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# Dandelion (*Taraxacum mongolicum* Hand.-Mazz.) suppresses the decrease in physical strength and exercise capacity caused by insufficient liver function

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## Abstract

In this study, D-galactose was used to establish a model of liver dysfunction caused by oxidative stress in mice, and the effect of dandelion on improving the exercise capacity of mice with liver dysfunction was observed and its mechanism was expounded. This study examined the role and mechanism of dandelion in improving running ability, swimming endurance, blood biochemical indices, histopathological changes, and tissue mRNA expression changes. The animal results showed that dandelion extended the running and swimming time to exhaustion in liver dysfunctional mice, reduced the serum levels of blood urea nitrogen (BUN), blood lactic acid (BLA) and malondialdehyde (MDA) in the liver, and increased hepatic glycogen (HG) and muscle glycogen (MG) levels as well as superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px) activities. Histopathological observations suggested that dandelion alleviated lesions in the liver. The quantitative polymerase chain reaction (qPCR) analysis results showed that dandelion downregulated inducible nitric oxide synthase (iNOS) and tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ) mRNA expression and neuronal nitric oxide synthase (nNOS), copper/zinc-superoxide dismutase (Cu/Zn-SOD), manganese-superoxide dismutase (Mn-SOD), and catalase (CAT) expression in the liver and skeletal muscle of the liver-dysfunctional mice. In contrast, dandelion downregulated syncytin-1 mRNA expression in skeletal muscle of mice with a dysregulated liver. The positional analysis showed that the main components of dandelion were gallic acid, protocatechuic acid, chlorogenic acid, caffeic acid, p-coumaric acid, rutin, myricitrin, isoquercitrin, isochlorogenic acid A, and luteolin.

**Keywords:** Dandelion, Aging, d-galactose, Exercise capacity, Liver function

## Introduction

The functions of organs weaken with age, including muscle activity, due to the decline in organ and muscle function, particularly a decline in liver function. Studies have shown that long-distance runners have higher leukocyte telomerase activity than ordinary people, indicating that exercise may delay aging. Long-distance runners have slower heart rates and lower blood pressure and cholesterol levels, which directly reflect the association between

exercise and aging [1]. Maintaining continuous exercise enhances the function of the cardiovascular system. Exercise promotes metabolism, delays arteriosclerosis, and reduces the frequency of diseases caused by a declining liver function [2]. In addition, the reasons for fatigue caused by aging are more complex, including an imbalance in the absorption and consumption of energy, the accumulation of harmful metabolic substances, and disorder of the internal circulation in the body. The decline in liver function caused by aging, metabolic imbalance, and fatigue decreases exercise capacity, so maintaining exercise improves liver function, relieves fatigue, and maintains body vitality, so aging and exercise mutually

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affect body health. Good exercise capacity promotes continuous exercise; thus, delaying aging [3].

The liver relies on oxidative metabolism to provide energy, so the liver is very vulnerable to oxidative stress, and insufficient antioxidant enzyme synthesis leads to dysfunction in the liver mitochondrial respiratory complex, eventually leading to aging of hepatocytes, increased autophagy, and damage from fibrinogen nitrication [4]. The aging damage of the liver causes the continuous accumulation of free radicals, leading to effects on normal metabolism, enhancing fatigue, and decreasing exercise capacity, resulting in a decline in the quality of life in older subjects. Maintaining the normal operation and health of organs, particularly the health of the liver, enables the body to continuously resist oxidative stress and slow down the aging caused by reactive oxygen species (ROS) [5]. Therefore, liver function, aging, and motor ability are closely linked. Enhancing motor function is an effective way to delay aging by intervening in liver function, thus strengthening motor function.

Dandelion is a perennial herb that has been used as traditional medicine or functional foods in China. The dandelion plant body contains a variety of healthy nutrients used to treat acute mastitis, lymphadenitis, malignant boils, acute conjunctivitis, cold fever, acute tonsillitis, acute bronchitis, gastritis, hepatitis, cholecystitis, and urinary tract infection [6–10]. Functional foods from plants show good liver protection and liver function-promoting effects, among which improving liver function through antioxidants is an important effect of these natural functional foods [11, 12]. This study induced aging in mice liver with D-galactose, which resulted in insufficient liver function. We observed that dandelion improved liver function, which improved exercise capacity. We also analyzed the active ingredients in dandelion to provide a theoretical basis for further research and use of dandelion.

## Materials and methods

### Dandelion extract

The dandelion (*Taraxacum mongolicum* Hand.Mazz.) was produced in Luyi County, Zhoukou City, Henan Province, and was collected in 2021. The dandelion was identified by Professor Yu Qian in Collaborative Innovation Center for Child Nutrition and Health Development, Chongqing University of Education, China. A 200 g freeze-dried dandelion was crushed into a fine powder, 4 L of 70% (v/v) ethanol was added to the powder, and the mixture was extracted at 60 °C for 3 h. The extract was filtered and the solvent was dried in a rotary evaporator to obtain the dandelion extract.

### Animal model

Fifty ICR mice (25 males and 25 females) were acclimated (temperature:  $25 \pm 2$  °C, humidity:  $50 \pm 5\%$ ) to adaptive feeding for 7 d, and were then divided into five groups of ten mice/group (weighing  $25 \pm 2$  g, five females and five males in each group): the normal group, the model group, the vitamin C (Vc) group, the dandelion low dose group (L-dandelion), and the dandelion high dose gastric group (H-dandelion). The experimental cycle was 10 weeks. After the start of the experiment, mice in each group, except the normal group, were injected with a 5% (w/w) D-galactose solution at a daily dose of 100 mg/kg b.w. for 6 weeks [13]. The normal group of mice was injected intraperitoneally with normal saline (0.01 mL/kg b.w.). Mice in the normal and model groups were gavaged with distilled water (0.01 mL/kg b.w.) beginning on week 7. Mice in the Vc group were infused with Vc at a daily dose of 100 mg/kg b.w. Mice in the L-dandelion and H-dandelion groups were infused daily with dandelion at doses of 50 and 100 mg/kg b.w., respectively, which was continued for 4 weeks. After 10 weeks, the mice underwent running and swimming to exhaustion experiments, and all mice were killed by extracting capillary orbital blood and cutting the neck. The liver and skeletal muscle were dissected for future use. This study was implemented according to the Declaration of Helsinki, and the laboratory animals purchased from Shaanxi Experimental Animal Center the protocol was approved (SCXK (Shaanxi) 2020–001).

### Running experiment

After the end of the gavage experiment, the running rollers were set to 20 r/min, and the mice were forced to run, with five consecutive shocks until exhaustion (YH-CS, Wuhan Yihong Technology Co., Ltd, Wuhan, Hubei, China). The running time was recorded.

### Exhaustive swimming experiment

After the gastric sample experiment, the mice were placed in a homemade thermostatic water tank with a water temperature of  $28 \pm 2$  °C and a water depth of 20 cm and were forced to swim until they could not float to the surface for more than 10 s, which was determined to be exhaustion, and swimming time was recorded.

### Determination of the mice serum indices

Orbital blood was harvested, centrifuged (4 °C, 1500 rpm for 10 min), the serum layer was isolated, and blood urea nitrogen (BUN), blood lactic acid (BLA), malondialdehyde (MDA), hepatic glycogen (HG) and muscle glycogen (MG) levels, as well as superoxide dismutase (SOD) and

glutathione peroxidase (GSH-Px) activities, were determined with assay kits (Nanjing Jiancheng Bioengineering Institute, Nanjing, Jiangsu, China) [14].

### Preparation of the mouse tissue sections

The liver and skeletal muscle tissues were harvested and fixed in 10% (v/v) formalin by volume fraction immediately after three washes in saline. The fixed tissue was dehydrated at 4 °C for 48 h, embedded in paraffin, cut into 5–10 µm slices, and stained with hematoxylin and eosin. The tissue pathology was observed under a light microscope (BX53, Olympus, Tokyo, Japan) [15].

### Detection of mRNA expression in mouse tissues

A 0.2 g portion of mouse liver or skeletal muscle tissue was weighed, washed with saline, 1.8 mL of saline was added, and the tissue was homogenized with 1.0 mL RNazol (Beijing Solarbio Science & Technology Co., Ltd., Beijing, China) to extract the RNA. The 260 and 280 nm absorbance values of the RNA extract were determined, and the concentration of the RNA was adjusted after the OD260/OD280 was calculated at 1 µg/µL. The cDNA reaction system was prepared after reverse transcription, and the system solution (Thermo Fisher Scientific, Waltham, MA, USA) included cDNA (1 µL), SYBR Green PCR Master Mix (10 µL), upstream primers (1 µL, Table 1), downstream primers (1 µL), and sterile distilled water (7 µL). After the reaction solution was prepared, it was placed in the real-time fluorescent quantitative polymerase chain reaction instrument and the mRNA was amplified under the set conditions (60 s at 95 °C and 15 s at 95 °C for 40 cycles, then 30 s at 55 °C, 35 s at 72 °C, 30 s at 95 °C and 35 s at 55 °C, StepOnePlus, Thermo Fisher Scientific), with GAPDH as the internal standard. The relative expression intensity of each gene was calculated using the  $2^{-\Delta\Delta C_t}$  method [16].

### High performance liquid chromatography

Standards and the dandelion sample extracts were dissolved in methanol and tested. The composition of the compounds in the dandelion samples was measured using the following chromatographic conditions: AcclaimTM120 C18 column (4.6 mm × 150 mm, 5 µm); mobile phase: methanol and 0.5% glacial acetic acid; detection wavelength: 328 nm; column temperature: 35 °C; flow rate: 0.6 mL/min; sample volume: 20 µL (Ulti-Mate 3000, Thermo Fisher Scientific).

### Statistical analysis

Data were calculated for all mice, and the final experimental results are expressed as mean ± standard deviation. One-way analysis of variance and SPSS software (SPSS Inc., Chicago, IL, USA) were used to detect differences. A *P*-value < 0.05 was considered significant.

## Results

### Effect of dandelion on motor capacity of mice

According to Table 2, the longest running and swimming times to exhaustion were observed in the normal group, while the model group had the shortest times. Dandelion and Vc significantly (*P* < 0.05) prolonged the running and swimming time to exhaustion of the mice compared with the model group. H-dandelion had the best effect and

**Table 2** The running and exhaustive swimming times in mice

Group	Running time (s)	Exhaustive swimming time (s)
Normal	54.82 ± 2.15 <sup>a</sup>	311.83 ± 21.55 <sup>a</sup>
Model	33.20 ± 2.31 <sup>e</sup>	36.61 ± 20.68 <sup>e</sup>
Vc	43.09 ± 2.03 <sup>c</sup>	241.08 ± 19.88 <sup>c</sup>
L-dandelion	37.90 ± 1.92 <sup>d</sup>	186.06 ± 23.46 <sup>d</sup>
H-dandelion	48.72 ± 2.21 <sup>b</sup>	282.43 ± 16.77 <sup>b</sup>

<sup>a–e</sup>The same lowercase letters indicate no significant difference between the two groups, and different lowercase letters indicate a significant difference between the two groups, *P* < 0.05

**Table 1** The qPCR primer sequences used in this study

Gene	Upstream primer sequence	Downstream primer sequence
Cu/Zn-SOD	5'-AACCAGTTGTGTTGAGGAC-3'	5'-CCACCATGTTTCTTAGAGTGAGG-3'
Mn-SOD	5'-CAGACCTGCCTTACGACTATGG-3'	5'-CTCGGTGGCGTTGAGATTGTT-3'
CAT	5'-GGAGGCGGGAACCAATAG-3'	5'-GTGTGCCATCTCGTCAGTGAA-3'
nNOS	5'-TCGTCCAACCTCTGGGCTCTT-3'	5'-CCTTCTCTCTCTCCCTCTCTTC-3'
iNOS	5'-CAAAGGCTGTGAGTCCTGCAC-3'	5'-ACTTTGATCAGAAGCTGTCCC-3'
TNF-α	5'-ATGGGGGGCTTCCAGAA-3	5'-CCTTTGGGGACCGATCA-3'
Syncytin-1	5'-GTTAACCTTGTCTCTCCAGAATCGA-3'	5'-CATCAGTACGTGGGCTAGCA-3'
GAPDH	5'-TGACCTCAACTACATGGTCTACA-3'	5'-CTTCCCATCTCGGCCTTG-3'

was significantly ( $P<0.05$ ) better than the L-dandelion or Vc treatments.

**Mouse serum levels of BUN, BLA, MDA, HG, MG, SOD, and GSH-Px**

As shown in Table 3, the serum levels of BUN, BLA, and MDA were significantly lower ( $P<0.05$ ) in the normal group than those in the other groups, while the HG and MG levels, as well as SOD and GSH-Px activities, were significantly higher ( $P<0.05$ ) than those in the other groups. However, these serum indicators showed the opposite trend in the model group to those in the normal group. In the relative model group, dandelion and Vc reduced BUN, BLA, and MDA levels in the serum of mice with decreased liver function, while increasing the

HG and MG levels as well as the SOD and GSH-Px activities. The values for the H-dandelion group were close to those of the normal group.

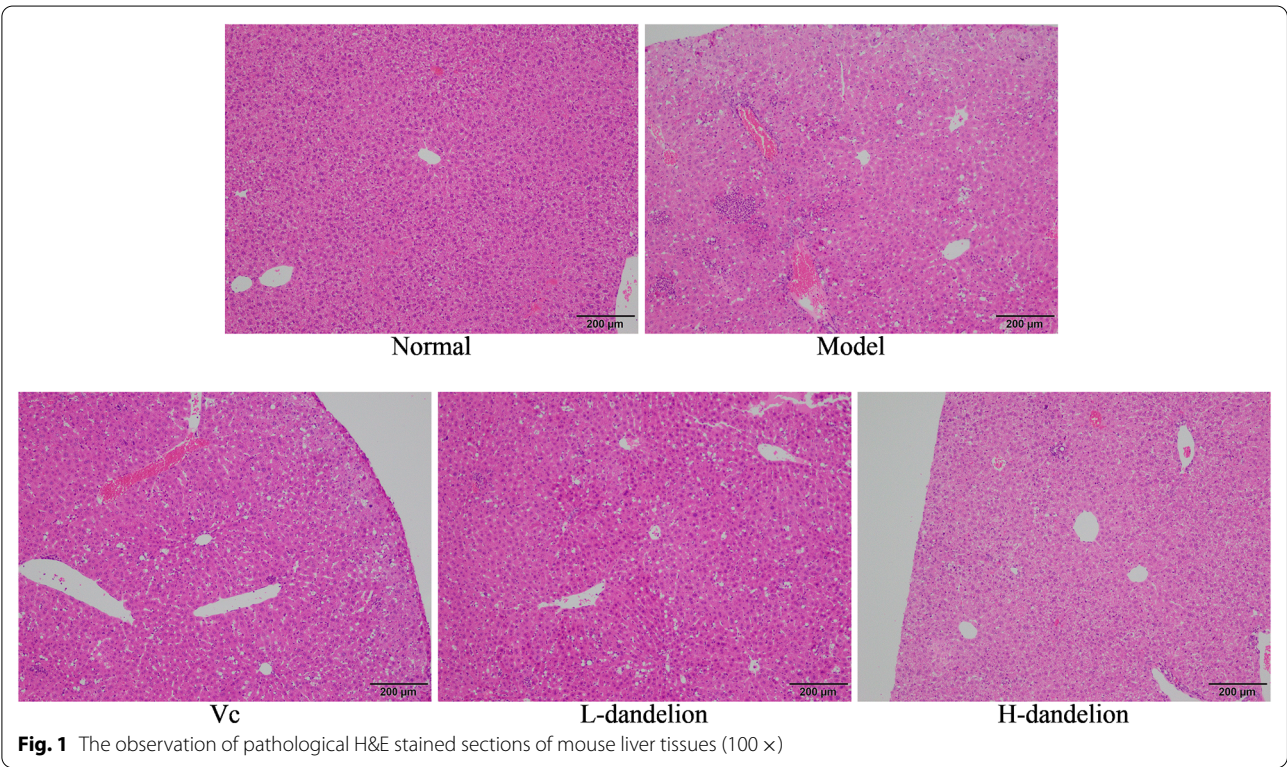
**Pathological changes in the mouse liver**

The microscopic liver tissue morphology of the mice is shown in Fig. 1. The normal group had clear and intact liver lobules and the hepatocytes were arranged radially centered around the central vein. The hepatic leaflet structure was disrupted in the model group, the arrangement of hepatocytes was chaotic, some cell and nuclear membranes were ruptured, and apoptotic bodies appeared in the microscopic view. Dandelion and Vc reduced the damage to hepatocytes. The liver lobular

**Table 3** The BUN, BLA, MDA, HG, MG, SOD, and GSH-Px levels in mouse serum

Group	BUN (mmol/L)	BLA (mmol/L)	MDA (mmol/mL)	HG(mg/mL)	MG (mg/mL)	SOD (U/mL)	GSH-Px (μmol/mL)
Normal	6.04 ± 0.36 <sup>e</sup>	15.98 ± 0.87 <sup>e</sup>	6.11 ± 0.49 <sup>e</sup>	12.06 ± 0.56 <sup>a</sup>	1.86 ± 0.31 <sup>a</sup>	332.52 ± 28.79 <sup>a</sup>	232.52 ± 18.63 <sup>a</sup>
Model	18.37 ± 1.52 <sup>a</sup>	34.88 ± 1.05 <sup>a</sup>	14.83 ± 0.72 <sup>a</sup>	4.93 ± 0.39 <sup>e</sup>	0.53 ± 0.17 <sup>e</sup>	94.65 ± 8.36 <sup>e</sup>	82.91 ± 6.89 <sup>e</sup>
Vc	10.99 ± 1.01 <sup>c</sup>	23.05 ± 0.81 <sup>c</sup>	10.86 ± 0.62 <sup>c</sup>	8.61 ± 0.48 <sup>c</sup>	1.15 ± 0.18 <sup>c</sup>	189.99 ± 13.06 <sup>c</sup>	136.52 ± 9.92 <sup>c</sup>
L-dandelion	14.30 ± 1.26 <sup>b</sup>	29.84 ± 0.74 <sup>b</sup>	12.35 ± 0.51 <sup>b</sup>	6.83 ± 0.45 <sup>d</sup>	0.80 ± 0.12 <sup>d</sup>	148.63 ± 11.22 <sup>d</sup>	106.82 ± 7.31 <sup>d</sup>
H-dandelion	8.35 ± 0.62 <sup>d</sup>	19.22 ± 0.69 <sup>d</sup>	8.16 ± 0.41 <sup>d</sup>	10.70 ± 0.61 <sup>b</sup>	1.18 ± 0.16 <sup>b</sup>	261.08 ± 20.63 <sup>b</sup>	185.65 ± 10.50 <sup>b</sup>

<sup>a–e</sup>The same lowercase letters indicate no significant difference between the two groups, and different lowercase letters indicate a significant difference between the two groups,  $P<0.05$



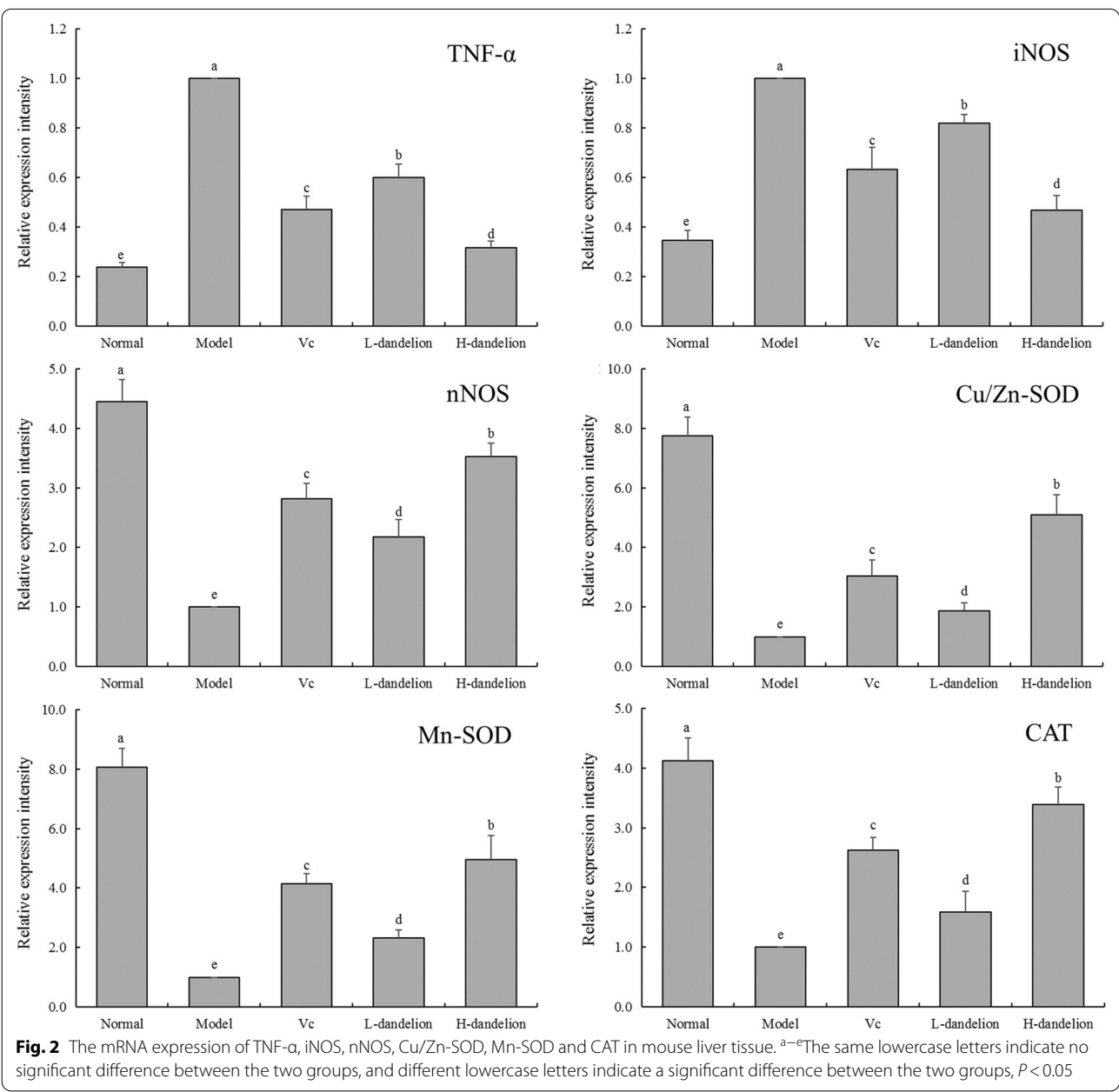


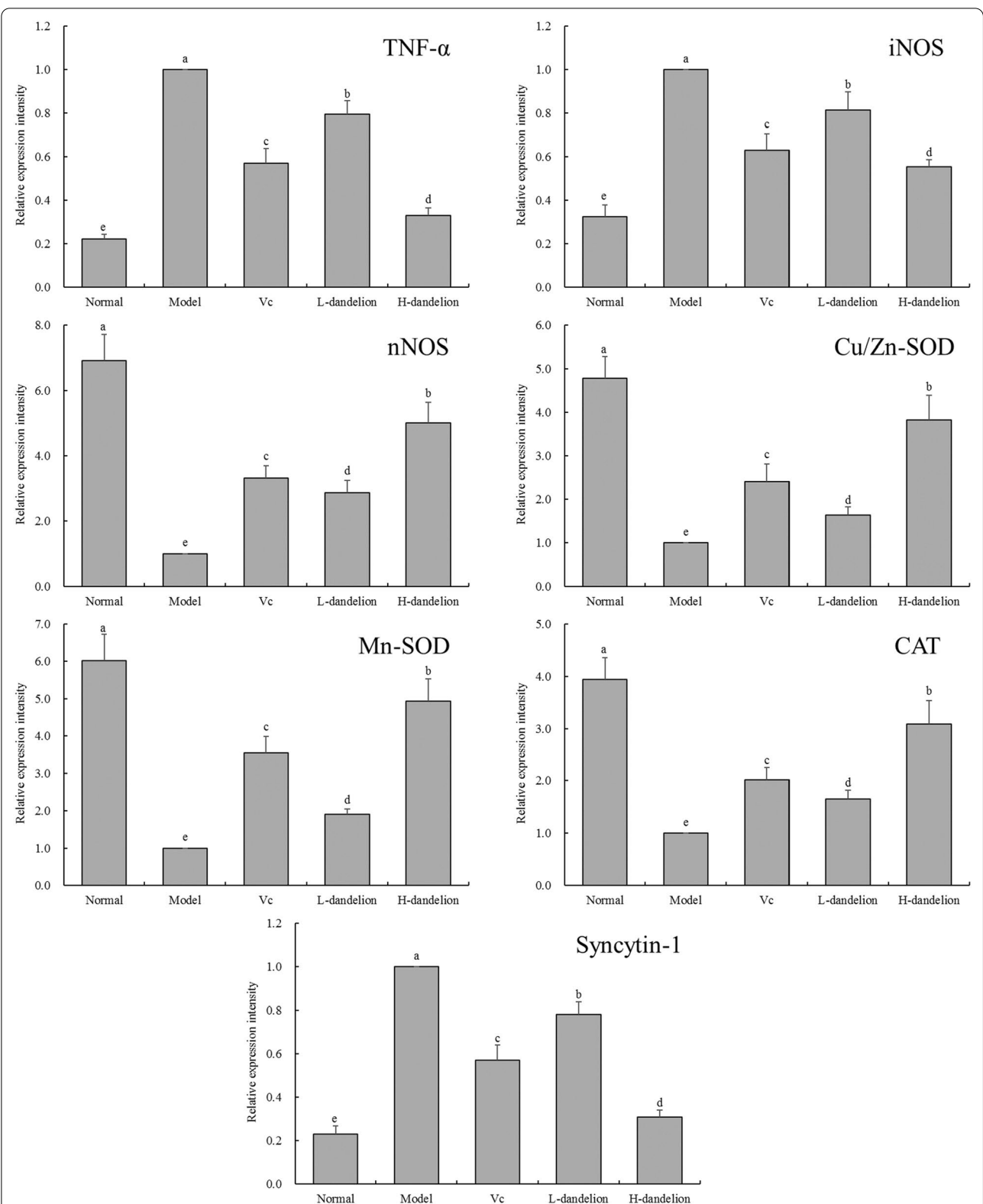
structure was intact after adding H-dandelion, but some cells were damaged in the L-dandelion and Vc groups.

**mRNA Expression in mouse liver and skeletal muscle**

As shown in Fig. 2, in addition to the normal group, the other four groups of mice experienced oxidative damage and aging due to the injection of D-galactose, therefore, the intensity of inducible nitric oxide synthase (iNOS) and tumor necrosis factor (TNF)- $\alpha$  mRNA expression was the weakest in the liver of the normal group of mice, while neuronal nitric oxide synthase (nNOS), Cu/Zn-SOD, Mn-SOD, and catalase (CAT)

expression were the strongest; however, the model group of mice had the strongest iNOS and TNF- $\alpha$  expression levels, but the weakest nNOS, Cu/Zn-SOD, Mn-SOD, and CAT expression levels. Dandelion down-regulated iNOS and TNF- $\alpha$  expression and upregulated nNOS, Cu/Zn-SOD, Mn-SOD, and CAT expression in the liver tissues of mice with decreased liver function. H-dandelion was the best regulator of these indices, as the expression levels were close to the normal group of mice. The results of the mRNA expression analysis in skeletal muscle were the same as those of TNF- $\alpha$ , iNOS, nNOS, Cu/Zn-SOD, Mn-SOD, and CAT in liver tissue



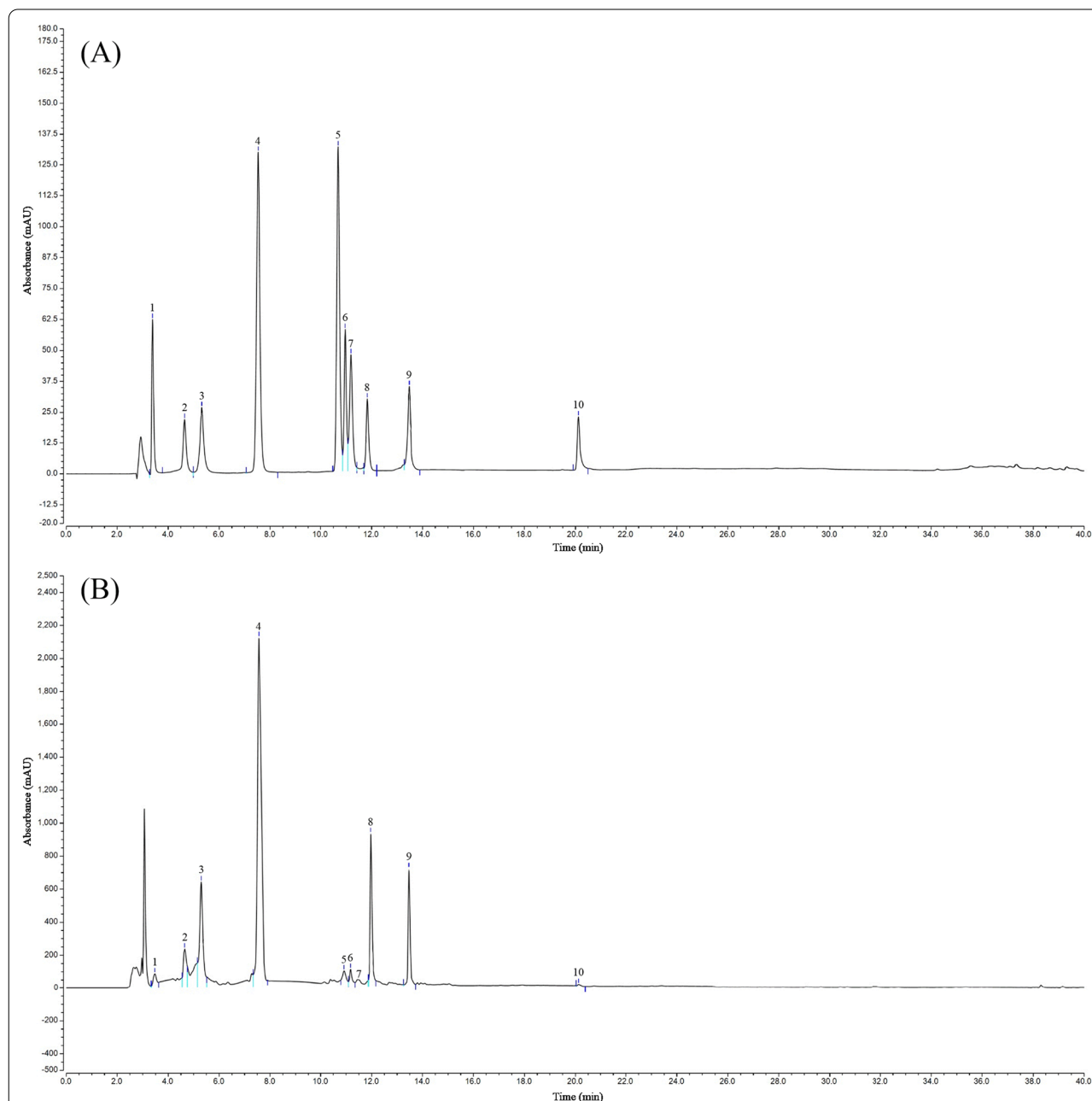


**Fig. 3** The mRNA expression of TNF- $\alpha$ , iNOS, nNOS, Cu/Zn-SOD, Mn-SOD, CAT and syncytin-1 in mouse skeletal muscle tissue. <sup>a-e</sup> The same lowercase letters indicate no significant difference between the two groups, and different lowercase letters indicate a significant difference between the two groups,  $P < 0.05$

(Fig. 3). In addition, the skeletal muscle of mice in the model group had the strongest syncytin-1 expression, and H-dandelion, L-dandelion, and Vc upregulated syncytin-1 expression in the skeletal muscle of mice with decreased liver function. H-dandelion had the strongest upregulating effect, making syncytin-1 expression most similar to the skeletal muscle in the normal group of mice.

#### Dandelion compound composition

Figure 4A is the chromatographic diagram of the standard, after comparing with the chromatogram of the standard product, it was found that the dandelion mainly contained 10 compounds, including gallic acid, protocatechuic acid, chlorogenic acid, caffeic acid, p-coumaric acid, rutin, myricitrin, isoquercitrin, isochlorogenic acid A, and luteolin (Fig. 4B). The chlorogenic acid and caffeic



**Fig. 4** Compound composition of dandelion analyzed by HPLC, **A** chromatographic diagram of the standard, **B** chromatographic diagram of the dandelion extract, 1: gallic acid, 2: protocatechuic acid, 3: chlorogenic acid, 4: caffeic acid, 5: p-coumaric acid, 6: rutin, 7: myricitrin, 8: isoquercitrin, 9: isochlorogenic acid A, 10: luteolin

acid contents were relatively high and could be the most critical active ingredients.

## Discussion

Aging of the body causes liver dysfunction, liver metabolic disorder, and further increases in free radicals. Excessive accumulation of free radicals causes organ damage and functional decline in movement-related organs, eventually decreasing movement ability and increasing fatigue [17]. The liver also plays the role of metabolic organ in the human body. Substances such as carbohydrates and proteins need to be metabolized through the liver. If the liver function is not good, it will not be able to provide enough energy for the body, and the physical strength will be significantly decreased. Therefore, the liver function level decreases after liver problems, and the fatigue performance of physical strength decreases. Appropriate exercise reduces free radical damage to cell membranes, enables normal cells to maintain normal oxidative respiratory chain activity and mitochondrial structure and function, and promotes the health of the liver [18]. Endurance running and swimming exhaustion experiments are commonly used to verify exercise capacity, as these are the most direct manifestation of fatigue resistance and a key manifestation of body resistance to metabolic imbalance [19]. In this study, D-galactose caused oxidative damage to the body, and promoted mouse aging and liver function decline, the experimental results confirmed that dandelion prolonged endurance running and swimming to exhaustion by the mice, suggesting that dandelion enhanced exercise capacity and restored liver function.

Sugar and lipid metabolism is abnormal after functional damage to the liver, so proteins and amino acids are metabolized, which increases BUN excessively. The body is temporarily in a state of hypoxia during strenuous exercise, so that normal energy metabolism changes, and a large amount of lactic acid accumulates in the muscle in a short time, leading to fatigue. The lactic acid that accumulates in muscle gradually enters the blood circulation to form BLA, which is an important indicator of fatigue [20]. In addition, the magnesium content in the body is directly related to exercise endurance and fatigue resistance. When HG and MG contents decrease, the energy supply to the muscle decreases, forcing MG to provide the necessary energy through glycolysis. A large amount of lactic acid accumulates during this process and is released into the blood, which weakens the ability of the muscle to contract and affects the ability to exercise [21]. Excessive exercise causes a large increase in free radicals in the body, which is particularly evident in the state of liver dysfunction. CAT, GSH-Px, and SOD enzymes maintain normal oxidative stress levels in the body and

remove excess free radicals. GSH-Px plays an important role in catalyzing hydrogen peroxide, protecting the structure and functional integrity of cell membranes by removing hydrogen peroxide and lipid hydrogen peroxide [22]. SOD is an important enzyme regulating the balance between oxidation and antioxidation. SOD decomposes oxygen radicals into oxygen and hydrogen peroxide, effectively removing superoxide anion radicals, thus reducing the production of toxic hydroxyl radicals, and protecting cells. If the accumulated free base far exceeds the antioxidant enzyme defense ability, unsaturated lipids in cell membranes will be destroyed by free radicals, and membrane fluidity and cell function will decrease, leading to lipid peroxidation and the production of MDA, which further destroys the membranes, leading to cell swelling and necrosis. Thus, the level of MDA also indirectly reflects the free radical metabolic changes and the degree of tissue peroxidation damage [23]. The level of lipid peroxides increases as liver dysfunction worsens, and the strength of the antioxidant enzyme activities is a decisive factor in the degree of free radical damage to cells. Detecting the activities of the antioxidant enzymes reflects the state of resistance to oxidative damage and reflects the degree of fatigue and exercise capacity [24]. The experimental results of this study showed that dandelion significantly changed the serum levels of BUN, BLA, HG, MG, MDA, SOD, and GSH-Px in the liver-dysfunctional mice; thus, inhibiting liver dysfunction and enhancing exercise endurance and capacity.

The liver is the central organ of the oxidative stress response, which significantly reflects the degree of oxidation in the body, and is an organ where free radicals and lipid peroxides are easily produced. High levels of free radicals can cause lesions in liver tissue. In addition, liver dysfunction leads to a decrease in the frequency of vasoconstriction in the liver and reduces the blood supply to various organs, which directly reduces exercise capacity [25]. In this study, the pathological observations confirmed the lesions in the liver tissues. Dandelion effectively reduced these lesions and inhibited liver dysfunction. One of its important roles may be to indirectly improve motor function.

The main role of CAT is to metabolize hydrogen peroxide; thus, removing the products of peroxidative stress found in erythrocytes, peroxisomes, and mitochondria, which prevent the cell damage caused by ROS [26]. Cu/Zn-SOD and Mn-SOD are two forms of SOD in mammals. Cu/Zn-SOD is a SOD with  $\text{Cu}^{2+}$  and  $\text{Zn}^{2+}$  in the cytoplasm; Mn-SOD is a SOD with  $\text{Mn}^{4+}$  in the mitochondria. Both SODs play an extremely important role in inhibiting oxidative stress in vivo. With liver dysfunction and decreased exercise capacity, the body contains more oxidative stress and produces more free radicals after



exercise. To maintain better exercise capacity and inhibit liver dysfunction, higher CAT, Cu/Zn-SOD, and Mn-SOD activities are needed to inhibit free radicals, reduce fatigue, and improve exercise ability [27]. The results of this study also confirmed that liver and skeletal muscle accumulate large amounts of free radicals due to a series of oxidative stress responses. Dandelion improved the activities of CAT, Cu/Zn-SOD, and Mn-SOD, suggesting that dandelion can inhibit liver dysfunction, protect the body, and improve the exercise ability of mice.

NOS has been divided into nNOS, which is expressed in the normal state, and endothelial nitric oxide synthase and iNOS, which are induced after injury. The generation of NO is closely related to NOS. NO is continuously produced in skeletal muscle. When the body is in a static state, the NO content in skeletal muscle is relatively low, but skeletal muscle contraction during exercise increases the NO level. The nNOS is distributed on the myofiber membrane, and is significantly upregulated after exhaustive exercise, but decreased after recovery from exercise fatigue. iNOS expression is induced by in vivo endotoxins and various cytokines, particularly the inflammatory cytokines, and is mainly distributed in hepatocytes, macrophages, and neutrophils. iNOS is a NOS subtype that generates more NO. NO is involved in liver lesions and is an important mediator of liver lesions [28]. TNF- $\alpha$  is a multifunctional cytokine secreted by monocytes and macrophages that promotes the secretion of ROS, such as  $O_2^-$ ,  $H_2O_2$ , and NO by macrophages; thus, promoting aging [29]. Therefore, the expression levels of iNOS, nNOS, and TNF- $\alpha$  reflect the oxidative stress level. The syncytin-1 gene plays a role in immune regulation, and the high expression of syncytin-1 in skeletal muscle impairs motor neurons. High expression of syncytin-1 in skeletal muscle also leads to the accumulation of oxygen free radicals and mitochondrial damage. Activation of syncytin-1 by oxidative stress causes muscle and nerve damage and affects motor function [30]. As the aging process progresses, muscle mass atrophies, and after age 40, muscle mass is lost at a rate of 8% per decade. After the age of 70, the loss of muscle mass accelerates at a rate of 15 percent per decade. The loss of muscle mass affects the degree of bone protection, reduces the ability to exercise, and leads to a significant decline in physical strength. By ingesting active substances to reduce the degree of muscle aging, avoid amyloidosis and mitochondrial dysfunction in muscle, so as to restore muscle vitality, improve motor function, restore physical strength, and delay aging [31]. In this study, dandelion significantly downregulated the expression of iNOS, nNOS, TNF- $\alpha$ , and syncytin-1, indicating that dandelion relieved exercise fatigue and oxidative stress, improved free radical scavenging activity, and had the ability to restore muscle

vitality and increase muscle mass, thus enhancing the exercise ability of mice.

Gallic acid, protocatechuic acid, chlorogenic acid, caffeic acid, p-coumaric acid, rutin, myricitrin, isoquercitrin, isochlorogenic acid A, and luteolin are antioxidants that inhibit liver lesions caused by oxidative stress and thus inhibit liver dysfunction [32–41]. The combined effect of these active substances inhibited liver dysfunction.

#### Acknowledgements

Not applicable.

#### Author contributions

Investigation, methodology, writing-original draft: YLZ; Formal analysis, methodology, writing-review, and editing: BL; Conceptualization, supervision, writing-review, and editing: GLW. All authors read and approved the final manuscript.

#### Funding

Not applicable.

#### Availability of data and materials

Not applicable.

#### Declarations

#### Ethical approval and consent to participate

This study was approved by the Ethics Committee of Renmin University of China (Beijing, China).

#### Competing interests

All authors declare that they have no competing interests.

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Received: 3 September 2022 Accepted: 19 December 2022

Published online: 31 December 2022

#### References

1. Zhang Y, Oliveira AN, Hood DA (2020) The intersection of exercise and aging on mitochondrial protein quality control. *Exper Gerontol* 131:110824
2. Georgakouli K, Manthou E, Fatouros IG, Deli CK, Spandidos DA, Tsatsakis AM, Kouretas D, Koutedakis Y, Theodorakis Y, Jamurtas AZ (2015) Effects of acute exercise on liver function and blood redox status in heavy drinkers. *Exper Ther Med* 10:2015–2022
3. Kristensen CM, Brandt CT, Ringholm S, Pilegaard H (2017) PGC-1 $\alpha$  in aging and lifelong exercise training-mediated regulation of UPR in mouse liver. *Exper Gerontol* 98:124–133
4. Luo XL, Liu WX, Zhong H, Yan YQ, Feng FQ, Zhao MJ (2021) Synergistic effect of combined oyster peptide and ginseng extracts on anti-exercise-fatigue and promotion of sexual interest activity in male ICR mice. *J Funct Foods* 86:104700
5. Orita I, Morikita I, Watanabe M, Oh Z, Kanai S (2021) Effects of facial isometric exercise on antioxidant capacity. *Health* 13:1171–1180
6. Gargouri M, Ghorbel-Koubaa F, Bonenfant-Magné M, Magné C, Dauvergne X, Ksouri R, Krichen Y, Abdely C, El Feki A (2012) Spirulina or dandelion-enriched diet of mothers alleviates lead-induced damages in brain and cerebellum of newborn rats. *Food Chem Toxicol* 50:2303–2310
7. González-Castejón M, Visioli F, Rodríguez-Casado A (2012) Diverse biological activities of dandelion. *Nutr Rev* 70:534–547

8. Davaatseren M, Hur HJ, Yang HJ, Hwang JT, Park JH, Kim HJ, Kim MJ, Kwon DY, Sung MJ (2013) Taraxacum officinal (dandelion) leaf extract alleviates high-fat diet-induced nonalcoholic fatty liver. *Food Chem Toxicol* 58:30–36
9. Ghaima KK, Hashim NM, Ali SA (2013) Antibacterial and antioxidant activities of ethyl acetate extract of nettle (*Urtica dioica*) and dandelion (*Taraxacum officinale*). *J Appl Pharm Sci* 3:96–99
10. Ovadje P, Hamm C, Pandey S (2012) Efficient induction of extrinsic cell death by dandelion root extract in human chronic myelomonocytic leukemia (CMML) cells. *PLoS ONE* 7:e30604
11. Zhu KX, Nie SP, Tan LH, Li C, Gong DM, Xie MY (2016) A polysaccharide from *Ganoderma atrum* improves liver function in type 2 diabetic rats via antioxidant action and short-chain fatty acids excretion. *J Agri Food Chem* 64:1938–1944
12. Mahesh R, Bhuvana S, Begum VM (2009) Effect of Terminalia chebula aqueous extract on oxidative stress and antioxidant status in the liver and kidney of young and aged rats. *Cell Biochem Funct* 27:358–363
13. Zhang J, Chen L, Zhang L, Chen Q, Tan F, Zhao X (2021) Effect of *Lactobacillus fermentum* HFY03 on the antifatigue and antioxidation ability of running exhausted mice. *Oxid Med Cell Longe* 2021:8013681
14. Li F, Huang H, Wu Y, Lu Z, Zhou X, Tan F, Zhao X (2021) *Lactobacillus fermentum* HFY06 attenuates D-galactose-induced oxidative stress and inflammation in male Kunming mice. *Food Funct* 12:12479–12489
15. Hu TT, Chen R, Qian Y, Ye K, Long XY, Park KY, Zhao X (2022) Antioxidant effect of *Lactobacillus fermentum* HFY02-fermented soy milk on D-galactose-induced aging mouse model. *Food Sci Human Well* 11:1362–1372
16. Long XY, Wu HB, Zhou YJ, Wan YX, Kan XM, Gong JJ, Zhao X (2022) Preventive effect of *Limosilactobacillus fermentum* SCHY34 on lead acetate-induced neurological damage in SD rats. *Front Nutr* 9:852012
17. Carvalho DZ, St Louis EK, Boeve BF, Mielke MM, Przybelski SA, Knopman DS, Machulda MM, Roberts RO, Geda YE, Petersen RC, Jack CR Jr, Vemuri P (2016) Excessive daytime sleepiness and fatigue may indicate accelerated brain aging in cognitively normal late middle-aged and older adults. *Sleep Med* 32:236–243
18. Zhang Y, Sheng L, Liu XW, Wei J, Liu XJ, Zhang NY, Wang ZY (2022) Effects of different exercise on liver lipid accumulation and FGF21 secretion in obese rats. *Chinese J Appl Physiol* 38:47–52
19. Wu JZ, Chen HJ, Wang D, Zhao X (2022) Effect of *Clerodendranthus spicatus* (Thunb.) C. Y. Wu on the exercise ability of D-galactose-induced oxidative aging mice. *Food Sci Technol* 42:e09822
20. Li XL, Xiong W, Xie CC (2006) The effect of L-arginine on the indexes of NO and BUN in serum of rats if increase by degree load motion to fatigue. *J Guangzhou Phys Educ Inst* 26:85–87
21. Jia L, Nie XJ, Fang M, Zhang J (2010) Review about influences of *Sphalerocarpus Gracilis* polysaccharides on serum CK, LDH, muscle glycogen, hepatic glycogen and sport endurance in mice. *J Jilin Inst Phys Educ* 26:4–6
22. Zhang ZG, Wang F (2022) Effects of aerobic exercise combined with D-ribose supplementation on anti-fatigue and anti-oxidation in mice. *Sci Technol Food Ind* 43:368–375
23. Song YH, Sun XW, Meng CH (2015) Effect of hongjintian decoction on serum MDA, SOD, NGF, MBP and BFGF levels of exercise-induced fatigue athletes. *Chinese J Biochem Pharm Sci Technol Food In* 2015:119–121
24. Qiu WM, Zhao ZM, Pan HS, Lai QY, Jing CX, Feng YC (2014) Study of exercise-induced fatigue rat model established by different periodical swimming training based on evaluation of liver function. *J Guangzhou Univ Tradit Chinese Med* 2014:983–987
25. Liu J, Han SK, Ma Y, Chen XH, He YM, Sun XM (2021) Aerobic exercise combined with vitamin D intervention regulates glucose and lipid metabolism and improves liver inflammation and oxidative stress in db/db mice. *Chinese J Sports Med* 40:629–637
26. Hu P, Tirelli N (2012) Scavenging ROS: superoxide dismutase/catalase mimetics by the use of an oxidation-sensitive nanocarrier/enzyme conjugate. *Bioconjugate Chem* 23:438–449
27. Yi RK, Tan F, Zhou XR, Mu JF, Li L, Du XP, Yang ZN, Zhao X (2020) Effects of *Lactobacillus fermentum* CQPC04 on lipid reduction in C57BL/6J mice. *Front Microbiol* 11:573586
28. Kim TW, Park SS, Kim BK, Sim YJ, Shin MS (2009) Effects of sildenafil citrate on peripheral fatigue and exercise performance after exhaustive swimming exercise in rats. *J Exer Rehabil* 15:751–756
29. Hou CW, Chen IC, Shu FR, Feng CH, Hung CT (2019) Protective effect of supplementation with *Lycium ruthenicum* Murray extract from exhaustive exercise-induced cardiac injury in rats. *Chinese Med J* 132:1005–1006
30. Chen HY, Yao YR (2021) Effect of Syncytin-1 overexpression in skeletal muscle cells on co-culture model of spinal cord anterior horn motor neurons, Schwann cells, and skeletal muscle cells. *Chinese J Neuromed* 20:649–655
31. Romani M, Sorrentino V, Oh CM, Li H, de Lima TI, Zhang H, Shong M, Auwerx J (2021) NAD<sup>+</sup> boosting reduces age-associated amyloidosis and restores mitochondrial homeostasis in muscle. *Cell Rep* 34:108660
32. Wang J, Tang L, White J, Fang J (2014) Inhibitory effect of gallic acid on CCl<sub>4</sub>-mediated liver fibrosis in mice. *Cell Biochem Biophys* 69:21–26
33. Nakamura Y, Torikai K, Ohigashi H (2001) Toxic dose of a simple phenolic antioxidant, protocatechuic acid, attenuates the glutathione level in ICR mouse liver and kidney. *J Agri Food Chem* 49:5674–5678
34. Shi HT, Dong L, Jiang J, Zhao JH, Zhao G, Dang XY, Lu XL, Jia M (2013) Chlorogenic acid reduces liver inflammation and fibrosis through inhibition of toll-like receptor 4 signaling pathway. *Toxicology* 303:107–114
35. Pari L, Prasath A (2008) Efficacy of caffeic acid in preventing nickel induced oxidative damage in liver of rats. *Chem-biol Interact* 731:77–83
36. Xie W, Zhang S, Lei F, Ouyang X, Du L (2014) Ananas comosus L leaf phenols and p-coumaric acid regulate liver fat metabolism by upregulating CPT-1 expression. *Evid-based Comple Alter Med* 2014:903258
37. Kandemir FM, Ileriturk M, Gur C (2022) Rutin protects rat liver and kidney from sodium valproate-induced damage by attenuating oxidative stress, ER stress, inflammation, apoptosis and autophagy. *Mol Biol Rep* 49:6063–6074
38. Shen YT, Shen XR, Cheng Y, Liu YL (2020) Myricitrin pretreatment ameliorates mouse liver ischemia reperfusion injury. *Int Immunopharmacol* 89:107005
39. Xie W, Wang M, Chen C, Zhang X, Zhang X, Melzig MF (2016) Hepatoprotective effect of isoquercitrin against acetaminophen-induced liver injury. *Life Sci* 152:180–189
40. Liu X, Huang K, Zhang RJ, Mei D, Zhang B (2020) Isochlorogenic acid A attenuates the progression of liver fibrosis through regulating HMGB1/TLR4/NF- $\kappa$ B signaling pathway. *Front Pharmacol* 11:582
41. Birman H, Dar KA, Kapucu A, Acar S, Uzüm G (2012) Effects of luteolin on liver, kidney and brain in pentylenetetrazol-induced seizures: involvement of metalloproteinases and NOS activities. *Balkan Med J* 29:188–196

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