MPTP intoxication significantly increased nitrite and citrulline levels along
with enhanced expression of vital pro-inflammatory (TNF-α, IL-6, IL-1β,
iNOS, GFAP, COX-2), pro-apoptotic (Bax, Cytochrome C and Caspase-3)
factors and depleted the expression of anti-apoptotic marker Bcl-2. However,
pre-treatment with CNB-001 diminished these modulations which might be
mechanism responsible for its neuroprotective activity.

Administration of MPTP (30 mg/kg for four consecutive days) reduced
expression of PD specific protein (tyrosine hydrolase, dopamine transporter
and vesicular monoamine transporter 2) which is responsible for inhibition of
dopamine biosynthesis and resultant behavioral anomalies. Whereas, pre-
treatment with CNB-001(24 mg/kg) enhanced expressions of TH, DAT and
VMAT2 resulting in increased dopamine synthesis and normal motor
coordination.

Electron microscopic analysis of mitochondria showed that MPTP
intoxication induced ultrastructural changes such as distorted cristae and
mitochondrial enlargement in substantia nigra and striatum region.
Alternatively, pre-treatment with CNB-001 protected mitochondria which
are elucidated by normal mitochondrial morphology, size with distinct
nucleus and cristae. These findings support the neuroprotective property of
CNB-001 which may have strong therapeutic potential for treatment of PD.