



Neuroscience Portfolio

A DIVERSE PORTFOLIO OF RODENT
MODELS AND SERVICES FOR
NEUROSCIENCE RESEARCH

Taconic Biosciences Neuroscience Portfolio

A major challenge in neuroscience research and drug discovery is the lack of easy access to relevant animal models for use in screening drug candidates.

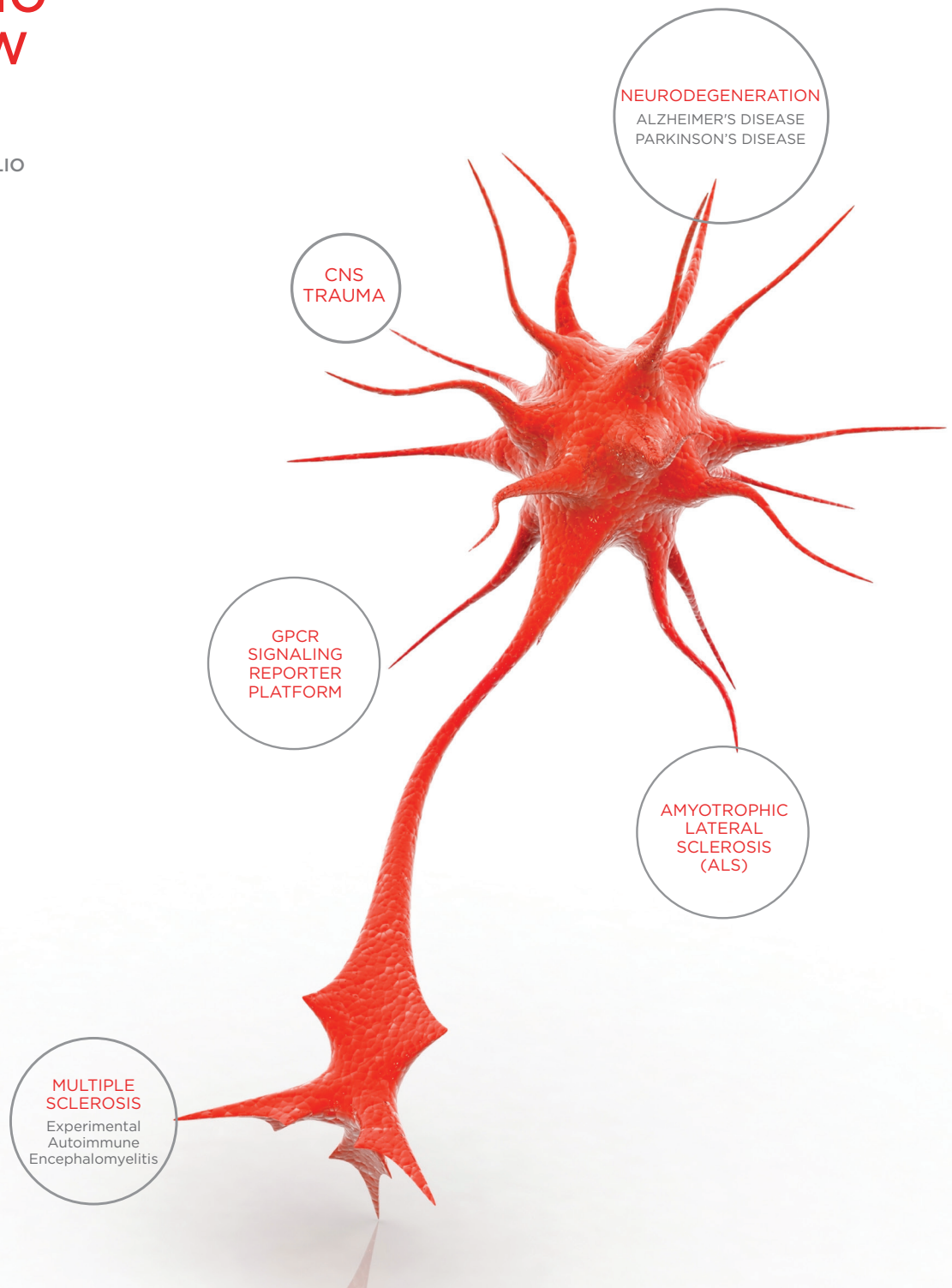
From neuro-developmental disorders to neurodegeneration, Taconic's Neuroscience Portfolio provides relevant solutions for pre-clinical research.

Taconic's Neuroscience Portfolio provides scientists with easy access to models and services for the study of neuronal disease and signaling. Many of these models are available exclusively from Taconic with rights to use. Together with scientific support from PhD scientists, extensive phenotyping data, and Taconic's customized breeding and aging services, we help you accelerate your neuroscience research efforts.

[Taconic.com/neuroscience](https://www.taconic.com/neuroscience)

PORTFOLIO OVERVIEW

MODELS AND SERVICES
COVERED IN THE
NEUROSCIENCE PORTFOLIO



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Neuroscience Portfolio

ALZHEIMER'S DISEASE MODELS

Amyloid beta (A β) plaques and neurofibrillary tangles (NFTs) combined with deficits in learning and memory are hallmarks of Alzheimer's Disease. Understanding how plaques and tangles are formed and discovering effective therapeutics that prevent these neurodegenerative processes are important factors for winning the battle against Alzheimer's Disease.

Taconic offers a variety of transgenic rodent models that develop plaques and tangles and can be used for screening of novel drug candidates for treating Alzheimer's and other neurodegenerative diseases.

FAMILIAL ALZHEIMER'S DISEASE MODELS

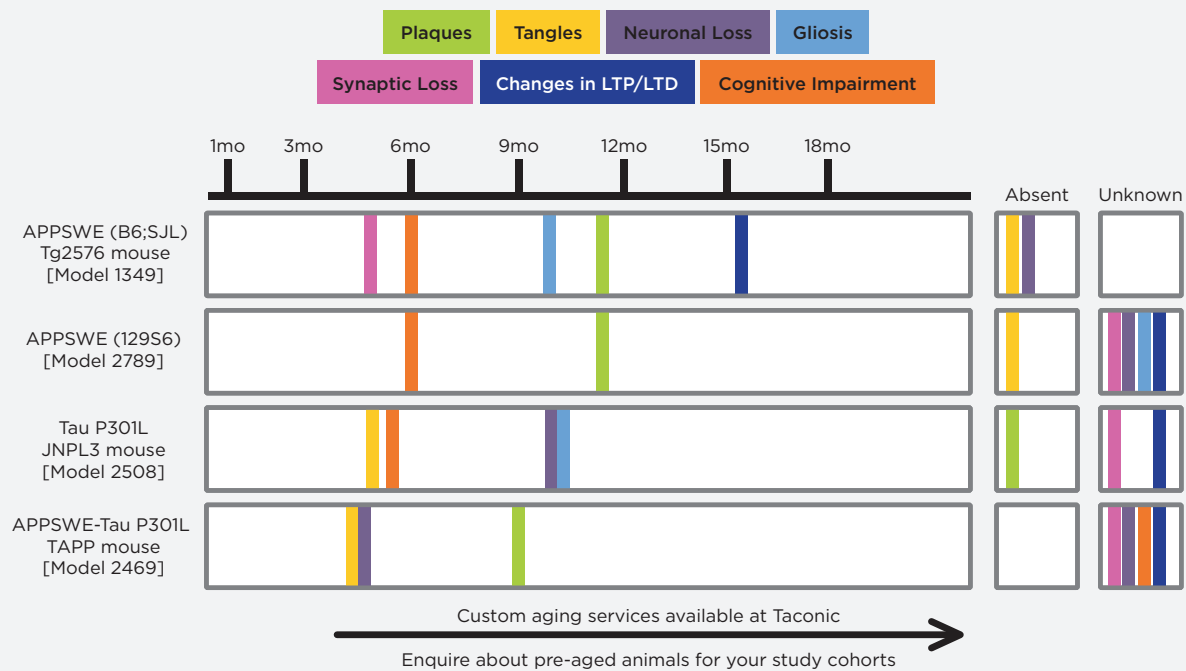
- APPSWE (Tg2576)
- TAU P301L (JNPL3)
- APPSWE-TAU P301L (TAPP)

SPORADIC ALZHEIMER'S DISEASE MODELS

- HUMANIZED APOE 2/3/4

Timeline of neuropathology of popular Alzheimer's mouse models available from Taconic

Graphic adapted from 'Research Models Visualization' at www.alzforum.org



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MODELS OF FAMILIAL ALZHEIMER'S DISEASE

APPSWE (Tg2576)

SWEDISH MUTATIONS K670N AND M671
RANDOM TRANSGENIC (B6;SJL MIXED BACKGROUND)

- Also known as the Tg2576 mouse, one of the most widely used models of Alzheimer's Disease pathology.
- Carries a transgene coding for the 695-amino acid isoform of human amyloid β precursor protein (APP) carrying the Swedish mutations (K670N, M671).
- Expresses high concentrations of the mutant A β protein, develops significant amyloid plaques, and displays memory deficits.
- Useful for the study of APP expression, amyloid plaque formation, neuronal decline, and memory loss associated with Alzheimer's.
- Carries the Pde6b^{rd1} retinal degeneration mutation observed in many inbred strains of mice.

B6;SJL-Tg(APP^{SWE})2576Kha

NOMENCLATURE

This may impact the results of behavioral testing.**

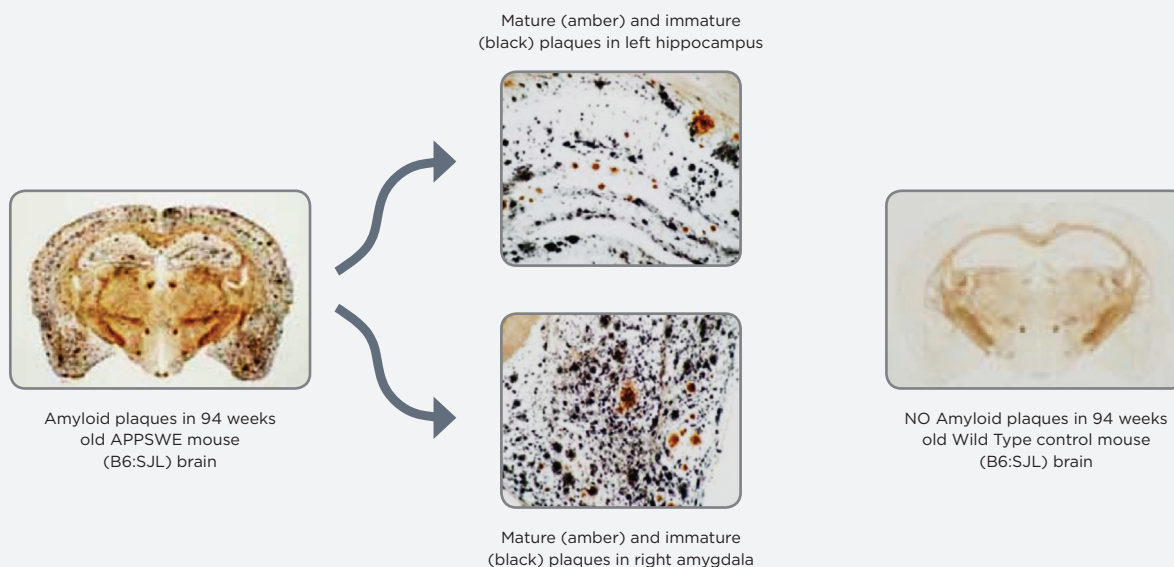
** Pink eyed animals, associated with certain coat colors, and the Pde6b^{rd1} retinal degeneration mutation found in several inbred strains of mice can cause light sensitivity and/or blindness in some animals. This may impact the results of behavioral testing. Upon request, Taconic can screen for eye color, coat color, and/or rd1 homozygosity for an additional fee.

MODEL NUMBER **1349**

MODEL NUMBER **1349-RD1**

(Screened for Pde6b^{rd1} mutation)

AMYLOID PLAQUE DEVELOPMENT IN APPSWE MOUSE BRAIN



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APPSWE

NOMENCLATURE

SWEDISH MUTATIONS K670N AND M67I
RANDOM TRANSGENIC (129S6 BACKGROUND)

129S6.Cg-Tg(APPSE)2576Kha N20+?

- Carries the same mutation as Tg2576 mouse, but on a different genetic background.
- Carries a transgene coding for the 695-amino acid isoform of human Alzheimer β -amyloid precursor protein (APP) carrying the Swedish mutations (K670N, M67I).
- Expresses high concentrations of the mutant A β protein, develops significant amyloid plaques and displays memory deficits correlating with the development of amyloid plaques.
- This background does not carry the Pde6brd1 retinal degeneration mutation, but as with all 129 substrains does carry a mutated Disc1 gene

MODEL NUMBER **2789**

TAU P301L (JNPL3 mouse)

NOMENCLATURE

P301L MUTATION IN HUMAN TAU
RANDOM TRANSGENIC (C57BL/6, DBA/2,
SW MIXED BACKGROUND)

STOCK Tg(PrnpMAPT*P301L) jNPL3Hlmc

- Also known as the JNPL3 mouse.
- Carries the transgene for the human P301L mutation of the microtubule associated protein tau gene (MAPT).
- Aggregates of filaments of TAU result in neurofibrillary tangles which are associated with Alzheimer's disease, Pick disease, and other neurological syndromes.
- Develops behavioral and motor disturbances related to development of neurofibrillary tangles.
- Sex differences in transgene expression have been observed with females expressing higher levels of protein than males.
- Carries the Pde6b^{rd1} retinal degeneration mutation observed

in many inbred strains of mice. This may impact the results of behavioral testing.**

** Pink eyed animals, associated with certain coat colors, and the Pde6brd1 retinal degeneration mutation found in several inbred strains of mice can cause light sensitivity and/or blindness in some animals. This may impact the results of behavioral testing. Upon request, Taconic can screen for eye color, coat color, and/or rd1 homozygosity for an additional fee.

MODEL NUMBER **2508**

MODEL NUMBER **1638**
(Wild Type control)

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APPSWE-TAU P301L (TAPP mouse)

NOMENCLATURE

SWEDISH MUTATIONS K670N, M671 IN HUMAN APP
AND P301L MUTATION IN HUMAN TAU
RANDOM TRANSGENIC (C57BL/6, DBA/2, SJL,
SW MIXED BACKGROUND)

STOCK Tg(APPswe)2576Kha
Tg(Prnp-MAPT*P301L)JNPL3HImc

- Carries the transgene coding for the 695-amino acid isoform of human amyloid β precursor protein (APP) in addition to the transgene for the human P301L mutation of the microtubule-associated protein tau gene (MAPT).
 - Amyloid plaque distribution, number, morphology and density is similar between APPSWE-TAU and APPSWE mice.
 - Motor disturbances and morphology of neurofibrillary tangles are comparable between APPSWE-TAU and Tau mice.
 - Useful for studies that focus on formation of β -amyloid plaques and neurofibrillary tangles, and for developing novel therapeutics for the prevention and treatment of Alzheimer's Disease.
 - Carries the Pde6b^{rd1} retinal degeneration mutation observed in many inbred strains of mice. This may impact the results of behavioral testing.**
- ** Pink eyed animals, associated with certain coat colors, and the Pde6b^{rd1} retinal degeneration mutation found in several inbred strains of mice can cause light sensitivity and/or blindness in some animals. This may impact the results of behavioral testing. Upon request, Taconic can screen for eye color, coat color, and/or rd1 homozygosity for an additional fee.

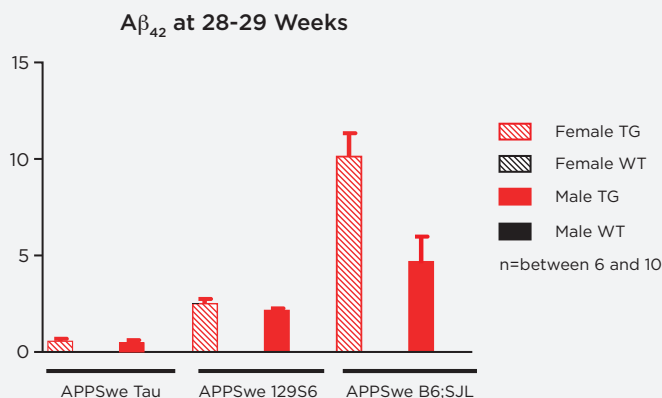
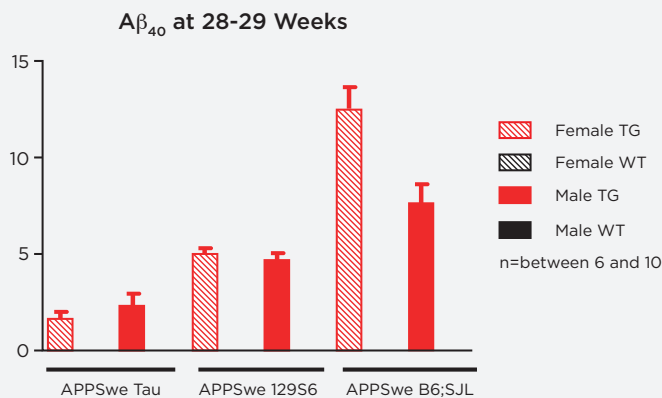
MODEL NUMBER **2469**

MODEL NUMBER **3723**
(Wild Type control)

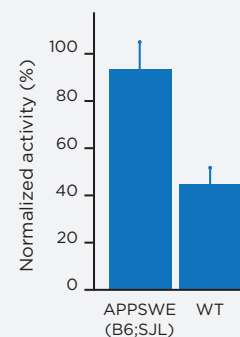
MODEL NUMBER **2469-RD1**
(Screened for Pde6b^{rd1} mutation)

MODEL NUMBER **3723-RD1**
(Screened for Pde6b^{rd1} mutation)

PATHOGENIC A β IN ALZHEIMER'S MOUSE MODELS



LEARNING DEFICITS IN APPSWE MICE



Impaired trace fear conditioning in 54 weeks old APPSWE mice

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MODELS OF SPORADIC ALZHEIMER'S DISEASE

HUMANIZED APOLIPOPROTEIN E (ApoE) BASED NEURODEGENERATION MODELS

ApoE and Neurodegeneration Quick Facts

ApoE is a plasma protein involved in cholesterol transport, with three human isoforms: E2, E3, and E4. In addition to genotype-phenotype associations with cardiovascular disease, isoforms of apoE have also been implicated as risk factors for sporadic forms of late-onset Alzheimer's Disease.

- In the brain, apoE is synthesized primarily by astrocytes and microglia. It is then lipidated by the ABCA1 transporter to form lipoprotein particles. Once formed, lipidated apoE binds to soluble A β and facilitates A β uptake via cell surface receptors.
- The association between specific apoE isoform expression and human neurodegenerative disorders has focused on the role of apoE isoforms in lipoprotein receptor-dependent synaptic modulation.
- Among the three isoforms, apoE4 appears to drive amyloid pathology by inhibiting brain clearance of A β peptides, and by promoting A β aggregation.
- Targeting apoE and apoE receptor pathways may offer novel therapeutic strategies to combat neurodegenerative diseases.

Taconic provides three humanized apoE mouse models that are useful for studying the role of human APOE polymorphism in neurodegenerative disorders.



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APOE2

HUMANIZED KNOCK IN (C57BL/6 BACKGROUND)

- Homozygous for a human APOE2 gene targeted replacement of the endogenous mouse Apoe gene.
- Expresses human apolipoprotein E2 isoform under the control of the murine Apoe regulatory sequences.
- E2 is the least common isoform in the human population.
- In humans, the E2 allele decreases the risk and delays onset of Alzheimer's Disease, but increases the risk of type III hyperlipoproteinemia.

MODEL NUMBER **1547**

NOMENCLATURE

B6.129P2-Apoe^{tm1(APOE*2)Mae} N9

- These mice develop spontaneous atherogenesis, which is exacerbated by a high fat diet.

APOE3

HUMANIZED KNOCK IN (C57BL/6 BACKGROUND)

- Homozygous for a human APOE3 gene targeted replacement of the endogenous mouse Apoe gene.
- Expresses human apolipoprotein E3 isoform under the control of the murine Apoe regulatory sequences.
- E3 is the most common isoform, expressed by almost 80% of the human population.
- On a normal diet these mice have normal plasma cholesterol and triglyceride levels, but relative quantities of plasma lipoprotein particles are altered, and clearance of vLDL particles is delayed.
- On a high-fat diet, these mice develop abnormal serum lipid profiles and atherosclerotic plaques.

MODEL NUMBER **1548**

NOMENCLATURE

B6.129P2-Apoe^{tm2(APOE*3)Mae} N8

- On a high-fat diet, these mice exhibit an increased risk of atherosclerosis and hypercholesterolemia relative to wild type mice.

APOE4

HUMANIZED KNOCK IN (C57BL/6 BACKGROUND)

- Homozygous for a human APOE4 gene targeted replacement of the endogenous mouse Apoe gene.
- Expresses human apolipoprotein E4 isoform under the control of the murine Apoe regulatory sequences.
- E4 occurs in approximately 14% of the human population and has been implicated as a risk factor for developing Alzheimer's disease.
- On a normal diet, these mice have normal plasma cholesterol and triglyceride levels, but relative quantities of plasma lipoprotein particles are altered, and clearance of vLDL particles is delayed.

MODEL NUMBER **1549**

NOMENCLATURE

B6.129P2-Apoe^{tm2(APOE*4)Mae} N8

- On a high-fat diet, these mice exhibit an increased risk of atherosclerosis relative to wild type, and APOE3 targeted replacement mice.

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AMYOTROPHIC LATERAL SCLEROSIS (ALS) MODEL

The hallmarks of Amyotrophic Lateral Sclerosis (ALS), also known as Lou Gehrig's disease, are muscle weakness, reduced motor function, and paralysis. Understanding how motor neurons are targeted for degeneration and discovering effective therapeutics that prevent motor neuron degeneration are important factors for winning the battle against ALS.

Taconic offers a humanized animal model sponsored by the ALS Association. This model allows investigators to screen novel compounds for efficacy against the various pathologies associated with ALS.

SOD1 RAT

SOD1 G93A MUTATION
RANDOM TRANSGENIC (SPRAGUE DAWLEY BACKGROUND)

NOMENCLATURE

NTac:SD-Tg(SOD1G93A)L26H

- Carries the transgene encoding the human SOD1 gene with the G93A mutation.
- Hemizygous rats express SOD1G93A in the spinal cord approximately 8-fold above endogenous levels, and develop motor neuron disease with abnormal gait and hind limb paralysis.
- SOD1G93A is also expressed across many brain regions as well as peripheral tissues.
- By end stage, mutant SOD1 levels accumulate approximately 16-fold over endogenous levels, representing an additional 2-fold increase in SOD1G93A compared with levels in young, presymptomatic rats (6 weeks old).
- Rapid decline of SOD1G93A rats coincides with substantial loss of spinal cord motor neurons as well as marked increases in gliosis and degeneration of muscle integrity and function.

MODEL NUMBER **2148**



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PARKINSON'S DISEASE RESEARCH TOOLS

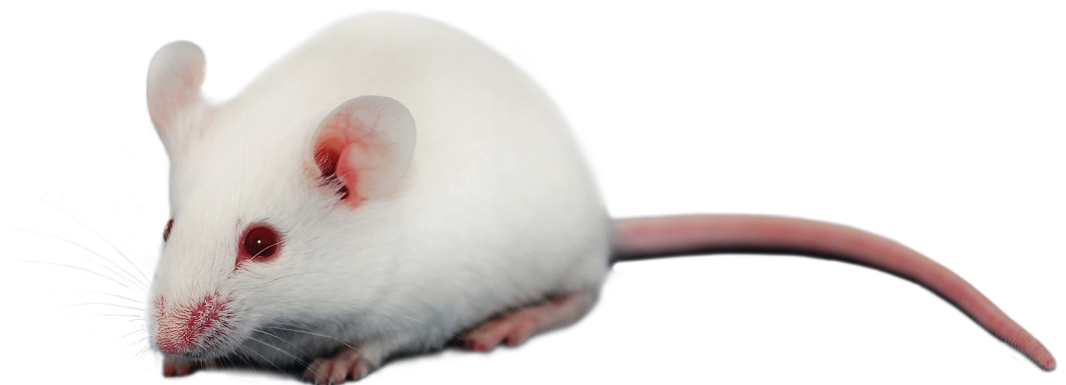
Rat Tools For Parkinson's Research Quick Facts

- Loss of nigrostriatal dopamine neurons and reduced motor abilities are hallmarks of Parkinson's Disease.
- Mutations in the *LRRK2* and alpha synuclein (*SNCA*) genes are associated with familial Parkinson's Disease and to affect the nigrostriatal pathway.
- The rat as an experimental organism can offer some unique strengths compared to mice:
 - Rats can perform more sophisticated behavioral tasks.
 - Rats are better suited for electrophysiological multichannel recordings.
 - Nigrostriatal circuit of the rat is more sensitive to insults compared to that of mice.

Alpha Synuclein (SNCA)
mutations (A53T, A30P, E46K) and gene duplication or triplication can lead to Parkinson's disease.

LRRK2 G2019S is the most prevalent mutation found in familial and sporadic Parkinson's disease.

6-OHDA insult to brain dopamine neurons mimic pathologies associated with Parkinson's disease.



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THE MICHAEL J FOX FOUNDATION SPONSORED RAT TOOLS FOR PARKINSON'S RESEARCH

TACONIC OFFERS SEVERAL TRANSGENIC RATS, SPONSORED BY THE MICHAEL J FOX FOUNDATION, THAT CAN BE USED AS TOOLS TO DEVELOP THERAPIES AGAINST PARKINSON'S DISEASE.

HUMAN ALPHA SYNUCLEIN RAT

NOMENCLATURE

EXPRESSES HUMAN SNCA
RANDOM TRANSGENIC (SD BACKGROUND)

Tac:SD-Tg(SNCA*WT)446CJli

- Carries the wild type human alpha synuclein gene (SNCA)

MODEL NUMBER **10680**

HUMAN ALPHA SYNUCLEIN A53T RAT

NOMENCLATURE

EXPRESSES A53T ON HUMAN SNCA GENE
RANDOM TRANSGENIC (SD BACKGROUND)

NTac:SD-Tg(SNCA*A53T)268Cjli

- Carries the A53T mutation on the human alpha synuclein gene (SNCA).
- Neuronal loss and Lewy Bodies in the substantia nigra and locus ceruleus are associated with the A53T alpha-synuclein mutation

MODEL NUMBER **10678**

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HUMAN ALPHA SYNUCLEIN E46K RAT

[NOMENCLATURE](#)

EXPRESSES E46K ON HUMAN SNCA GENE
RANDOM TRANSGENIC (SD BACKGROUND)

NTac:SD-Tg(SNCA*E46K)70CJLi

- Carries the E46K mutation on the human alpha synuclein gene (SNCA).
- Atrophy of the substantia nigra and build-up of Lewy bodies are associated with the E46K alpha synuclein mutation.

MODEL NUMBER **10679**

HUMAN LRRK2 G2019S RAT

[NOMENCLATURE](#)

EXPRESSES G2019S ON HUMAN LRRK2 GENE
RANDOM TRANSGENIC (SD BACKGROUND)

NTac:SD-Tg(LRRK2*G2019S)571Cjli

- Carries the G2019S mutation on the human LRRK2 gene.
- Non-specific deterioration of the substantia nigra and absence of Lewy bodies is associated with the G2019S mutation.

MODEL NUMBER **10681**

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hTH-GFP RAT

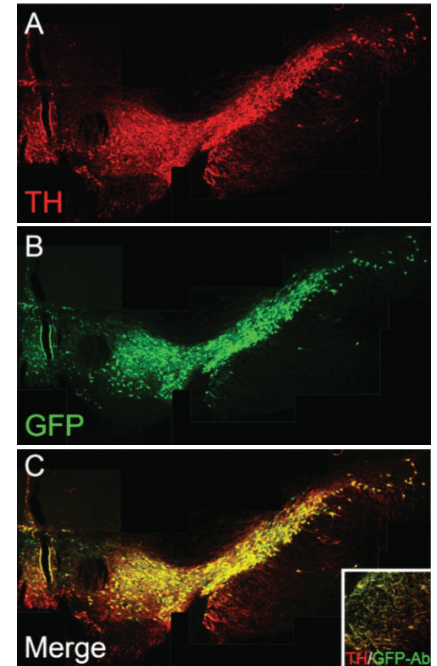
GFP EXPRESSION UNDER HUMAN
TYROSINE HYDROXYLASE PROMOTER
RANDOM TRANSGENIC (SD BACKGROUND)

NOMENCLATURE

NTac:SD-Tg(TH-EGFP)24Xen

- Carries an EGFP transgene driven by the human Tyrosine Hydroxylase (hTH) promoter
- Robust and specific EGFP expression observed in dopaminergic neurons of various brain structures including the substantia nigra, ventral tegmental area, striatum, olfactory bulb and hypothalamus. Minimal ectopic expression observed in other regions of the brain
- Dopamine neurons are susceptible to damage/loss in Parkinson's disease and therefore this rat can be used as a tool to study damage/loss of dopamine neurons, e.g., after MPTP or 6-OHDA treatment.
- Useful for *in vivo* anatomical visualization and micro-dissection of rat midbrain structures and axonal projections. High EGFP expression permits fluorescence imaging of brain slices. Also useful for FACS purification and *in vitro* culture of dopamine neurons for studies of disease pathogenesis in culture
- This rat is also practical for study of early embryonic development of dopamine neurons, since EGFP is more easily detected than TH immunostaining at early developmental stages

MODEL NUMBER **12141**



GFP expression overlaps with TH expression in the midbrain.

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SURGICALLY INDUCED MODEL OF PARKINSON'S DISEASE

Unilateral 6-Hydroxydopamine Lesion of the Nigrostriatal Pathway

- Animal models with unilaterally destroyed central dopamine neurons are important tools in the study of neurodegenerative disease processes associated with Parkinson's Disease.
- The neurotoxin 6-hydroxydopamine (6-OHDA) is administered into certain brain areas. This results in a selective destruction of catecholamine (adrenaline, noradrenaline, and dopamine) neurotransmitter neurons.
- The unilateral destruction of dopamine neurons results in a chemical imbalance of dopamine content across the brain hemispheres.
- As a result of this chemical imbalance or asymmetry, administration of certain dopamine agonists, such as apomorphine, causes stimulation of intact dopamine neurons in the unaffected brain hemisphere.
- This asymmetric stimulation is behaviorally manifested by locomotion in the direction of the unaffected hemisphere (i.e. the animal runs in circles).
- The quantification of circling behavior can be used to assess the efficacy of therapeutic agents which may be used in the treatment of Parkinson's Disease.



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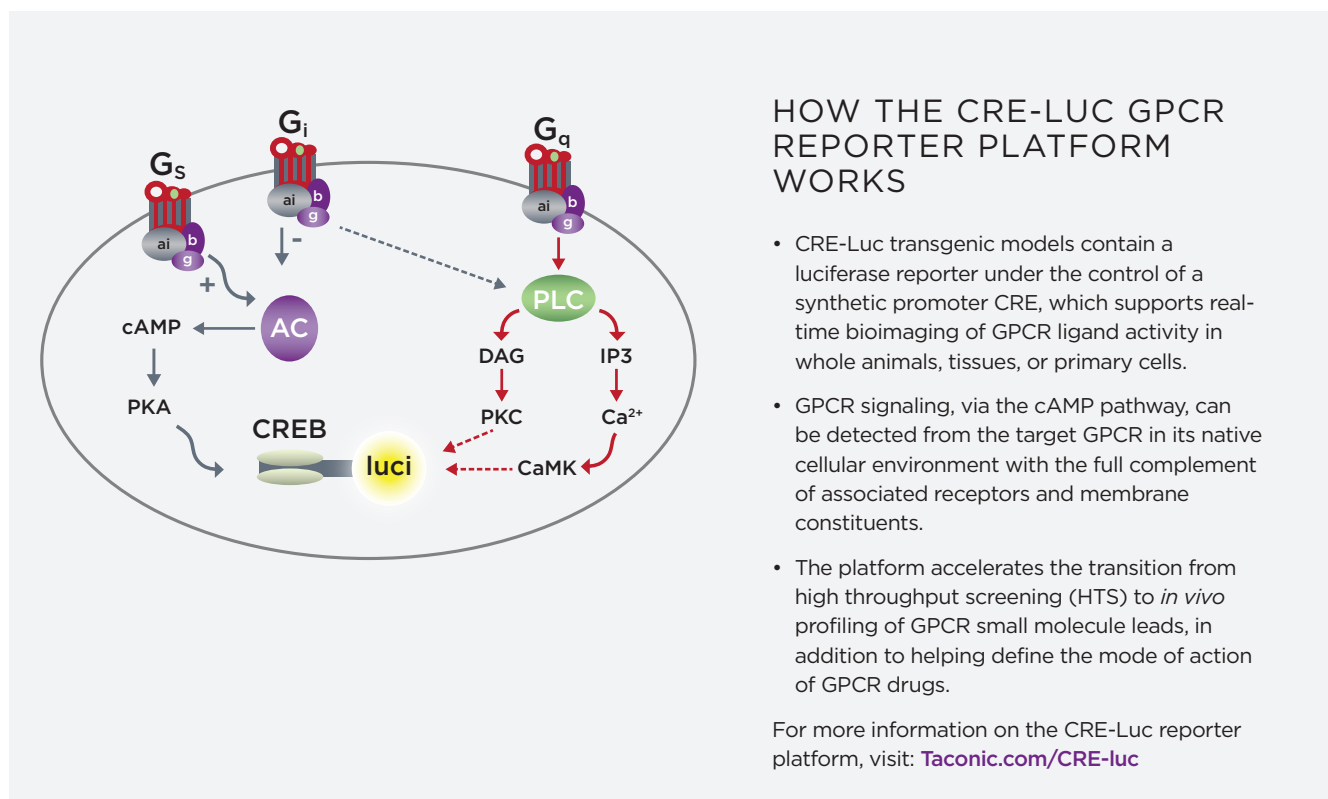
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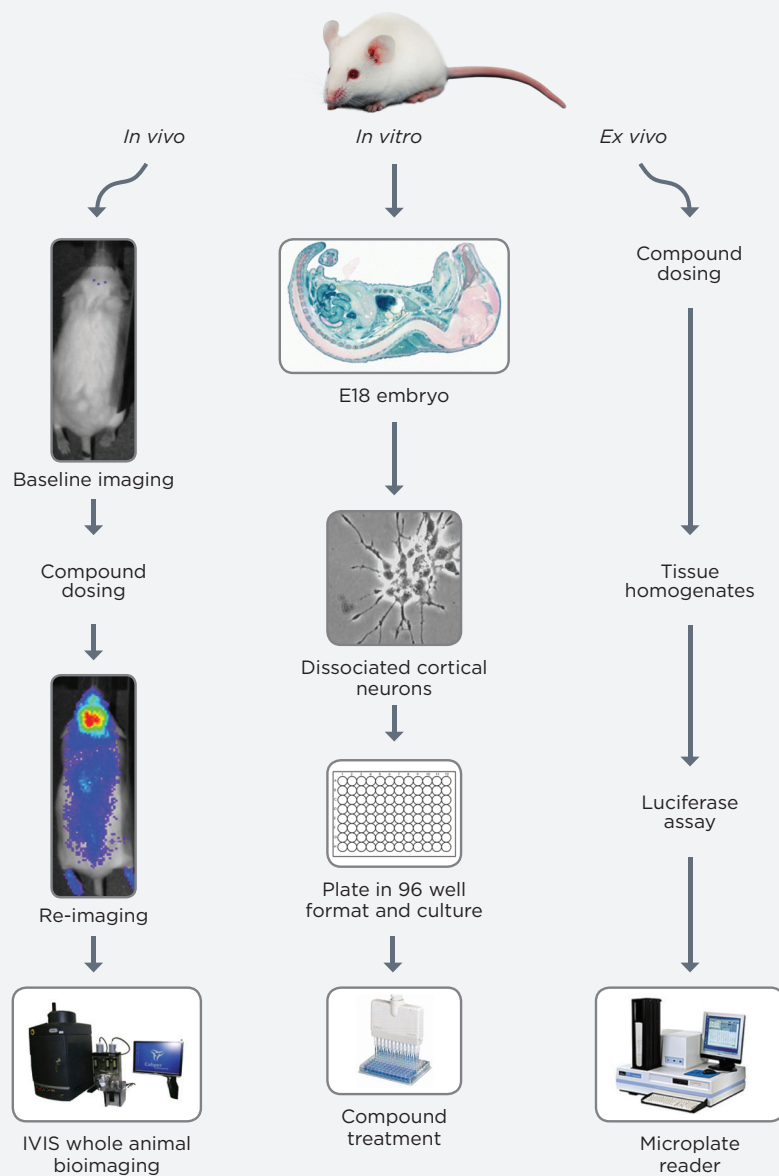
CRE-LUC GPCR REPORTER MOUSE PLATFORM

AN *IN VITRO*/*IN VIVO* LUCIFERASE REPORTER PLATFORM FOR PROFILING OF LEADS IN GPCR DRUG DEVELOPMENT

- A panel of luciferase reporter mice are available that allow monitoring of GPCR pathway activation (via the two main GPCR classes, G_s and G_i) in various tissues, and help better profile leads in GPCR drug development.
- The CRE-Luc GPCR reporter mouse platform enables investigators to rapidly conduct *in vivo* PK/PD profiling of compounds with quantitative data to compare pharmacological action.
- The central nervous system CRE-Luc reporter is specifically expressed in the brain and spinal cord, and can be leveraged in a variety of assays including *in vitro* (primary neuronal cultures), *in vivo* (whole animal), and *ex vivo* (brain slices).



CRE-LUC MOUSE PLATFORM USED IN DIFFERENT ASSAY SYSTEMS



KEY STRENGTHS OF THE PLATFORM

- Helps accelerate the difficult transition from *in vitro* to *in vivo* assays in GPCR pharmaceutical programs.
- Multiple assay formats can enhance lead optimization and progression.
- Supports monitoring of any GPCR signaling through the camp pathway in a native environment where the critical membrane interfaces are interacting with the targeted GPCR.

MODEL NUMBER	REPORTER EXPRESSION
11515	Pancreas
11516	Intestine, liver, pancreas, lungs
11517	Kidney, liver
11518	Spleen, kidney, liver
11519	Brain, lung
11520	Brain, spinal cord
11521	Kidney, brain, pancreas, lungs

TO ORDER

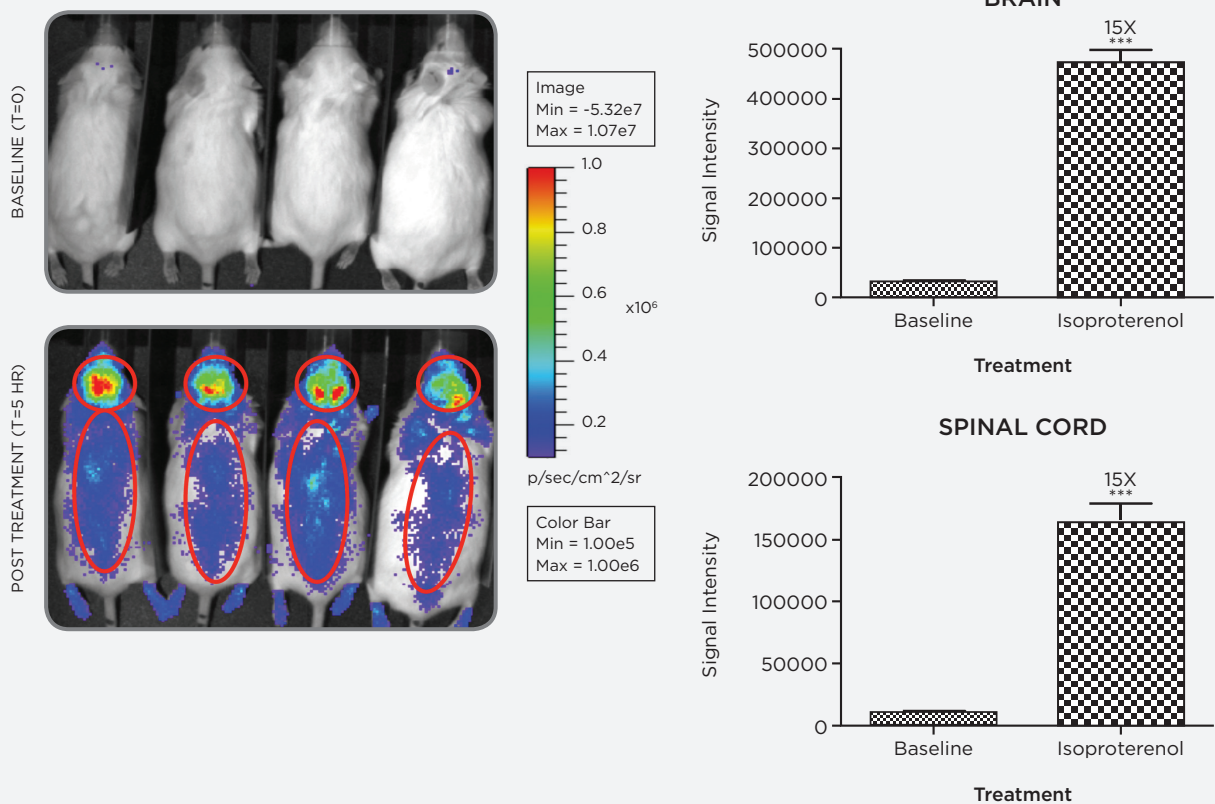
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APPLICATIONS IN STUDIES OF GPCR SIGNALING IN THE CNS

APPLICATIONS IN STUDIES OF GPCR SIGNALING IN THE CNS

Whole Animal Imaging



Treatment of mice (Model #11520) with Isoproterenol (β -adrenergic receptor agonist) shows CNS response

EXAMPLE WORKFLOW WITH THE CRE-LUC REPORTER PLATFORM

The CRE-Luc lines can serve as a source of primary cells with the GPCR reporter in its native environment. Therefore *in vitro* studies can be first performed followed by *in vivo* studies.

IN VITRO STUDIES (PRIMARY CELLS)

Primary cell cultures derived from CRE-Luc models can be used to confirm ligand activation. For example, CRE-Luc cultures support GPCR receptor specificity assays, like the use of RNAi or ligand competition assays. These assays are an important validation step since it is possible that any receptor (or combination of receptors) can be activated by a single ligand.

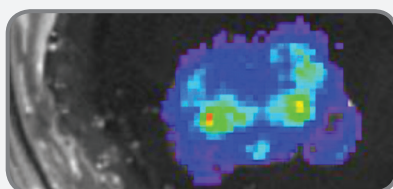
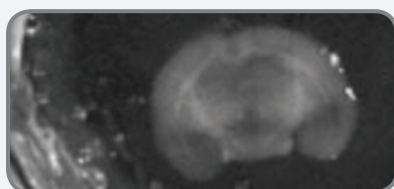
Once ligand activation has been profiled in primary cells, more complex tissue profiles can be assayed for luciferase enzyme levels either *ex vivo* or using tissue homogenates. Although tissue homogenate analyses can be time consuming, it is especially valuable when combined with dosing in whole animals, as it allows investigators to generate tissue-specific, and quantitative ligand activation profiles.

IN VIVO STUDIES (WHOLE ANIMAL)

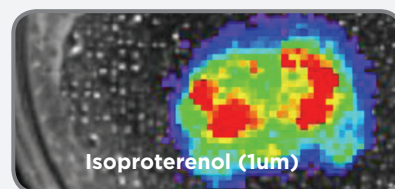
Once the activation profiles have been established using primary cells, ligand profiles can be probed in whole animals using bioimaging techniques, while also incorporating dose-response and time-course assays. Data analysis can occur in the same day as the imaging session which allows unknown endpoints or results in the assay to be defined as the study progresses. This feature impacts flexibility in the animal study and can save significant time in avoiding repetitive studies to capture overlooked data.

The whole animal bioimaging assay can quantitatively define the site and magnitude of ligand activation, and can support a quantitative comparison of similar compounds which can be useful for selecting optimal lead structures, and SAR.

BRAIN SLICE IMAGING (MODEL #11520)



Baseline



Gs agonist

Imaging of compound induced changes in luciferase levels by a β -adrenergic receptor agonist

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GFAP-LUC: CNS TRAUMA MODEL

CNS TRAUMA AND GFAP Quick Facts

- Glial fibrillary acidic protein (GFAP) is an intermediate filament protein found in the cytoskeleton of astroglia. GFAP may serve as a traumatic brain injury and central nervous system cell damage.
- Physical trauma, chemical treatment, and bacterial infections resulting in cellular damages in the CNS or changes in pressure within the cerebral spinal fluid are most likely to regulate GFAP expression.
- The GFAP-Luc mouse provides an easy to use tool to study CNS damage.

GFAP-Luc MOUSE

EXPRESSES LUCIFERASE UNDER GFAP PROMOTER
RANDOM TRANSGENIC (FVB BACKGROUND)

NOMENCLATURE

FVB/N-Tg(Gfap-luc)53Xen

- The GFAP-Luc model carries a luciferase reporter gene under the control of the GFAP promoter.
- The reporter is inducible following injury to the CNS and the model is useful for studying changes in the health of the CNS that result in GFAP gene regulation.
- Useful for studying brain trauma, CNS regeneration, astrocyte regeneration, meningitis, physical trauma, chemical insult, glial scarring.
- Animals have albino coat color making them suitable for whole body imaging.

MODEL NUMBER **10501**

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GFAP-LUC-B6 ALBINO: BRAIN INFLAMMATION MODEL

Experimental Autoimmune Encephalomyelitis Quick Facts

- Experimental Autoimmune Encephalomyelitis (EAE) is an animal model of brain inflammation.
- It is widely studied as an animal model of the human CNS demyelinating diseases, including multiple sclerosis (MS) and acute disseminated encephalomyelitis.
- EAE is also the prototype for T-cell-mediated autoimmune disease in general.
- EAE can be induced in rodents, for example by administering antigens such as spinal cord homogenate (SCH) or purified myelin, myelin protein such as MBP.

GFAP-Luc-B6 Albino

EXPRESSES LUCIFERASE UNDER GFAP PROMOTER
RANDOM TRANSGENIC (FVB BACKGROUND)

- GFAP-luc in B6 albino x FVB F1 mouse is susceptible to Experimental Autoimmune Encephalomyelitis (EAE).
- This mouse can be used for studying brain inflammation, and CNS demyelination disorders such as MS.
- Animals have albino coat color making them suitable for whole body imaging.

ENQUIRE FOR AVAILABILITY

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MOLECULAR ANALYSIS

Pde6b^{rd1} Retinal Degeneration Genotyping Assay

- Animals homozygous for the *Pde6b^{rd1}* allele become blind by weaning. The presence of this allele may be very important in mice used for behavioural testing. This assay may be used to screen out *Pde6b^{rd1}* homozygotes prior to behavioral work.

To learn more about the *rd1* mutation and order our screening service please contact: info@taconic.com

MODEL NUMBER **GENO_RD1_PCR**

Nnt Mutation Testing

Nnt (nicotinamide nucleotide transhydrogenase) is a gene present in humans, mice and bacteria. The enzyme encoded by the *Nnt* gene plays an important role in cellular metabolism, and also helps in the detoxification of cells under conditions of stress.

When a mutation of the *Nnt* gene occurs, this often results in abnormal glucose metabolism under conditions

of stress. This property makes *Nnt* important in research that spans across a variety of biological systems such as neurodegenerative diseases, diabetes, cancer, aging, and cardiomyopathy.

Taconic provides an *Nnt* mutation screening service that can help ensure your transgenics are free of the *Nnt* mutation.

To learn more about the *Nnt* mutation and order our screening service please contact: info@taconic.com

MODEL NUMBER **GENO-NNT-TEST**

STUDY READY SERVICES FOR NEUROSCIENCE RESEARCH

TO EFFECTIVELY EVALUATE DRUG EFFICACY OR DISEASE PROGRESSION WHEN USING *IN VIVO* MODELS, IT IS CRITICAL TO HAVE THE ABILITY TO PERFORM MANIPULATIONS AND CONDUCT BIOLOGICAL SAMPLING.

TACONIC PROVIDES A WIDE ARRAY OF SERVICES THAT CAN BE PERFORMED ON OUR RODENT MODELS TO HELP YOU ACCELERATE YOUR DISCOVERY PIPELINE. THESE SERVICES INCLUDE CUSTOM AGING, ADMINISTRATION OF SPECIALIZED DIETS, MICRODIALYSIS IMPLANTS, BRAIN CANNULATIONS, AND MURINE BIOSPECIMEN COLLECTION SERVICES. BELOW IS A DESCRIPTION OF EACH SERVICE.

Telemetry

- Subcutaneous or intraperitoneal placement of transmitters.
- Allows for continuous monitoring of neurological data (EEG, EMG, temperature, and activity) in fully awake and freely moving laboratory animals.
- Taconic staff have been trained by Data Sciences International.

Aging And Specialized Diets

- Rodent models of neurodegeneration often need to be aged in order for the pathological phenotype to develop. Taconic offers a wide range of customized aging services including resources to allow for natural aging.
- Animals are housed in our Isolated Barrier Units
- Animals can be maintained on specialized diets (e.g., high- fat diet to induce atherosclerotic plaques)
- Taconic's PhD scientists discuss with clients the aging and diet conditions, and help in selecting starting cohort sizes to help offset the mortality rate inherent in aged neurodegeneration models.

Microdialysis Implants

- Minimally invasive *in vivo* sampling technique for extracellular tissue fluid allowing for the continuous measurement of small particles such as neurotransmitters.
- General applications include: drug metabolism and pharmacokinetics, behavioral studies, psychological research, neurological and neurochemical studies, addiction and chemical dependency.
- A microdialysis guide cannula is implanted in targeted areas of the brain such as hippocampus, prefrontal and striatal portions of the brain. Custom brain coordinates available upon request.
- Cannula can be placed both in rats and mice.

TO ORDER

US: 1-888-822-6642 | EU: +45 70 23 04 05 | INFO@TACONIC.COM

Neuroscience Portfolio

Murine Biospecimens

- Brain and other biospecimens (e.g., brain sections, eye, embryo,) can serve as important components to any research project, including comparative evaluations.
- Superior quality organs (e.g., brain, heart, lung), fluids (e.g., CNS fluid, whole blood, plasma), tissue (e.g., skin, muscle), and embryo products can be harvested from all Taconic animal models.
- Biospecimens are available fresh harvest, frozen, or flash frozen using dry ice and in RNA later.

Brain Cannulations

- Nominally intrusive surgery for placement of cannulas into the brain.
- Cannulas enable researchers to inject substances directly into the brain.
- Cannulation options include: Intracerebroventricular and Third Ventricle.
- Custom coordinate brain cannulations available upon request.
- Brain cannulations can be performed in both rats and mice.

Rodent Identification

- The identification of an animal is a valuable link to your key data.

Knock Out Repository and The GEM Collection

The Knock Out Repository provides unparalleled access to over 4000 fully-licensed, healthy knockout mouse models. These knock-out mice are highly valuable research tools that rapidly accelerate the drug discovery and development processes.

Visit [Taconic.com/KO](https://www.taconic.com/KO) to search for the knock-out model that you need.

The GEM Collection (for access by non-profit organizations only). The GEM Collection contains several hundred proprietary mouse lines which include conditional (cKO) and constitutive (KO) Knock Out mice, transgenic (Tg) over-expressing lines, conditional (cTTG) and constitutive targeted transgenesis (TTG) lines.

Scientific Support

HAVE QUESTIONS RELATED TO YOUR ANIMAL RESEARCH? TACONIC'S EXPERTS CAN HELP YOU WITH MODEL SELECTION, STUDY DESIGN, OPTIMAL USE OF VARIOUS DISEASE MODELS, DIET QUESTIONS, AND MORE!

CONSULT WITH TACONIC'S PHD NEUROSCIENTISTS TO HELP GET YOUR RESEARCH OFF ON THE RIGHT TRACK. CONTACT US TO SET UP YOUR CONSULTATION.

DISCUSS YOUR NEEDS

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Take Your Research Further



GEMS DESIGN

Taconic Biosciences GEMs Design empowers our clients to develop research models specifically suited to the unique needs of their discovery and development studies or therapeutic programs.

- Gene Inactivation
- Gene Mutation or Replacement
- CRISPR Gene Editing
- Transgene Expression
- miRNA Expression
- Cohort Production Packages

PRECISION RESEARCH MODELS

Research organizations demand precision tools that better reflect human physiology. Taconic Biosciences leads the field delivering innovative solutions to meet these continually evolving needs. Our core competencies include the delivery of complex strategies that both integrate human genetic sequences and engraft human cells and tissues into custom mouse and rat models.

- Human Gene Replacement
- Human Cell and Tissue Engraftment

GEMS MANAGEMENT

Taconic's fully integrated GEMs Management brings innovative models from design to study-ready cohorts with unprecedented speed and transparency.

- Embryology
- Rapid Colony Expansion
- Contract Breeding
- Surgical Services
- Tissue Collection
- Genotyping and Molecular Analysis
- Microbiome and Germ-Free Research Models and Services

CHOOSE TACONIC

For more than 60 years, Taconic has anticipated the needs of the scientific community to deliver models and services that meet the diverse needs of biomedical and biopharmaceutical researchers.

Today that forward thinking and commitment to working collaboratively has resulted in a client-centric environment infused with a knowledge bank that allows you to select the optimum model for your study based on informed insight into the generation of genetically engineered mouse and rat models.

YOUR COLLABORATIVE PARTNER

As a full-service biosciences company, Taconic can help you acquire, test, develop, breed, cryopreserve, prepare, and distribute highly relevant research lines worldwide. Whether you require custom genetically engineered, cell or tissue engrafted models or traditional models, Taconic's scientists will partner with you to rapidly and efficiently deliver the highest quality models.

TALK TO A SCIENTIST

Our scientific teams are happy to meet and talk with you about the most efficient way to achieve your study goals. Working in partnership with clients the world over, our scientific teams offer expert advice that can help you speed up your research and reduce your overall costs.

TALK TO A REPRESENTATIVE

For general information, you can talk to a member of our customer service team. Our customer service team is here to help you make the right decisions and get the models you need fast. Contact us at info@taconic.com

VISIT TACONIC.COM

For more information on the entire Taconic portfolio of products and services designed to help further your research, visit Taconic.com

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