

# The Potential Ameliorative Role of Ashwagandha (*Withania somnifera*) Roots Extract on Oxidative Stress

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## Abstract

The present study was intended to investigate the potential ameliorative role of ashwagandha roots extract (ARE) on oxidative stress. Thirty (30) adult male albino rats were randomly distributed into 2 main groups. The first main group (6 rats) fed on the basal diet as a negative control group (normal rats). The second main group (24 rats) injected by a single intraperitoneal dose of potassium bromate at dose of 125 mg/kg body weight to induce oxidative stress then reclassified into 4 sub-groups one of them left as a positive control group and other three groups were given three doses of ashwagandha roots ethanolic extract (150, 300 and 450 mg/kg body weight) orally, respectively. The treatment lasted for 28 days. Also, the potential effect of ashwagandha roots extract against the human liver carcinoma cell line (HEPG2) has been studied in vitro. The obtained results revealed that oral administration of ashwagandha roots extract for 28 days (150, 300, 450 mg / kg body weight) caused significant ( $P \leq 0.05$ ) increases in HDL, Catalase and Glutathione transferase but with significant ( $P \leq 0.05$ ) decreases in serum glucose level, TC, TG, LDL, VLDL, MDA, kidney function and liver function markers. Also, ashwagandha roots extract caused death of liver cancer cells. In conclusion, the study recommended using ashwagandha in our daily diets to block many complications by controlling oxidative stress.

**Key words:** *Withania somnifera*, *Potassium bromate*, *Free radicals*, *Liver cancer cells*.

## Introduction

The close association between oxidative stress and lifestyle-related diseases has become well known. Oxidative stress, defined as a disturbance in the balance between the production of reactive oxygen species (free radicals) and antioxidant defenses (**Radak et al., 2013**). Overproduction of free radicals can cause oxidative damage to biomolecules, (lipids, proteins & DNA), eventually leading to many chronic diseases such as atherosclerosis, cancer, diabetics, rheumatoid arthritis, post-ischemic perfusion injury, myocardial infarction, cardiovascular diseases, chronic inflammation and stroke (**Phaniendra et al., 2015**).

Potassium bromate ( $\text{KBrO}_3$ ) is widely used as a food additive. The toxic effects of  $\text{KBrO}_3$  are attributed to its ability to induce oxidative stress leading to enhanced production of reactive oxygen species which cause oxidative damage to essential macromolecules. The reactive oxygen species are widely thought to be generated in the cell because of reduction of  $\text{KBrO}_3$  to bromide by intracellular reductants (**Ahmad and Mahmood, 2016**).

In the last few years, there has been an exponential growth in the field of herbal medicine, and these drugs are gaining popularity both in developing and developed countries because of their natural origin and less side effects (**Bhardwaj et al., 2018**). Among these *Withania somnifera* is popularly known as ashwagandha belonging to the family *Solanaceae*, is a traditional herb known worldwide for its numerous beneficial health activities since ancient times. Ashwagandha is an important source of many pharmacologically and medicinally important chemicals (**Dutta et al., 2019**). The various chemical constituents present in ashwagandha are anaferine, anahygrine,  $\beta$ -Sitosterol, chlorogenic acid, cysteine, cuscohygrine, iron, pseudotropine, scopoletin, somniferinine, somniferiene, tropanol, withanine, withananine and withanolides A-Y (steroidal lactones) (**Dubey and Chinnathambi, 2019**). Presence of diverse bioactive constituents and their low toxicity profiles, and novel mechanism of action makes ashwagandha a suitable drug candidate for the treatment of various diseases (**Mahrous et al., 2019**). This medicinal plant provides benefits against many human illnesses such as epilepsy, depression, arthritis, diabetes and palliative effects such as analgesic, rejuvenating, regenerating, and growth-promoting effects. Several clinical trials of the different parts of the herb have demonstrated safety in patients suffering from these diseases (**Tripathi et al., 2020**). Therefore, the present study was designed to evaluate the utilization of ashwagandha roots ethanolic extract (ARE) as natural antioxidant to prevent the side effect of oxidative stress.

## Materials and methods

**Plants:** Ashwagandha roots powder was purchased from Harraz for Food Industry and Natural Products, Bab Alkhalq, Cairo, Egypt.

**Chemicals:** Potassium bromate ( $\text{KBrO}_3$ ) was purchased from El-Gomhoriya Company for Trading Drugs, Chemicals and Medical instruments.

**Diets:** The basic diet prepared according to the following formula as mentioned by **AIN (1993)** as follow: protein (10%), corn oil (10%), vitamins mixture (1%), mineral mixture (4%), choline chloride (0.2%), methionine (0.3%), cellulose (5%), and the remained is corn starch (69.5%). The used vitamin mixture component was that recommended by **Campbell (1963)** while the salts mixture used was formulated

according to **Hegsted *et al.*, (1941)**. Diet contents were purchased from El-Gomhoriya Company for Trading Drugs, Chemicals and Medical instruments.

**Ethanolic extract:** Extraction from ashwagandha roots powder was carried with 70% ethanol at room temperature by cold percolation process. The extracted material was filtered and concentrated using rotary evaporator at 45 °C. Concentrated material of herb was lyophilised to obtain lyophilised ethanolic extract of herb according powder was used for further study (**Jain *et al.*, 2010**).

**Experimental design:** Thirty (30) adult male albino rats weighting  $150 \pm 10$  g were obtained from Research Institute of Ophthalmology, Medical Analysis Department, Giza, Egypt and housed individually in wire cages in a room maintained at  $25 \pm 2$  °C and kept under normal healthy conditions. All rats were fed on basal diet for one-week before starting the experiment for acclimatization. After one-week period, rats were randomly distributed into 2 main groups. The first main group fed on the basal diet as a negative control group (normal rats). The second main group injected by a single intraperitoneal dose of potassium bromate at dose of 125 mg/kg body weight to induce oxidative stress (**Khan and Sultana, 2004**), then reclassified into 4 sub-groups one of them left as a positive control group. The other three groups fed on three doses of ashwagandha roots extract (ARE) (150, 300 and 450 mg/kg body weight) orally, respectively. The treatment lasted for 28 days.

**Blood Sampling:** At the end of the experiment, rats were fasted overnight and anesthetized with diethyl ether. Blood samples were collected in clean dry centrifuge tubes from hepatic portal vein; serum obtained by centrifugation was carefully aspirated, transferred into clean cuvette tubes and stored frozen at -20°C for analysis (**Malhotra, 2003**).

**biochemical analysis:** The serum glucose level was determined according to **Kaplan (1984)**. Alanine amino transferase (ALT), aspartate amino transferase (AST) and alkaline phosphatase (ALP) were estimated according to **Tietz (1976), Henry (1974) and Moss (1982)**, respectively. Serum total cholesterol, triglycerides and HDL were determined according to **Allain (1974), Fossati and prncipe (1982) and Lopez (1977)**, respectively. Determination of LDL and VLDL was carried out according to the method of **Lee and Nieman (1996)**. Urea, uric acid and creatinine were determined according to **Malhotra (2003), Fossati and Principe (1980) and Bohmer (1971)**. Malondialdehyde (MDA), glutathion transferase (GTF) and Catalase enzyme (CAT) were assayed according to **Ohkawa *et al.*, (1979), HU (1994) and Diego (2011)**, respectively.

**The sulphorhodamine (SRB) assay:** The potential effect of AREE against the human liver carcinoma cell line (HEPG2) was tested in vitro by SRB assay according to Skehane *et al.*, (1990) in National Cancer Institute, Cairo, Egypt.

**Statistical Analysis:** The data were statistically analyzed using a computerized program by one way ANOVA .The results are presented as mean  $\pm$  SD. Differences between treatments at ( $p \leq 0.05$ ) were considered significant (Steel and Torrie, 1980).

## Results and discussion

### Effect of ashwagandha roots extract on liver functions of intoxicated rats with $KBrO_3$

The activities of liver functions marker enzymes namely AST, ALT and ALP were significantly increased in intoxicated rats as compared to normal control group as presented in Table (1). The elevated levels of AST, ALT and ALP were significantly decreased after the administration of ARE (150, 300 and 400 mg/kg B.Wt.) in treated rats when compared with positive control group.

**Table (1): Effect of ashwagandha roots extract on liver functions of intoxicated rats with  $KBrO_3$**

Groups	AST (U/L)	ALT (U/L)	ALP (U/L)
	Mean $\pm$ SD	Mean $\pm$ SD	Mean $\pm$ SD
(1) control (-)	59.53 <sup>e</sup> $\pm$ 3.16	76.46 <sup>e</sup> $\pm$ 1.55	120.23 <sup>e</sup> $\pm$ 1.80
(2) control (+)	95 <sup>a</sup> $\pm$ 2	173.80 <sup>a</sup> $\pm$ 1.94	252.86 <sup>a</sup> $\pm$ 2.69
(3) ARE (150 mg/kg B.Wt.)	80.76 <sup>b</sup> $\pm$ 2.15	164.83 <sup>b</sup> $\pm$ 2.37	213 <sup>b</sup> $\pm$ 3
(4) ARE (300 mg/kg B.Wt.)	74.4 <sup>c</sup> $\pm$ 1.83	121 <sup>c</sup> $\pm$ 1.74	178.56 <sup>c</sup> $\pm$ 2.69
(5) ARE (450 mg/kg B.Wt.)	64 <sup>d</sup> $\pm$ 2.64	82.76 <sup>d</sup> $\pm$ 2.47	131 <sup>d</sup> $\pm$ 2.64
L.S.D	4.381	3.730	4.727

Values are mean  $\pm$  SD. Values in the same column sharing the same superscript letters are not statistically significantly different ( $p \leq 0.05$ ).

The results agreed with the once reported by Saxena *et al.*, (2007) who studied the hepato-protective effect of ashwagandha and found a decrease in the activity of liver enzymes, which indicates the important role that ashwagandha plays in improving liver function. Also, Sabiba *et al.*, (2013) and Jamuna *et al.*, (2018) suggested that ashwagandha possesses promising hepato-protective effects. Treatment of the diabetic rats with ashwagandha root extract restored the changes of AST, ALT and ALP to their normal level (Swamy *et al.*, 2019). The hepato-protective effect of ashwagandha may be due to its high antioxidant content (Harikrishnan *et al.*, 2008).

## Effect of ashwagandha roots extract on kidney functions of intoxicated rats with $KBrO_3$

Table (2) shows the effect of ashwagandha roots extract on kidney functions of negative and intoxicated groups. Data showed that exposure of rats to  $KBrO_3$  can increase kidney function markers (urea, creatinine and uric acid) in positive control group compared with negative group. All treated groups with ashwagandha roots extract showed significantly ( $p \leq 0.05$ ) decrease in kidney function markers, as compared to the positive control group, on the other hand, kidney function markers decreased gradually with increasing the level of ashwagandha roots extract.

**Table (2): Effect of ashwagandha roots extract on kidney functions of intoxicated rats with  $KBrO_3$**

Groups	Urea (mg/dl)	Creatinine (mg /dl)	Uric acid (mg/dl)
	Mean $\pm$ SD	Mean $\pm$ SD	Mean $\pm$ SD
(1) Control (-)	30.43 <sup>c</sup> $\pm$ 1.59	0.65 <sup>c</sup> $\pm$ 0.15	2.01 <sup>c</sup> $\pm$ 0.08
(2) Control (+)	59.93 <sup>a</sup> $\pm$ 2.45	1.53 <sup>a</sup> $\pm$ 0.15	4.63 <sup>a</sup> $\pm$ 0.32
(3) ARE (150 mg/kg B.Wt.)	51.83 <sup>b</sup> $\pm$ 2.07	1.13 <sup>b</sup> $\pm$ 0.25	4.03 <sup>b</sup> $\pm$ 0.15
(4) ARE (300 mg/kg B.Wt.)	42.16 <sup>c</sup> $\pm$ 1.53	1 <sup>bc</sup> $\pm$ 0.10	3.7 <sup>b</sup> $\pm$ 0.20
(5) ARE (450 mg/kg B.Wt.)	34.76 <sup>d</sup> $\pm$ 1.42	0.68 <sup>c</sup> $\pm$ 0.10	2.36 <sup>c</sup> $\pm$ 0.15
L.S.D	3.381	0.293	0.361

Values are mean  $\pm$  SD. Values in the same column sharing the same superscript letters are not statistically significantly different ( $p \leq 0.05$ )

The present study is in accordance with **Grunz-Borgmann *et al.*, (2015)** who suggested that ashwagandha may represent a botanical approach for the management of renal dysfunction. Also, **Rasheed *et al.*, (2020)** illustrated that cisplatin induced renal toxicity in albino wistar rats may be corrected by ashwagandha root extract through the renal function analysis (serum urea and creatinine levels). Ashwoganda's ability to improve kidney function is due to its content of flavonoids and phenolic compounds, which have the ability to remove excess ammonia, urea, uric acid, non-protein, nitrogen and creatinine and offer protection against hyper-ammonemic and nephrotoxic conditions (**Harikrishnan *et al.*, 2008**). Also, **Govindappa *et al.*, (2019)** found that ashwagandha with its antioxidant function significantly alleviates adverse effects of gentamicin, ashwagandha decreases MDA, total protein, BUN and Cr levels.

## Effect of ashwagandha roots extract on lipid profiles of intoxicated rats with $KBrO_3$

Data in table (3) showed that administration with  $KBrO_3$  can lead to increase levels of TC, TG, LDL and VLDL in control positive group compared with negative control group while HDL had opposite trend. The results indicated that treatment with (150, 300 and 450 mg/kg B.Wt.) ARE can decrease LDL, TC, TG and VLDL levels and increase HDL level compared with positive control group.

**Table (3): Effect of ashwagandha roots extract on lipid profiles of intoxicated rats with  $KBrO_3$**

Group	T.C	T.G	HDL	LDL	VLDL
	(mg/dl)	(mg/dl)	(mg/dl)	(mg/dl)	(mg/dl)
	Mean $\pm$ SD	Mean $\pm$ SD	Mean $\pm$ SD	Mean $\pm$ SD	Mean $\pm$ SD
(1) control (-)	118.9 <sup>e</sup> $\pm$ 2.45	134.46 <sup>e</sup> $\pm$ 2.56	83.1 <sup>a</sup> $\pm$ 1.95	8.90 <sup>e</sup> $\pm$ 2.91	26.89 <sup>e</sup> $\pm$ 0.51
(2) control (+)	189.66 <sup>a</sup> $\pm$ 7.13	183.76 <sup>a</sup> $\pm$ 3.94	32.5 <sup>d</sup> $\pm$ 2.55	120.41 <sup>a</sup> $\pm$ 2.14	36.75 <sup>a</sup> $\pm$ 0.78
(3) ARE (150 mg/kg B.Wt.)	170 <sup>b</sup> $\pm$ 2.64	176.83 <sup>b</sup> $\pm$ 2.21	43.63 <sup>c</sup> $\pm$ 2.56	91.28 <sup>b</sup> $\pm$ 3.72	35.36 <sup>b</sup> $\pm$ 0.44
(4) ARE (300 mg/kg B.Wt.)	143.3 <sup>c</sup> $\pm$ 2.56	156.3 <sup>c</sup> $\pm$ 2.66	64 <sup>b</sup> $\pm$ 2.64	48.04 <sup>c</sup> $\pm$ 3.48	31.26 <sup>c</sup> $\pm$ 0.50
(5) ARE (450 mg/kg B.Wt.)	123.9 <sup>d</sup> $\pm$ 1.87	141 <sup>d</sup> $\pm$ 2	81 <sup>a</sup> $\pm$ 1.73	14.7 <sup>d</sup> $\pm$ 0.5	28.2 <sup>d</sup> $\pm$ 0.40
<b>L.S.D</b>	<b>4.103</b>	<b>5.027</b>	<b>4.221</b>	<b>5.106</b>	<b>1.005</b>

Values are mean  $\pm$  SD. Values in the same column sharing the same superscript letters are not statistically significantly different ( $p \leq 0.05$ )

**Verma and kumar (2011)** results came to a conclusion indicating that hypocholesterolemic effects of ashwagandha root were assessed in human subjects, significant decreases in serum cholesterol, triglycerides and low-density lipoproteins were seen. Also, **Pal et al., (2015)** illustrated that ashwagandha root powder has been found to decrease total lipids, cholesterol and triglycerides in hypercholesteremic animals. On the other hand, the extract also significantly increased plasma HDL-cholesterol levels and bile acid content of liver.

Similar results were obtained by **Ojha and Arya, (2015)** who reported that herbal cocktail containing ashwagandha used as an adjunct to conventional anti-

ischemic drugs has been found to reduce total cholesterol, triglycerides and increase high density lipoprotein cholesterol in the post myocardial infarction patients.

Treatment of the diabetic rats with ashwagandha root extract restored the changes of serum lipids except high density lipoprotein-bound cholesterol (HDL-c) and tissues like liver, kidney and heart lipids to their normal level, indicating that ashwagandha root extract possess hypolipidaemic activities in alloxan-induced diabetes mellitus rats (Swamy *et al.*, 2019).

### Effect of ashwagandha roots extract on serum glucose level of intoxicated rats with $KBrO_3$

Effect of ashwagandha roots extract on serum glucose level of intoxicated rats with  $KBrO_3$  is recorded in Table (4). Results indicated that the mean value of positive control group was significantly higher than that of negative control group (healthy rats) concerning serum glucose level. The mean values of G3, G4, and G5 were lower than that of positive control group. Rats fed on 450 mg kg B.Wt. ashwagandha roots extract (G5) recorded as the best treatment for serum glucose level.

**Table (4): Effect of ashwagandha roots extract on serum glucose level of intoxicated rats with  $KBrO_3$**

Groups	Serum glucose (mg/dl)
	Mean $\pm$ SD
(1) Control (-)	74.73 <sup>e</sup> $\pm$ 4.05
(2) Control (+)	138.83 <sup>a</sup> $\pm$ 1.68
(3) ARE (150 mg/kg B.Wt.)	130.5 <sup>b</sup> $\pm$ 1.55
(4) ARE (300mg/kg B.Wt.)	107.5 <sup>e</sup> $\pm$ 2.45
(5) ARE (450 mg/kg B.Wt.)	83.43 <sup>d</sup> $\pm$ 3.15
L.S.D	4.988

Values are mean  $\pm$  SD. Values in the same column sharing the same superscript letters are not statistically significantly different ( $p \leq 0.05$ ).

These results are supported by the results published by Khalili (2009) who illustrated that ashwagandha may be considered as a potential treatment for painful diabetic neuropathy. Also, Belal *et al.*, (2012) showed that ashwagandha utilise different mechanisms to exert hypoglycaemic effect and has phytochemical component which can prevent stress induced hyperglycaemia.

The same trend of change was found by Nagaraj and Veeresham, (2018) who reported also that the blood glucose levels also significantly reduced when pretreated with ashwagandha than glimepiride alone treated diabetic rats. The effect of the

ashwagandha might be due to the inhibition of drug metabolizing enzyme CYP2C9 in the liver microsomes.

### Effect of ashwagandha roots extract on MDA, CAT and GTF level of intoxicated rats with $KBrO_3$

Data in Table (5) shows the effect of ashwagandha roots extract on MDA, CAT and GTF level of intoxicated rats with  $KBrO_3$ . Treatment with ARE (150,300 and 450 mg/kg) significantly improved the levels of CAT and GTF in a dose dependent manner when compared to positive control rats. However, administration of ashwagandha roots extract led to decrease in MDA level. The highest reduction in MDA was observed in rats treated with 450 mg/ kg B.Wt. ARE.

**Table (5): Effect of ashwagandha roots ethanolic extract on MDA, CAT and GTF level of intoxicated rats with  $KBrO_3$**

Groups	MDA (mol/L)	CAT(mol/L)	GTF(mol/L)
	Mean $\pm$ SD	Mean $\pm$ SD	Mean $\pm$ SD
(1) Control (-)	17.76 <sup>d</sup> $\pm$ 2.54	287.96 <sup>a</sup> $\pm$ 2.95	622 <sup>a</sup> $\pm$ 2.64
(2) Control (+)	37.53 <sup>a</sup> $\pm$ 1.74	130.26 <sup>c</sup> $\pm$ 2.19	423 <sup>c</sup> $\pm$ 3
(3) AREE (150 mg/kg B.Wt.)	31.26 <sup>b</sup> $\pm$ 1.95	174.9 <sup>d</sup> $\pm$ 3.70	463.8 <sup>d</sup> $\pm$ 2.62
(4) AREE (300 mg/kg B.Wt.)	25 <sup>c</sup> $\pm$ 1	224.3 <sup>c</sup> $\pm$ 3.10	586.86 <sup>c</sup> $\pm$ 2.27
(5) AREE (450 mg/kg B.Wt.)	19.83 <sup>d</sup> $\pm$ 2.17	263 <sup>b</sup> $\pm$ 2	617 <sup>b</sup> $\pm$ 2.64
L.S.D	3.55	5.203	4.816

Values are mean  $\pm$  SD. Values in the same column sharing the same superscript letters are not statistically significantly different ( $p \leq 0.05$ )

These results are in agreement with **Verma and Kumar, (2011)** who reported that ashwagandha extract significantly decreased lipid peroxidation in the liver homogenate and significantly increased catalase activity, promoting scavenging of free radicals that can cause cellular damage.

Studies conducted in rat brains showed that ashwagandha produced an increased level of the three natural antioxidants SOD, CAT and GPx (**Alam et al., 2012**). The levels of GSH, SOD, CAT, and GPX in the exposed tissue returned to near normal values following administration of ashwagandha extract (**Pal et al., 2012**).

These results are completely in agreement with **Sabiba et al., (2013)** who suggested that ashwagandha was able to restore the levels of antioxidants (superoxide dismutase, catalase, reduced glutathione and glutathione-S- transferase) of paracetamol intoxicated rats. Also, **Dar et al., (2015)** reported that glycol-withanolides from ashwagandha exhibited significant anti-oxidant activity in cortex and striatum of rat brain by inducing



a dose-related increase in superoxide dismutase, catalase, and glutathione peroxidase activity.

Ashwagandha glycol-withanolides tended to normalize the augmented SOD and LPO activities and enhanced the activities of CAT and GPX (Sengupta *et al.*, 2018).

### Effect of ashwagandha roots extract on human liver cancer cells

The effect of ashwagandha roots extract on human liver cancer cells is shown in table (6) and figure (1). The previous mentioned table results indicated firstly that ashwagandha roots extract had apoptotic action on human cancer liver cells. Apoptosis occurs normally during development and aging and a homeostatic mechanism to maintain cell population in tissues.

Irradiation or drugs used for cancer chemotherapy results in DNA damage in some cells, which can lead to apoptotic death through so called p53 dependent pathway (Elmore, 2007). Meanwhile nothing is found in the previous literature saying that ashwagandha roots extract has any toxicity for human. On the other side data of Table (6) as illustrated in figure (1) indicate the value of ashwagandha roots extract to combat human liver cancer.

It is evident that surviving fraction, showing a level of 23 ug/ml HEPG2-1, ashwagandha roots extract encouraging apoptosis and death of cancer cells may not be exactly that encouraging metabolism and avoid deterioration of the organs functions. Anyhow ARE proved to show appreciable cancer cells apoptosis.

**Table (6): Median lethal concentration (LC50) of ashwagandha roots extract that kills 50% of human liver cancer cells (surviving cells)**

Concentration (ug/ml)	Survival fraction of cancer cell
	HEPG2
0.000	1.000
12.500	0.751
25.000	0.375
50.000	0.216
100.000	0.216
IC50(ug/ml)	23

HEPG2 (liver carcinoma cell line). IC50 (does of the tested compound which reduces survival cells to 50%).

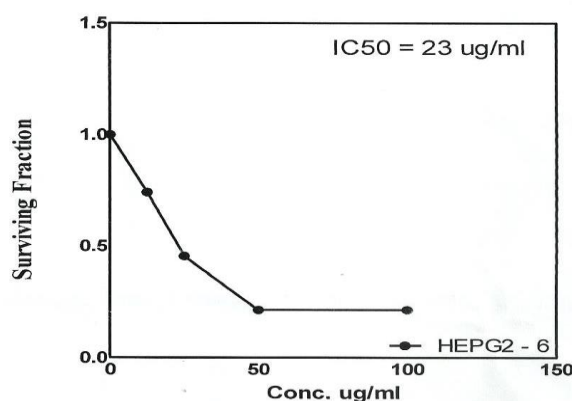


Fig. (1): Median lethal concentration (LC50) of ashwagandha roots

extract that kills 50% of human liver cancer cells (surviving cells)

Results of present work are in line with that of **Dutta et al., (2019)** who reported that ashwagandha extracts containing complex mixtures of diverse components including withaferin A, which have shown inhibitory properties against many cancers, (breast, colon, prostate, colon, ovarian, lung, brain), along with their mechanism of actions and pathways involved. **Nile et al., (2019)** concluded that the withaferin A showed significant reduction in cell viability of cervical cancer (HeLa) cells, with IC50 values 10 mg/ml and 8.5  $\mu$ M/ml, respectively but no cytotoxic effect for normal cells (MDCK). Also, **Sundar et al., (2019)** suggested that withanolides may serve as highly potent anticancer compounds for treatment of cancers harboring a p53 mutation.

Studies of **Guo et al., (2019)** and **Hassannia et al., (2020)** agree with results of present work in that withaferin A, an anticancer constituent of the plant ashwagandha, inhibits tumor growth in association with apoptosis induction.

In conclusion, ashwagandha is a medically important herb and has a proven impact on human health. The findings from this study suggest that ethanolic extract of ashwagandha root was associated with a significant reduction of oxidative stress. Hence, using this herb as a supplement to decrease the oxidative stress could be an excellent alternative options so we should use ashwagandha in our daily diets.

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## الدور المحسن المحتمل لمستخلص جذور الاشوجندا على الاجهاد التأكسدي

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### ملخص البحث:

تهدف الدراسة الحالية إلى معرفة الدور المحسن المحتمل لمستخلص جذور الاشوجندا على الاجهاد التأكسدي. تم توزيع ثلاثين (30) من ذكور الفئران بشكل عشوائي على مجموعتين رئيسيتين: المجموعة الأولى ( 6 فئران ) تغذت على الوجبة الاساسية كمجموعة ضابطة سالبة (فئران طبيعية). وتم حقن المجموعة الرئيسية الثانية ( 24 فأر ) بجرعة واحدة داخل الصفاق من برومات البوتاسيوم بجرعة 125 مجم / كجم من وزن الجسم لاحداث الإجهاد التأكسدي ثم أعيد تصنيفها إلى 4 مجموعات فرعية ، تركت إحداها كمجموعة ضابطة موجبة وتم اعطاء الثلاث مجموعات الاخرى ثلاث جرعات من المستخلص الكحولي لجذور الاشوجندا (150 و 300 و 450 ملجم / كجم من وزن الجسم) عن طريق الفم ، على التوالي. وقد استمر العلاج لمدة 28 يومًا. كما تم دراسة التأثير المحتمل لمستخلص جذور الاشوجندا ضد خلايا سرطان الكبد البشري (HEPG2) معمليا خارج الجسم . وقد أظهرت النتائج المتحصل عليها أن تناول مستخلص جذور الاشوجندا عن طريق الفم لمدة 28 يومًا (150 ، 300، 450 ملجم / كجم من وزن الجسم) تسبب في زيادة معنوية ( $P \leq 0.05$ ) في الليبوبروتين مرتفع الكثافة والكتاليز والجلوتاثيون ترانسفيريزوحدوث انخفاض معنوي في مستوى جلوكوز الدم ، الدهون الثلاثية ، الكوليسترول الكلى ، الليبوبروتين منخفض الكثافة ، الليبوبروتين منخفض الكثافة جدا ، المالمونالدهيد ، وظائف الكلى ووظائف الكبد. كما تسبب مستخلص جذور الاشوجندا في تشجيع موت الخلايا السرطانية. وقد أوصت الدراسة باستخدام الاشوجندا في وجباتنا الغذائية اليومية لمنع العديد من المضاعفات عن طريق التحكم في الإجهاد التأكسدي.

**الكلمات المفتاحية:** الاشوجندا، برومات البوتاسيوم، الجذور الحرة، خلايا الكبد السرطانية.