

Original Article

BENEFICIAL EFFECTS OF ETHANOLIC LEAF EXTRACT OF *CORIANDRUM SATIVUM* ON METANIL YELLOW INDUCED ALTERATION INACTIVITY OF CATALASE AND LEVEL OF LIPID PEROXIDATION IN HERCINE CARDIAC TISSUE *IN VITRO*

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ABSTRACT

Objective: The primary aim of the study was to find out a suitable effective dose of metanil yellow that can cause alterations of lipid peroxidation and catalase activity in the heart *in vitro* and to determine the effectiveness of graded doses of *Coriandrum sativum* as an antioxidant in preventing lipid peroxidative damage to cardiac tissue and back up catalase activity.

Methods: Metanil yellow at different doses (10, 25, 35, 50 μ l in phosphate buffer) were applied on isolated hircine heart homogenate (10%) *in vitro* followed by estimation of lipid peroxidation and catalase activity after completion of the incubation period. Another set of the experiment included incubation of the heart homogenates with graded dilutions of ethanolic leaf extracts of *Coriandrum sativum* (CsEth) at concentrations of 10, 20, 30, 40 μ l in phosphate buffer medium followed by quantification of the same biochemical parameters as above. This was followed by co-administration of the selected effective dose of metanil yellow (35 μ l) and ethanolic leaf extract of *Coriandrum sativum* (30 μ l) on heart homogenate to track the variation in lipid peroxidation and catalase activity in heart homogenate *in vitro*.

Results: Metanil yellow significantly lowered catalase activity in isolated cardiac tissue that was reflected in elevated lipid peroxidation assuming free radical induced oxidative injury to cardiac tissue ($p < 0.01$). However ethanolic leaf extract of *Coriandrum sativum* buffered such changes ($p < 0.01$) significantly, presuming a protective action against oxidative cardiac damage.

Conclusion: It may be concluded that ethanolic leaf extract of *Coriandrum sativum* is a promising cardioprotective remedy not only in mitigating free radical mediated lipid peroxidative damage to cardiac tissue but also in reinstating normal catalase activity that may be used as a unique herbal based remedy for such cardiac ailments in future.

Keywords: Cardioprotection, *Coriandrum sativum*, Metanil yellow, Methanolic leaf extract, Myocardial infarction

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INTRODUCTION

Reactive oxygen species (ROS) are an important group of molecules that are generated usually by oxygen slippage either during mitochondrial electron transport or extra-mitochondrial electron transport systems [1]. An imbalance between the generation of such free radicals and scavenging activity of these free radical by cellular antioxidant enzyme cascade may be a cause for generation of oxidative stress. Oxidative stress may be the etiology of a multitude of ailments ranging from neurological, haematological, immunological, cardiovascular, and genetic to developmental disorders [2].

Mammalian heart is highly aerobic and oxygen demanding organ. In addition, cardiac tissue is normally dynamic with highly metabolic demand and mitochondrial rich myocardial work out may be a source of oxygen slippage turning out as oxidative injury to the heart. ROS generation in the heart may affect normal myocardial contractility induce maladaptive myocardial remodelling and subsequently, heart failure may be an outcome [3].

Cellular antioxidant mechanisms work in cascade in the heart to combat the stress mediated cardiac injury. Formerly superoxide anion generation are prevented by vitamin E, formation of hydrogen peroxides from superoxide radicals are again checked by sulphur containing amino acids like cysteine and methionine, and later generation of hydroxyl radicals are counteracted by coordinated actions by superoxide dismutase and catalase which otherwise may interact with polyphenolic lipids to form lipid hydroxides, a product of lipid peroxidation. Although SOD and glutathione peroxidase are adequate in cardiac tissue to combat oxidative damage, catalase is very crucial for H₂O₂ detoxification in cardiac myocytes [4].

Amongst the spectrum of sources for free radical mediated oxidative stress, changing lifestyle pattern nowadays may be a good booster of oxidative stress induced systemic disorders [5]. Eye attracting coloured foods consumption in our country like roadside cheap and hotel made biriyani, mango and milk shakes, cheese and sweets, laddoos has become a common fashion amongst inhabitants belonging to low and medium socio-economic status in metropolitan cities. Such foods are generally adulterated with metanil yellow (as observed from our laboratory studies) [6]. Consumption of such food adulterant may alter a metabolic pattern that may account for the generation of free radicals leading to oxidative stress [7].

Antioxidant supplementation is widely followed the regime to combat cellular oxidative insult. Amongst these groups, synthetic antioxidant supplementation has been criticized by many and shifting of therapy towards the use of indigenous plant-based chemicals (including polyphenols, flavonoids, phenolic acids, tannins etc.) is recently gaining recognition [8].

One such plant widely used by Indians in diet, popular for its delicious tastemaker is the annual herb, *Coriandrum sativum*.

Apart from raw consumption of its leaves, seeds of this plant are also used as common Indian spice. Essential oils extracted from leaves, seeds, fruits etc. is used as folk medicine for curing bacterial, fungal diseases and antioxidant therapy [9]. *Coriandrum* leaves have been reported to have antidiabetic, anti ulcer, anti-arthritis and several other medicinal uses [10]. Studies showed that *Coriandrum* leaves extract has anticancer activity [11]. Studies also showed that reversal of memory deficit in mice model is possible using *Coriandrum* leaves extract [12]. *Coriandrum sativum* leaves extract has been found to have radical scavenging potential and it has the

ability to protect DNA damage [13]. *Coriandrum sativum* leaves extract has been reported to scavenge DPPH and has antibacterial [14]. Our earlier studies reveal that methanolic extract of the leaves of *Coriandrum sativum* has hepatoprotective effect *in vitro* [5]. However cardioprotective effects of ethanolic leaf extract of this plant is yet to be scrupulously determined.

One of the principal aims of this study was to explore the possible effects of metanil yellow on H₂O₂ metabolism by monitoring catalase activity which may partially anticipate the mechanism of activation of lipid peroxidation in cardiac tissue *in vitro*. The secondary aspect of this study was to find out an effective dose of this drug in producing such oxidative insult.

MATERIALS AND METHODS

Collection of *Coriandrum sativum* plant

The shoots of *Coriandrum sativum* were collected from the local market. Plant along with its seeds was collected from Chinsurah Hooghly, West Bengal, herbarium was prepared and the specimen was identified by a concerned expert from the Botanical Survey of India, Central National Herbarium as *Coriandrum sativum* [Specimen voucher number is HMC/SG/PHYSIO-02].

Processing of the leaves

Fresh leaves were isolated from the plant, washed and dried on filter paper at normal room temperature. The dried leaves were weighed. The leaves were then placed in a hot air oven maintained at 50 °C until they became crispy and dry. The dried leaves were then grinded in mortar and pestle to dry powder. The powder was then stored in air tight sealed container until further use. For ethanolic extract formation, 0.1g dried powder was dissolved in 20 ml of ethanol and kept for 30 min which was then filtered. This process of extraction was practiced freshly for each study.

Physical examination

The colour and constituency of the dried powder were monitored regularly. pH of the ethanolic leaf extract was determined using a digital pH meter before use by measuring the pH of a standard buffer solution of pH 9 and another of pH 3 respectively.

Relative moisture content was measured by the following protocol:

Fresh weight (FW) of the leaves was measured followed by placement in 9 cm Petri dish containing distilled water; maintained as such for 24 h at 4 °C in the dark. The leaves were then blotted dry and the turgor fresh weight (TW) was determined. Subsequently, the leaves were dried at 70 °C for 24 h. Finally, the dry weight (DW) was measured [Barr HD]. The relative water content was finally calculated by using the equation:

$$RWC (\%) = [(W - DW)/(TW - DW)] \times 100$$

Where,

W-sample fresh weight

TW-sample turgid weight

DW-sample dry weight

Chemical examination (Qualitative screening)

Saponin content of the leaves extract were estimated by the methods of Kuganathan *et al.* 2008 and Srivastava *et al.*, 2010. [16, 17], tannins and phenols content of the leaves extract were estimated by the method of Srivastava *et al.*, 2010 [17], alkaloids content of the leaves extract were estimated by using Mayer's, Dragendorff's, Hager's and Wagner's reagents, gum and mucilage and flavonoids content of the leaves extract were estimated by the method of Srivastava *et al.*, 2010 [17].

Metanil yellow solution preparation (MY)

1 mg metanil yellow (Sigma brand) was dissolved in distilled water at 1:1 (weight/volume, i. e, 1 mg/ml).

Procuration of goat heart

Goat heart was collected from local shops immediately after slaughtering; parcelled in ice container and stored in laboratory at -20 °C for future use (n=8).

Preparation of tissue homogenate

A 10% tissue homogenate of heart was prepared (mostly with the fresh tissues) in ice cold 0.1M phosphate buffer (ph 7.4) using a Potter-Elvehjem homogenizer. The homogenates prepared were used for further *in vitro* studies.

In vitro studies

Isolated heart homogenates were incubated in a graded concentration of Metanil Yellow (MY) in the following series 10, 25, 35, 50 µl in phosphate buffer medium. Another set of experiments included incubation of the heart homogenates with graded dilutions of ethanolic leaf extracts of *Coriandrum sativum* (CsEth) at concentrations of 10, 20, 30, 40 µl in phosphate buffer medium. A subsequent series of experiment was carried out co-incubation of heart homogenate with selected dose of MY and above mentioned concentrations of CsEth. The final set of incubation included homogenate incubation with selected dose of MY, selected dose of CsEth and co-incubation of selected dose of CsEth and MY in phosphate buffer solution. Control solution for all experiments included 10% heart homogenate in phosphate buffer solution. The incubation period for all sets of experiments included a 30 min duration at 37 °C [18, 19].

Biochemical analysis

Lipid peroxidation

The level of lipid peroxidation in heart tissue of goat was estimated by the method of Buege and Aust with modifications [5, 20].

Catalase activity

The activity of catalase enzyme was estimated by the method of Beer and Seizer 1952 [21] and as modified by Mitra *et al.* 2012 [22].

Protein estimation

The protein content of cardiac tissue was estimated by the method of Lowry [23].

RESULTS

Phytoconstituents like saponins, tannins and phenols, gum and mucilage and flavonoids etc. were qualitatively estimated by using standardized protocols. We found that except saponin all three other constituents were present in the ethanolic extract of *Coriandrum sativum* (table 1).

Table 1: Qualitative analysis of the phytoconstituents of ethanolic leaves extract of *Coriandrum sativum*

Qualitative test	Method Used	Results
Saponins	Foam method	-
Tannins and Phenols	Ferric chloride method	+
Gum and Mucilage	Alcohol method	+
Flavonoids	Sodium hydroxide method	+

+: Present, -: Absent

Qualitative analysis of the phytoconstituents of ethanolic leaves extracts of *Coriandrum sativum* revealed the presence of tannins and phenols, gum and mucilage and flavonoids. Saponin was found to be absent.

Our studies reveals that there was no significant alteration in the level of lipid peroxidation or activity of the antioxidant enzyme, catalase in goat heart when the tissues were treated *in vitro* with various doses of *Coriandrum sativum* (10 µl, 20 µl, 30 µl, 40 µl, 50 µl) [table 2].

Table 2: Effect of various doses of ethanolic extract of *Coriandrum sativum* on the level of lipid peroxidation and catalase activity of goat heart

Parameters	Control	CsEth 10	CsEth 20	CsEth 30	CsEth 40	CsEth 50
Level of lipid peroxidation [nmoles of TBARS per mg protein]	13.97±1.0234	13.78±1.0432	14.18±1.0232	13.71±1.0487	14.01±1.0384	14.14±1.0329
Catalase activity [μ moles of H ₂ O ₂ consumed per minute per mg protein]	60.54±0.4942	61.55±0.15899	62.34±0.2127	62.55±0.3150	62.60±0.1159	62.76±0.1822

n= 6, Values are mean+SE

Our studies reveal that increasing doses of metanil yellow caused an increase in the level of lipid peroxidation in a dose-response manner in goat heart *in vitro*. Metanil yellow at a dose of 35 μ l

(concentration: 1 mg/ml) caused a significant increase in the level of lipid peroxidation in goat heart *in vitro* compared to that of control (P<0.01 Vs. Control) [fig. 1.].

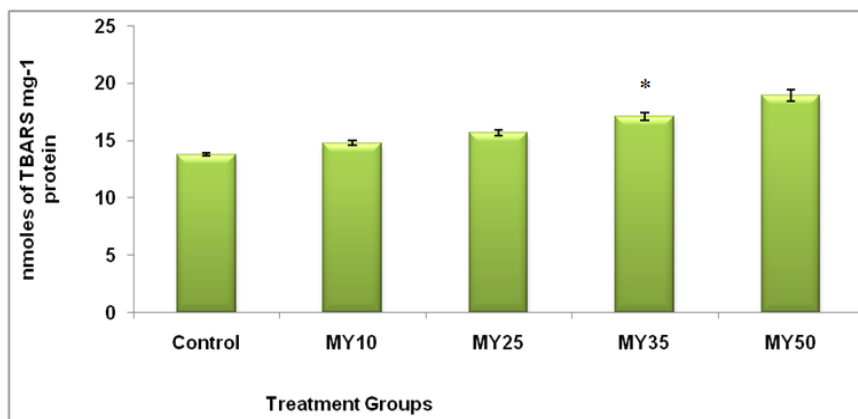


Fig. 1: Effects of various doses of metanil yellow on lipid peroxidation level in heart tissue of goat (n=6); Values are mean+SE, *P<0.01 Vs. control

Our studies reveal that increasing doses of metanil yellow caused a gradual decrease in the activity of catalase in a dose-response manner in goat heart *in vitro*. Metanil yellow at a dose of 35 μ l (concentration: 1 mg/ml) (MY35) caused a significant decrease in

the activity of catalase in goat heart *in vitro* compared to that of control (*P<0.01 Vs. Control) [fig. 2.]. We used the dose of 35 μ l (concentration: 1 mg/ml) of metanil yellow for the rest of our study.

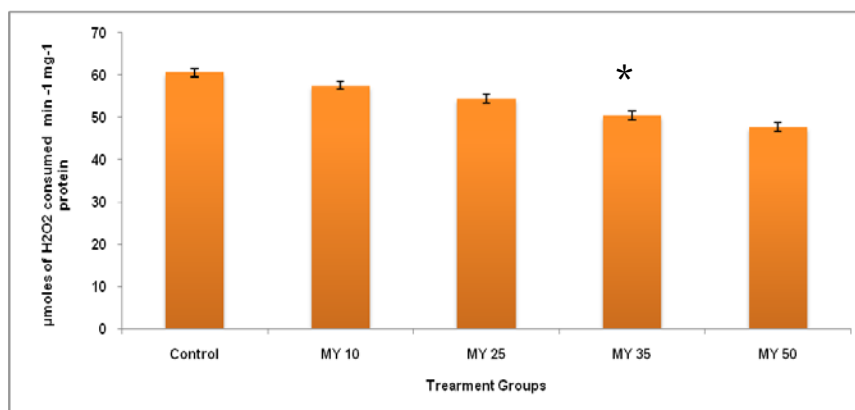


Fig. 2: Effects of various doses of metanil yellow on catalase activity in heart tissue of goat (n=6); Values are mean+SE; *P<0.01 Vs. control

Our studies reveal that metanil yellow at a dose of 35 μ l (concentration: 1 mg/ml) caused a significant increase in the level of lipid peroxidation compared to that of control in goat heart *in vitro*. Metanil yellow at a dose of 35 μ l (MY35) caused a significant decrease in the activity of catalase in goat heart *in vitro* compared to that of control (*P<0.01 Vs. Control) [fig. 3].

We found that co-treatment of heart tissues of goat *in vitro* ethanolic extract of *Coriandrum sativum* (EthCs) at various doses (10 μ l, 20 μ l, 30 μ l, 40 μ l, 50 μ l) protected the level of lipid peroxidation in goat heart from being increased in a dose response manner compared to

that of metanil yellow treated heart. Our studies further reveal that co-treatment of goat heart tissue with ethanolic extract of *Coriandrum sativum* at the dose of 30 μ l protected the level of lipid peroxidation from being increased in the cardiac tissue mediated by metanil yellow exposure at the dose of 35 μ l (MY35) (concentration: 1 mg/ml).

The level of lipid peroxidation in the 35 μ l (concentration: 1 mg/ml) metanil yellow and 30 μ l ethanolic extract of *Coriandrum sativum* (MY35+EthCs30) co-treated tissue was found to be significantly lowered than that of metanil yellow exposed tissue (MY35) (**P<0.01) [fig. 3, 5].

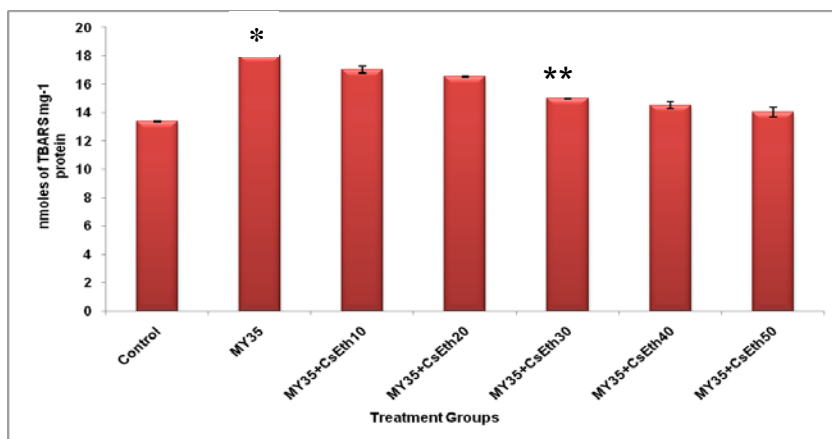


Fig. 3: Dose-response study of ethanolic leaves extract of *Coriandrum sativum* against metanil yellow induced lipid peroxidation in heart tissue of goat; (n=6); values are mean+SE; *P<0.01 Vs control; **P<0.01 Vs MY35 [metanil yellow at a dose of 35 µl (MY35) (concentration: 1 mg/ml)]

Our studies reveal that metanil yellow at a dose of 35 µl (concentration: 1 mg/ml) caused a significant decrease in the activity of catalase enzyme in heart tissue of goat (n=6 each) compared to that of control. We found that co-treatment of heart tissues of goat *in vitro* ethanolic extract of *Coriandrum sativum* (EthCs) at various doses (10 µl, 20 µl, 30 µl, 40 µl, 50 µl) protected the activity of catalase enzyme in goat heart from being increased in a dose response manner compared to that of metanil yellow treated heart. Our studies further reveal that co-treatment of goat heart

tissue with ethanolic extract of *Coriandrum sativum* at the dose of 30 µl protected the activity of catalase enzyme from being decreased in the cardiac tissue mediated by metanil yellow exposure at the dose of 35 µl (concentration: 1 mg/ml).

The activity of catalase enzyme in the 35 µl (concentration: 1 mg/ml) metanil yellow and 30 µl ethanolic extract of *Coriandrum sativum* (MY35+EthCs30) co-treated tissue was found to be significantly higher than that of metanil yellow exposed tissue (MY35) (**P<0.01) [fig. 4, 6].

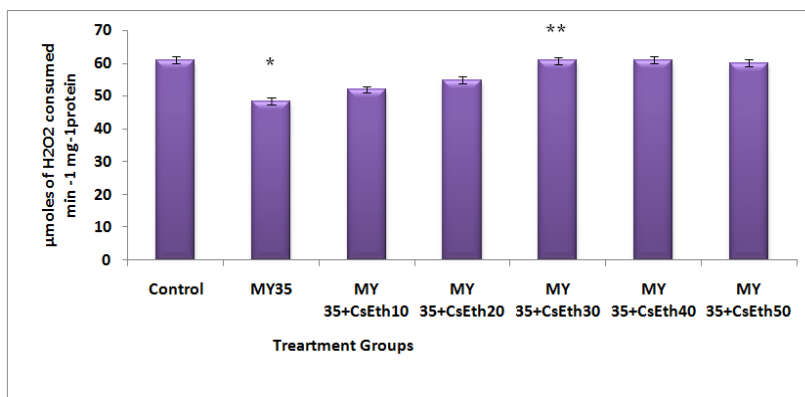


Fig. 4: Dose response study of ethanolic leaves extract of *Coriandrum sativum* against metanil yellow induced changes in the activity of catalase enzyme in heart tissue of goat (n=6). Values are mean+SE; *P<0.01 Vs Control; **P<0.01 Vs MY35 [metanil yellow at a dose of 35 µl (MY35) (concentration: 1 mg/ml)]

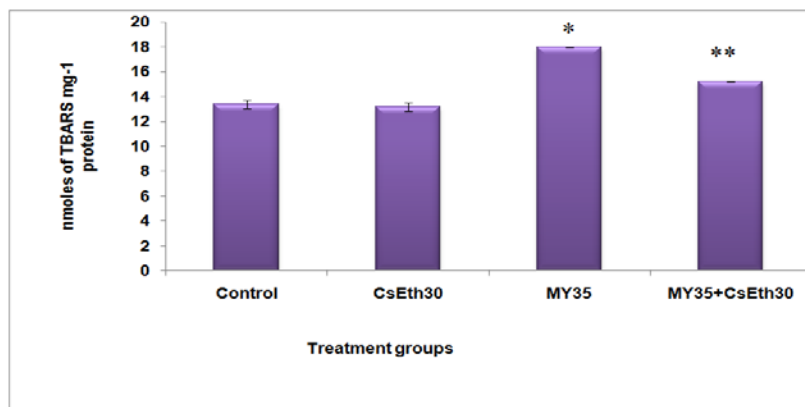


Fig. 5: Protective effect of ethanolic leaves extract of *Coriandrum sativum* against Metanil yellow induced changes in the level of lipid peroxidation in hearts of goat (n=6) values are mean+SE; *P<0.01 Vs Control; **P<0.01 Vs MY35 [metanil yellow at a dose of 35 µl (MY35) (concentration: 1 mg/ml)]

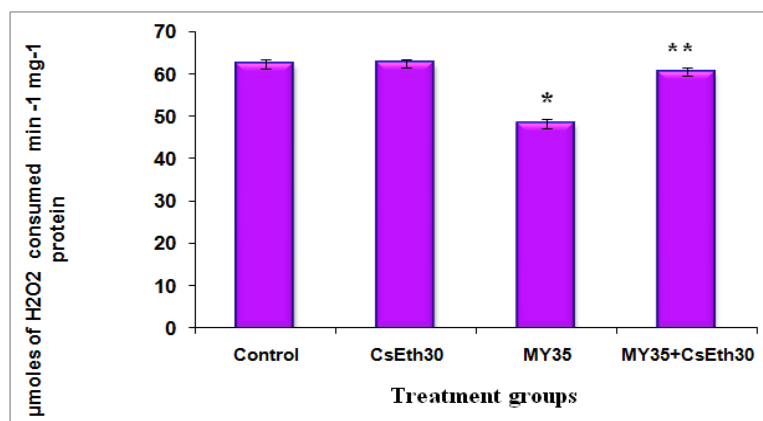


Fig. 6: Effect of effective dose of ethanolic leaves extract of *Coriandrum sativum* on effective dose of Metanil yellow on activity of catalase in hearts of goat (n=6); values are mean+SE; *P<0.01 Vs Control; **P<0.01 Vs MY35 [metanil yellow at a dose of 35 µl (MY35) (concentration: 1 mg/ml)]

DISCUSSION

Catalase is a conjugative homotetrameric enzyme having Fe³⁺centred heme group. It has a molecular weight of 64 kDa. Eukaryotic enzyme having both catalase and peroxidase activity. It is one of the chief subcellular antioxidant defence enzyme with the highest activity in liver and erythrocytes, followed by kidney and adipocytes, moderate in lungs and pancreas and least in brain and heart. It is one of the key detoxifier of H₂O₂ converting the former into water and oxygen. It scavenges about 80% of H₂O₂ in cardiac myocytes yet its overall activity is low. This may partially account for the high sensitivity of the heart to oxidative insult [24]. Studies revealed that in ischemia-reperfusion injury, subsequent supplementation of catalase in perfusion fluid can efficiently detoxify H₂O₂ in the isolated heart [25].

Heavy metals induced cardiac toxicity is known to reduce catalase activity significantly [26]. Metanil yellow, when administered in single dose parenterally, induces P-450 and its dependent monooxygenases which is related to its hepatic metabolism and toxicity [27]. P-450 dependent monooxygenase is also adequate in the heart. This heme-thiolate enzyme is involved in the metabolism of environmentally toxic chemicals and endogenous substrates through oxidative, peroxidative and reductive changes and some of the subfamily of enzymes may be detrimental to cardiac function as it may induce the generation of ROS [28]. Metanil yellow exposure to isolated heart perhaps by some likely mechanism induces ROS production in the heart. A significant reduction in catalase activity in isolated goat heart exposed to metanil yellow *in vitro* may be attributed to excess free radical generation by metanil yellow [5] analogous to acute myocardial infarction which is a consequence of an excess of H₂O₂ and superoxide production resulting in decreased activity of catalase [29].

LPO activity in the metanil yellow exposed isolated hearts was observed to be significantly enhanced. LPO is approximated by the accumulation of malonic dialdehyde [30]. There occurs a spectrum of cellular alterations including membrane phospholipid peroxidation, thiol oxidation, utilization of major chain-breaking membrane anti-per oxidants. Further oxidative alterations of myocardial membrane polyunsaturated fatty acids are related to increased level of lipid peroxidation which may be a probable manifestation of progression of myocardial infarction [31].

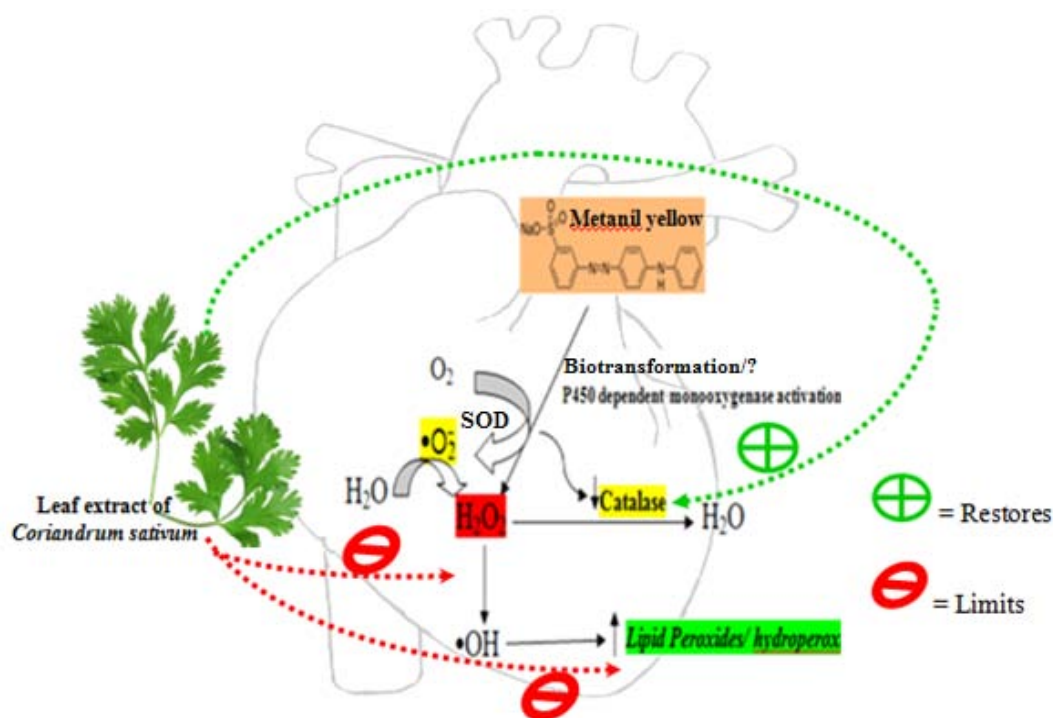
Amongst the various mechanisms involved in free radical induced lipid peroxidation followed by myocardial ischaemia, a deterioration of antioxidant system in the myocardium is suggested [32]. In the present study, increased lipid peroxidation may be attributed to decreased catalase activity since the available literature described catalase as a significant enzyme in cardiac myocytes in terms of its low content but highest activity in scavenging hydrogen peroxides which otherwise generates lipid peroxides and lipid hydroperoxides.

The findings of the present work also suggest that metanil yellow is cardiotoxic and may be an inducer of myocardial infarction mediated through oxidative stress.

Cardioprotection by phytochemicals or herbal medicine is widely accepted trend and is already in use recently. Many plants are known to be a good source of carotenoids, triterpenes, flavonoids, and alkaloids that are potent to combat free radical mediated cardiac toxicity including myocardial infarction and ischemic heart diseases [33]. Some of such commonly listed plants are *Terminalia arjuna*, *Andrographis paniculate*, *Citrus aurantium*, *Vitis Vinifera*, *Cocos nucifera*, *Zingiber officinale*, *Allium cepa*, *Curcuma longa*, *Terminalia chebula*, *Azadirachta indica* etc and many others [32]. *Coriandrum sativum*, however, is not enlisted. Our observations showed that ethanolic leaf extract of *Coriandrum* has a good potential to counteract metanil yellow lipid peroxidative damage to cardiac tissue and restoring the normal activity of catalase. It has already been admitted that plants containing tannins, polyphenols and flavonoids have cardioprotective action [34]. Of the various phytoconstituents, qualitative estimation of ethanolic leaf extract of *Coriandrum* has shown the presence of a spectrum of compounds having antioxidant potential. Amongst such compounds phenols have been established as a good inhibitor of lipid peroxidation in *in vitro* studies [35]. Polyphenols and tannins are competent metal chelators and their high redox potentials bestow them with free radical quenching and hydrogen donating potential [36]. Flavonoids and tannins can also check superoxide radical generation [37]. Further, it has been reported that flavonoid content in tea leaf extract may be accountable for the decrease in lipid peroxidation and restoration of catalase activity in cadmium-induced oxidatively stressed rats [38]. The observations of the present study mimic such similar findings and it may be thus summated phenolic constituents, flavonoids and tannins present in *Coriandrum* leaves may be good detoxifiers of free radical mediated peroxidative damage in goat heart exposed to metanil yellow and efficiently refurbish normal catalase activity in the heart.

CONCLUSION

Thus it may be concluded that altered metabolic pattern induced by metanil yellow in the isolated hircine heart may induce cardiotoxicity by the generation of ROS which includes superoxide anion radical followed by excess hydroxyl radical which impinges normal catalase activity subsequently leading to peroxidative damage to lipids of isolated heart *in vitro*. Ethanolic leaf extract of *Coriandrum sativum* may alleviate such free radical mediated cardiotoxicity because of the presence of indigenous phytochemicals like tannins, flavonoids and phenols that quench the free radicals with the beneficiary effect of restoring catalase activity in *in vitro*. Thus the study may indicate that the leaf extract of this plant might be promising in the development of new drugs to treat various diseases including cardiac ailments.



Scheme 1: Mechanism of amelioration of metanil yellow induced cardiotoxicity by ethanolic leaf extract of *Coriandrum sativum* in goat heart

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Dr. SG and Dr. DG designed and conceived the study and analyzed the findings and together composed the manuscript. RND and SH performed the experiments and calculated and arranged the data. Finally, all authors read and approved the manuscript.

CONFLICT OF INTERESTS

Declared none

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