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Fermented Seed of *Parkia biglobosa* (Jacq.) Benth (African Mustard) Diet Prevents Myocardial Infarction in Rat

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Abstract: Hypertension and diabetes are traditional major common risk factors of ischemic heart diseases. Antidiabetic and anhypertensive property of *Parkia biglobosa* (PB) are well documented. In our previous study, we demonstrated that stem bark of PB possessed cardioprotective effects in rats. Fermented seeds of PB (African mustard) is a natural nutritional condiment frequently used to season sauces in African diets. This study evaluated fermented seed of PB (FSPB) dietary preventing heart attacks in isoproterenol (ISO) induced Myocardial Infarction (MI). Four groups of five *Sprague dawley* rats of each were used. Control group, received the saline solution as vehicle (5ml/kg) per os (p.o) daily, for 15 days and was injected subcutaneously with vehicle (2ml/kg) at an interval of 24 hours, on day 14 and 15. Infarcted group received vehicle p.o and was injected subcutaneously ISO (100 mg/kg) as in control group. FSPB pre-treated groups received p.o FSPB (1g eq mv/kg and 2g eq mv/kg respectively) daily, for 15 days and was injected subcutaneously ISO (100mg/kg). ISO has induced MI, characterized by significant ($p < 0.001$) heart hypertrophy; histological heart changes; increased lipid peroxidation (MDA); decreased serum total Antioxidant Activity (AOA); increased transaminases, LDH and CPK activities and lipids metabolism alteration in infarcted rats. FSPB reduced significantly ($p < 0.01$) ISO induced biochemical alterations, morphological and histological changes, MDA and increased AOA in pre-treated rats. FSPB has cardioprotective property and its consumption, not only maintain health, may prevent heart attacks development in human.

Key words: *Parkia biglobosa*, fermented seed, isoproterenol, myocardial infarction

INTRODUCTION

Cardiovascular diseases remain the principal cause of mortality both in developed and developing countries, accounting for roughly 20% of mortality (Premisly *et al.*, 2010; Aman *et al.*, 2011). In among these evils disease, ischemic heart diseases, such as myocardial infarction, in particular are one major direct causes of death. In 2002, World Health Organization (WHO) estimated that 7.2 millions of deaths worldwide were issued from ischemic heart attacks (Sakande, 2009). Damorou *et al.* (2008) predicted that, heart attacks will be more frequent in Africa in the following decades because of rapid health transition in lifestyle and eating habits. Myocardial infarction is a systematic ischemic necrosis of the heart muscle, usually caused by sudden occlusive thrombosis of a coronary artery that supplies blood to the heart (Sakande, 2009). A lot of risk-factors especially hypertension, diabetes obesity overweight, dyslipidaemia, physical inactivity and use of tobacco work in concert, leading to a metabolic syndrome for

growing burden of cardiovascular diseases, increase prevalence of MI (Aman *et al.*, 2011).

Commonly, synthetics products used to treat myocardial infarction are too expensive, therefore, often unavailable to most population in developing countries. These medical drugs are efficient but not without side effects. Then, a range of natural products which may be used as dietary supplement, have been tried with considerable success to prevent the development of acute MI (Aman *et al.*, 2011). Yet, species of African locust bean tree, *P. biglobosa*, a perennial tree legume, belonging to the *Mimosaceae*, family, has been found to be used as traditional food and medicine (El-Mahmoud *et al.*, 2007). African pharmacopeia attributed to this plant therapeutic properties against: dental caries, pneumonia, bronchitis diarrhea, wounds, otitis, dermatosis, bilharziosis, leprosy, ankylosis tracheitis, conjunctivitis and cardiovascular diseases (hypertension, cardiac failure, cardiac disturbances and chest pains) (Adjanooun *et al.*, 1987; Millogo-Kone *et al.*, 2008; Kane *et al.*, 2009).

The fermented seeds mustard of *P. biglobosa* has been commonly used in the north of Togo and, indeed, the west coast of Africa to season traditional soups.

Hence, previous ethnopharmacological investigations reported that seeds and stem bark had hypotensive and cardioinhibitory biological activities (Bonnah *et al.*, 1998; Kassi *et al.*, 2008), anti-inflammatory and analgesic effects Kouadio *et al.* (2000), protection against neurotoxic, hemotoxic and cytotoxic effects of poisonous snake venom (Asuzu and Harvey, 2003). Phytochemistry investigations identified antioxidants (catechins and ferulates) in stem bark extracts (Tringali *et al.*, 2000), reducing compounds, cardiac glycosides, alkaloids and polyphenols, protein and amino acid in seed of *P. biglobosa* (Ndir *et al.*, 2000; Ikpeme *et al.*, 2012).

In our previous study, we demonstrated that hydro alcoholic extract of *P. biglobosa* stem bark had cardioprotective effects in rats (Adi *et al.*, 2013) and fermented seeds had anti-inflammatory and antimicrobial activities (Adi *et al.*, 2006). Odetola *et al.* (2008) demonstrate that both aqueous and methanolic extracts of fermented seeds of *P. biglobosa* exert an antidiabetic property in rats. However, only the aqueous extract of *P. biglobosa* ameliorated the loss of bodyweight usually associated with diabetes and had a favorable lipid profile which is probably an indication of its possible anti-arteriogenic property (hypertension and ischemic heart diseases). Fermented seed of *Parkia biglobosa* is well appreciated by African for its organoleptic and pharmacological properties and is frequently used in sauce as a natural nutritional condiment. As stem bark extract has cardioprotective effects, in this study, we aimed to find out that, African mustard consuming, either maintain health, may prevent ISO induced heart attack in rats.

MATERIALS AND METHODS

Plant material: The African locust bean seed needed for this research was purchased from local market of Kara in the north of Togo.

Experimental animals: All the experiments were carried out with male and female *Sprague dawley* rats, weighing between 150-200g and obtained from the Animal House of Faculty of Sciences of the University of Lomé. They were housed in polypropylene cages (47 x 34 x 20cm) under standard conditions. The rats were fed on standard diet and water was provided ad libitum.

Fermented seed mustard preparation: Traditional method of processing African locust beans to mustard was used. 750g of *P. biglobosa* seed were boiled in tap water in ratio of (1: 5, w/v) for 5 hours. The evaporated water was replaced every 2 hours in order to keep the seeds covered. When seed are well cooked,

they are softened i.e., the integument are easily removable by simple friction between two fingers. Cooked seed were pounded gently in a cyclical manner in a mortar with sand in order to separate the seed coat from the cotyledons. The flaps of integument are carefully removed by scrubbing and washing with water. The cotyledons obtained were boiled again for 45 minutes, before washed and placed in a gift basket which, bottom and sides were covered with banana leaves. The gift basket was wrapped with jute sack tightly immediately to prevent the heat from escaping and incubated at room temperature for 72 hours to obtain fermented seed. Fermented seed well almonds were finely crushed softened. We add to the paste of fermented seeds, 10% of *P. biglobosa* tree branches ash and then we homogenize. The homogeneous mixture ash and paste was put into small lumps on a plateau and dried for 4 consecutive days out from dust and we obtained mustard of seeds fermented of African locust.

Preparation of fermented seed aqueous extract:

Fermented seed aqueous extract was freshly prepared by dissolving 10g eq mv of African mustard in 100 ml of NaCl 0.9% solution.

Experimental design: The experimental designs were carried out according to the method of Ponnian *et al.* (2008) modified. The rats were divided into 4 groups of 5 animals: Control rats received 9% of saline solution as vehicle (5 ml/ kg body weight) p.o daily for 15 days and was injected subcutaneously with vehicle (2ml/kg) at an interval of 24 hours on day 14 and 15. Group referred as infarcted rats, received p.o vehicle in the same conditions as in control group and was injected subcutaneously with Isoproterenol (Sigma, St. Louis, USA) (100 mg/kg body weight) dissolved in saline solution, at an interval of 24 hours on day 14 and 15. FSPB pretreated groups received p.o FSPB (1g eq mv/kg and 2g eq mv/kg body weight respectively), daily for 15 days and was injected subcutaneously with Isoproterenol (100 mg/kg body weight) at an interval of 24 hours on day 14 and 15. At the end of day 15, 12 hours after Isoproterenol injection, all the rats were weighed and anesthetized using anesthetic ether before, blood was collected via retro-orbital puncture and centrifuged at 2500 rpm for 10 minutes with electric centrifuge (SPN- 400 Shimadzu Scientific Corporation, Tokyo, Japan). Then serum was separated and used for estimation of various biochemical parameters.

After blood collection, all the rats were sacrificed by cervical dislocation. The heart was dissected out, washed immediately in ice chilled saline, blotted and weighed. 150mg weight of heart tissue was homogenized in 2.5ml of 0.1M Tris-HCl (pH7.4) buffer

solution and the homogenate was used for tissue lipid peroxidation assay. Heart from 2 rats in each of the groups, were randomly selected for histological examinations.

Estimation of hart relative weight: In each group, heart relative weight was determined by the method of Arvindkumar *et al.* (2009). Hart relative weight = hart weight/animal body weight x 100.

Serum parameters estimation: Total cholesterol, triglycerides, High Density Lipoprotein (HDL), serum activities of lactate Lactate Dehydrogenase (LDH) and creatinkinase (CK) GPT and GOT were estimated by using an automated spectrophotometric analyzer (Model: GB 300 PLUS. Version 3.8, Germany) and standard SPRINREACT kits reagents (S.A.U. Centra Coloma). Serum Low Density Lipoproteins (LDL) and Very Low Density Lipoproteins (VLDL) were calculated using Friedewald *et al.* (1972) equations:

$$\text{VLDL} = \text{triglycerides}/5$$

$$\text{LDL} = \text{Total cholesterol} - (\text{HDL cholesterol} + \text{VLDL cholesterol})$$

Lipid peroxidation assay: Lipid peroxides were estimated using Pattola *et al.* (2009) method. We initially activated Methyl-2-Phenyl-Indol. We have, mixed methyl-2-Phenyl-Indole solution 10 nM to iron chloride solution 32 μM in the respective proportions of 75 and 25%. Methyl-2-Phenyl-Indole was dissolved in acetonitrile and the iron chloride in methanol. The reaction mixture contained: 650 μL of methyl-2-Phenyl-Indole activated, 250 μL of MDA or homogenate, 150 μL of 12 N HCl solution and 10 μL of 2, 6-Di-tert-butyl-4-methylphenol (BHT) at a concentration of 0.1M. We incubate tubes at 45°C for 60 minutes. We centrifuge them at 3000 rpm for 10 minutes. We read the absorbance in a spectrophotometer (Thermo Fisher Scientific- Genesys 20, USA) at 526 nm. The concentrations of MDA standard used are 0.625, 1.25, 2.5, 5, 10 and 20 nM. White is composed of 75% acetonitrile, 25% iron chloride and 200 μL of Tris, pH 7.4

Serum antioxidant activity assay: Total Antioxidant Activity (TAC) designs were carried out according the method of Koracevic *et al.* (2001). A standardized solution of Fe-EDTA complex reacts with hydrogen peroxide by a Fenton type reaction, leading to the formation of radicals (OH). These reactive oxygen species degrade benzoate, resulting in the release of Thiobarbuturic Acid Reactive Substances (TBARS). Antioxidants from the added sample of rat serum cause suppression of the production of TBARS. This reaction was measured spectrophotometrically using thermo Fisher Scientific-Genesys 20, USA.

The inhibition of color development defined the antioxidant activity (AOA).

Histological studies: Histological evaluation was performed on the apical portion of the heart tissue. Fresh heart tissue were excised and then fixed in 10% formalin for 24 hours. The fixative was removed by washing through running tape water overnight. After dehydration through graded series of alcohols (70°, 90°, 95° and 100°) the tissues were cleaned in methyl benzoate, embedded in paraffin wax. Section were cut into 5 μm thickness and stained with hematoxylin and eosin. The sections were mounted and observed under light microscope with magnification of 100X for histological changes examinations (necrosis, inflammatory cells).

Statistical analysis: The result was express as mean \pm standard deviation for six rats for each group. The statistical analysis was performed using Graph Pad Prism software version 4.00. Analysis was made using one way analysis (ANOVA) followed by Duncan's multiple comparison tests. A P-value <0.05 was considered statistically significant

RESULTS

Heart relative weight of the infarcted rats (5.48 \pm 0.17%) was significantly higher (P<0.001) compared to control (3.57 \pm 0.09%). FSPB (1g eq mv/kg and 2g eq mv/kg body weight) lowered significantly (respectively P<0.01 and P<0.05) heart relative weight when compared to ISO group (Table 1).

Table 1 showed also significant increased of MDA concentration (P<0.001) and serum uric acid (P<0.01) which indicated the decreased of antioxidant activity in serum of infarcted rats when compared to control. MDA concentration in FSPB pre-treated groups was significantly (P<0.01, P<0.001, respectively) lowered, while antioxidant activity increased, when compared to infarcted rats group.

LDH, CK, SGOT and GPT serum activities were increased significantly (p<0.001) in infarcted rats when compared with control group. FSPB (1g eq mv/kg, 2g eq mv/kg body weight) decreased significantly (p<0.001) these enzyme activities in pretreated rats when compared to infarcted rats group (Table 2).

Table 3 showed the lipid profile in different groups. There was in infarcted group significant increased (p<0.001) in serum total cholesterol, triglycerides, LDL and VLDL concentrations while HDL level decreased significantly (p<0.001) when compared to control group. FSPB pre-treatment decreased significantly the concentration of total cholesterol (p<0.01), triglycerides (p<0.05), LDL (p<0.001), VLDL (p<0.05) and increased HDL concentration in serum.

Table 1: Effect of FSPB on heart weight ratio, MDA and antioxidant activity

Groups (n = 5)	Heart relative weight (%)	MDA (n M/L)	AOA = uric acid (µM/L)
Control	3.57±0.09	248.80±10.32	614.16±39.82
ISO (100 mg/kg)	5.48±0.17***	432.54±40.49***	836.47±14.23●●
FSPB (1g eq mv/kg)	4.52±0.23##	272.20±17.14##	638.98±33.04##
FSPB (2g eq mv/kg)	4.62±0.19◆	250.01±20.73###	643.01±31.97##

FSPB was administered to rats daily for period of 15 days. Values are mean ± S.D for 6 rats in each group. *** p<0.001 and ●● p<0.01 VS control; ## p<0.01 and ◆ p<0.05 VS ISO

Table 2: Effect of FSPB on ISO induced changes in the activities of enzymes

Groups (n= 5)	SGOT (UI/L)	SGPT (UI/L)	CPK (UI/L)	LDH (UI/L)
Control	104.60±5.24	68.60±4.41	471.60±59.73	1621.20±139.41
ISO (100 mg/kg)	385.60±46.45***	139.60±13.08***	673.00±40.65Ω	4345.20±227.48***
FSPB (1g eq mv/kg)	186.20±24.21◆◆◆	90.80±8.82##	463.40±48.12●	1698.20±215.39◆◆◆
FSPB (2g eq mv/kg)	163.80±13.87◆◆◆	72.80±4.30◆◆◆	360.00±19.73##	1392.80±115.10◆◆◆

FSPB was administered to rats daily for period of 15 days. Values are mean ± S.D for 6 rats in each group. *** p<0.001 and Ω p<0.05 VS control; ◆◆◆ p<0.001, ## p<0.01 and ● p<0.05 VS ISO.

Table 3: Effect of FSPB on ISO induced changes in serum lipid

Lot (n= 5)	Cholestérol (mg /dl)	Triglycérides (mg /dl)	HDL (mg /dl)	LDL (mg /dl)	VLDL (mg /dl)
Control	116.00±3.84	50.60±5.21	112.80±4.99	5.04±2.31	10.56±0.73
ISO (100 mg/kg)	144.40±4.40Ω	78.40±2.85***	60.20±4.017***	68.72±4.83***	15.68±0.57***
FSPB (1g eq mv/kg)	100.80±10.94##	62.00±4.03●	85.40±8.10●	23.28±9.06◆◆◆	12.60±0.88●
FSPB (2g eq mv/kg)	107.80±3.42##	66.40±2.2494	69.20±3.30	25.28±4.67◆◆◆	13.28±0.44

FSPB was administered to rats daily for period of 15 days. Values are mean±S .D for 6 rats in each. *** p<0.001 and Ω p<0.05 VS control; ◆◆◆ p<0.001, ## p<0.01 and ● p<0.05 VS ISO.

Histological examination of control rat heart tissue showed normal myocardial architecture (Fig. 1A). Infarcted rats tissue showed severe necrosis and massive infiltrations of inflammatory cells (Fig. 1B) while there was slightly necrotic damage and infiltrations of inflammatory cells FSPB pre-treated d rats myocardium tissue (Fig. 1C and D).

DISCUSSION

In the present study, we evaluated *P. biglobosa* fermented seed mustard dietary preventing MI development using isoproterenol induced myocardial infarction model.

Previous studies after myocardial infarction in human reported: Cardiac hypertrophy (Vlasov and Volkov, 2004), increased of lipid peroxidation (Moselhy and Demerdash, 2004; Kasap *et al.*, 2007), elevated cardiac enzymes activity in serum (Damourou *et al.*, 2005) and high concentration of serum lipids (Pasupathi *et al.*, 2009).

Experimental studies on rats (Rajadurai and Prince, 2005; Arvindkumar *et al.*, 2009; Nivethetha *et al.*, 2009; Panda and Naik, 2009; Ponnian *et al.*, 2008) indicated that, all these pathophysiological abnormalities observed in humans after an acute myocardial infarction were similar to abnormalities exhibited in rats after isoproterenol administration.

Isoproterenol (L-beta-(3,4-dihydroxyphenyl)-alpha isopropylaminoethanol hydrochloride), a sympathomimetic beta-adrenergic receptor agonist, at high doses causes oxidative stress to myocardium resulting infarct like necrosis of heart muscle (Arvindkumar *et al.*, 2009;

Aman *et al.*, 2011), mainly via the beta-1 adrenergic receptor stimulation especially present in the heart muscle (Rang *et al.*, 2003). ISO exert positive inotropic and chronotropic effect on heart and it endogenous store lead to functional hypoxia then may deplete energy reserve of cardiomyocytes and thus result in biochemical and structural changes which are responsible for the development of irreversible damage leading to ischemia (Prabhu *et al.*, 2006; Aman *et al.*, 2011). If ischemia is severe and prolonged, it can induce cardiac cell death (necrosis or myocardial infarction) and the release of their enzyme content in the extra cellular medium. Ischemic muscle quickly generates the radical species derived from oxygen (ROS) when the capacity of cellular antioxidant enzymes decreased (Arvindkumar *et al.*, 2009).

The role of ROS is quite well-known. They play an important pathogen role in myocardial infarction. They are able to react with unsaturated lipids and initiate a chain reaction of lipid peroxidation in the cell membrane. They can also cause the oxidation of sulphhydryl groups in proteins and the separation of nucleic acid strands is possible, leading to irreversible destruction of heart cells (Kasap *et al.*, 2007; Pasupathi *et al.*, 2009). Free radical-mediated lipid peroxidation and consequent changes in membrane permeability are primary factor of cardiotoxicity. ISO caused lipid metabolic alteration (Ponnian *et al.*, 2008; Rajadurai *et al.*, 2005; Panda *et al.*, 2009 and Kasap *et al.*, 2007) which deposition in the arteries promotes cardiovascular diseases (Rajadurai *et al.*, 2009). In addition it causes lipolysis in the myocardium resulting in the release of cardiac lipids bloodstream (Ponnian *et al.*, 2008).

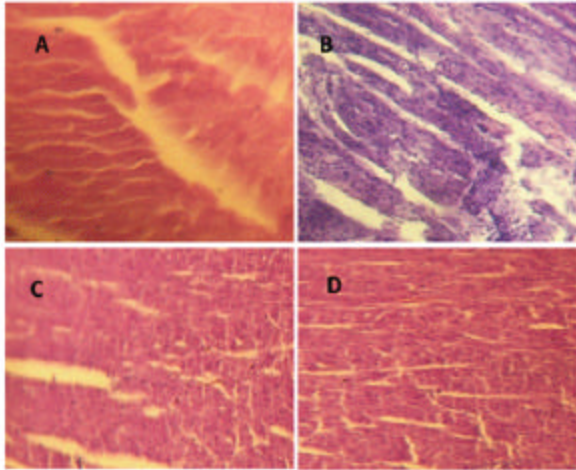


Fig. 1: Hematoxylin and eosin staining of heart 10x X 10x = 100X

- A: Control: Normal cardiac tissue integrity
- B: Infarcted rats (ISO): Tissue necrosis and with massive accumulation of inflammatory cells
- C: Pretreated FSPB (1g éq mv/kg): Slightly inflammatory cells infiltration
- D: Pretreated FSPB (2g éq mv/kg): Slightly inflammatory cells infiltration

In this study, heart relative weight higher significantly ($P < 0.001$) compared with the control rats. Our results were in line with previous studies (Arvindkumar *et al.*, 2009; Heather *et al.*, 2009). Heart relative weight increase traduced hart hypertrophy. Hypertrophy of the heart is an adaptive response to any intrinsic or extrinsic stimuli during the remodelling that occurs in the evolution of ischemic heart disease (Choukroun *et al.*, 2002; Arvindkumar *et al.*, 2009). Hypertrophy observed is a compensatory response to necrosis of the heart muscle, caused by the severe stress of the heart induced by administration of ISO (Ennis *et al.*, 2003).

Heart relative weight increase is due to the increase in overall protein biosynthesis during development of hypertrophy accompanied by oedema or over expression of genes encoding proteins involved in the contractile unit (Heather *et al.*, 2009; Choukroun *et al.*, 2002). Fermented seed of *P. biglobasa*, reduced significantly ($p < 0.001$) hart relative weight in pre-treated groups when compared to infarcted rats group. Our extract may prevent heart cells from the stress caused by ISO.

Lipid peroxidation is more probably one of the main mechanism through which ISO exert it toxic effects (Aman *et al.*, 2011). Malondialdheyde (MDA) is the only stable indicator marker of installation process of lipid peroxidation (Aznar *et al.*, 1983). MDA concentration in infarcted rats increased significantly ($p < 0.001$). Our results were in concord with previous results (Panda *et al.*, 2009, Prabhu *et al.*, 2006). ISO has the ability to

interact with sulphhydryl groups of various proteins and also lead to production of superoxide anions and subsequently hydrogen peroxide. This results in change in microsomal permeability, mitochondrial C^{2+} uptake, decrease in ATP production and the formation of reactive hydroxyl radicals which causes lipid peroxidation, protein and DNA irreversible damage (Pasupathi *et al.*, 2009; Dhalla *et al.*, 2010). Oxidative damage is commonly viewed as a consequence of oxidative stress that itself is defined classically as dynamic balance between pro-oxidants and antioxidant (Dhalla *et al.*, 2010)

Significant increase of serum uric acid concentration in infarcted rats indicated the decreased of cellular antioxidant capacity when compared to control.

Uric acid has potential pro-atherogenic effects, as it can stimulate the proliferation of vascular smooth muscle cells and causes endothelial dysfunction. Indeed, increased plasma concentrations of uric acid correlate with markers for coronary heart disease and predict the severity of coronary atherosclerosis (Lonn *et al.*, 2012). Hence, MDA level is found significantly decreased and the antioxidant capacity increased in animals pretreated with FSPB, when compared with ISO injected group. The combination treatments probably may prevent excessive superoxide anions production and then lipid peroxidation and cardiomyocytes damage. It is also possible that FSPB cardioprotective effects may pass by inhibiting cells membrane permeability disturbance or reinforcing myocardium antioxidant activities

Enzyme markers of cells necrosis SGOT, SGPT, CK and LDH level in infarcted rats group increased significantly ($p < 0.001$) when compared to control group. Cardiomyocytes contain large amounts of SGOT, SGPT, CK and LDH (Prabhu *et al.*, 2006; Sathish *et al.*, 2003). Their activity increased in infarcted rats serum, indicated the extent of cellular injury caused by excessive formation of free radicals induced by ISO which are responsible for impairment of cardiac cells membrane permeability and subsequent cardiac cells content release into bloodstream (Panda *et al.*, 2009; Nivethetha *et al.*, 2009). FSPB pre-treatment reduced significantly ($p < 0.001$) SGOT, SGPT, CK and LDH activity. These results suggested two lines of defense in protecting cardiac cell against injury owing to inhibiting lipids peroxidation by free radical scavenger activity and directly by increasing antioxidant capacity to resist against the cytotoxic effect of ISO, even to protein and DNA irreversible damage, consequently cardiac cells death.

Biochemical alterations reported in ISO induced myocardial infarction include altered lipids metabolism. Total cholesterol, triglycerides, LDL and VLDL concentration in serum increased, while, HDL level decreased in infarcted rats group. These results corroborated previous studies (Ponnian *et al.*, 2008; Rajadurai *et al.*, 2005). The primary disturbances of ISO induced myocardial infarction has been reported to

enhance cardiac cyclic adenosine monophosphate formation which in turn would lead to higher lipid accumulation (Prince and Rajadurai, 2005; Aman *et al.*, 2011). Increasing in cholesterol concentration, could be due to HDL decreasing, since it was known that HDL inhibits the uptake of LDL by the arterial wall and facilitates the transport of cholesterol from tissue to liver where it is catabolized and excreted from body (Ponnanian *et al.*, 2008; Rajadurai *et al.*, 2005). Increased triglycerides level may be due to the inhibition of protein lipase activity and therefore their transport into the circulation. Previous studies (Pasupathi *et al.*, 2009) have shown that high concentrations of total cholesterol, triglycerides, LDL, VLDL have positive correlation with the development of cardiovascular disease while, low level of HDL have negative correlation. Conversely, HDL level increased, protects the heart against the risk of cardiovascular disease (Rang *et al.*, 2003; Rajadurai *et al.*, 2005). Pre-treatment with FSPB decreased significantly total cholesterol, triglycerides VLDL and LDL, when compared infarcted rats. This suggested that, FSPB may prevent ISO induced lipid metabolism alteration. There it favorable lipid profile could represent a protective mechanism against the development of atherosclerosis in human by antihyperlipidemic effect.

Conclusion: *P. biglobosa* is widely traditionally used to treat various cardiovascular diseases like diabetes, hypertension, cardiac failure and cardiac disturbances. Fermented seed of *P. biglobosa* (African mustard) prevent significantly biochemical alterations, heart hypertrophy, histological changes and oxidative stress induced by isoproterenol. African mustard, used as dietary supplement, have favorable lipid profile effect and antioxidant property which may constitute two mechanisms of defense in cardiovascular diseases. Therefore, African mustard consumption, not only maintains health but may also prevent the development of myocardial infarction disease in human.

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