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# MELATONIN PROTECTIVE EFFECT AGAINST Aβ- INDUCED NEUROTOXICITY AND MEMORY IMPAIRMENT IN ICV Aβ1-42 RAT MODEL WITH PINEALECTOMY

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Melatonin is a multifunctional molecule, and its role in the regulation and protection of the central nervous system is well documented. Melatonin has a higher preference than other available antioxidants in the therapeutic strategy of Alzheimer's disease (AD) due to its inhibitory effect on amyloid-beta (Aβ1-42) accumulations, low toxicity effect in high doses, and readily passage through blood-brain barrier. Therefore, pinealectomy appears to be a good model to study melatonin deficiency and pathway dysfunction in the pathogenesis of neurodegenerative diseases. The present study aimed to explore the role of melatonin on behavioral and biochemical changes in an Aβ1-42 model with concomitant melatonin deficit (pinealectomy+icvAβ1-42). Adult rats that undergone icv infusion of Aβ1-42/vehicle were injected with melatonin (50 mg/kg, intraperitoneally (i.p.)/vehicle about two hours before the onset of the dark phase for 40 days. The pinealectomy+icvAβ1-42 model disturbed the control on oxidative stress and led to Aβ1-42 accumulation in the frontal cortex and the hippocampus. Melatonin treatment mitigated an enhanced anxiety response, corrected memory decline, as well as suppressed Aβ1-42 accumulation and lipid peroxidation in the hippocampus. The present data suggest pinealectomy+icvAβ1-42 rat model as a more reliable AD model. Melatonin could substantially restore behavioral deficit via suppression of Aβ1-42 accumulation and disturbed oxidative stress homeostasis in the brain.

**Key words:** icvAβ1-42; pinealectomy; behavior; Aβ1-42 accumulation; oxidative stress; melatonin

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## Biography

Jana Tchekalarova is from Institute of Neurobiology, Bulgarian Academy of Sciences. He has attended many international conferences and published his research in many Journals.

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