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Effect of Dietary Coenzyme Q_{10} on Lipid Peroxidation in Adriamycin-treated Rats

- II. Effect on Mitochondrial Coenzyme Q_{10} Level and Fatty Acid Composition -

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ABSTRACT

The present study was designed to evaluate the effects of dietary coenzyme Q_{10} on mitochondrial coenzyme Q_{10} and fatty acid composition in adriamycin (ADR)-treated rats. Two experiments were conducted in rats. Experiment 1 was undertaken under the condition of simultaneous administration of ADR and coenzyme Q_{10} for 4 weeks. Experiment 2 was undertaken under the same condition as experiment 1 after feeding the experimental diets alone without administration of ADR for 4 weeks.

Heart mitochondrial coenzyme Q_{10} level of rats was greatly decreased by ADR treatment, but higher level of dietary coenzyme Q_{10} elevated this decrease to control ranges. Pretreatment with dietary supplementation of coenzyme Q_{10} showed a significant increase in myocardial coenzyme Q_{10} level. With ADR treatment, polyunsaturated fatty acids such as arachidonic acid (20 : 4) and docosahexaenoic acid (22 : 6) were decreased. However, dietary supplementation of coenzyme Q_{10} modified this decrement to some extent. In both experiment 1 and 2, the polyunsaturated fatty acids/saturated and monounsaturated fatty acids (P/S+M) ratio of ADR-treated rats tended to be lower than that of control rats.

KEY WORDS : coenzyme Q_{10} · adriamycin · fatty acid composition.

Introduction

The peroxidation of lipids is commonly described as an oxidative, oxygen-dependant deterioration of fats, notably the unsaturated fatty acids[1]. Unsaturated fatty acids undergo peroxidation as free acids, triacylglycerols or as components of phospholipid. Peroxidation of unsaturated fatty acids is usually initiated by free radicals[2]. Free radical attack on polyunsaturated fatty acids yields lipid radicals with allylic double bond. An allylic hydrogen atom is relatively easily removed to produce a radical site subject to the addition of an oxygen molecule. The addition of oxygen yields a lipid peroxy radical, which is considered a hallmark of peroxidizing lipids[3]. The significa-

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nce of lipid peroxidation and cross-linking is the reduction in celullar integrity. Biomembranes and subcelullar organelles are major sites of lipid peroxidation damage[4].

Drug-induced structural and functional damage to subcelullar organelles is believed to be responsible for myocyte mortality[5]. It was suggested that lipid peroxidation in vivo may play a major role in the cardiotoxicity of adriamycin(ADR) and other structurally similar anthracycline anticancer drugs[6][7]. But this effect is thought to be blocked by the concomitant administration of a free radical scavenger.

Several investigators[8-10] reported that coenzyme Q_{10} has membrane stabilizing and protective activities an ADR-induced toxicity. More recently, Shinozawa et al[11] stated that coenzyme Q_{10} tended to inhibit rises in plasma and liver lipid peroxidation levels induced by ADR administration, but there was no statistically significant difference between treatments. Besides, some workers suggested that dietary coenzyme Q_{10} could affect the blood and tissue coenzyme Q_{10} concentrations. Kamikawa et al[12] reported that the average coenzyme Q_{10} plasma concentration increased after coenzyme Q_{10} treatment. Another point was demonstrated by Okamoto et al[13] who adressed that it is still obscure how exogenous coenzyme Q affects the biochemical functions and the metabolism of endogenous coenzyme Q. But the interesting thing is the finding that ADR inhibits the biosynthesis of coenzyme Q_{10} by one or more mechanisms. This observation suggested that this antibiotic might act as an antagonist of coenzyme Q_{10}[14].

In this study we examined the effects of dietary coenzyme Q_{10} on mitochondrial coenzyme Q_{10} level and fatty acid composition in hearts of ADR-treated rats.

Materials and Methods

To investigate the effects of dietary coenzyme Q_{10} on mitochondrial coenzyme Q_{10} level and fatty acid composition in ADR-treated rats, two experiments were conducted. Experimental design, experimental animal and diet were the same as in the previous paper[15].

1. Biochemical analyses

At the end of each experimental period, rats were anesthetized with ethyl ether after 16-hour fasting. Heart were promptly removed, rinsed with 0.02 M tris-buffer (pH 7.4) and blotted in filer paper and weighed. Heart homogenate and mitochondrial fraction of rats were prepared as described previously[15]. Coenzyme Q_{10} of the heart mitochondrial fraction was assayed by the method of Ikenoya et al[16]. All extraction steps for mitochondrial fraction were performed in the absence of direct sunlight and incandescent light to minimize the photochemical degradation of coenzyme Q. The concentration of coenzyme Q_{10} was determined by comparing the peak areas on HPLC with those of the correspondig authentic coenzyme Q_{10} treated in the same manner as the sample. Operating condition of HPLC was shown in Table 1. Lipids in heart mitochondria were extracted by the modified method of Folch et al[17]. Nonvolatile fatty acids in lipid extracts were transesterified to volatile methyl esters by heating with BF_3-methanol by the method of Delomore and Lupien[18]. The esters were extracted by adding 2 ml of heptane, shaking briefly, and centrifuging until both layers were clear. Then the supernatants, resulting methyl esters wers used for analysis of fatty acid in mitochondrial lipid extracts by gas chromatography. Instrument and operating conditions for gas-liguid chromatography were

Table 1. Operation condition for the determination of coenzyme Q_{10} by high performance liquid chromatography

Instrument	HPLC, Waters
Column	C_{18} reverse phase, 30cm
Mobile phase	Mixed solution of Ethanol : Methanol : $HClO_4$ (700 : 300 : 1) with 7.0g/l $NaClO_4$ H_2O
Flow rate	1.2ml/min
Detector	UV 275nm
Sample injection	5μℓ
Temperature	25℃
Attenuation	16
Chart speed	2cm

described in Table 2. Identification of the esters was performed by comparison of retention times with those of the standard esters chromatographed under the same conditions. Peak areas of each fraction were calculated as % of total area.

2. Statistical analyses

In the statistical analysis, the treatment effects on both experiment 1 and 2 were followed by one-way analysis of variance and Duncan's new multiple range test. Linear-regression analysis between mitochondrial coenzyme Q_{10} and lipid peroxide levels was conducted by the methods described in Ott[19].

Table 2. Operation condition for the determination of mitochondrial fatty acid in rat heart by gas chromatography

Instrument	G.C.(Hewlett Packard 5890A), Integrator(Hewlett Packard 3390A)
Column	PEG20M SCOT fused silica capillary column(25m, 0.32mm)
Temperature	Injection : 250℃ Detector(FID) : 250℃ Column oven : 160℃ to 210℃, 4℃/min
Flow rate	Carrier(He) : 3mℓ/min, split ratio 60 : 1 H_2 : 20mℓ/min Air : 300mℓ/min
Sample injection	3μℓ
Attenuation	2
Chart speed	1cm/min
Peak width	0.02
Threshold	2

Results and Discussion

1. Effects of dietary coenzyme Q_{10} on mitochondrial coenzyme Q_{10} level of rat heart

Mitochondrial coenzyme Q_{10} level of rat heart in both experiment 1 and 2 was determined by HPLC. As shown in Table 3, dietary coenzyme Q_{10} significantly influenced on heart mitochondrial coenzyme Q_{10}. In general, heart mitochond-

Table 3. Effect of dietary coenzyme Q_{10} on heart mitochondrial coenzyme Q_{10} in ADR-treated rat

Group	Coenzyme Q_{10}			
	Exp. 1		Exp. 2	
	ng/mg protein	%	ng/mg protein	%
C	142.4± 20.0[a]	0	132.7± 25.8[a]	0
A1Q0	76.2± 19.0[b]	-46.5	84.0± 28.1[b]	-38.8
A1Q1	44.7± 12.6[c]	-68.6	64.2± 28.1[bc]	-53.2
A1Q2	110.1± 16.7[d]	-22.7	176.9± 50.6[a]	28.9
A2Q0	57.9± 16.7[bc]	-59.3	39.6± 14.3[c]	-71.1
A2Q1	77.6± 23.9[b]	-45.5	94.7± 24.9[b]	-31.0
A2Q2	112.6± 17.3[d]	-20.9	172.1± 38.7[a]	-25.4

1) Values shown are the mean± S. D.(n=5)

2) Values with a common superscript letter within the same column are not significantlly different($p<0.05$).

rial coenzyme Q_{10} level was apparently decreased by ADR treatment but higher level of exogenous coenzyme Q_{10} elevated this decrease to control ranges. Besides, prior supplementation of dietary coenzyme Q_{10} increased all the more myocardial coenzyme Q_{10} level. Although there appeared to be a trend toward increasing heart mitochondrial coenzyme Q_{10} content as the supplementation level of dietary coenzyme Q_{10} was increased, great variation was noted.

Several investigators suggested that dietary coenzyme Q_{10} could affect the blood and tissue coenzyme Q_{10} concentrations[12)13)]. On the other hand, Folkers et al[14)] reported that ADR inhibited the mitochondrial biosynthesis of coenzyme Q_{10}. They also suggested that ADR could inhibit the biosynthesis of coenzyme Q_{10} either by direct competition at one or more of the sites of three quinone precursors or by indirect depression of the biosynthesis through damage of the mitochondria. Particularly, it was found that supplementary coenzyme Q_{10} prevented the in vitro inhibiton by ADR of coenzyme Q_{10}-enzymes. It is needed to note that, although the antioxidant function of coenzyme Q_{10} has not yet been clarified, it may antagonize the deteriorative reaction of membrane lipid caused by ADR because of their structural similarity. Therefore, it may be speculated that the supplementation of coenzyme Q_{10} to ADR-treated cancer patients might circumvent the depressed biosynthesis and the inhibited functionality of coenzyme Q_{10}-enzymes to improve bioenergetics and, in turn, reduce the cardiotoxicity.

Correlations of lipid peroxide value shown in the previous paper[15)] with coenzyme Q_{10} in rat heart mitochondrial fraction of both experiments are presented in Fig. 1 and 2. Lipid peroxide value of heart mitochondrial fraction was negatively associated with cardiac coenzyme Q_{10} level in both

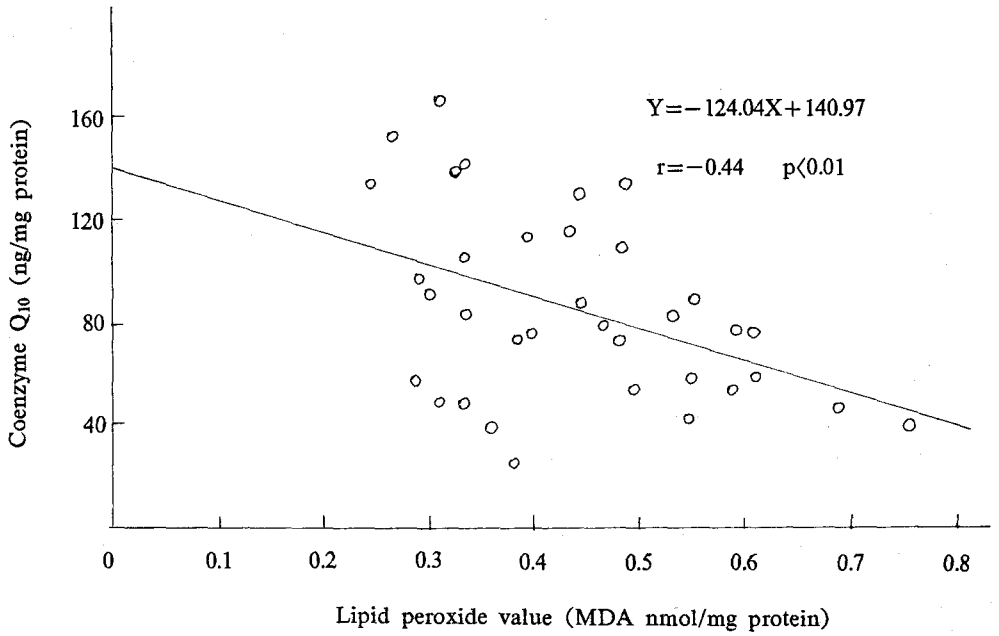

Fig. 1. Correlation of lipid peroxide value with coenzyme Q_{10} in rat heart mitochondrial fraction of rats in Exp. 1.

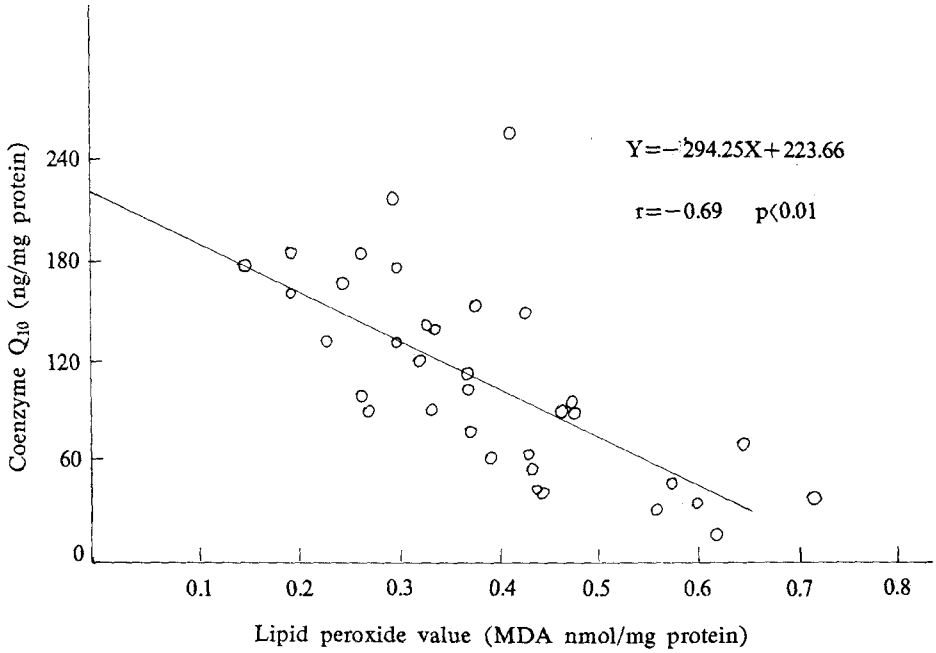

Fig. 2 Correlation of lipid peroxide value with coenzyme Q_{10} in rat heart mitochondrial fraction of rats in Exp. 2.

experiments. This finding supports the report by Marubayashi et al[20] that preservation of mitochondrial function by coenzyme Q_{10} resulted from its action as an antioxidant on lipid peroxidation.

2. Effects of dietary coenzyme Q_{10} mitochondrial fatty acid composition of rat heart

Table 4 and 5 presented the fatty acid composition of heart mitochondrial fractions of rats as influenced by ADR treatment and dietary coenzyme Q_{10}. Palmitic (16 : 0), stearic (18 : 0), linoleic (18 : 2) and arachidonic (20 : 4) acids were the major fatty acids in the heart mitochondrial fraction. With ADR treatment, polyunsaturated fatty acids such as arachidonic acid (20 : 4) and docosahexaenoic acid (22 : 6) were decreased. But exogenous coenzyme Q_{10} modified this decrement to some extent.

The polyunsaturated fatty acid/saturated and monounsaturated fatty acids (P/S+M) ratio in membrane lipid seemed to be an important determinant of membrane fluidity[21]. Various membrane functions such as selective permeability, enzyme activity, receptor availability, and ion transport have been shown to be influenced by membrane fluidity. The changes in the fatty acids of membranes occurring with lipid peroxidation could lead to membrane dysfunction through changes in fluidity[22]. Therefore, the P/S+M ratio in heart mitochondria was calculated and tabulated in Table 4 and 5. In both experiment 1 and 2, the P/S+M ratio of ADR-treated groups tended to be lower than that of control group. Also, present results showed that exogenous coenzyme Q_{10} increased P/S+M ratio. This phenomenon indicated that ADR treatment and dietary coenzyme Q_{10} seems to have influenced on the composition of fatty acid of mitochondrial membrane and thus the membrane fluidity.

At present, though the relationship between

Table 4. Fatty acid composition of the heart mitochondria of rats as influenced by ADR treatment and dietary coenzyme Q_{10} in Exp. 1

(%)

Group / F.A.	C	A1Q0	A1Q1	A1Q2	A2Q0	A2Q1	A2Q2
12 : 0	2.01	7.41	0.67	1.36	0.62	7.64	1.58
14 : 0	2.20	1.91	1.72	2.83	1.14	2.06	1.69
15 : 0	—	—	0.28	1.36	—	—	—
16 : 0	20.58	21.34	21.47	23.69	23.30	17.60	19.10
18 : 0	33.66	35.76	35.89	40.38	41.61	29.49	37.14
18 : 1	5.46	4.56	6.69	1.49	4.49	6.08	5.14
18 : 2	11.43	12.88	12.35	8.35	13.70	12.74	9.60
20 : 0	—	—	—	—	—	0.38	—
20 : 1	—	—	1.00	1.24	0.69	0.71	—
20 : 2	—	—	—	2.19	—	—	—
20 : 4	18.58	15.09	16.81	12.84	12.30	17.38	21.11
20 : 3	—	—	—	—	—	1.01	—
20 : 5	—	—	—	0.57	—	1.23	—
22 : 6	6.09	1.06	3.12	3.70	2.16	3.68	4.66
Total	100.01	100.01	100.00	100.00	100.01	100.00	100.02
P/S+M	0.565	0.409	0.477	0.382	0.392	0.563	0.547

1)Fatty acids of $C_{18:3}$, $C_{18:4}$, and $C_{19:0}$ were not detected.

2) P/S+M ; Polyunsaturated fatty acids/ Saturated and Monounsaturated fatty acids.

Table 5. Fatty acid composition of the heart mitochondria of rats as influenced by ADR treatment and dietary ccenzyme Q_{10} in Exp. 2

(%)

Group / F.A.	C	A1Q0	A1Q1	A1Q2	A2Q0	A2Q1	A2Q2
12 : 0	0.68	1.68	1.49	0.60	1.73	0.61	1.01
14 : 0	1.71	2.39	1.24	1.10	0.83	1.80	1.32
15 : 0	0.44	—	—	0.29	—	0.48	0.36
16 : 0	23.86	25.06	17.26	15.96	21.67	19.94	16.51
18 : 0	33.22	44.95	29.59	30.27	38.97	36.11	31.69
18 : 1	2.51	1.89	7.25	7.05	3.19	5.92	5.88
18 : 2	11.29	18.50	15.07	13.26	11.78	11.11	13.46
18 : 4	—	0.47	—	—	0.48	—	—
20 : 4	16.90	13.71	23.19	22.55	19.31	18.39	20.38
22 : 6	9.39	1.35	4.91	8.92	2.04	5.66	9.39
Total	100.00	100.00	100.00	100.00	100.00	100.02	100.00
P/S+M	0.602	0.316	0.760	0.809	0.506	0.542	0.761

1) Fatty acids of $C_{18:3}$, $C_{19:0}$, $C_{20:0}$, $C_{20:1}$, $C_{20:2}$, $C_{20:3}$, and $C_{20:5}$ were not detected.

specific lipid composition and membrane function is not well understood, it is generally considered that the fatty acid composition of membrane phospholipids may affect the permeability of membranes and therefore the function of the membrane[23)24)]. It is well known that polyunsaturated fatty acids are easily oxidized and form hydroperoxides both in vivo and in vitro. Recently, Mouri et al[25)] reported that feeding of the marine oil changed the composition of fatty acids in rat liver phospholipids, especially comprising reduction of arachidonic acid. They also demonstrated that the levels of TBA-reacting substances in the 100% cod liver oil group were raised four times higher than those in the corn oil group and interpreted these phenomena to be associated with lipid peroxidation. Similar data were reported by several investigators. Kameda et al[26)] suggested that once erythrocytes from vitamin E deficient rats were exposed to H_2O_2, the erythrocyte membrane fluidity was significantly reduced, which was presumed to be accompanied by an increase in the hemolysis and lipid peroxide formation. And they showed in the same study that large decreases in arachidonic acid and phospholipid were also observed. The decrease in arachidonic acid, caused by lipid peroxide formation, was also shown in liver microsomes by May and McCay[27)]. They reported that the polyunsaturated fatty acids (primarily arachidonic acid) in phosphatidylethanolamine and phosphatidylcholine were consumed by the NADPH oxidase-catalized lipid peroxide formation. They also showed that most of the fatty acid loss could be accounted for by the decrease in arachidonic acid in the β-position. The loss of the phospholipids from the membranes might have resulted in disorganization of the lipid bilayer matrix, and could have caused damage to the cellular function.

On the other hand, Ohki et al[28)] observed that NADPH-dependent lipid peroxidation decreased the content of polyunsaturated fatty acids, arachidonic acid (20 : 4) and docosahexaenoic acid (22 : 6). But it was reported that addition of α-tocopherol after peroxidation resulted in a slight inhibition of peroxide and small alteration in fatty acid composition. These reports were consistent with the present data that ADR-induced lipid peroxidation reduced the levels of polyunsaturated fatty acids. There was a tendency of higher polyunsaturated fatty acid levels in coenzyme Q_{10}-supplemented groups.

In conclusion, pretreatment of dietary coenzyme Q_{10} increased more myocardial coenzyme Q_{10} level. With ADR treatment, polyunsaturated fatty acids such as arachidonic acid and docosahexaenoic acid were decreased. However, dietary coenzyme Q_{10} modified this decrement to some extent.

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식이 중의 Coenzyme Q_{10}첨가가 Adriamycin을 투여한 흰쥐의 체내 지질과산화에 미치는 영향

-II. 미토콘드리아내의 Coenzyme Q_{10} 수준과 지방산 조성에 미치는 영향-

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식이 중에 첨가된 coenzyme Q_{10}이 ADR을 투여한 흰쥐의 심장 미토콘드리아 분획의 coenzyme Q_{10} 수준과 지방산 조성에 미치는 영향을 구명하기 위하여 두가지 실험을 실시하였다. 실험 1에서는 4주간 실험식이를 공급함과 동시에 ADR을 투여하였으나 실험 2에서는 4주간 실험식이만을 급여한 후 다시 4주간 실험1과 같은 방법으로 ADR투여와 실험식이 공급을 병행하였다. 실험군은 실험1과 2에서 모두 ADR 2수준 (1.0mg/kg B.W./week, 2.0mg/kg B.W./weed)과 coenzyme Q_{10} 3수준 (무첨가군, 0.1g/kg diet 및 0.5g/kg diet)에 의한 6개의 실험군과 basal diet만을 공급하는 대조군을 설정하였다. 본 실험에서 얻어진 실험결과를 요약하면 심장 미토콘드리아 내의 coenzyme Q_{10}의 함량은 ADR투여로 크게 감소되었으나 특히 식이중의 coenzyme Q_{10}을 고수준으로 급여함에 따라 유의적으로 증가되었으며 실험1에 비해 실험2에서 coenzyme Q_{10} 급여효과가 더욱 크게 나타났다. 지방산 조성은 모든 실험군에서 plamitic(16 : 0), stearic(18 : 0), linoleic acid(18 : 2)와 arachidonic acid(20 : 4)가 주요지방산으로서 80% 이상을 차지하였고 ADR투여로 인해 arachidonic acid(20 : 4)와 docosahexaenoic acid(22 : 6)등의 고불포화지방산 함량이 감소되는 경향이었으며 coenzyme Q_{10} 급여에 의해 이러한 감소가 조절되었다.