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Using of Gum Arabic to Ameliorate Liver Disorders Induced by Benzo(*a*)pyrene in Rats

Prof. Dr.SherifSabryRagab[1], Abeer N. Abdel Rahman[2], Hoda Atef Abd El-Aziz[3]

Department of Nutrition and Food Science, Faculty of Home Economics, Menoufia University, Shebin El-kom, Egypt[1,2].Department Nutrition and Food Science, Faculty of Home Economics, Al-Azhar University, Nawag, Egypt[3].

Abstract:
This study was carried out to evaluate the effects of Gum Arabic (GA) in liver functions of rats treated withbenzo(a)pyrene(B(*a*)P).Thirty sixmale mature albino rats weighted 130-150g per each, were used in this study and divided two main groups, the first group(Group 1, 5 rats as a negative control group) still fed on basal diet and injected with the vehicle alone (5 ml/kg body weight), while the other main group (25 rats) was were challenged with an ip injection of B[a]P (100 mg/5 ml/kg body weight) dissolved in 0.9% NaCL solution containing 0.1% Tween 20 to induce liver impaired. The tested plant powder were given to the rats as a percent of 1,2,3 and 4% from the basel diet for 28 days . At the end of the experiment,Glotamic oxalic transaminase (AST),Glotamicpyrofic transaminase (ALT), alkaline phosphatase (ALP), GSH was determined by HPLC , Liver glycogen levels determined after digestion of liver and precipitation of glycogen by glycogen assay kit II,lipid peroxide levels measured as malondialdehyde in serum and liver were determined as thiobarbituric acid reactive substances (TBARS). According to the results,Gum Arabic (GA) could be used for improvement of liver functions and the best treatment was for group 6 (treated with 4% GA powder).

Key words: Gum Arabic,Benzo(a) Pyrene, GSH, Malonaldhyde,Liver functions andliver glycogen

Introduction

The liver is a critical organ in the human body. It performs an array of functions that help support metabolism, immunity, digestion, detoxification, vitamin storage among other functions. It comprises around 2% of an adult's body weight. The liver is a unique organ due to its dual blood supply from the portal vein (approximately 75%) and the hepatic artery (approximately 25%). It is intertwined with nearly every system in the body; hence, it is prone to a variety of pathologies ***(Karla and Tuma, 2018).*** Its primary function is to control the flow and safety of substances absorbed from the digestive system before distribution of these substances to the systemic circulatory system. A total loss of liver function could leads to death within minutes, demonstrating the liver‘s great importance ***(Ozougwu , 2017).*** The liver is necessary for survival; there is currently no way to compensate for the absence of liver function long term, although liver dialysis can be used short time.It has been known that cooking can produce toxic compounds in foods, if the appropriate precursors are present (**Elhassaneen and Tawfik, 1998)**. Among of these compounds, polycyclic aromatic hydrocarbons (PAH) from incomplete combustion occur in several foods such as charcoal broiled and smoked goods **(Emerole*et al.*, 1982; Larsson *et al.*, 1983 andBassiouny, 1999).**Benzo(*a*)pyrene [B(*a*)P] is a member of the family, polycyclic aromatic hydrocarbon (PAH) that is a by-product of incomplete combustion or burning of organic (carbon-containing) items, e.g., cigarettes, gasoline, and wood. B(*a*)P is commonly found with other PAHs in cigarette smoke, in grilled and broiled foods, and as a by-product of many industrial processes (**Elhassaneen, 2004**). Benzo (a) Pyrene is also found in ambient (outdoor) air, indoor air, and in some water sources **(U.S. Environmental Protection Agency, 2005**). Many of PAH compounds including B(*a*)P have been shown to be toxic, mutagenic and/or carcinogenic by extensive experiments *in vivo* (**Harvey, 1985 and Hawkins *et al.*, 1990)** and in vitro (**Elhassaneen, 1996 andElhassaneen*et al.*, 1997**) systems. Also, B(*a*)P exposure is associated with the development of liver cancer in mammals, rodent and fish **(Harvey, 1985 and Hawkins *et al.*, 1988 andElhassaneen, 1996).** It is known that the toxic, tumorigenic and carcinogenic effects of B[*a*]P correlate with the cellular metabolism of this compounds to arene oxides, phenols, quinones, dihydrodiols, and epoxides and with their subsequent formation of recative

intermediates that interact covalently with DNA to form adducts **(Harvey, 1985 andElhassaneen, 1996).**The Fixation of a biochemical changes by cell proliferation is considered the next step. The mutagenicity of BP is dependent upon metabolic activation. So, BP is considered a promutagen. The modern pharmacological therapy is costly and associated with multiple side effects resulting in patient non-compliance. Thus there is a need to explore alternative therapies particularly from natural sources as these are cost effective and possess minimal side effects. In this attention, Gum Arabic (GA) or Acacia Gum is an edible biopolymer obtained as exudates of mature trees of *Acacia Senegal* and *Acacia Seyal* which grow principally in the African region of Sahe in Sudan. Structurally, GA is a neutral or slightly acidic salt of a complex polysaccharide composed of galactose, arabinose, rhamnose, glucuronic acid, 4-*O*-methylglucuronic acid, calcium, magnesium, and potassium **(Anderson and Dea, 1971 andHegazy, 2009**). The chemical composition of GA is complex and consists of a group of macromolecules characterized by a high proportion of carbohydrates (~ 97%), which are predominantly composed of *D*-galactose and *L*-arabinose units and a low proportion of proteins (<3%) **(Mohamed, 2013).**

The effective biological role of GA has confirmed in the last twenty years including reduction in plasma cholesterol level in animals and humans**,**anticarcinogenic effect and antioxidant effect **(Ali *et al.*, 2003; Trommer and Neubert, 2005 and Ali and AlMoundhri, 2006)** with a protective role against hepatic and cardiac toxicities. In addition, it has been claimed that GA alleviates effects of chronic renal failure in humans **(Ali *et al.*, 2008; Glover *et al.*, 2009 and Ali *et al.*, 2010).** Also, GA is indigestible to both humans and animals, not degraded in the intestine, but fermented in the colon to give short-chain fatty acids, leading to a large range of possible health benefits, one of these benefits is its prebiotic effect which reported by**(Phillips and Philips, 2011)**that four week supplementation with Gum Arabic (10 g/day) led to significant increases in Bifidobacteria, Lactobacteria, and Bacteriodes indicating a prebiotic effect. Several epidemiological studies suggest that a high intake of dietary fiber, including GA (dietary fiber > 80%), is associated with beneficial effects on fat metabolism **(Ali *et al.*, 2009)**. For all of these reasons, GA has been widely used around the world in folk medicine. It has been reported to be used internally for the treatment of inflammation

of the intestinal mucosa, and externally to cover inflamed surfaces **(GamalEl-din *et al.*, 2003).** Despite the fact that GA is widely used as a vehicle for drugs in experimental physiological and pharmacological experiments, and is assumed to be an ‘‘inert” substance, some recent reports have claimed that GA possesses anti-oxidant, nephroprotectant and other effects **(GamalEl-din *et al.*, 2003 and Ali *et al.*, 2008).** Clinically, it has been tried in patients with chronic renal failure, and it was claimed that it helps reduce urea and creatinine plasma concentrations and reduces the need for dialysis from 2 to 3 times per week **(Sulimane*t al.*, 2011).** According to our knowledge, the studies regarding the potential effects of GA on liver disease/cancer are so limited. Therefore, in this study, we examined the influence of powders of GA in the liver cancer of rats induced by benzo(*a*)pyrene.

Materials and methods

Plant materials: Gum Arabic (GA) was purchased from El-Ghomhorya Company for Drug, Chemical and Medical Equipments, Cairo, Egypt.Casein was obtained from Morgan Chemical Co., Cairo, Egypt. All organic solvents and other chemicals were of analytical grade were purchased from El-Ghomhorya for Drug and Chemical Trading Co. (Cairo, Egypt).

Rats and diets: Male albino rats weighing 130-150g per each were obtained from Helwan Station, Ministry of Health and Population, Helwan, Cairo, Egypt.

Chemicals: The basic diet prepared according to thefollowing formula as mentioned by **(AIN, 1993)** as follow: protein (10%), corn oil (10%), vitamin 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 salt mixture used was formulated according to **(Hegsted, 1941)**.

Experimental Design:

All biological experiments performed a complied with the rulings of the Institute of Laboratory Animal Resources, Commission on life Sciences, National Research Council. Rats (n=50 rats), 130-150g per each, were housed individually in wire cages in a room maintained at 25 ± 2^{O}C, relative humidity (55±5%), a 12-hr lighting cycle 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 rats were divided into two main groups, the first group (Group 1, 5 rats, as a negative control group) still fed on basal diet and injected with the vehicle alone (5 ml/kg body weight) and the other main group (25 rats) were challenged with an ip injection of B[a]P (100 mg/5 ml/kg body weight) dissolved in 0.9% NaCL solution containing 0.1% Tween 20 to induce liver impaired rats then classified into eight sub- groups as follow:

- Group (2): fed on standard diet only as a positive control
- Group (3): B(a)P injection then feed on standard diet containing 1% GA powder.
- Group (4): B(a)P injection then feed on standard diet containing 2% GA powder.
- Group (5): B(a)P injection then feed on standard diet containing 3% GA powder.
- Group (6): B(a)P injection then feed on standard diet containing 4% GA powder.

At the end of experiment period, 4 weeks, blood samples were collected after 12 hours fasting using the abdominal aorta and rats were scarified under ether anesthetized. Blood samples were received into clean dry centrifuge tubes and left to clot at room temperature, then centrifuged for 10 minutes at 3000 rpm to separate the serum according to **Drury and Wallington, (1980)**. Serum was carefully aspirate, transferred into clean covet tubes and stored frozen at -20oC until analysis.

Liver functions assay:

Glotamic oxalic transaminase (GOT), Glotamicpyrofic transaminase (GPT) and alkaline phosphatase (ALP) were determined according to the methods described by**Yound, (1975) , Tietz, (1976) and Belfield and Goldberg,(1971)** respectively.GSH was determined by HPLC according to the method of **McFarris and Reed, (1987).**Liver glycogen levels were determined after digestion of liver and precipitation of glycogen by Glycogen Assay Kit II **(Colorimetric, abcam kits Co., ab169558, www.abcam.com).**Lipid peroxide levels measured as malondialdehyde in serum and liver were determined by as thiobarbituric acid reactive substances (TBARS) as described by **Buege and Aust, (1978).**

Statistical Analysis:

All measurements were done in triplicate and recorded as mean±SD. Statistical analysis was performed with the Student t-test and MINITAB 12 computer program **(Minitab Inc., State College, PA).**

Results and Discussion

Effects of GA in liver functions of rats treated with B(*a*)P

Liver functions of rats injected B[a]P and consumed GA powders were shown in Table (1). From such data it could be noticed that treatment of animals with B[a]P caused a significant increased ($p \leq 0.05$) in AST (110.38%), ALT (67.85%) and ALP (116.47%) compared to negative controls. Supplementation of diets with GA powders (1 to 4 g/100g) prevented the rise of mean serum AST, ALT and ALP activities. The rate of preventative was increased with the increasing of the GA powder concentration. The percent of change in the liver enzymatic activities were recorded 47.56, 29.01, 15.40 and 13.34% (For ALT); 64.55, 47.14, 26.86 and 21.36% (for AST) and 73.23, 42.41, 35.12 and 19.69% (for ALP) with the rat diets supplemented by 1, 2, 3 and 4 g/100g of GA powder, respectively.

B[a]P is one of the most commonly used hepatotoxins in the experimental study of liver diseases. The hepatotoxic effects of B[a]P are largely due to its active metabolites, oxides, hydroxyls, polyhydroxyls and quinones radicals **(Elhassaneen, 1996)**. These activated radicals bind covalently to the macromolecules and induce peroxidative degradation of membrane lipids of cell wall membrane, mitochondria, lysosomes and endoplasmic reticulum rich in polyunsaturated fatty acids **(Elhassaneen, 1996 and Elhassaneen*et al.*,1997).** This leads to the formation of lipid peroxides. This lipid peroxidative degradation of biomembranes is one of the principle causes of hepatotoxicity of B[a]P (**Elhassaneen, 2004**). This is evidenced by an elevation in the serum marker enzymes namely AST, ALT, ALP and decrease in protein.In the assessment of liver damage by B[a]P the determination of enzyme levels such as AST, ALT and ALP is largely used. Necrosis or membrane damage releases the enzyme into circulation and hence it can be measured in the serum. High levels of AST indicates liver damage, such as that caused by viral hepatitis as well as cardiac infarction and muscle injury, AST catalyses the conversion of alanine to pyruvate and glutamate and is released in a similar manner. Therefore ALT is more specific to the liver, and is thus a better parameter

for detecting liver injury. Elevated levels of serum enzymes are indicative of cellular leakage and loss of functional integrity of cell membrane in liver **(Drotman and Lawhan, 1978).** Serum ALP, bilirubin and total protein levels on other hand are related to the function of hepatic cell. Increase in serum level of ALP is due to increased synthesis, in presence of increasing biliary pressure **(Muriel and Garcipiana, 1992).**

The present data reported that feeding of rats suffering from liver disorders with GA induced significant ($p \leq 0.05$) improving in the liver functions. In this concern, **GamalEl-din *et al*., (2003)** studied that protective effect of GA against acetaminophen-induced hepatotoxicity in mice. Mice were given Arabic gum orally (1000 mg/100g body weight) for 5 days before a hepatotoxic dose of acetaminophen (500 mg/kg) intraperitoneally. GA administration dramatically reduced acetaminophen-induced hepatotoxicity as evidenced by reduced serum ALT and AST activities. And conclusion, Arabic gum is effective in protecting mice against acetaminophen-induced hepatotoxicity. This protection may involve the reduction of oxidative stress. Also, **Mochida *et al.*, (1996)** studied the effect of GA on macrophage, which play an important role in the regulation of immunological process in rats, activation by their ability to produce superoxide anions in vitro, and found that GA suppresses macrophage activation in vitro. This confirms an earlier report that GA is capable of almost completely blocking the macrophage function **(Mochida *et al*., 1990; Fujiwara *et al*., 1995)**. The authors inferred that such effects of GA would merit consideration in the therapy for chronic liver disease, as deranged function of Kupffer cells and hepatic macrophages occurs in this disease and is involved in its complications, such as endotoxemia. Finally, many authors found that pre-treatment with some phytochemicals such as found in GA were able to reduce the damage of liver i.e. suppress the elevation of AST and ALT through the improvement of antioxidant defense system in red blood cells **(Beattic*et al*., 2005).**

Table 1. Effects of GA in liver functions of rats treated with B(a)P

Value	Control (-)	Control (+)	GA (%, w/w)			
			1	2	3	4
Serum alanine aminotransferase (ALT,U/L)						
Mean	**73.72**	**123.74**	**108.79**	**95.11**	**85.08**	**83.56**
SD	**5.68**	**9.35**	**10.34**	**8.80**	**9.77**	**8.82**
% of Change	**0.00**	**67.85**	**47.56**	**29.01**	**15.40**	**13.34**
Serum aspartate aminotransferase (AST,U/L)						
Mean	**41.84**	**88.03**	**68.85**	**61.57**	**53.08**	**50.78**
SD	**4.55**	**8.81**	**9.37**	**9.84**	**5.46**	**7.81**
% of Change	**0.00**	**110.38**	**64.55**	**47.14**	**26.86**	**21.36**
Serum alkaline phosphatase (ALP,U/L)						
Mean	**138.51**	**299.83**	**239.94**	**197.25**	**187.15**	**165.78**
SD	**12.23**	**20.36**	**13.36**	**14.23**	**12.48**	**14.18**
% of Change	**0.00**	**116.47**	**73.23**	**42.41**	**35.12**	**19.69**

* Means in the same row with different letters are significantly different at ($p \leq 0.05$) **B(a)P** =benzo(a)pyrene

Effects of GA on Glycogen concentration (mg/g tissue) levels of rats treated with B(*a*)P

Liver glycogen content of rats injected with B[*a*]P and consumed GA were shown in Table (2). From such data it could be noticed that the liver glycogen content was decreased 86.64% by B[*a*]P, and this decrease was significantly elevated in the B[*a*]P+GApowder. The rate of glycogen elevation was increased with the increasing of the GA powder concentration. The percent of change increasing in glycogen was recorded 79.71, 69.21, 56.82 and 40.86%with the rat diets supplemented by 1, 2, 3 and 4 g/100g of GA powder, respectively. In similar study carried out by **Hasegawa *et al.*, (1995)** found that previous drinking of phytochemicals content of GA clearly protected against the changes in liver glycogen content. Such data with the others **(Hasegawa *et al.*, 1995)** suggested that secretion of lipoprotein from liver to blood might be blocked because of

intracellular structural failure and/or because of the energy depletion suggested by the marked decrease in glycogen content.

Table (2): Effects of GA on Glycogen concentration (mg/g tissue) levels of rats treated with B(*a*)P

Value	Control (-)	Control (+)	GA (%, w/w)			
			1	2	3	4
Mean	13.91	1.86	2.82	4.28	6.01	8.23
SD	1.06	0.34	0.47	0.69	1.19	1.60
% of Change	0.00	-86.64	-79.71	-69.21	-56.82	-40.86

* Means in the same row with different letters are significantly different at p≤0.05.) **B(a)P** =benzo(a)pyrene.

Effects of GA in biological antioxidants (serum glutathione fractions) levels of rats treated with B(*a*)P

Data presented in Table (3) showed effect of feeding GApowder on liver glutathione content of rats treated with B[*a*]P. It was observed that the mean value of GSH, GSSG and GSH/GSSG for control (-) group were 38.35, 19.30 and 23.61%. On the other hand feeding with GA powder, lead to significantly (P ≤0.05) increased in GSH and GSSG concentrations and GSH/GSSG ratio in plasma by 31.09, 26.00, 18.91 and 11.32%; 21.51, 19.17, 13.84 and 8.79%; and 12.21, 8.45, 5.88 and 2.77% compared to normal controls, respectively.

Reduced glutathion (GSH) is a tripeptide-thiol (γ-glutamylcysteinyl-glycine) that has received considerable attention in terms of its biosynthesis, regulation, and various intracellular functions (Reed and Beatty, 1980; Larsson et al., 1983). Among these function are two constructing roles in detoxifications: (1) as a key conjugate of electrophilic intermediates, principally via glutathione-s-transferase activities in phase II metabolism, and (2) as an important antioxidant. The antioxidant functions of GSH includes its role in the activities of GSH enzymes family including glutathione peroxidase (GSH-Px) and peroxiredoxins (PRXs). In addition, GSH can apparently serve as a nonenzymatic scavenger of oxyradicals**(Halliwell and Gutteridge, 1985)**. A fall in glutathione fractions observed in obese rats group generally accompanied by a concomitant decreased in the ratio of GSH/GSSG. **Di Giulio (1991)** mentioned that a more fundamental effect of oxyradical-generating compounds as the obesity development, however, is their effect on what can be referred to as the redox status (GSH/GSSG) of cells or tissues. Few

studies have been addressed directly the issue of effects of pro-oxidants on redox status. **Elhassaneen*et al*., (2004)** mentioned that increased fluxes of oxyradicals might be decreased in the GSH/GSSG ratio, due either to direct radical scavenging or to increased peroxidase activity. This effect could also occur indirectly due to reduced NADPH availability [necessary for glutathione reductase (GSH-Rd) activity] resulting, for example, from oxidations in the first step of the redox cycle **(Champe and Harvey, 1994 and Bedard, and Krause, 2007)**. In this context, **Bedard and Krause (2007)** reported that various enzymes inside the cells including adipocytes can also produce ROS. Particularly, the family of NAPDH oxidases (NOX) is considered to be an important source of ROS generation. Such effect could be one of the most important reasons for reducing the GSH/GSSG ratio in obese rats. The GA feeding is rich in bioactive compounds which exhibited antioxidant effects against ROS formation as the obesity development through several mechanism of action including the raising of redox status (GSH/GSSG ratio) in the body.

Table 3. Effects of GA in biological antioxidants (serum glutathione fractions) levels of rats treated with B(*a*)P

Value	Control (-)	Control (+)	GA (%, w/w)			
			1	2	3	4
Reduced glutathione concentration (GSH, µmol/L)						
Mean	**10.90**	**6.72**	**7.51**	**8.07**	**8.84**	**9.67**
SD	**1.29**	**1.15**	**1.28**	**0.90**	**1.18**	**1.99**
% of Change	**0.00**	**-38.35**	**-31.09**	**-26.00**	**-18.91**	**-11.32**
Oxidized glutathione concentration (GSSG, µmol/L)						
Mean	**0.99**	**0.80**	**0.78**	**0.80**	**0.86**	**0.91**
SD	**0.25**	**0.25**	**0.35**	**0.21**	**0.16**	**0.22**
% of Change	**0.00**	**-19.30**	**-21.51**	**-19.17**	**-13.84**	**-8.79**
GSH/GSSG ratio						
Mean	**10.96**	**8.37**	**9.62**	**10.03**	**10.31**	**10.65**
SD	**1.21**	**1.21**	**0.98**	**1.77**	**1.11**	**1.07**
% of Change	**0.00**	**-23.61**	**-12.21**	**-8.45**	**-5.88**	**-2.77**

* Means in the same row with different letters are significantly different at $p \leq 0.05$). **B(a)P** =benzo(a)pyrene.

Effects of GA on serum and liver tissue biological oxidants levels of rats treated with B(*a*)P

Liver lipid peroxidation of rats injected with B[*a*]P and consumed GA powder were shown in **Table (4a,b)**. From such data it could be noticed that the liver lipid peroxidation (MDA and NO_2) level was increased 39.43 and 23.55% (in serum) and 249.43 and 186.14% (in liver) by B[*a*]P, and this increase was significantly reduced in the B[*a*]P+ GApowder. The rate of MDA and NO_2 reducing was increased with the increasing of the GA powder concentration. The percent of change in MDA and NO_2 were recorded 33.79, 32.25, 26.36 and 24.53%; 20.65, 17.75, 15.22 and 9.06% (In serum); and 201.53, 102.40, 86.00 and 47.43; and 111.39, 67.57, 50.00 and 41.83% (in liver) with the rat diets supplemented by 1, 2, 3 and 4 g/100g of GA powder, respectively.

Several reports have documented the potent antioxidant capacity of GA where by mitigation of lipid peroxidation and oxidative stress in several tissues were demonstrated **(Nabavi*et al*., 2012b)**. Also, **Hasegawa *etal*., (1995)** found that previous drinking of phytochemicals such found in GA clearly protected against the changes in serum and liver lipid peroxide levels. On the other hand, serum lipid peroxide levels dropped in all B[a]P-treated animals. Such data with the others **(Hasegawa *et al*., 1995)** suggested that secretion of lipoprotein from liver to blood might be blocked because of intracellular structural failure and/or because of the energy depletion suggested by the marked decrease in glycogen content. The increases of serum enzyme activities were clearly inhibited by turmeric and partly by curcumin at this time.

Conclusion

The**GA** in the present study were effective in protecting rats treated with B(a)P. These results supported our hypothesis that tested **GA** contain several important compounds such as fibers, minerals, polyphenols, flavonoids and carotenoids which are able to protecting rats treated with B(a)P. Therefore, data recommended**GA** by a moderate amount to be included in our daily diets.

Table (4a): Effects of GA on serum biological oxidants levels of rats treated with B(*a*)P

Value	Control (-)	Control (+)	GA (%, w/w)			
			1	2	3	4
Malondialdehyde (MDA) concentration (nmol/mL)						
Mean	0.182	0.110	0.120	0.123	0.134	0.137
SD	0.027	0.014	0.058	0.065	0.047	0.049
% of Change	0.00	-39.43	-33.79	-32.25	-26.36	-24.53
Nitrite (NO_2, nmol/L)						
Mean	2.99	2.29	2.38	2.46	2.54	2.72
SD	0.59	0.35	0.47	0.24	0.53	0.68
% of Change	0.00	-23.55	-20.65	-17.75	-15.22	-9.06

* Means in the same row with different letters are significantly different at ($p \leq 0.05$). **B(a)P** =benzo(a)pyrene

Table (4b): Effects of GA on liver biological oxidants levels of rats treated with B(*a*)P

Value	Control (-)	Control (+)	GA (%, w/w)			
			1	2	3	4
Malondialdehyde (MDA) concentration (nmol/mg tissue protein)						
Mean	3.80	13.29	11.47	7.70	7.07	5.61
SD	0.35	1.21	1.13	1.03	0.90	1.01
% of Change	0.00	249.43	201.53	102.40	86.00	47.43
Nitrite (NO2, nmol/g tissue protein)						
Mean	4.26	12.19	9.01	7.14	6.39	6.04
SD	0.66	2.22	1.52	1.36	1.04	1.25
% of Change	0.00	186.14	111.39	67.57	50.00	41.83

* Means in the same row with different letters are significantly different at ($p \leq 0.05$) **B(a)P** =benzo(a)pyrene

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إستخدام الصمغ العربي لتحسين المضاعفات الناتجة عن أمراض الكبد في الفئران

شريف صبري رجب مكاوي[1]، عبير نزيه عبد الرحمن[2]، هدى عاطف عبد العزيز[3]
قسم التغذية وعلوم الأطعمة ،كلية الاقتصاد المنزلي ،جامعة المنوفية ، شبين الكوم،مصر، [1،2].
بكالوريوس الاقتصاد المنزلي- كلية الاقتصاد المنزلي – جامعة الأزهر- نواج[3]

الملخص العربى:

تم إجراء الدراسة الحالية لمعرفة تأثير استخدام الصمغ العربي لتحسين المضاعفات الناتجة عن أمراض الكبد في الفئران المصابة بالفابنزوبيرين. تم استخدام 36فأر أبيض بالغ يتراوح وزن كل منهما علي130-150 جم وتم تقسيمهم إلى مجموعتين رئيسيتين إحداهما كمجموعة ضابطة سالبة أما المجموعة الأخرى فتم إحداث تسمم بالكبد فيها باستخدام الفابنزوبيرين (100 mg/5 ml/kg body weight)المذاب في محلول NaCLبنسبه9.% يحتوي علي (20 Tween %.1)للحث علي اصابه الكبد. وأضيف النبات المستخدم بالنسب الاتيه1، 3,2، 4% من الوجبة الأساسية على هيئة مطحون منالصمغ العربي لمدة 28 يوم. وتم قياس إنزيمات الكبد (الجلوتاميك أوكسالوأسيتكترانسأمينيز، الجلوتاميكبيرو فيك تر انس أمينيز و الألكالين فوسفاتيز) و الجلوتاثيون ، ثنائي ألدهيد المالون ، جليكوجين الكبد. وقد أظهرت نتائج هذه الدراسة أن تناول نبات الصمغ العربي نتج عنه تحسن في وظائف الكبد.وكانت أفضل المجموعات التي تغذت على الوجبة الاساسيه مضافا إليها 4% من بودر الصمغ العربي.

الكلمات المفتاحية:الصمغ العربي- وظائف الكبد- الجلوتاثيون - ثنائي ألدهيد المالون - جليكوجين الكبد.