Effect of Digoxigenin-3-O-rutin isolated from *Trigonella foenum graecum* on T₄-induced hyperthyroidism and serum lipid concentrations

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ABSTRACT

In this study, effect of digoxigenin-3-O-rutin isolated from the seeds of *Trigonella foenum graecum* on thyroid hormones and serum lipid concentrations was evaluated in L-thyroxine (L-T₄)-induced hyperthyroidism in rats. Digoxigenin-3-O-rutin was administered (10mg/kg) to L-thyroxine (L-T₄)-induced hyperthyroidic rats and alterations in the concentrations of serum thyroid hormones, insulin, glucose, hepatic 5′-monodeiodinase (5′DI) and glucose-6-phosphatase (G-6-Pase) activity were analyzed. Antioxidant status was estimated by determining the levels of antioxidative enzymes and lipidperoxidation. L-T₄ (500μg/kg, s.c./d) administration increased the serum levels of thyroxine (T₄), triidothyronine (T₃), glucose, insulin, different lipids, activity of hepatic 5′-DI and G-6-Pase. High lipidperoxidation level was observed both in liver and cardiac tissues with a depletion in cellular antioxidants. On the contrary, test drug (10mg/kg) treatment improved the alterations with respect to hormonal levels, lipid concentrations and lipid peroxidation towards normalcy and enhanced the antioxidant activities. Rats treated with PTU generally gave lower results compared to groups treated with the test drug. The antithyroidic role of the test compound is mediated possibly through the inhibition in 5′DI activity. Improvement in lipid profile by the test drug might have protective effect on cardiovascular health in vivo.

Keywords Digoxigenin-3-O-rutin, hyperthyroidism, 5′DI, serum lipids, insulin.

INTRODUCTION

Cardiac glycosides have a long history of therapeutic use for the treatment of heart-diseases. The relationship between thyroid hormone and the cardiovascular system has been extensively demonstrated in numerous experimental and clinical studies.[1,2] Heart is one of the main target organs for the action of thyroid hormone, and any change in the thyroid hormone status indirectly affects the cardiac function.[3]

Thyrotoxicosis is a term given for the clinical manifestation of hyperthyroidism which can invoke heart and vascular abnormalities through the mechanism at heart muscle cells nuclear level. Also the cardiac contractility, resting heart rate and cardiac output are increased. Hyperthyroidism may cause cardiac complications because of the increase in heart rate, myocardial contractility, oxygen demand and give rise to conditions silent coronary artery disease or compensated heart failure.[4]

Therefore, amelioration of hyperthyroidism is sometimes considered as an important aspect in controlling cardiovascular problems. For its regulation some conventional medicines such as neomarcazole, methimazole and propryl-thiouracil are prescribed. Despite the fact that many patients give preference to herbal drugs that are known to be safe and economic, nothing much has been investigated on cardiac glycoside which can ameliorate hyperthyroidism. Most of the investigations made so far on cardiac glycosides are in relation to the regulation of ischemic stroke, cancer and neurodegenerative diseases.[5,6]

Since natural products are considered as a major source of potential drugs, in the present investigation I isolated and evaluated a novel phytochemical for its potential to...
ameliorate hyperthyroidism and related cardiovascular abnormalities.

*Trigonella foenum gracium* (TFG) seeds have been reported to protect against dyslipidemia, cardiac problems, hypercholesterolemia, and hyperthyroidism. Recently we isolated and reported the cardio-protective potential of digoxigenin-3-O-rutin a novel compound of TFG in isoproterenol induced myocardial infarction. In the present investigation, an attempt has been made to evaluate the effect of digoxigenin-3-O-rutin on T₄-induced hyperthyroidism with reference to changes in serum thyroid hormones, hepatic 5'DI activity, lipids, antioxidative enzymes, blood glucose and insulin levels, keeping in mind the association of CVD and thyroid problems.

**EXPERIMENTAL**

**Chemicals**

L-thyroxine (L-T₄) was purchased from Sigma Chemical Co. Ltd. St. Louise, USA. Radioimmunoassay (RIA) kits for the estimation of serum T₄, T₃, and Insulin were supplied by Bhabha Atomic Research Center. Assay kits for different lipids and glucose were purchased from Ranbaxy Pvt. Ltd., Mumbai, India, thiobarbituric acid, sodium dodecyl sulphate, sulphuric acid and ethylene diamine tetra-acetic acid were obtained from E. Merck (India) Ltd., Mumbai, India.

**Plant material and extraction**

Dried fenugreek seeds (1kg) were finely powdered and subjected to 70% ethanolic extraction in a soxhlet apparatus at 60°C for 12h. The extract was vacuum dried to obtain it in ethanol-free powdered and was then processed for the isolation of cardenolide following as done in our laboratory previously and the modified method of Lei et al.[12,13] In brief, it was partitioned between n-hexane and water and the aqueous layer (110g) was subjected to silica gel chromatography followed by elution with water and 40% MeOH. The elute (14g) was subjected to column chromatography using sephadex LH-20 and silica gel to obtain the final compound (25mg).

**Animals**

Experiment was carried out in Wistar albino rats, weighing 190–200gm, housed in a standard photoperiod (14h light: 10h dark) and temperature (27 ± 1°C) controlled room with the provision of laboratory feed (Gold Mohur feed, Hindustan Lever Limited, Mumbai, India) and water ad libitum. Ethical guidelines of the Committee for the Purpose of Control and Supervision on Experiments in Animals, Ministry of Social Justice and Empowerment, Government of India, (Regd. No. 779/2012-13) were followed.

**Experimental design**

Thirty five healthy rats were divided in to five groups of seven each. While Group I animals receiving distilled water (0.1ml/day/animal) served as control, animals of group II, III, IV and V were made hyperthyroidic by administering L-T₄ (500µg/kg, s.c.) for 12 consecutive days as done earlier. After 12 days of thyroxine treatment, group II animals, serving as hyperthyroidic control were administered with distilled water while animals of group III and IV received two different concentrations of digoxigenin-3-O-rutin dissolved in distilled water (5.0 and 10.0mg/kg, p.o) respectively. Group V was administered with 10mg/kg of PTU. The administered doses of digoxigenin-3-O-rutin and L-T₄ and PTU were taken from our earlier studies.[11,12,14] Experiment was continued for 4 weeks and then terminated. On the day of termination over night fasted animals were sacrificed by cervical decapitation and blood from each animal was collected, and serum was separated for the estimations of thyroid hormones and lipids.

**Biochemical estimations**

After exsanguinations, heart and liver of each animal were removed quickly, washed and homogenized with phosphate buffered saline (PBS, pH 7.4). The homogenates were centrifuged at 17,000g for 30 min at 4°C and the supernatant was used for biochemical estimations including lipid peroxidation (LPO), superoxide dismutase (SOD), catalase (CAT), and reduced glutathione (GSH) and glutathione peroxidase (GPxs).

**Radioimmunoassay of thyroid hormones and insulin**

Total circulating T₃ and T₄, hepatic 5’-DI and insulin were estimated by radioimmunoassay (RIA) in serum samples following the protocol provided in the RIA kits supplied by Bhabha Atomic Research Centre (BARC), Mumbai, India, as done routinely in our laboratory.[16,17] Intra-assay variations for T₄ and T₃ were <5% and <1%, respectively, and for insulin assay it was <4.9%.

**Estimation of marker enzymes, lipids and glucose**

Different serum lipids were estimated using standard commercial kits. Low-density lipoprotein
choline (LDL-C) and very low-density lipoprotein cholesterol (VLDL-C) were calculated using the formula of Friedwald et al.\textsuperscript{[18]} Serum glucose concentration was measured by the glucose oxidase/peroxidase method of Trinder.\textsuperscript{[19]}

**Study of lipid peroxidation (LPO)**

Tissue lipid peroxide level in heart was measured by the method of Ohkawa et al.\textsuperscript{[20]} and finally LPO was expressed as nM of MDA formed/h/mg protein. The levels of lipid hydro peroxide (LOOH) were measured by the method of Jiang et al.\textsuperscript{[21]}

The activities of antioxidants such as catalase, superoxide dismutase (SOD), glutathione peroxidase (GPx) and reduced glutathione (GSH) levels were assayed by the methods of Marklund and Marklund,\textsuperscript{[22]} Aebi,\textsuperscript{[23]} Rotruck et al.\textsuperscript{[24]} and Ellman\textsuperscript{[25]} respectively. G-6-Pase was assayed by the inorganic phosphate release method as described by Baginski et al.\textsuperscript{[26]} Protein content was determined following our routine method of Lowry et al.\textsuperscript{[27]}

**Statistical analysis**

All values were expressed as mean±S.E.M. Differences in mean values were compared using version Prism 4 software for windows, Inc., La jolla, CA, USA and by one way analysis of variance (ANOVA) followed by post hoc Newman-Keuls multiple comparison tests. \( P<0.05 \) was considered as statistically significant.

**RESULTS**

Digoxigenin-3-O-rutin, obtained as yellow crystalline powder and the molecular formula was assigned as C\(_{56}\)H\(_{72}\)O\(_{25}\) which was deduced from ESI. The isolated compound revealed its UV, IR and NMR data consistent with our earlier report\textsuperscript{[10]} (Fig 1).

**Effects on serum concentration of thyroid hormones**

The effects of L-T\(_4\) in serum T\(_3\), T\(_4\), hepatic 5’DI, G-6-pase activity and body weight are shown in Fig. 2. In T\(_4\) treated rats T\(_3\), T\(_4\), hepatic 5’DI, G-6-pase activity enhanced significantly \( P<0.001 \) as compared to control values. A significant decrease \( P<0.001 \) in body weight was observed in T\(_4\)-induced animals as compared to control. Administration of the test drug 5mg/kg to hyperthyroid animals significantly decreased the T\(_3\), hepatic 5’DI, G-6-pase activity \( (P<0.001, P<0.01 \) and \( P<0.05 \) respectively) as compared to T\(_4\) treated animals. However, at 10mg/kg the test drug administration significantly decreased T\(_3\), T\(_4\), hepatic 5’DI, G-6-pase activity with an increase in body weight (\( P<0.001 \) or \( P<0.01 \)) as compared to T\(_4\)-induced animals. In PTU+T\(_4\) treated group also thyroid hormones decreased significantly (\( P<0.001 \) or \( P<0.01 \) as compared to T\(_4\)-induced animals).

![Figure 2](image-url). Changes in concentrations of serum T\(_3\) (ng/ml), T\(_4\) (ng/ml X 10), 5’DI (ng/ml), G-6-Pase (\( \mu \)M of inorganic phosphate liberated/h/mg protein\( \times 10^{-3} \)), % increase in body wt. following the administration of digoxigenin-3-O-rutin (5.0 and 10.0mg/kg/d) to the L-T\(_4\)-induced animals. Each vertical bar represents the mean±S.E.M. (n=7); \( ^{1}P<0.001 \) as compared to the respective control value, whereas \( ^{2}P<0.001 \), \( ^{3}P<0.01 \) and \( ^{4}P<0.05 \) as compared to the respective value of thyroxine treated animals.

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**Figure 1.** The structure of the isolated compound, derived from UV, IR, NMR and Mass spectra, which was identified as digoxigenin-3-O-rutin.
Effects on serum glucose, insulin and lipid levels

T<sub>4</sub>-induced rats revealed a significant decrease in TG, TC, LDL-C, HDL-C and VLDL-C (P<0.001 or P<0.01 or P<0.05 as compared to control animals (Table 1). Treatment with of 10.0mg/kg the test drug to hyperthyroid animals significantly (P<0.001 increased HDL-C and rest of the lipids were nearly to normal values. At the dose of 5.0mg/kg only cholesterol and triglycerides levels were increased significantly (P<0.001 and P<0.05 as compared to T<sub>4</sub>-induced animals).

The test drug at 5.0mg/kg and 10mg/kg decreased both serum glucose and insulin levels significantly (P<0.001, P<0.01; P<0.001 respectively, as compared to T<sub>4</sub>-induced animals). In T<sub>4</sub>+PTU treated group TC and TG increased significantly (P<0.001, P<0.01 respectively as compared to T<sub>4</sub>-treated animals). Serum glucose and insulin levels were also decreased significantly (P<0.001, P<0.01 respectively as compared to T<sub>4</sub>-treated animals) (Table 1).

Effects on LPO and antioxidants

As shown in table 2, the amount of MDA and LOOH was significantly increased in cardiac tissue in T<sub>4</sub>-treated animals (P<0.01 as compared to control animals) and a significant decrease in SOD, CAT, GPx and total GSH content (P<0.001 as compared to control animals was observed. In hepatic tissues also a significant increase in LPO and LOOH levels with a decrease in antioxidants were observed in T<sub>4</sub>-induced animals (P<0.001 or P<0.01 or P<0.05) as compared to normal controls. The antioxidant levels in hepatic tissues were increased significantly following the administration of 10mg/kg of the test drug (P<0.001 or P<0.01 or P<0.05) as compared to T<sub>4</sub> alone treated animals (Table 3). In 5mg/kg of digoxigenin-3-O-rutin and PTU+T<sub>4</sub>-treated group GSH remain unaltered in cardiac and hepatic tissues other antioxidative enzymes were enhanced significantly (P<0.001 or P<0.05). The dose of 10mg/kg found to be highly effective in decreasing MDA and LOOH levels and increasing SOD, CAT, GPx and total GSH (P<0.001 or P<0.01 as compared to T<sub>4</sub>-induced animals).

**DISCUSSION**

A significant increase in serum T<sub>3</sub>, T<sub>4</sub> concentrations and 5'DI activity in L-T<sub>4</sub> induced animals indicates clearly the hyperthyroidic condition as has been observed by us previously.[16,17] Interestingly, following the administration of digoxigenin-3-O-rutin in hyperthyroid

### Table 1. Effects of digoxigenin-3-O-rutin (TD-5, 10mg/kg and T<sub>4</sub>+PTU) in the alterations in serum concentrations of total cholesterol (TC), low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C), very low density lipoprotein cholesterol (VLDL-C), and triglyceride (TG), all expressed in mg/dl, serum insulin (IU/ml) and serum glucose concentration (mg/dl) in thyroxine (T<sub>4</sub>) treated rats.

<table>
<thead>
<tr>
<th></th>
<th>CHOL</th>
<th>HDL</th>
<th>TG</th>
<th>VLDL</th>
<th>LDL</th>
<th>Glucose</th>
<th>Insulin</th>
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<tbody>
<tr>
<td>CTRL</td>
<td>98.97±3.85</td>
<td>40.29±2.16</td>
<td>85.96±4.39</td>
<td>17.09±1.14</td>
<td>41.12±2.99</td>
<td>96.36±3.48</td>
<td>6.85±0.27</td>
</tr>
<tr>
<td>T&lt;sub&gt;4&lt;/sub&gt;</td>
<td>59.35±3.19&lt;sup&gt;a&lt;/sup&gt;</td>
<td>24.54±1.71&lt;sup&gt;a&lt;/sup&gt;</td>
<td>61.17±3.94</td>
<td>12.23±1.99&lt;sup&gt;a&lt;/sup&gt;</td>
<td>23.14±1.94&lt;sup&gt;a&lt;/sup&gt;</td>
<td>168.00±4.35&lt;sup&gt;a&lt;/sup&gt;</td>
<td>12.26±0.69&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>T&lt;sub&gt;4&lt;/sub&gt;+TD(5mg)</td>
<td>92.03±4.63&lt;sup&gt;a&lt;/sup&gt;</td>
<td>30.96±3.68</td>
<td>89.02±5.16&lt;sup&gt;a&lt;/sup&gt;</td>
<td>17.80±1.76</td>
<td>45.28±3.11&lt;sup&gt;a&lt;/sup&gt;</td>
<td>132.24±5.67&lt;sup&gt;a&lt;/sup&gt;</td>
<td>8.02±0.37&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>T&lt;sub&gt;4&lt;/sub&gt;+TD(10mg)</td>
<td>99.34±5.52&lt;sup&gt;a&lt;/sup&gt;</td>
<td>63.78±5.02&lt;sup&gt;a&lt;/sup&gt;</td>
<td>80.46±3.69&lt;sup&gt;a&lt;/sup&gt;</td>
<td>16.09±1.09&lt;sup&gt;a&lt;/sup&gt;</td>
<td>20.92±0.79</td>
<td>112.24±3.43&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6.52±0.44&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>T&lt;sub&gt;4&lt;/sub&gt;+PTU(10mg)</td>
<td>97.07±6.16&lt;sup&gt;a&lt;/sup&gt;</td>
<td>38.67±3.99</td>
<td>83.78±5.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>16.75±1.46</td>
<td>43.66±2.17</td>
<td>102.68±5.08&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7.99±0.76&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
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</table>

Data are mean±S.E.M. (n=7). *P<0.001, **P<0.01 and *P<0.05 as compared to their respective control values; whereas *P<0.001, **P<0.01 and *P<0.05 as compared to the respective value of thyroxine treated animals. TD=test drug.

### Table 2. Alterations in lipid peroxidation and antioxidants following T<sub>4</sub> (500µg/kg), T<sub>4</sub>+TD(5mg/kg), T<sub>4</sub>+TD (10mg/kg) in the cardiac tissues of rats.

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>T&lt;sub&gt;4&lt;/sub&gt;</th>
<th>T&lt;sub&gt;4&lt;/sub&gt;+TD 5.0mg</th>
<th>T&lt;sub&gt;4&lt;/sub&gt;+TD 10.0mg</th>
<th>T&lt;sub&gt;4&lt;/sub&gt;+PTU</th>
</tr>
</thead>
<tbody>
<tr>
<td>LPO (nmol MDA formed/h/mg protein)</td>
<td>0.87±0.06</td>
<td>1.94±0.08&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.29±0.09&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0.67±0.05&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.16±0.04&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>LOOH (nmol/mg protein)</td>
<td>3.02±0.67</td>
<td>7.62±1.05&lt;sup&gt;a&lt;/sup&gt;</td>
<td>5.67±0.44&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.10±0.78&lt;sup&gt;a&lt;/sup&gt;</td>
<td>5.99±0.57</td>
</tr>
<tr>
<td>SOD (U/mg protein)</td>
<td>6.16±0.32</td>
<td>2.29±0.23&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.76±0.34&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7.49±0.52&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.03±0.46</td>
</tr>
<tr>
<td>CAT (μmoles of H&lt;sub&gt;2&lt;/sub&gt;O&lt;sub&gt;2&lt;/sub&gt; decomposed/ min/mg protein)</td>
<td>21.21±3.50</td>
<td>12.70±2.30&lt;sup&gt;a&lt;/sup&gt;</td>
<td>19.72±2.11&lt;sup&gt;a&lt;/sup&gt;</td>
<td>26.14±3.69&lt;sup&gt;a&lt;/sup&gt;</td>
<td>20.14±1.74&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>GSH (μmoles GSH/mg protein)</td>
<td>6.99±0.57</td>
<td>3.06±0.47&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.85±0.26</td>
<td>6.09±0.42&lt;sup&gt;a&lt;/sup&gt;</td>
<td>3.64±0.51</td>
</tr>
<tr>
<td>GPx (μmoles of GSH oxidized/mg protein)</td>
<td>6.27±0.37</td>
<td>3.14±0.23&lt;sup&gt;a&lt;/sup&gt;</td>
<td>5.97±0.48&lt;sup&gt;a&lt;/sup&gt;</td>
<td>7.93±0.96&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6.01±0.55&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
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</table>

Data are mean±SEM, n=7. *P<0.001 and **P<0.01, as compared to the respective control values. *P<0.001, **P<0.01, *P<0.05, as compared to the respective values of the T<sub>4</sub>-induced animals.
animals could decrease the T₃ concentration and 5’DI activity, suggesting the inhibition of peripheral mono-deiodination of T₄, the principal source of generation of T₃. Thus it is presumed that inhibition of 5’DI activity is likely to be responsible for hypothyroidic action of the test drug. This finding corroborate with our earlier observation on crude seed extract of *Trigonella foenum graecum* that had indicated its thyro-inhibitory action in mice.[31]

Hyperthyroidism is characterized by decreased body weight, increased insulin and glucose indicative of insulin resistance as well as decreases in plasma lipids such as plasma cholesterol and triglycerides.[32,33] Thyroxine (T₄) stimulates hepatic gluconeogenesis, and even a small change in T₄ levels can affect glucose metabolism.[34] In the present study, following exogenous L-T₄ administration leads to enhanced glucose levels in this condition may be explained by increased endogenous glucose production through more rapid glycogenolysis and gluconeogenesis.[35] The hyperthyroid rats were also found to be insulin resistant, with increased insulin levels. Interestingly, the administration of test compound to hyperthyroid animals normalized the serum insulin and glucose levels. This reduction could be the result of the direct effects of thyroid hormones on the β-cells for insulin secretion as suggested earlier.[36]

A significant reduction in hepatic-glucose-6-phosphatase activity following the digoxigenin-3-O-rutin administration further supports the inhibitory role in T₃ formation as the activity of this enzyme is commonly related to the alteration of the thyroid hormone levels.

Hyperthyroidism is generally associated with a loss in body weight. In the present study a decrease in body weight in hyperthyroid animals and an increase in test drug treated animals were observed. Since body weight changes are very often associated with the alterations in T₃ level, these observations further support the T₃ inhibitory nature of the test drug.[37]

Thyroid hormones play an important role in regulating lipid metabolism, and thyroid dysfunction can result in lipid abnormalities which increase the risk of endothelial dysfunction, hypertension and cardiovascular disease. Thyroid dysfunction has a great impact on lipids as well as a number of other cardiovascular risk factors. A significant decrease in the level of TC, TG, HDL-C, LDL-C and VLDL-C was observed in L-T₃-induced rats.[38] The reduction in total cholesterol due to thyroxine treatment was due to decrease in HDL and LDL cholesterol.[39,40]

However, treatment with the isolated compound normalized most of these changes induced by hyperthyroidism. Interestingly, 10mg/kg of digoxigenin-3-O-rutin increased HDL-C levels significantly as compared to that of hyperthyroidic animals maintaining the LDL-C nearly to normal values. This increase in HDL-C might also be the result of its antithyroidic property, as T₃ is believed to reduce serum HDL levels.[35,36] Thus an increase in HDL-C following the treatment with digoxigenin-3-O-rutin, suggests its beneficial role in the regulation of hyperthyroid-induced cardiovascular problems.

Most of the available data indicates that an experimentally induced hyperthyroid state causes an increased radical production.[37,38]

It has been reported that the increase in reactive oxygen species induced by thyroid hormone leads to an oxidative stress condition in liver, cardiac and some skeletal muscles with a consequent lipid peroxidative response.[39,40] Hyperthyroidism is a hyper metabolic state accompanied by increased oxygen utilization, increased production of reactive oxygen species and consequently measurable changes in anti oxidative factors.[41,42]
In this study also T₃ administration enhanced MDA level in both the hepatic and cardiac tissues and antioxidants such as SOD, CAT, GPx and total GSH were found to be decreased. The increase in lipid peroxidation may be due to increased free radical production. These observations are also in agreement with previous findings. Interestingly, the isolated compound at the dose of 10mg/kg prevented the oxidative damage in both the cardiac and hepatic tissues with an increase in antioxidants in hyperthyroid rats suggesting its free radical scavenging activity.

CONCLUSIONS

In conclusion the present investigation suggests that the isolated compound, digoxigenin-3-O-rutin from fenugreek seeds appears to attenuate oxidative stress, not only by fortifying antioxidant defense capacity but also by lowering lipid peroxidation and helped to maintain the levels of lipids in the serum. The antithyroidal activity exhibited by the compound is through the inhibition of 5’DI activity.

CONFLICT OF INTEREST

Author declares no conflict of interest.

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