

## **Effect of Feeding Cake Fortified with Different Concentrations of Ashwagandha Root against Sodium Arsenite Toxicity in Male Rats**

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### **ABSTRACT**

**I**n ayurvedic medicine, herbal plants as ashwagandha (*Withania somnifera*) were used as a powerful plant to cure many of diseases. The present study was carried out to evaluate the protective effect of fortified cake by different concentrations of ashwagandha root powder against sodium arsenite induced toxicity in rats. Thirty male Sprague-Dawley rats weighing ( $120 \pm 10$ g). Rats classified into five groups (6 rats). The first group was kept as a negative control and fed on the basal diet only. Other four groups were administered sodium arsenite at a single dose of 5 mg/kg/day to induce toxicity injury. One of these groups left as positive control (group 2). The third group was treated with 100% wheat flour fortified cake. The fourth group was treated with 10 % ashwagandha powder fortified cake. The fifth group was treated with 20 % ashwagandha powder fortified cake. Laboratory analysis showed that fortification with ashwagandha inhibited the levels of liver injury biomarkers, also improved the kidney function enzymes. These results suggested that ashwagandha has a powerful antioxidant effect which can reduce organ injury through its ability by scavenge the free radical.

**Key words:** *Ashwagandha, Sodium Arsenite , Anti-amnesiac, Hepatotoxicity.*

## INTRODUCTION

Ashwagandha" *Withania somnifera*" also known as winter cherry is one of the most powerful traditional Indian herbs used in Ayurvedic medicine (**Chatterjee and Pakrashi, 1995**). Ashwagandha is belonging to family *Solanaceae* with oval leaves and yellow flowers. It bears red fruit which is native to the dry regions of India, and the Middle East (**Gupta, et al., 2011**). The chemical composition of ashwagandha roots contain high level of protein (10.72%), ash (5.41%), crude fiber (14.58%) and total carbohydrates (65.80%) (**Pingali, et al., 2014**). Many pharmacological studies and investigations have been conducted to study the effects of ashwagandha for human health in an attempt to find its use as a medicinal agent (**Mirjalili, et al., 2009**) in Ayurveda medicine or alternative medicine, ashwagandha is used for improving thinking ability, arthritis, decreasing pain, anxiety, chronic liver disease, asthma and as an adaptogen to

increase energy to stand the daily stress (**Ingrid Hehmeyer and Hanne Schönig Herbal Medicine in Yemen, 2012**).

Recently, ashwagandha shows an anti-amnesiac effect against streptozotocin in neurological degenerative disease which can be a promising alternative treatment for Alzheimer's disease as it improves the formation of memories and physical performance (**Baitharu, et al., 2013**).

Arsenic is a naturally occurring element and released into the environment through agriculture and industry that ubiquitously exists as trivalent ( $\text{As}^{3+}$ , arsenite) and pentavalent ( $\text{As}^{5+}$ , arsenate) forms, and arsenite has been considered to be more toxic when compared with arsenate (**Domingo, 1995 and Baitharu, et al., 2013**).

Sodium arsenite is an inorganic compound with the formula  $\text{NaAsO}_2$  which may be inhaled or absorbed through the skin (**Grund, et al., 2005**). According to clinical observations, the chronic arsenic exposure is a cause of

various disorders such as diabetes mellitus, cardiovascular diseases, nephrotoxicity, and cancer of the skin, bladder and neurotoxicity. In addition, it has been suggested to affect the liver function and to induce hepatotoxicity. During arsenic metabolism, oxygen radicals can be produced frequently which lead to overproduction or accumulation of oxygen free radicals in cells causing damage of DNA, lipids and proteins (Gurr, et al., 2003). Acute sodium arsenite poisoning may lead to abdominal pain, diarrhea, poor appetite and nausea (Ratnaike, 2003). Accumulation of arsenite in vital tissues and organs due to chronic poisoning of sodium arsenite can lead to convulsions, decreased blood pressure, nervous system damage, headache, weakness, eventual paralysis and death (ICPEMC, 1990).

Food fortification is a modern strategy which refers to the addition of micronutrients to processed foods (World Health Organization and Food and Agriculture Organization of

the United Nations, 2006). Reducing micronutrient malnutrition is considered as prime goal of food Fortification to provide adequate levels of the respective nutrients in the diet. In many situations, this strategy can lead to use this nutritional supplementation as medicine which is used in treating many disorders including cancers, also it can used to ensure approaching nutrient deficiencies among people (Gerrior, et al., 2004). The purpose of this study is to review and evaluate the influence of fortified cakes with ashwagnda on the levels of some physiological parameters as kidneys function, liver function and lipid profile of chronic renal failure in rats.

## **MATERIALS &METHODS**

### *Materials*

**Animals:** Thirty male albino rats, *Sprague Dawley* strain, weighing (120 ± 10g) were purchased from the animal house of Agriculture Research Center, Giza, Egypt. The animals were housed in plastic cages, maintained on a natural

light-dark cycle at room temperature of  $26 \pm 2^\circ$  C and fed standard diet according to (Reeves, et al., 1993).

***Chemical and plant products:***

-Sodium Arsenite<sup>®</sup>: (NaAsO<sub>2</sub>) was obtained from Sigma Chemical Co. (St Louis, Mo, USA). Minerals and vitamins constituent, sucrose, glucose and absolute ethanol were obtained from El-Gomhoriya Pharm. and Chem. Ind. Co. Cairo, Egypt.

-Casein (> 85% protein) was obtained from Misr Scientific Company, Giza, Egypt. Cellulose and DL- methionine were purchased from Morgan Company, Cairo, Egypt.

- Corn oil was obtained from the local market. Corn starch was obtained from Starch and Glucose Company, Helwan, Egypt.

-Ashwagandha (*Withania somnifera*) roots and were purchased as dried material from local market in Cairo. Egypt.

-American Wheat flour (72%), sucrose, skim milk, whole egg, salt, baking powder, vanillin,

margarine were purchased from local market in Cairo. Egypt.

***Methods:***

***Phytochemical composition:***

Analyzed phytochemical constituents of the roots of the major phytoconstituents were determined by using the methods of (Adesanya and Sofowora, 1983).

***Preparation of Fortified cakes:***

Fortified cakes were prepared according to the common method of (Penfield and Campbell, 1990). Preparation of cake was carried out by using wheat flour (72%), samples replaced separately with 10 and 20% ashwagandha powder.

***Experimental design:***

The experiment was performed in Animal House in the Food Technology Research Institute, Agriculture Research Center, Giza. After the acclimatization period, rats were divided randomly into two main groups, the first main group (n= 6 rats) fed on the basal diet only

as a negative control. While, the second main group (n= 24 rats) were sodium arsenite administration (Sa, 5 mg/kg BW per day) the selection of arsenic dose and procedure of administration were based on the prior study to (**Chattopadhyay, et al., 2003**). The second main group was given orally 10 ml of water containing sodium arsenite at the dose of 5 mg per kg body weight per day) which classified into positive control (+ve) group and three treated rat groups that treated with cake 100% wheat flour (10g /100 g Basel diet), fortified cake with 10% ashwagandha powder (10g /100 g Basel diet), fortified cake with 20% ashwagandha powder (10g /100 g Basel diet). Body weight (BW) was recorded weekly during the experimental period and feed intake was measured daily during the experimental periods. At the end of the experiment, biological evaluation of the tested diets was carried out by determining total feed intake, body weight gain (BWG) and food efficiency ratio (FER).

### ***Blood sampling:***

At the end of the experiment period (8 weeks), rats were sacrificed after overnight fasting under ether anesthesia. Blood samples were taken from hepatic portal vein and were left to clot by standing at room temperature for 15 minutes, and then centrifuged at 3000 rpm for 20 minutes. Serum was carefully separated and transferred into clean quite fit plastic tubes and kept frozen at -20°C until the time of analysis.

### ***Biochemical analysis:***

Serum alanine and aspartate aminotransferases (ALT & AST) were estimated according to (**Reitman and Frankel, 1957**) while alkaline phosphatase (ALP) was assayed by (**Kind and King, 1954**) and bilirubin levels were estimated according to **Bartholomev and Delany, (1966)**. Serum creatinine, uric acid and urea were determined according to the methods described by (**Bohmer, 1971; Fossati et al., 1980 and Patton and Crouch, 1977**), respectively. Enzymatic colorimetric determination of

triglycerides was carried out according to (**Fossati and Prencipe, 1982**). Total cholesterol was determined by colorimetric method according to (**Allian, et al., 1974**). Determination of HDL-c (high density lipoprotein) was carried out according to the method of (**Fnedewaid, 1972**). The determination of VLDL-c (very low density lipoproteins) and LDL-c (low density lipoproteins) were carried out according to the method of (**Lee and Nieman, 1996**) by calculation as follows:

\* VLDL-c (mg/dl) = Triglycerides /5

\* LDL-c (mg/dl) = Total cholesterol –HDL-c-VLDL-c

Acetyl cholinesterase (AChE) activity was determined according to (**Knedel and Boottger, 1967**). Superoxide dismutase (SOD) activity, total antioxidants capacity (TAC), and malondialdehyde (MDA) were determined according to (**Nishikimi, et al., 1972; Cao, et al., 1993 and Oh-kawa, et al., 1979**), respectively.

### **Statistical analysis:**

The obtained data were statistically analyzed using computerized SPSS (Statistic Program Sigmastat, Statistical Soft-Ware, SAS Institute, Cary, NC). Effects of different treatments were analyzed by one way ANOVA (Analysis of variance) test using Duncan's multiple range test and  $p < 0.05$  was used to indicate significance between different groups (**Snedecor and Cochran, 1967**).

## **RESULTS & DISCUSSION**

Fig (1) shows the phytonutrient constituents of ashwagandha roots, the highest phenolic compound concentration in ashwagandha roots was chlorogenic acid. ashwagandha roots also contain pyrocatechol, caffeic acid, ferutic acid, protocatechuic acid, vanillic acid and pyrogallol.

Table (1) discusses the effect of sodium arsenite and the other treated groups on body weight gain and feed intake. The results indicated that the control group (+ve) Showed a significant decrease in body

weight gain and feed intake compared to Normal control group while the other treated groups showed significant increase in these parameter compared with control group (+ve). There wasn't a significant difference between the treating with ashwagandha powder and wheat flour on body weight gain and feed intake. Our results were matchd with **(Mane, et al., 2012)**, who reported that Inclusion of aswaganda powder in broiler food was beneficial in improving the body weight and feed intake ratio.

The effect of ashwagandha powder fortified cake on liver function enzymes in rats received NaAsO<sub>2</sub> are presented in Table 2. The elevation of liver function enzymes was due to the injury caused by NaAsO<sub>2</sub>, as the positive group (+ve) showed a significant increase in total bilirubin, AST and ALT in comparing to normal control group (-ve). On the other hand, the treated groups with different concentrations of ashwagandha powder improved the results as those levels were found to be

significantly lower in comparing to positive group (+ve). specifically, the cake fortified with ashwagandha powder at different concentrations was better than 100% wheat flour cake in lowering the elevation of hepatic enzymes caused by NaAsO<sub>2</sub>.

Similarly **Udayakumar, (2009)** clarified that both ashwaganda root and leaf have a great role in decreasing the circulating liver enzymes In diabetic animals induced by alloxan at 200mg/kg. Moreover, **(Hosny and Farouk, 2012)** aimed to investigate the protective effect of ashwaganda extract against irradiation of gamma-induced oxidative stress in hepatic cells. His results were closely matched to our study as he suggested that ashwaganda extract abrogated the increases in liver enzymes so it could be used as a preventive drug which protects the hepatic cells in radiotherapy.

Thus, it may be due to the hepatoprotective action of Withanolide A which is exist in ashwaganda by alleviating oxido-nitrosative stress and

inflammatory via inhibition of interleukin-1(IL-1 $\beta$ ), cyclooxygenase activity (COX-2), tumor necrosis factor-alpha (TNF- $\alpha$ ) and total nitric oxide (INOS). Hence, ashwaganda exerts a hepatoprotective effect through several mechanisms which could be attributed to the ability to remove the urea related compounds and antioxidant properties (**Dhuley, 2000**).

In Table (3), results showed that kidney function tests were elevated by NaAsO<sub>2</sub> as it shown in positive group (+ve). The changes in the kidney function tests were due to NaAsO<sub>2</sub> induced kidney injury, as there was significant increase in urea, uric acid and creatinine compared to normal group (-ve). Meanwhile, the other treated groups reversed those results as there was a significant decrease in the levels of creatinine, urea and uric acid except the 100% wheat flour group, as there is a non significant difference in uric acid level compared to positive group. Fortified cake with 20% ashwaganda powder showed the

best results as shown in table (3). Recently (**Jeyanthi and Subramanian, 2009**) investigated the nephroprotective role of withania somnifera on gentamicin induced nephrotoxicity in mice. These studies showed that 500mg/kg of root extract of ashwagandha given to albino rats for two weeks prior to gentamicin induced renal toxicity was able to attenuate the increase in urinary protein and serum creatinine.

Furthermore, ashwagandha contain many of withanolides and alkaloids. (**Harikrishnan, et al., 2008**) withaferin A, 1-oxo-5b, 6b-epoxy-witha-2-ene-27-ethoxy-olide, etc, 32, 33 are the main active compounds in ashwagandha (**Jayaprakasam, 2003**). They have the ability to increase the glomerular filtration rate (GFR) which decrease the serum urea and creatinine levels.

NaAsO<sub>2</sub> induced toxicity (positive group) caused a significant rise in total cholesterol, triglycerides,

VLDL-C and LDL-C while, there was significant decrease in HDL-C in comparing to normal group (-ve) as it shown in table (4). This is due to accumulation of sodium arsenite in liver and kidneys after excessive doses (**Engel, et al., 1994**).

On the other hand, the other treated groups showed significant decrease in serum total cholesterol, triglycerides, VLDL-C and LDL-C while increase in HDL-C compared to positive group (+ve). The fortification with 10% and 20% of ashwagandha demolishes the negative impact of NaAsO<sub>2</sub> on lipid profile more than 100% wheat flour group.

Our results were matched with (**Visavadiya and Narasimhacharya, 2011**) who found that the supplementation of ashwagandha in albino rats with normal cholesterol level for four weeks was seems to decrease the total cholesterol (10.5-16.6%). He also investigated the effect of ashwagandha in diet-induced hypercholesterolemic, founding that there was significant decreasing in LDL-C (49.2-

62.7%) with a significant increase in HDL-C (15.1-17.7%). In other study, there was a reduction in LDL level in otherwise healthy persons who did not have elevated LDL cholesterol at baseline when the subjects given 750-1,250mg of the water extract (**Raut, 2012**). (**Bhattacharya, et al., 1997**) have suggested that ashwagandha exerts the Hypolipidaemic effect due to the antioxidant effect which may be responsible for its pharmacological properties.

Table (5) represent the results of the Effect of ashwagandha on antioxidant parameters in rats received NaAsO<sub>2</sub>. NaAsO<sub>2</sub> induced toxicity caused significant decrease in serum antioxidant enzyme activity; superoxide dismutase SOD and total antioxidants level while increase in malondialdehyde (MDA) activity compared to normal group (-ve). On the other hand, all other treated groups significantly attenuated the decreased levels of MDA and elevated the serum antioxidant enzyme levels in comparing to

positive group (+ve). This result was agreed with (**Visavadiya and Narasimhacharya, 2011**) who noted that lipid peroxidation (MDA) is reduced 12.4-18.2% when rats fed 0.75-1.5% of the diet as ashwagandha root. These anti-peroxidative action observed in are most likely due to free radical scavenging activity of ashwagandha specially at concentration 20%. The potent antioxidant power in ashwagandha is may be due to the presence of several compounds like, flavanoids, alkaloids withanolides and withaferin-A. The reduction in the increased levels of lipid peroxidation because the ashwagandha is known as a good source of flavanoids and polyphenolic compounds which they are consider as potent free radical scavengers, including hydroxyl and superoxide anions ( **Jovanovic and Simic, 2000**). Also ashwagandha root powder proved to have another compounds such as sitoindosides VII–X which have also an antioxidant activity (**Bhattacharya, et al., 1997**).

## **CONCLUSION**

From the above study results, it may be concluded that fortification with ashwagandha (*Withania somnifera*) root powder at different concentration possess antioxidant and antihyperlipidaemic activities in NaAsO<sub>2</sub>-induced toxicity in rats and may have some role in decreasing serum urea and creatinine levels. Ashwagandha root powder appears to prevent the oxidative damage to kidney and liver tissue.

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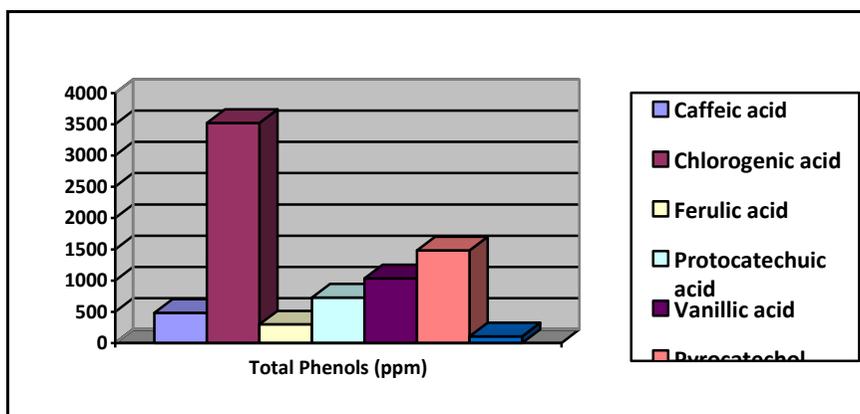
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**Fig 1: Phytonutrient constituent of ashwagandha roots**



**Table (1): Effect of fortified with ashwagandha on feed intake, body weight gain % in rats received NaAsO<sub>2</sub>**

Parameters		Feed intake g/day	Body weight gain %
Groups			
Normal control group		19.43 ±2.31 <sup>a</sup>	94.10 ±9.80 <sup>a</sup>
Control group (+ve)		12.86 ±2.75 <sup>c</sup>	51.15 ±7.62 <sup>c</sup>
<i>Treated groups</i>	Wheat flour 100%	16.85 ±2.24 <sup>b</sup>	77.46 ±8.07 <sup>b</sup>
	Ashwagandha powder 10%	16.99 ±2.19 <sup>b</sup>	71.37 ±10.8 <sup>b</sup>
	Ashwagandha powder 20%	17.72 ±2.37 <sup>b</sup>	72.12 ±9.19 <sup>b</sup>

Values are expressed as mean ± SD. Significance at  $p < 0.05$ .

Values which don't share the same letter in each column are significantly different.

**Table (2): Effect of fortified with ashwagandha on liver functions in rats received NaAsO<sub>2</sub>**

Parameters		AST (μ /ml)	ALT (μ /ml)	Total bilirubin mg / dl
Normal control group		43.63 ±8.50 b	39.07 ±3.43 c	0.49 ±0.03d
control group (+ve)		79.67 ±9.03 a	74.67 ±6.84 a	1.06 ±0.08 a
<i>Treated groups</i>	Wheat flour 100%	64.67 ±8.15 a	54.67 ±5.51 b	0.75 ±0.24 b
	Ashwagandha powder 10%	46.23 ±6.27 b	47.33 ±8.92 bc	0.57 ±0.39 c
	Ashwagandha powder 20%	43.01 ±7.48 b	39.67 ±8.08 bc	0.48 ±0.13 d

Values are expressed as mean ± SD. Significance at p<0.05.

Values which don't share the same letter in each column are significantly different.

AST: Aspartate aminotransferase.

ALT: Alanine aminotransferase.

**Table (3): Effect of fortified with ashwagandha on kidney functions in rats received NaAsO<sub>2</sub>**

Parameters		Uric acid mg/dl	Creatinine mg/dl	Urea mg/dl
Normal control group		2.73 ±0.78 b	1.02 ±0.07 b	40.67 ±3.06 d
Control group (+ve)		5.18 ±0.52 a	1.97 ±0.31 a	64.03 ±2.65 a
<i>Treated groups</i>	Wheat flour 100%	4.80 ±0.57 a	1.47 ±0.15 b	54.0 ±7.64 b
	Ashwagandha powder10%	2.91 ±0.72 b	1.51 ±0.22 b	50.67 ±6.35 b
	Ashwagandha powder 20%	2.82 ±0.93 b	1.19 ±0.06 b	42.01 ±3.21 c

Values are expressed as mean ± SD. Significance at p<0.05.

Values which don't share the same letter in each column are significantly different.

**Table (4): Effect of fortified with ashwagandha on lipid profile in rats received NaAsO<sub>2</sub>**

Parameters		TC mg/dl	TG mg/dl	VLDL-c mg/dl	LDL-c mg/dl	HDL-c mg/dl
Groups						
Normal control group		82.27 ±4.16 d	68.13 ±2.97 d	13.62 ±0.59d	31.16 ±5.99e	37.50 ±2.29a
Control group (+ve)		101.57 ±4.71 a	114.33 ±10.66a	20.78 ±2.13a	54.56 ±3.55a	26.23 ±1.37c
Treated groups	Wheat flour 100%	96.67 ±7.26 b	89.60 ±3.14 b	17.94 ±0.63b	46.56 ±5.76b	32.17 ±1.76b
	Ashwagandha powder 10%	89.80 ±2.44 c	79.47 ±5.22bc	15.89 ±1.04bc	39.77 ±1.34c	34.53 ±3.02b
	Ashwagandha powder 20%	84.27 ±2.89c	74.27 ±1.42cd	14.98 ±0.28cd	32.91 ±3.66d	36.70 ±2.07a

Values are expressed as mean ± SD. Significance at p<0.05.

Values which don't share the same letter in each column are significantly different.

TG: Triglyceride TC: total Cholesterol  
density lipoprotein cholesterol

VLDLc: Very low

LDLc: Low density lipoprotein cholesterol  
lipoprotein cholesterol

HDLc: High density

**Table (5): Effect of fortified with ashwagandha on antioxidant parameters in rats received NaAsO<sub>2</sub>**

Parameters		SOD U/mL	Total antioxidants mmol/L	MDA mmol/L
Normal control group		198.23 ±12.74 a	3.12 ±0.75 a	4.47 ±0.76 c
Control group (+ve)		114.33 ±12.01 c	1.17 ±0.55 b	11.05 ±1.16 a
<b>Treated groups</b>	Wheat flour 100%	147.12 ±13.01 bc	1.23 ±0.65 b	8.2 ±0.86 b
	Ashwagandha powder 10%	163.33 ±14.81b	1.99 ±0.80 ab	5.11 ±0.62 c
	Ashwagandha powder 20%	173.33 ±14.15 a	2.73 ±0.68 a	4.76 ±0.43 c

Values are expressed as mean ± SD.

Significance at p<0.05.

Values which don't share the same letter in each column are significantly different.

SOD: Superoxide dismutase.

MDA: Malondialdehyde.

# تأثير التغذية بالكيك المدعم بواسطة تركيزات مختلفة من جذور الاشواجاندا ضد السمية الناتجة عن تناول زرنخ الصوديوم في ذكور الجرذان

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## الملخص العربي

في الطب الهندي القديم، كانت النباتات العشبية مثل نباتالاشواجاندا تعتبر من النباتات القوية المستخدمة لعلاج العديد من الأمراض. وأجريت هذه الدراسة لتقييم التأثير الوقائي للكيك المدعم بتركيزات مختلفة من مستخلص جذورالاشواجاندا ضد السمية الناتجة عن زرنخ الصوديوم في الفئران. ثلاثون من ذكور الفئران البيضاء (سبراج داولي) وزنها  $120 \pm 10$  جرام تم تقسيمهم إلى خمسة مجموعات (٦ فئران). المجموعة الأولى (الضابطة السالبة) والتي تغذت على الوجبة القياسية فقط، أما المجموعات الأربعة الأخرى تم اعطاها زرنخ الصوديوم بجرعة ٥ ملجم / كجم/من وزن الجسم في لاحداث الإصابة بالسمية. ثم قسمت إلى ٤ مجموعات على النحو التالي : مجموعة (٢) ابققت كمجموعة الضابطة الموجبة. المجموعة (٣) تغذت على كيك مدعم ب١٠٠% دقيق القمح. عولجت المجموعة (٤) بكيك مدعم ب١٠% من مستخلص جذور الاشواجاندا. بينما عولجت المجموعة (٥) بكيك مدعم ب٢٠% من مستخلص جذور الاشواجاندا. اظهرت النتائج المختبرية ان التدهيم بجذور الاشواجاندا تثبط مستوي المؤشرات الحيوية الخاصة بالكبد، كما انها تحسن من انزيمات وظائف الكلي. وتشير هذه النتائج إلى أن نبات الاشواجاندا لها تأثير قوي كمضاد للاكسدة والتي تقلل من إصابة الاعضاء الحيوية عن طريق مسح الشقوق الحرة.