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levels of methylmalonate and homocysteine in Parkinson's disease, progressive supranuclear palsy and amyotrophic lateral sclerosis. Dementia and geriatric cognitive disorders, 29(6), pp.553-59. 14. Quadri, P., Fragiacomo, C., Pezzati, R., Zanda, E., Forloni, G., Tettamanti, M. and Lucca, U., 2004. Homocysteine, folate, and vitamin B-12 in mild cognitive impairment, Alzheimer disease, and vascular dementia. The American journal of clinical nutrition, 80(1), pp.114-22. 15. Morris, M.S., 2003. Homocysteine and Alzheimer's disease. The Lancet Neurology, 2 (7), pp.425-28. 16. Mattson, M.P. and Shea, T.B., 2003. Folate and homocysteine metabolism in neural plasticity and neurodegenerative disorders. Trends in neurosciences, 26(3), pp.137-46. 17. Lamberti, P., Zoccolella, S., Armenise, E., Lamberti, S.V., Fraddosio, A., De Mari, M., Iliceto, G. and Livrea, P., 2005. Hyperhomocysteinemia in l-dopa treated Parkinson's disease patients: effect of cobalamin and folate administration. European journal of neurology, 12(5), pp.365-68. 18. O'Suilleabhain, P.E., Sung, V., Hernandez, C., Lacritz, L., Dewey Jr, R.B., Bottiglieri, T. and Diaz-Arrastia, R., 2004. Elevated plasma homocysteine level in patients with Parkinson disease: motor, affective, and cognitive associations. Archives of neurology, 61(6), pp.865-68. 19. Zoccolella, S., dell'Aquila, C., Abruzzese, G., Antonini, A., Bonuccelli, U., Canesi, M., Cristina, S., Marchese, R., Pacchetti, C., Zagaglia, R. and Logroscino, G., 2009. Hyperhomocysteinemia in levodopa-treated patients with Parkinson's disease dementia. Movement Disorders, 24(7), pp.1028-33. 20. Hassin-Baer, S., Cohen, O., Vakil, E., Sela, B.A., Nitsan, Z., Schwartz, R., Chapman, J. and Tanne, D., 2006. Plasma homocysteine levels and Parkinson disease: disease progression, carotid intima-media thickness and neuropsychiatric complications. Clinical neuropharmacology, 29(6), pp.305-11.

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Physiological level of melatonin and its role on pathophysiological processes in Parkinson's disease: evidences from experimental parkinsonism and Parkinson's disease patient population

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Objective: To investigate the role of circulating levels of melatonin in relation with behavioural and biochemical dysfunction in experimental model of PD. Also to investigate the circulating levels of melatonin in subjects with PD and to correlate with disease severity.

Background: Parkinson's disease (PD), with motor and non-motor disabilities have abnormal circadian rhythm disturbances. Circulating melatonin is closely associated with circadian rhythms.

Methods: The PD neurotoxin, 1-methyl-4-phenyl-1,-2,3,6-tetrahydropyridine (MPTP) is administered at different doses (20 or 30 mg/kg) in pinealectomized mice. Behavioral assays and neurotransmitters estimation employing HPLC-electrochemical detection procedure were performed on the 6thday and 7thday, respectively. Plasma melatonin level from mice and PD patients are estimated using melatonin assay kit, and severity of parkinsonian symptoms are measured with Hoehn and Yahr scale.

Results: In human PD patients, melatonin concentration in plasma increased with severity of parkinsonian motor symptoms. MPTP administration in pinealectomized mice could not show any significant alterations on behaviour parameters and striatal neurotransmitter levels from the experimental controls.

Conclusion: Decreased levels of circulating melatonin could not show any sensitivity to MPTP neurotoxicity in pinealectomized mice, whereas plasma melatonin level increased with degree of severity of parkinsonian motor symptoms in human PD patients.

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Rotenone-induced α-syn aggregation promotes mitochondrial dysfunction via Parkin/Pink1 signaling in SNpc region: Implication for PD pathology

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Objective: In this study, we investigated the neuroprotective effects of *Tinospora cordifolia* and the underlying mechanisms in classic rotenone-induced Parkinsonism.

Background: Phosphorylated α-syn (pS129) accumulation as well as parkin downregulation are associated with Parkinson's disease (PD) pathogenesis. *Tinospora cordifolia* has emerged as a novel medicinal plant that protects neurons from oxidative stress and mitochondrial dysfunction.

Methods: Mice were divided into four experimental groups: control, rotenone (2 mg/kg body wt., subcutaneous), *Tinospora cordifolia* extract (TCE, 200 mg/kg body wt., oral) + rotenone, and TCE only]. Mice were pre-treated with TCE for a week and then simultaneously injected with ROT for 35 days.

Results: TCE administration significantly improved locomotor performance and increased tyrosine hydroxylase (TH) expression in the substantia nigra pars compacta (SNpc) of rotenone-intoxicated mice. Furthermore, TCE improved mitochondrial dysfunction *via* counteracting the decline in mitochondrial electron transport chain complex activity evoked by ROT. Similarly, TCE suppressed ROT-induced imbalance of Bax/Bcl-2 ratio and activation of caspase-3. Furthermore, TCE also significantly decreased the expressions of caspase-3, caspase-9, and increased pink1 and parkin expression.

Conclusion: The Bax/Bcl-2 ratio, mitochondrial dysfunction, and expression of caspase-3, and caspase-9 were seen to be significantly increased on rotenone intoxication. The expression of pink1 and parkin are also decreased in ROT group leading to mitochondrial dysfunction. However, TCE was potent in protecting the neurons against rotenone-induced cytotoxicity through the regulation of oxidative stress-mediated mitochondrial dysfunction and apoptosis in the mouse model of PD.

Taken together, our results suggested that TCE attenuated rotenone-induced oxidative stress, through the regulation of mitochondrial functions.

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Glial neutrophic factor as a nonspecific factor in the progression of Parkinson's disease

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Objective: To study the dynamics of the level of glial neurotrophic factor in blood plasma in patients with Parkinson's disease.

Background: The progression of Parkinson's disease (PD) depends on many factors, including trophic factors. Glial neutrophic factor (GDNF) is the main trophic factor in dopaminergic neurons.

Methods: We examined 48 patients with PD aged 32 to 60 years (mean age 46.0±4.8), including 24 men and 24 women. The control group consisted of 10 patients without PD, which matched in age and gender. All patients underwent clinical neurological, neuroimaging and biochemical studies. In the dynamics of the level of GDNF was determined by enzyme immunoassay. The second re-analysis was performed after 1.5 years, in the same patients.

Results: The obtained results showed that in the blood serum of patients of the main group, the amount of glial neurotic factor was 46.8±7.5 pg/ml, in the control group 80.668±5.2 pg/ml ($p<0.05$). In the main group, the content of GDNF was two times lower than in the control group. After 1.5 years in PD patients, the GDNF level was 31.89±3.8 pg/ml, while in the control group the GDNF level was 76.7±4.3 pg/ml. Glial neurotrophic factor responds to the trophism of dopaminergic neurons, and the death of these neurons depends on the level of the factor. A decrease in GDNF levels aggravates the clinical course of motor and non-motor symptoms of PD.

Conclusion: Based on this study, it can be concluded that glial neurotrophic factor may be a non-specific factor in the progression of Parkinson's disease.

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Influence of WT and A53T alpha-synuclein overexpression in SH-SY5Y and DA neurons derived from iPSCs on its intracellular calcium response its related toolkit and on membrane fluidity

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Objective: To assess the effect of Wildtype (WT) and A53T mutant α-synuclein(α-Syn) on expression of phosphorylated a-Syn serine