IRX4204

A Phase II Clinical Stage Potent and Selective RXR Agonist Compound for Treatment of Neurodegenerative and Autoimmune Diseases

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Io Therapeutics, Inc.
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What We Are Seeking

Io Therapeutics is seeking equity financing or corporate partnerships with established biopharmaceutical companies for world-wide development and commercialization of a platform of products based on the phase II clinical stage, highly potent and selective RXR nuclear receptor agonist compound IRX4204, for treatment of neurodegenerative and autoimmune diseases.

IRX4204 Platform Product Rationale

- IRX4204 already has demonstrated oral drug delivery, clinical safety, dose range, and clinical activity in humans with cancers or Parkinson's disease.
- Imbalance of Tregs and Th17 cells and overproduction of IL-17, TNF, and IL-6 are well documented pathologic features of many autoimmune and inflammatory CNS, skin musculoskeletal, gastrointestinal, and lung diseases.
- IL-17, TNFα, IL-6 are already clinically well validated targets in humans for treatment of multiple autoimmune diseases including rheumatoid arthritis, psoriasis, psoriatic arthritis, ankylosing spondylitis, Crohn's disease, and ulcerative colitis.
- IRX4204 has effects promoting dopaminergic and cortical neuron survival and function in PD and AD models.
- IRX4204 has myelin protective and reparative effects.
- IRX4204 can be formulated for oral, parenteral, topical for skin and eye, and inhaled for lung administration, providing a platform of product opportunities for CNS and autoimmune diseases.

IRX4204 Intellectual Property for Neurodegenerative and Autoimmune Diseases

- **Issued US Patents for Neurodegenerative Diseases:**
  1. Monotherapy treatment with IRX4204 of any neurologic condition, either central or peripheral, which involves both neuroinflammation and demyelination;
  2. Combination treatments of IRX4204 with thyroid hormone, and optionally, neurotrophic factors, for aging related neurodegeneration, AD, PD, MS, ALS, Stroke, brain or spinal cord trauma, hypoxic brain injury, schizophrenia, depression, autism.
- **Pending US patent for**: in vitro growth of myelinated glial cells for tissue repair
- **Issued US Patents for Autoimmune Diseases:**
  Autoimmune inflammatory arthritis (RA, OA, PSA, AS, Juvenile arthritis); psoriasis, eczema, and other autoimmune skin diseases; Crohn's disease, ulcerative colitis, celiac disease; asthma and other allergic diseases
- **Pending US Patents for Other Autoimmune/Immune Indications**
- solid organ transplant rejection, GVHD, retinopathies
- **Issued International Patents for Neurodegenerative and Autoimmune Diseases:**
  The company owns issued patents for Parkinson's disease; as well as for many of the above listed indications in Europe, Japan, South Korea, China, Hong Kong, Singapore, Australia, New Zealand, Canada, Mexico, Israel, and South Africa.

Io's development interests for IRX4204 for Neurodegenerative and Autoimmune Diseases are:

- Parkinson's Disease
- Alzheimer's Disease
- Aging-related Neurodegeneration
- Amyotrophic Lateral Sclerosis
- Multiple Sclerosis
- Psoriasis and Other Autoimmune Skin Diseases
- Inflammatory Bowel Diseases
- Inflammatory Arthritides
- Other Th17-mediated Autoimmune Diseases

Summary of IRX4204: A Phase II Clinical Stage RXR Agonist for Neurodegenerative and Autoimmune Diseases

- IRX4204 is a potent, highly selective RXR agonist with immunomodulatory, anti-inflammatory, myelin protective, myelin reparative, neuroprotective and neurorestorative activities.
- IRX4204 promotes T-reg differentiation; inhibits Th17 differentiation; inhibits Th17 production of proinflammatory IL-17s, TNFs; inhibits microglial production of IL-6 and proinflammatory chemokines; promotes differentiation of oligodendrocyte precursor cells into myelin-producing oligodendrocytes; promotes axonal protection and remyelination in vivo; promotes in vitro cortical neuron neurite outgrowth; promotes in vitro and in vivo survival of dopaminergic neurons; and inhibits beta amyloid deposition in Abeta transgenic mice.
- IRX4204 is effective in animal models of AD, PD, MS, adoptively transferred Th17 EAE, cuprizone-induced axonal transection and demyelination; db/db mouse model of diabetes; mouse GVHD.
- Orally administered IRX4204 is brain penetrant. IRX4204 can be developed as a nasally inhaled formulation for nose to brain delivery for CNS conditions. Topical formulations can be developed for treatment of diverse immunoinflammatory Th17-mediated diseases of skin, lung and eyes.
- 100 patients have been treated with oral IRX4204, including 15 with early-stage Parkinson's disease (PD), and 85 with various cancers.
- IRX4204 has been safe and well tolerated for up to 20 months of continuous treatment in 100 humans with cancers and Parkinson's disease.
- Open label clinical trial data supports potential clinical utility in Parkinson's disease and cancers.

Summary of IRX4204 Preclinical Pharmacology Relevant for Neurodegenerative and Autoimmune Diseases

- **Immunomodulatory**: increases Treg differentiation; decreases Th17 differentiation in vitro and in vivo; effective in acute and chronic GVHD in which it inhibits skin, lung, and GI manifestations.
- **Anti-inflammatory:** inhibits in vitro human T-cell production of IL-17s, TNFs, IL-9; and mouse microglia and macrophage production of IL-6, MIP-1α, MIP-2, COX-2, and nitric oxide.
- **Myelin Protective and Myelin Reparative:** promotes OPC differentiation into myelin-producing oligodendrocytes in vitro; is axon protective and myelin reparative *in vivo* in cuprizone models; combination effects with thyroid hormone on OPCs in vitro, and in cuprizone demyelination models.
- **Neuroprotective and Neurorestorative:** increases rat dopaminergic neuron survival in vitro and in vivo, alone and in combination with thyroid hormone; activates Nurr1 and increases expression of downstream genes DAT, DDC, and GDNFR; preserves dopamine content in dopaminergic neurons in vivo; increases in vitro cortical neuron survival and mitochondrial respiration; promotes cortical neuron neurite outgrowth in vitro alone, and in combination with thyroid hormone or insulin.
- **Insulin sensitizing:** reduces blood glucose in db/db mouse model of T2DM.
- **Effective in EAE mouse models of MS:** treats MOG-induced EAE, SJL EAE, and adoptively transferred Th1 or Th17 EAE; protects and restores motor function in EAE; protects myelin in vivo in adoptive Th17 and MOG EAE models; has combination treatment effects with thyroid hormone in MOG EAE.
- **Effective in 6-OH dopamine rat model of PD:** preserves motor functions, and TH+ dopaminergic neurons in vivo; has combination motor and neuroprotective effects with thyroid hormone.
- **Effective in TgCRND8 β amyloid transgenic mouse model of AD:** improves cognitive functions; decreases deposition of brain beta amyloid; restores long term potentiation in hippocampal slices; increases APO E expression in primary cultured neurons; inhibits Aβ peptide oligomerization.

IRX4204: Clinical Development Status

- 100 patients have been treated with IRX4204, including 85 with various cancers, and 15 with early Parkinson's disease.
- IRX4204 was well-tolerated for up to 20 months of continuous oral treatment.
- PK in humans is consistent with once per day oral dosing.
- Common side effects include dose related reversible suppression of TSH with resultant treatable hypothyroidism; mild to moderate reversible increased triglycerides; and mild to moderate reversible leukopenia.
- Open label uncontrolled phase I and II clinical trials in cancers and Parkinson's disease support clinical safety, efficacy, and effective doses.
- Synthesis of GMP clinical trial drug supply API is completed. Manufacture of drug and placebo capsules for Phase II trials completed.
- Planning phase II clinical trials in PD, psoriasis, and Her2+ breast cancer.

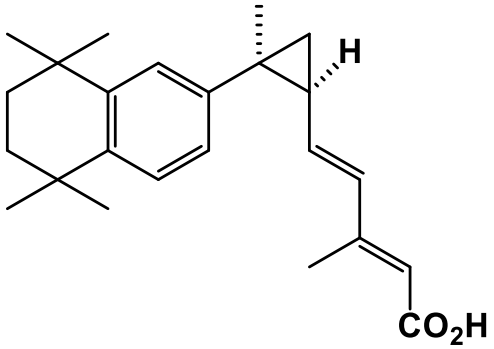
IRX4204: Product Development Status

- INDs for IRX4204 for treatment of cancers and Parkinson's disease GLP 6-month rat, 9-month dog toxicology completed; dose related ocular cataracts noted in rats and dogs; milder findings than in bexarotene toxicology (which also showed ocular cataracts)
- PK in rats, dogs and humans consistent with once daily oral dosing
- Brain penetrance demonstrated
- GLP gene toxicology panel negative
- GLP *in vivo* safety pharmacology panel (CNS, CV, Resp) negative
- GMP manufacturing completed on multi-kg scale; chemical and chiral purity 99+%
- Multi-year compound stability demonstrated
- Formulated in oral capsules for Phase II clinical trials
- Can be formulated for intravenous, inhaled nose to brain, inhaled lung, dermal, and ocular administration

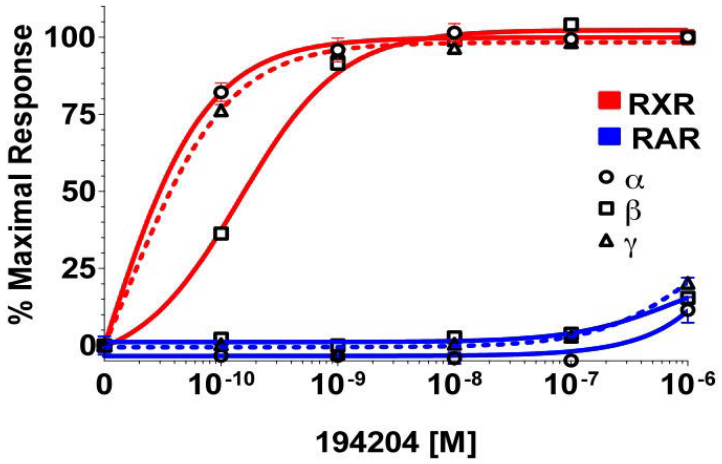
Proprietary IRX4204 Synthesis Methods and Formulations

- IRX4204 has two chiral centers and four enantiomers; only one enantiomer has the desired RXR agonist pharmacologic activities.
- The company developed and owns issued US and international patents for proprietary IRX4204 synthetic processes; and for specific chemical structures of multiple IRX4204 synthetic intermediates. The proprietary process and intermediates are usable for multi-kilogram commercial scale manufacturing of >99% chemically pure and >99% enantiomerically pure IRX4204 GMP API, without requiring a terminal chromatographic purification, at low cost of goods. The company believes these patented methods of synthesis and compound intermediates provide a substantial barrier to manufacturing generic versions of IRX4204 without patent infringement.
- The company discovered and has patented specific salts and polymorphs of IRX4204 with substantially increased aqueous solubility as New Chemical Entities. These are suitable for developing IRX4204 salts and polymorphs for oral, inhaled nose to brain, inhaled mouth to lung, and topically applied dermal and ocular formulations for diverse clinical indications.
- Patents for methods of synthesis are issued or pending in US, EP, and over 20 other non-EP countries. Patents for salt/polymorph NCEs are PCT applications and will be nationalized widely in PCT and a select non-PCT countries.

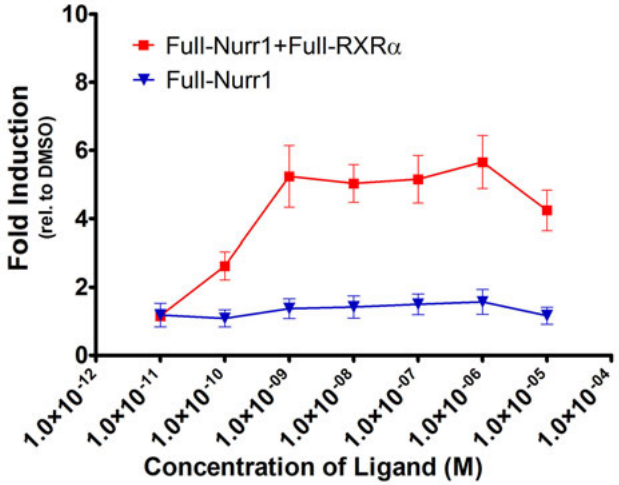
IRX4204 Activates RXR and Nurr1, But Not RAR Nuclear Receptors at Nanomolar Concentrations



IRX4204 – A Potent RXR Homodimer Agonist with no RAR Activity at nM Concentrations

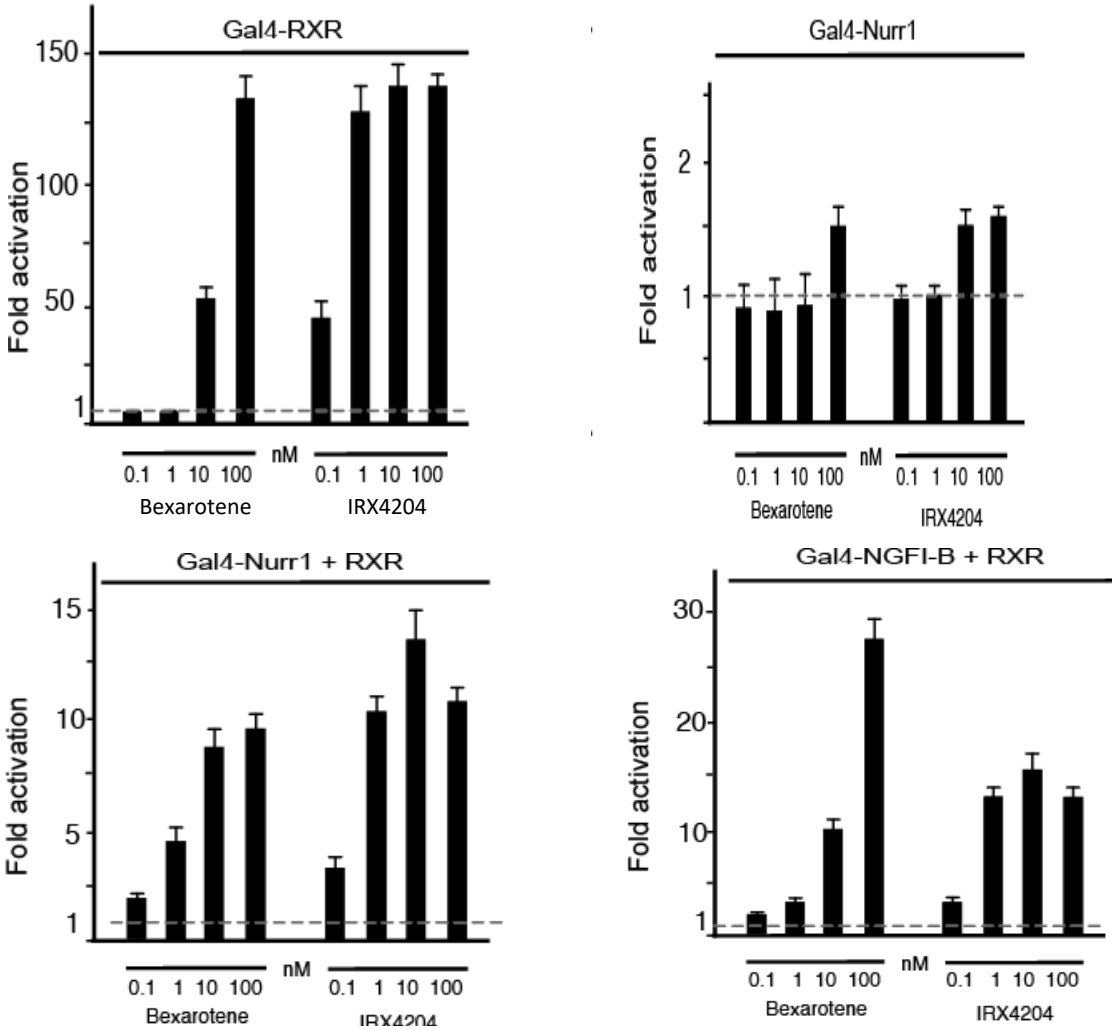


IRX4204 Transactivates Nurr1/RXR Heterodimers through RXR



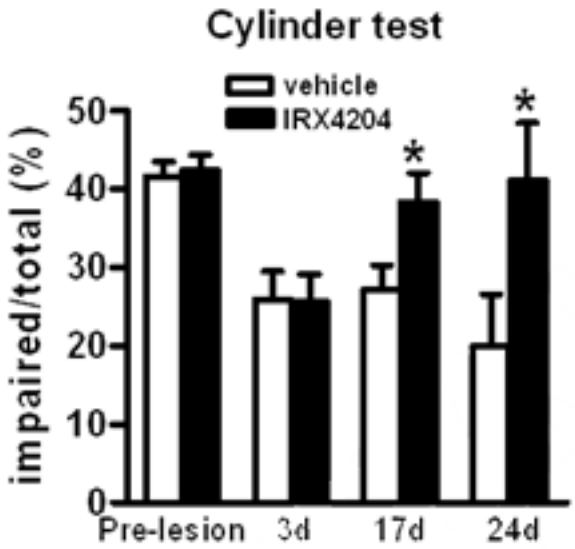
Parkinson's Models and Dopaminergic Neuron Protective Effects of IRX4204

Reporter Assays with RXR Ligands Demonstrate IRX4204 is More Potent Than Bexarotene for Activation of RXR, Nurr1, and NGFI-B (Nur77)



Data provided by
Thomas Perlmann
Karolinska Institute
Stockholm, Sweden

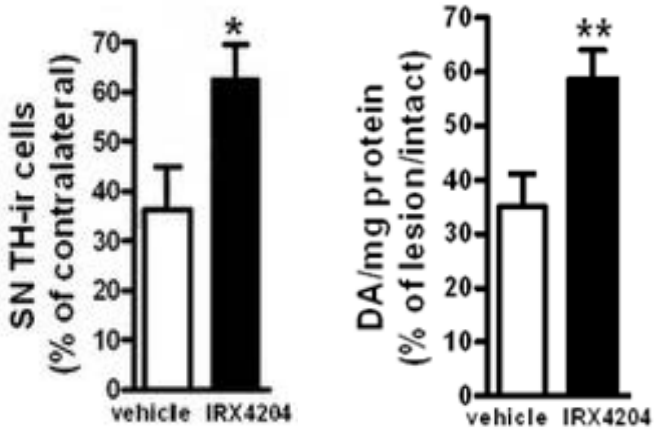
IRX4204 Reverses Asymmetric Impairment in Limb Usage Induced by 6-OHDA Lesions in Rat PD Model



Cylinder Test (paw placement test, rearing test, limb use asymmetry test)

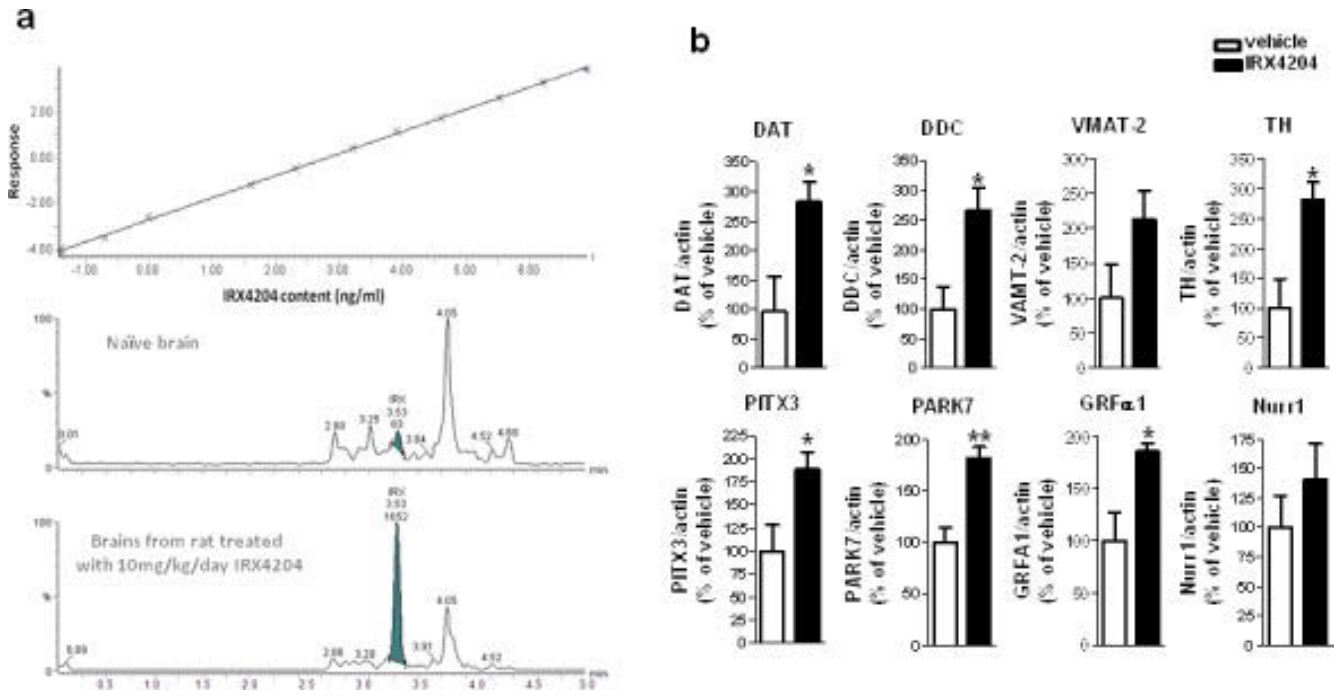
Detects forelimb impairments, specifically asymmetry in limb use for weight bearing movements when leaning on walls of glass cylinder during exploration of a novel environment. Compares use of the affected side to the unaffected side, therefore each animal serves as its own control.

IRX4204 Treatment Protects Against Loss of TH-Positive Midbrain Neurons and Reduces Loss of Dopamine in 6-OHDA Lesioned Rat PD Model



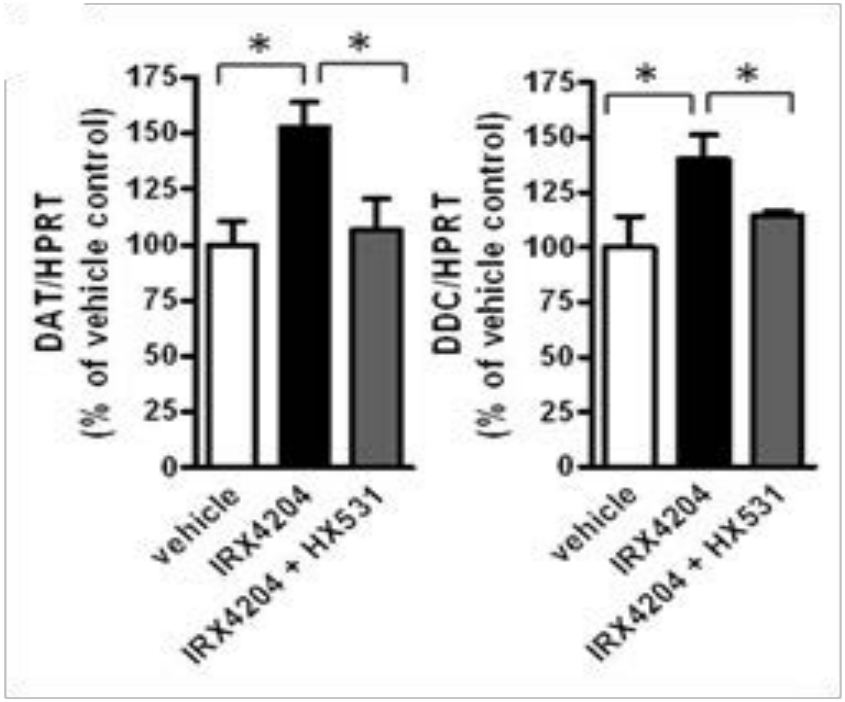
Rats were stereotaxically injected with 6-OHDA (40µg in 2µl) into right striatum. Rats were treated with IRX4204 (10mg/kg/d) or vehicle by oral gavage for 21 days starting 3 days post-lesion.

IRX4204 is Bioactive in Rat Brains Following Oral Administration and Activates Nurr1 Downstream Genes in the Substantia Nigra

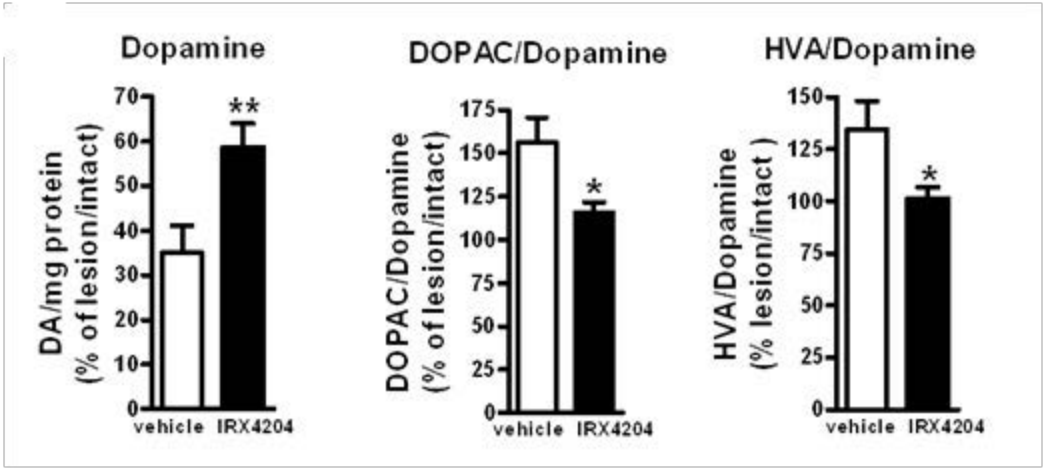


a. Detection of IRX4204 in the brains: Dose-response calibration curve of IRX4204 detection in the brain matrix with detection limit of 0.25nM (top panel); Representative IRX4204 LC/MS/MS chromatograms from brains of vehicle and IRX4204 treated rats (middle and bottom panel) **b.** Expression of Nurr1 downstream genes in the SN following 7 days oral treatment with 10 mg/kg/day IRX4204. Real-time PCR data are expressed as the percentage of vehicle treated controls.

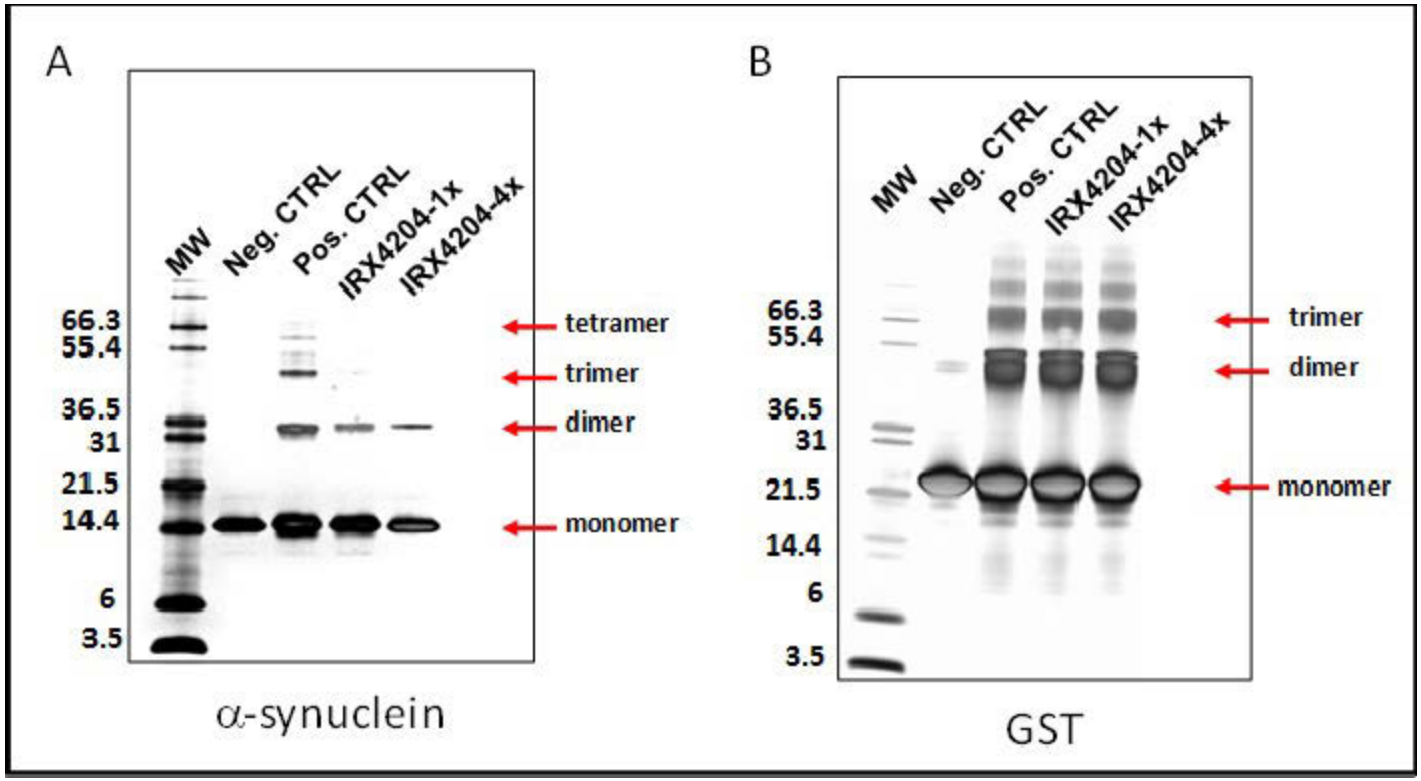
IRX4204 Induces Expression of Nurr1/RXR Regulated Genes DAT and DDC in Cultured Primary Ventral Midbrain Neurons in an RXR Dependent Manner



IRX4204 Treatment Reduces Loss of Dopamine and Reduces Dopamine Catabolism in 6-OHDA Lesioned Rats

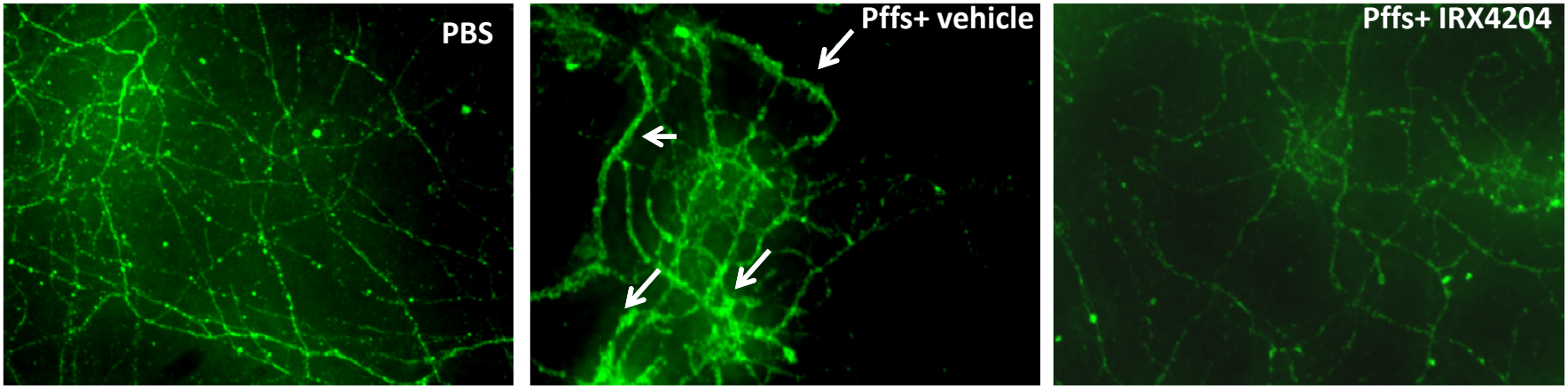


IRX4204 Interferes with Alpha-Synuclein Oligomerization in Photo-induced Cross-Linking of Unmodified Proteins (PICUP) Assay



SDS-PAGE of (A) alpha synuclein and (B) glutathione synthetase (GST) crossed-linked in the presence or absence of 4X molar excess of the indicated compounds. Negative controls are non-cross-linked alpha-synuclein or GST. Positive control is aggregated alpha-synuclein or GST.

IRX4204 prevent α-synuclein pre-formed fibrils (Pffs)-induced fibrillar-like structure formation



Primary neurons were treated with human α-synuclein pre-formed fibrils (Pffs) for 14 days and fixed with 4% PFA+1% triton 100. The cells were immunostained with anti-α-synuclein antibody. In PBS treated neurons, endogenous mouse α-synuclein localized to presynaptic puncta while in pffs-treated neurons, α-synuclein formed fibrillar-like structures. Treatment of IRX4204 prevented the formation of fibrillar-structure induced by Pffs.

Phase I/II Clinical Trial in Early Parkinson's Disease

Mean Changes from Baseline to Week 2 in Total Motor Score, UPDRS, and TSH Indicate CNS Effects and Support Potential for Clinical Efficacy of IRX4204 in PD

Dose	Total Motor Change*^	Total UPDRS Change*^	Changes in TSH
20 mg/day	-14.5%	-6.6%	-98.8%
10 mg/day	-11.7%	-13.6%	-36.6%
5 mg/day	-31.4%	-18.7%	-28.9%

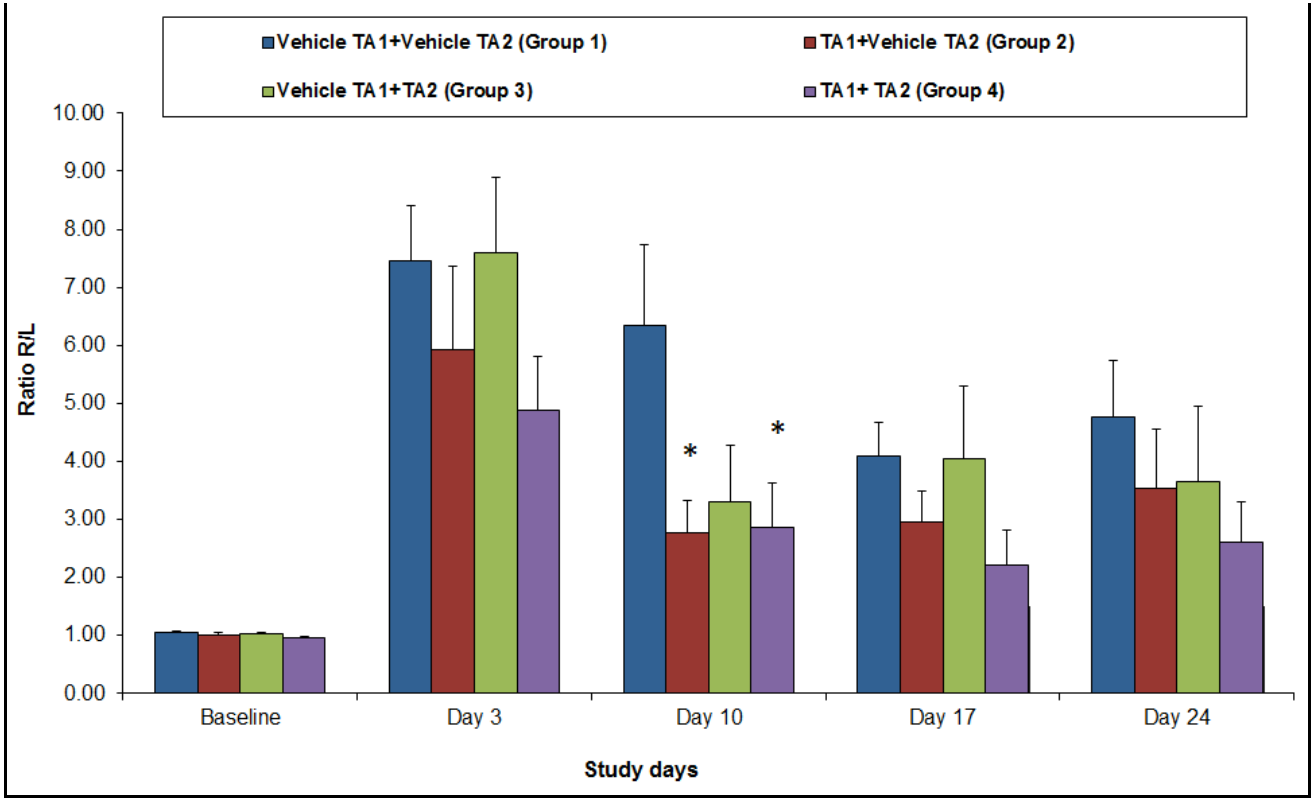
*Reduction in UPDRS and Total Motor scores represent clinical improvement

^Mean changes, n=5 per group

TSH Reduction is a class effect of RXR agonists, caused by inhibition of hypothalamic TRF synthesis; and is a biomarker of brain penetrance of IRX4204.

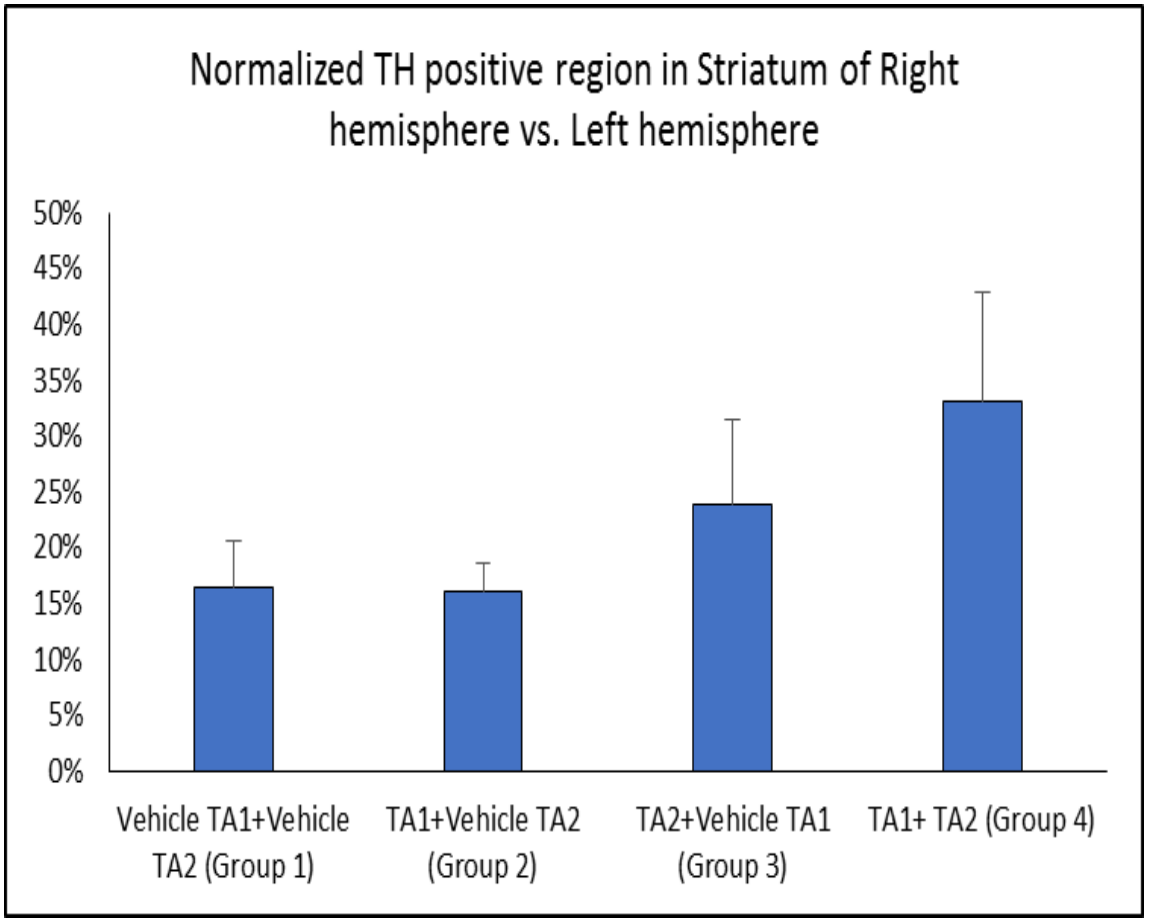
Clinical trial conducted at Molecular Neuroimaging, Inc.

Thyroid Hormone Potentiates Effects of IRX4204 on Motor Function in the 6-OH Dopamine Rat PD Model



TA1 IRX4204 10 mg/kg/d po, TA2 Thyroid Hormone sc

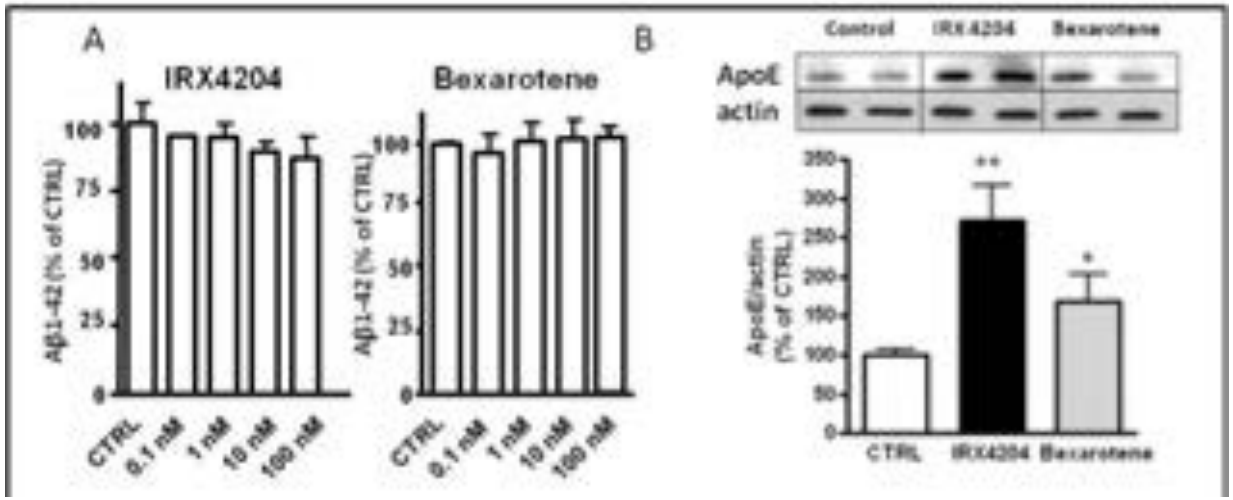
IRX4204 + Thyroid Hormone Have Combination Protective Effects on TH Positive Striatal Neurons in the 6-OH Dopamine Rat PD Model



TA1 IRX4204 10 mg/kg/d po, TA2 Thyroid Hormone sc

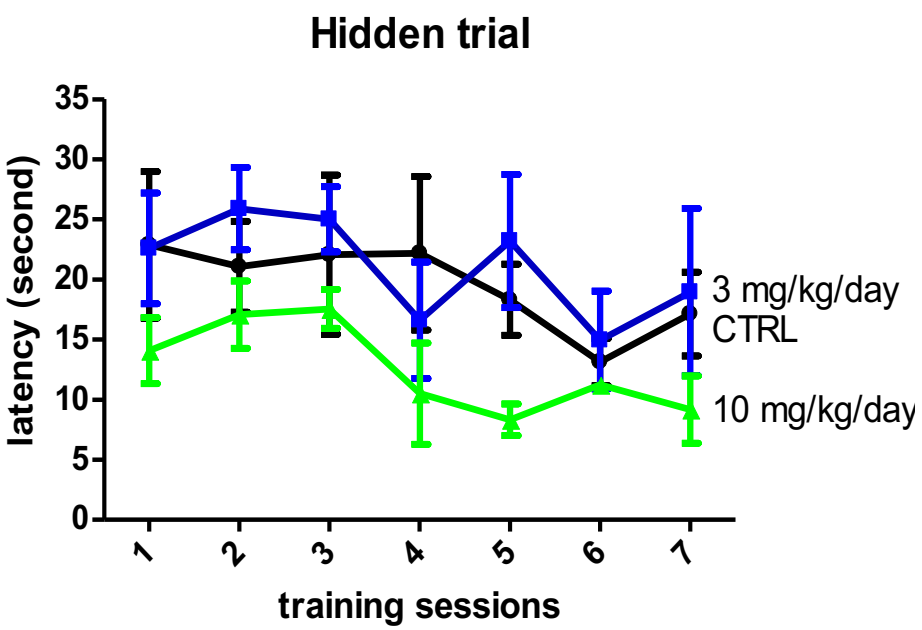
Alzheimer's Disease Models and Cortical Neuron Protective Effects of IRX4204

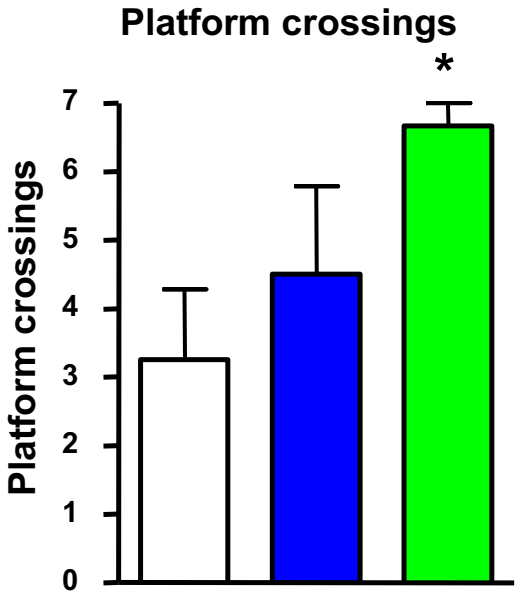
IRX4204 Decreases Beta Amyloid Expression and Increases ApoE Expression in Primary Cultured Mouse Neurons



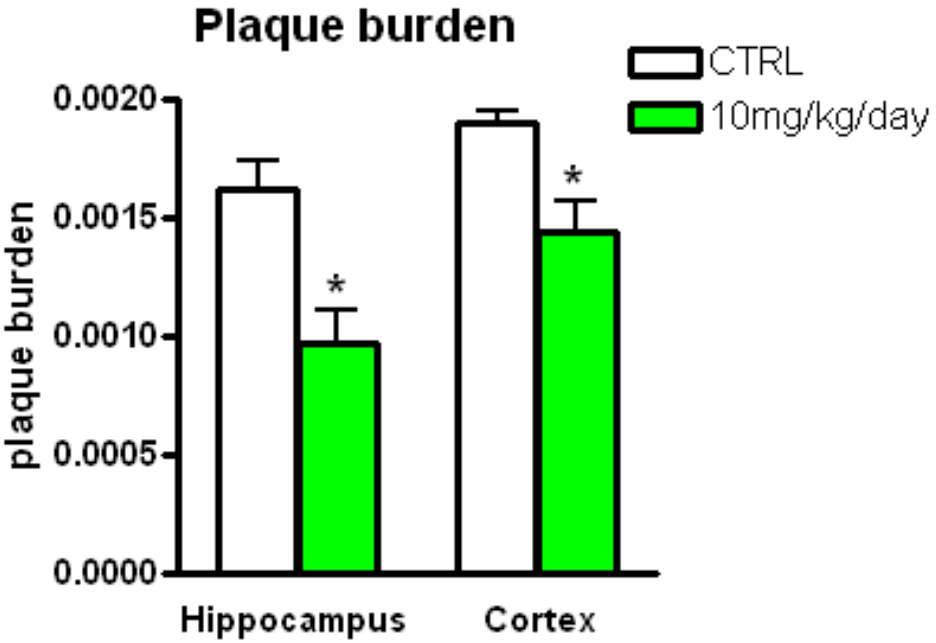
Beta Amyloid peptide was assayed in cultured cortico-hippocampal neurons from heterozygous TG2576 transgenic mouse primary neurons after 16 hours of culture in indicated concentrations of IRX4204 or bexarotene (Panel A). Primary cortico-hippocampal neurons from C57B6 wild type mice were cultured with 100 nM of IRX4204 or bexarotene, and total proteins were isolated and examined by Western Blot analysis for ApoE expression.

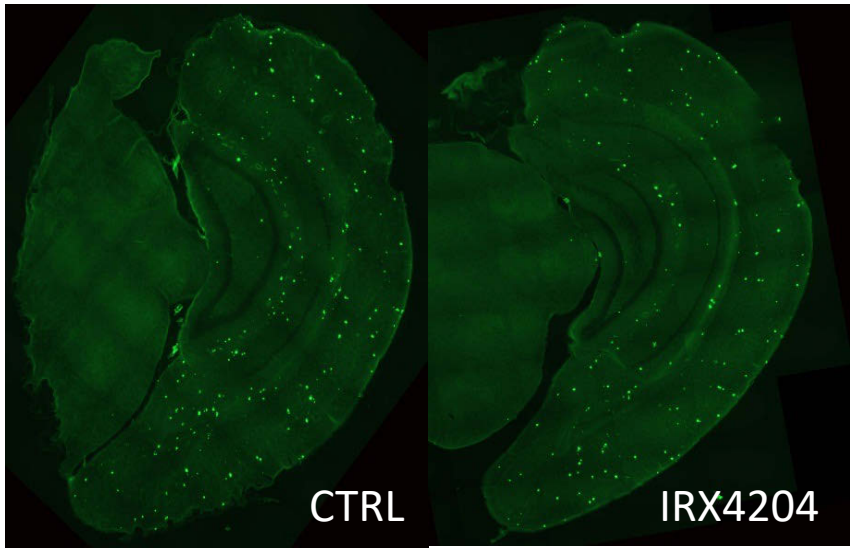
Oral Administration of IRX4204 Attenuates Cognitive Deterioration in TgCRND8 AD Model in Mice as Assessed by Morris Water Maze Test



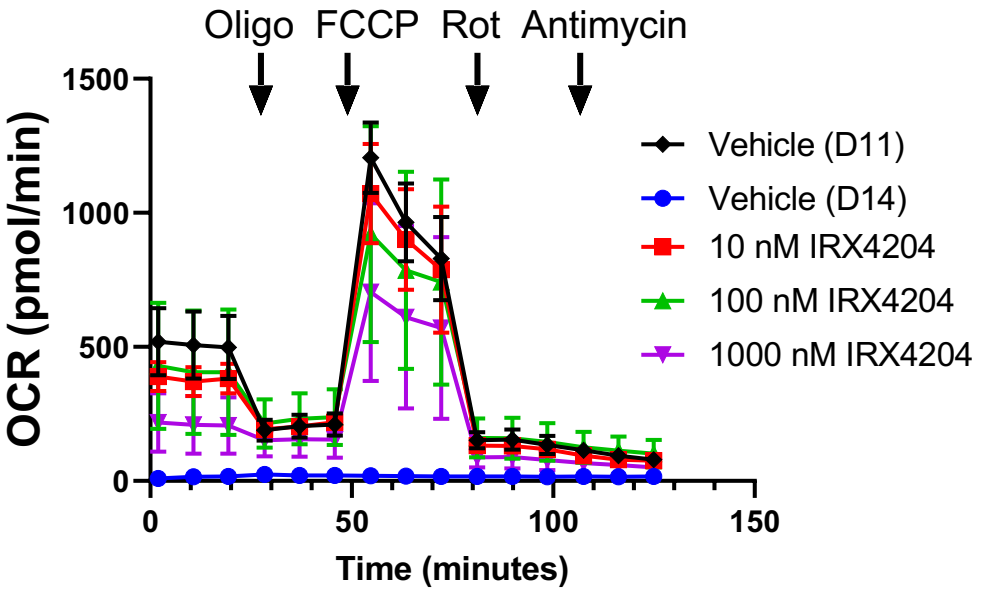


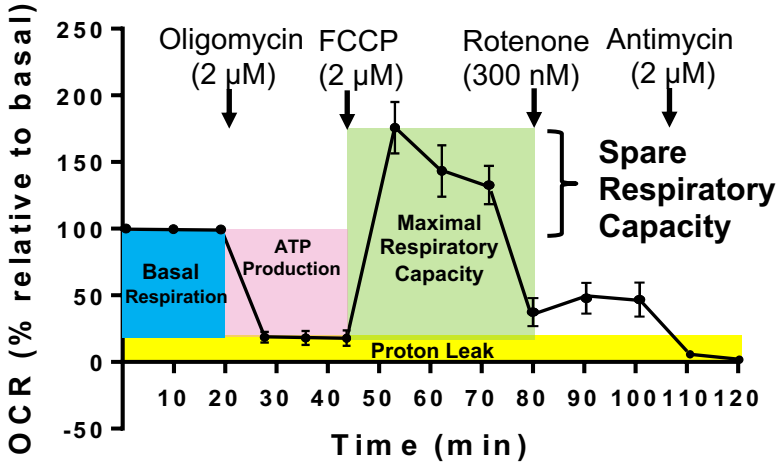
Oral Administration of IRX4204 Attenuates Amyloid Neuropathology in TgCRND8 AD Model in Mice





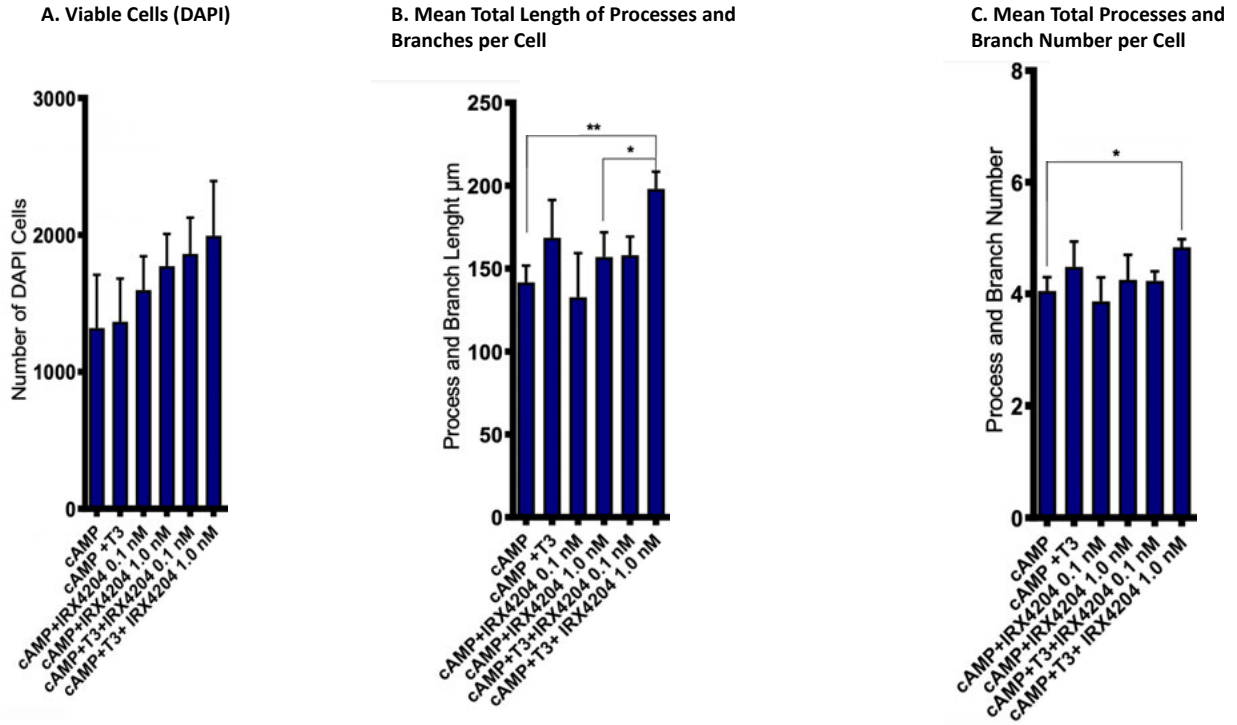
IRX4204 Preserves and Increases Mitochondrial Respiration in Cultured Murine Cortical Neurons *in Vitro* in Seahorse Assay





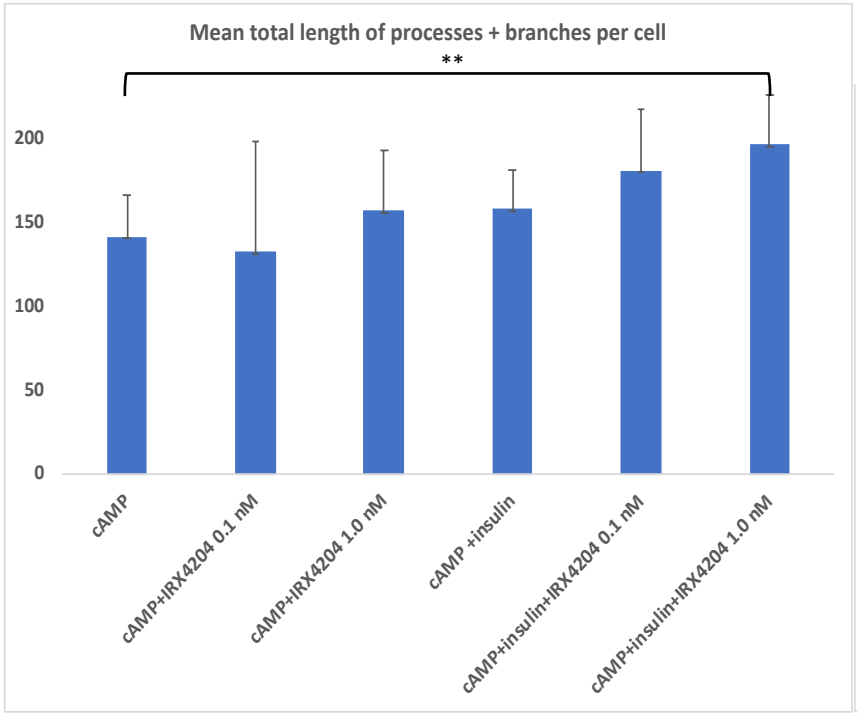
IRX4204 prevented the loss of mitochondrial respiration caused by culturing cortical neurons for a prolonged time (left panel). Relative to *in vitro* day 11 (D11) primary cultures of mouse cortical neurons treated with vehicle (0.01% DMSO), D14 cortical neurons showed a profound loss of mitochondrial respiration. Treatment with IRX4204 (10, 100 or 1000 nM) from D11-14 (72 hr drug treatment) prevented this loss of mitochondrial respiration. Relative to D14 vehicle treated cultures, IRX4204 elevated basal respiration, ATP production and spare respiratory capacity. Seahorse XF24 extracellular flux instrumentation was used to assess basal respiration, ATP production and spare respiratory capacity (right panel). Each data point and bar represent the mean ± SD for 6-8 biological replicates.

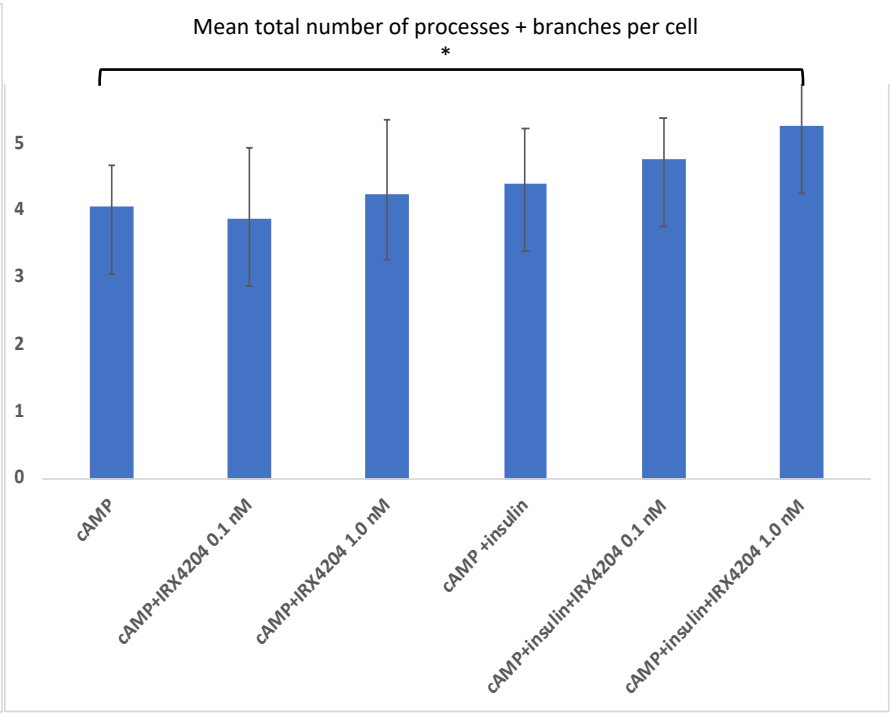
IRX4204 + T3 Have Combination Effects on Rat Cortical Neuron Survival and Neurite Outgrowth in Vitro



Data from 48 hour cultures of rat cortical neurons, supplemented with cyclic adenosine monophosphate (cAMP) as a metabolic energy source. DAPI is a stain that identifies viable cells. Panel A shows that IRX4204 alone, and in combination with thyroid hormone, increased mean survival of treated cortical neurons. Panels B and C show data from an automated imaging and quantitation system for assessing outgrowth of neurites. The combination of IRX4204 plus thyroid hormone statistically significantly increased the length of neurites relative to cAMP alone (**p<.01); and IRX4204 alone (*p<.05); and increased the total number of neurites per cell relative to cAMP alone (*p<.05).

IRX4204 + Insulin Have Combination Effects on Rat Cortical Neuron Neurite Outgrowth in Vitro

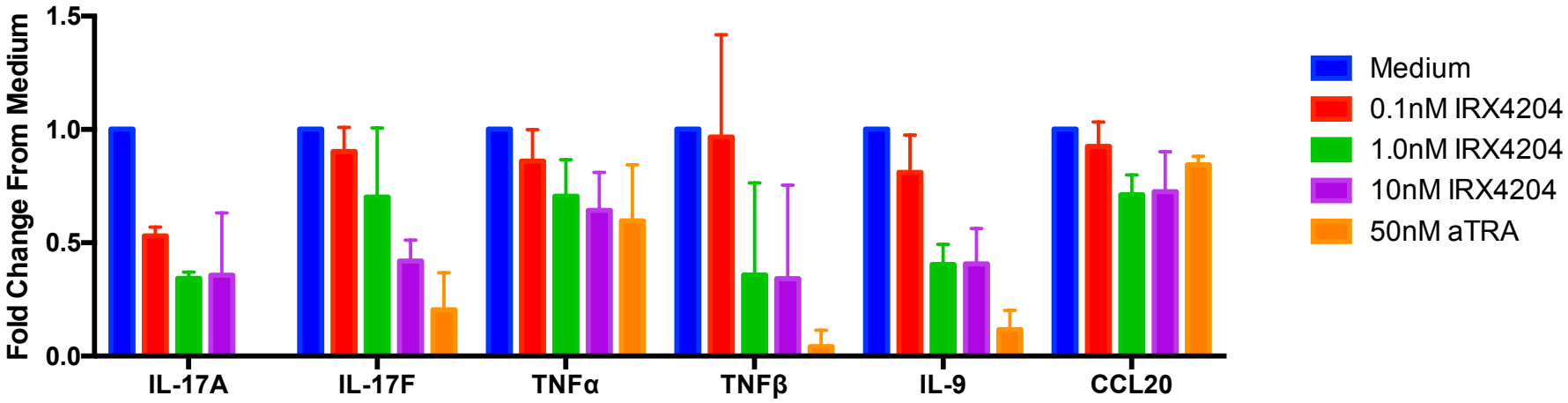




Data are means ± SEM, N=6 per group.
Statistical significance (Welch's t-test): ** p< 0.01, * p< 0.05.

Autoimmune Diseases, Treg and Th17 Effects of IRX4204

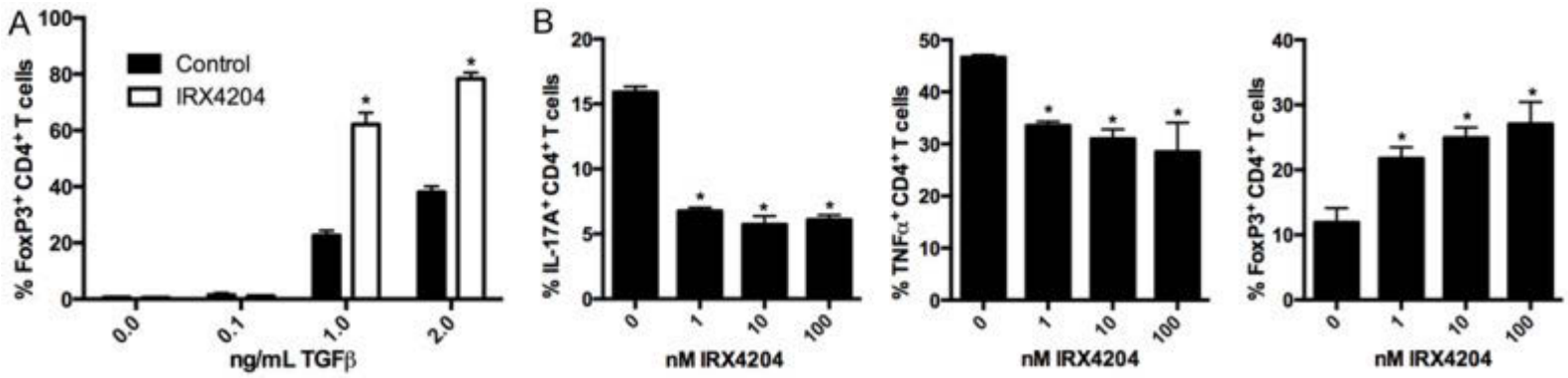
IRX4204 inhibits Th17-associated cytokine secretion by human T-cells cultured in Th17 skewing conditions



Normal Human PBMCs were isolated from whole blood, then magnetically separated to enrich a ~95% pure CD4+CD45RA+ naïve T cell fraction. Cells were cultured under TH17 skewing conditions for 5 days and stimulated anti-CD3/CD28 beads. and supernatants collected following 5 days in culture.

Therapeutic agents which are bioactivity neutralizing for IL-17 and TNF-α are effective in randomized controlled clinical trials, and FDA approved, for treatment of rheumatoid arthritis, psoriasis, psoriatic arthritis, ankylosing spondylitis, Crohn's disease, ulcerative colitis, and uveitis; validating IL-17 and TNF-α as therapeutic targets for diverse autoimmune diseases. The preclinical data shown above, using human T-cells in vitro, provides strong support for potential clinical utility of IRX4204 for treatment of these same IL-17 and TNF-α mediated diseases.

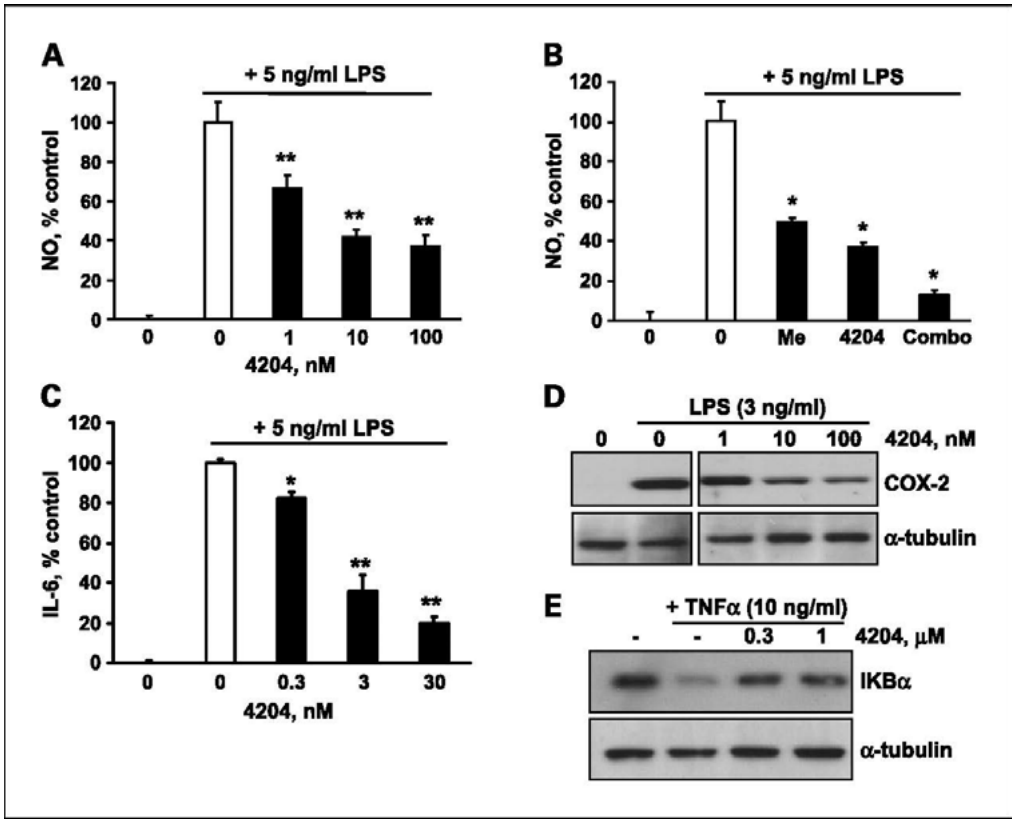
IRX4204 promotes iTreg and inhibits Th17 differentiation *in vitro* in Mouse CD4+ T-cells



Naïve $CD4^+$ T cells were cultured under iTreg (A) or Th17 (B) differentiation conditions after re-stimulation and graphed as Mean ± SD. Asterik (*) indicates the group is statistically significant from the controls. Results are representative of 3 independent experiments. for four days in the presence of vehicle control (0 nM group) or IRX4204 at 100 nM or indicated concentration. At the end of culture cells were assessed for FoxP3 expression and cytokine production.

Chandraratna, et al.
Am J Transl Res, 2016

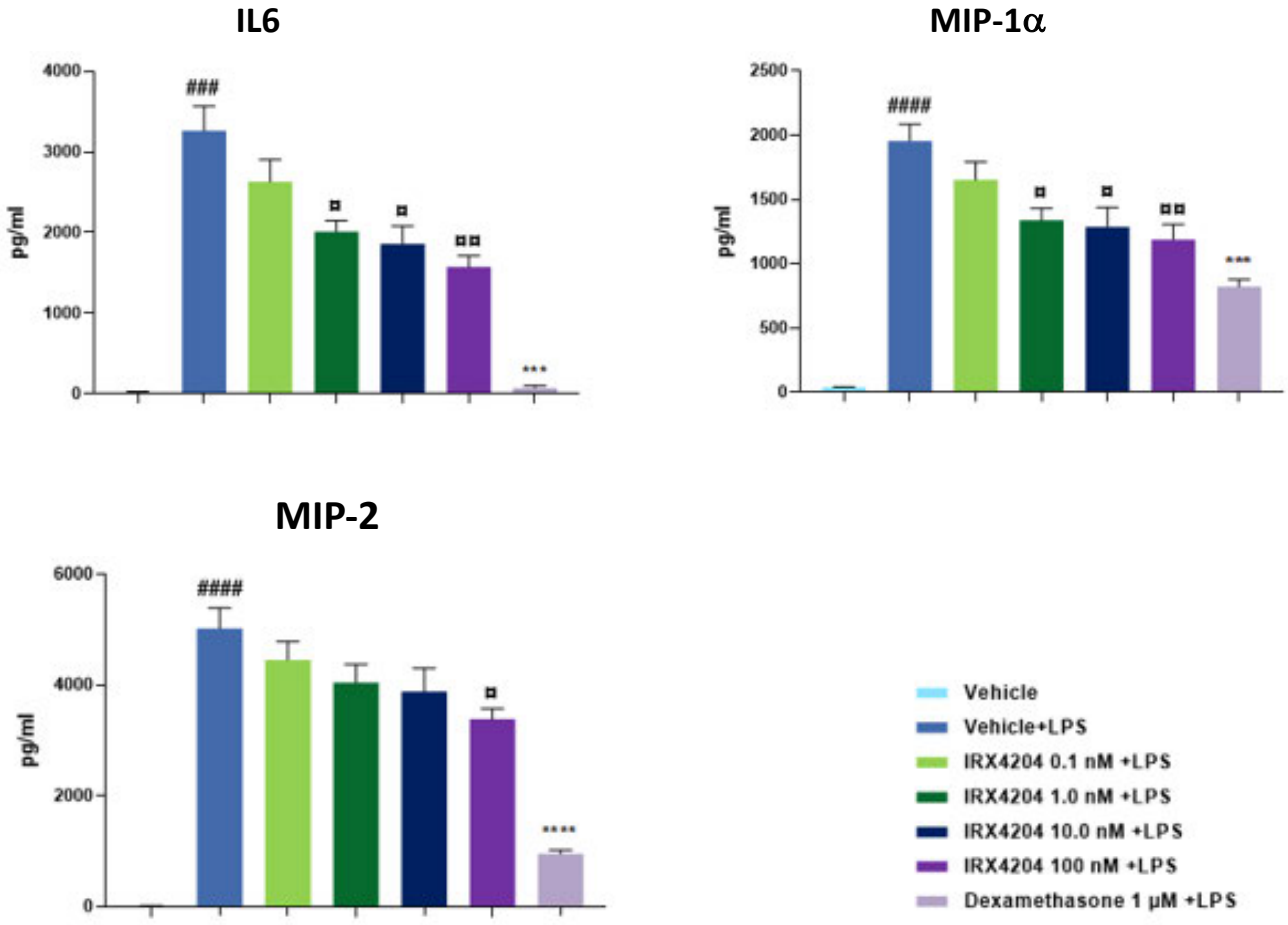
IRX4204 Inhibits Macrophage IL-6 and Nitric Oxide Production



Liby, et al.
Clin Cancer Res 2007

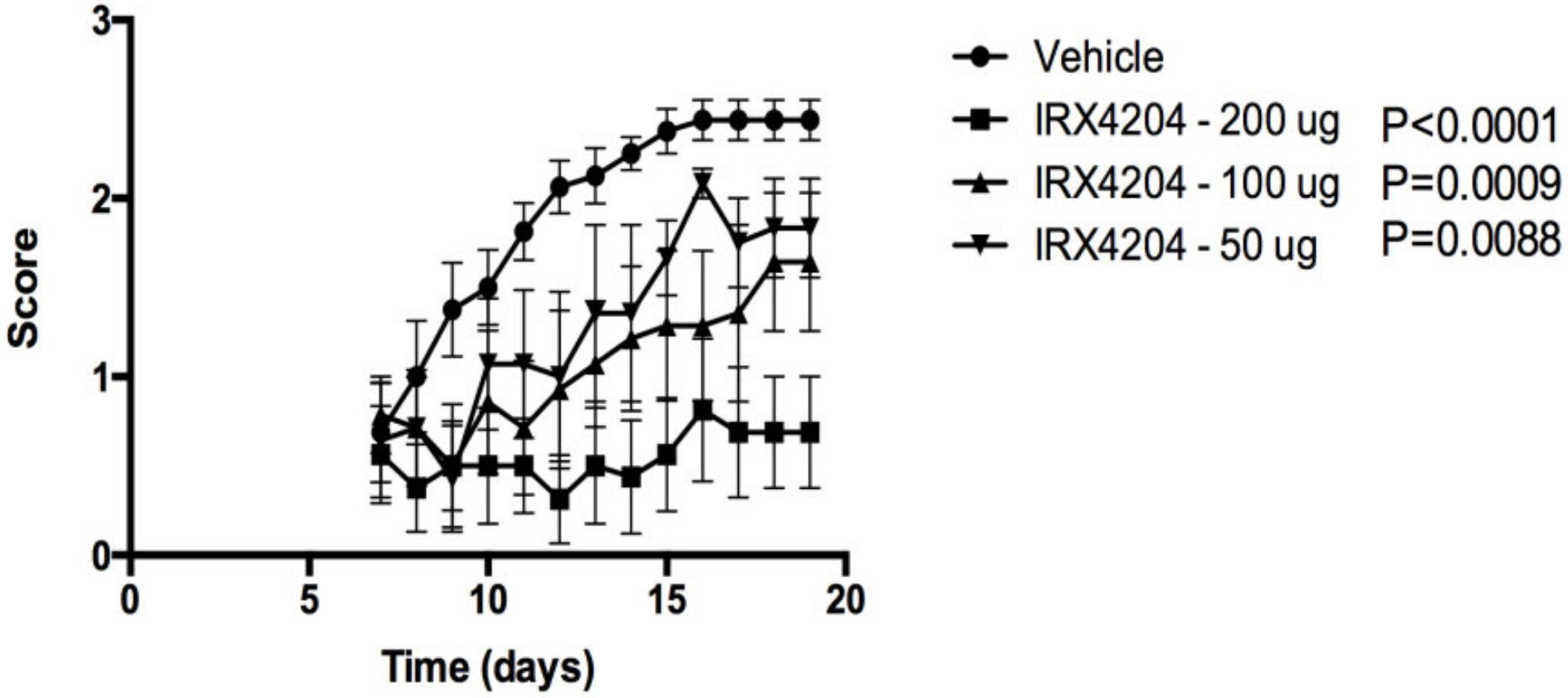
4204 inhibits the effects of lipopolysaccharide and tumor necrosis factor-α. RAW264.7 mouse macrophage–like cells were treated either with NRX194204 (4204) alone (A) or with 4204 (100 nmol/L) alone and in combination (B) with CDDO-Me (1 nmol/L) and then stimulated with lipopolysaccharide (LPS) for 24 h. The Griess reaction was used to measure nitric oxide in the medium. RAW cells were also treated with 4204 and lipopolysaccharide for 24 h (C and D), and either the amount of interleukin 6 released into the media was measured using an ELISA (C) or cell lysates were immunoblotted with cyclooxygenase-2 antibodies (D). E, RAW cells were treated with 4204 for 24 h, the cells were then stimulated with tumor necrosis factor-α for 15 min, and cell lysates were immunoblotted using IKBα antibodies. *, $P < 0.05$ versus control; **, $P < 0.001$ versus control.

IRX4204 inhibits LPS-induced rat cortical microglial production of IL-6 and pro-inflammatory chemokines



Cultures were 80% purified microglia, 20% astrocytes

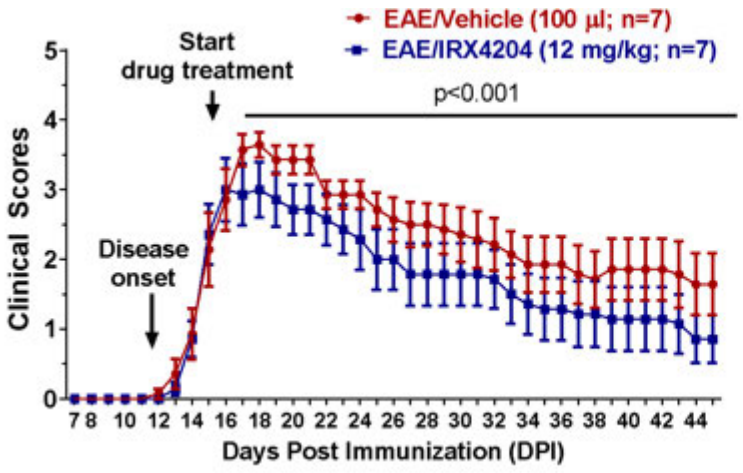
IRX4204 delays and inhibits disease in EAE mouse models of MS



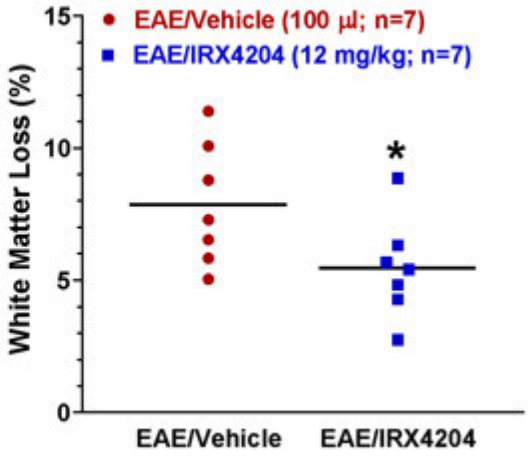
B6 Mouse Model: IRX4204 administered i.p. every other day starting day 7 after immunization.

IRX4204 Reduces Myelin Loss in MOG-Induced EAE

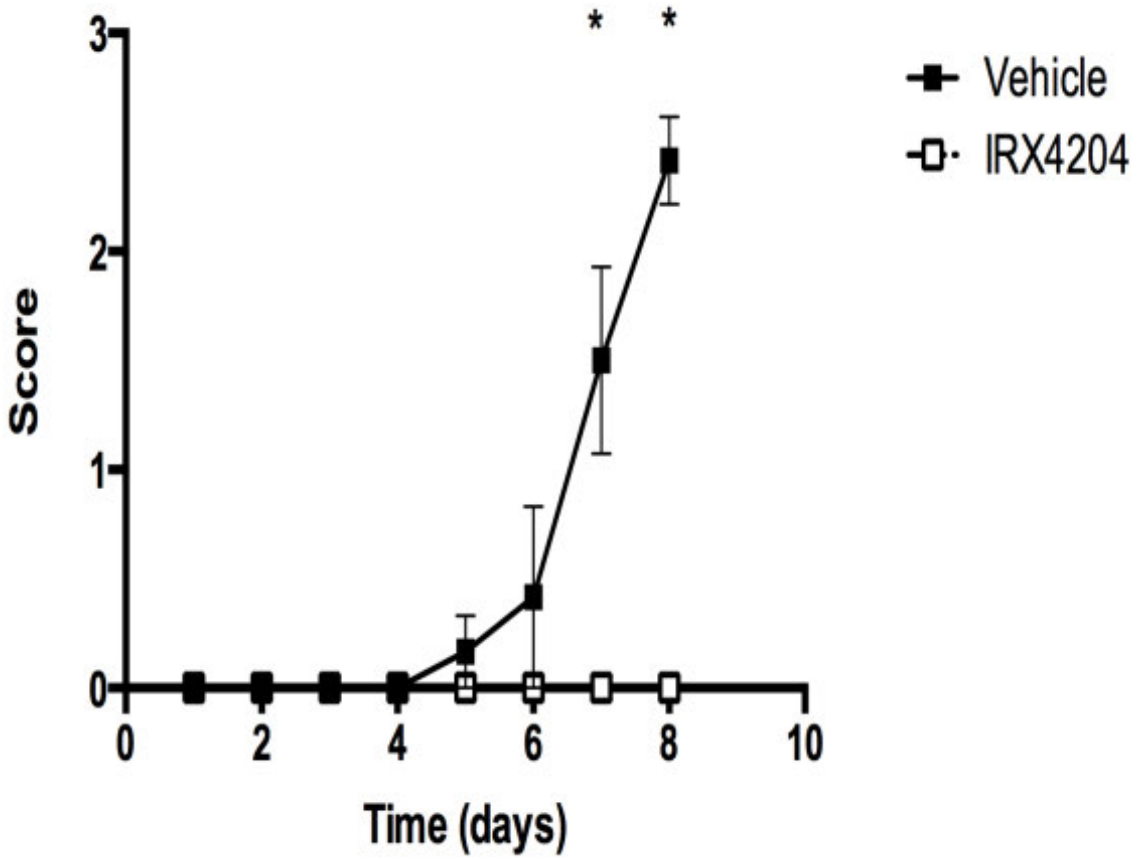
IRX4204 Reduces EAE Disease Severity



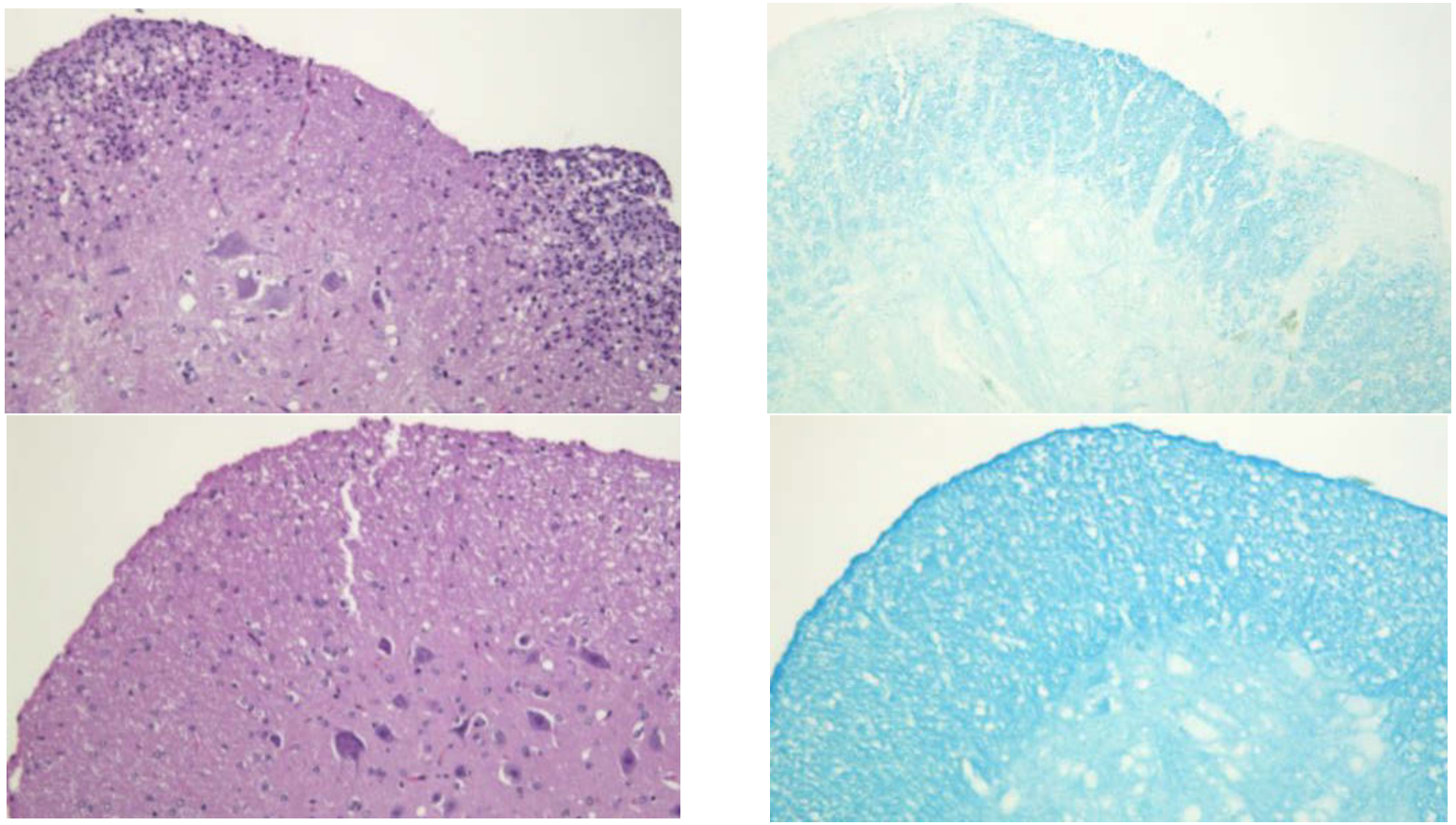
IRX4204 Reduces Spinal Cord White Matter Loss in EAE Mice



IRX4204 is 100% Effective in an Adoptive Transfer Model of EAE Using MOG-sensitized Th17 T-Cells



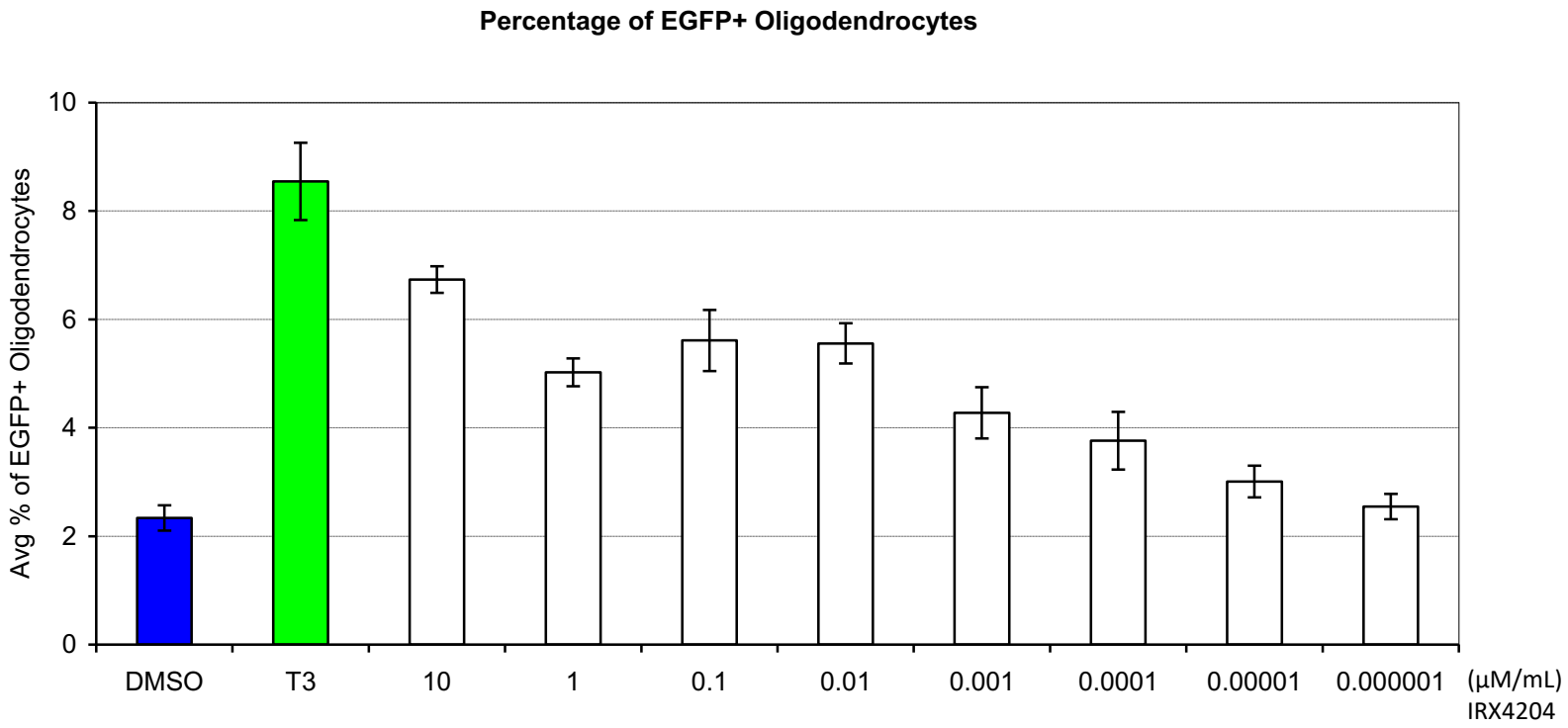
IRX4204 Inhibits CNS Infiltration of Leukocytes and Loss of Myelin in Th17 Adoptive Transfer EAE Model



Brain tissues from the Th17 adoptive transfer EAE model were stained for detection of infiltrating leukocytes (with hematoxylin and eosin, left), and integrity of myelin (with Luxol Fast Blue, right); IRX4204 treated animals (lower), vehicle treated animals (upper).

Oligodendrocyte Differentiation, Axon Protection, and Myelin Repair Effects of IRX4204

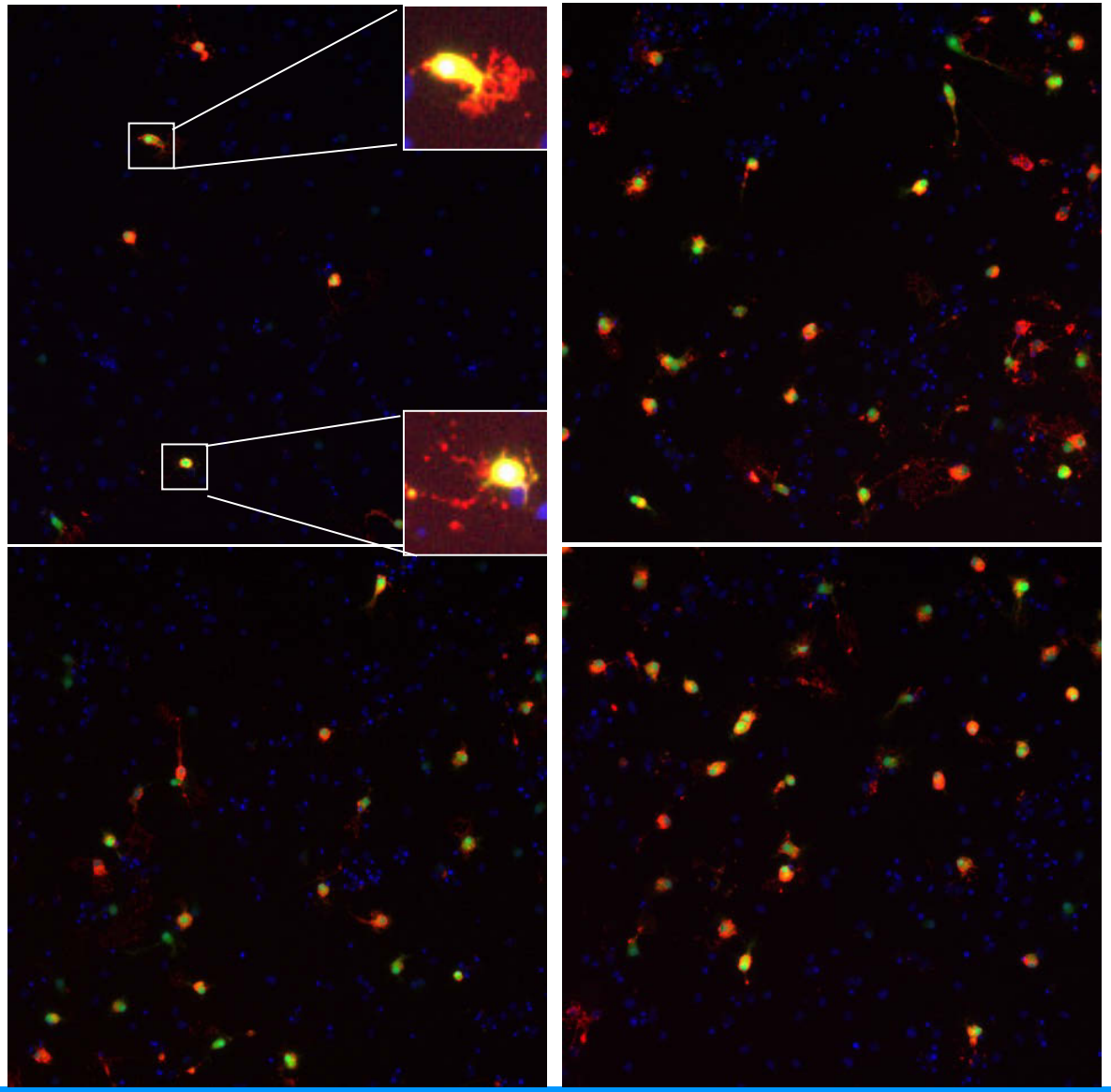
IRX4204 Promotes Differentiation of Oligodendrocyte Precursor Cells into Myelin Producing Oligodendrocytes



MBP/PLP Overlay Images Show IRX4204 Differentiated OL Produce Myelin

DMSO

T3 (0.1 ng/ml)



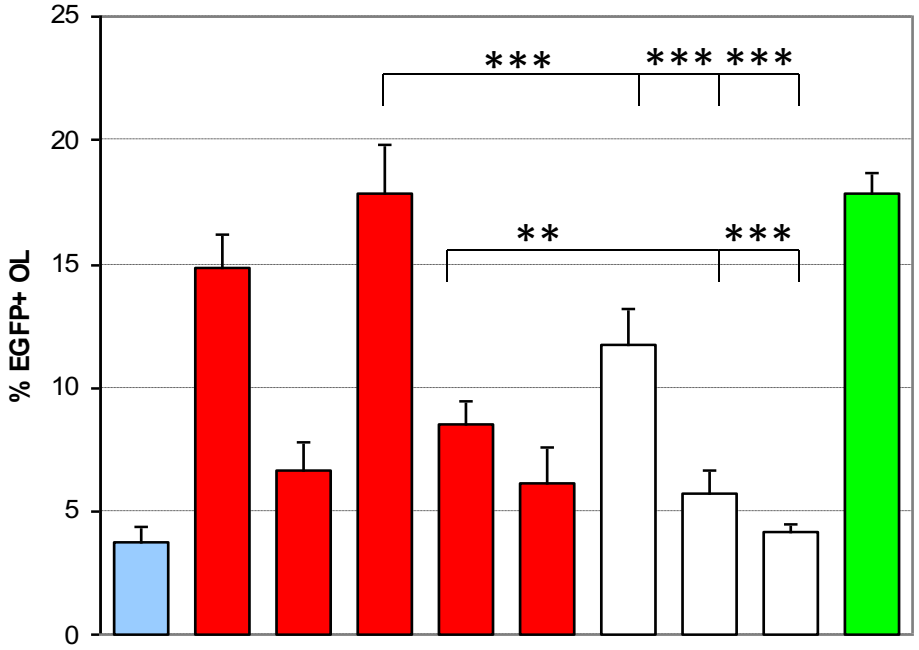
EGFP+ OLs
MBP+ OLs
Nuclei

IRX4204
10 nM

IRX4204 (10nM)
T3 (0.1ng/ml)

IRX4204 and Thyroid Hormone T3
Have Additive or Synergistic Effects on OPC Differentiation in Vitro

Percent of EGFP+ Oligodendrocytes

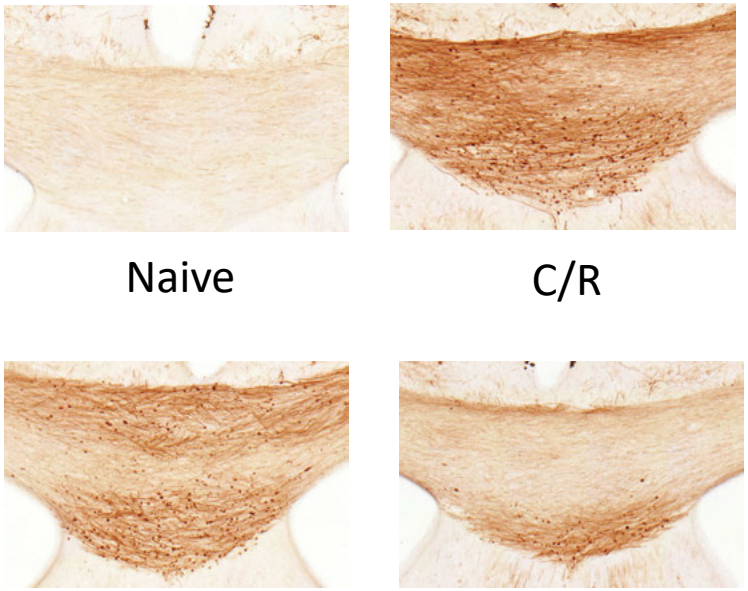


** P = < 0.01
*** P = < 0.0001

Col	2	3	4	5	6	7	8	9	10	11	
IRX	0	10uM	1nM				0	0	0		
T3	0	0	0	0.1	0.01	0.001	0.1	0.01	0.001	10	ng/ml

IRX4204 + Thyroid Hormone Have Combination Axonal Protective Effects in Murine Model of Cuprizone-induced Axonal Transection

SMI-32+ Ovoids are Transected Axons – Represetative Images of Corpus Callosum

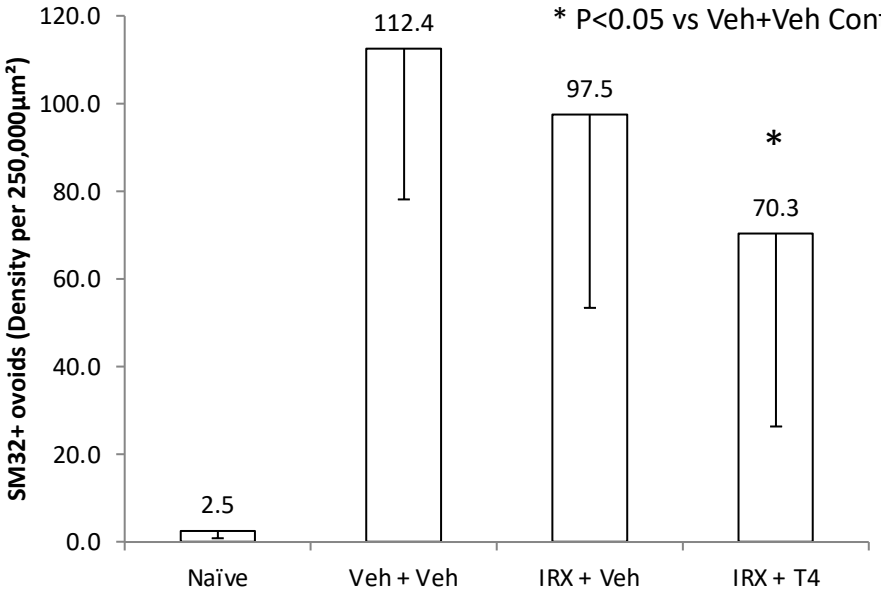


Naive C/R

C/R IRX4204 C/R IRX4204, T4

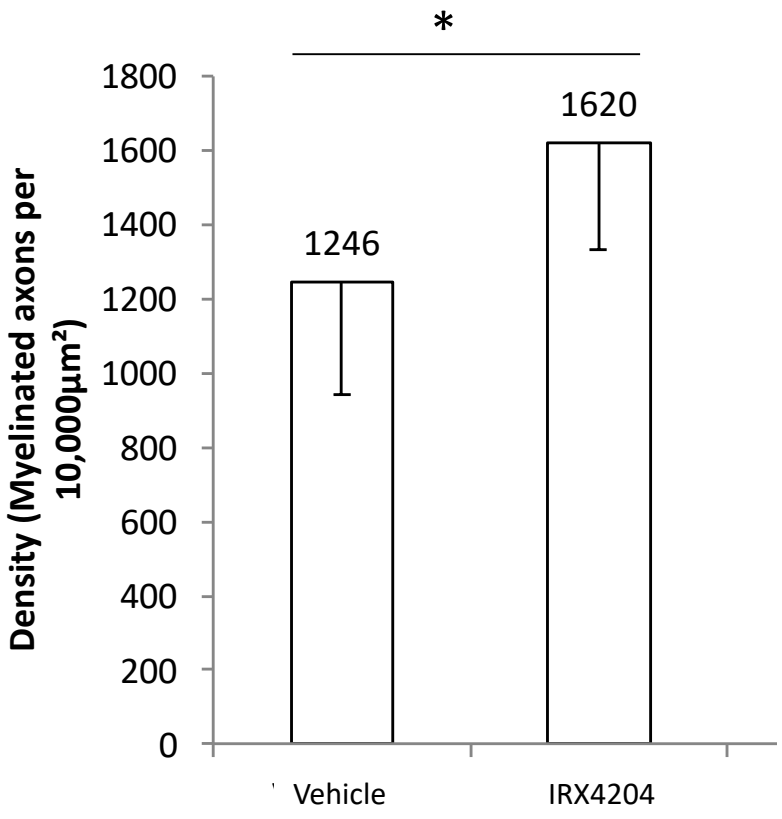
Axonal Transection Induced by Cuprizone (C) plus Rapamycin (R)

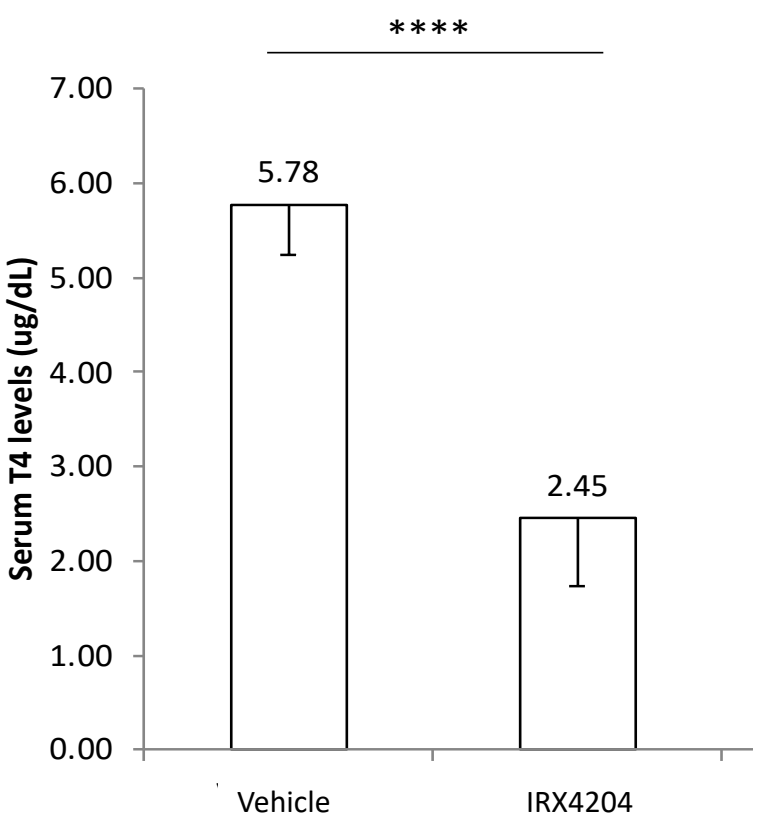
SMI-32 Quantification of Transected Axons



There was a significant reduction in corpus callosal axonal transection as shown by the number of SMI-32 positive ovoids in the IRX4204+T4 treatment group when compared to vehicle.

IRX4204 Promotes Remyelination in Mouse Model of Cuprizone-induced Chronic Demyelination





IRX4204 treatment resulted in a 30% statistically significant increase in myelin relative to vehicle ($p<0.05$), notwithstanding inducing highly significant hypothyroidism.
Study done at Renovo Neural, Inc.

Collaborators and Acknowledgements

Elizabeth Nowak, Randy Noelle; Dartmouth University
Murine Treg and Th17 differentiation studies; EAE models; EAE model of combination treatment with IRX4204 + thyroid hormone

Daniel Mielcarz, Jacqueline Channon; DARTLab, Dartmouth University
Human Treg and Th17 differentiation and Th17 production of proinflammatory cytokines studies

George Robertson; Dalhousie University
EAE models; myelin protection in EAE; neuronal mitochondrial respiration studies

Jun Wang, Giulio Pasinetti; Mount Sinai School of Medicine
Alzheimer's and Parkinson's disease models; studies of IRX4204 effects on α–synuclein, β-amyloid, and tau proteins

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OPC differentiation in vitro studies; axonal protection and remyelination studies in cuprizone-induced demyelination models

Raj Thangavelu, Bruce Blazar; University of Minnesota
GVHD studies

Thomas Perlmann; Karolinska Institute
RXR reporter assays with IRX4204 qnd bexarotene

Danna Jennings, Ken Marek; Molecular Neuroimaging, Inc.
Parkinson's disease phase I clinical trial

Charles River Laboratories
Cortical neuron neurite outgrowth studies; microglial production of proinflammatory cytokines and chemokines studies

MD Biosciences, Ltd.
PD model of combination treatment with IRX4204 + thyroid hormone