

Accepted Manuscript

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PII: S0304-3940(18)30678-5
DOI: <https://doi.org/10.1016/j.neulet.2018.10.008>
Reference: NSL 33863

To appear in: *Neuroscience Letters*

Received date: 30-7-2018
Revised date: 24-9-2018
Accepted date: 5-10-2018

Please cite this article as: Fonseca-Fonseca LA, Wong-Guerra M, Ramírez-Sánchez J, Montano-Peguero Y, Padrón Yaquis AS, Rodríguez AM, da Silva VDA, Costa SL, Pardo-Andreu GL, Núñez-Figueroa Y, JM-20, a novel hybrid molecule, protects against rotenone-induced neurotoxicity in experimental model of Parkinson's disease, *Neuroscience Letters* (2018), <https://doi.org/10.1016/j.neulet.2018.10.008>

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JM-20, a novel hybrid molecule, protects against rotenone-induced neurotoxicity in experimental model of Parkinson's disease

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Highlights:

- Novel hybrid molecule (JM-20) prevents rotenone-induced cell death.
- JM-20 reduced oxidative stress and improved mitochondrial functions in rotenone-treated rats.
- JM-20 prevents the increase in sensory indifference (apathy) in rats chronically treated with rotenone.
- JM-20 increased survival and body weight gain of animals treated with rotenone.

Abstract

Oxidative stress and mitochondrial dysfunction are two pathophysiological factors often associated with the neurodegenerative process involved in Parkinson's disease (PD). The aim of this study was to investigate the effects of a novel hybrid molecule, named JM-20, in different *in vitro* and *in vivo* models of PD induced by rotenone. To perform *in vitro* studies, SHSY-5Y cells were exposed to rotenone and/or treated with JM-20. To perform *in vivo* studies male Wistar rats were intoxicated with rotenone (2.5 mg/kg) via intraperitoneal injection and/or treated with JM-20 (40 mg/kg) administered via oral (for 25 days, both

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