

Protective Efficacy of Emodin against γ -Rays Induced Acute Hepatorenal Injury in Rats

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EMODIN(C₁₆H₁₂O₅), an active principle extracted from *Rheum palmatum*. Its protective effect was evaluated against γ -rays-induced biochemical alterations in rats.

The purpose of recent study is to demonstrate protective efficacy of emodin against γ -rays induced acute hepatorenal injury in rats.

γ -irradiation (6 Gy) caused significant elevation in the release of serum alanine and aspartate transaminases, (ALT & AST), alkaline phosphatase (SALP), lactate dehydrogenase (LDH), bilirubin (Br) and glucose (Gu) with concomitant decrease in haemoglobin (Hb) after 24 h of its exposure.

Toxicant exposure intensified the lipid peroxidation (LPO, measured as MDA units), total cholesterol (TC) and activity of acid phosphatase (TAC) and altered glutathione status (GSH), activities of adenosine triphosphatase (ATP), alkaline phosphatase (TALP), glutamate dehydrogenase (GDH) as well as major cellular constituents; total proteins (TP) and glycogen (Gn) in liver and kidney, compared to control measures.

Emodin, oral treatment, significantly lessened the toxicity by protecting γ -rays-induced alterations in various blood and tissue biochemical variables, compared to irradiated groups.

Thus, the study concluded that emodin at a dose of 40 mg/kg body wt possesses optimum hepatorenal protective ability in γ -irradiated toxicant rats.

Keywords: Emodin, hepatorenal distress, γ -rays, rats.

Ionizing radiation induces the production of free radicals such as hydrogen radicals, hydroxyl, singlet oxygen and peroxy radicals, in a cascade pathway. This irradiation can lead to mortality in mammals, so it is important to protect biological systems from radiation-induced tissue damage (Rzeszowska-Wolny *et al.*, 2009). Natural plant extracts have been shown to protect cells and tissues against ionizing radiation without adverse reactions (Tawfik and Mansour, 2008).

Plant-derived natural products including flavonoids, terpenoids and steroids *etc.* have received considerable attention due to their diverse pharmacological properties (DeFeudis *et al.*, 2003). Antioxidants play an important role in inhibiting and scavenging free radicals and provide protection against infection and degenerative diseases (Bhadauria, 2009).

Emodin is an important component of traditional Chinese herbs and has well-documented anti-inflammatory effect (Song *et al.*, 2012). It has been shown to possess several biological activities like immuneosuppressive (Zhao *et al.*, 2012) and as a useful chemotherapeutic agent against hepatocellular carcinoma (Jeon *et al.*, 2012).

The present study was undertaken to investigate the possible protective effect of emodin against γ -rays-induced hepatorenal damage in rats.

Materials and Methods

Animals and chemical administration

Sprague-Dawley rats (110 ± 10 g) were randomly selected from the departmental animal facility where they were inbred and housed under standard husbandry conditions ($25 \pm 2^\circ\text{C}$ temperature, 60-70 % relative humidity and 12 h photoperiod). All rats were given a standard rat diet and water *ad libitum*. Emodin and other chemicals were purchased from Sigma-Aldrich Co., USA. Emodin was dissolved in a little amount of saturated solution of NaHCO_3 and diluted with double distilled water making a dose of 40 mg/ kg/ 5ml and were administered orally according to Bhadauria (2009).

Irradiation

Whole-body γ -irradiation was performed at NCRRT, Cairo, Egypt, using Gamma Cell-40 biological irradiator (^{137}Cs). The dose rate was 0.46 Gy/ min at the time of the experiment.

Experimental plan and samples preparations

The rats were divided to four groups, each of 8 rats. Control group, received 5 ml distilled water contains the same amount of saturated solution of NaHCO_3 as vehicle. Emodin group, received dose of emodin (40 mg/ kg orally). γ -irradiated group, received vehicle and submitted to a dose of 6 Gy of γ -rays. Protected group, received emodin (40 mg/ kg orally) 24 h before exposure to 6

Egypt. J. Rad. Sci. Applic., Vol. 25, No. 1-2 (2012)

Gy of γ -rays. 24 h after end of the experiments, blood and liver tissues samples were harvested and immediately processed for biochemical analysis. Blood was kept for 1 h at room temperature after that serum was separated by centrifugation at 1000g for 15 min and stored at -20°C . Liver samples were homogenized with ice-cold 150 mM KCl and 1% sucrose for the determination of MDA and GSH. Homogenates of liver was prepared in chilled hypotonic solution (10% w/v) for other biochemical assays

Estimation of various biochemical endpoints in serum and blood

Serum was used for the estimation of ALT & AST (Reitman and Frankel, 1957), SALP (Roy *et al.*, 1970), LDH (Taffs and Sitkovsky, 1991), Br (Perry *et al.*, 1983), Hb (Swarup *et al.*, 1992), Gu (Teitz, 1986).

Liver and kidney tissue biochemical assay

LPO measured as MDA (Ohkawa *et al.*, 1979), GSH content (Brehe and Burch, 1976). Activities of ATP (Seth and Tangari, 1966), TAC (Barrett and Health, 1977), TALP (Roy *et al.*, 1970), GDH (Plummer, 1989), Gn (Seifter *et al.*, 1950), TC (Zlatkis *et al.*, 1953).

Serum and tissue TP was measured according to Bradford (1976) method. The results are reported as means \pm S.E of 8 rats. The results were analyzed by using one way analysis of variance (ANOVA) considering significant at $P < 0.05$ followed by student's *t*-test (Snedecor and Cochran, 1994).

Results

Emodin treated group showed non-significant changes in all blood, serum and tissues biochemical's parameters, Tables 1- 4.

TABLE 1. Serum liver biomarker enzymes; transaminases, (ALT & AST), alkaline phosphatase (SALP). Glucose (Gu) and lactate dehydrogenase (LDH) in different rat groups.

Rat groups	ALT (IU/L)	AST (IU/L)	SALP (IU/L)	Gu (mg/dl)	LDH (U/L)
Control	38.5 \pm 1.82 ^a	55.3 \pm 2.65 ^a	17.4 \pm 1.01 ^a	117.1 \pm 5.88 ^a	162.6 \pm 8.31 ^a
Emodin	38.6 \pm 1.87 ^a	57.2 \pm 2.47 ^a	17.6 \pm 1.12 ^a	121.2 \pm 5.76 ^a	164.4 \pm 8.46 ^a
γ-rays	82.3 \pm 4.05 ^b	121.5 \pm 5.43 ^b	28.2 \pm 1.71 ^b	177.4 \pm 9.65 ^b	321.3 \pm 16.24 ^b
Protected	51.1 \pm 2.6 ^c	61.2 \pm 2.86 ^c	21.6 \pm 1.15 ^c	115.7 \pm 5.43 ^c	201.1 \pm 11.13 ^c

^{a-c}Means in the same column with different superscript letters differ significantly at $P < 0.05$.

Significant increases of ALT, AST, SALP, Gu and LDH activities were noticed in serum after γ -rays exposure as shown in Table 1. Emodin treatment down regulated the activities of all these enzymes towards control and exhibited 23-50% protection, Table 1.

TABLE 2. Blood haemoglobin (Hb) and serum bilirubin (Br) in different rat groups.

Rat groups	Hb (g/dl)	Br (mg/dl)
Control	16.2± 1.01 ^a	0.26± 0.014 ^a
Emodin	16.5± 1.11 ^a	0.27± 0.019 ^a
γ -rays	12.2± 1.32 ^b	0.43± 0.021 ^b
Protected	15.1± 1.12 ^c	0.31± 0.016 ^c

Legends as in Table 1.

The γ -rays exposure significantly decreased blood Hb, where as increased serum Br level. Emodin in protected group lessened the toxic effects of γ -rays and showed significant protection in the two blood biochemical parameters and displayed 24-28% protection, Table 2.

TABLE 3. Tissue contents of lipid peroxidation (MDA) and glutathione (GSH) in different rat groups.

Rat groups	MDA (nmole/mg protein)	GSH (μ mole/g tissue)	
Control	Liver	0.56± 0.043 ^a	8.81± 0.341 ^a
	kidney	0.81± 0.041 ^a	4.81± 0.242 ^a
Emodin	Liver	0.54± 0.041 ^a	8.83± 0.343 ^a
	kidney	0.80± 0.044 ^a	4.83± 0.241 ^a
γ -rays	Liver	1.26± 0.66 ^b	4.31± 0.216 ^b
	kidney	1.81± 0.088 ^b	2.12± 0.105 ^b
Protected	Liver	0.81± 0.043 ^c	7.32± 0.365 ^c
	kidney	1.22± 0.064 ^c	4.34± 0.222 ^c

Legends as in Table 1.

The γ -rays exposure significantly increased liver and MDA levels, where as decreased tissues GSH levels. Emodin in protected group lessened the toxic effects of γ -rays and showed significant protection in all the tissues biochemical parameters, showing 33-105% protection, Table 3.

Activity of TALP, ATP and GDH were diminished after γ -rays exposure in both organs, Table 4. Emodin treatment caused significant improvements in enzymatic activities of the three enzymes. γ -rays significantly increased TAC activity in liver and kidney, where as emodin dosage reduced its activity, Table 4.

TABLE 4. Tissue enzymes activity of acid phosphatase (TAC), alkaline phosphatase (TALP), adenosine triphosphatase (ATP) and glutamate dehydrogenase (GDH) in different rat groups.

Rat groups		TAC (mgPi/100ml/h)	TALP (mgPi/100ml/h)	ATP (mgPi/100ml/S)	GDH (U/g protein)
Control	Liver	194.4± 9.9 ^a	53.4± 2.58 ^a	32.3± 1.57 ^a	2123± 116.4 ^a
	kidney	224.8± 11.4 ^a	1911.5± 92.75 ^a	42.2± 2.12 ^a	934± 46 ^a
Emodin	Liver	197.6± 9.4 ^a	55.6± 2.46 ^a	32.7± 1.53 ^a	2206± 156.5 ^a
	kidney	229.2± 11.7 ^a	1941.3± 91.23 ^a	44.1± 2.43 ^a	947± 48 ^a
γ-rays	Liver	292.3± 15.24 ^b	18.1± 0.87 ^b	15.8± 0.79 ^b	1244± 66 ^b
	kidney	281.1± 14.68 ^b	624.4± 31.19 ^b	20.3± 1.21 ^b	453± 23 ^b
Protected	Liver	203.4± 9.87 ^c	27.9± 1.54 ^c	26.1± 1.46 ^c	1813± 93 ^c
	kidney	235.1± 11.18 ^c	1073.1± 55.46 ^c	34.4± 1.87 ^c	764± 39 ^c

Legends as in Table 1.

The γ -rays exposure significantly decreased TP contents in liver and kidney as well as hepatorenal glycogen contents, Table 5. Emodin therapy did not show protective effect on renal proteins, where as it enhanced hepatic proteins significantly. Emodin significantly reversed hepatic and renal glycogen towards control. The γ -rays exposure increased TC contents in liver and kidney. Treatment of emodin reduced TC contents significantly in both of liver and kidney, respectively, Table 5.

TABLE 5. Tissue constituents; total protein (TP), glycogen (Gn) and total cholesterol (TC) levels in different rat groups.

Rat groups		TP (mg/100 mg)	Gn (mg/100 mg)	TC (mg/100 g)
Control	Liver	19.3± 0.92 ^a	2591± 129.6 ^a	2.9± 0.17 ^a
	kidney	17.9± 0.86 ^a	78± 3.9 ^a	1.5± 0.08 ^a
Emodin	Liver	19.5± 0.76 ^a	2598± 116.6 ^a	3.0± 0.14 ^a
	kidney	18.1± 0.79 ^a	82± 3.7 ^a	1.6± 0.07 ^a
γ-rays	Liver	14.6± 0.68 ^b	1564± 78.3 ^b	4.2± 0.21 ^b
	kidney	14.3± 0.66 ^b	51± 3.5 ^b	3.0± 0.16 ^b
Protected	Liver	17.7± 0.86 ^c	2218± 109.3 ^c	3.3± 0.17 ^c
	kidney	16.3± 0.83 ^b	73± 2.7 ^c	2.0± 0.10 ^c

Legends as in Table 1.

Discussion

In the present study γ -rays-induced hepatorenal injury was evidenced by biochemical measurements. Increased level of serum ALT, AST, SALP, Gu and

LDH indicated deterioration in the hepatic functions due to damaging effects of γ -rays. Increase in MDA accompanied by reduction in GSH implicated hepatorenal oxidative damage. Emodin intake helped in mitigating γ -rays-induced toxic consequences on liver and kidney. Since, involvement of free radicals in the pathogenesis of γ -rays-induced hepatotoxic effects is well-known (Ping *et al.*, 2012), thus; free radical scavenging property of emodin has also been well-supported.

The γ -rays-induced depletion of cytosolic and mitochondrial GSH content lead to the loss of cellular homeostasis leading to liver injury (Limon-Pacheco and Gonsebatt, 2009). The γ -rays-induced depletion of GSH was restored towards control by emodin treatment, which is in agreement with the fact that exogenous administration of antioxidants also influences the GSH metabolism. Therefore, emodin might play a key role in protection against γ -rays intoxication by modulating the cellular GSH pool. The MDA is a good indicator of the degree of LPO (Adaramoye *et al.*, 2012), which is closely related to γ -rays-induced tissue damage. γ -rays exposure induced LPO and subsequent hepatorenal injury supports the findings of previous study (El-Khafif *et al.*, 2003).

In this study, γ -rays-induced cellular alterations were supposed on the basis of significant elevation in release of AST and ALT (Omran *et al.*, 2009). Prolonged destruction in hepatic cells results in more hepatic releases to exacerbate hepatic dysfunction and causes an elevation of SALP, LDH and Br in serum (Schmidt, 1978). Elevation in these parameters due to γ -rays challenge is well-reported with protective effects of stem Extract of *Eucalyptus maculata* on them (Mohamed *et al.*, 2005). Alteration of liver function tests after administration of emodin signifies its strong hepatoprotective activity.

Hematopoietic stem cells are highly sensitive to ionising-radiation. Hematopoietic dysfunction is the most common clinical complications of radiation exposures (Chen *et al.*, 2007). Radiation-induced destruction of the hematopoietic systems causes depletion of peripheral blood elements, leading to a loss of function. Subsequently, the exposed individuals become susceptible to opportunistic pathogens (Walsh *et al.*, 2009). In addition, emodin repairs damaged DNA in γ -irradiated rats indicates that it protects cells against

radiation-induced damage which may be attributed to its ROS scavenging activity (Heo *et al.*, 2010). These functions of emodin are likely to be factors in the protective efficacy of the irradiated rats.

Radiation induced damages to membranes of the sub cellular organelles marker enzymes; TAC, TALP and GDH could be attributed to peroxidation of membrane lipid portion monitored by the increases in MDA (Azab, 2007). In addition, hepatic injury elicits intracellular stress that leads to peroxidation of membrane lipids accompanied by alteration in the structural and the functional characteristic of the membrane, which affects the activities of the membrane-bound ATP (Devi *et al.*, 2004).

In the present study, decreased activities of ATP, TALP and GDH might be due to the membrane fragility and/or altered permeability, whereas increased activity of TAC might be due to lysosomal injury. Emodin maintained the activity of these metabolic enzymes towards control either by preventing peroxidation of membrane or by stabilizing permeability or by both (Bhadauria, 2010).

Impairments in cellular metabolism due to initiation of LPO altered major cellular components *ie.*, TP, Gn and TC. The main source of energy in liver is Gn and it is utilized to maintain blood Gu level (Gustavsson *et al.*, 2010). Exposure to γ -rays increases Gu release and glycolysis from endogenous Gn (glycogenolysis) and inhibits oxygen uptake (Kang *et al.*, 2010 and Verspohl *et al.*, 2003). Hepatorenal Gn was reduced markedly after exposure to γ -rays in this study. Emodin therapy significantly prevented the alterations of Gn in liver and kidney, which was an indication of improved metabolic functioning. Emodin maintained Gn status probably by its antifibrotic mechanism because fibrosis disrupts the normal architecture and blood flow into the liver, therefore, inhibits the nutrients to be absorbed by the hepatocytes (Chavez *et al.*, 2008).

Diminishment in hepatorenal TP and Gn contents indicated the alterations in their synthesis, whereas increase in TC might be due to the defect in the lipid metabolism and its decreased utilization by cells in toxic conditions (Baker *et al.*, 2009 and Seyama, 2003). Emodin treatment prevented γ -rays-induced biochemical alterations in the cell components towards normal by improving

the cellular metabolism and by providing protection against tissue necrosis (Bhadauria, 2010).

Conclusion

Emodin has the ability to regulate γ -rays-induced alterations in liver function tests, metabolic enzymatic activities, GSH and major cellular components as well as in mitigating hepatorenal cellular damage by diminishing oxidative stress.

Recommendations

Emodin needs an attention to be considered as a potential agent in limiting γ -rays-induced toxic effects.

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كفاءة الايمودين في الوقاية من الإصابات الحادة التي تحدثها أشعة جاما في كبد و كلي الجرذان

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قسم بيولوجيا الإشعاع ، المركز القومي لبحوث وتكنولوجيا الإشعاع ، ص. ب. ٢٩ مدينة نصر ، و*بحوث التطبيقات البيولوجية ، مركز البحوث النووية ، انشاص ، ص. ب. ١٣٧٥٩ مصر.

يستخرج الايمودين من جذر نبات الفنتيلاجو الهندي ، و قد تم تقييم دوره في حماية الجرذان من التغييرات الكيموحيوية التي تحدثها أشعة جاما. تبين وجود ارتفاعا احصائيا في نشاط انزيمات الترانس أمينيزس (ALT&AST) و الفوسفاتيز القلوي (SALP) و لاكتيت ديهيدروجينيز (LDH) و مستوي كل من البيلوروبين (Br) و الجلوكوز (Gu) في سيرم الدم و صاحب ذلك نقص في مستوي الهيموجلوبين بالدم بعد ٢٤ ساعة من التعرض لأشعة جاما (جرعة ٦ جراي). كما سبب التعرض للأشعة السامة زيادة مستوي كل من أكسدة الليبيدات (LPO ، المقدره بوحدات MDA) و الكوليستيرول الكلي (TC) و انزيم الفوسفاتيز الحمضي (TAC) ، و ادت الي نقص مستوي كل من الجلوتاثيون (GSH) و البروتين الكلي (TP) و الجليكوجين (Gn) و انزيمات أدينوسين الفوسفات الثلاثي (ATP) و الفوسفاتيز القلوي (TALP) و جلوتيميت ديهيدروجينيز (GD) في نسيج كل من الكبد و الكلي و ذلك عند مقارنتها بقياسات المجموعة الضابطة. أدى تناول عقار الايمودين بالفم إلي السيطرة علي سمية أشعة جاما و تحسين مستوي كل القياسات الكيموحيوية الدموية و النسيجية و ذلك عند مقارنتها بقياسات المجموعة المعرضة للإشعاع. وقد أثبتت الدراسة أن الايمودين (جرعة ٤٠ ملليجرام/ كجم من وزن الجرذ) يقي من اصابات الكبد و الكلي التي تحدث في الفئران نتيجة التعرض لأشعة جاما.