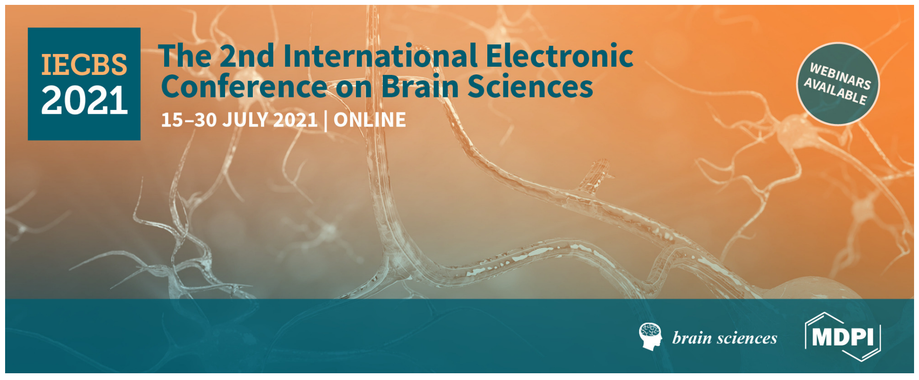

NEUROPROTECTIVE POTENTIAL OF CRANBERRY JUICE AGAINST PARKINSON'S DISEASE

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INTRODUCTION

Cranberry juice (CJ) is a rich source of polyphenols with strong antioxidant activity 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 in the brain. [1].

AIM

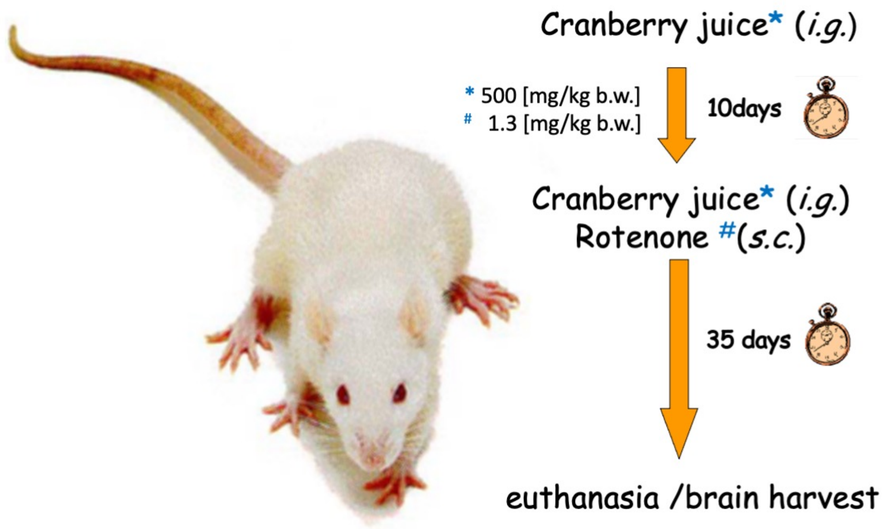
The present studies were designed to examine the capability of CJ for protection against Parkinson's disease in a rat model of parkinsonism induced by rotenone (ROT).

MATERIALS AND METHODS

Cranberry Juice

Commercial 6-fold concentrated cranberry juice (CJ) rich in quercetin, epicatechin, chlorogenic acid, cyanidin-3-O-glucoside [1] was obtained from Alter Medica (Zywiec, Poland).

EXPERIMENTAL DESIGN



WESTERN BLOTTING

Western blot analysis was performed to determine the Bax, caspase-9, cytochrome c, and α-synuclein protein levels in the midbrain. The samples were separated on 16% SDS-PAGE gels and transferred to nitrocellulose membranes. After blocking with bovine serum albumin, the proteins were probed with rabbit Bax, caspase-9, cytochrome c, and α-synuclein (Cell Signaling Technology) antibodies. As the secondary antibodies, anti-rabbit IgG HRP-linked antibody (Cell Signaling Technology) was used. The GAPDH was used as an internal control.

ELISA ASSAY

To quantify tumor necrosis factor-alpha (TNF-α) ELISA kit (RTA00, Bio Techne-R&D System) was used.

HISTOPATHOLOGICAL EXAMINATION

Samples of the midbrain were stained with hematoxilin & eosin (H&E) and examined by light microscopy.

SUMMARY AND CONCLUSIONS

1. CJ treatment provided neuroprotection as evidenced by enhancement of neuronal survival, which correlated well with decreased expression of pro-apoptotic caspase-9 and Bax and normalization of cytochrome c level.
2. Importantly, treatment with CJ declined α-synuclein level.
3. The expression of TNF-α was similar across all groups with no statistically significant differences (data not shown).

ACKNOWLEDGEMENTS

altermedica LABORATORIES

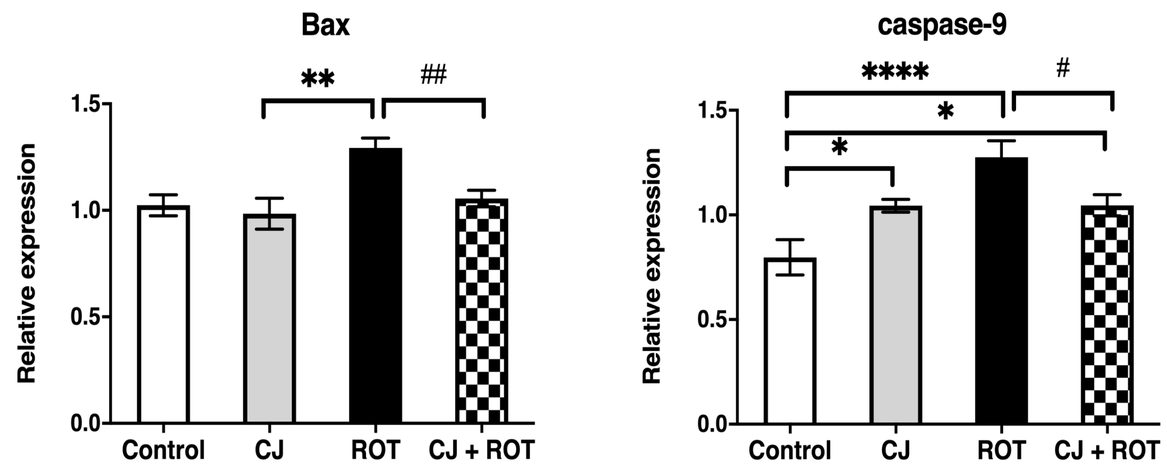
REFERENCES

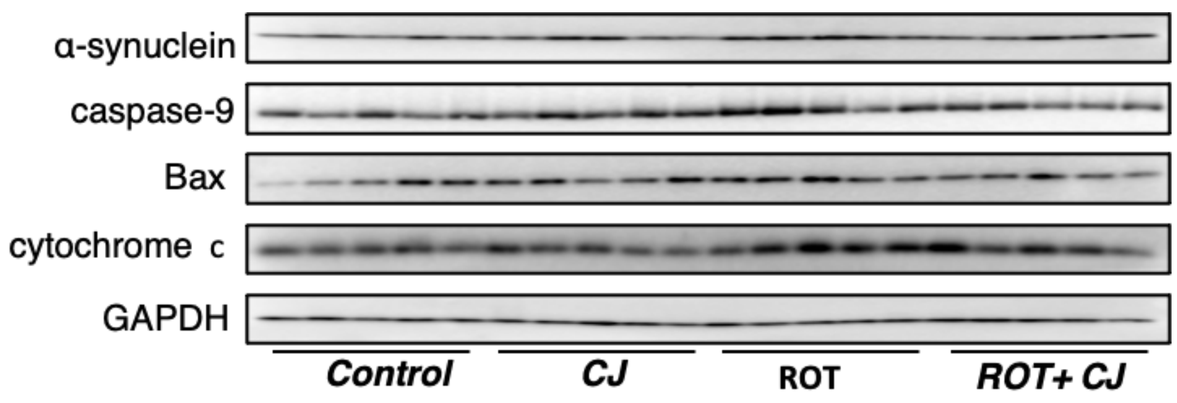
[1] Kurpik, M.; Zalewski, P.; Kujawska, M.; Ewertowska, M.; Ignatowicz, E.; Cielecka-Piontek, J.; Jodynis-Liebert, J. Can Cranberry Juice Protect against Rotenone Induced Toxicity in Rats? Nutrients 2021, 13, 1050. https://doi.org/10.3390/nu13041050

RESULTS

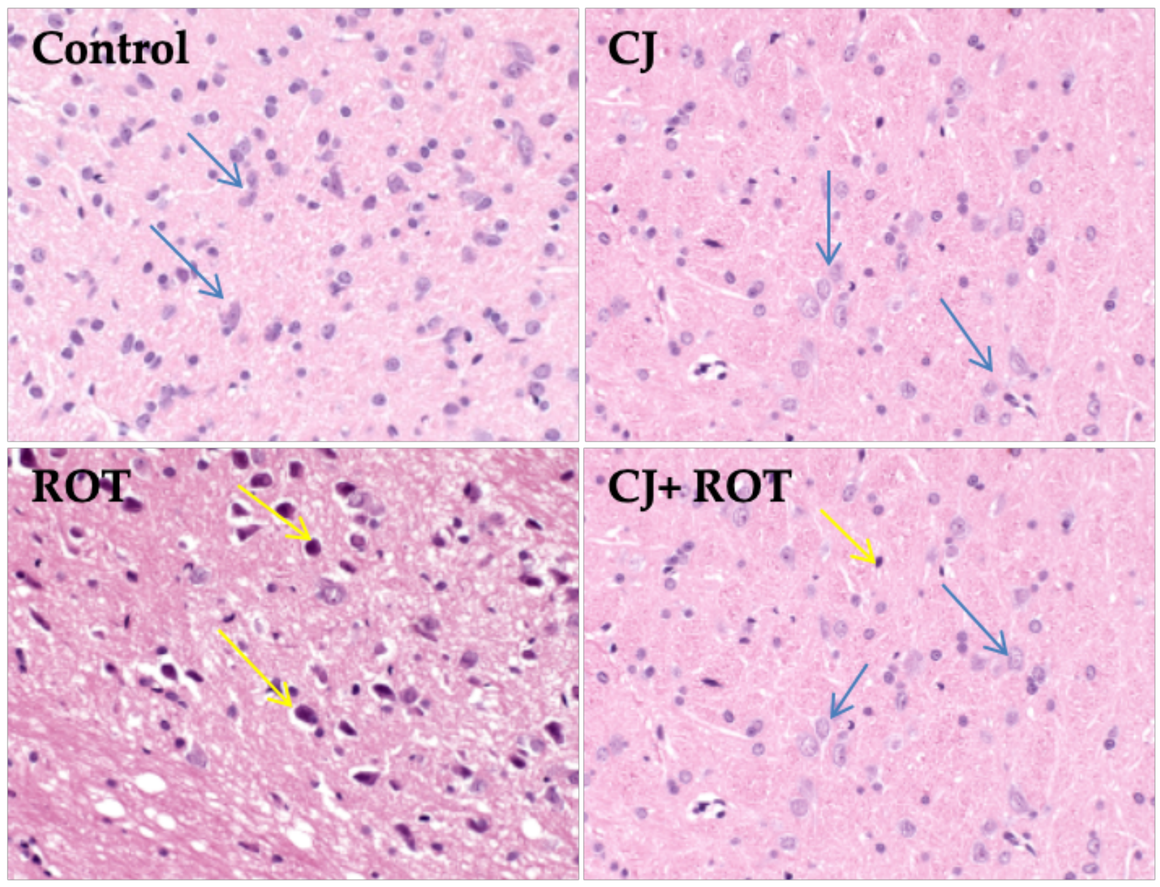
Apoptosis markers and α-synuclein

Data are presented as mean values ± SEM of five rats per group and analyzed using one-way analysis of variance (ANOVA) followed by Fisher's LSD test.
*** - p < 0.001 vs. Control; ** - p < 0.01 vs. Control; * - p < 0.05 vs. Control
# - p < 0.05 vs. ROT.





HISTOPATHOLOGICAL FINDINGS



Representative photomicrographs (× 40) of H&E stained midbrain sections of rats.

Control and cranberry juice alone treated rats (CJ) show normal neurons (blue arrows). Rotenone (ROT) administration caused prominent degeneration of neurons (yellow arrows). Rats treated with cranberry juice and rotenone (CJ + ROT) show normal neurons (blue arrows) and a few cells with signs of degeneration (yellow arrows).