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Research Article

Effect of *Cucumis sativus* Linn. on Heat Stress in Albino Rats

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Abstract

In this world, everybody suffers from heat particularly in places during the work traffic snarls and meeting deadlines. It produces not only physical but also mental sickness. A variety of studies have recommended on relationship between heat exposure and the occurrence of disorders involving nervous, endocrine, and cardiovascular disorders. In this study, Wistar strain albino rats were pretreated with *Cucumis sativus, Linn* extract at the dose of 250 and 500mg/kg body weight for 2 days. The normal group and disease control received only vehicle. On 2nd day after treating animals, rats were kept in a hot chamber at the temperature of 40°C for about an hour until their rectal temperature (Tr) reached above 40°C but within 42°C. After an hour the blood was collected from retro-orbital puncture and analyzed for various biochemical parameters like lipid profile, anti oxidant, glucose, protein and rectal temperature. The seed of *Cucumis sativus, Linn* extract at the dose of 500mg/ kg, body weight had anti heat stress activity and normalize the lipid profile, oxidative stress and body temperature.

Key words: Heat-stress, lipid profile, oxidative-stress, *Cucumis sativus*

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Introduction

Heat-induced and heat-related illnesses remain highly prevalent in many working and living environments, in particular, during a sudden increase of ambient temperature or a heat wave when acclimatization of the human body has not been established (Bouchama and Knochel, 2002). Such illnesses include heat syncope, heat rash, heat cramps, heat exhaustion, and heat stroke in the order of increasing severity (Alzeer *et al*, 1997).

The heat shock or stress response is a highly conserved response of all cells, tissues and organisms on exposure to elevated temperatures, to a variety of environmental stresses, and to pathological stimuli such as infections, fever, inflammation, malignancy, and autoimmunity (Lindquist and Craig, 1988; Tan Morimoto *et al*, 1994).

Heat-stress is known to produce ROS, probably due to increased flux of oxygen through the mitochondrial electron transport chain (Tsong and Su 1999).

Elevated body temperature (Ikeda *et al*., 1999) and high metabolic rate (Ji, 1999 and Niess 1999) are known to increase the production of reactive oxygen species which react with various cellular molecules such as lipids, proteins and nucleic acids and result in cellular injury (Wells *et al*., 1997).

Antioxidants play a major role in protecting cells from the actions of reactive oxygen species (ROS) by reducing chemical radicals and preventing the process of lipid peroxidation (Nishigaki *et al*., 1992 & Yu 1994).

Endogenous antioxidant enzymes such as catalase, copper-zinc-containing superoxide dismutase (Cu,Zn-SOD), and Mn-SOD synergize with the GSH redox system to provide a comprehensive defence against the damaging effects of oxidative stress. The homeostatic balance between antioxidant defence mechanisms appears to be critical in maintaining normal cellular function (Palomero *et al*., 2001, Pansarasa *et al*., 1999) and Sanz *et al*., 1997)

Ayurveda, the traditional Indian system of medicine has given great emphasis to the promotion of health. Rasayanas are a group of non-toxic single or polyherbal preparation commonly used in indigenous medical practice in India to improve the health and longevity. Rasayanas improve memory, intelligence, and youthfulness, good lusture, complexion and efficiency

(Govindasa 1884, Hanumanthachar and Milind 2006, Mohandas Rao *et al*., 2006).

A number of standard Rasayanas such as Narasimha Rasayana, Ashwagandha Rasayana, Amruthaprasham and Chyavanprash have been reported to inhibit the oxidative stress and spontaneous mutations (Vayalil *et al*., 2002).

Cucumis sativus, Linn a herb belong to the family cucurbitaceae is found to distribute in the Himalayas from Kumaon to Sikkim, but it is cultivated throughout India. Fruit is nutrient and demulcent. Seeds are cool and diuretic (Ayurvedic Pharmacopoeia, 1927).

The present experiment was performed with objectives to determine the efficacy of *Cucumis sativus, Linn,* in heat-stress induced animals.

Materials and Methods

Plant material and extract preparation

The seed of *cucumis sativus, linn* was collected from Tamil nadu, India. The seed was identified and authenticated by experts in the R

abinat herbarium, St. Joseph College, Trichy, St. Xavier's college, Palayamkottai and Botanical survey, CCRAS unit, Govt. medical college, Palayamkottai. The collected materials were washed thoroughly in sterile water and dried in shade, pulverized by a mechanical grinder and passed through a 45-mesh sieve to get the course powder and stored in an airtight container. The pulverized seed of *Cucumis sativus* (seed,) was used for preparing hydroalcoholic extract. Then, it was filtered and allowed to cool and it was used for treatment.

Experimental animals

Healthy young adult male Albino wistar rats (200-250 gm) were procured from CARISM, SASTRA University, Tanjore. They were kept separately in individual polypropylene cages with stainless steel hopper. At the commencement of the study, the weight variations of the animals were kept minimal without exceeding ± 20 % of the mean weight for individual animals. (Lingjia Qian *et al*., 2004). The temperature in the experimental animal room was maintained 22 ± 3 ºC. For feeding, conventional laboratory diets was used with an unlimited supply of drinking water. The animals were fasted prior to dosing by withholding food overnight.

Treatment of animals

Animals were randomly divided into 4 groups with 8 animals in each. Group 1 Normal vehicle, 0.5 ml, p.o. Group 2 (Control) received vehicle 0.5 ml, p.o, Group 3 and 4 received extract of *Cucumis sativus* at the dose of 250 and 500 mg/kg, p.o, respectively.

Experimental procedure

Heat-stressed animal model

Animals were pretreated with extract at the dose of 250 and 500-mg/kg body weight for 2 days. The normal group and disease control received only vehicle. On 2nd day after treating animals, rats were kept in a hot chamber at the temperature 40°C with relative humidity of 75–85% for about an hour until their rectal temperature (Tr) reached between 40°C and 42°C. After an hour, the blood was collected from retro orbital puncture and analyzed for various biochemical parameters and the rectal temperature was measured before and after administration of the drug. The heat exposure was done in the morning from 6:00 am to 7:00 am. All rats were housed in a pathogen-free environment at room temperature (22ºC ± 3ºC.) and maintained on food and water *ad libitum* after heat exposure. (Lingjia Qian *et al*., 2004).

Analysis of various biochemical parameters includes glucose (RANDOX GPO PAP method, Cat no GL 2623, GL 2614, GL 2610), cholesterol (RANDOX Enzymatic end point method, Cat no CH 200, CH 201, CH 202, CH 280, CH 259), separation of serum high density lipoprotein, low density lipoprotein (Warnick and Albers,1978), triacylglycerol (RANDOX GPO PAP method, Cat no TR 1697/TR 2347), total protein (Lowry et al., 1951), RANDOX Biuret method) lipid peroxidation (Ohkawa et al., 1979), catalase (Aebi 1983), reduced glutathione (Ellman 1959), glutathione peroxidase (Rotruck et al., 1973) and vitamin C (Omaye et al., 1979)

Results and Discussion

Lipid profile

While concentration of total cholesterol, HDL, TGL and VLDL levels were noted down to be in the decreasing order ($p<0.01$). lipid profile in treated animals with extract at different doses, was found to be, increased significantly ($p<0.01$, Table 2). LDL was observed to be significantly decreasing ($p<0.01$) in animals, when subjected to higher temperature. But on treating with extract, it was significantly increased but dose dependently ($p<0.01$).

Antioxidants

Heat-stress causes increased oxidative stress and damage in the liver of young and old animals (Hall *et al.*, 2000, Hall , Xu , *et al*., 2000, Zhang *et al*., 2004 and Zhang HJ *et al*., 2003). Heat-induced uncoupling of the mitochondrial electron transport chain proteins may contribute to the increased free radical production during and after heat-stress by increasing the leakiness of the electron transport chain (Tsong and Su 1999).

The increased malondialdehyde and other unsaturated aldehydes generated by heat was found to be decreased significantly ($P<0.01$) when treated with the formulation (Table 2).

Similar results have been observed by Ando *et al*., 1994). They have reported that heat stress over 24 hours used to increase the lipid peroxidation level in the livers of rats and guinea pigs.

Feenster 1985) and Richards 1997) have mentioned that heat-stress causes the release of corticosterone and catecholamines and initiates lipid peroxidation in cell membranes. Sahin *et al*., 2001) have stated that at temperatures above or below thermoneutral zone, corticosteroid secretion increases, as a response to stress, causing oxidative damage on membrane of hepatic cells,

The glutathione concentration is thought to be one of the major cellular redox buffering systems in mammals. Hydrogen peroxide formed by the dismutation of superoxide is catabolized at the expense of reducing equivalents from GSH by glutathione peroxidase to form glutathione disulfide or mixed protein disulfides.

The reduced glutathione is noticed to increase significantly ($P<0.01$, Table 2) when treated with the extract. Similar results have also been observed by Freeman and Meredith 1988). They have determined that mitochondrial GSH compartmentalization as the limiting factor in cell survival after treatment.

Ramnath *et al*., 2007) have reported that glutathione peroxidase is an enzyme associated with conversion of reduced glutathione to oxidized form and back. Liver is

the chief organ concerned with detoxification process and availability of increased amount of GSH in the liver, which improves this function.

From our present findings, it is observed that reduced glutathione concentration is accounted to decrease. This might be due to the utilization of reduced glutathione by the enzymes like glutathione peroxidase and glutathione reductase, which are released from the tissues in to the circulation, to protect the cell from peroxidation. But on treating the animals with either extract, glutathione peroxidase level is registered to remain in the increased state itself. This may be due to the increased synthesis and release of reduced glutathione and glutathione peroxidase from liver.

Yin *et al*., 1993), Gey 1998) Luadicina and Marnett 1990) have mentioned that vitamin C and vitamin E are the primary antioxidants in biological systems and use to break the chain of lipid peroxidation in cell membranes. Overall antioxidant potential has been reported to possibly be more efficient and crucial than single antioxidant nutrients.

Tappel (1968) and McKee and Harrison (1995) have said that vitamin C and vitamin E work together such that vitamin E is the major chain-breaking antioxidant in lipid phases such as cellular membrane or low density lipoproteins, and the oxidizing free radical chain reactions are terminated in aqueous compartments, with vitamin C as terminal reductant.

Vitamin C level was found to be decreased significantly ($p<0.01$) in diseased control animals against the normal animals. But on treating animals with extract, vitamin C concentration was found to be increased significantly ($p<0.01$) while the disease control animals behave in the other way. The results are given in the Table 2.Results of our present findings in the concentration of vitamin C is found to be corroborated with earlier findings. (Njoku 1986), Richards 1997) & Osman and Tanios 1983).

Kutlu and Forbes 1993) have reported the decrease in adrenal size and vitamin C secretion rate, at high temperatures.

Glucose

Serum glucose concentration is found to be decreased significantly ($P<0.01$, Table 3) when treated with the extract

Febbraio *et al*., 1994), Fink *et al*., 1975) and Rowell *et al*., 1968) have also observed the increased level of glucose during exercise under high temperature. They have mentioned that the increase in glucose level may be due to liver glucose output exceeding muscle glucose uptake. Yasplekis *et al*., 1993) have demonstrated that splanchnic glucose output is increased during exercise and heat stress

Mark hargreaves *et al*., 1996) have quoted certain reasons for increase in glucose level during exercise in heat condition as, greater increase in liver glucose output without any alteration in whole body glucose utilization, increased oxidation of carbohydrate and muscle glycogenolysis.

Jansson *et al*., 1986) have demonstrated, the decreased muscle glucose uptake during exercise which may cause the decreased disappearance of glucose from plasma and an increase in intramuscular glucose – 6- phosphate concentration.

The increased level of serum glucose from our present findings may be attributed to the high temperature causing the blood to flow at a maximum to skin when compared to the muscle. This decreased uptake of glucose by muscle causes the increased level in plasma. Stress due to high temperature may increase the neither level of nor epinephrine level in blood, which may activate the sympathetic nervous system. The increased sympathetic activity may also have contributed to the greater liver glucose output. We have no data in the present study to either support or refute such a mechanism. On treating animals with the extract, plasma glucose level is found to decrease when compared to the control rats. This may be due to the increased utilization and decreased release of glucose by other organs when compared to the control animals. Thus, direct measurement of glucose uptake by contracting skeletal muscle may be required to assess the effects of heat stress on muscle glucose uptake in both control and treated rats.

Protein

Kazim Sahin *et al*., 2003) have surmised the decrease in protein level to the adjustment of the pancreas in birds accustomed to a hot environment.

Wallis and Balnava 1984) have seen that the digestibility of amino acids is decreased at a high environmental temperature in broilers. Similarly, Zuprizal et. al 1993) have shown that true digestibility of protein and amino acids of two different protein sources (rapeseed and soybean meals) decreased, as the temperature increased from 21 to 32^{o}C. Hai *et al*. 2000) have reported the significant decrease of the activities of trypsin, chymotrypsin, and amylase by a high temperature (32^{o}C).

Serum protein concentration is found to be increased significantly ($p<0.01$), on treating animals with extract or formulation when related to the control animals. The decrease in activity in disease control animals may be due to the activity of pancreas, decreased digestibility of amino acids or anabolism of proteins.

Conclusion

On treating animals with extract of *Cucumis sativus, Linn* the stress induced by heat was found to be decreased. It was observed by estimating various biochemical parameters like lipid profile, oxidative enzyme, glucose and protein. The rectal temperature was also observed. The extracts exhibit its activity as dose dependent.

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Table 1 Observation of lipid profile on animals subjected to heat stress and after treatment with extract (Values are mean ± SD)

Treatment	Cholesterol (mg/dl)	HDL (mg/dl)	LDL (mg/dl)	TGL (mg/dl)	VLDL (mg/dl)
Normal	65.0 ± 1.6	15.9 ± 0.8	32.9 ± 2.1	63.5 ± 1.5	12.7 ± 0.2
Control	20.3 ± 0.9**	7.8 ± 0.5**	10.9 ± 1.0**	20.3 ± 1.2**	4.06 ± 0.3**
CS 250 mg/kg	45.6 ± 3.2*	10.9 ± 1.3*	21.8 ± 1.5 **	40.5 ± 3.9*	8.1 ± 0.9*
CS 500 mg/kg	63.5 ± 1.8**	15.3 ± 1.2**	30.5 ± 1.3**	56.3 ± 2.5**	11.3 ± 0.9**

Note - *- p<0.05, ** - p<0.01
HDL – High Density Lipoprotein; LDL – Low-density Lipoprotein; VLDL – Very Low density Lipoprotein; TGL - Triacylglycerol.

Table 2 Observation of antioxidants on animals subjected to heat stress and after treatment with extract (Values are mean ± SD)

Treatment	Lipid peroxidation (µg/mg of protein)	Reduced Glutathione (µg/mg of protein)	Glutathione peroxidase (µg of GSH used /min/mg of protein)	Vitamin C (µg/dl)
Normal	600 ± 11.1	253.8 ± 12.9	65.3 ± 1.2	3000.1 ± 50.1
Control	1100 ± 20.1 **	123.5 ± 11.3 **	108.3 ± 2.1**	1200 ± 41.2**
CS 250 mg/kg	950 ± 31.2 *	183.6 ± 9.5 *	88.5 ± 3.2 *	2100 ± 51.2 *
CS 500 mg/kg	720 ± 15.3 *	245.9 ± 11.5 **	71.3 ± 2.5 **	2900 ± 33.5 **

Note - *- p<0.05, ** - p<0.01

Table 3 Observation of other bio molecules on animals subjected to heat stress and after treatment with extract (Values are mean ± SD)

Treatment	Glucose (mg/dl)	Protein (g/dl)
Normal	63.42 ± 2.0	7.5 ± 0.3
Control	178.0 ± 3.8 **	4.1 ± 0.2 *
CS 250 mg/kg	123.5 ± 4.9 *	6.3 ± 0.5*
CS 500 mg/kg	86.2 ± 5.4 **	7.2 ± 0.6 *

Note - *- $p<0.05$, ** - $p<0.01$

Table 4 Observation of rectal temperature in heat-stress in pretreatment and after treatment extract (Values are mean ± SD)

Treatment	0 hr -Pre treated (ºC)	1hr-After treated (ºC)
Normal	30.0± 1.3	30.1± 0.9
Control	33.8± 1.7*	36.9± 0.8*
CS 250 mg/kg	32.6± 1.9*	34.8± 0.6*
CS 500 mg/kg	33.5± 2.1*	35.5± 0.5*

Note - *- $p<0.05$,