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Evaluation of the antioxidant potential of defatted extract from *Astragalus spruneri* in spontaneously hypertensive rats (SHRs)

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The aim of this study was to evaluate the antioxidant potential of *Astragalus spruneri* (Fabaceae) in spontaneously hypertensive rats (SHR). Hypertension is a non-communicable disease and oxidative stress is regarded as one of the main pathophysiological mechanisms. Defatted extract of *A. spruneri* (EAS) was administered at a dose of 100 mg/kg bw (1/20 LD_{50}) for 14 days. At the end of the treatment period the animals were euthanized and the activities of catalase (CAT), superoxide dismutase (SOD) and glutathione peroxidase (GPx) as well as the levels of non-enzyme cell protector reduced glutathione (GSH) were assessed in the brain, liver, kidney and spleen of SHR. In comparison to normotensive Wistar rats, in control, non-treated SHRs the GSH level and the activity of GPx were decreased in all organs, while the activity of CAT and SOD was decreased in brain, liver and kidney, and unchanged in spleen. Compared to the control SHRs *A. spruneri* exerted antioxidant activity, discerned by statistically significant increased activities of CAT and SOD in liver and kidney, of GPx and GSH in liver, kidney and spleen. It is worth to be noted that the extract did not exert any effect in the brain. This might be due to the fact that it cannot penetrate the blood brain barrier. Based on the results of our study we could conclude that the lyophilized extract of *A. spruneri* showed antioxidant potential in spontaneously hypertensive rats – a model of essential hypertension in humans.

Keywords: *Astragalus spruneri*, Oxidative stress, SHR, Antioxidant enzymes

INTRODUCTION

Recently it has been shown that oxidative stress is a key player in the pathogenesis of hypertension. A reduction in superoxide dismutase (SOD) and glutathione peroxidase (GPx) activity has been observed in newly diagnosed and untreated hypertensive subjects, which are inversely correlated with blood pressure [1]. Hypertension (HTN) is the most important cardiovascular risk factor, contributing to coronary heart disease and cerebrovascular diseases [2].

Oxidative stress is an independent risk factor in the development of hypertension in experimental animal models, as spontaneously hypertensive rats (SHRs) [3].

If oxidative stress is indeed a cause of hypertension, then, antioxidants should have beneficial effects on hypertension control and reduction of oxidative damage should result in a reduction in blood pressure. A significant body of experimental [4] and clinical trial data [5-7] suggest that diets known to contain significant concentrations of naturally occurring antioxidants appear to reduce blood pressure and may reduce cardiovascular risk.

In relation to this information, dietary and plant-derived antioxidants may have beneficial effects on hypertension and cardiovascular risk factors [8]

Astragalus is a large genus of herbs and small shrubs, belonging to the legume family Fabaceae. In the folk medicine *Astragalus* plants are used for treatment of common cold, upper respiratory infections, allergies, fibromyalgia, anemia, and to regulate the immune system. It is also used for chronic fatigue syndrome (CFS), kidney disease, diabetes, and high blood pressure [9]. The species have been proved to accumulate three main groups of pharmacologically significant metabolites – saponins, flavonoids and polysaccharides. Many flavonoids isolated from some *Astragalus* species have been proved to exert antioxidant effects that inhibit free radical production and act as their scavengers and cytoprotectors. In the body, free

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radicals damage cells and are linked to many health problems including hypertension [10].

Astragalus spruneri Boiss. (Spruner's Milk-vetch) is a clump-forming perennial plant endemic for the Balkan Peninsula and Turkey [11]. The total and differential flavonoid content of the species has been recently evaluated [12]. Up to date there is no information present on its pharmacological action.

In light of these data, we aimed to evaluate the antioxidant and antihypertensive potential of defatted extract from *A. spruneri* in spontaneously hypertensive rats (SHRs), a model of essential hypertension in humans.

MATERIALS AND METHODS

Plant material and preparation of defatted extract

The overground parts of *A. spruneri* were collected during flowering period from Kozhuh Mountain, Rupite area, Bulgaria, in April, 2014. The species was identified by Dr. D. Pavlova from the Faculty of Biology, Sofia University, Bulgaria, where a voucher specimen was deposited (SO-107625).

Air-dried plant material (120 g) was defatted with dichloromethane (800 ml, 24 h) and then extracted with 100% (200 ml) and 80% methanol (1000 ml) *via* percolation for 72 h. The methanol extract was concentrated on a rotary evaporator in order to eliminate the solvent and lyophilized. There were 12 main flavonoids in the extract and the total flavonoid content was 14 mg/g dry weight. Three main saponins were proved as well with total quantity of 8 mg/g dry weight. Quantitative analysis of the extract was performed by a HPLC method (RP C18 column 250 × 4.6 mm, linear gradient of MeOH and 0.1% H_3PO_4 in water as described in [12].

Animals

Experiments were performed with 12 male SHRs (initial body weight 180-230 g) and 12 male NTRs (initial body weight 200-250 g), obtained from Charles River Laboratories (Sülzfeld, Germany). The animals were housed in Plexiglas cages (3 per cage) at 20 ± 2 °C and 12/12 h light/dark cycle. Food and water were provided *ad libitum*. All procedures were approved by the Bulgarian Agency of Food Safety (№ of permission 169) and performed strictly following the principles stated in the European Convention for the Protection of Vertebrate Animals used for Experimental and other Scientific Purposes (ETS 123).

Chemicals

All the reagents used were of analytical grade. Streptozotocin, as well as other chemicals, bovine serum albumin (fraction V), beta–nicotinamide, adenine dinucleotide 2`-phosphate reduced tetrasodium salt (NADPH), reduced glutathione (GSH), oxidized glutathione (GSSG), glutathione reductase (GR) enzyme, and cumene hydroperoxide were purchased from Sigma Chemical Co. (Taufkirchen, Germany). 2,2- Dinitro-5,5 dithiodibenzoic acid (DTNB) was obtained from Merck (Darmstadt, Germany).

Blood pressure measurement

Blood pressure was measured in conscious animals using an automated tail-cuff device (CODA non-invasive blood pressure system, Kent Scientific Corporation, USA). Before the experimental period, the rats were conditioned to the restraining cylinders. Rats were pre-warmed for 10 min using a temperature-controlled warming holder (37 °C) to facilitate tail blood flow before their blood pressure was measured. The mean of three tail-cuff readings was used as the systolic and diastolic blood pressure value. SHRs with highest blood pressure values were taken for the *in vivo* experiment.

Design of the in vivo experiment

The rats were randomly divided into four groups (n=6) as follows:

Group 1: control NTRs, treated with the saline vehicle, administered by gavage at 5 mL/kg bw/day, 14 days.

Group 2: NTRs treated with EAS alone at 100 mg/kg bw/day, 14 days [13].

Group 3: control SHRs, treated with the saline vehicle, administered by gavage at 5 mL/kg bw/day, 14 days.

Group 4: SHRs treated with EAS alone at 100 mg/kg bw/day, 14 days.

The animals in all groups were sacrificed on the 15^{th} day from the beginning of the experiment. Brains, livers, kidneys and spleens from SHRs and NTRs were taken for assessment of parameters of antioxidant status. For all following experiments the excised organs were perfused with cold saline solution (0.9% NaCl), blotted dry, weighed, and homogenized with corresponding buffers (see *Markers of oxidative stress* below).

Markers of oxidative stress

Reduced glutathione (GSH) was assessed by measuring non-protein sulfhydryls after precipitation of proteins with 5% trichloroacetic acid (TCA), using the method described by Bump *et al.* (1983) [14]. A total of 10% homogenates were prepared in 0.05M phosphate buffer (pH 7.4) and centrifuged at 7 000 × g and the supernatant was used for antioxidant enzymes assay.

Glutathione peroxidase (GPx) was measured by NADPH oxidation, using a coupled reaction system consisting of GSH, GR, and cumene hydroperoxide (Tappel, 1978) [15]. Catalase (CAT) activity was determined by measuring the decrease in absorbance at 240 nm of a reaction mixture consisting of H_2O_2 in phosphate buffer, pH 7.0, and requisite volume of supernatant sample. The molar extinction coefficient of 43.6 M cm^{-1} was used to determine catalase activity. The specific activity was calculated and was expressed as μmol/min/mg of total protein (Aebi, 1974) [16]. Superoxide dismutase activity (SOD) was measured according to the method of Misura and Fridovich (1972) [17], following spectrophotometrically the autoxidation of epinephrine at pH=10.4, 30 °C, using the molar extinction coefficient of 4.02 mM^{-1} cm^{-1}.

Histopathological examination

For light microscopic evaluation, pre-sectioned brain, liver, kidney and spleen samples (n=10 per group) were fixed in 10% buffered formalin and thin sections (4 μm) were subsequently stained with hematoxylin/eosin (HE) for general histological features determination [18]. Sections were studied under light microscope Leica DM 500 (DMR + 550, Leica, Wetzlar, Germany)

Statistical analysis

Statistical programme 'MEDCALC' was used. The results were expressed as mean ± SEM for six rats in each group. The significance of the data was assessed using the nonparametric Mann–Whitney test. For both statistical methods, values of $P \leq 0.05$ were considered statistically significant.

RESULTS

Blood pressure

Blood presure values are shown in Table 1. SHRs have higher sistolic blood pressure (SBP) by 45% ($p<0.05$) and higher diastolic blood pressure (DBP) by 44% ($p<0.05$), compared with the normotensive rats. EAS did not change the blood pressure in NTRs. In SHRs, however, the EAS decreased SBP by 18% ($p<0.05$) and DBP by 23% ($p<0.05$).

Table 1. Blood pressure values measured in conscius NTR and SHR rats and in those treated with defatted EAS.

Blood pressure	NTR	SHR	NTR+EAS	SHR + EAS
SBP	136.6 ± 3.4	198 ± 9.5*	128.6 ± 7.2	162.2 ± 9.1+
DBP	78 ± 3.6	112.2 ± 5.2*	82.5 ± 2.1	86.6 ± 6.7+

*p< 0.05 *vs* NTR control; +p<0.05 *vs* SHR control

GSH levels and activity of antioxidant enzymes

The differences in the GSH level and activity of antioxidant enzymes between normotensive and hypertensive rats are presented in Table 2. The results showed that SHRs have lower GSH level and antioxidant capacity in all investigated organs than the respective NTR control group. GSH level was statistically significantly ($p<0.05$) lower in the liver by 22%, in the kidney by 21%, in the brain by 34% and in the spleen by 26 %. CAT activity was significantly ($p<0.05$) lower in the liver, kidney and brain of SHR by 20%, 21% and 37%, respectively. SOD activity was lower in the liver by 18% ($p<0.05$), in the kidney by 22% ($p<0.05$) and in the brain – by 31% ($p<0.05$). GPx activity was lower in the liver by 15% ($p<0.05$), in the kidney and brain by 21% ($p<0.05$), and in the spleen – by 23% ($p<0.05$).

Table 2. GSH levels, antioxidant enzymes GPx, CAT and SOD activity

Parameters	GSH[a]		GPx[b]		CAT[c]		SOD[d]	
Controls	NTR	SHR	NTR	SHR	NTR	SHR	NTR	SHR
Liver	6.62±0.38	5.22±0.15*	0.33±0.01	0.28±0.02*	21.2±0.87	16.9±1.3*	0.28±0.01	0.23±0.01*
Kidney	4.46±0.29	3.55±0.21*	0.24±0.01	0.19±0.01*	16.3±1.02	12.9±0.5*	0.23±0.01	0.18±0.02*
Brain	1.69±0.17	1.11±0.1*	0.43±0.01	0.34±0.02*	42.1±1.1	26.8±1.26*	0.32±0.02	0.22±0.02*
Spleen	3.23±0.24	2.39±0.23*	0.31±0.02	0.27±0.01*	18.9±1.1	16.8±0.7	0.33±0.02	0.35±0.01

*p<0.05 *vs* control NTR; [a]nmol/g tissue; [b]nmol/min/mg protein; [c]μmol /min/mg protein

The effects of the defatted extract from *A. spruneri* (EAS) on the antioxidant capacity of SHR are shown in Fig. 1. Fourteen-day treatment with the EAS increased the level of GSH statistically significantly ($p<0.05$) in the liver, kidney and spleen of SHR by 27%, 26% and 35%, respectively, when compared to control SHR group. CAT activity was increased in the liver and kidney by 25% ($p<0.05$). SOD activity was also increased by the EAS in the liver and kidney in statistically significant manner ($p<0.05$) by 22% and 28%, respectively. GPx activity was increased significantly in the same organs, in the liver by 18% ($p<0.05$) and in the kidney by 26% ($p<0.05$).

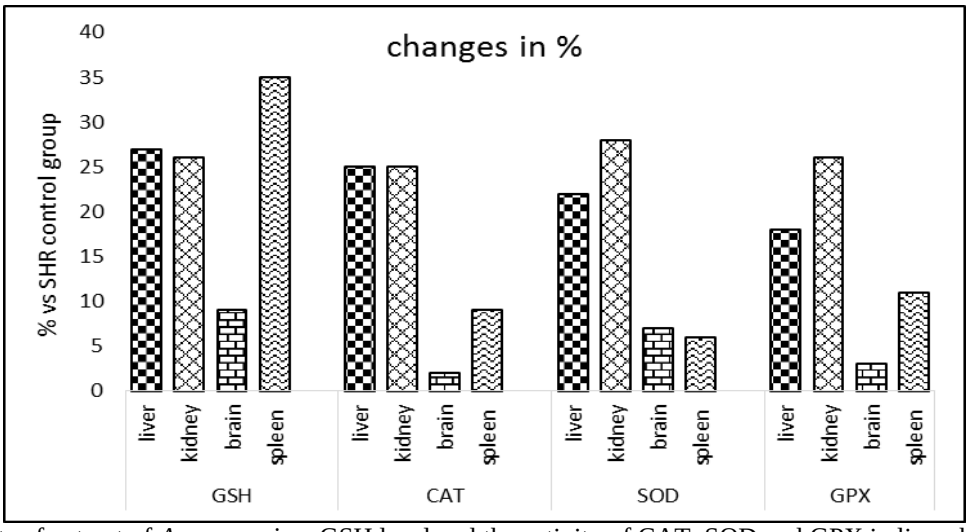

Figure 1. Effects of extract of *A. spruneri* on GSH level and the activity of CAT, SOD and GPX in liver, kidney, brain and spleen from SHR, compared to the control SHR.

Histopathological evaluation

The results from the histopathological examination are shown in Fig. 2. It is visible that EAS did not affect the investigated organs and did not change their morphological structure.

DISCUSSION

In both humans and animals, essential hypertension acts as a risk factor for subclinical organ damages [19] but mechanisms that correlate hypertension and organ damages have not been elucidated extensively. Several experimental and clinical data prove that hypertension and oxidative stress are closely related [20, 21], although it is unclear whether oxidative stress is a cause or an effect of hypertension [22, 23]. The important pathophysiological role of ROS in hypertension development is due, in a large part, to oxygen excess and decreased NO bioavailability in vasculature [24]. Increased oxidative stress has been revealed in genetic and experimental models of hypertension although the effectiveness of antioxidant treatments in reducing blood pressure has been not fully verified [25]. Oxidative stress seems to be a salient feature also in human hypertension. There is evidence that hypertensive patients show both increased oxidative stress and reduced antioxidant capacity [26].

In the current study, we used a genetic model of hypertension, the spontaneously hypertensive rats (SHRs), derived from the normotensive Wistar Kyoto rats (WKY) [27]. We hypothesized that the increased oxidative stress in genetic models of hypertension may be attenuated by natural antioxidants contained in *A. spruneri.*

On the basis of this assumption, the aim of the current study was to investigate the possible antihypertensive and antioxidant activity of the defatted extract from *A. spruneri* on SHR, a model of essential hypertension in humans.

EAS exerted a pronounced antihypertensive effect in SHRs, probably due to flavonoids in this extract. Evidence exists to support several potential mechanisms whereby flavonoids might decrease blood pressure and decrease the severity of hypertension in animals and humans. These mechanisms are: decrease in oxidative stress, interference with the renin-angiotensin-aldosterone system (RAAS), and/or improving vascular function in an endothelium-dependent or independent manner [28].

Our results clearly show a significant depletion in the efficiency of antioxidant enzymes in SHR. In comparison to normotensive Wistar rats, in control, non-treated SHRs the GSH level and the activity of GPx were decreased in all investigated organs, e.g. liver, kidney, brain and spleen by approximately 25%.

The activity of CAT and SOD was decreased in brain, liver and kidney by around 30%, and unchanged in spleen. Our results confirmed literature reports in animal models [24].

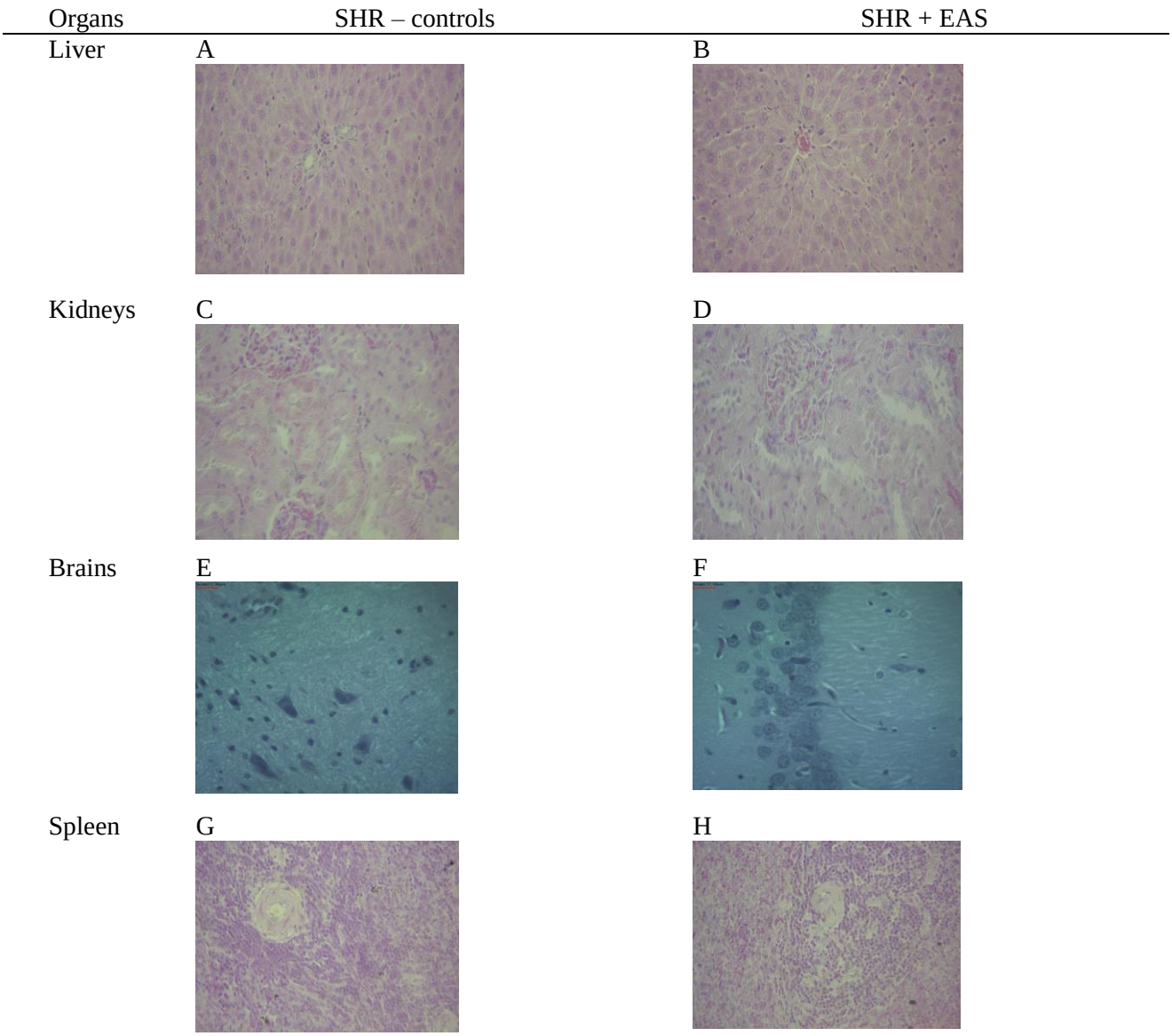

Figure 2. Histological examination of liver, kidney, brain and spleen of control and EAS gavaged SHR.

Compared to the control SHRs *A. spruneri* exerted antioxidant activity, discerned by statistically significant increased activities of CAT and SOD in liver and kidney, of GPx and GSH in liver, kidney and spleen (see Fig. 1). The antioxidant effects observed in our study can be attributed to many phytochemicals in the EAS as they are reported to possess the antioxidant activity. Among these, flavonoids have been very frequently correlated with the antioxidant potential of any plant extract. It has been proposed by Ye *et al.* [29] that flavonoids have a very strong capacity to eliminate free radicals in the blood and promote the activities of antioxidant enzymes such as SOD, GPx, and CAT. These actions of flavonoids are also dose-dependent. This information could explain the observed increase in the activity of the antioxidant enzymes in our study. The extract did not exert any effect in the brain, which might be due to the fact that it cannot penetrate the blood brain barrier. EAS did not affect the activity of antioxidant enzymes in the spleen neither, which might be due to affected blood circulation in this organ in SHR.

Hypertension causes target organ damage (TOD) that involves vasculature, heart, brain and kidneys. Complex biochemical, hormonal and hemodynamic mechanisms are involved in the pathogenesis of TOD. Common to all these processes is an increased bioavailability of reactive oxygen species (ROS). Both *in vitro* and *in vivo* studies explored the role of mitochondrial oxidative stress as a mechanism involved in the pathogenesis of TOD in hypertension, especially focusing on atherosclerosis, heart disease, renal failure, cerebrovascular disease [30]. The SHRs, besides being the most widely used model for essential hypertension, are also an excellent model for studying the development of TOD in the context of

human hypertension. We utilized kidney and brain as target organs and liver and spleen as control tissues, since these organs do not appear particularly susceptible to hypertensive damage [31].

Our results from the histopathological evaluation did not show morphological changes in the investigated organs neither in control SHR nor in EAS treat SHR (fig. 2).

Based on the results of our study we could conclude that under the conditions of our study the defatted extract of *A. spruneri* showed antihypertensive effect and antioxidant potential in the liver and kidney from SHRs – a model of essential hypertension in humans.

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ОЦЕНКА НА АНТИОКСИДАНТНИЯ ПОТЕНЦИАЛ НА ОБЕЗМАСЛЕН ЕКСТРАКТ ОТ *Astragalus spruneri* В СПОНТАННО ХИПЕРТОНИЧНИ ПЛЪХОВЕ

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(Резюме)

Целта на това изследване е да се оцени антиоксидантният потенциал на *Astragalus spruneri* (Fabaceae) в спонтанно хипертонични плъхове (SHR). Хипертонията е незаразна болест и оксидативният стрес се счита за един от основните патофизиологични механизми. Обезмаслен екстракт от *A. spruneri* (EAS) е приложен в доза от 100 mg/kg bw (1/20 LD_{50}) в продължение на 14 дни. В края на този период животните са евтаназирани и активностите на каталаза (CAT), супероксид дисмутаза (SOD) и глутатион пероксидаза (GPx), както и нивата на неензимния клетъчен протектор редуциран глутатион (GSH) са оценени в мозъка, черния дроб, бъбреците и далака на SHR. В сравнение с Wistar плъхове с нормално кръвно налягане, в контролните нетретирани SHR нивото на GSH и активността на GPx са понижени във всички органи, докато активността на CAT и SOD е понижена в мозъка, черния дроб и бъбреците, но е непроменена в далака. В сравнение с контролните SHR, *A. spruneri* проявява антиоксидантна активност, което личи от статистически значимо нарасналите активности на CAT и SOD в черния дроб и бъбреците, и на GPx и GSH в черния дроб, бъбреците и далака. Трябва да се отбележи, че екстрактът няма ефект върху мозъка. Това може да се дължи на факта, че екстрактът не може да проникне през кръвно-мозъчната бариера. От получените резултати може да се заключи, че лиофилизираният екстракт от *A. spruneri* проявява антиоксидантен ефект в спонтанно-хипертонични плъхове – модел за есенциална хипертония при хора.