

Naringenin Mitigates α -synuclein Pathology And Stress Signaling In SH (SNCA) Cells Via Redox Modulation.

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KEY TAKEAWAY: Naringenin protects α -synuclein-overexpressing SH-SY5Y cells against rotenone-induced neurotoxicity by reducing oxidative stress, suppressing stress kinase activation, and decreasing α -synuclein aggregation, highlighting its potential as a therapeutic candidate for Parkinson's disease.

INTRODUCTION

Parkinson's disease (PD) is characterized by progressive dopaminergic neurodegeneration associated with oxidative stress, mitochondrial dysfunction, neuroinflammation, and abnormal aggregation of α -synuclein.

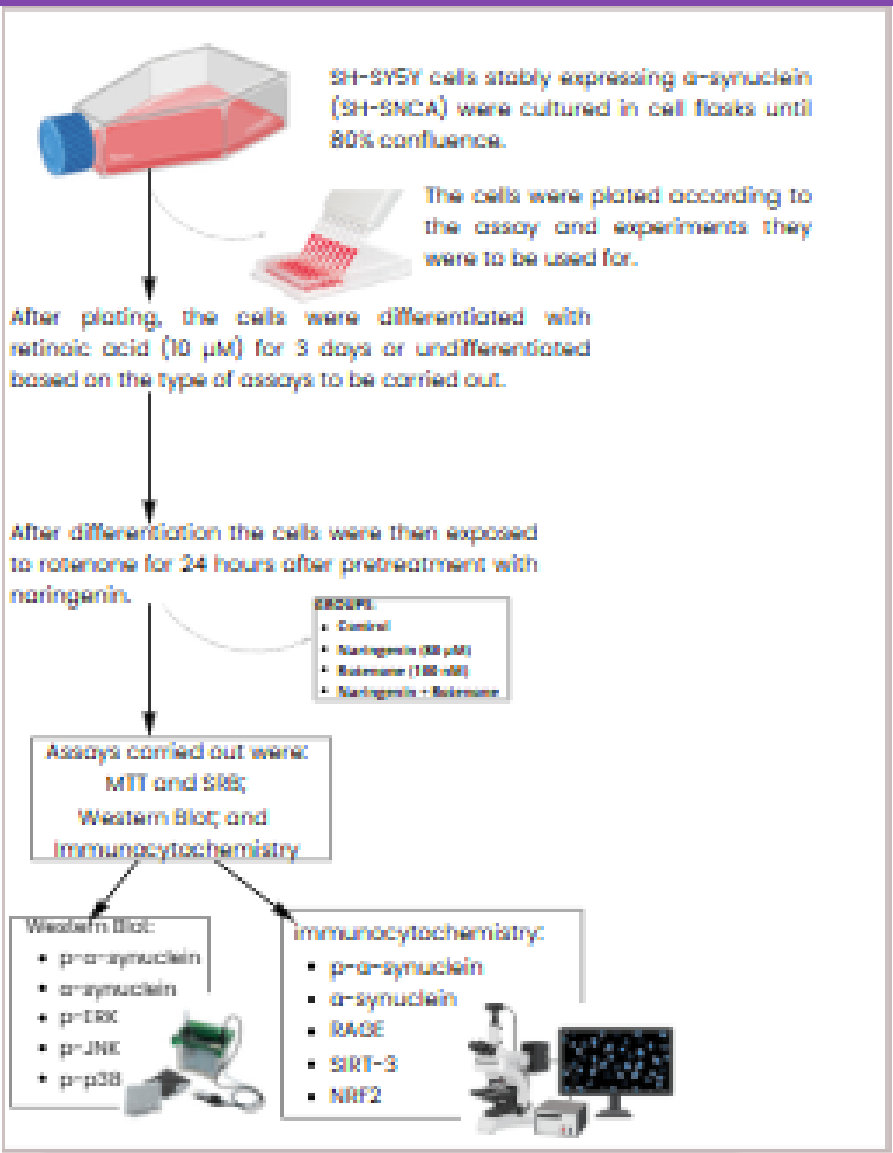
Excessive α -synuclein phosphorylation and aggregation contribute to neuronal dysfunction and disease progression. Although current therapies alleviate symptoms, they do not prevent neurodegeneration.

Naringenin, a citrus-derived flavonoid, possesses antioxidant, anti-inflammatory, and neuroprotective properties. This study evaluated whether naringenin protects SH-SY5Y cells overexpressing α -synuclein against rotenone-induced toxicity.

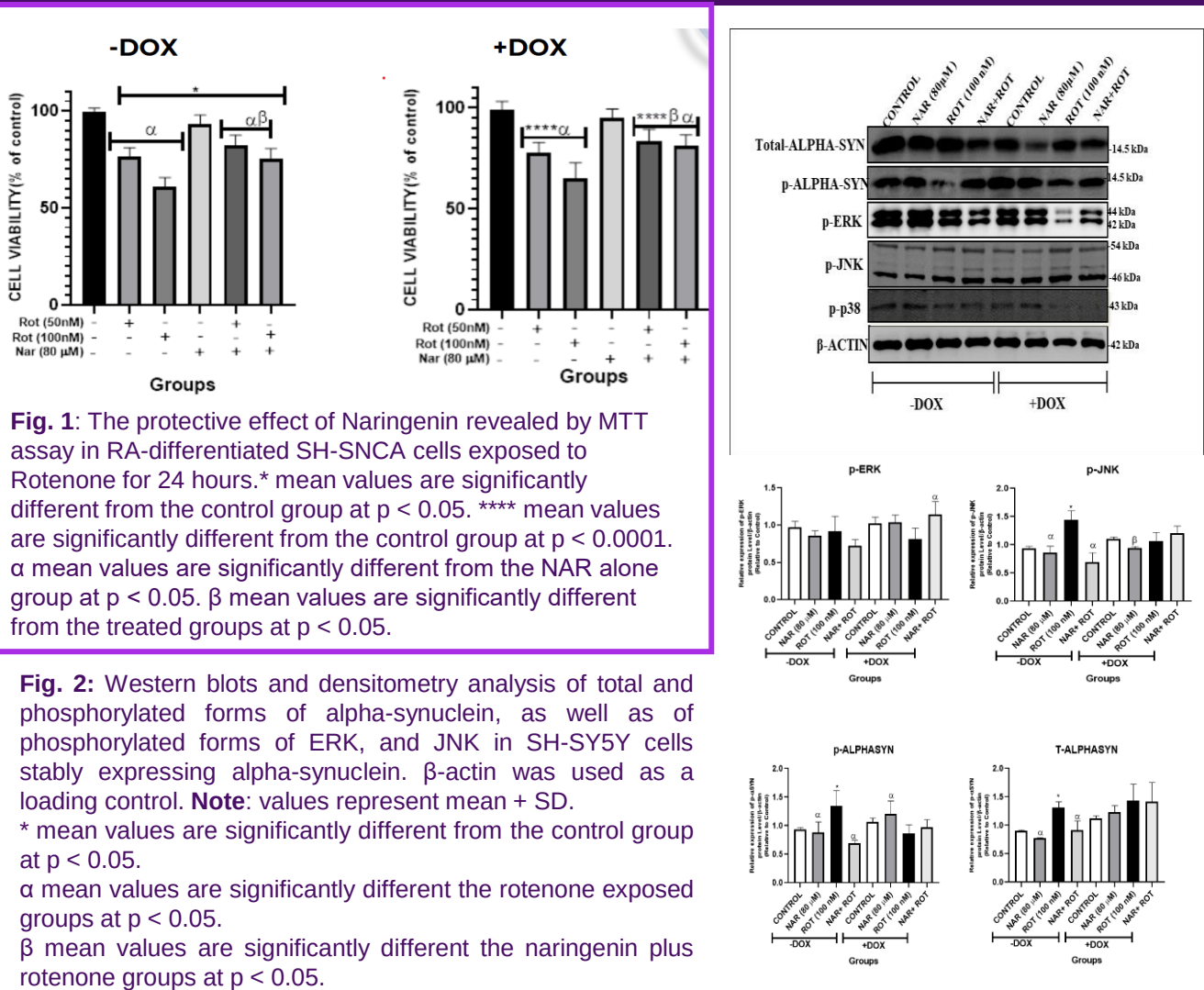
OBJECTIVES

- To evaluate the effect of naringenin on cell viability and survival in rotenone-exposed SH-SNCA cells using MTT and SRB assays.
- To determine whether naringenin modulates α -synuclein expression, phosphorylation, and RAGE-mediated inflammatory signaling.
- To elucidate the molecular mechanisms underlying its neuroprotection by examining changes in oxidative stress markers (SIRT-3, Nrf2) and apoptotic/stress kinases (p-JNK, p-p38, ERK).

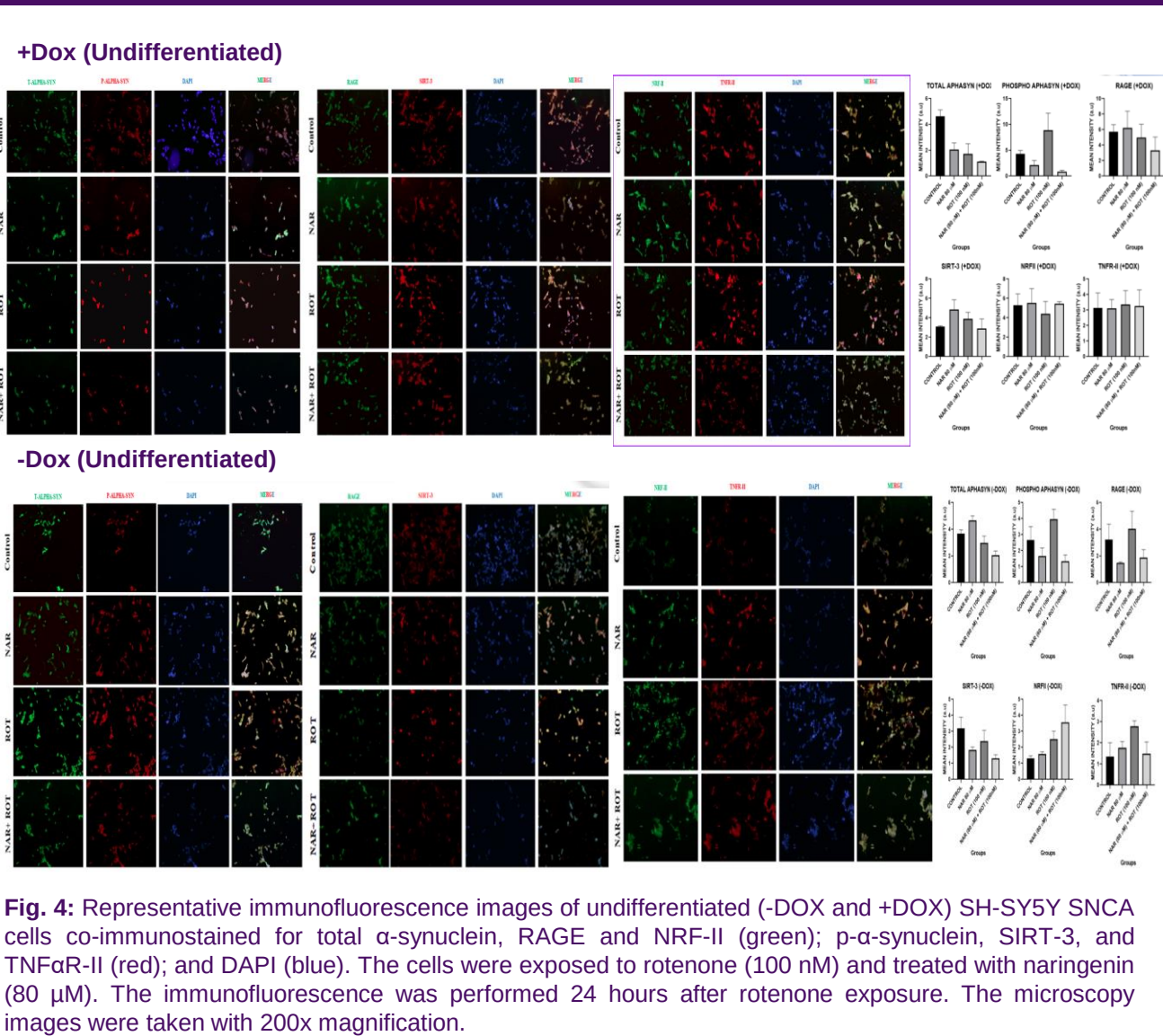
METHODS



RESULTS



RESULTS CONTD.



CONCLUSIONS

- Naringenin significantly protected SH-SY5Y cells overexpressing α -synuclein from rotenone-induced toxicity by enhancing cell viability and reducing α -synuclein aggregation.
- It modulated key oxidative, inflammatory, and apoptotic pathways through the activation of Nrf2 and inhibition of RAGE.
- These findings suggest naringenin as a promising neuroprotective compound for managing Parkinson's disease.

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