ABSTRACT E-BOOK

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ported. The present study was focused to investigate the effects of NPFCs (metanil yellow-MY, malachite green-MG and sudan III-SIII) on learning and memory of rats, oxidative stress, mitochondrial complex activity, neuroenzyme activity, neurotransmitters and histopathology in the hippocampus of rats.
Methods: Rats were divided into 5 groups and treated with MY (430 mg/kg), MG (13.75 mg/kg), SIII (250 mg/kg), mixture (YGR) (MY 143.33 + MG 4.52 + SIII 83.33 mg/kg) and 1 % gum acacia serve as control p.o. for 60 days. Learning and memory were assessed through Y maze and Morris water maze then one rat from each group was transcardially perfused for histopathology and the remaining were decapitated for hippocampus isolation and processed for biochemical, neurochemicals, neurotransmitters analysis through valid standard protocols.
Results: The treatment groups showed impairment in learning and memory and a significantly enhanced oxidative stress (higher lipid peroxidation, decreased level of reduced glutathione, superoxide dismutase and catalase activity), reduced mitochondrial complex enzyme activity (I and II), higher acetylcholinesterase activity, lower monoamine Oxidase –B activity in the hippocampus of rats. Levels of serotonin, dopamine and nor-adrenaline were higher in treatment groups compared to the control. Finally, significant damage in the architectures of neurons of hippocampus was observed in the treatment groups.
Conclusions: The results of the present study demonstrated that chronic exposure of NPFCs impaired learning and memory due to cholinergic and dopaminergic dysfunctions and an increase in the level of serotonin, dopamine and nor-adrenaline and neuronal damages due to enhanced oxidative stress and mitochondrial dysfunction which could be further associated with impaired motor dysfunction and pathological alterations like neurodegenerative diseases.

P 046
Correlation between NREM Sleep EEG characteristics and Mild Cognitive Impairment in patients with Parkinson's disease

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Background: Purpose of the study was to assess the relevance between NREM Sleep EEG characteristics and Mild Cognitive Impairment (MCI) in patients with Parkinson's disease (PD)
Methods: 34 patients were enrolled in this study and were divided into two groups according to the cognitive status. First group consists of 18 patients with PD-MCI while second group includes 16 patients with PD and normal cognitive function(PD-NCF). All patients underwent an overnight polysomnography study (PSG). Sleep EEG signals were removed and clarified from the PSG and subjected to a conventional power spectral analysis and detrended fluctuation analysis (DFA) during wakefulness, nonrapid eye movement (NREM) sleep, and rapid eye movement (REM) sleep.
Results: The alpha activity and fast ratio (alpha + beta/delta + theta) were lower during wakefulness and NREM sleep in patients with PD-MCI than in those with PD-NCF ($p < 0,05$). DFA increased in patients with PD-MCI during wakefulness and NREM sleep compared to those with PD-NCF ($p < 0,05$).
The results showed that PD-MCI patients had a decreased EEG fast ratio ($0,63 \pm 0,22$ vs $1,23 \pm 0,59$, $p = 0,001$) and increased DFA ($0.87 \pm 0,12$ vs $0,78 \pm 0,12$, $p = 0,005$) during NREM compared to PD-NCF patients

($p < 0{,}005$). Mild cognitive dysfunction was positively correlated with DFA in NREM ($r = 0{,}432$, $p = 0{,}005$) and negatively correlated with the fast ratio in NREM ($r = -0{,}524$, $p = 0{,}001$) in channel O1 during NREM sleep.

Conclusions: In conclusion, this study showed that the power spectral analysis and DFA characteristics of NREM sleep EEG were related to MCI in patients with PD. DFA can provide an estimation for cognitive function. Slowing of EEG activity during NREM sleep may reflect the decline in NREM physiological function and is therefore a marker in patients with PD-MCI.

P 047 (GPT)
The role of dopaminergic therapy on cognition in Parkinson's disease

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Background: Parkinson's Disease (PD) is a neurodegenerative disease that causes debilitating cognitive deficiencies in addition to motor impairments. Although dopaminergic medications are designed to improve motor symptoms, their effect on cognition is complex and varies based on individual differences. Recent studies have used verbal Digit Span as a tool to study working memory in PD patients and have shown that Digit Span Backward can be used as a screening tool for mild cognitive impairment (MCI) in PD.

Our previous work demonstrated that Digit Span performance could discriminate patients whose working memory benefits from dopaminergic therapy.

Our goal was to assess whether Digit Span performance reveals the dopamine effect on decision-making impacted by memories.

Methods: Participants (25 PD patients without MCI) performed a Digit Span task ON and OFF dopaminergic medication. While OFF medication using a median split, they were divided into low and high working memory capacity groups (LM and HM). Consequently, they performed a decision task and made choices between pairs of familiar food items. The reaction time (RT) and choice performance were measured.

Results: Working memory performance varied across participants. We found the performance improvement due to dopaminergic therapy in the Verbal Digit Task was restricted to LM group; the HM group was not affected. The decision-making task revealed that dopamine increased RT in the HM group more than in the LM group. This increase in RT only was beneficial for the LM group resulting in improved performance.

Conclusions: This study offers evidence that working memory measured with the Verbal Digit Span test can be used as a screening tool for the effect of dopamine on other cognitive tasks. Moreover, the dopaminergic medication affects decision-making performance in PD patients, but this effect depends on the underlying baseline of the PD patients.