

## Therapeutic value of frankincense and myrrh In liver recovery after exposure to aflatoxin B<sub>1</sub>

Taha A. Kumosani\*, Jehad M. Yousif\*\* and Omayma A. Abou Zeid\*\*

\*Biochemistry Department, Faculty of Science, King Abdulaziz University;

\*\*Chemistry Department, Faculty of Science, Girl's Collage of Education,  
Jeddah, Saudi Arabia

### ABSTRACT

Frankincense, (*Gum Olibanum*), and Myrrh, (*Commiphora merrha*), are of plant resins produce by the *Burseraceae* family, growing in Somali, India and Yemen. They were known for thousands of years as one of hoarding in the east. In order to study the therapeutic value of such resins on liver recovery after exposure to aflatoxin B<sub>1</sub>, it was administrated intra- peritoneal to male Wister Albino rats for 10 days, after which Frankincense and Myrrh, (each one alone), were given in the form of water extract to rats for 20 days. At the end of the study blood from all experimental animals was analyzed for some biochemical parameters including glucose, triglycerides, cholesterol, urea, uric acid, creatinine, bilirubin, hemoglobin and some key liver enzymes as asparate amino transferase (AST), alanine amino transferase (ALT), gamma- glutamyl transferase (GGT). Liver tissue samples were analysed for their content of total proteins, deoxyribonucleic acid (DNA), ribonucleic acid (RNA) and in addition to histopathological examination. This study demonstrated that Frankincense and Myrrh are of certain therapeutic recovery value in liver after exposure to AFB<sub>1</sub>.

### INTRODUCTION

Today, one of the most urgent problems of public health is the development of effective methods to block the environmental carcinogenesis sequential events. Liver cancer (Primary hepatocellular carcinoma), is a major public health hazard in the developing countries of Africa and Asia. The etiology of this disease implicates both infection with hepatitis B and C and exposure to aflatoxin B<sub>1</sub> (AFB<sub>1</sub>), as a food contaminant, (Montalto *et al.*, 2002)<sup>1</sup>.

Chemoprevention is a concept defined as prevention of cancer by the

administration of natural or synthetic pure chemicals, or through daily foods rich in cancer preventive components. Several compounds have been discovered with inhibitory effects on the tumor-promoting stage, interestingly many of them were derived from plants, (Vimala *et al.*, 1999 and Borrelli & Izzo, 2000)<sup>2,3</sup>. However, primary cancer prevention has two aspects in its methodology; exclusion or avoidance of the environmental carcinogens and other chemical factors closely related to carcinogenesis such as tumor promoters; and the administration of inhibitory or suppressive agents

against carcinogenesis (Elegbede *et al.*, 2002)<sup>4</sup>.

Both Frankincense, (*Gum Olibanum*) and Myrrh, (*Commiphora merrha*), of the botanical family *Burseraceae* are resins from small trees or shrubs. Their natural abundance is limited, but this has been overcome by systematic regular cultivation to meet world wide demands. Today, most Frankincense and Myrrh are produced in the Southern Arabian Peninsula, (Oman and Yemen) and in southeast Africa, (Somalia). When referring to this pair of herbs, western people might immediately think of their historic importance in religion, (Hostanska *et al.*, 2002)<sup>5</sup>.

The resins obtained from different plants have long been known in the traditional medicine of different countries. The main components of Frankincense are alpha-pinene, boswellic acid together with many other compounds, (Hostanska *et al.*, 2002 and Shi *et al.*, 2002)<sup>5,6</sup>. As a treatment it was used as anti-inflammatory, anti-proliferative toward a variety of malignant cells and in the treatment of non-insulin dependent diabetes mellitus, (Al-wadi *et al.*, 1991, Liu *et al.*, 2002 and Park *et al.*, 2002)<sup>7,8,9</sup>.

The chemical composition of resin Myrrh which is obtained from the stem of different commiphora species is sesquiterpenoids, volatile oils and many active component, (Zhu *et al.*, 2001 and El Ashry *et al.*, 2003)<sup>10,11</sup>. It is highly reputed and commonly used in Arab medicine as anti-inflammatory, anti-ulcer, anti-thrombotic, anti-pyretic, anti-septic, for lowering serum cholesterol and

triglycerides, inhibiting cholera toxin, anti-microbial and fascioliasis. When medicinally tested in a variety of diseases it caused a decrease in the contents of nucleic acids and proteins, (Tariq *et al.*, 1986; Michie & Cooper, 1991, Al-Harbi *et al.*, 1997, Olajide, 1999 and Massoud *et al.*, 2000)<sup>12-16</sup>. The aim of this research is to study the therapeutic value of Frankincense and Myrrh extracts in the recovery of liver after exposure to aflatoxin B<sub>1</sub>.

## MATERIAL & METHODS

### Animals

In this study, 84 Male Wister Albino Rats, weighing 70-100 gm were kept on commercial laboratory standard diet. The rats were divided into 4 groups of 21 rats each. With the exception of the normal control group, all groups were injected i.p with 20 µl of 0.1ml/ 100 gm body weight aflatoxin B<sub>1</sub> solution, once a day for 10 days, (Qin *et al.*, 1997)<sup>17</sup>. After 10 days of aflatoxin B<sub>1</sub> injection one group was left without treatment and served as AFB<sub>1</sub> non-treated group. The other two groups namely; the Frankincense treated and the Myrrh treated groups were allowed to drink the respective resin extracts ad-libitum.

### Methods

Resins extracts were prepared by stirring of 20 gm of the resin in 400 ml distilled water for 60 min at 80°C, after which the extract was cooled to room temperature, filtered and administered to the animals in drinking bottles, (Zhou *et al.* 2000)<sup>18</sup>.

On day 20 of treatment, blood samples were withdrawn from all animals for the determination of

serum enzymes; aspartate aminotransferase, AST, (Saris, 1978)<sup>19</sup>, alanine aminotransferase, ALT, (Bergmeyer *et al.*, 1978)<sup>20</sup> and gamma glutamyl aminotransferase, GGT, (Shaw *et al.*, 1983)<sup>21</sup>. Other analysis included: bilirubin, (Jendrassik & Grof, 1938)<sup>22</sup>, urea, (Talke & Schubert, 1965)<sup>23</sup>, uric acid, (Bulgar & Johns, 1941)<sup>24</sup>, creatinine, (Larsen, 1972)<sup>25</sup>, cholesterol, (Stadtman, 1957)<sup>26</sup>, triacylglycerols, (Rautela *et al.*, 1974)<sup>27</sup>, glucose, (Henry, 1974)<sup>28</sup>, hemoglobin, (Van Assendelf, 1970)<sup>29</sup>. Liver tissue samples from each group were obtained for the determination of RNA, DNA, (Bregman, 1983)<sup>30</sup> and total protein, (Lowry *et al.*, 1951)<sup>31</sup>. Histopathological examination was performed according to Bancroft & Steven, 1996<sup>32</sup>.

#### Statistical analysis

Data were statistically analyzed using the student t-test with SPSS program version 13.

## RESULTS

Results of biochemical analysis are shown in table (1), while those of liver tissue analysis are shown in table (2), followed by the results of histological examination of liver tissue.

It is clear from table (1) that there have been a statistically very highly significant increase in serum GGT, bilirubin and glucose, ( $p < 0.0001$ ) in the AFB<sub>1</sub> non-treated group, accompanied by a statistically very highly significant decrease in serum cholesterol and triglycerides, ( $p < 0.0001$ ), as compared to the

normal control group, (t-1) with statistically non-significant changes in all other measured parameters.

With Frankincense, a notable statistically significant decrease in serum bilirubin and an increase of cholesterol to the normal levels are found. Serum uric acid, however, appears on increase, ( $p < 0.05$ ) over its normal level, while blood hemoglobin continues to decrease to reach a statistically significant, ( $p < 0.005$ ) lower value. This picture changed a little on treatment with Myrrh, as serum AST and urea appear to be statistically increased, ( $p < 0.05$ ). The same applies for serum GGT, with the return of serum uric acid and glucose to their normal values.

On comparison with AFB<sub>1</sub> non-treated group, (t-2), treatment with Frankincense statistically decreased serum bilirubin and blood hemoglobin significantly, ( $p < 0.05$ ) and serum triglycerides very highly significantly, ( $p < 0.0001$ ) with a notable increase in serum cholesterol, ( $p < 0.0001$ ).

With Myrrh treatment, serum AST remains higher, ( $p < 0.01$ ) with statistically significantly lower serum bilirubin, ( $p < 0.001$ ), urea, ( $p < 0.05$ ), glucose, ( $p < 0.0005$ ) and blood hemoglobin, ( $p < 0.05$ ).

On statistical comparison of treatment with Frankincense and Myrrh, (t-3) non-significant differences were found in serum AST, ALT, bilirubin, creatinine and blood hemoglobin, while serum urea, uric acid, cholesterol and glucose were statistically lower and triglycerides higher in the Myrrh treated over the Frankincense treated group, ( $p < 0.05 - p < 0.0001$ ).

Table (1): Mean  $\pm$  SEM and t-test\* of Biochemical Parameters.

Group Parameter	Normal Control Group	AFB <sub>1</sub> Non-treated Group	AFB <sub>1</sub> Frankincense Treated Group	AFB <sub>1</sub> Myrrh Treated Group
AST (U/L) t-1 t-2 t-3	259.9 $\pm$ 41.5	242.3 $\pm$ 43.7 N.S.	227.3 $\pm$ 142.2 N.S. N.S.	425 $\pm$ 56.8 p < 0.05 p < 0.01 N.S.
ALT (U/L) t-1 t-2 t-3	88.8 $\pm$ 20.2	66.5 $\pm$ 11.1 N.S.	120 $\pm$ 49.2 N.S. N.S.	79.3 $\pm$ 11.6 N.S. N.S. N.S.
GGT (U/L) t-1 t-2 t-3	10 $\pm$ 0.76	13.6 $\pm$ 0.34 p < 0.0001	15 $\pm$ 5.8 N.S. N.S.	14.2 $\pm$ 0.6 p < 0.0001 N.S. N.S.
Bilirubin (mg/dl) t-1 t-2 t-3	0.17 $\pm$ 0.2	1.26 $\pm$ 0.15 p < 0.0001	0.25 $\pm$ 0.46 N.S. p < 0.05	0.41 $\pm$ 0.19 N.S. p < 0.001 N.S.
Urea (mg/dl) t-1 t-2 t-3	26.3 $\pm$ 1.03	24.9 $\pm$ 1.05 N.S.	27.6 $\pm$ 1.5 N.S. N.S.	21.5 $\pm$ 1.7 p < 0.05 p < 0.05 p < 0.01
Uric acid (mg/dl) t-1 t-2 t-3	2.2 $\pm$ 0.3	3.11 $\pm$ 0.5 N.S.	3.8 $\pm$ 0.63 p < 0.05 N.S.	2.1 $\pm$ 0.76 N.S. N.S. p < 0.05
Creatinine (mg/dl) t-1 t-2 t-3	0.3 $\pm$ 0.27	0.4 $\pm$ 0.24 N.S.	0.33 $\pm$ 0.42 N.S. N.S.	0.38 $\pm$ 0.2 N.S. N.S. N.S.
Cholesterol (mg/dl) t-1 t-2 t-3	115.5 $\pm$ 2.9	65.3 $\pm$ 2.5 p < 0.0001	121.3 $\pm$ 5.2 N.S. p < 0.0001	59.8 $\pm$ 0.9 p < 0.0001 N.S. p < 0.0001
Triglycerides (mg/dl) t-1 t-2 t-3	149.9 $\pm$ 6.2	66.4 $\pm$ 2.1 p < 0.0001	39.1 $\pm$ 5.2 p < 0.0001 p < 0.0001	74.2 $\pm$ 8.99 p < 0.0001 N.S. p < 0.001
Glucose (mg/dl) t-1 t-2 t-3	134.5 $\pm$ 4.6	168.5 $\pm$ 5.3 p < 0.0001	202 $\pm$ 14.6 p < 0.0001 N.S.	134.5 $\pm$ 10.3 N.S. p < 0.005 p < 0.0005
Hemoglobin (mmol/L) t-1 t-2 t-3	10.5 $\pm$ 0.7	9.4 $\pm$ 0.41 N.S.	8.2 $\pm$ 0.43 p < 0.005 p < 0.05	7.9 $\pm$ 0.77 p < 0.01 p < 0.05 N.S.

\*t-test: t-1 = v.s. Normal control group.

t-2 = v.s. AFB<sub>1</sub> non-treated group.t-3 = v.s. AFB<sub>1</sub> Frankincense treated group.

Table (2), shows the results of liver tissue analysis of the four experimental groups, viz. total proteins, RNA and DNA. It was found that AFB<sub>1</sub> injection had caused a statistically significant, (t-1) decrease in liver total protein content, ( $p < 0.05$ ) together with non-significant decrease of both RNA and DNA, as compared to the normal control group.

Treatment with Frankincense showed a non-significant increase in all measured parameters, while treatment with Myrrh showed statistically significant increase in total protein, ( $p < 0.005$ ) and DNA, ( $p$

$< 0.05$ ) in comparison to the normal control group. However, there has been a statistically non-significant increase, (t-2) in all measured parameters in the Frankincense treated group on comparison to the AFB<sub>1</sub> non-treated group, while only a highly significant increase in total protein was observed in the Myrrh treated group on the same comparison. Neither Frankincense nor Myrrh showed any statistically significant difference, (t-3) between each other in their effects on the concerned parameters.

**Table (2): Mean  $\pm$  SEM and t-test\* of liver Total Proteins, RNA, DNA, ratio of RNA/ DNA % and Ratio of RNA and DNA to Total Protein %.**

Group Parameter	Normal Control Group	AFB <sub>1</sub> Non-treated Group	AFB <sub>1</sub> Frankincense Treated Group	AFB <sub>1</sub> Myrrh Treated Group
Total protein (gm/100gm) t-1 t-2 t-3	6.84 $\pm$ 0.39	5.54 $\pm$ 0.85 $p < 0.05$	7.24 $\pm$ 1.77 N.S. N.S.	8.6 $\pm$ 0.38 $p < 0.005$ $p < 0.005$ N.S.
RNA (gm/100gm) t-1 t-2 t-3	2.06 $\pm$ 0.40	1.29 $\pm$ 0.52 N.S.	2.19 $\pm$ 0.14 N.S. N.S.	2.15 $\pm$ 0.18 N.S. N.S. N.S.
RNA/Total Protein %	30.12	23.29	30.25	25.00
DNA (gm/100gm) t-1 t-2 t-3	0.75 $\pm$ 0.34	0.47 $\pm$ 0.69 N.S.	1.44 $\pm$ 0.98 N.S. N.S.	1.55 $\pm$ 0.15 $p < 0.05$ N.S. N.S.
DNA/Total Protein %	10.96	8.43	19.89	18.02
RNA/DNA %	274.7	274.5	152.1	138.7

\*t-test: t-1 = v.s. Normal control group.

t-2 = v.s. AFB<sub>1</sub> non-treated group.

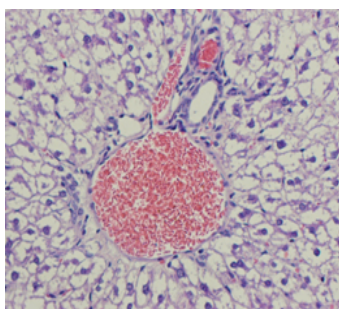
t-3 = v.s. AFB<sub>1</sub> Frankincens treated group.

RNA and DNA to total protein ratio % were found to decrease in the AFB<sub>1</sub> non-treated group. However, RNA/DNA ratio % remained

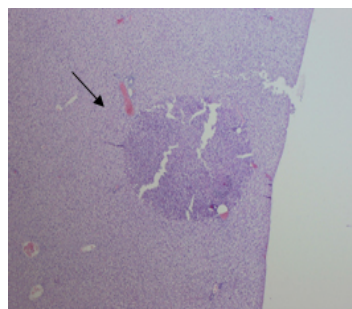
unchanged. Upon treatment with Frankincense RNA/total protein ratio % returned to its normal but DNA/total protein ratio % doubled its

normal value, while upon treatment with Myrrh RNA/total protein ratio did not differ much from AFB1 non-treated group and DNA/total protein ratio % remained at double its normal value. RNA/DNA ratio % decreased to almost one-half its normal value on either treatment.

Histological examination of the liver tissue of AFB<sub>1</sub> non-treated group showed liver cells without nucleus, degenerative and necrotic with decreased number of kupffer cells, and hemorrhage in the portal area, (Fig.1-a) and hepatoma focci, (Fig. 1-b, arrow).



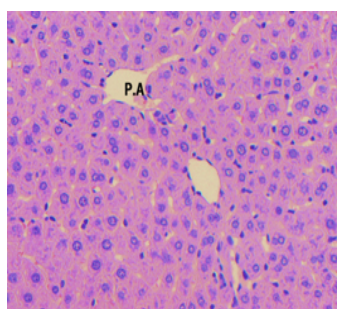
*Fig. (1-a)*



*Fig. (1-b)*

After treatment with Frankincense, liver sections showed an increase in number of kupffer cells and blood stasis in central and portal veins and swelling bile ducts, with

marked advance in histological composition in portal area (P.A) of hepatic cells (H.C) and central vein (C.V) (Figs. 2-a,b).



*Fig. (2-a)*



*Fig. (2-b)*

Treatment with Myrrh, showed dilatation and degenerative of blood vessels, still hepatic focci, (Fig.3-a,

arrow), necrotic hepatic cells, central veins and hemorrhage, (Fig. 3-b, arrow).

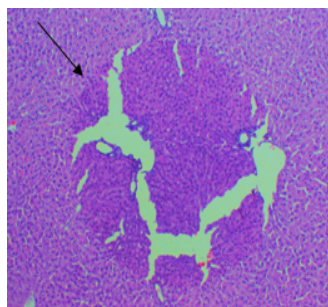


Fig. (3-a)

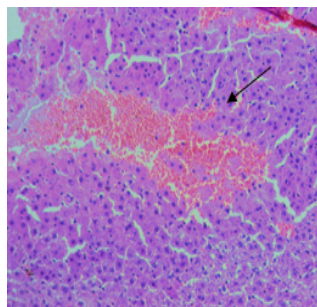


Fig. (3-b)

## DISCUSSION

Primary hepatocellular carcinoma is a major health hazard that might implicate exposure to aflatoxin B<sub>1</sub>, (AFB<sub>1</sub>), as a food contaminant, (Montalto *et al.*, 2002)<sup>1</sup>. Chemoprevention could be achieved with daily foods rich in cancer preventive components, (Vimala *et al.*, 1999 and Borrelli & Izzo, 2000)<sup>2,3</sup> and inhibitory or suppressive agents against carcinogenesis, (Elegbede *et al.*, 2002)<sup>4</sup>.

Both Frankincense, (*Gum Olibanum*) and Myrrh, (*Commiphora merrha*), of the family *Burseraceae* are plant resins used in Arab medicine. The main components of Frankincense are alpha-pinene and boswellic acid, (Hostanska *et al.*, 2002 and Shi *et al.*, 2002)<sup>5,6</sup>. It is used in the treatment of a variety of malignancies and in treatment of type II diabetes mellitus, (Al-wadi *et al.*, 1991, Liu *et al.*, 2002 and Park *et al.*, 2002)<sup>7,8,9</sup>. Myrrh is rich in sesquiterpenoids and volatile oils, (Zhu *et al.*, 2001 and El Ashry *et al.*, 2003)<sup>10,11</sup>. It is used as anti-inflammatory, anti-ulcer, anti-thrombotic, anti-pyretic, anti-septic, anti-hyperlipidemic. It decreases the

content of nucleic acids and proteins, (Tariq *et al.*, 1986; Michie & Cooper, 1991, Al-Harbi *et al.*, 1997, Olajide, 1999 and Massoud *et al.*, 2000)<sup>12-16</sup>. The aim of this research was to study the therapeutic value of Frankincense and Myrrh in the recovery of liver after exposure to aflatoxin B<sub>1</sub>.

It is clear from table (1) that there have been a statistically very highly significant increase in serum GGT, bilirubin and glucose, ( $p < 0.0001$ ) in the AFB<sub>1</sub> non-treated group, accompanied by a statistically very highly significant decrease in serum cholesterol and triglycerides, ( $p < 0.0001$ ), as compared to the normal control group, (t-1) with statistically non-significant changes in all other measured parameters. The increases in serum bilirubin after aflatoxin B<sub>1</sub> injection could be due to degeneration of RBCs. This is in contradiction with Guerre *et al.*, 1997<sup>33</sup> and Rastogi *et al.*, 2001<sup>34</sup>. The decrease in serum lipids and increase in glucose is considered as one of cancer recognition. This agrees with Burt *et al.*, 1981<sup>35</sup> who reported that glucose level was significantly higher due to increased gluconeogenesis. The decrease in triglycerides and cholesterol agrees with Singh and

Venkitasubramanian, 1975<sup>36</sup> and Ekman *et al.*, 1982<sup>37</sup>. The very highly significant increase in serum GGT is probably due to the degeneration of liver cells as shown by the histopathological examination.

Compared to the normal control group, (t-1) treatment with Frankincense showed a statistically significant decrease in serum bilirubin and an increase of cholesterol to the normal levels which might be due to the high content of boswellic acid in Frankincense with its anti-inflammatory and anti-cancer properties, (Liu *et al.*, 2002)<sup>8</sup>. Serum uric acid significantly increased, ( $p < 0.05$ ), with significant decreased blood hemoglobin, ( $p < 0.005$ ) lower value, indicating some amelioration in liver condition. This picture changed a little on treatment with Myrrh, as serum AST and urea appear to be statistically increased, ( $p < 0.05$ ). The same applies for serum GGT, with the return of serum uric acid and glucose to their normal values which may be due to the increased gluconeogenesis in liver cancer, (Al-wadi & Gumaa, 1987)<sup>38</sup>.

On comparison with AFB<sub>1</sub> non-treated group, (t-2), treatment with Frankincense statistically decreased serum bilirubin and blood hemoglobin significantly, ( $p < 0.05$ ) and serum triglycerides very highly significantly, ( $p < 0.0001$ ) with a notable increase in serum cholesterol, ( $p < 0.0001$ ), indicating slight amelioration in liver condition.

With Myrrh treatment, serum AST remains higher, ( $p < 0.01$ ) with statistically significantly lower serum bilirubin, ( $p < 0.001$ ) which might be due to the terpenes content in the Myrrh which is resistant to the

carcinogenesis, (Al-Harbi *et al.*, 1994)<sup>39</sup>. A decrease in urea, ( $p < 0.05$ ), glucose, ( $p < 0.0005$ ) and blood hemoglobin, ( $p < 0.05$ ) was also found.

On statistical comparison of treatment with Frankincense and Myrrh, (t-3) non-significant differences were found in serum AST, ALT, bilirubin, creatinine and blood hemoglobin, while serum urea, uric acid, cholesterol and glucose were statistically lower and triglycerides higher in the Myrrh treated over the Frankincense treated group, ( $p < 0.05 - p < 0.0001$ ).

Table (2), shows that AFB<sub>1</sub> injection had caused a statistically significant, (t-1) decrease in liver total protein content, ( $p < 0.05$ ) with non-significant decrease of both RNA and DNA, as compared to the normal control group. Raju & Devegowada, 2000<sup>40</sup> and Ekman *et al.*, 1982<sup>39</sup> attributed this to AFB<sub>1</sub> DNA adduct which may interrupt the transcription of RNA necessary for protein synthesis.

Treatment with Frankincense showed a non-significant increase in all measured parameters, while treatment with Myrrh showed statistically significant increase in total protein, ( $p < 0.005$ ) and DNA, ( $p < 0.05$ ) in comparison to the normal control group. Al-Harbi *et al.*, 1994<sup>37</sup> reported significant increases in RNA, total protein, with very highly significant increase in DNA of liver tissues on treatment with Myrrh.

RNA and DNA to total protein ratio % were found to decrease in the AFB<sub>1</sub> non-treated group, paralleling the decrease in total protein with constant RNA/DNA ratio %. Upon treatment with Frankincense



RNA/total protein ratio % returned to its normal, and so did the total protein, but DNA/total protein ratio % doubled its normal value, which might indicate an increased DNA replication, while upon treatment with Myrrh RNA/total protein ratio did not differ much from AFB<sub>1</sub> non-treated group and DNA/total protein ratio % remained at double its normal value. RNA/DNA ratio % decreased to almost one-half its normal value on either treatment which might indicate an increased DNA replication in relation to RNA transcription.

However, there has been a statistically non-significant increase, (t-2) in all measured parameters in the Frankincense treated group on comparison to the AFB<sub>1</sub> non-treated group, while only a highly significant increase in total protein was observed in the Myrrh treated group on the same comparison, which gives the Myrrh an advantage over Frankincense. Neither Frankincense nor Myrrh showed any statistically significant difference, (t-3) between each other in their effects on the concerned parameters.

Histopathological examination of liver sections of the AFB<sub>1</sub> non-treated group showed hepatocytic foci, necrotic liver cells and dilatation of blood vessels within central veins and hemorrhage and decrease in the number of kupffer cells. This agrees well with Al-Harbi *et al.*, 1997<sup>14</sup> and Baptista *et al.*, 2002<sup>41</sup>.

In conclusion this demonstrated that Frankincense and Myrrh can ameliorate the liver biochemical and histological condition after exposure to AFB<sub>1</sub>.

## REFERENCES

1. **Montalto, G., Cervello, M., Giannitrapani, L., Dantona, F., Terranova, A. And Castagnetta, L. A. (2002).** Epidemiology, risk factors, and natural history of hepatocellular carcinoma. *Ann. NY. Acad. Sci.*, 963:13-20.
2. **Vimala, S., Norhanom, A. W. and Yadav, M. (1999).** Antitumor promoter activity in Malaysian ginger rhizobia used in traditional medicine. *Br. J. Cancer.*, 80(1-2):110-6.
3. **Borrelli, F. and Izzo, A. A. (2000).** The plant kingdom as a source of antiulcer remedies. *Phytother. Res.*, 14(8):581-91.
4. **Elegbede, J. A. and Gould, M. N. (2002).** Monoterpene reduced adducts formation in rats exposed to aflatoxin. *Afr. J. Biotech.*, 1(2): 46-49.
5. **Hostanska, K., Daum, G. and Saller, R. (2002).** Cytostatic and apoptosis-inducing activity of boswellic acids toward malignant cell lines *in vitro*. *Anticancer. Res.*, 22(5):2853-62.
6. **Shi, S.M., Tian, J.G. and Wang, B.Q. (2002).** Study on the detecting methods of the imported medica-olibanum. *Zhongguo Zhong Yao Za Zhi*, 27(3):170-3.
7. **Al-Wadi, F., Fatania, H. and Shamte, U. (1991).** The effect of a plant mixture extract on liver gluconeogenesis in streptozotoin induced diabetic rats. *Diabetes. Res.*, 18(4):163-8.
8. **Liu, J. J., Nilsson, A., Oredsson, S., Badmaev, V., Zhao, W. Z.**

- and Duan, R. D. (2002). Boswellic acids trigger apoptosis via a pathway dependent on caspase- activation but independent on fas/fas ligand interaction in colon cancer H T-29 cells. *Carcinogenesis.*, 23(12):2087-93.
9. Park, Y. S., Lee, J. H., Bondar, J., Harwalke, J. A., Safayhi, H. and Golubic, M. (2002). Cytotoxic action of acetyl-11-keto-beta-boswellic acid (AKBA) on meningioma cells. *Plants Med.*, 68(5):397-401.
10. Zhu, N., Kikuzaki, H., Sheng, S., Sang, S., Rafi, M. M., Wang, M., Nakatani, N., Dipaola, R. S., Rosan, R. T. and No, C. T. (2001). Furanosquiteroenoids of commiphora myrrha. *J. Nat. Prod.*, 64(11):1460-2.
11. El Ashry, E. S., Rashed, N., Salma, O.M. and Saleh, A. (2003). Component, therapeutic value and uses of myrrh. *Pharmazie*, 58(3):163-8.
12. Tariq, M., Ageel, A. M., Al-Yhya, M. A., Mossa, J. S., Al-Said, M. S. and Parmar, N. S. (1986). Anti - inflammatory activity of *Commiphora molmol*. *Agents & Actions.*, 17(3-4): 381-2.
13. Michie, C. A. and Cooper, E. (1991). Frankincense and myrrh as remedies in children. *J. R. Soc. Med.*, 84(10):602-5.
14. Al-Harbi, M. M., Qureshi, S., Raza, M., Ahmed, M. M., Afzal, M. and Shah, A. H. (1997). Gastric antiulcer and cytoprotective effect of *Commiphora molmol* in rats. *J. Ethnopharmacol.*, 55(2):141-50.
15. Olajide, O. A. (1999). Investigation of the effect of selected medicinal plants on experimental thrombosis. *Phytother. Res.*, 13(3):231-2.
16. Massoud, A. M. and Labib, I. M. (2000). Larvicidal activity of *Commiphora molmol* against *Culex pipiens* and *Aedes caspius* larvae. *J. Egypt. Soc. Parasitol.*, 30(1):101-15.
17. Qin, G., Gopalan-Kriczky, P., Su, J., Ning, Y. and Lotlikar, D. P. (1997). Inhibition of aflatoxin B1 induced initiation of hepatocarcinogenesis in the rat by green tea. *Cancer Lett.*, 112:149-154.
18. Zhou, R., Zhou, Y., Chen, D., Li, S. and Hang, A. (2000). Effects of soaking temperature and soaking time during preparation of water extract of tea on anticlastogenicity against environmental tobacco smoke in the sister-chromatid exchange assay. *Toxicology. Letters*. 115:23-32.
19. Saris, N. E. (1978). Revised IFCC method for aspartate aminotransferase. *Clin. Chem.*, 24:720-721.
20. Bergmeyer, H. U, Scheibe, P. and Wahlefeld, W.W. (1978). Optimization of methods for aspartate amino transferase and alanine amino transferase. *Clin. Chem.*, 24/1:58-73.
21. Shaw, L. M, Stromme, J. L, London, J. L. and Theodorsen, L, (1983). IFFCC methods for the determination of enzymes part 4. IFFCC method for gamma glutamyl transferase [(gamma-glutamyl)-peptide; amino acid

- gamma glutamyl transferase, EC 2.3.2.2]. *Clinica Chemica Acta.*, 15F-338F.
22. **Jendrassik, L. and Grof, P. (1938).** Vereinfachte photometrische methoden zur bestimmung des blutbilirubin. *Biochem.*, 297:81.
  23. **Talke, H. and Schubert, G. E. (1965).** Enzymatische Harnstoffbestimmung in Blut und serum in optischen test nach Warburg, *Klin Wschr*, 41:174.
  24. **Bulgar, H. M. and Johns, H. E. (1941).** The determination of plasma uric acid. *J. Biol. Chem.*, 140:427. Chemical composition and antioxidant activity of *strobilanthes crispus* leaf extract.
  25. **Larsen, K. (1972).** Creatinine assay by a reaction-kinetic approach. *Clin Chem Acta.*, 41: 209-217.
  26. **Stadtman, T. C. (1957).** Methods in Enzymology, Vol III, colowick, SP, and caplan, NO, (Eds.), Academy Press, New York, NY, PP 392-394, 678-681.
  27. **Rautela, G. S, Hall, R. G, Bekiesz, C. L, Wermus, G. R. (1974).** A kinetic method for the rapid and automatic measurement of triglycerides in biological fluids. *Clin. Chem.*, 20:857.
  28. **Henry, R. J. (1974).** Clinical Principles and Technics, Harper and Row, New York, NY, PP 1283.
  29. **Van Assendelft, O.W. (1970).** Spectrophotometry of hemoglobin derivatives, Royal van Gorem Ltd, The Netherlands.
  30. **Bregman, A.A. (1983).** Laboratory investigations and cell biology. New York, Wiley, PP 51-60.
  31. **Lowry, O.H, Roesebrough, N.S, Farr, A. L. and Randall, R.J. (1951).** Protein measurement with folin phenol reagent. *J. Biol. Chem.*, 193:265-275.
  32. **Bancroft, J.D. and Stevens, A. (1996).** Theory and practice of histological technique. 4<sup>th</sup> Ed. Churchill, Livingston, Edinburgh, London, Melbourne and New York pp50-56.
  33. **Guerre, P., Burgat, V. and Galtier, P. (1997).** Dose-related increase in liver heme catabolism during rabbit of lathyrism. *Toxicol. Lett*, 92(2):101-8.
  34. **Rastogi, R., Srivastara, A. K. and Rostog, A. K. (2001).** Biochemocal changes induced in liver and serum of aflatoxin B1-treated male wister rats, preventive effect of picroliv. *Pharmacol. Toxicol*, 88(2):53-8.
  35. **Burt, M. E., Lowry, S. f., Gorschboth, C. and Brennan, M. F. (1981).** Metabolic alterations in noncachectic animal tumor system. *Cancer*, 47:2138-46.
  36. **Singh, N. and Venkitasubramanian, T. A. (1975).** Effect of aflatoxin B1 on lipids of rat tissues. *Environ. Pysiol. Biochem*, 5(3):147-57.
  37. **Ekman, L., Korlberg, I., Edstrom, S., Lindmark, L., Scherston, T. And Lundholm, K. (1982).** Metabolic alteration in liver, skeletal muscle and fat tissue in response to different tumor burdens in growing sarcoma bearing rats. *J. Surg. Res*, 33:23-31.

38. Al-Wadi, F. M. and Gumaa, K. A. (1987). Studies on the activity of individual plants of an antidiabetic plant mixture. *Acta. Diabetol. Lat.*, 24(1):37-41.
39. Al-Harbi, M. M., Qureshi, S., Raza, M., Ahmed, M. M., Giangreco, A. B. and Shah, A.H. (1994). Anticarcinogenic effect of *Commiphora molmol* on solid tumors induced by Ehrlich carcinoma cells in mice. *Chemotherapy.*, 40(5):337-47.
40. Raju, M.V. and Devegowada, G. (2000). Influence of esterified-glucomannan on performance and organ morphology, serum biochemistry and haematology in broilers exposed to individual and combined mycotoxicosis (aflatoxin, ochratoxin and T-2 toxin). *Br. Poult. Sci.*, 41(5):640-50.
41. Baptista, A. S., Horii, J., Calori-Domingues, M. A., Glória, E. M., Salgado, J. M. and Vizoli, M. R. (2002). Thermolysed and active yeast to reduce the toxicity of aflatoxin. *Sci. agric. (Piracicaba, Braz.)*, (59) no.2 Piracicaba.

### القيمة العلاجية للبان الذكر و المر في شفاء الكبد بعد التعرض لمركب "أفلاتوكسين ب ١"

طه ع. قمصاني\* وجهاد م. يوسف\*\* وأميمة أ. أبو زيد\*\*

\* قسم الكيمياء الحيوية، كلية العلوم، جامعة الملك عبد العزيز، جدة، المملكة العربية السعودية  
\*\* قسم الكيمياء، الأقسام العلمية، كلية التربية للبنات، جدة، المملكة العربية السعودية

يعتبر اللبان الذكر (الكندر) والمر من أقدم العلاجات النباتية التي وجدت في بلاد العرب ، وينتميان إلى العائلة البلسمية وينمو شجر الكندر في الهند والصومال وجبال اليمن، وقد اعتنى به الأطباء القدامى ووصفوه في كثير من علاجاتهم . أما المر فيتحصل عليه من شجيرة تنبت في جزيرة العرب والصومال وقد اعتبر لآلاف السنين كأحد كنوز الشرق. تهدف هذه الدراسة إلى معرفة القيمة العلاجية لهاتين المادتين (من مجموعة الراتنجيات)، كل على حده، في شفاء الكبد بعد التعرض لمركب "أفلاتوكسين ب ١".

ولإجراء هذه الدراسة تم حقن ٠.١ مل/ ١٠٠ جم من مادة "الأفلاتوكسين ب ١" داخل الغشاء البريتوني لذكور فئران التجارب المعملية البيضاء وتركها لمدة ١٠ أيام ثم تمت معالجتها بأعطائها خلاصة اللبان الذكر والمر لمدة ٢٠ يوماً، وفي نهاية التجربة أخذت عينات الدم لدراسة تأثير اللبان الذكر والمر على بعض الدلائل البيوكيميائية والتي شملت بعض الإنزيمات مثل إنزيم الأسبرتات أمينو ترانسفيريز (AST) وإنزيم ألانين أمينو ترانسفيريز (ALT) وإنزيم جاما-جلوتاميل ترانسفيريز (GGT) . والبليروبين، البولين، حمض البوليك، الكرياتينين، الكوليسستيرول، الجليسيريدات الثلاثية، الجلوكوز، الهيموجلوبين، كما تم أخذ عينة من الكبد لتقدير مستوى (الدنا DNA) و (الرنا RNA) والبروتين الكلي وإجراء الفحص النسيجي المجهرى. كل ذلك بالمقارنة بمجموعة ضابطة طبيعية.

اثبتت هذه الدراسة أن اللبان الذكر والمر يساعدان في تحسين الصورة البيوكيميائية والنسجية للكبد بعد التعرض للتأثيرات الضارة لمركب "الأفلاتوكسين ب ١".