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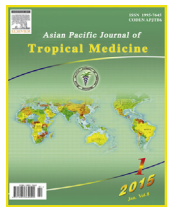

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An overview on cardioprotective and anti-diabetic effects of thymoquinone

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ABSTRACT

Thymoquinone (TQ), one of the active components of *Nigella sativa* exhibited to have many biological effects. Several beneficial effects of TQ such as its antidiabetic, antioxidant, anticancer, hypolipidemic, and anti-inflammatory activities have been recognized. The present review focuses on the findings of recent studies on the protective effects of TQ against cardiovascular diseases. In the current review, we additionally concluded that TQ may be therapeutically effective agents for controlling diabetes and hyperlipidemia by decreasing the oxidative stress and inflammatory responses.

1. Introduction

1.1. General knowledge

Cardiovascular diseases (CVDs) are a group of disorders in the heart and blood vessels that are responsible for the death of approximately 23.6 million people around the world [1]. According to WHO reports, unhealthy diet, tobacco use, cigarette smoking, environmental pollution, physical inactivity, and alcohol addiction are the most important risk factors for developing CVDs [1]. However, improving quality of life by avoiding these factors has been shown to reduce the risk of CVDs. The consumption of healthy food with low in free sugar, salt, and fat, is rich with natural plant products may be one of the major effective factors for protection against CVDs [2]. Plants containing flavonoids have been used for the treatment of various illnesses in many years [3,4]. In the modern pharmacology, flavonoids are receiving much more attention as an effective treatment for CVDs due to its anti-inflammatory, antioxidant, and vasodilatory effects [5]. Thymoquinone (TQ) has several bioactive components [6]. It has been found in the seeds of *Nigella sativa*, a plant species belonging to the Ranzmculaceae family [6]. TQ was also observed in various plants belonging to the Lamiaceae including *Agastache*, *Coridothymus*, *Origanum*, *Monarda*, *Mosla*, *Satureja*, *Thymbra*, and *Thymus* [7–9]. It has also been found in genus *Telraclinis,* and in a glycosidic form in the genera of *Cupressus* and *Juniperus* of the Cupressaceae family [10]. Many plant species contain TQ with its dimeric and reduced forms dithymoquinone (DTQ) and thymohydroquinone (THQ) [11]. (Table 1)

TQ, the major component of Lamiaceae family, exhibits protective effects against coronary artery diseases, respiratory failures, urinary system failures, hypertension, diabetes, nemodegenerative diseases, apoptosis, inflammation, and oxidative stress. The anti-oxidant and anti-inflammatory activities of TQ may cause its clinical effect against various diseases [12]. The anti-inflammatory effect of TQ is associated to its inhibitory effects on cyclooxygenase and 5-lipoxygenase and its antioxidant effect is associated with the scavenging activity against reactive oxygen species (ROS) [13]. TQ penetrates to physiological barriers and access to subcellular compartments, and exhibits the radical scavenging effects [14,15]. TQ also reacts with glutathione (GSH), NADH, and NADPH to form glutathionyl-dihydro-TQ (reduced species) [16] and combats

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Table 1

A summary of cardio protective effects of thymoquinone.

TQ dose	Rout of exposure	Experimental model	Effects	Ref
20 mg/kg	Gastric gavage	Hypercholesterolemic rabbit	Prevented the progression of atherosclerosis by decreasing LDL-C, TC/HDL-C, and TG, MDA levels and increasing HDL-C level	[28]
3.5 mg/kg	p.o.	Hypercholesterolemic rabbit	Prevented the progression of atherosclerosis by decreasing LDL-C, TC/HDL-C, and TG, MDA levels and increasing HDL-C level	[29]
10 mg/kg	p.o.	Hypercholesterolemic rabbit	Prevented the progression of atherosclerosis by decreasing LDL-C, TC/HDL-C, and TG, MDA and PC levels and also increasing HDL-C level	[30]
1 mL of 10 mg TQ	Gastric Gavage	Hypercholesterolemic rat	Prevented the progression of atherosclerosis by enhancing in the activity of arylesterase, decreasing in the activity of HMG-CoA reductase, MDA level, and shortened the lb-LDL, sd-LDL and LDL log times.	[31]
5 mg/kg	i.p.	STZ-diabetic rat	Prevented the progression diabetes by decreasing the expression of COX-2 enzyme MDA levels and increasing the level of SOD in the pancreatic tissue.	[50]
20 mg/kg	p.o.	Diabetic mice during gestation and lactation	Prevented the progression diabetes in their offspring by decreasing in the levels of blood glucose, free radicals, plasma pro-inflammatory cytokines (IL-1β, IL-6, and TNF-α), and lipids, and restoring the number of circulating lymphocytes, the proliferation of SEB-stimulated lymphocytes and aberrant AKT phosphorylation	[51]
50 mg/kg	Gastric gavage	Gestational diabetic hamster	Prevented the progression diabetes by inhibiting the synthesis of gluconeogenic enzymes	[52]
80 mg/kg	Gastric gavage	STZ-diabetic rat	Prevented the progression diabetes by decreasing the activities of the glucose-6-phosphtse, fructose-1,6-bisphosphatase, MDA, and increasing the levels of GST, GPx, CAT, GSH, Vit E and Vit C	[53]
3 mg/mL	i.p.	STZ-diabetic rat	Prevented the progression diabetes and also improved the toxic activities of STZ, such as heterochromatin aggregates, DNA damage, segregated nucleoli, and fragmentation and vacuolization of mitochondria by decreasing the MDA level and increasing the SOD level	[54]
12.5, 25, 50 mg/kg	p.o.	Rat exposed to Iso	Prevented cardiotoxicity caused by Iso via decreasing TBARS level and increasing GSH/GSSG ratio of myocardial tissue, and plasma GR & SOD	[55]
50 mg/L	p.o.	Rat exposed to CP	Prevented cardiotoxicity caused by CP via decreasing serum TBARS, TNF-α, TC, TG, CK, LDH, Urea, and also increasing CAT, ATP, GPx, and GSH levels of the heart tissue	[56]
8 mg/kg	p.o.	Mice exposed to DOX	Prevented cardiotoxicity caused by DOX via decreasing serum CK and LDH	[57]

TQ: thymoquinone, STZ: streptozotocin, DOX: doxorubicin, Iso: isoproterenol, CP: cyclophosphamide, TG: triglycerides, TC: total cholesterol, LDL-C: low density lipoprotein cholesterol, LDH: lactate dehydrogenase, CK: creatine kinase, COX-2: cyclooxygenase-2, HMG-CoA reductase: 3-hydroxy-3-methyl-glutaryl-coenzyme A reductase, IL-6: interleukin-6, TNF-α: tumor necrosis factor-α, MDA: malondialdehyde, HDL-C: high density lipoprotein cholesterol, GSH: glutathione, SOD: superoxide dismutase, CAT: Catalase, GST: glutathione-S- transferase, GR: glutathione reductase, ATP: adenosine triphosphate, TBARS: thiobarbituric acid reactive substances, SEB: *Staphylococcus* enterotoxin B, p.o.: Per Os, i.p.: intraperitoneal.

with free radicals [17]. It has been observed that TQ possesses protective aspects for inhibition and treatment of CVDs [18]. The present review aims to increase our knowledge on the protective effects of TQ against CVDs by gathering the present scientific literature. In addition, the isolated TQ from Ranzmculaceae family may be used as an emerging potential therapeutic drug for treatment of CVDs in the future.

1.2. Pharmacokinetics properties

TQ (2-Isopropyl-5-methylbenzo-1, 4-quinone) is the most bioactive ingredients of seeds with molecular formula $C_{10}H_{12}O_2$ and molar mass 164.20 g mol^{-1} [19]. The Figure 1 shows the

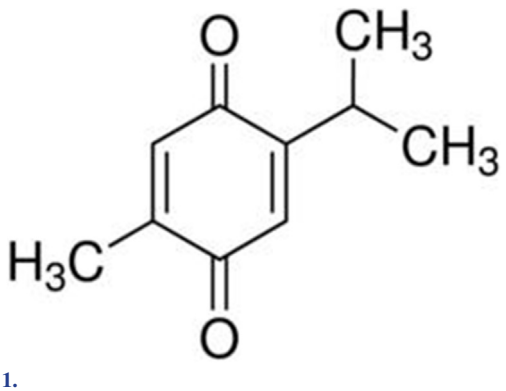

Figure 1.

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