HYPOLIPIDAEMIC EFFECTS OF *CURCUMA LONGA* L AND *NARDOSTACHYS JATAMANSI*, DC IN TRITON-INDUCED HYPERLIPIDAMIC RATS

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Summary : Fifty per cent ethanolic extract of *Curcuma longa* (tuber) and *Nardostachys jatamansi* (whole plant) feeding elevates HDL-cholesterol/total cholesterol ratio. The extracts also caused a significant reduction in the ratio of total cholesterol/phospholipids. *Curcuma longa* exhibited better cholesterol and triglyceride lowering activity [Ch=−85%; Tg=−88%] as compared to *N. jatamansi* in triton-induced hyperlipidaemic rats. In view of the protective action of HDL against heart disease and atherogenecity, *C. longa* consumption is recommended.

Key words : *Curcuma longa* HDL-cholesterol/total cholesterol triglyceride triton WR-1339

INTRODUCTION

Elevated plasma LDL and or abnormal VLDL with hypercholesterolemia is one of the principal risk factor in the development of atherosclerosis (12). Induction of hyperlipidaemia in rats with Triton WR–1339 is a method for screening new lipid lowering agents (15).

Increased interest in the atherosclerosis prevention and the identification of hyperlipidaemia as a risk factor have stimulated the study of drugs which prevent or lessen the risk (9). Very few plants todate have been investigated for antiatherosclerotic or hypolipidaemic activity in man and experimental animals (1, 13, 5, 6, 7, 8).

Curcuma longa L. (Family-Zingiberaceae) and *Nardostachys-jatamansi* DC. (Family-Valerianaceae) are well reputed in Ayurvedic system of medicine for their various therapeutic properties (4). Ethanolic (50% v/v) extract of *C. longa* and *N. jatamansi* have been screened

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for their possible liquid lowering activity in hyperlipidaemic rats so as to develop a cheap and effective antiatherosclerotic drug from plant source.

MATERIAL AND METHODS

Fresh tuber of *C. longa* and whole plant of *N. jatamansi* were separately powdered and defatted with petroleum ether (60-80°). The defatted materials were subjected to Soxhlet extraction with ethanol (50% v/v) for 24 hrs. Ethanol was removed under reduced pressure to obtain a brown solid.

30 Adult healthy male albino rats weighing 200-250 *g* of the inbred colony were used. They were maintained in an air conditioned room (26°±1°C) and were given rat feed (Hindustan Lever Pvt. Ltd.) and water *ad libitum*. The animals were divided into three groups of 10 each. The experiment was designed in such a way that for each drug there was a group of 10 rats and each drug was screened one after the other and not simultaneously. Each rat was injected intraperitoneally with Triton-WR-1339 (40 *mg*/100 *g* body weight/day in 0.15 M NaCl for 2 days).

Blood was withdrawn after 48 *hr* for triton administration and analysed to assess the induction of hyperlipidaemic state. This sample of blood is referred to as 0 *hr* sample, when the rats were found in a definite hyperlipidaemic state. Each drug was administered by similar mode and at the same time (i e. 300 *mg*/100 *g* body weight : drugs given repeatedly every 6 *hr* orally; 300 *mg* fifty per cent ethanolic extract v/v of *Curcuma longa* is equivalent to 3 *g* fresh tuber; 300 *mg* 50% ethanolic extract v/v of *N. jatamansi* is equivalent to 2.4 *g* dry weight of whole plant). Blood was taken from tail vein at 0, 6, 12, 24, 36 and 48 *hr* after the drug administration. Serum was separated by slow centifugation and analysed for total cholesterol (18), phospholipid (17), triglyceride (11), HDL-cholesterol (2), and VLDL-cholesterol and LDL-cholesterol (3). Student 't' test was applied in comparing means.

RESULTS AND DISCUSSION

Serum analysis : A significant lowering ($P \leq 0.01$) of serum total-cholesterol was observed 6 *hr* after the administeration of *C. longa* or *N. jatamansi*. Maximum fall of 80.8% with *C. longa* extracts and 55.6% with *N. jatamansi* extracts was recorded at 36 and 48 *hr* interval respectively (Table I).

A maximum fall of 85.3% and 56.2% was observed in serum triglycerides after 48 *hr* of *C. longa* and *N. jatamansi* extract feeding respectively. The maximum fall in mean value of phospholipid was 64.5% and 44.7% after treatment with *C. longa* and *N. jatamansi* respectively.

TABLE I : Serum total Cholesterol, Triglyceride, Phospholipid in Triton-induced hyperlipidaemic rats after *C. longa* and *N. jatamansi* extract feeding.

	Total cholesterol (mg/dl)			*Triglyceride (mg/dl)*			*Phospholipid (mg/dl)*		
	No treatment	*C. longa*	*N. jatamansi*	*No treatment*	*C. longa*	*N. jatamansi*	*No treatment*	*C. longa*	*N. jatamansi*
Fasting	92.8±5.5	100.2±5	104.5±7	105.9±4	118±3	109±4	143.7±7	150.7±6	147.5±8.0
0 hr	682±49.9	704±52.8	710±15	310±10	351±18	370±21	200±14.3	250±17	380±25
+6 hr	724±91.2[a]	532.9±41.1[c]	620±10[c]	316±8[a]	302.8±12[b]	305±16.2[b]	205±72[a]	240.6±17[a]	350±18[a]
% fall	—	24.2	12.7	—	13.7	17.6	2.5	3.73	7.9
+12 hr	694±74[a]	308.5±13.7[d]	560±12[d]	310±10.5[a]	165 2±9.5[d]	290±15.5[b]	190±10.2[a]	196±18[d]	329±15[a]
% fall	—	56.2	21.1	—	52.9	21.6	5	21.6	13.4
+24 hr	660±10[a]	151.4±15.7[d]	480±8[d]	308±9[a]	79.1±5[d]	250±14.8[d]	180±9.5[a]	156.6±6.8[d]	290±14[a]
% fall	3.2	78.5	32.4	0.64	77.5	32.4	10	37.3	23.7

P values, a = NS; b≤0.05; c≤0.01; d≤0.001 compared with 0 hr.

TABLE II : Serum HDL-cholesterol/total cholesterol ratio, VLDL-cholesterol and LDL-cholesterol in Triton-induced hyperlipidaemic rats after *C. longa* and *N. jatamansi* extract feeding.

	HDL-Chol./Total Chol. Ratio			*VLDL-Cholesterol (mg/dl)*		
	No treatment	*C. longa*	*N. jatamansi*	*No treatment*	*C. longa*	*N. jatamansi*
Fasting	0.592±0.098	0.525±0.078	0.561±0.081	21.18±1.0	23.6±2.0	21.9±2.1
0 hr	0.270±0.03	0.260±0.03	0.240±0.02	62.0±2.0	70.2±12.5	74.0±4.5
+6 hr	0.280±0.01[a]	0.320±0.023[a]	0.280±0.02[a]	63.2±1.6[a]	60.6±3.5[a]	62.0±4.2[a]
% deviation	+3.7	+23.08	+16.66	+1.93	−13.7	−17.6
+12 hr	0.290±0.02[a]	0.350±0.038[a]	0.290±0.03[a]	62.0±4[a]	33.0±3[b]	58.0±3.8[b]
% deviation	+7.4	+34.61	+20.83	—	−52.9	−21.6
+24 hr	0.290±0.015[a]	0.420±0.031[c]	0.300±0.03[a]	61.6±3.8[a]	15.82±1.8[c]	50.0±3[c]
% deviation	+7.4	+61.54	+25.0	−0.64	−77.4	−32.43

	LDL-Choleaterol (mg/dl)			*HDL-Cholesterol (mg/dl)*		
	No treatment	*C. longa*	*N. jatamansi*	*No treatment*	*C. longa*	*N. jatamansi*
Fasting	16.68±1.0	23.9±1.0	23.9±3.5	54.9±54	52.6±0.39	58.6±0.56
0 hr	430.0±10	436.3±16.0	465±18.5	184.1±16	183.0±15.5	170.4±2.5
+6 hr	430±19.5[a]	301.8±14.0[d]	385±10[c]	202.7±15[a]	170.5±9.5[a]	173.6±4.1[a]
% deviation	—	−30.8	−17.3	+10.1	+6.85	+1.87
+12 hr	442±15[a]	167.5±13.0[d]	339±8.7[d]	201.3±14.5[a]	107.9±5.2[c]	162.4±4.0[a]
% deviation	−2.8	−61.6	−27.1	+9.3	−41.06	−4.7
+24 hr	435±13[a]	71.9±8.5[d]	286±6.8[d]	191.4±13[a]	43.9±3.5[c]	144.0±2.5[c]
% deviation	−1.16	−83.5	−38.5	+3.96	−76.02	−15.5

P values, a = NS; b≤0.05; c≤0.01; d≤0.001 compared with 0 hr.

Total cholesterol/phospholipid ratio was also decreased significantly after these treatments (*C. longa*- from 2.81±0.3 to 0.90±0.13 : $P \leq 0.001$; *N. jatamansi*- from 1.86±0.06 to 1.5±0.04 : $P \leq 0.01$ at 48 *hr* interval).

LDL-cholesterol, VLDL-cholesterol and HDL-cholesterol/total cholesterol ratio (Table II) : A reduction of 88.6% and 65% respectively was noted in the LDL-cholesterol levels after 48 *hr* of *C. longa* and *N. jatamansi* treatment compared to 0 *hr* level. A maximum fall of 48.3% and 56.2% in VLDL-cholesterol was observed after 48 *hr* of *C. longa* and *N. jatamansi* treatment compared to 0 *hr* level.

Serum HDL-cholesterol/total cholesterol ratio in *C. longa* treated rats was significantly higher at all intervals while in *N. jatamansi* treated rats the increase occurred only after 48 *hr* of the drug administration as compared to their initial values (Table II, *N. jatamansi* ; initial 0.24±0.02; after 48 *hr* drug administration : 0.38±0.05 : $P > 0.05$).

Miller and Miller (14) have presented evidences that HDL is inversely related to total body cholesterol. They postulated that the mechanism of action may involve transport of cholesterol back to the liver, the only organ which can catabolize and excrete quantitatively important amounts of cholesterol. Glomset (10) showed that HDL alters the balance of unesterified cholesterol between plasma and cells by increasing its utilization in the lecithin/cholesterol acyltransferase (LCAT) system to form cholesterol-ester. The serum cholesterol lowering actions of *C. longa* and *N. jatamausi* extracts reflect increased serum cholesterol (LDL and VLDL) catabolism and the mechanism for this effect remains to be elucidated. The extracts also caused significant reduction in the ratio of total cholesterol/phospholipid, suggesting the protective effect of the drug on the myocardial lipids (16). The fact that *C. longa* extract exhibited better cholesterol and triglyceride lowering activity warrants further detailed studies.

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