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Therapeutic and Protection Effectiveness of Quercetin in γ-Irradiated Rats

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QUERCETIN (QTN); 3,5,7,3',4'-pentahydroxy flavone is a well known phenolic compound widely present in the plant kingdom.

This study investigates the potential of QTN to protect the hepatic, myocardial and renal tissues against global reperfusion injury induced by γ-irradiation. The study was performed in Sprage Dawlly rats that were administered during 7 days with QTN (50 mg/ kg body wt), before or after exposure to 6 Gy γ-rays with appropriate controls.

The irradiation data revealed significant elevation in plasma malondialdehyde (MDA) and conjugated dienes (CD) accompanied by reduced levels of total glutathione (GSH) and nitric oxide (NO). In addition, reduced activity of glutathione reductase (GR) and increased lactate dehydrogenase (LDH) activity were determined in tissue homogenates of liver, heart and kidney due to radiation damage when compared with respective control values.

Treatment with QTN either before or after radiation-exposure improved organs function and reduced the severity of liver, heart and kidney injuries reflected by the lower concentrations of lipid peroxidation and the pro-inflammatory and tissue damaging LDH-indicator. In addition, improvement in the oxidative-stress' indicators confirms the therapeutic and protection effectiveness of QTN.

These effects were linked to peroxyl-radical trapping capacity of QTN, which quenches free radicals induced by exposure of rats to γ-rays, thus improving regeneration of the biological tissues. It may be concluded that QTN is effective in this model of organs injury and cell damage and could become useful supplement in the treatment of liver, heart and kidney inflammatory status and oxidative stress.

Keywords: Quercetin, liver, heart, kidney, γ-rays, rats.

Numerous studies have clearly demonstrated that polyphenolic compounds such as flavonols, exhibit beneficial health effects due to their antioxidant properties (Huang *et al.,* 2012). Flavonoids; including the flavonol QTN, are reported to exhibit a wide range of biological activities related to their antioxidant capacity as free radical scavenger (Larson *et al.,* 2012). Recently, radiobiologists have focussed their attention towards the phytochemicals due to their lower toxicity. The dietary flavonoid; QTN is a major flavonoid widely distributed in apples, onions, berries red wine and tea, and in the propolis of bee hives (Larson *et al.*, 2012).

The destructive effects of radiation arise from direct and indirect ionization events that disrupt chemical bonds in bio molecules such as proteins, lipids and DNA, which result in important cellular damage. All animals and human beings are being exposed to radiation from natural as well as man-made sources and exposure to such radiations can induce alterations in the cellular macromolecules and affect their functions (Azzam *et al.,* 2011). In addition, inflammation can play a key role in ionising-radiation induced cells and organs dysfunctions (Morales *et al.,* 2006).

In spite of the large number of in-*vitro* studies published on the effects of QTN, the literature on their effects in-*vivo* is inadequate. Therefore, the aim of the present investigation was to evaluate the therapeutic and protective effectiveness of QTN against inflammatory status and oxidative stress developed in rat model by exposure to γ-rays on liver, heart and kidney.

Materials and Methods

Irradiation

Irradiation was performed with ^{137}Cesium, γ-radiation source (γ-Cell-40 manufactured by the Atomic Energy of Canada, Ltd.) at NCRRT. The dose rate was 0.42 Gy/ min.

QTN treatment

QTN was purchased from Sigma/Aldrich, St. Louis, MO, USA. Animals received QTN dosage according to the protocol of Ikizler *et al.* (2007).

Animals, experimental design and sampling

Sprague-Dawley rats (n= 10, both sexes in each group) were purchased from the *Laboratory Animal Centre of the Holding Company for Biological Products and Vaccines,* Cairo, Egypt. Rats were housed under normal standardized conditions with free access to food and tap water. Rats were handled according to the National Academy of Sciences standards (1996). The animals were randomly divided into 5 groups. Group 1: animals were kept as controls and received the vehicle; 0.2 % (vol/ vol) ethanol in tap water via intra-gastric tubes once daily for 7 days. Group 2: Rats received 50 mg QTN/ kg body wt dissolved in the vehicle once daily for 7 days. Group 3: Rats exposed to single dose (6 Gy) of whole body γ-rays. Group 4: Rats received QTN dosage for 7 days and after 24 h exposed to 6 Gy of γ-rays. Group 5: Rats were submitted to 6 Gy γ-rays then after 24 h, treated with QTN for 7 consecutive days. All treated rats were sacrificed 24 h after treatment by cervical dislocation. Blood samples were withdrawn from the heart, collected in tubes containing EDTA as anticoagulant for preparing plasma, centrifuged (1000x g for 10 min), collected in test tubes with screw caps and stored at -20 °C until analyzed. Tissues homogenates were prepared in ice-cold phosphate buffer (0.1M, pH 7.4) using a Potter-Elvehjem homogenizer (Poland) to give a 10 % homogenates.

Biochemical assay

Cytotoxicity indicators were determined in plasma by assaying lipid peroxidation as MDA (Ohkawa *et al.*, 1979) and CD as described previously (Benito *et al.*, 2004). Tissue damages were assessed using LDH (Asha and Radha, 1985). Evidence of oxidative stress was determined from the tissue homogenates using GSH and NO levels (Beutler, 1957 and Wang *et al.,* 1998) and GR activity (Manso and Wroblewski, 1957). Protein concentrations were measured according to the method of Lowry *et al.* (1951).

Statistical analysis

All results are expressed as mean± SD. Comparisons among groups were performed by one-way analysis of variance (ANOVA) followed by Tukey's post-test, when indicated. The level of statistical significance was set at $p< 0.05$ (Zar, 1999).

Results

No significant differences in MDA and CD levels in plasma were observed between the QTN-treated and the control group. The MDA and CD in plasma of irradiated groups were significantly higher than in control and QTN-treated rats. MDA was restored in both the protected and therapeutic groups and not significantly different in comparison with QTN-treated and untreated control. CD level in both the protected and therapeutic groups was similarly ameliorated but remained significantly different in comparison with the control and irradiated groups (Table 1).

TABLE 1. Plasma malondialdehyde (MDA) and conjugated dienes (CD) in different groups of animals.

Groups	**MDA** (μM/ mg protein)	**CD** (n mol/ mg protein)
Control	6.78± 0.542**A**	0.76± 0.087**A**
QTN-treated	6.72± 0.433**A**	0.74± 0.081**A**
Irradiated (6 Gy)	9.34± 0.784**B**	0.95± 0.094**B**
QTN+ Irradiated	7.72± 0.683**A**	0.83± 0.075**C**
Irradiated+ QTN	8.32± 0.743**A**	0.87± 0.088**C**

Groups not sharing common superscripts (**A**, **B**, **C**) differ significantly at $p< 0.05$ level.

QTN-intake revealed insignificant increase in LDH activity in tissue of different tested organs (Table 2). However, the irradiated animals showed significant elevation in LDH activity in liver, heart and kidney relative to their corresponding control and QTN-treated values. Additionally, QTN-treatment prior- and post- irradiation markedly ameliorated the rise in LDH activity and partially restored the enzyme activity in the tested tissues.

TABLE 2. Lactate dehydrogenase (LDH) activity (U/ mg protein) in examined tissues in different groups of animals.

Groups	Liver	Heart	Kidney
Control	122.5± 10.36**A**	13.1± 1.21**A**	5.6± 0.54**A**
QTN-treatd	124.6± 10.67**A**	12.9± 1.32**A**	5.5± 0.63**A**
Irradiated (6 Gy)	212.7± 18.55**B**	19.5± 1.68**B**	10.2± 1.16**B**
QTN+ Irradiated	151.2± 15.12**C**	15.4± 1.71**C**	6.1± 0.61**C**
Irradiated+ QTN	177.6± 15.67D	16.8± 1.59**D**	8.4± 0.87**D**

Groups not sharing common superscripts (**A**, **B**, **C, D)** differ significantly at $p< 0.05$ level.

No significant differences in levels of GSH and NO and activity of GR in the tested tissues were observed between the QTN-treated and the control group

(Table 3). A marked depletion in GSH and NO levels was observed in the examined tissues of irradiated groups.

TABLE 3. Levels of total glutathione; GSH (mg/g tissue) and nitric oxide; NO (n mol/g wet tissue/min) and activity of glutathione reductase; GR (U/g wet tissue/min) in examined tissues in different groups of animals.

Groups	Liver	Heart	Kidney
Control			
GSH	64.4± 5.22**A**	41.6± 3.91**A**	32.7± 3.12**A**
NO	21.1± 2.23**A**	17.2± 1.66**A**	7.3± 0.82**A**
GR	764.1± 57.74**A**	453.2± 42.4**2A**	156.7± 14.44**A**
QTN-treated			
GSH	63.2± 5.45**A**	42.2± 3.24**A**	33.7± 3.32**A**
NO	20.3± 1.93**A**	17.1± 1.35**A**	7.2± 0.6**6A**
GR	772.1± 53.49**A**	459.7± 44.8**1A**	155.6± 15.11**A**
Irradiated (6 Gy)			
GSH	37.8± 5.23**B**	36.7± 3.24**B**	21.9± 2.01**B**
NO	9.4± 1.02**B**	14.3± 1.61**B**	4.2± 0/36**B**
GR	406.4± 37.76**B**	276.6± 24.42**B**	67.4± 6.11**B**
QTN+ Irradiated			
GSH	55.6± 4.67**C**	40.4± 3.77**A**	31.8± 32.89**A**
NO	17.6± 1.56**A**	16.5± 1.46**A**	6.4± 0.58**C**
GR	612.7± 58.77**C**	312.2± 29.8**2C**	99.4± 9.54**C**
Irradiated+ QTN			
GSH	48.6± 4.24**D**	41.6± 4.13**A**	28.1± 2.55**A**
NO	16.2± 1.47**A**	16.0± 1.39**A**	5.4± 0.51**D**
GR	533.5± 32.66**D**	378.3± 36.33**D**	121.7± 11.26**D**

Groups not sharing common superscripts (**A**, **B**, **C, D)** differ significantly at $p < 0.05$ level.

Treatment with QTN revealed significantly elevated level of GSH contents and NO in the tissues of both the protected and treated groups in comparison with irradiated group, which resulted in non-significant GSH-changes in heart and kidney and non-significant NO-changes in liver and heart in comparison with control values. A marked inhibition in GR activity resulted in the 3 tissues tested post irradiation. Here, treatment of rats with QTN pre- and post-radiation exposure resulted in significant augmentation comparing with irradiated values in the liver, heart and kidney homogenates.

Discussion

Flavonoids have preventive effects on degenerative diseases (Palafox-Carlos *et al.*, 2011). QTN is a dietary flavonoid ubiquitous in nature. It is found

in many plants, such as onions, broccoli and tea (Martinez *et al.*, 2008). QTN inhibits the expression of free radical-generating enzymes and thus, function as strong antioxidants (Frei and Higdon, 2003).

The MDA and CD in plasma of irradiated groups was significantly elevated than in control and QTN-treated rat groups. In the study we present, the levels of MDA and DC, which are indicators of the damage induced by free radicals on lipid tissues were found to be lower in the QTN protected and treated groups compared to control group. Lipid peroxidation is relevant to the pathophysiological aspects including aging and various diseases such as cancer, inflammation. The preventive strategy against it has been directed to the use of medicinal and natural antioxidants (Tsuchiya *et al.*, 2008). QTN is a free radical scavenging antioxidant (Boots *et al.*, 2007), protects cell from oxidant injury and cell death by scavenging oxygen radicals (Erden Inal *et al.*, 2001) and prevents propagation of lipid peroxidation (Noaman *et al.*, 2006).

Among other pharmacological and biochemical effects, QTN prevents lipid peroxidation and scavenges superoxide radicals (O'Brien *et al.*, 2000). These effects are due to the presence of several aromatic rings containing hydroxyl radicals and the essential part of the free radical-scavenging activity; the o-dihydroxyl group in the B-ring (catechol group) in their diphenylpropane structure (Terao, 2009). On the basis of these biochemical properties, it is expected that, in the present study, the simultaneous use of QTN led to significant attenuation of the γ-irradiation-induced increases in the levels of MDA and DC. Similar effects of QTN on tissue levels of MDA have been described in other animal models of organ injury (Singh *et al.*, 2004). In the present study, the levels of MDA and DC were also significantly lower in the protected and treated groups than in the irradiated group, strongly supporting the role for QTN as an inhibitor of lipid peroxidation.

The significant increase in LDH activity has been previously mentioned in tissues of animals exposed to radiation (Tanito *et al.*, 2002 and Cai *et al.*, 2004), which indicated the cytotoxicity of radiation-exposure. Sridharan and Shyamaladevi (2002) attributed the elevation in LDH concentration to the excessive production of free radicals and lipid peroxides, which might have caused the leakage of cytosolic enzymes such as LDH. In a similar manner,

levels of LDH that was defence mechanisms against ROS damage were found to be higher in irradiated group of the study. QTN treatment before- and after-irradiation decrease oxidative stress through its antioxidant properties. Thus, lowers level of LDH enzyme these groups indicates that QTN has ameliorative and curative roles on irradiation-injury in the tested tissues.

The results of the present study showed that QTN treatment prior- and post-irradiation ameliorated radiation induced Cytotoxicity, tissue damage and oxidative stresses in plasma and examined tissues relative to corresponding controls. QTN attenuates the hepatic injury induced by γ-irradiation. This effect was characterized by a reduction in oxidative stress of liver. In addition, QTN has been shown to have anti-inflammatory properties in hepatic cirrhosis in various in-*vitro* and in-*vivo* systems (Pavanato *et al.,* 2003). Furthermore, QTN inhibited the proliferation and collagen synthesis of hepatic rat-derived stellate cells that played a central role in experimental models of chronic liver disease as well as in humans with chronic liver disease (Kang *et al.,* 2001).

Oxidative stress and inflammation have been strongly implicated in the low-antioxidant status. Interplay between inflammatory reaction and the cell membranes of heart and kidney cause relative NO-deficiency as a result of reaction of NO with oxygen radicals (Bongartz *et al.,* 2005) and by further increasing production of ROS, the balance between NO and ROS are twisted towards the latter. Exposure of animals to γ-rays caused significant decreases in NO levels in the 3 tested tissue homogenates. QTN-treatment produces cytoprotective action where, NO increases thereby protecting the tissues from irradiation damage. Therefore, increasing attention has been paid to searching for effective approaches to the prevention and treatment of γ-rays-induced tissue injuries, including the use of flavonoid antioxidants.

It was confirmed that both antiradical and chelating effects are involved in the protective effect of QTN (Cheng and Breen, 2000). Furthermore, QTN treatment has also been reported to significantly improve the decrease of GR activity induced by oxidative stress (Erden Inal *et al.,* 2001). In our experiments, GR activity was significantly decreased in irradiated animal group. We suggest that decreased GR activity may be a consequence of γ-rays-induced oxidative stress. The finding that oxidative stress depletes the activity of GR

supports this hypothesis (Leutner *et al.,* 2001). In animals exposed to γ-rays pre- and post-treated with QTN, there was a significant elevation in GR activity as compared to the γ-irradiated animals. We suggest that this can be a consequence of a QTN antioxidative effect.

Conclusion

As a major dietary flavonoid, due to its antioxidant and cytoprotective actions, QTN has the capacity to protect the hepatic, myocardial and renal tissues against global reperfusion injury induced by γ-irradiation.

Recommendation

Possible implications of such research include the development of radio-protecting and radio-therapeutic strategy.

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الكفاءة العلاجية و الوقائية للكوريستين في الجرذان المعرضة لأشعة جاما

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قسمي بحوث البيولوجيا الإشعاعية و *البحوث الصحية الإشعاعية ، المركز القومي لبحوث وتكنولوجيا الإشعاع ، ص. ب. ٢٩ مدينة نصر، مصر.

يعتبر الكوريستين من الفينولينات واسعة الآنتشار في المملكة النباتية. تم دراسة الكفاءة الوقائية و العلاجية للكوريستين (٥٠ ميلليجرام / كجم) بتجريع الكوريستين لمجموعة من الجرذان لمدة ٧ أيام ، و تعريض مجموعة أخري لجرعة ٦ جراي من أشعة جاما ، و مجموعة تجرعت الكوريستين ثم عرضت لأشعة جاما ، و مجموعة تعرضت لأشعة جاما ثم تجرعت الكوريستين وذلك بالمقارنة بمجموعة ضابطة من الجرذان. أظهرت النتائج حدوث زيادة إحصائية في مستوي كل من معيار أكسدة الليبيات (MDA) و المواد المرتبطة بها (CD) في بلازما الدم وهذه الزيادة مرتبطة بنقص إحصائي بمستوي كل من الجلوتاثيون الكلي (GSH) و والنيترات المؤكسدة (NO). و كذلك زيادة نشاط انزيم اللاكتيت ديهيدروجينيز (LDH) و نقص نشاط إنزيم الجلوتاثيون المختزل (GR) في أنسجة الكبد و القلب و الكلي بسبب تأثير أشعة جاما علي تلك الأنسجة و ذلك عند مقارنتها بنتائج المجموعة الضابطة.

كما بينت النتائج أن الوقاية و العلاج بالكوريستين حسنت من وظائف الكبد و القلب و الكلي و قللت من التأثير المدمر لأشعة جاما علي تلك الأجهزة الحيوية. أدي استخدام الكوريستين إلي التحكم في نسبة تأكسد الدهون (lipid peroxidation) و مستوي تدمير الخلايا و ارتفاع نشاط إنزيم اللاكتيت ديهيدروجينيز.

و كذلك أدي إلي تحسن مستويات معايير الضغط التأكسدي (GSH, GR & NO) مما يؤكد الفاعلية العلاجية و الوقائية للكوريستين في الجرذان المعرضة لأشعة جاما. هذه الفاعلية مرتبطة بقدرة الكوريستين علي التقاط الشوارد الحرة - التي يحدثها الإشعاع من الدم و خلايا أنسجة الكبد و القلب و الكلي. و من ثم يعمل علي استعادة الوظائف الحيوية لأنسجة جسم الجرذ.

الخلاصة: الكوريستين فعال في وقاية و علاج خلايا أنسجة الكبد و القلب و الكلي في الحيوانات التي تتعرض للأضرار التدميرية لأشعة جاما و بالتالي فأنه مفيد عند إضافته لطعامها.