Cardioprotective effect of resveratrol on atherogenic diet-fed rats

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Abstract: Atherogenic or high fat diets were known to induce cardiovascular diseases, and several active compounds were tested to protect/prevent the risk of cardiovascular diseases. We aimed to investigate the cardio protective effect of resveratrol against atherogenic diet fed rats. Male Wistar rats were administered atherogenic diet for 30 days and further continued for 15 days with or without resveratrol in the diet. The serum lipid profile, antioxidant enzyme activity, lipid peroxidation, lipid metabolic proteins and cardiac tissue markers were examined. The histopathology of myocardium and aorta were also examined. The abnormal serum lipid profile found in atherogenic rats was reversed by the administration of resveratrol. Similarly, the enzymatic (catalase, superoxide dismutase, glutathione-peroxidase), non-enzymatic (reduced-glutathione, Vitamin C, E) antioxidants were improved by the resveratrol fed against atherogenic diet. Interestingly, resveratrol activated the lipid metabolic proteins (SIRT1, eNOS and AMPKa), suggesting its protective effect on lipid metabolism. Further analysis on tissue damage revealed that resveratrol had significantly protected the tissue damage and maintains the morphology of cardiac tissue. Altogether, our results suggest that resveratrol played a significant role in the prevention of cardiovascular system against the high fat diet. Emphasising the anti-atherogenic property of resveratrol, we propose resveratrol as a potential compound to be consumed for the healthy lifestyle.

Keywords: Antioxidants, atherosclerosis, resveratrol, lipid metabolism

Introduction

Coronary heart disease (CHD) is a major and preventable cause of morbidity and death in the United States. Studies have shown that despite an increased prevalence of smoking and consumption of diets containing significant amounts of saturated fats, the incidence of cardiovascular disease is actually lower in the French population than in the American population [1]. The prophylactic and therapeutic effect of many plant foods and extracts in reducing cardiovascular disease has been reviewed [2].

Resveratrol, trans-3, 5, 4'-trihydroxy stilbene is a naturally occurring phytoalexin present in many different types of nutrients, which we consume on daily basis. Resveratrol is a polyphenol found in the skin of grapes, berries and peanuts that can activate AMPK and sirtuins [3]. Resveratrol possess antioxidant properties [3], increases nitric oxide synthase production [4] and improves mitochondrial function by activating AMPK and sirtuins [3-5]. Resveratrol has anti-cancer and anti-inflammatory effects and beneficial cardiovascular effects [6]. It is well known that resveratrol has beneficial effects on the cardiovascular system. It plays the most important role in the epidemiological phenomenon called “French paradox” meaning that existence of cardiovascular risk factors with low incidence/mortality rates which may attribute to moderate consumption of red wine [7, 8].

Resveratrol protects the cardiovascular system by a number of mechanisms, including resveratrol-mediated inhibition of low-density lipoprotein oxidation, inhibition of platelet aggregation, synthesis of proatherogenic eicosanoids, inhibition of cell proliferation, and increased vasorelaxation. Recent studies have also shown that resveratrol suppresses the induction of procoagulant tissue factor, one of the key components thought to be responsible for high mortality from cardiovascular disease [9-11].
The present study was designed to evaluate the protective effect of resveratrol against atherogenic diet particularly focusing on its possible role of cardio protection in rat model.

Materials and methods

Experimental animals and grouping

The Male Wistar rats (n = 24) were housed in room temperature with regular 12 h day/night cycle, the animals were accessed to food and water ad libitum. The animals were used in accordance with institutional guidelines and approved protocols by the Animal Ethical Committee. The animals were initially divided into three groups. A control group (C, n = 6) was given access to water and normal standard diet, ad libitum. To study influence of resveratrol in standard diet-fed conditions, a (Res, n = 6) group received standard diet and 6 mg/l resveratrol (trans-3, 40, 5-trihydroxystilbene) in its drinking water, approximately 1 mg/kg body weight/day. In order to study the effects of resveratrol in atherogenic diet-fed condition, a group (AD, n = 12) received water and atherogenic diet formulated based on Diniz [12]. After 30 days of the experimental period, the AD group was randomly divided into two subgroups (n = 6/group): (AD) group remained receiving atherogenic diet and water, and (AD-Res) group given atherogenic diet and 6 mg/l resveratrol in its drinking water. The experiment with AD and AD-Res group continues further for 15 days. Rats in the C and Res groups remained with the same treatment during whole experimental period of 45 days.

Sample preparation

After sacrificing the animals the blood samples, serum was separated and a haemolysate was prepared according to the modified procedure of Quist [13]. A lipid profile analysis was performed on the serum samples, while antioxidant levels were analyzed in haemolysate samples. All the samples were stored at -80°C until analysis. Prior to biochemical analysis, cardiac tissue (100 mg tissue/ml buffer) was homogenized in 50 mM phosphate buffer (pH 7.2); the homogenate was then centrifuged at 1200 g for 15 min and the supernatant was used for biochemical analysis. The protein concentration in each fraction was determined by the method of Bradford [14], using crystalline bovine serum albumin as a standard.

Evaluation of serum lipid profile

The lipid profile includes total cholesterol (TC), triglycerides (TG), low-density lipoprotein cholesterol (LDL), very low-density lipoprotein cholesterol (VLDL) and high-density lipoprotein cholesterol (HDL). The serum levels lipid profile was determined by using standard assay kits (Diasys, Holzheim, Germany). The units were expressed as mg/dl.

Determination of lipid peroxidation

The lipid peroxidation was evaluated in both tissue homogenate and haemolysate samples. The mean concentration of malondialdehyde (MDA) was determined as a measure of lipid peroxidation, and MDA was assayed in the form of thiobarbituric acid-reacting substances (TBARS) by the method of Ohkawa [15].

Enzymatic antioxidant activities

The activity of enzymes in antioxidant system was evaluated in both tissue homogenate and haemolysate samples by following the previously reported methods. Catalase (CAT) activity was determined by the method of Sinha [16]. The activity of CAT was expressed as units/mg protein (µmol of H2O2 consumed/min/mg protein). Superoxide dismutase (SOD) activity was determined by the method of Marklund and Marklund [17]. The enzyme activity was expressed as units/mg protein. Glutathione peroxidase (Gpx) was determined essentially as described by Rotruck [18]. The activity of Gpx was expressed in terms of µg of GSH consumed/min/mg protein. The enzyme activity was expressed as lmol of CDNB formed/min/mg protein.

Non-enzymatic antioxidant levels

The levels of non-enzymatic antioxidants in cardiac tissue homogenate samples were determined by following the previously reported methods. Reduced glutathione (GSH) content was estimated by the method of Moron [19]. Ascorbate (vitamin C) was measured by the method of Omaye [20]. α-Tocopherol (vitamin E) was estimated by the method of Desai [21]. For
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Sections of thickness 4-6 µm were cut, stained by aqueous haematoxylin and alcoholic eosin and the sections were examined by bright-field microscopy (Carl Zeiss Axioskop 2 plus; Jena, Gera, Germany).

Statistical analysis

The values are expressed as mean ± standard deviation (SD) for six animals in each group. Differences between groups were assessed by a one-way analysis of variance (ANOVA) and students T-test using SPSS software package for Windows (Version 11.5; SPSS Inc., Chicago, IL, USA). Post hoc testing was performed for intergroup comparisons using the least significance difference (LSD) test.

Results

Resveratrol improves serum lipid profile

A significant \( (P < 0.001) \) increased levels of serum TC, TG, bad cholesterols (LDL, VLDL) and the cardiac risk ratio values were measured in AD fed rats than those fed with a normal diet or Res fed rats. However, AD + Res fed rats had significantly \( (P < 0.001) \) decreased these levels and showed a significant \( (P < 0.001) \) increase in the level of HDL cholesterol. Rats fed with resveratrol alone showed improved lipid profile compared to that of normal diet fed (control) rats (Table 1).

Resveratrol precludes lipid peroxidation both in cardiac tissue and haemolysate

The lipid peroxidation (LPO) is determined by measuring the mean concentration of MDA. The cardiac tissue and haemolysate samples of AD rats showed significantly \( (P < 0.001) \) high-

### Table 1. Administration of Resveratrol improves serum lipid profile

<table>
<thead>
<tr>
<th>Lipid Profile</th>
<th>Control</th>
<th>Res</th>
<th>AD</th>
<th>AD + Res</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC</td>
<td>50 ± 3.76</td>
<td>40 ± 5.07</td>
<td>425 ± 7.34*</td>
<td>125 ± 5.38*</td>
</tr>
<tr>
<td>TG</td>
<td>80 ± 3.4</td>
<td>75 ± 3.5</td>
<td>180 ± 5.6*</td>
<td>115 ± 3.8*</td>
</tr>
<tr>
<td>LDL</td>
<td>20 ± 2.2</td>
<td>17 ± 2.8</td>
<td>205 ± 2.1*</td>
<td>60 ± 2.6*</td>
</tr>
<tr>
<td>HDL</td>
<td>70 ± 2.3</td>
<td>76 ± 2.5</td>
<td>38 ± 3.1*</td>
<td>50 ± 3.2*</td>
</tr>
<tr>
<td>VLDL</td>
<td>20 ± 1.5</td>
<td>16 ± 1.2</td>
<td>50 ± 1.9*</td>
<td>23 ± 1.3*</td>
</tr>
<tr>
<td>Cardiac ratio</td>
<td>5 ± 0.2</td>
<td>5 ± 0.1</td>
<td>17 ± 1.3*</td>
<td>6 ± 0.2*</td>
</tr>
</tbody>
</table>

TC: Total cholesterol; TG: Triglycerides; LDL: low-density lipoprotein, VLDL: Very low-density lipoprotein, HDL: High-density lipoprotein. Values are expressed as mean ± SD of six animals. Statistical analyses \*represents significance at \( P < 0.01 \). \*Control vs. AD values; \*AD vs. AD + Res values.

### Table 2. Resveratrol prevent lipid peroxidation (LPO)

<table>
<thead>
<tr>
<th>LPO</th>
<th>Control</th>
<th>Res</th>
<th>AD</th>
<th>AD + Res</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tissue</td>
<td>0.8</td>
<td>0.6</td>
<td>1.5*</td>
<td>1.0*</td>
</tr>
<tr>
<td>Haemolysate</td>
<td>1.7</td>
<td>1.5</td>
<td>3.2*</td>
<td>2.0*</td>
</tr>
</tbody>
</table>

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Assessment for cardiac tissue damage markers

The activity of cardiac tissue damage markers such as lactate dehydrogenase (LDH), alkaline phosphatase (ALP), aspartate transaminases (AST) and alanine transaminases (ALT) were determined by following the methodology of King [22].

Histopathological examination

Conventional techniques of paraffin wax sectioning and haematoxylin-eosin (HE) staining were used for this study. Slices of fresh thoracic aorta were cut and fixed in buffered neutral formalin fixative for 24 h. Following fixation, the tissue slices were washed and processed through an ascending series of alcohol (30%, 50%, 70%, 90% and 100%), cleared in methyl salicylate and infiltrated with wax at 57°C. The aorta, thus cleared was embedded in paraffin.

Sections of thickness 4-6 µm were cut, stained by aqueous haematoxylin and alcoholic eosin and the sections were examined by bright-field microscopy (Carl Zeiss Axioskop 2 plus; Jena, Gera, Germany).

Western blot analysis

Cells were washed with Hanks buffer and scraped in 50-100 mL of lysis buffer (with protease inhibitors), centrifuged, and the supernatant was collected. Protein content was determined by BCA protein assay. Total cell extracts containing 16-20 mg of protein were prepared in SDS sample buffer and subjected to SDS-PAGE and western blot analysis. Proteins were transferred to nitrocellulose membranes prior to immuno-detection. The antibodies for SIRT1, P-AMPKα (Thr172) and endothelial nitric oxide synthase (eNOS) were purchased from Cell Signaling (Beverly, MA), and GAPDH (Cambridge, MA) were used to detect the protein level in the heart tissues.

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Cardio protective effect of resveratrol

Figure 1. Resveratrol improves antioxidant system. Represents the levels of enzymatic antioxidants activities from cardiac tissue (A), haemolysate (B) and non-enzymatic antioxidants levels from cardiac tissue (C). Values are expressed as mean ± SD of six rats in each group and compared between AD vs. Control, & AD + Res. *Represents significance (P < 0.001); #Represents significance (P < 0.05).

Figure 2. Represent the western blot analysis of lipid metabolic proteins in rat heart tissues of experimental groups. Resveratrol regulates the key proteins SIRT1, eNOS, P-AMPKα involved in lipid metabolic pathway.

Resveratrol improves enzymatic antioxidant activity and non-enzymatic antioxidants levels

The mean activity of CAT, SOD and GPx found significantly (P < 0.01) lower in cardiac tissue and haemolysate samples of AD fed rats while compared to that of control or Res fed rats (Table 2). The mean MDA concentrations in cardiac tissue of AD + Res fed rats were significantly (P < 0.05) lower than that in AD rats. Similarly, pattern of MDA concentration was found in haemolysate sample. However, the Res fed rats showed much improved protection of LPO than that of control rats (Table 2).

Resveratrol regulates lipid metabolic pathway

The western blot analysis of key proteins involved in lipid metabolism showed that resveratrol significantly activated the protein levels of SIRT1, eNOS and decreased the phosphorylated AMPKα in heart tissue (Figure 2). However, the AD fed rats showed the reverse effect.
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These results might be a key evidence to speculate the mechanism of action of resveratrol against lipid deposition in heart tissue.

Resveratrol improves the activity of cardiac marker enzymes

The marker enzymes lactate dehydrogenate (LDH), alkaline phosphatase (ALP), alanine amino transferase (ALT) and aspartate amino transferase (AST) were found significantly (P < 0.01) elevated in AD rats than that of control/Res fed rats (Table 3). There was a significant (P < 0.001) decrease in the activity of cardiac markers in AD + Res fed rats when compared to AD rats. On administration of resveratrol, the changes in the activity of cardiac markers were reverted to near normal (Table 3).

Resveratrol prevents structural integrity of heart muscles

We had examined the histopathological studies on myocardial tissue and aorta. The control and Res fed rats shows healthy morphology of myocardial tissue. The AD fed rats show extensive myocardial damage like edema, leukocyte infiltration and necrosis. However, these damages were found significantly decreased in AD + Res fed rats (Figure 3A). The intima layer of the aorta was found thickened (blocks of fat deposition) in the AD fed rats while compared with control or Res fed rats. However, the AD + Res fed rat showed the intima of the aorta was found slightly thickened than that of rats fed with control rats (Figure 3B).

Discussion

The results of our present study represent the possible implications of resveratrol as a therapeutic agent for cardiovascular disease with an emphasis on resveratrol's effect on cholesterol metabolism. The cholesterol diet influences the cholesterol deposit in the aorta and other tissues in the form of cholesterol esters [23]. Our study demonstrated that the experimental animals fed with atherogenic diet (AD) exhibited augmented lipid levels in the cardiac tissue. The deposited cholesterol esters in the tissue need hydrolysis to release free cholesterol. One of the hydrolysis factors is HDL, since HDL-cholesterol level was found to be decreased in atherogenic diet fed rats [24], the insufficient HDL level may lead to free cholesterol in plasma, enhancing the pathogenesis. Lipoproteins are the vehicle for transporting plasma lipids to the blood. Resveratrol significantly alleviates the serum lipid profile, considering the importance of HDL to the reverse transport of cholesterol. Our results showed that resveratrol enhanced HDL in Res-fed rats. The most obvious effect of resveratrol on lipid profile was its action on in vivo ox-LDL. Resveratrol reduced the ox-LDL in both dietary conditions. Ox-LDL promotes atherosclerosis both by providing lipids signals that initially activate macrophages, and by stimulating foam cell formation [25].

Oxidative stress is one of the causative factors that link hypercholesterolemia with atherogenesis. There was enhanced lipid peroxidation or MDA level observed in the group II animals due to atherogenic diet that induces free radical production. Lipid peroxidation is a chain event that enhances MDA production [26]. Resveratrol also induced a strong decrease in alcohol-induced lipid peroxidation of heart; this could partly explain the cardiovascular beneficial effects of red wine consumption [27]. The lipophilic property of resveratrol enables it to associate with the lipid moiety of lipoproteins and prevent the oxidation of their unsaturated fatty acids [28]. Resveratrol increases the expression of antioxidant enzymes like superoxide dismutase, catalase, and glutathione peroxidase, thereby reducing the formation of free radicals and preventing endothelial injury [28]. Consistently, our results in present study; resveratrol...
Cardio protective effect of resveratrol

Resveratrol administration had improved the enzymatic and non-enzymatic antioxidant system against the atherogenic diet as well as in normal condition. These effects were similar in both heart tissue and haemolsate which is consistent with the previous study, treatment of apoE knockout mice with resveratrol for 7 days results in the upregulation of superoxide dismutase, glutathione peroxidase, and catalase in heart tissue [29]. Some researchers believe that the expression of these enzymes is the actual mechanism by which resveratrol prevents oxidative injury rather than the direct scavenging activity of reactive oxygen species.

Lipid metabolism in macrophages is an important process in the context of hypercholesterolemia. Uptake of excessive amounts of native and modified lipoproteins leads to their conversion into foam cells, which accumulate to create fatty streaks, a central feature of the early phase of atherosclerotic lesion development. Networks of proteins associated with macrophage lipid metabolism have been found in recent years to be affected by resveratrol [30]. In the present study, resveratrol activated the SIRT1, eNOS and regulated the phosphorylation of AMPK against the atherogenic diet. Resveratrol was discovered to be a strong activator of SIRT1, which regulates lipid metabolism by de-acetylation of modified lysine residues on histones and various transcriptional regulators [31]. SIRT1 has several effects associated with protection from the development of atherosclerosis. SIRT1 is an important signaling molecule in endothelium, which improves its function. SIRT1 binds directly to eNOS and has been shown to target eNOS for deacetylation, thereby stimulating nitric oxide production and promoting vascular relaxation [32]. Endothelial-derived nitric oxide controls vascular tone and has atheroprotective effects. AMPK is a sensor of cellular energy status and a key controller in the regulation of whole-body energy homeostasis [33]. It plays an integral role in lipid metabolism by switching on the oxidative process for fatty acids and by inhibiting the synthesis of lipids [34]. It also aids in endothelial relaxation and dilation.

Next, we examined the tissue damage induced by high cholesterol diet. The cardiac tissue markers were measured. The rats exposed to atherogenic diet had increased the activity of cardiac markers such as LDH, ALP, AST and ALT. This tissue damage causes the leakage of these markers in the plasma [35]. Resveratrol administration prevents this damage. Similarly, our histopathological examination of myocardium and aorta showed the abnormal morphology in atherogenic diet fed rats. However these changes have been prevented in resveratrol fed rats. These results were supported by the earlier reports with similar studies but with difference in treatments like fluvastatin and methanol extract of sorbus cortex [35, 36]. Moreover, resveratrol has potent antiatherosclerotic effects in in-vitro and in-vivo that indicate potential clinical utility in preventing the onset and/or progression of atherosclerotic cardiovascular disease. Despite a good deal of preclinical evidence, data on cardiovascular effects in humans are quite limited [36].

**Figure 3.** Represent the histopathological studies on rat myocardial tissue (A) and aorta (B) of experimental groups. The resveratrol had reduced the damage induced by atherogenic diet. Scale bar 200× and 100× respectively.
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In conclusion, the results of the present study have demonstrated that administration of resveratrol had significantly prevented cardiac abnormalities induced by atherogenic diet. Our data revealed that resveratrol possess the cardio protective effect by improving the serum lipid profile, antioxidant system, improving lipid metabolism and cardiac tissue damages either in myocardium or aorta. Our findings support a role for regular consumption of dietary resveratrol by consumption of resveratrol rich fruits or vegetables to avoid the risk of coronary artery disease.

Disclosure of conflict of interest

None.

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