Disturbances in Pituitary-Thyroid Axis
Hormones in Rats Exposed to CCL₄ and/ or Gamma-Irradiation

N. Abdel Aziz

Radiation Biology Dept., National Centre for Radiation Research and Technology (NCRRT), P. O. Box; 29 Nasr City, Egypt.

THIS WORK aims to detect the disturbances in hormones of pituitary-thyroid axis in male rats exposed to liver injury by carbon tetrachloride (CCL₄) and/ or γ-irradiation, as well as modulating these disturbances by supplementation of hepato-protective agent, silymarin-plus (S+). Subcutaneous injection of CCL₄ as a hepatotoxic agent, 1 ml/ Kg body wt two times/ week for 3 weeks alone or combined with 6Gy fractionated doses of whole body γ-irradiation (1Gy two times/ week for 3 weeks) induced hepatotoxicity as manifested biochemically by an elevation of liver marker enzymes; transaminases (ALT & AST) and alkaline phosphatase (ALP).

Oxidative stress in liver was evidenced by a significant increase of malondialdehyde (MDA) along with reduction of glutathione (GSH) content. Liver damage induced by CCL₄ and/or γ-irradiation was accompanied by a significant decrease in the levels of serum triiodothyronine (T₃) and thyroxin (T₄), while thyroid-stimulating hormone (TSH) showed a significant increase. In addition, significant increases were recorded in total cholesterol and triglycerides levels whereas; significant decrease was recorded in glucose level in group exposed to CCL₄ and γ-irradiation. Oral supplementation of S+ ameliorated the changes induced by exposure to CCL₄ and/ or γ-irradiation.

In conclusion, the present data demonstrated that exposure to chemical as well as physical environmental biohazards induced liver injury concomitant with a hypothyroid state. This disturbance can be modulated by supplementation of hepato-protective agent.

Keywords: CCL₄, γ-rays, rats, liver, thyroid hormones, silymarin.

Exposure to xenobiotic and ionizing radiation induce complex cellular responses in both experimental animals and humans which may lead to adverse outcomes. Several investigations indicated that damaging effects of radiation and xenobiotic as CCL₄ result from production of highly reactive free radicals.
and their consequent action on biological molecules which leads to injury of different tissues (Ashry, 2008 and Hanafy et al., 2008).

Liver injury is a prevalent pathology that involves a variety of disorders including oxidative stress, hepatitis, fibrosis, cirrhosis, apoptosis and hepatocellular carcinoma. Numerous reports have shown an indirect effect of liver disorders on serum levels of thyroid hormones (Moustafa et al., 2009), where the liver plays an important role in the metabolism of thyroid hormones and regulates their systemic endocrine effects (Malik and Hodgson, 2002). In turn, thyroid hormones regulate the basal metabolic rate of hepatocytes and modulate hepatic function. Hence, liver and thyroid hormones closely connected, and dysfunction of one causes a disturbance in the other (Malik and Hodgson, 2002 and Khan, 2012). Consequently, treatment of liver injury is important in modulation of the disturbance in pituitary-thyroid axis hormones resulting from exposure to chemical and/ or physical environmental biohazards.

Silymarin is a flavonoid derived from milk thistle that has been reported to afford hepato-protection in vitro and in vivo by inhibiting the production of pro-inflammatory and pro-fibrogenic factors (Grattagliano et al., 2013). S+ is a combination of silymarin and other standard antioxidants that include acetyl cysteine, vitamin E, vitamin C, vitamin A, selenium and zinc. It was reported that combination of antioxidants exerts synergistic action in scavenging free radicals (Fang et al., 2002) and employs a series of redox reactions (Blokhina et al., 2003 and Mandelker, 2004) since deficiency in one component may affect the efficiency of the other (Vertuani et al., 2004).

This work aims to underscore the similarity of the toxic effect of CCl₄ metabolites and its highly reactive derivatives to those of gamma irradiation, as well as, their synergistic impact on pituitary-thyroid axis hormones. Investigating the impact of the hepato-protective drug, S+ also is intended.

**Material and Methods**

Male albino rats weighing 120-150 g (45-60 days) were obtained from the National Research Centre, Giza, Egypt. The animals were maintained under conventional conditions with free access to water and standard pellet concentrated diet.
Radiation exposure

Whole body γ-irradiation was performed at the NCRRT, Cairo, Egypt using a ventilated Canadian $^{137}$Cs Gamma Cell-40 at a dose rate of 0.47 Gy/min. Rats were exposed to fractionated doses, 1Gy two times/week for three weeks.

Chemicals

CCl$_4$ was purchased from El-Nasr Pharmaceutical Chemical Co., Egypt. Animals were injected subcutaneously 1ml/kg body wt (Ibrahim and Abdel Aziz, 2009) two times/week for a period of 3 weeks. The S+ produced by SEDICO pharmaceutical Co, Egypt. It was available in the form of sachets containing Silymarin (200 mg), acetylcysteine (200 mg), vitamin A (300 IU), ascorbic acid (30 mg), vitamin E (5 IU), selenium (18.3 µg) and zinc (3.6 mg). It was orally administered 150 mg (dissolved in water)/kg/day (Omar et al., 2012).

Experimental design

Animals were divided into 7 groups: Control untreated normal rats, CCl$_4$: Rats injected with CCl$_4$ (1ml/ kg body wt) subcutaneously two times/week for a period of 3 weeks, S+ plus CCl$_4$: Rats orally supplemented with S+ daily for one week before and then through CCl$_4$ administration for a period of 3 weeks. IRR: Rats whole body exposed to γ-irradiation (1Gy) two times/week for 3 weeks, S+ plus IRR: Rats supplemented with S+ daily for one week before and then through exposure to γ-irradiation for a period of 3 weeks. CCl$_4$ plus IRR: Rats injected with CCl$_4$ (1ml/ kg body wt) two times/week for 3 weeks and exposed to γ-irradiation (1Gy) two times/week for 3 weeks. S+ plus CCl$_4$ plus IRR: Rats treated with S+ and CCl$_4$ and exposed to irradiation as described above. The animals were sacrificed one day after the last dose of irradiation and/or treatment.

Biochemical analysis

Animals were lightly anesthetized with ether and blood was collected by heart puncture. Serum was separated by blood centrifugation at 3000 rpm. Liver was quickly removed and homogenized in ice-cold saline. Both serum and homogenate were stored at −20°C till biochemical analysis. Serum transaminases (ALT & AST) and ALP were determined according to Reitman and Frankel (1957) and Belfield and Goldberg (1971), respectively. Total T$_3$ and T$_4$ in serum were measured using radioimmunoassay kits, TSH was
measured using immunoradiometric assay kit produced by Diagnostic Products Corporation, USA. Liver GSH and MDA contents were determined according to Beutler et al. (1963) and Yoshioka et al. (1979), respectively. Serum cholesterol, triglycerides and glucose levels were determined according to Richmond (1973), Fossati and Principe (1982) and Trinder (1969), respectively.

**Statistical analysis**

The SPSS computer program was used for statistical analysis of the results. Values were expressed as mean± S.E.. Statistical comparison between groups was done by using one way ANOVA. Differences were considered significant at \( P < 0.05 \).

**Results**

The results of the present study revealed that exposure of rats to \( \text{CCl}_4 \) and/or \( \gamma \)-radiation induced a significant increase \( (P < 0.05) \) in hepatic MDA associated with a significant decrease \( (P < 0.05) \) in GSH content as compared to control values. Oral supplementation with \( S^+ \) induced significant amelioration of the changes induced by \( \text{CCl}_4 \) or irradiation as well as their combination (Table 1).

**TABLE 1. Changes in liver MDA and GSH levels of adult male albino rats in different groups.**

<table>
<thead>
<tr>
<th>Groups</th>
<th>MDA (( \mu \text{mol/g tissue} ))</th>
<th>GSH (mg/g tissue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>125.40± 6.06</td>
<td>2.64± 0.08</td>
</tr>
<tr>
<td>( \text{CCl}_4 )</td>
<td>170.80± 5.67&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>1.95± 0.07&lt;sup&gt;ad&lt;/sup&gt;</td>
</tr>
<tr>
<td>( S^+ ) plus ( \text{CCl}_4 )</td>
<td>138.00± 5.91&lt;sup&gt;bd&lt;/sup&gt;</td>
<td>2.48± 0.05&lt;sup&gt;bc&lt;/sup&gt;</td>
</tr>
<tr>
<td>IRR</td>
<td>146.80± 4.91&lt;sup&gt;abc&lt;/sup&gt;</td>
<td>1.93± 0.08&lt;sup&gt;ab&lt;/sup&gt;</td>
</tr>
<tr>
<td>( S^+ ) plus IRR</td>
<td>132.80± 4.07&lt;sup&gt;bd&lt;/sup&gt;</td>
<td>2.45± 0.07&lt;sup&gt;ad&lt;/sup&gt;</td>
</tr>
<tr>
<td>( \text{CCl}_4 ) plus IRR</td>
<td>175.80± 4.37&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>1.16± 0.04&lt;sup&gt;abc&lt;/sup&gt;</td>
</tr>
<tr>
<td>( S^+ ) plus ( \text{CCl}_4 ) plus IRR</td>
<td>144.00± 5.45&lt;sup&gt;bc&lt;/sup&gt;</td>
<td>2.13± 0.07&lt;sup&gt;ad&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Data are represented as means± S.E. \( (n=5) \). <sup>a</sup>Significantly different from control. <sup>b</sup>Significantly different from \( \text{CCl}_4 \). <sup>c</sup>Significantly different from irradiated. <sup>d</sup>Significantly different from \( \text{CCl}_4 \) plus irradiation.

As shown in Table 2., the administration of \( \text{CCl}_4 \) and/or irradiation resulted in a significant increase \( (P < 0.05) \) of serum ALT, AST and ALP activities as compared to control values, indicating liver injury. Oral supplementation of \( S^+ \) induced significant decrease in the activity of these enzymes in \( \text{CCl}_4 \), irradiated, or dual treated group.
TABLE 2. Changes in serum ALT, AST and ALP enzyme activities of adult male albino rats in different groups.

<table>
<thead>
<tr>
<th>Groups</th>
<th>ALT (U/L)</th>
<th>AST (U/L)</th>
<th>ALP (U/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>16.78± 0.45</td>
<td>55.86± 1.24</td>
<td>23.40± 0.81</td>
</tr>
<tr>
<td>CCl₄</td>
<td>29.72± 1.70₁</td>
<td>72.60± 1.43₁</td>
<td>34.90± 0.87₁</td>
</tr>
<tr>
<td>S+ plus CCl₄</td>
<td>17.64± 0.50₁</td>
<td>55.96± 0.85₁</td>
<td>22.56± 1.09₁</td>
</tr>
<tr>
<td>IRR</td>
<td>28.12± 0.78₁</td>
<td>71.22± 0.98₁</td>
<td>31.08± 0.68₁</td>
</tr>
<tr>
<td>S+ plus IRR</td>
<td>19.08± 1.15₁</td>
<td>58.80± 1.77₁</td>
<td>25.36± 1.18₁</td>
</tr>
<tr>
<td>CCl₄ plus IRR</td>
<td>34.58± 1.44₁</td>
<td>78.70± 0.66₁</td>
<td>40.4± 1.16₁</td>
</tr>
<tr>
<td>S+ plus CCl₄ plus IRR</td>
<td>21.76± 0.90₁</td>
<td>60.10± 1.14₁</td>
<td>25.38± 1.42₁</td>
</tr>
</tbody>
</table>

Legends as Table 1.

The results in Table 3 showed that exposure of rats to CCl₄ and/ or γ-radiation induced a significant decrease (P< 0.05) in both total T₃ and T₄ and a significant increase (P< 0.05) in TSH level compared to control values. Administration of S+ significantly elevated the levels of T₃ and T₄ and normalized TSH level in the treated groups.

TABLE 3. Changes in serum levels of T₃, T₄ and TSH of adult male albino rats in different groups.

<table>
<thead>
<tr>
<th>Groups</th>
<th>T₃ (ng/dl)</th>
<th>T₄ (µg/dl)</th>
<th>TSH (µIU/ml)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>91.80± 1.91</td>
<td>5.21± 0.09</td>
<td>0.203± 0.012</td>
</tr>
<tr>
<td>CCl₄</td>
<td>77.74± 1.63₁</td>
<td>4.63± 0.11₁</td>
<td>0.288± 0.029₁</td>
</tr>
<tr>
<td>S+ plus CCl₄</td>
<td>89.02± 1.17₁</td>
<td>4.96± 0.18₁</td>
<td>0.216± 0.011₁</td>
</tr>
<tr>
<td>IRR</td>
<td>63.84± 1.21₁</td>
<td>4.58± 0.12₁</td>
<td>0.310± 0.015₁</td>
</tr>
<tr>
<td>S+ plus IRR</td>
<td>88.42± 1.29₁</td>
<td>5.03± 0.08₁</td>
<td>0.232± 0.009₁</td>
</tr>
<tr>
<td>CCl¢ plus IRR</td>
<td>63.44± 1.76₁</td>
<td>3.10± 0.08₁</td>
<td>0.328± 0.024₁</td>
</tr>
<tr>
<td>S+ plus CCl₄ plus IRR</td>
<td>79.16± 2.37₁</td>
<td>4.17± 0.25₁</td>
<td>0.236± 0.019₁</td>
</tr>
</tbody>
</table>

Legends as Table 1.

Table 4. showed a significant decrease (P< 0.05) in glucose level after exposure to both CCl₄ and γ-rays as compared to control value. Oral supplementation of S+ significantly increased (P< 0.05) the glucose level in this group. Whereas, non-significant changes were observed in glucose level in the other groups. A significant elevation (P< 0.05) in triglycerides level was recorded in all groups exposed to CCl₄ and/ or γ-rays, compared to control value. Exposure to CCl₄ alone or combined with irradiation induced a significant increase (P< 0.05) in cholesterol level. Oral supplementation of S+ significantly decreased (P< 0.05) cholesterol and triglycerides levels in these groups to near normal values.

TABLE 4. Changes in serum cholesterol, triglycerides and glucose levels of adult male rats in different groups.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Cholesterol (mg/dl)</th>
<th>Triglycerides (mg/dl)</th>
<th>Glucose (mg/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control</td>
<td>69.40± 2.50</td>
<td>83.80± 3.06</td>
<td>124.60± 3.54</td>
</tr>
<tr>
<td>CCl&lt;sub&gt;4&lt;/sub&gt;</td>
<td>79.00± 2.85&lt;sup&gt;a&lt;/sup&gt;</td>
<td>95.80± 4.13&lt;sup&gt;a&lt;/sup&gt;</td>
<td>117.00± 4.05&lt;sup&gt;cd&lt;/sup&gt;</td>
</tr>
<tr>
<td>S+ plus CCl&lt;sub&gt;4&lt;/sub&gt;</td>
<td>70.80± 3.02&lt;sup&gt;d&lt;/sup&gt;</td>
<td>85.00± 3.02&lt;sup&gt;bcd&lt;/sup&gt;</td>
<td>121.20± 4.86&lt;sup&gt;cd&lt;/sup&gt;</td>
</tr>
<tr>
<td>IRR</td>
<td>78.00± 2.58</td>
<td>99.80± 3.26&lt;sup&gt;d&lt;/sup&gt;</td>
<td>135.80± 5.28&lt;sup&gt;bcd&lt;/sup&gt;</td>
</tr>
<tr>
<td>S+ plus IRR</td>
<td>76.80± 4.35&lt;sup&gt;d&lt;/sup&gt;</td>
<td>85.64± 2.78&lt;sup&gt;bcd&lt;/sup&gt;</td>
<td>130.00± 4.65&lt;sup&gt;bcd&lt;/sup&gt;</td>
</tr>
<tr>
<td>CCl&lt;sub&gt;4&lt;/sub&gt; plus IRR</td>
<td>86.40± 2.29&lt;sup&gt;d&lt;/sup&gt;</td>
<td>101.80± 1.85&lt;sup&gt;d&lt;/sup&gt;</td>
<td>93.80± 1.66&lt;sup&gt;bcd&lt;/sup&gt;</td>
</tr>
<tr>
<td>S+ plus CCl&lt;sub&gt;4&lt;/sub&gt; plus IRR</td>
<td>77.80± 2.56</td>
<td>90.64± 2.65&lt;sup&gt;d&lt;/sup&gt;</td>
<td>115.80± 5.85&lt;sup&gt;cd&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Legends as Table 1.

Discussion

In this study subcutaneous administration of CCl<sub>4</sub> two times a week for three weeks induced significant elevation in the activity of liver marker enzymes; ALT, AST and ALP. Elevated activities of these enzymes are indicative of cellular leakage and loss of functional integrity of liver cell membrane (Rajesh and Latha, 2004) and damage of hepatocyte cells (Shah and Shah, 2012). The additional findings of the elevated levels of hepatic MDA and decreased GSH content in liver corroborated the hepatic toxicity of CCl<sub>4</sub>.

The change in the pituitary-thyroid axis hormones, a decrease in total T<sub>3</sub> and T<sub>4</sub> levels and an increase in TSH may be an outcome of CCl<sub>4</sub>-induced hepatotoxicity (Itoh et al., 1989, Jatwa and Kar, 2008 and Khan et al., 2011). Significant decrease in total T<sub>3</sub> level was also reported in patients with various hepatic disorders (Moustafa et al., 2009). According to Costa et al. (2001), Torlak et al. (2007) and Khan et al. (2011) serum total T<sub>4</sub> entirely originate from thyroid gland, while more than 80% of total T<sub>3</sub> is produced by deiodination of T<sub>4</sub> in other tissues, especially the liver and kidney through iodothyronine 5'-monodeiodinase enzyme. In particular, the hepatic enzyme is thought to be responsible for the major part of peripheral total T<sub>3</sub> production. It was established that CCl<sub>4</sub>-induces liver injuries in rat (Ashry, 2008 and Sreelatha et al., 2009), reduction in serum T<sub>3</sub> level could partly be due to the decreased conversion of T<sub>4</sub> to T<sub>3</sub> on account of low activity of iodothyronine 5'-monodeiodinase enzyme (Jatwa and Kar, 2008). Furthermore, T<sub>3</sub> concentration appears to parallel the severity of liver dysfunction (Moustafa et al., 2009). However, injuries of CCl<sub>4</sub> metabolites leading to dysfunction of thyroid cannot be excluded (Khan et al., 2011). It could be suggested that the disturbances in thyroid function or thyroid hormone levels in blood after CCl<sub>4</sub> administration may be secondary to hepatotoxicity of CCl<sub>4</sub>.

CCL₄ hepatotoxicity derives from its metabolic activation in the liver. Adewole et al. (2007) explained that CCL₄ is metabolized by cytochrome P450 2E1 to trichloromethyl radical (CCL₃⁺). The CCL₃⁺ and its highly reactive derivative, the trichloromethyl peroxyl radical (C(I3)COO⁻), are assumed to initiate free radical-mediated lipid peroxidation leading to accumulation of lipid peroxidation products that causes hepatic and renal injuries. These radicals are capable of initiating a chain of lipid peroxidation reactions by abstracting hydrogen from polyunsaturated fatty acids (PUFA). Peroxidation of lipids, particularly those containing PUFA, can dramatically change the properties of biological membranes at cellular and subcellular levels. These changes lead to hepatocyte destruction and release of intracellular enzymes; ALT, AST and ALP as observed in this study. This phenomenon results in the generation of reactive oxygen species (ROS), (like superoxide anion O₂⁻, H₂O₂ and hydroxyl radical OH⁻). Therefore, the elevation of hepatic lipid peroxidation observed in this study could be an outcome of CCL₄-induced cellular damage. It could also be due to the decreased GSH level as observed in this study as well as endogenous antioxidant enzymes, SOD, CAT and GPX (Ashry, 2008 and Chiu et al., 2012). The decreased GSH level after CCL₄ administration might be due to its increased utilization by the hepatocytes in scavenging toxic radicals of CCL₄ (Bhadauria et al., 2008). On the other hand, ionizing radiation induced lipid peroxidation through the generation of ROS which attack the PUFA constituent of the cell membrane and other cell components (Zahran et al., 2006). The increase in ALT, AST and ALP activities in serum-observed in this study reflects an increase of plasma membrane permeability, which may be associated with cell death (Gharib, 2007). In this study, γ-irradiation induced non-significant increase in total cholesterol level and significant increase in triglycerides levels, which runs in agreement with the results of Ashry et al. (2009) and Ashry et al. (2010). They attributed this increase to the suppression of lipoprotein lipase activity that reduces the uptake of lipids by adipose cells in addition to decreased fatty acid oxidation and increased rate of cholesterol biosynthesis in the liver and other tissues.

The thyroid status disturbance-observed after irradiation-seemed to be due to both inhibited T₄ and T₃ biosynthesis in thyroid and disturbed hormone peripheral metabolism under radiation exposure (Nadol'nik et al., 2004).
Consequently, TSH level in serum increased - as observed in this result-in order to activate the thyroid gland to increase its production of T3 and T4 (Abdel Fattah et al., 2003). From the above results, the extent of liver damage and thyroid hormone disturbances upon simultaneous CCl4 and radiation exposure is greater than that caused by each of them alone.

The significant decrease in glucose level observed in this work can be discussed on the bases that, thyroid hormones stimulate almost all aspects of carbohydrate metabolism, decreasing of these hormones under the effect of free radicals generated from xenobiotics reduced hepatic glucose uptake (Arai et al., 2010). Moreover, studies have demonstrated decreased hepatic glycogen content after treatment with CCl4 (Morsy et al., 2002 and Muriel et al., 2001), reflecting decreased gluconeogenesis by the liver (Althnaian et al., 2013). Furthermore, hypoglycaemia was observed previously in liver cirrhotic rats (Mion et al., 1996). As well as, all aspect of fat metabolism are also enhanced under the influence of thyroid hormones. Decreases of thyroid hormones secretion increase the plasma concentration of cholesterol and triglycerides and almost always cause excessive deposition of fat in the liver as well. In addition intracellular T3 acts as an antioxidant by reducing ROS accumulation through producing the NADPH required for regeneration of reduced GSH, a potent endogenous antioxidant (Moustafa et al., 2009).

Oral supplementation of S+ during exposure to CCl4 and/ or γ-rays ameliorated the disturbances in oxidative stress in liver tissue and decreased the activity of liver enzymes and consequently modulated the disturbances in pituitary-thyroid axis hormones, decreased cholesterol and triglycerides and increased glucose levels in serum. This ameliorating effect of S+ observed in this study is attributed to its components; mainly silymarin which have the ability to reduce membrane permeability and restore impaired liver function (Ozturk et al., 2012 ), acetyl cysteine which exerts its hepato-protective effect by counteracting accumulated ROS in the liver through its antioxidant properties and a GSH precursor (Liu et al., 2007), zinc which plays an important role in treatment of liver injury due to its catalytic, structural and regulatory role in a large number of enzymes (Fatimah and Mahboob, 2012), vitamin C which is a major water-soluble antioxidant is believed to decrease lipid peroxidation either directly or indirectly by regenerating vitamin E (Adikwu and Deo, 2013), vitamin E which prevents ROS damage in PUFA as a liposoluble antioxidant acting against damage caused to phospholipids as a membrane-stabilizing agent (UBOH et al., 2013).
The ameliorating effect of S+ on thyroid hormones may be due to the activation of 5’-deiodinase enzyme in the liver which stimulates conversion of T4 to T3. It is well known that deiodinases contain Se in the form of the amino acid selenocystein located at their active sites, so injection of an antioxidant drug including Se may cause changes in deiodinases activities (Abdel-Fattah et al., 2003). Also vitamin E, a lipid soluble antioxidant in the cell membrane, could increase the release of stored T4 through the membrane of thyroid cells into the blood (Abdel-Fattah et al., 2003). The benefit of S+ might be attributed to the synergistic effect of the antioxidants content therein which act as free radical scavengers and lipid peroxidation inhibitor, acting thus to prevent the membrane permeability changes especially those of the hepatocyte membrane transport, thereby improving the xenobiotics induced changes in liver function.

According to the results obtained in the current study, oxidative damage resulting from the metabolism of CCl4 in the liver as well as exposure to γ-ray exhibit the same effect on liver and pituitary-thyroid axis hormones. As well as, exposure to CCl4 and ionizing radiation exert a synergistic effect on liver injury associated to a hypothyroid state. Treatment of liver injury by hepato-protective drug S+ modulated liver function and consequently modulated the disturbances in pituitary-thyroid axis hormones.

References


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اضطرابات هرمونات محور النخامية-الدرقية في الجرذان
المعرضة لرابع كلوئيد الكربون أو أشعة جاما

ناهد عبد العزيز
قسم بحوث البيولوجيا الإشعاعية، المركز القومي لبحوث وتقنية الإشعاع،
صر. ب. 29 مدينة نصر، مصر.

يهدف هذا البحث إلى دراسة الاضطرابات التي تحدث في هرمونات الغدة النخامية المحفزة لافراز الغدة الدرقية وكذلك هرمونات الغدة الدرقية، من خلال ملاحظة التأثيرات المورثة لارتفاع كلوئيد الكربون عند زراعة الأشعة الجاما. وقد تم فحص الجرذان برابع كلوئيد الكربون لكل جرذان مرتين أسبوعيًا، لمدة ثلاثة أسابيع مع/أو بدون تعرض لمادة جاما المجردة بجرعات قدرها 1 جرعة مرتين أسبوعيًا، لمدة ثلاثة أسابيع. وقد أدى ذلك إلى زيادة مستوى اللاندكان والدهم مصحوباً بنقص في مستوي الجلوكوزات في الكبد. وتم ارتباط الإجهاد التأكسدي الناتج عن الإشعاع زيادة في الالزيمات الدائرة على تلف وظيفة الكبد (اللامينتين ترانس أميناز الانتراتيسفرابز والزيم محور للنخامية). وكذلك أدى إلى نقص في هرمونات الدم المورثة. فيما تم فحص جرذان آخرين لمتابعة تأثيرات الإشعاع.