

Abstract

# Neuroprotective Potential of Cranberry Juice against Parkinson's Disease <sup>†</sup>

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**Abstract:** Parkinson's disease (PD) is a neurodegenerative disorder characterized by motor dysfunction associated with a loss of dopaminergic (DAergic) neurons in the substantia nigra pars compacta (SNpc) and the presence of Lewy bodies, mainly composed of aggregated  $\alpha$ -synuclein (ASN), in the brain [1]. Cranberry juice (CJ) is a rich source of polyphenols with strong antioxidant activity, which is believed to contribute to this fruit's wide range of health benefits. However, to date, our knowledge of cranberry neuroprotective potential is very scarce and limited to only a few in vitro studies. Recently, we have reported that treatment with CJ controls oxidative stress in several organs, with the most noticeable effect occurring in the brain [2]. It has been shown that rats exposed to prolonged low-dose of rotenone (ROT) treatment develop PD-like neurodegeneration due to complex I inhibition and associated oxidative damage, ASN aggregation, and loss of DAergic neurons in the SNpc [3]. This study aimed to examine CJ's ability to modulate the apoptosis mechanism and thereby provide neuroprotection in an ROT-induced rat model of parkinsonism. Wistar rats were given treatment with CJ in a dose of 500 mg/kg b.w./day (*i.g.*) and injected with ROT (1.3 mg/kg b.w./day, *s.c.*) from the 11th day. The experiment lasted a total of 45 days, including 10 days pre-treatment with CJ and 35 days combined treatment with CJ and ROT. To evaluate its neuroprotective effect, a microscopic examination, determination of inflammation and apoptosis markers, and ASN level were performed in the midbrain. Our results indicated that the CJ treatment provided neuroprotection, as evidenced by an enhancement of neuronal survival, which correlated well with the decreased expression of pro-apoptotic caspase-9 and Bax and normalization of cytochrome c levels. Importantly, treatment with CJ declined  $\alpha$ -synuclein levels. The expression of tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ) was similar across all groups, with no statistically significant differences.

**Keywords:** cranberry juice; polyphenols; neuroprotective; rotenone;  $\alpha$ -synuclein; neuronal survival



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**Supplementary Materials:** The following are available online at <https://www.mdpi.com/article/10.3390/IECBS2021-10673/s1>, Figure S1: Apoptosis marker and  $\alpha$ -synuclein, Figure S2: Representative photomicrographs (\* 40) of H&E stained midbrain sections of rats.

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**Informed Consent Statement:** Not applicable.

**Data Availability Statement:** Original data are available with the authors according to their contribution but not archived in databases elsewhere.

**Conflicts of Interest:** The authors declare no conflict of interest.

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