Abstract

Aging is a process that is characterized by the progressive loss of the tissue and organ function. Aging causes changes in the brain size, vasculature and cognition.The brain shrinks with increasing age and there are changes at all levels from molecules to morphology. Age related oxidative stress is caused by combined effect of increased production of the free radicals, decreased antioxidant levels, reduced activities of antioxidant enzymes, and lessened repairs of oxidative damage. This study was aimed to evaluate anti-aging and antioxidant potential of sericin and Se-AgNPs on brain of experimental mice. For this purpose, artificial aging was induced in mice with the help of D-galactose. Mice were divided in 9 groups. Group 1 was treated with D-galactose alone, group two and three were prevention groups and were treated with sericin (120 mg/Kg) and Se-AgNPs (120 mg/Kg). Group 4 and 5 were given sericin (120 mg/Kg and (150 mg/Kg), while group 6 and 7 were treated with Se-AgNPs (120 mg/Kg and 150 mg/Kg). group 8 was treated with metformin (1 mg/Kg) and while group 9 was kept without any treatment. To evaluate effect of different doses of our treatment seven different parameters e.g; glutathione (GSH), glutathione Peroxidase (GSH Px), low density lipoproteins(LDL), high density lipoproteins (HDL), superoxide dismutase (SOD), catalase (CAT) and Malondialdehyde (MDA) levels were measured. D-galactose treatment caused a decrease in GSH, CAT, SOD and GSH Px levels while there was an increase in LDL, HDL and MDA levels. Treatment with Se-AgNPs in prevention group caused highest significant decrease in MDA level (6.96±0.57 mmol/ml). Treatment of mice with d-galactose caused a significant decrease in GSH level in brains of mice (20±4.26 mg/l), while treatment with Se-AgNPs in prevention group caused a highest significant increase in GSH level (36.2±0.9 mg/l). Treatment of mice with d-galactose causes a significant decrease in catalase level in brain of mice (0.58±0.03 mmol/ml), while in prevention group treatment with Se-AgNPs caused a highest significant increase in catalase value compared to all other treatments (3±0.153 mmol/ml). Treatment of mice with d-galactose caused a significant decrease in level of SOD (61.4±2.11 U/ml), but treatment of these mice with Se-AgNPs in prevention group caused a highest significant increase in SOD levels in brain of these mice (230.4±13.75 U/ml). Treatment of mice with D-galactose caused a significant increase in LDL level (30.6±1.02 mg/dl) and treatment of these mice with Se-AgNPs in prevention group caused a highly significant increase in LDL level (6.1±0.43 mg/dl). Level of HDL was significantly increased in brain of mice treated with d-

galactose (147.2±3.65 mg/dl), while Se-AgNP treatment in prevention group caused a highly significant decrease in HDL level in brain of mice (67.4±4.26 mg/dl). D-galactose treatment caused a significant decrease in GSH Px level (64.8±2.35 U/l), while treatment with SeAgNPs in prevention group caused a highly significant increase in GSH Px level (153.8±3.00 U/l).Our data provides experimental proof that silk sericin and Se-AgNPs exhibit the antioxidant potential that can eliminate the oxidative stress induced by ROS, as a result of D-galactose administration. However, further studies are warranted to investigate the anti-aging activity exhibited by silk sericin and Se-AgNPs.