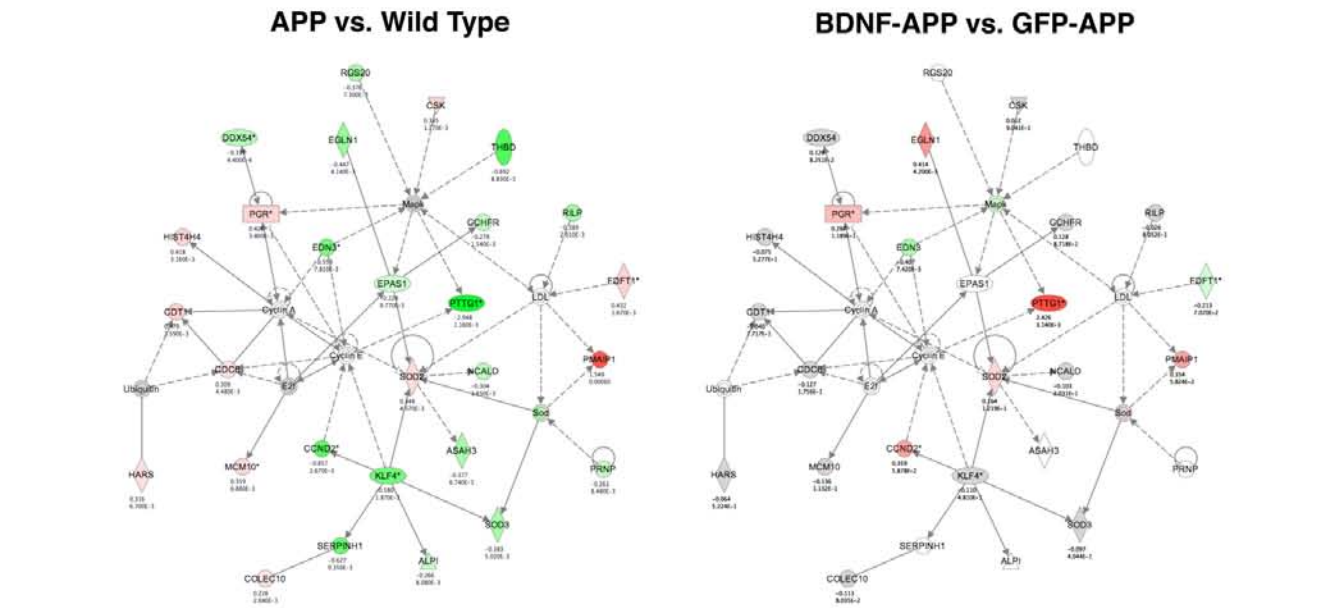
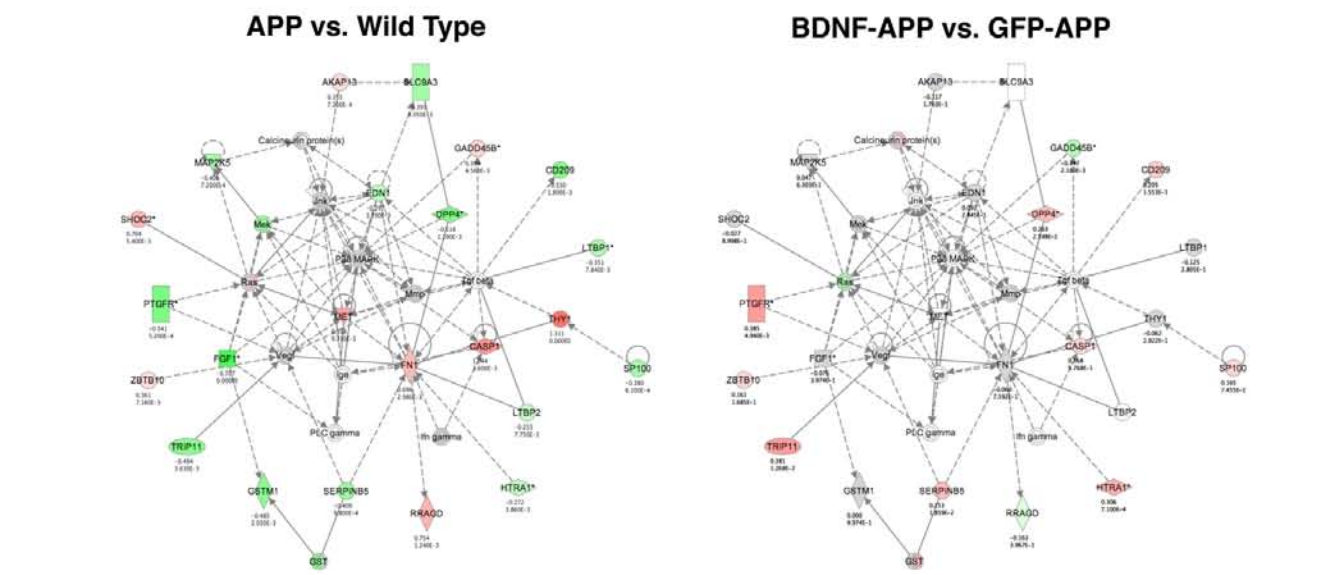


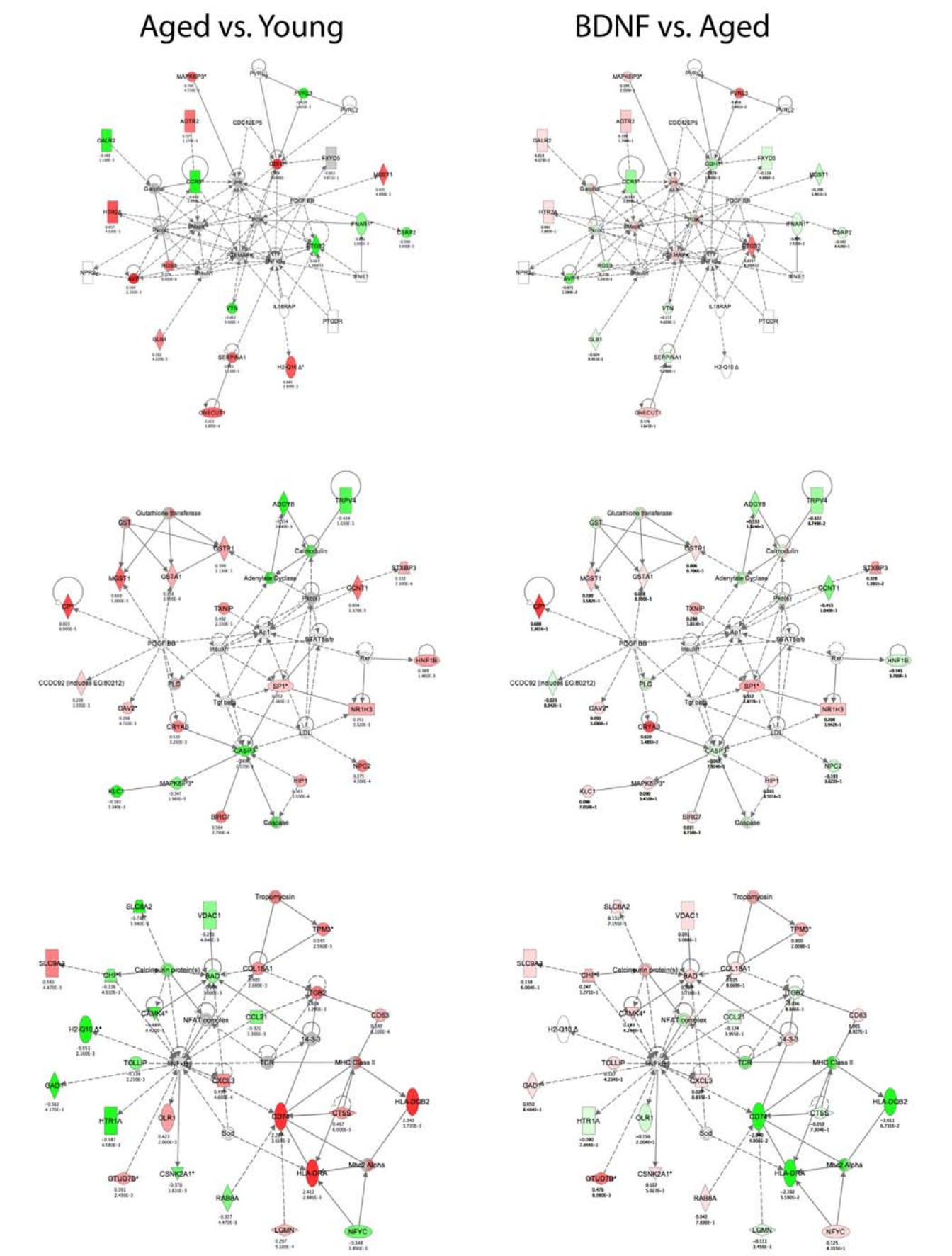
Supplemental Figure 1: APP mutant mice. (**a**) Performance on cued conditioning task, which is not hippocampal-dependent. As expected, APP transgenic mice show no impairments on a cued conditioning task that is not dependent on entorhinal-hippocampal circuitry (ANOVA $p = 0.74$). (**b**) Stereological quantification demonstrates that both Lenti-BDNF-treated and Lenti-GFP-injected APP mutant mice exhibit significant cell loss in entorhinal cortical layers II, III, and V-VI compared to wild-type littermates (ANOVA $p = 0.0001$; post-hoc Fisher's $p = 0.0001$). (**c**) ß-amyloid plaque labeling in APP transgenic mouse. Scale bar, 85µm. (**d**) Quantification of plaque numbers in the hippocampus indicates that BDNF treatment does not alter plaque density over the 8-week experimental period (ANOVA $p = 0.0005$; post-hoc Fisher's TG^{BDNF} vs. TG^{GFP}; $p = 0.88$). Thus, the beneficial effects of BDNF in this model are exerted independent of direct effects on Aß plaque number.



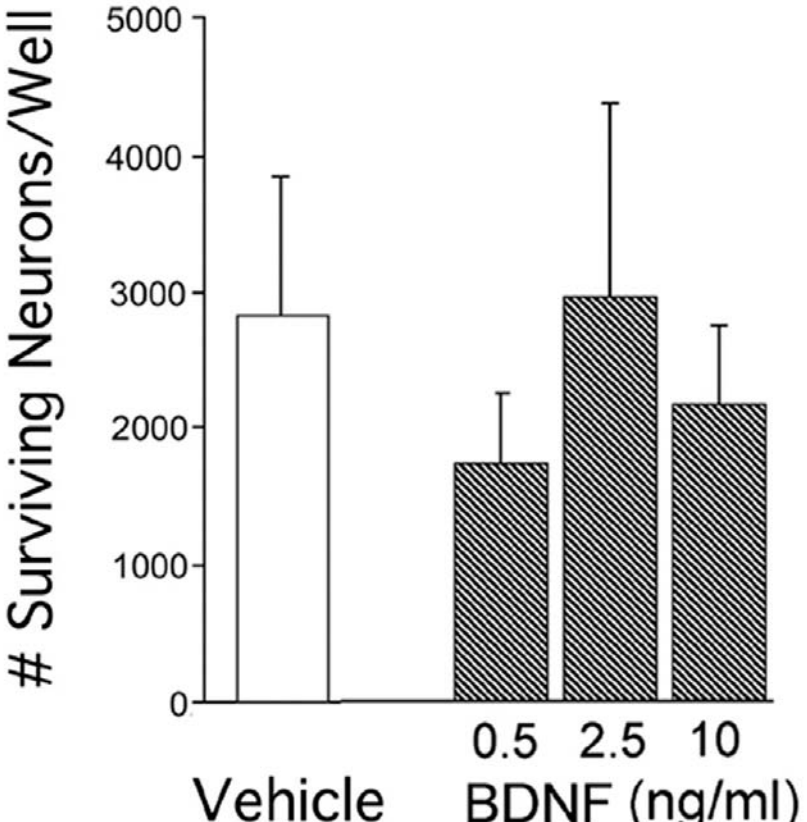




Supplemental Figure 2: Pathway Analysis in APP Transgenic Mice
Compared to wild-type control mice, expression of mutant APP results in perturbations in canonical pathways related to oxidative stress, lipid metabolism and cell function. Each sample network illustrates perturbations in entorhinal cortex of APP transgenics compared to wild-type mice on left side, and tendencies to reverse perturbations in these pathways after BDNF treatment (right side). Color coding of the gene symbols corresponds to fold changes in TG vs WT (left) and TGBDNF mice vs. TGGFP mice (right). Examples of three separate canonical pathways are shown.



Supplemental Figure 3: Pathway Analysis in Aged Rats
Compared to young rats, aging results in perturbations in a number of canonical pathways, including oxidative stress, lipid, and glutathione metabolism. Each sample network illustrates perturbations in aged rats compared to young on left side, and trend to reverse perturbations in these pathways after BDNF administration, in entorhinal cortex (top) and hippocampus (middle and bottom panel) of aged rats. Color coding of the gene symbols corresponds to fold changes in aged vs. young rats (left) and BDNF-aged rats vs. GFP-aged rats (right).



Supplemental Figure 4: BDNF Specifically Reduces Aß Toxicity.
BDNF specifically prevents Aß-induced toxicity rather than generally increasing the number of cultured entorhinal cortical neurons. Addition of BDNF to culture conditions in the absence of Aß does not significantly increase cell survival after 24 hr in culture (comparing **Vehicle** to **BDNF** group).

SUPPLEMENTAL MATERIAL

METHODS:

All procedures were conducted with experimenters blinded to group identity.

Transgenic Mouse Study

Subjects: Heterozygotic transgenic mice on a BI/6 background expressed the APP Indiana (V717F) and Swedish (K670M) mutations (J20 strain)[1, 2]. Subjects received injections of lentiviral vectors expressing either recombinant human BDNF, enhanced GFP at the same dose, or sham surgery (needle passage into the same coordinates of the EC). A total of 5µl of vector ($1.25x10^8$ infectious units/ml) was injected into each subject at a rate of 1µl/min. Pre-operative baseline learning and memory were assessed using hidden platform testing in the Morris water maze[3, 4]. One week following water maze testing, mice were tested on a fear conditioning paradigm in Habitest chambers (Coulbourn Instruments). Animal movements were tracked using an infrared body heat activity monitor (Coulbourn Instruments). After completion of behavioral testing, mice were sacrificed and brains were post-fixed in 2% paraformaldehyde/0.2% parabenozquinone. Brains were sectioned on a microtome set at 30µm thickness. For unbiased stereological analyses, a series of 1-in-6 Nissl-stained sections were examined using StereoInvestigator software (Microbrightfield) on a Ludl motorized system. Cell number was quantified in layer II of medial EC on both intact and lesioned sides, as described[4]. Layer II of medial EC was outlined at low magnification (4x) and cell counts were performed at high magnification (60x). A 30µm x 30µm counting frame was used within a 120µm x 120µm sampling grid, and cell counts were made within a 15µm-thick zone that began 5% below the measured thickness of the section at each site. Only cells

with nuclei falling within the 15-µm-thick sampling zone at each site were counted. Cross-sectional area of the soma at the level of the nucleus was also stereologically measured based on intersection of 6-ray extensions from the cell nucleus. Entorhinal cortex layers II, III, and V-VI were outlined, cells were quantified as noted above, and cell body size was measured. Immunocytochemical labeling was performed for BDNF (1:1000; ABCAM), GFP (1:1500; Rockland); beta-amyloid (4G8; 1:1000; Signet), synaptophysin (1:1000; Chemicon), and phosphorylated-ERK (1:300; Cell Signaling). Processing continued using either fluorescent secondary antibodies (Molecular Probe) or avidin/biotin amplification and Vector ABC kit with DAB reaction. Quantification of immunolabeled sections was performed in a blinded fashion on sections/images, and statistical analysis was performed using ANOVA and post-hoc Fisher tests with a significance criterion of $P<0.05$.

Aged Rat Study

Rat Water Maze Procedure/Pump Implantation: Spatial learning and memory were tested in the Morris water maze following established protocols[3, 4]. Aged and young rats were pre-operatively screened to quantify their ability to learn the location of a hidden platform in a 6-foot circular pool of opaque water using spatial cues in the environment. Distance and latency to locate the hidden platform were measured over 4 pre-operative testing blocks, each consisting of 6 total trials (3 trials per day of maximum duration 90 sec/trial). The last trial of each block consisted of a probe trial, in which the hidden platform was removed from the pool to measure the platform search strategy of each animal (the number of times that rats crossed a 10 cm annulus of space centered about the

goal platform, or "annulus crossings"). 30 of 37 aged subjects were cognitively impaired, performing below the range of the worst-performing young subject on both latency and distance measures during the fourth block of testing, a standard criterion of age impairment[5]. Following pre-operative screening, Aged-Impaired subjects received implants of 4-week Alzet minipumps (model 2004) that delivered either 120ng recombinant human BDNF/day (20 ng/ml; Chemicon) in artificial CSF, or artificial CSF alone. Infusion devices were placed bilaterally into the entorhinal cortices at coordinates relative to Bregma of -9.3 mm A/P, ± 5.6 mm M/L, 6 mm V/D. Post-operative testing began two full weeks after surgery, also consisting of 4 testing blocks of 6 trials per block. At the conclusion of non-visible platform testing, rats were-tested on a visible platform placed in the pool at a novel location to ensure comparable visual and motor capabilities across groups. No significant differences in performance were found across groups in this "cued" visible platform condition (latency: P=0.68; distance: P=0.35). Impaired spatial learning with intact cue learning is a behavioral profile consistent with results observed in young rats with hippocampal or entorhinal-perirhinal lesions[6]. Comparisons between groups were made using repeated-measures 2-way ANOVA (learning vs. time across groups). Collection of the data was performed using a Columbus Instruments tracking system. Rats were then either decapitated rapidly for immunoblots or perfused transcardially with 2% paraformaldehyde for histological analysis. For histology, 1-in-12 50µm-thick sections were immunolabeled for BDNF using rabbit anti-BDNF antibody @ 1:6000 (gift of Q. Yan, Amgen), with avidin/biotin amplification and $NiCl_2$ intensification. Antibody specificity was verified by primary antibody omission, which completely eliminated labeling. Immunoblots were performed per published

protocols[7]. Subjects were young (*n*=8), non-operated aged-unimpaired (*n*=4), non-operated aged-impaired (*n*=4), BDNF-infused aged (*n*=4), and vehicle-infused aged (*n*=4). Briefly, the medial entorhinal cortex was rapidly dissected, sonicated in ice-cold RIPA buffer (50 mM Tris, pH 8, 150 mM NaCl, 1% NP-40, 0.5% deoxycholate, and 0.1% SDS) and boiled in 1X Laemmli sample buffer. Tissue from pairs of animals within each treatment group was pooled. Proteins were resolved by SDS-PAGE, transferred onto PVDF membrane (Millipore), and analyzed by immunoblotting using the following primary antibodies: rabbit anti-phosphoAkt (Ser473), mouse anti-phosphoERK (Cell Signaling Technologies), mouse anti-Akt1 (Santa Cruz), or rabbit anti-pan ERK (Santa Cruz). Proteins of interest were visualized by incubation with appropriate horseradish peroxidase-conjugated secondary antibodies (Jackson Laboratories) and enhanced chemiluminescence (Amersham). Quantities of loaded protein were normalized to levels of non-phosphorylated ERK. Blots were quantified using NIH Image software and statistical analysis was performed using ANOVA and post-hoc Fisher's. For microarray studies, entorhinal cortices and hippocampi were rapidly dissected and snap frozen to –80^{o}C. Amplified and biotin-labeled cRNA was hybridized to Affymetrix Mouse Genome 430_2 and Rat Genome 230_2 arrays per manufacturer instructions. Raw data was analyzed using Bioconductor packages (www.bioconductor.org[8]). Quality assessment was performed by assessment of the inter-array Pearson correlation and RNA degradation plots. Clustering based on top variant genes was used to assess overall data coherence. Contrast analysis of differential expression was performed using the LIMMA package[9]. After linear model fitting, a Bayesian estimate of differential expression was calculated and the threshold for statistical significance was set at $P<0.005$ (Bayesian modified t-

test). Pathway and gene ontology analysis was performed using Ingenuity Pathways Analysis (www.ingenuity.com). Data analysis aimed to identify APP- or aging-related genes and the effect of BDNF therapy on their expression levels. For the BDNF effect analysis, APP- or aging-related genes that were differentially expressed after GFP viral infection alone were not considered BDNF-specific and were excluded, resulting in a more stringent analysis of the BDNF effect. A trend towards normalization was defined as a relative difference (delta) of at 30% towards normal levels when comparing BDNF effect vs. APP/aging effect.

In Vitro Aß Toxicity Study

Entorhinal cortices were dissected from postnatal-day-3 rats in ice cold Hibernate A medium. Tissue was trypsinized and mechanically dissociated by gentle pipetting. Dissociated cortical neurons were collected and seeded on poly-L-lysine-coated 3.5 cm dishes and grown in DMEM/F12 medium supplemented with B27 for 24 hours before start of the Aß assay. Entorhinal neurons were treated with Aß peptide 1-40 or 1-42 at 5 or 10 μM (gift of C. Glabe). Aß was dissolved in 1% DMSO and added to culture media for 24 hr; control cultures received DMSO alone. Aß 1-40 and 1-42 caused equivalent toxic effects on entorhinal neurons, whereas scrambled Aß had no toxic effect. Because these levels of Aß were equally toxic, further studies focused solely on addition of the 1-40 peptide at 10μM concentration. BDNF or NGF (concentrations of 0, 0.5, 2.5, or 10 ng ml^{-1}) were provided to the cultures at the same time that Aß was added. 24 hr later, cell viability was determined using the LIVE/DEAD Viability/Cytotoxicity Kit; all neurons

per culture dish were counted, and experiments were repeated in triplicate. Differences were determined by ANOVA with post-hoc Dunnet's.

Perforant Path Lesion Study

Three-month-old male Fischer 344 rats received injections of lentivirus expressing BDNF and (via an internal ribosomal entry site, IRES) GFP (BDNF-GFP; n=8 animals). Controls received injections of lentivirus expressing NGF and GFP (NGF-GFP; n=8 animals) or GFP alone (n=6 animals). Vector was injected into three sites distributed evenly through the right entorhinal cortex. A total of 5µl of vector (concentration $1.25x10^8$ infectious units/ml) was injected into each subject at a rate of 1µl/min. Four days later, rats underwent transection of the right perforant pathway using a Kopf wire knife lowered to coordinates relative to bregma of AP: -7.8 mm, ML –4.1mm, VD –6.0 mm. The wire knife was extended 2.2 mm and raised 3.7 mm, transecting the perforant pathway. Two weeks later, rats were sacrificed and brains were sectioned in the horizontal plane on a microtome set at 40µm thickness. Stereological analyses were performed in layer II medical EC using methods described above on 1-in-6 Nissl-stained sections. Differences among groups were determined by ANOVA with post-hoc Fisher's, significance criterion $P<0.05$. Data are presented as mean ± SEM. All analyses were performed in a blinded manner.

Primate Perforant Path Lesion Study

Six adult rhesus monkeys underwent perforant path transections to examine BDNF-mediated neuroprotection of neurons in the lateral entorhinal cortex. To create bilateral

perforant path lesions, a heat-generating lesion probe (Radionics) was passed into the angular bundle using MRI-generated coordinates located rostral to the entorhinal cortex and caudal to the hippocampus. The probe temperature was raised to 80^{o}C for one minute in each of nine sites in a 3x3mm grid using the lesion device (Radionics, Burlington, MA). Monkeys then received injections of lentiviral vectors expressing either: BDNF (*n*=6 monkeys) into the right entorhinal cortex, GFP (*n*=4 monkeys) into the left entorhinal cortex, or lesions alone (*n*=2 monkeys) in the left entorhinal cortex. Vector was injected into four sites selected on pre-operative MRI scans to encompass the rostral-to-caudal extent of the medical entorhinal cortex. 20µl of vector solution were injected per site at a rate of 1µl per minute, vector concentration 5x10^{8} infectious units/ml. After a one-month survival period, subjects were transcardially perfused and entorhinal cortices were sectioned at intervals set at 40µm. Cells in layer II of the lateral entorhinal cortex were quantified on serial Nissl-stained sections using stereological methods, as previously reported[10]. Briefly, with the nucleolus as a reference, an optical sampling frame of 10%, and a z-axis inclusion/exclusion zone of 5% at the top and bottom of each section, cell number was quantified in a 1:12 series of sections within lateral entorhinal subregions based on laminar landmarks[10] using StereoInvestigator software. Values were compared using ANOVA and post-hoc Fisher's with a significance criterion of $p<0.05$.

Aged Primate Study

Nine aged and six young rhesus monkeys were subjects of this study. The mean age of BDNF-treated subjects (mean age 24.3±1.3 years, range 21–27 years; two males and two females) was slightly older than GFP-treated monkeys (mean age 22.4±0.6 years, range

21–24 years, all males) but did not differ significantly (P =0.25). The mean age of young monkeys was 7.8±0.7 years (range 6–10 years; 3 females and 3 males). Prior to gene transfer, all subjects were trained to criterion levels of performance on a visuospatial learning task[11, 12] using a home cage-based computerized cognitive task adapted from the Cambridge Neuropsychological Test Automated Battery (CANTAB)[13]; details of the task are reported in[12, 14]. Briefly, subjects were shown a stimulus on a computer screen, the screen then became blank, and after a 2-second delay three choice objects were presented; the monkey was required to choose the original object. Twenty trials per session were presented over 20 consecutive training sessions. The number of correct choices on this task was quantified pre-operatively in all subjects and compared among groups. Aged subjects as a group were significantly impaired compared to young subjects at the completion of pre-operative testing. Aged subjects were then divided into two groups: four subjects underwent lenti-BDNF gene transfer to the entorhinal cortex and five subjects underwent lenti-GFP gene transfer, using targeting methods and vector doses described in the primate perforant path lesion experiment. Beginning one month after gene delivery, performance of aged animals on the task was re-assessed daily over 10 sessions, and each subject's change in performance compared to post-operative session 1 baseline was calculated. Mean improvement in performance between BDNF-treated and control aged groups was compared using 2-tailed Student's t-test. On completion of behavioral testing, subjects were transcardially perfused and entorhinal cortices were coronally sectioned at set intervals of 40 μm. The size of neurons in layer II of the entorhinal cortex was quantified on Nissl-stained sections using stereological methods, as previously reported[10, 15]. Mean neuronal size was compared among groups

using analysis of variance and post-hoc differences were assessed by Fisher's least square difference. Aged primates do not exhibit reductions in entorhinal neuronal numbers compared to young monkeys[10], hence cell numbers were not quantified.

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Supplementary File 1 (SupplFile1.xls)

Differentially expressed genes in APP-TG mice compared to WT, and effect of BDNF gene delivery.

Columns and tab bars present full data set, using following key to comparisons: E, entorhinal cortex; H, hippocampus; APP_GFP is TG^{GFP}; NTG_Sham is WT^{Sham}; BDNF is TG^{BDNF}; NTG_GFP is WT^{GFP}; APP is TG^{sham}.

For example, in Sheet 'TG-EC', Column D (E.APP_GFPvsNTG_Sham) indicates comparison of entorhinal cortex in TG^{GFP} vs. WT^{Sham}. Column E (H.APP_GFPvsNTG_Sham) indicates comparison of hippocampus in TG^{GFP} vs. WT^{Sham}.

Sheets represent following data:

1) Legend: Column names, legend
2) TG-EC: 424 differentially expressed probesets in entorhinal cortex (EC) of TG vs. WT mice (APP ratio in column D, E.APP_GFPvsNTG_Sham).
3) GO_TC-EC: Gene ontology of genelist in Sheet #2 (TG-EC).
4) TG-EC_BDNF: *BDNF effect on genes differentially expressed in APP mice, entorhinal cortex*: 107 probesets are differentially expressed in the entorhinal cortex (EC) of TG vs. WT mice, after exclusion of probesets differentially expressed after GFP viral infection alone (ratios in Column D, E.APP_GFPvsNTG_Sham). In bold the 59 probesets (55%) showing at least a 30% change towards normal levels (column N, Delta_E.BDNFvsAPP) after BDNF treatment.

5) TG-Hipp: 146 differentially expressed probesets in hippocampus (Hipp) of TG vs. WT mice (APP ratio in column E).
6) GO_TG-Hipp: Gene ontology of genelist in Sheet #5 (TG-Hipp).
7) TG-Hipp_BDNF: *BDNF effect on genes differentially expressed in APP mice, hippocampus*: 37 probesets are differentially expressed in the hippocampus (Hipp) of TG vs. WT mice after exclusion of probesets differentially expressed after GFP viral infection alone (ratios in Column E, H.APP_GFPvsNTG_Sham). In bold the 20 probesets (54%) showing at least a 30% change towards normal levels (column N, Delta_H.BDNFvsAPP) after BDNF treatment.
8) BDNF-EC: General BDNF effect on TG animals, entorhinal cortex (300 differentially expressed probesets, ratios in Column L, E.BDNFvsAPP).
9) GO_BDNF-EC: Gene ontology of genelist in Sheet #8 (BDNF-EC).
10) BDNF-Hipp: General BDNF effect on TG animals, hippocampus (43 differentially expressed probesets, ratios in Column O, H.BDNFvsAPP).
11) GO_BDNF-Hipp: Gene ontology of genelist in Sheet #10 (BDNF-Hipp).

Supplementary File 2 (SupplFile2.xls)

Differentially expressed genes in aged rats compared to young, and effect of BDNF gene delivery to entorhinal cortex.

Columns and tab bars present full data set, using following key to comparisons: E, entorhinal cortex; H, hippocampus; Cy is control young; BDNF is aged-Lenti-BDNF; GFP is aged-Lenti-GFP; Ca is control aged (aged, unoperated).

For example, in Sheet 'CavsCy-EC', Column D (H.BDNFvsCy) indicates comparison of hippocampus in aged-Lenti-BDNF vs. Control young. Column E (H.BDNFvsGFP) indicates comparison of hippocampus in aged-Lenti-BDNF compared to aged-Lenti-GFP. Sheets represent following data:

Differentially expressed genes in aged rats compared to young rats, and effect of BDNF infusion.

1) Legend: Column names, legend.
2) CavsCy-EC: 114 differentially expressed probesets in entorhinal cortex (EC) of aged vs. young rats (ratios in column I, E.CavsCy).
3) GO_CavsCy-EC: Gene ontology of genelist in Sheet #2 (CavsCy-EC)
4) CavsCy-EC_BDNF: *BDNF effect on aging-related genes, entorhinal cortex*: 61 probesets are differentially expressed in the entorhinal cortex (EC) of aged vs. young rats after exclusion of probesets differentially expressed after GFP viral infection alone (ratios in column J, E.CavsCy). In bold the 26 probesets (43%) showing at least a 30% change towards normal levels (Column K, Delta_E.BDNFvsGFP) after BDNF treatment. Column C: overlap with Rowe et al[16].
5) CavsCy-Hipp: 534 differentially expressed probesets in hippocampus (Hipp) of aged vs. young rats (ratios in column F, H.CavsCy).
6) GO_ CavsCy-Hipp: Gene ontology of genelist in Sheet #5 (CavsCy-Hipp)
7) CavsCy-Hipp_BDNF: *BDNF effect on aging-related genes, hippocampus:* 181 probesets are differentially expressed in the hippocampus (Hipp) of aged vs. young rats after exclusion of probesets differentially expressed after GFP viral

infection alone (ratios in column G, H.CavsCy). In bold the 77 probesets (42%) showing at least a 30% change towards normal levels (Column K, Delta_H.BDNFvsGFP) after BDNF treatment.

8) BDNF-EC: General BDNF effect on aged animals, entorhinal cortex (206 differentially expressed probesets, ratios in Column H, E.BDNFvsGFP)
9) GO_BDNF-EC: Gene ontology of genelist in Sheet #8 (BDNF-EC).
10) BDNF-Hipp: General BDNF effect on aged animals, hippocampus (100 differentially expressed probesets, ratios in Column E, H.BDNFvsGFP)
11) GO_BDNF-Hipp: Gene ontology of genelist in Sheet #10 (BDNF-Hipp).