Research Article

Purple Sweet Potato Color Ameliorates Cognition Deficits and Attenuates Oxidative Damage and Inflammation in Aging Mouse Brain Induced by D-Galactose

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Purple sweet potato color (PSPC), a naturally occurring anthocyanin, has a powerful antioxidant activity in vitro and in vivo. This study explores whether PSPC has the neuroprotective effect on the aging mouse brain induced by D-galactose (D-gal). The mice administrated with PSPC (100 mg/kg·day, 4 weeks, from 9th week) via oral gavage showed significantly improved behavior performance in the open field and passive avoidance test compared with D-gal-treated mice (500 mg/kg·day, 8 weeks). We further investigate the mechanism involved in neuroprotective effects of PSPC on mouse brain. Interestingly, we found, PSPC decreased the expression level of glial fibrillary acidic protein (GFAP), inducible nitric oxide synthase (iNOS), and cyclooxygenase-2 (COX-2), inhibited nuclear translocation of nuclear factor-kappaB (NF-κB), increased the activity of copper/zinc superoxide dismutase (Cu/Zn-SOD) and catalase (CAT), and reduced the content of malondialdehyde (MDA), respectively. Our data suggested that PSPC attenuated D-gal-induced cognitive impairment partly via enhancing the antioxidant and anti-inflammatory capacity.

1. Introduction

There is considerable evidence that oxidative stress plays an important role during the pathogenesis of age-associated or neurodegenerative disease [1]. Oxidative stress is caused by the imbalance between the reactive oxygen species (ROS) production and ROS elimination in the biological system. And it leads to oxidative damage to cell and tissue paralleled by modifications in the morphology and function, resulting in aging and premature cell death. The aging animal is easily subjected to the alteration of observed behavior such as the reduction of spontaneous behavior, the decline of learning, and memory function. Now a decline in memory and cognitive function is considered to be a consequence of aging and age-related neurodegenerative disorders [2, 3]. The age-related impairment in learning and memory may be relieved by antioxidant treatment [4].

One approach to protect against ROS in aging and neurodegenerative disorders is to enhance biological defense activities via antioxidants. Recently, most efforts to prevent and treat neurodegenerative disorders focus on dietary antioxidant components, including vitamins and phenolic compounds [5]. Anthocyanins are a class of naturally polyphenolic compounds that give the intense color to many flowers and fruits in the high plants. They have been reported to show several effectively pharmacological properties such as anti-inflammation, antioxidation [6, 7], and antimutagen [8, 9]. Purple sweet potato (*Ipomoea batatas*) has been regarded as an abundant source of stable anthocyanins [10] and has been attracted extensive attentions. The previous studies on purple sweet potato color (PSPC) have focused on its stability, structure, and physiological functionality and functional food exploitation [11–13]. However, it is still uncertain whether PSPC

could improve spontaneous behavior, learning, and memory function, and whether PSPC could have a neuroprotective effect on brain damage induced by the oxidation and inflammation.

D-galactose (D-gal) is a naturally occurring chemical substance in the body; however, at high levels, it can cause the accumulation of ROS, finally resulting in oxidative stress. Recently, D-gal was used to inject mice or rats for pharmacological studies. Evidence has shown that D-gal caused aging-related changes including the increase of ROS and the decrease of antioxidant enzyme activity [14, 15]. D-gal could simultaneously cause the behavioral and neurochemical changes [15–18]. The D-gal-injected mouse has been utilized to be a model for antiaging pharmacology and brain aging studies.

In this study, we investigated the neuroprotective effect of PSPC on impaired explortary behavior, learning, and memory in the D-gal-treated mouse and the underlying neuroprotective mechanism of PSPC on mouse brain.

2. Material and Methods

2.1. Animals and Administration. Male Kunming mice (30.23 ± 5.12 g body weight) were purchased from the Branch of National Breeder Center of Rodents (Shanghai, China). They were housed in a ventilated, temperature-controlled (23 ± 1°C), and standardized sterile animal room. Eight mice were housed per cage and had free access to food and water under 12 hours light/dark schedule (lights on from 08:30 to 20:30 hour). After acclimatization to the laboratory for one week, group 1 and group 3 were injected (s.c.) with D-gal at dose of 500 mg/(kg.day) (Sigma-Aldrich, MO, USA) [19], and group 2 and group 4 were injected with 0.9% saline for 8 weeks. Then group 1 and group 4 were administrated by oral gavage with PSPC (Ipomoea batatas; Tsingtao Pengyuan Natural Pigment Research Institute, China; the major components of PSPC by HPLC analysis are cyanidin acyl glucosides and peonidin acyl glucosides (> 90%), and the others are other flavonoids) in distilled water containing 0.1% Tween 80 at dose of 100 mg/(kg.d); at the same time other two groups received distilled water containing 0.1% Tween 80 without PSPC for another four weeks. Before experiment, the mean body weights of each group were examined: control group: 30.08 ± 3.15 g; D-gal group: 31.13 ± 4.22 g; D-gal + PSPC group: 29.82 ± 4.71 g; PSPC group: 30.16 ± 2.27 g. After 12-week administration, the mean body weights of each group were also examined: control group: 38.28 ± 2.14 g; D-gal group: 39.13 ± 1.66 g; D-gal + PSPC group: 39.82 ± 2.59 g; PSPC group: 38.03 ± 2.33 g. The administration of D-gal and/or PSPC does not significantly influence on the body weight change and the feeding in the mice. The procedures used in the experiment were conducted according to the regulations of the ethics committee of the International Association on the use and care of laboratory animals and were approved by the respective university committees for animal experiments.

2.2. Behavioral Tests

2.2.1. Open-Field Testing. The open field has proven useful for evaluation of the effects of drugs on behavior. Locomotor activity and exploratory behavior were examined using a circular arena measuring 50 cm in diameter and 30 cm in height with a 40 W bulb (3000 lux). The floor of the arena was equally divided into 21 units by white lines. Tests were carried out in the animal room from 8:30 to 16:00 as reported previously [20]. Each mouse was placed at the center of the arena. The spontaneous behavioral variables were classified into 4 mutually exclusive categories, including the number of times of rearing, leaning, grooming, and ambulation recoded for 5 minutes.

2.2.2. Passive-Avoidance Testing. The apparatus consisted of an illuminated compartment (11.5 cm × 9.5 cm × 11 cm) and a dark compartment (23.5 cm × 9.5 cm × 11 cm). The two compartments were separated by a sliding door, and the dark one contained a metal floor that could deliver footshocks. The illuminated compartment was lit with a 25 W lamp. The passive avoidance test was performed as described previously [21]. The mouse was firstly placed in the dimly lit room to acclimate the new environment with the apparatus for 0.5 hour before entering the dark compartment. Latency was defined as the interval between the initial opening of the door and the fully entry of the mouse to the dark compartment. The mouse was individually placed in the illuminated compartment facing away from the door. Once the mouse escaped to the illuminated compartment, the door was closed and a mild footshock (0.3 mA, 50 Hz, 5 seconds) was triggered. Immediately the mouse was removed from the apparatus. To carry out the retention trial, the mouse was placed once again in the illuminated compartment 72 hours later. The retention trial followed the method mentioned above in the footshock. The latency (avoidance latency) was recorded. The retention session lasted a maximum ("cut-off") of 300 seconds. The test was conducted in 2 days immediately following the open-field testing.

2.3. Tissue Homogenarate. For biochemical analysis, animals were deeply anesthetized and sacrificed. Brains were promptly dissected and perfused with 50 mM cold phosphate buffer saline solution (PBS, pH7.4). Brains were homogenized in 1/5 (w/v) PBS containing Complete Mini Protease Inhibitor Cocktail tablets (Roche Diagnostics Corporation, Indianapolis, IN, USA) with 10 strokes at 1200 rpm in a Potter homogenizer. Homogenates were divided into two portions. One part was directly centrifuged at 8000x g for 10 minutes at 4°C, and the supernatants were collected to measure MDA content. The other part was sonicated four times for 30 seconds with 20 seconds intervals using a VWR Branson Scientific ultrasonicator (VWR Int. Ltd, Merch House Pool, UK). The homogenates were centrifuged at 5000x g for 10 minutes at 4°C to obtain the supernatants for determination of Cu/Zn-SOD and CAT activities.

For western blot analysis, the brain tissues were dissected and homogenized in 1/3 (w/v) Tris-buffered saline (TBS) containing Complete Mini Protease Inhibitor Cocktail

tablets with 10 strokes at 1200 rpm in a Potter homogenizer. The homogenates were centrifuged at 10000x g for 10 minutes at 4°C. And the supernatants were collected and stored at −70°C for the measurement of GFAP, COX-2, and iNOS. Nuclear and cytoplasmic proteins were extracted using an NE-PER kit (Pierce Biotechnology, Inc., Rockford, USA). Nuclear proteins and cytoplasmic proteins were performed by western blot analysis of NF-κB nuclear translocation. Protein content was determined by using the BCA assay (Pierce Biotechnology, Inc., Rockford, USA).

2.4. Assay of Cu/Zn-SOD and CAT Activity. Cu/Zn-SOD and CAT activities were measured using chemicals purchased from Sigma Chemical Company (USA). Cu/Zn-SOD activity was spectrophotometrically monitored at 550 nm according to the method described by Sun et al. [22]. SOD activity was showed as U/mg protein with reference to the activity of a standard curve of bovine Cu/Zn-SOD under the same condition.

CAT activity was measured on the basis of Aebi [23]. Briefly, 50 μL sample and 0.65 mLof 50 mM PBS (PH7.0) were added to a quarts cuvette, and 0.3 mLof 30 mM hydrogen peroxide was used as the substrate. The light absorbance at 240 nm (25°C) was determined. Enzymatic activity was expressed as nM H_2O_2 decomposed/min/mg protein.

2.5. Measurement of MDA Level. MDA content was measured using chemicals purchased from Sigma Chemical Company (USA). The level of MDA in brain tissue homogenates was assessed as an index of lipid peroxidation by the method of Mihara and Uchiyama [24]. 500 μL homogenate was mixed with 3 mLof H_3PO_4 solution (1%, v/v) followed by addition of 1 mLof thiobarbituric acid solution (0.67%, w/v). Then the mixture was heated in a water bath (95°C) for 45 minutes. The colored complex was extracted into *n*-butanol and cooled to the room temperature. The absorption at 532 nm was measured using tetramethoxypropane as standard. MDA level was expressed as nmol per mg of protein.

2.6. Western Blot Analysis of GFAP, COX-2, iNOS, and NF-κB. Sample buffer (0.1 M Tris-HCl, 20% glycerol, 4% SDS, and 0.01% bromophenol blue) was added to aliquot of the supernatant fraction from the lysates. Samples were boiled for 5 minutes. Aliquots of 80 μg protein were subjected to 10% SDS-PAGE. The separated proteins were transferred to PVDF membrane (Roche Diagnostics Corporation, Indianapolis, IN, USA) for 1 hour at 350 mA. The membrane was preblocked with 5% nonfat milk or 5% w/v BSA in TBS containing 0.1% Tween 20, incubated with the primary antibody at 4°C with gentle shaking, overnight. Each membrane was washed 3 times for 5 minutes and incubated with the secondary horseradish peroxidase-linked antibody (Santa Cruz Biotechnology, CA and Cell Signaling Technology, Beverly, MA, resp.). Quantitation of detected bands was performed with the Scion Image analysis software (Scion Corp., Frederick, MD, USA). Each density was normalized by corresponding β-actin (40 μg sample) or β-tubulin (40 μg

sample) as an internal control. We standardized the density of control group for relative comparison as 1.0 to compare other group.

2.7. Statistical Analysis. All statistical tests were performed with the statistical program SPSS 11.5. One-way analysis of variance (ANOVA) was applied with Newman-keuls or Tukey's HSD posthoc comparisons. The data were expressed as means ± SEM. Statistical significance was set at $P < .05$.

# 3. Results

*3.1. Effect of PSPC on the Behavior of D-Gal-Treated Mouse*

3.1.1. Open-Field. Open-field behaviors were observed to identify the behavioral difference in response to a novel environment. The behaviors included exploratory, adaptive, and locomotor abilities of animals expressed by line crossing, rearing/leaning and grooming. The data presented in Figure 1 indicated that, in comparison to the control group with injection of saline (0.9%), the behavior activities of mice group subcutaneously injected by using D-gal (D-gal model) cut down on the activities of line crossing ($F(3, 28) = 6.210$, $P < .01$), rearing/leaning ($F(3, 28) = 6.292$, $P < .01$), and grooming ($F(3, 28) = 7.484$, $P < .001$). This result showed that the impairment of the exploration activities appeared in the mice treated with D-gal. When D-gal-induced mice were received 100 mg/(kg.d) of PSPC, their activities of crossing ($P < .001$), rearing/leaning ($P < .001$), and grooming ($P < .001$) were significantly increased. However, there was no statistically significant between the control group and the study group only received PSPC.

3.1.2. Passive-Avoidance Testing. In the initial trial, the passive-avoidance latency showed no difference among the four groups ($F(3, 28) = 0.622$, $P > .05$) (See Figure 2). However, the latency in the 72 hours retention trial was significantly decreased ($F(3, 28) = 6.476$, $P < .01$) in the D-gal-treated mice, as compared with the control. In contrast to the D-gal model, the latency of the D-gal-treated mice fed with 100 mg/(kg.d) PSPC for 4 weeks was significantly increased ($P < .05$). However, no significant difference was found between the study group only treated with PSPC and the control group.

3.2. Effect of PSPC on the Cu/Zn-SOD and CAT Activity. Cu/Zn-SOD and CAT are two kinds of important enzymes in the antioxidant defense system. Cu/Zn-SOD can catalyze the conversion of O_2^- into H_2O_2; furthermore, CAT converts H_2O_2 to H_2O and O_2. To determine whether PSPC could ameliorate oxidative damage in the brain of D-gal-treated mouse, we measured the activities of antioxidant enzymes Cu/Zn-SOD and CAT. Figure 3 showed the activities of Cu/Zn-SOD and CAT in mouse brain. We found from the two figures, compared with control, that D-gal treatment significantly decreased Cu/Zn-SOD ($P < .001$, Figure 3(a)) and CAT ($P < .05$, Figure 3(b)) activities, while PSPC could reverse the above changes to the normal level. And there was
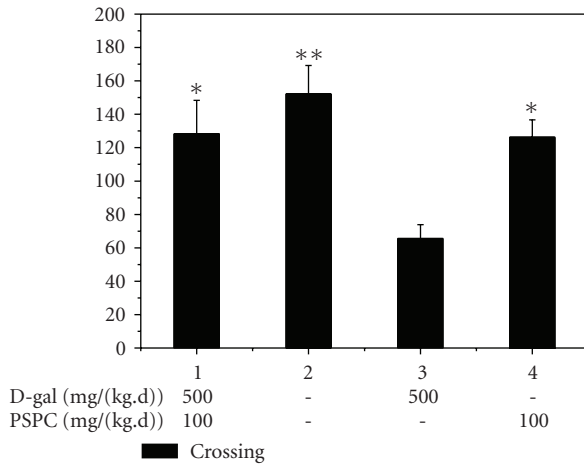
(a)
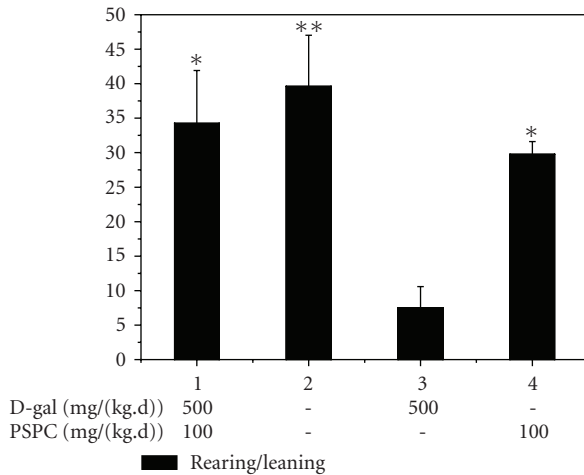
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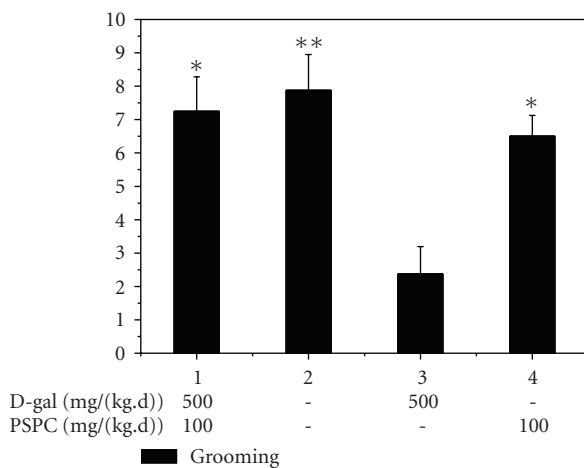
(c)

Figure 1: Effects of PSPC on open-field behaviors of mice as measured by (a) the number of grid crossing, (b) rearing and leaning, and (c) grooming. All values are expressed as means ± SEM ($n = 8$). *$P < .05$, **$P < .01$, ***$P < .001$ versus D-gal model. Scale bars: 100 μM.
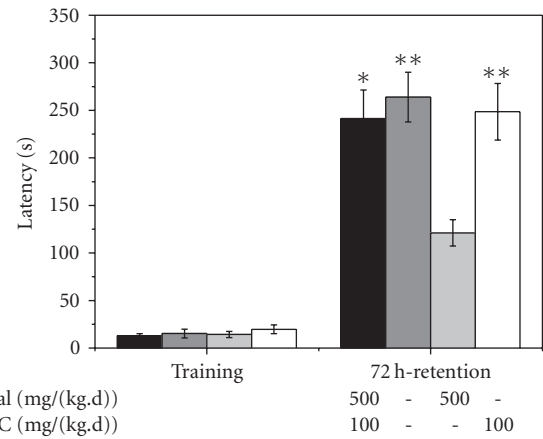

Figure 2: Performance in the passive avoidance test. All values are expressed as means ± SEM ($n = 8$). *$P < .05$, **$P < .01$, ***$P < .001$ versus D-gal model. Scale bars: 100 μM.

no significant difference between PSPC group and control group.

3.3. Effect of PSPC on the MDA Content. MDA is a highly reactive three carbon dialdehyde produced as a byproduct of polyunsaturated fatty acid peroxidation and arachidonic acid metabolism and readily combines with several functional groups on molecules including proteins, lipoproteins, and DNA. Therefore, MDA is often measured as a major marker of lipid peroxidation in the aging tissue [25]. As shown in Figure 4, MDA level in D-gal-treated group was higher than that in the control group ($P < .001$). PSPC depressed MDA enhancement in mouse brain (D-gal/PSPC versus D-gal model, $P < .01$). While compared with control group, both D-gal/PSPC group and PSPC group did not show significant change.

3.4. Effect of PSPC on the Expression of iNOS, COX-2, and Nuclear Translocation of NF-κB. A marker of CNS inflammation is the activation of microglia that produces neurotoxic factor nitric oxide (NO). Generally, the inducible NOS could generate NO, which played an important role in inflammatory response [26]. Our Western blot analysis revealed that D-gal upregulated the expression of iNOS protein ($P < .01$ versus control group), and its level was significantly decreased after PSPC administration. Compared with control group, there was no significant difference in D-gal/PSPC group and PSPC group (Figure 5(a)).

COX-2 is a rate-limiting enzyme in converting arachidonic acid to prostaglandins. The increasing introduction of prostaglandins in the body can lead to the inflammatory response. We found from Figure 5(b) that D-gal-treatment caused an increase in COX-2 expression in mouse brain ($P < .001$ versus control). When D-gal-treated mice were fed with PSPC, COX-2 level dramatically declined ($P < .001$ versus D-gal model), while PSPC treatment alone did not affect the basal COX-2 expression.

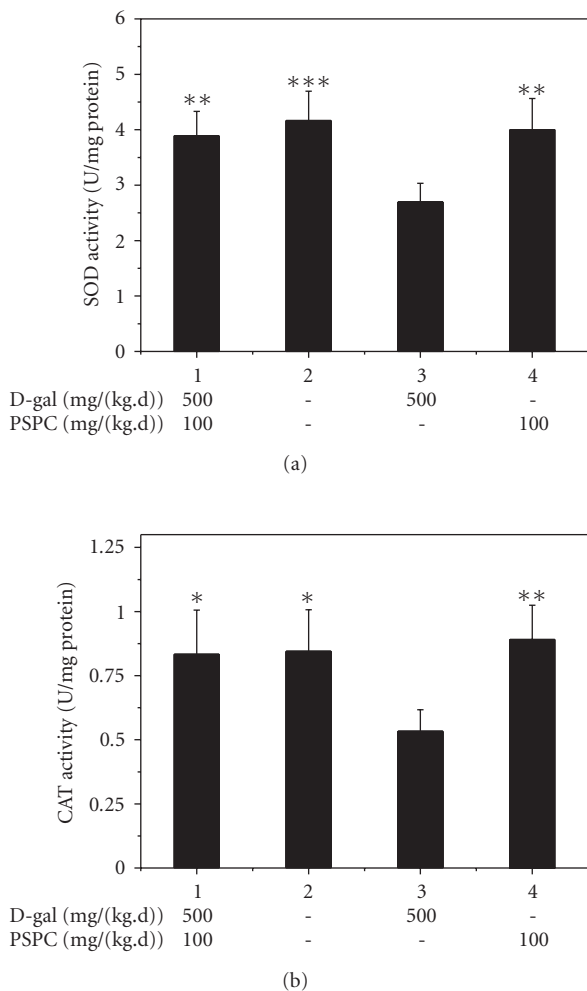COX-2 promoter contains the binding site for transcription factor of NF-κB [27]. NF-κB translocation means its

FIGURE 3: Pharmacological column for the effect of PSPC on the activity of Cu/Zn-SOD (a) and CAT (b). Each value is the means $\pm$ SEM ($n = 5$). $*P < .05$, $** P < .001$, $*** P < .001$ versus D-gal model.
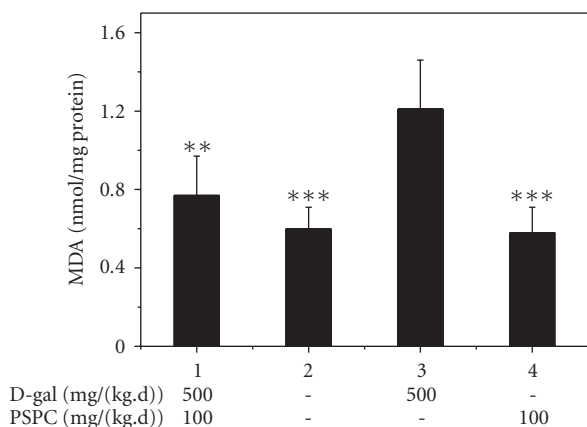

FIGURE 4: Pharmacological column for effect of PSPC on MDA content. Each value is the means $\pm$ SEM ($n = 5$). $*P < .05$, $** P < .01$, $*** P < .001$ versus D-gal model.

activation from the cytosol to the nucleus. At the same time we investigated NF-κB level in the nucleus. The results showed that PSPC markedly attenuated the activity of NF-κB induced by D-gal (D-gal/PSPC versus D-gal model, $P < .01$, Figure 5(c)). However, there is no statistical variation between PSPC group and control group.

3.5. Effect of PSPC on the Expression of GFAP Protein. GFAP was an intermediate filament that mediates the extension of astrocyte processes, and it has been considered as a reliable marker to document age-related increase in brain of rodents and humans subjects [28–31]. We measured the GFAP expression in mouse brain. As shown in Figure 6, there was a sharp contrast to control group in D-gal-treated group, where twofold amount of GFAP was detected ($P < .01$). Compared with control group, there was no significant variation in D-gal/PSPC group and PSPC group. Thus, the supplement of PSPC appears to attenuate the production of GFAP protein induced by D-gal.

4. Discussion

Aging represents a fundamental biological phenomenon; it can be considered as an inevitable progressive process related to the accumulation of oxidative damage. During normal aging, the brain undergoes morphologic and functional changes resulting in the observed behavioral retrogression such as declines in motor and cognitive performance. These declines are augmented by neurodegenerative diseases including amyotrophic lateral sclerosis (ALS), Alzheimer's disease (AD), and Parkinson's disease (PD) [32]. Currently it has been an encouraging challenge for genetic biology to discover an effective therapy for aging. Therapies in aging may be aimed at reversing or decelerating age-related modifications once they have occurred at various points in the life span [33]. D-gal is a reducing sugar and can be metabolized at normal concentration. However, at high levels, it can be converted into aldose and hydroperoxide under the catalysis of galactose oxidase, resulting in the generation of a superoxide anion and oxygen-derived free radicals [10]. D-gal also reacted readily with the free amines of amino acids in proteins and peptides in vivo to form advanced glycation endproducts (AGEs) [10]. Evidence showed that AGEs could remarkably cause the accumulation of ROS and also trigger neuronal inflammatory through activation of NF-κB pathway, which induced the behavioral impairment of learning and memory and decreased antioxidant enzyme activities in the brain. By this token, ROS, AGEs, and inflammatory response induced by D-gal contributed to the neurotoxic effect [10, 15, 20, 34]. These changes are considerably similar to the normal aging process. So the mouse treated with D-gal has been served as the aging model and has been utilized to investigate the mechanism related to the aging. Flavonoids are unique phytochemicals and have powerful antioxidants. They appear potential in antiaging and anti-inflammation properties [35, 36]. Purple sweet potato color is one of the major flavonoids and has displayed some physiological effects and food exploitation. In this study,
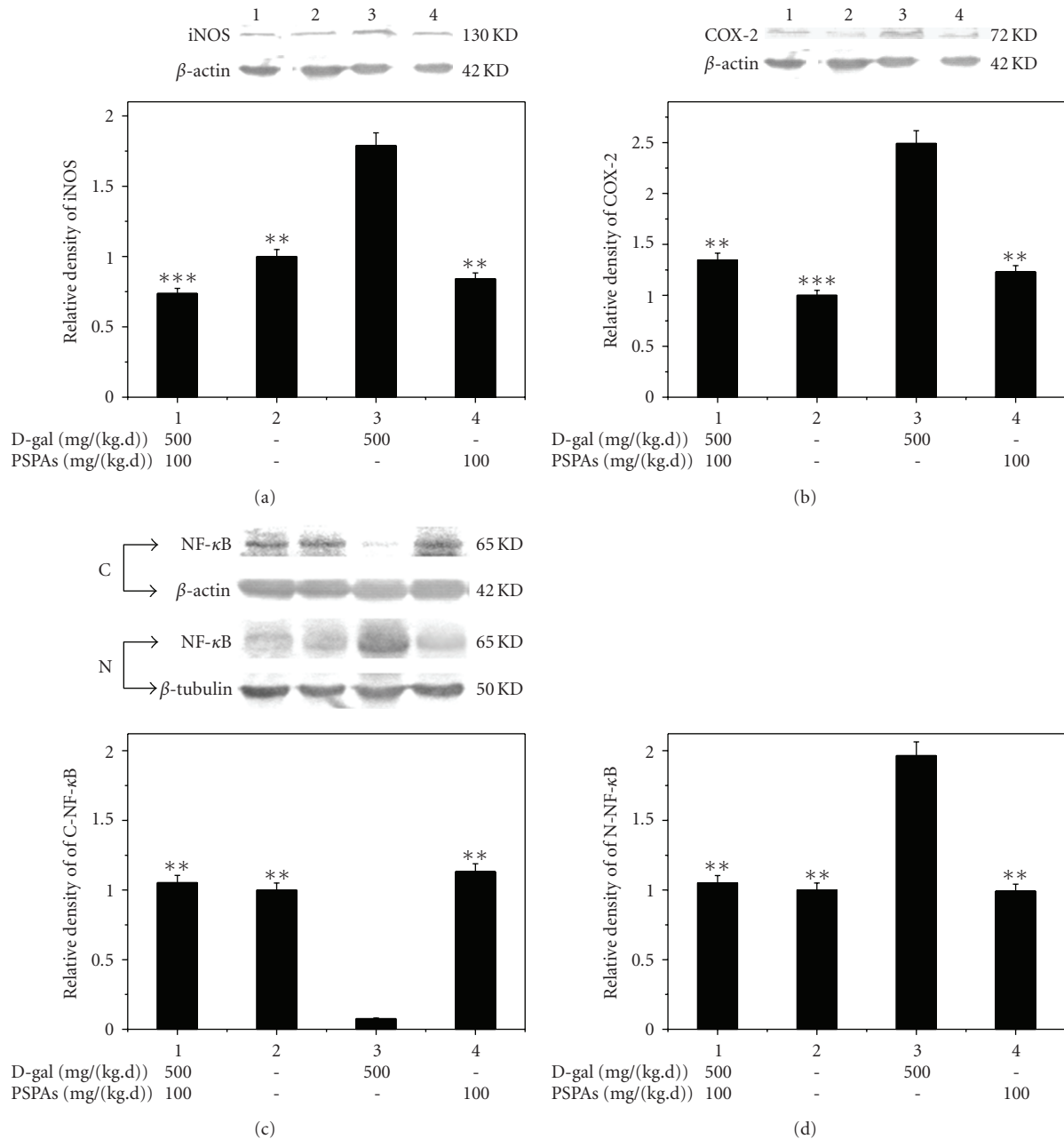
(a)

(b)

(c)

(d)

FIGURE 5: Western blot analysis of the expression level of representative protein in mouse brain ($n = 3$). (a) iNOS, (b) COX-2, (c) NF-κB: cytoplasmic (C) proteins were isolated and used for western blot analysis, (d) NF-κB: Nuclear (N) proteins were isolated and used for western blot analysis. Data are averages from three independent experiments. Each value is the means ± SEM $*P < .05, **P < .01, ***P < .001$ versus D-gal model.

PSPC is mostly composed of cyanidin acyl glucosides and peonidin acyl glucosides (peonidin 3-O-(6-O-(E)-caffeoyl-2-O-β-D-glucopyranosyl-β-D-glucopyranoside)-5-O-β-D-glucoside, peonidin 3-O-(2-O-(6-O-(E)-caffeoyl-β-D-glucopyranosyl)-6-O-(E)-caffeoyl-β-D-glucopyranoside)-5-O-β-D-glucopyranoside, peonidin 3-O-(2-O-(6-O-(E)-feruloyl-β-D-glucopyranosyl)-6-O-(E)-caffeoyl-β-D-glucopyranoside)-5-O-β-Dglucopyranoside, cyanidin 3-O-(6-O-p-coumaroyl)-β-D-glucopyranoside) by HPLC analysis [37].

Evidence showed that cyanidin acyl glucosides and peonidin acyl glucosides had higher antioxidant and anti-inflammatory capacity [38]. The acylated cyanidin and peonidin as the major components of PSPC played an important role in pharmacological effect of PSPC. Our results also demonstrated that the PSPC is an effective antioxidant and anti-inflammatory agent. It could alleviate or reverse the D-gal-caused neurotoxicity by regulating the ROS levels in mouse brain.
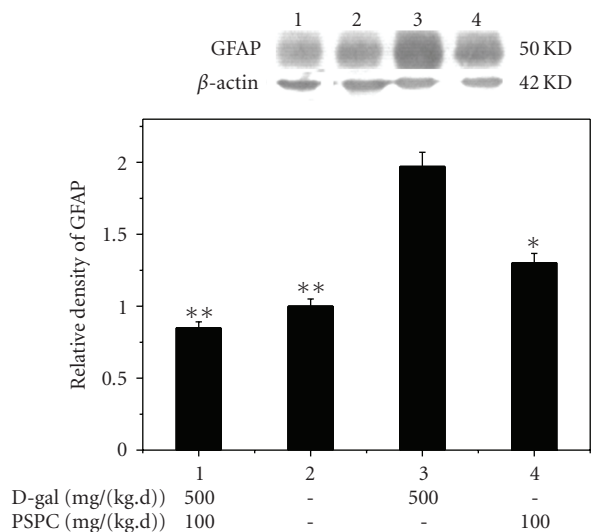
FIGURE 6: Western blot analysis of the expression level of GFAP protein in mouse brain (n = 3). Data are averages from three independent experiments. Each value is the means ± SEM $^*P <$ .05, $^{**}P <$ .01, $^{***}P <$ .001 versus D-gal model.

In our behavioral tests, we found that D-gal could remarkably lead to the impairment of learning and memory in mice. However, both open-field and passive avoidance task suggested that there appeared a novel effect of PSPC on the improvement of brain functions during aging. On one hand, PSPC significantly inhibited the behavioral retrogression, such as rearing/leaning, grooming, and crossing, induced by D-gal. PSPC could also reverse the impairment of learning and memory in D-gal-treated mice.

To further identify the molecular mechanism associated with the recovery capability of the aging brain by PSPC treatment following D-gal-induced injury, we utilized the western blot analysis and biochemistry evaluation to study the relative expression of representative protein or enzyme or metabolic product-associated aging in mouse brain. Brain aging is accompanied by behavioral deficits, including cognitive and motor performances. These deficits are probably induced by oxidative damage and inflammation [32, 39]. The excessive ROS produced would speed up the breakdown of biomacromolecules. thus the body could be subjected to the oxidative damage. However antioxidant enzymes can greatly attenuate oxidative damage, providing support for the free radical theory of aging [40]. D-gal could remarkably cause the decrease of antioxidant enzyme activity such as Cu/Zn-SOD and CAT (Figure 3) and the accumulation of ROS, whereas PSPC could restore the activity of Cu/Zn-SOD and CAT and significantly enhanced the antioxidant capacity of the body. The increased MDA content is an important indicator of oxidative damage in brain [40]. We also found that PSPC decreased the generation of MDA induced by D-gal and reduced the production of the free radical. Therefore, PSPC retarded the aging rate induced by D-gal through the alleviation of oxidative damage.

Oxidative damage is one of the main factors of brain aging. The studies also suggest that brain aging is associated with the neuroinflammation. Evidence indicated that oxidative stress and AGEs induced by D-gal both caused neuroinflammation [41, 42]. Neuroinflammation had been connected to the activation of inflammatory factors iNOS and COX-2, the upregulation of GFAP expression, and the promotion of nuclear translocation of NF-κB [43–47]. Figures 5(a)–5(c) and Figure 6 indicated that D-gal-treatment caused inflammatory response, in which iNOS, COX-2, and GFAP levels were increased, and NF-κB activity was also upregulated. It was remarkable that PSPC could reverse the injurious effect of D-gal. This kind of inhibitory function from PSPC is likely to be due to the impactful effect of the anthocyanins on the inflammation. Pergola et al. found that cyanidin from the blackberry extract effectively brought down the expression of iNOS protein induced by proinflammatory stimuli bacterial lipopolysaccharide (LPS) [48]. The other report revealed that cyanidin inhibited the expression of COX-2 and NF-κB induced by LPS as well [6]. Our results indicated that PSPC not only downregulated the expression of COX-2 and iNOS protein but also inhibited nuclear translocation of NF-κB which was associated with the decreased AGEs levels (data not shown). So PSPC significantly attenuated the progressive inflammation induced by D-gal in mouse brain. Actually, NF-κB was a transcriptional regulator that could control the expression of iNOS gene and COX-2 gene in modulating inflammation and oxidative stress associated with brain aging [49, 50]. NF-κB signaling system is one of the main stress signaling pathways and/or central mediator in response to oxidant damage [47]. The results suggested that PSPC showed anti-inflammatory effect in the neurotoxin pathway due to its strongly modulating the COX-2 and iNOS expression by reducing NF-κB activation. And PSPC ameliorated D-gal-induced brain impairment partly by inhibiting NF-κB signaling pathway.

Our group previously found that the impairment of memory and the loss of synaptic protein associated with memory formation in D-gal-treated mice may be improved by treatment with PSPC. Similarly the present study further demonstrated that PSPC could attenuate the impairment of memory and the changes of inflammation and aging factors in D-gal-treated mice. The results suggested that PSPC might improve the behavior performance of the mice not only by increasing the level of the synaptic proteins but also by enhancing the antioxidant ability which could resume activity of antioxidant enzymes, attenuate lipid peroxidation and result in inhibiting inflammatory response in mouse brain.

In conclusion, the present study demonstrated that PSPC administration attenuated D-gal-induced aging-related changes in mouse brain. PSPC increased the activity of Cu/Zn-SOD and CAT, decreased the expression of iNOS and COX-2, downregulated the expression of GFAP and MDA content, and inhibited nuclear translocation of NF-κB. PSPC consequently improved the spontaneous behavior and cognitive performance and enhanced the capacity of the brain inherent antioxidant or anti-inflammation. These findings about the pharmacological efficacy of PSPC would contribute to brain aging research or aging-related diseases research.

Abbreviations

PSPC: Purple sweet potato color
D-gal: D-galactose
ROS: Reactive oxygen species
GFAP: Glial fibrillary acidic protein
iNOS: inducible nitric oxide synthase
COX-2: Cyclooxygenase-2
NF-κB: Nuclear factor-kappaB
Cu/Zn-SOD: Copper/Zinc superoxide dismutase
CAT: Catalase
MDA: Malondialdehyde
PBS: Phosphate buffer saline solution
TBS: Tris-buffered saline
CNS: Central nervous system
NO: Nitric oxide
ALS: Amyotrophic lateral sclerosis
AD: Alzheimer's disease
PD: Parkinson's disease
LPS: Lipopolysaccharide
AGEs: Advanced glycation endproducts.

Acknowledgments

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