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CNS Models

We offer preclinical research services using validated rodent models for central nervous system (CNS) disorders to support neuroscience drug development. Our expertise includes models for cognition, depression, anxiety, and Parkinson's disease. With capabilities in behavioural testing, pharmacological evaluation, and neurochemical and biomarker analysis, we provide high-quality, reproducible data to accelerate your CNS research.

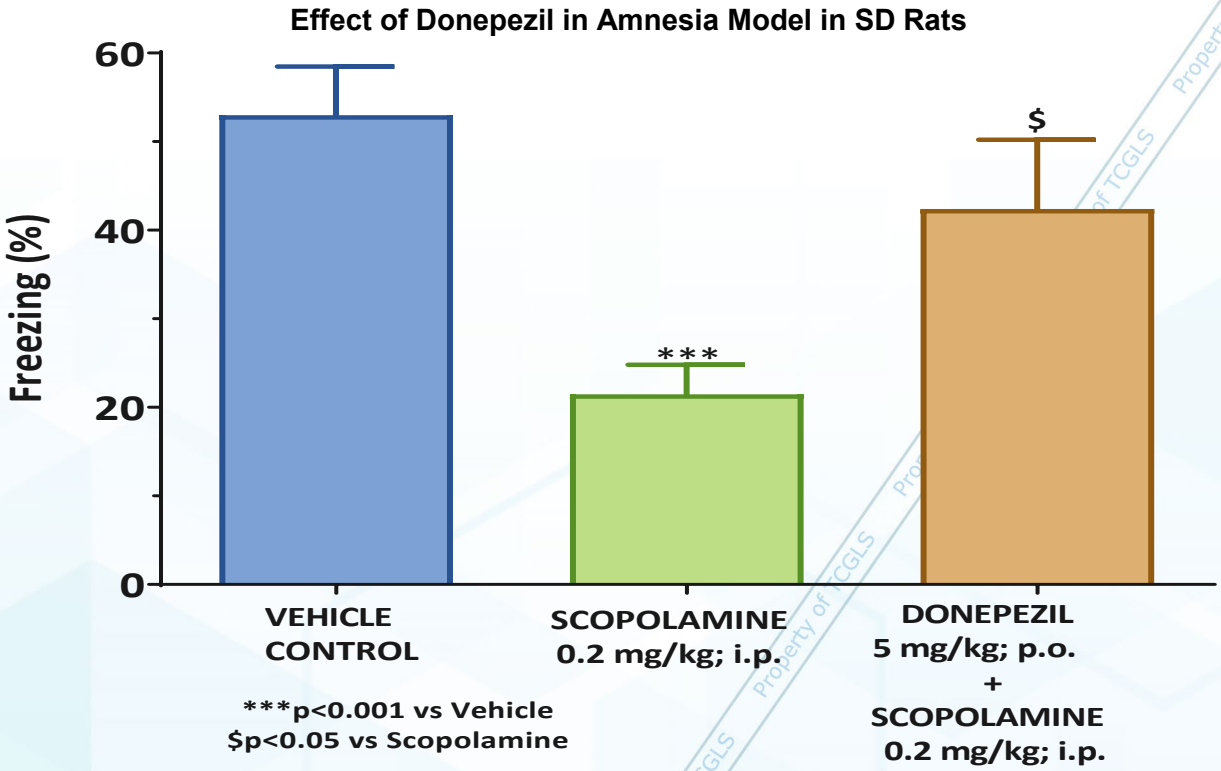
Cognition

- **Contextual Fear Conditioning (CFC)**

  The Contextual Fear Conditioning (CFC) test is a crucial tool in behavioural neuroscience for evaluating associative learning and memory, particularly those involving the hippocampus and amygdala. Its importance lies in its ability to model how organisms learn to associate environmental cues with aversive events, providing insights into the neural mechanisms underlying fear and memory. This test is widely used to study cognitive functions and emotional processing, making it valuable in research on neurodegenerative diseases (e.g., Alzheimer's), psychiatric disorders (e.g., PTSD, anxiety), and the effects of genetic or pharmacological interventions.

Validation results

- **Cognition development**: Conditioning of rats to a context for a definite time period (PSI) followed by unconditional stimulus of electric shock at foot
- **Cognition testing**: Observation of animals in the same environment on the following day for endpoint readout
- **Endpoint readout**: Rats show freezing behaviour, and the freezing time is recorded



- Scopolamine inhibited freezing behaviour which was reversed by Donepezil
- This platform can be used in screening compounds for reversal of amnesia

- Novel Object Recognition Test

Anxiety & Depression

- Forced Swim Test (FST)
- Marble Burying Test (MBT)
- Novelty Suppressed Feeding (NSF)
- Tail Suspension Test (TST)

Adverse Effect Profiling

- Locomotor Activity
- Rotarod Test

Neurodegenerative Disorders

- MPTP-PD (In progress)

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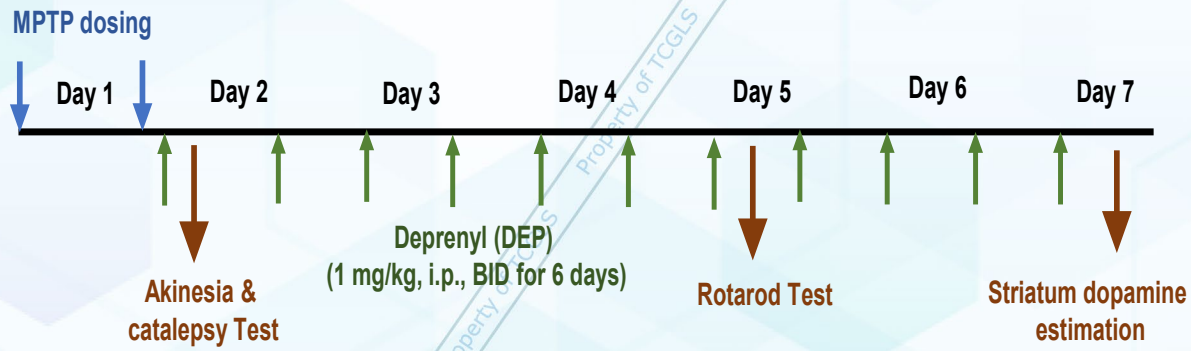
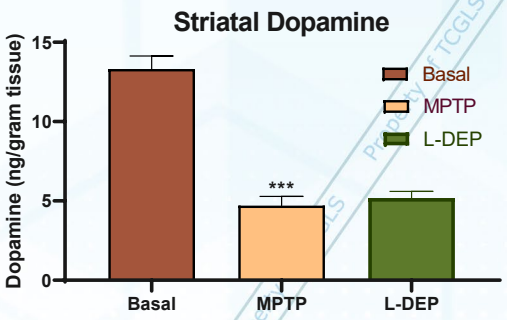
MPTP-Induced Parkinsonism Model

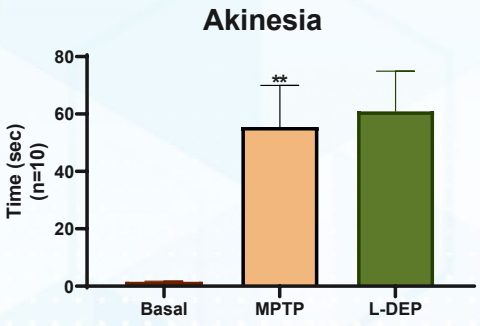
The MPTP-induced Parkinsonism model mimics Parkinson's disease by selectively destroying dopaminergic neurons in the substantia nigra. MPTP is converted to MPP^+ in the brain, leading to mitochondrial dysfunction and motor deficits. This model is widely used to study PD pathogenesis and evaluate potential treatments.

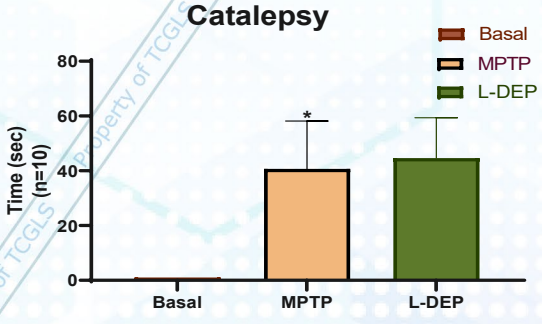
Validation results

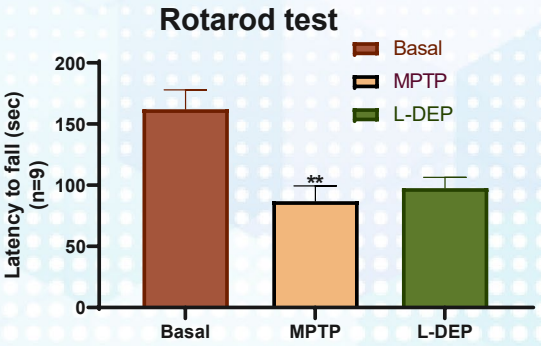
- **Animals:** Male C57BL/6 mice
- **Induction:** 2 doses of MPTP at 16-hour interval
- **Endpoint:** Striatal Dopamine deletion; Akinesia & Catalepsy duration
- **Behavioral Assessment:** Rotarod test performance











- MPTP administration (2 doses; IP at 16-hour interval) reduced striatal dopamine level by about 3 folds which was also well reflected in noticeable behavioural impairments in terms of prolonged akinesia & catalepsy as well as marked decline in rotarod performance.
- Deprenyl treatment (1 mg/kg, IP; BID for 6 days) could not reverse the MPTP-induced dopamine depletion at all and thereby, could not improve any behavioural impairment. Although improvement of behavioural impairment by DEP is reported in some literatures, the reversal effect in correlation with dopamine levels is not clearly depicted for DEP. In our study, probably pronounced dopaminergic neuronal loss by MPTP led to significant reduction in stratal dopamine levels which could not be reversed by DEP. Result in line with this hypothesis is also reported in few literatures (Neurochem Res. 1995 Apr;20(4):385-9. & J Neurosci Res. 1989 Jul;23(3):326-9).
- **Further validation is required to check different standard drug like L-Dopa/Carbidopa for reversal effect in MPTP-induced parkinsonism model.**

- **6-OHDA-PD (Subsequent plan)**

Field Potential Recording

- **LTP and LTD in Hippocampal Slices**