行政院國家科學委員會專題研究計畫 成果報告

輔?Q10 與維生素 A，C，E 對代謝症候群受試者之氧化壓力，抗氧化酵素活性與發炎相關性之探討

研究成果報告(精簡版)

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行政院國家科學委員會補助專題研究計畫 ■成果報告
□期中進度報告

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中文摘要

輔酶 Q10 為粒線體膜上的內生性脂溶性抗氧化劑。本研究目的是要了解代謝症候群之受試者輔酶 Q10 與脂質過氧化、抗氧化酵素活性與發炎反應之相關性。代謝症候群為根據行政院衛生署國民健康局 2007 年之診斷標準判斷之（病例組，72 位）與收集年齡配對之健康受試者（對照組，105 位）。測量受試者血漿輔酶 Q10、脂質過氧化指標（丙二醛，Malondialdehyde；MDA）、抗氧化酵素【過氧化氫酶（Catalase；CAT）、超氧化物歧化酶（Superoxidase dismutase；SOD）及麩胱甘肽過氧化酶(Glutathionine peroxidase;GPX)】活性及發炎指標(高敏感 C-反應性蛋白,high senstivity C-reactive protein;hs-CRP)濃度，並利用問卷調查了解受試者日常生活習慣。結果顯示，病例組之年齡、血壓、體位測量、血液生化值（禁食血糖及血脂質）及發炎指標（hs-CRP）顯著高於對照組。病例組之血漿輔酶 Q10 濃度顯著高於對照組，抗氧化酵素（CAT、SOD 及 GPX）活性則顯著低於對照組。利用複回歸分析發現，血漿輔酶 Q10 濃度與脂質過氧化物（MDA）濃度及抗氧化酵素（SOD 及 GPX）活性呈顯著之正相關，且在調整代謝症候群之危險因子後此顯著統計依然存在。本研究推論代謝症候群之患者，可能承受較高的氧化壓力而啟動了抗氧化機制，使輔酶 Q10 濃度有顯著增加。

關鍵字：輔酶 Q10、代謝症候群、抗氧化、氧化壓力、發炎

Abstract

Background & aim: A higher oxidative stress may contribute to the pathogenesis of metabolic syndrome (MS). Coenzyme Q10 is an endogenous lipid-soluble antioxidant located in the mitochondrial membrane. The purpose of this study was to investigate the relationship between coenzyme Q10 concentration and lipid peroxidation, and antioxidant enzymes activities.

Methods: The inclusion criteria of MS are according to the Bureau of Health Promotion, Department of Health (2007) in Taiwan were assigned to the case group (n = 72). The control group (n = 105) comprised healthy individuals with normal blood biochemical values. The plasma coenzyme Q10 concentration, lipid profiles, malondialdehyde (MDA) and antioxidant enzymes activities [catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx)] were measured.

Results: Subjects with MS had significant increases in plasma coenzyme Q10 level and the ratios of coenzyme Q10 to lipid profiles ($p < 0.01$), but antioxidant enzymes activities were lower compared to those of the control group ($p < 0.01$). However, the MDA level was not significantly different between case and control groups. The plasma coenzyme Q10 concentration and the ratio of coenzyme Q10 to lipid profiles were positively correlated with MDA activities and SOD activities, but not significant correlated with CAT or GPx.

Conclusion: Subjects with MS showed an incensement of plasma coenzyme Q10 concentration. An increase plasma coenzyme Q10 might indicate an adaptive response to oxidative stress in MS patients.

KEYWORDS: Coenzyme Q10, Metabolic syndrome, Oxidative stress, Antioxidant enzymes, Case-control study

Introduction

The metabolic syndrome (MS) represents a clustering of physiological or anthropometric abnormalities [1] which is now recognized as a significant risk factor of cardiovascular disease and type II diabetes [2]. Ford and Giles (2003) reported there were 24 % adults and 25% elderly in the US had MS [3, 4]. A recent report from the Elderly Nutrition and Health Survey in Taiwan (NAHSIT) conducted during 1999-2000, the prevalence rates of MS were 26% in men and 47% in women, respectively [5]. The MS, including insulin resistance, type II diabetes, hypertension, dyslipidemia and visceral obesity may increase oxidative stress [6-8]. Elevations in oxidative stress may then contribute to impaired vascular function, inflammation, thrombosis and atherosclerosis, and ultimately to vascular disease [9].

Coenzyme Q10 (also called ubiquinone) is a lipid-soluble benzoquinone with 10 isoprenyl units in the side chain and is a key component of the mitochondrial respiratory chain for adenosine triphosphate (ATP) synthesis [10, 11]. Coenzyme Q10 is an intracellular antioxidant that protects the membrane phospholipids, mitochondrial membrane protein and low-density lipoprotein-cholesterol (LDL-C) from free radical-induced oxidative damage [12, 13]. Adults with the MS have suboptimal concentrations of antioxidants [14]. Until to date, the relationship between coenzyme Q10 and the prevention of the MS are still unclear. In addition, the clinical trials have revealed that oxidative stress may increase free oxygen reactive species (ROS) formation and reduce antioxidants defenses [15, 16]. Antioxidant enzymes such as catalase (CAT), superoxide dismutase (SOD) and glutathione peroxidase (GPx) are the first line of defense against ROS, and a decrease in their activities contributes to the oxidant attack on cells [17]. As a result, we designed a case-control study to investigate the relationship between coenzyme Q10 concentration and oxidative stress, as well as antioxidant enzymes activities in subjects who suffering from MS.

Materials and Methods

Subjects

The current study was designed as a case-control study. Subjects with MS (n = 72) were recruited from the department of family and community medicine of Chung Shan Medical University Hospital in Taiwan. The inclusion criteria of MS in adult are according to the Bureau of Health Promotion, Department of Health in Taiwan (2007), if subjects had 3 of the following 5 characteristics: (1) abdominal obesity (waist circumference ≥ 90 cm in men and ≥ 80 cm in women), (2) impaired fasting glucose (≥ 5.6 mmol/L), (3) hypertriglyceridemia (≥ 1.7 mmol/L), (4) low HDL-C (< 1.0 mmol/L in men and < 1.3 mmol/L in women), and (5) increased blood pressure (SBP≥ 130 mmHg and DBP ≥ 85 mmHg). Subjects using antidiabetic or antihypertensive or lipid-lowering medications were considered to have elevated fasting blood glucose or elevated blood pressures or dyslipidemia, respectively. Case subjects with liver, renal diseases or undergoing statin therapy were excluded. Control subjects (n = 105) were exhibited normal blood biochemical values, including fasting glucose < 6.11 mmol/L, blood urea nitrogen (BUN) < 7.9 mmol/L, creatinine < 123.8 μmol/L, alkaline phosphates < 190 U/L, glutamic oxaoloacetic transaminase (GOT) < 35 U/L and glutamic pyruvate transaminase (GPT) < 45 U/L. Control subjects did not have any illnesses and a history of gastrointestinal disorder, hypertension, hyperlipidemia, liver and renal disease, diabetes, or other metabolic disease. Subjects currently taking vitamin supplements were also excluded. Informed consent was obtained from each subject. This study was approved by the Institutional Review Board of Chung Shan Medical Hospital in Taiwan. The age, blood pressures, smoking and exercise habits of all subjects were recorded.

Blood pressure was measured in each patient after resting for at least 5 min. Body weight, height, waist and hip circumferences were measured and the body mass index (BMI; kg/m^2) and the waist to hip ratio were then calculated.

Blood collection and biochemical measurement

Fasting venous blood samples (15 mL) were obtained to estimate hematological and vitamin status. Blood specimens were collected in Vacutainer tubes (Becton Dickinson, Rutherford, NJ, USA) with or without containing EDTA as an anticoagulant as needed. Serum and plasma were prepared and then frozen (-80℃) for storage until analysis. Blood lipid profiles [i.e., total cholesterol (TC), triacylglycerol, LDL-C, high density lipoprotein-cholesterol (HDL-C)] were measured using an automated biochemical analyzer. Automated measurements of high sensitivity C-reactive protein (hs-CRP) concentration were performed using particle-enhanced immunonephelometry with an Immage analyzer.

Plasma coenzyme Q10 was measured using high-performance liquid chromatography (HPLC) according to the method of Chu et al. [18] and Littarru et al. [19]. The mean intra- and inter-assay coefficients of fasting plasma coenzyme Q10 variability were 1.8% and 2.7%, respectively. The mean analytical recovery of plasma coenzyme Q10 was 101.1%. Plasma MDA was determined using the thiobarbituric acid reactive substances (TBARs) method, as described by Botsoglou [20] and Chung et al. [21]. The mean intra- and inter-assay coefficients of plasma MDA variability were 1.2% and 3.2%, respectively. Red blood cells (RBCs) were diluted with 25x sodium phosphate buffer for SOD and GPx measurements and 250x sodium phosphate buffer for CAT measurement. The methods for measuring CAT, SOD and GPx in RBCs have previously been described [21], and measurements were performed spectrophotometrically at 240 nm, 325 nm and 340 nm, respectively. Protein contents of RBCs were determined based on the Biuret reaction of the BCA kit (Thermo, Rockford, IL, USA). The mean intra- and inter-assay coefficients of protein variability were 0.2% and 3.2%, respectively, in RBCs. The antioxidant enzymes activity levels were expressed as unit/mg of protein. All analyses were performed in duplicate and the variations of repeated determinations were within 10% of the same sample. The analyses of plasma MDA and antioxidant enzymes activities were completed within 7 days.

Statistical analyses

Data were analyzed using SigmaStat statistical software (version 2.03; Jandel Scientific, San Rafael, CA, USA). The normal distribution of variables was evaluated using the Kolmogorov-Smirnov test. Differences in subjects' demographic data and the hematological measurement data between case and control groups were analyzed using the Student's t-test or the Mann-Whitney rank sum test. For categorical response variables, differences between two groups were assessed using the Chi-square test or the Fisher's exact test. To examine the relationships of the plasma coenzyme Q10 concentration and the ratio of coenzyme Q10 to lipid profiles with oxidative stress (MDA) and antioxidant enzymes activities (CAT, SOD, GPx), multiple linear regression analyses were used. We adjusted the potential confounders of MS, including age, fasting blood sugar, systolic blood pressure, waist circumference, and hs-CRP. Data were expressed as means ± standard deviations (SD). Results were considered statistically significant at $p < 0.05$.

Results

Table 1 shows the demographic data and health characteristics of the subjects. Subjects in the case group had significantly higher values for age, systolic blood pressure, diastolic blood pressure, body mass

index, waist circumference, waist to hip ratio, hematological parameters (i.e., fasting glucose, TG, LDL-C, TC/HDL-C, hs-CRP) and lower HDL-C level than the control group.

The plasma coenzyme Q10 concentration, lipid peroxidation and antioxidant enzymes activities are shown in Figure 1 and 2. Subjects in the case group had significant higher plasma coenzyme Q10 concentration ($p < 0.01$), the ratio of coenzyme Q10 to lipid profiles (Coenzyme Q10 / TC and Coenzyme Q10 / LDL-C) ($p < 0.01$) but lower CAT ($p = 0.02$), SOD ($p < 0.01$) and GPx activities ($p < 0.01$) than the control group. However, the MDA level was not significantly different between case and control groups ($p = 0.07$).

The correlations between coenzyme Q10 concentration, lipid peroxidation and antioxidant enzymes activities in the case group are shown in Table 2. The plasma coenzyme Q10 concentration and the ratio of coenzyme Q10 to lipid profiles were significantly positively correlated with MDA and SOD activities after adjusting for the potential confounders of MS. No significant correlation existed between the plasma coenzyme Q10 and CAT or GPx levels.

Discussion

The present study showed a statistically significant link between the plasma coenzyme Q10 concentration and MS. Subjects suffering from MS showed a significant higher level of plasma coenzyme Q10 than the control group (Figure 1) and the plasma coenzyme Q10 was significantly positively correlated with lipid peroxidase (MDA) ($\beta = 0.44$, $p < 0.01$) even after adjusted for the potential confounders of MS ($\beta = 0.43$, $p = 0.02$) (Table 2). This result was similar to a study conducted by Miles et al. (2004) [22]; they proposed that an increment in the level of coenzyme Q10 may be a part of the natural antioxidant defense response to certain components of MS.

In present study, we also examined the correlations between plasma coenzyme Q10 and the potential confounders of MS after adjusted for age (data not shown). The plasma coenzyme Q10 was significantly positively correlated with systolic blood pressure ($\beta = 15.03$, $p < 0.01$), diastolic blood pressure ($\beta = 9.48$, $p < 0.01$), body mass index ($\beta = 3.18$, $p = 0.03$), waist circumference ($\beta = 9.29$, $p = 0.04$), the ratio of waist to hip ($\beta = 0.07$, $p = 0.02$), fasting glucose ($\beta = 1.81$, $p < 0.01$), TC ($\beta = 0.56$, $p < 0.05$), and LDL-C ($\beta = 0.71$, $p < 0.01$), but not correlated with TG ($\beta = 0.03$, $p = 0.91$) or HDL-C ($\beta = 0.04$, $p = 0.69$). The MS represents a clustering of physiological and anthropometric abnormalities, subjects may under a higher oxidative stress [6-8] and an endogenous antioxidant defense should increase in response to oxidative stress [23], such as coenzyme Q10.

In addition to oxidative stress, we assessed the activities of the major antioxidant enzymes directly involved in the neutralization of ROS. The activities of CAT, SOD, and GPx were significantly lower in the case group compared to those of the control group (Figure 2). As shown in Table 2, plasma coenzyme Q10 was positively correlated with SOD activities, even after adjusting for the potential confounders. The activity of SOD may increase to protect against lipid peroxidation and against ROS [24, 25] and synergistic with coenzyme Q10to uptake of superoxide radical to form oxygen and hydrogen peroxide.

Coenzyme Q10 can be synthesized in tissue from farnesyl diphosphate and tyrosine [26, 27] and also can be obtained from food (especially in meat, fish, nuts, and some oils) [28], however, total absorption of coenzyme Q10 from food is thought to be less than 10% [27]. Coenzyme Q10 is transported by lipids and lipoprotein (especially LDL-C, 58%) in the blood [29]. Although we did not analysis the content of

coenzyme Q10 from food, we have examined the relations between the plasma coenzyme Q10 and lipid profiles. Subjects with MS showed a higher values of blood lipid (Table 1), and there was a significantly positive correlation between coenzyme Q10 and LDL-C ($\beta = 0.68$, $p < 0.01$). Coenzyme Q10 is a lipid-soluble antioxidant, therefore, the plasma coenzyme Q10 level was higher in the case group may due to the higher level of blood lipid.

Our study has two limitations. First, the number of participants was small, although we did recruit more subjects than we expected to recruit (sample size calculation: we expected the differences in mean levels of plasma coenzyme Q10 between case and control groups were to be 0.2 ± 0.3 μmol/L, hence the desired power was set at 0.8 to detect a true effect, and $\alpha = 0.05$ with a minimal simple size of 40 participants in each group). Second, this study was the absence of age matched between case and control groups; as a result, we try to limit these biases by adjusting for the potential confounders of MS in the statistical tests.

Subjects suffer from MS were exposed to a higher level of oxidative stress and coenzyme Q10 concentration. An increment in the level of coenzyme Q10 may be a part of the natural antioxidant defense response to certain components of MS. Lager studies are needed to be confirmed the effect of coenzyme Q10 in MS patients.

Conflict of Interest

The authors have no conflict of interest.

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Table 1 Characteristic of subjects

	Case (n = 72)	Control (n = 105)	*p* values
Male / Female (n)	43 / 29	52 / 53	0.24
Age (y)	54.8 ± 12.9	50.4 ±9.2	< 0.01
Systolic blood pressure (mmHg)	141.9 ± 11.8	118.8 ± 16.8	< 0.01
Diastolic blood pressure (mmHg)	88.5 ± 10.3	77.7 ± 9.8	< 0.01
Body mass index (kg/m^2)	29.1 ± 5.8	24.4 ± 3.5	< 0.01
Waist circumference (cm)	96.0 ± 12.4	80.6 ± 13.6	< 0.01
Waist to hip ratio	0.93 ± 0.07	0.85 ± 0.11	< 0.01
Fasting glucose (mmol/L)	7.5 ± 2.6	5.2 ± 1.1	< 0.01
TC (mmol/L)	4.9 ± 1.0	5.1 ± 0.9	0.19
TG (mmol/L)	1.9 ± 0.9	1.3 ± 0.6	< 0.01
LDL-C (mmol/L)	3.2 ± 0.9	2.9 ± 0.7	0.04
HDL-C (mmol/L)	1.2 ± 0.3	1.4 ± 0.4	< 0.01
TC / HDL-C	4.4 ± 1.2	3.9 ± 1.2	< 0.01
hs-CRP (mmol/L)	3.7 ± 7.0	1.9 ± 3.6	0.01
Current smoker[2], n (%)	15 (20.8%)	13 (12.4%)	0.19

[1]Mean ± SD. [2]Current smoker: individuals currently smoking one or more cigarettes per day.
HDL-C, high-density lipoprotein-cholesterol; hs-CRP, high sensitivity C-reactive protein; LDL-C, low density lipoprotein-cholesterol; TC, total cholesterol; TG, triglyceride.

Table 2 The correlations between plasma coenzyme Q10 and the ratios of coenzyme Q10 to lipid profiles, lipid peroxidation and antioxidant enzyme activities after adjusting for the potential confounders in the case group

	Plasma coenzyme Q10 (μmol/L)	Coenzyme Q10/TC (μmol/mmol)	Coenzyme Q10/TG (μmol/mmol)	Coenzyme Q10/LDL-C (μmol/mmol)
	β [1] (*p* value)			
MDA (μmol/L)				
Model 1[2]	0.44 (< 0.01)	2.20 (0.01)	0.09 (0.39)	1.51 (< 0.01)
Model 2[3]	0.43 (0.02)	2.04 (0.02)	0.12 (0.31)	1.42 (< 0.01)
CAT (unit/mg protein)				
Model 1	2.64 (0.45)	0.57 (0.97)	0.37 (0.87)	-1.54 (0.87)
Model 2	2.24 (0.53)	-2.06 (0.91)	-0.29 (0.90)	-3.69 (0.72)
SOD (unit/mg protein)				
Model 1	25.21 (< 0.01)	116.59 (< 0.01)	19.78 (< 0.01)	54.42 (0.02)
Model 2	23.79 (< 0.01)	107.02 (0.01)	19.17 (< 0.01)	49.76 (0.04)
GPx (unit/mg protein)				
Model 1	-0.99 (0.74)	-3.28 (0.83)	0.09 (0.96)	5.09 (0.54)
Model 2	-1.02 (0.74)	-6.01 (0.69)	0.13 (0.95)	3.46 (0.69)

[1]Regression coefficient.
[2]None adjusted.
[3]Adjusted for age, fasting blood sugar, systolic blood pressure, waist circumference and high sensitivity C-reactive protein.
CAT, catalase activity; GPx, glutathione peroxidase;; LDL-C, low density lipoprotein-cholesterol; MDA, malondialdehyde; SOD, superoxide dismutase; TC, total cholesterol; TG, triglyceride

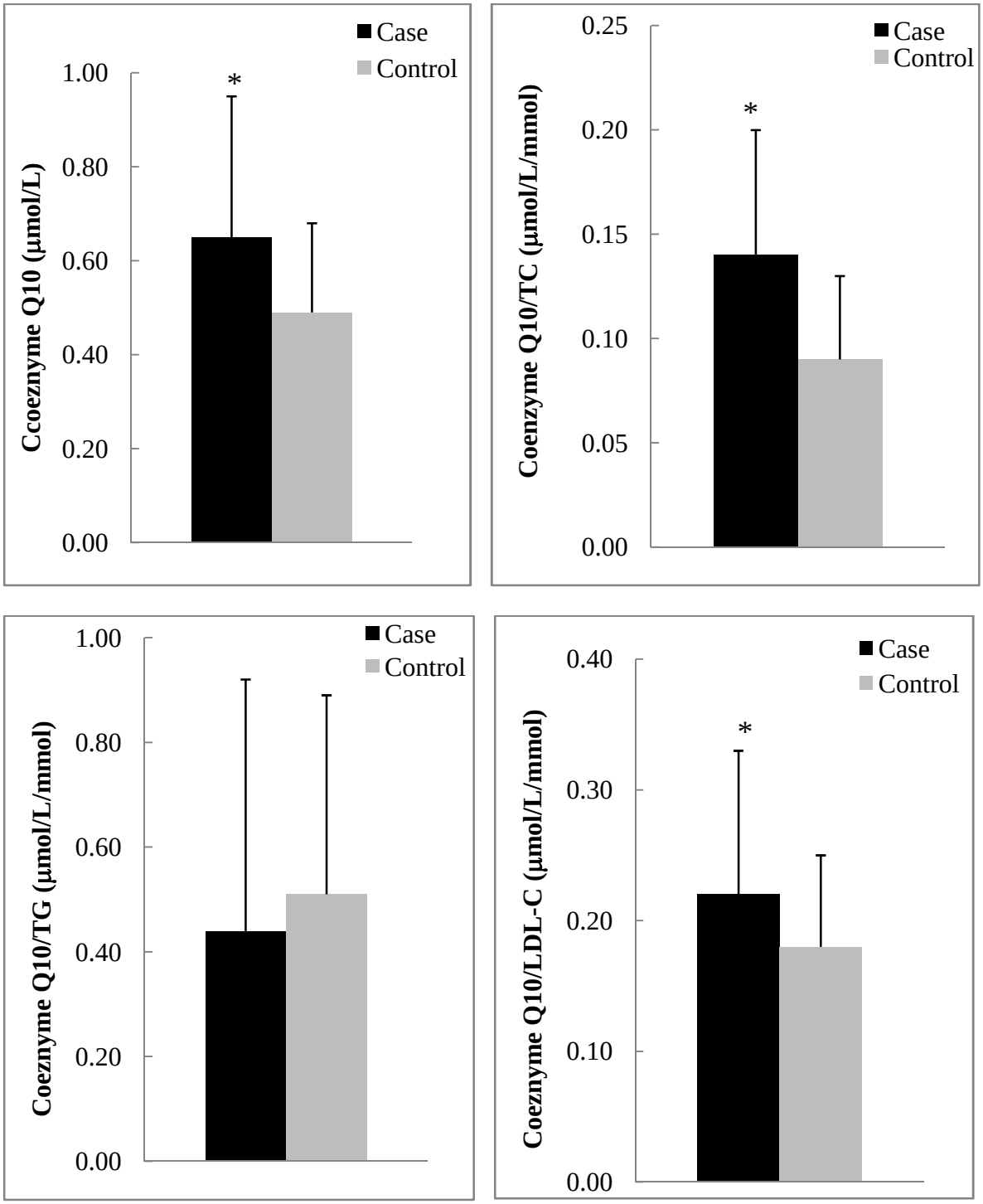

Figure 1 Concentrations of plasma coenzyme Q10 and the ratios of coenzyme Q10 to lipid profiles.

LDL-C, low density lipoprotein-cholesterol; TC, total cholesterol; TG, triglyceride.

*Values were significantly different between case and control groups; $p < 0.05$.

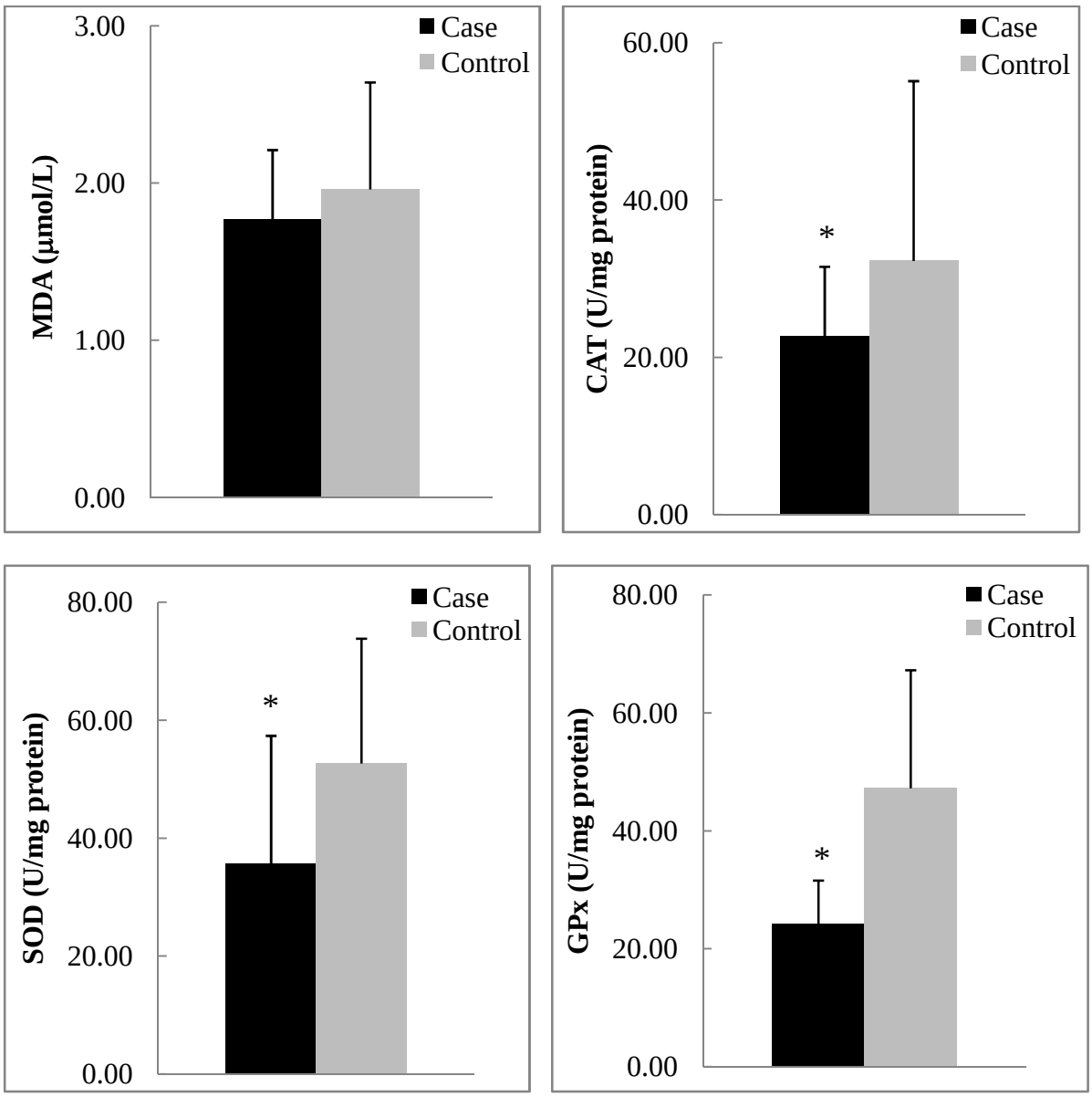

Figure 2 Concentration of lipid peroxidation and antioxidant enzymes activities.

CAT, catalase; GPx, glutathionine peroxidase; MDA, malondialdehyde; SOD, superoxide dismutase. *Values were significantly different between case and control groups; $p < 0.05$.

國科會補助專題研究計畫成果報告自評表

請就研究內容與原計畫相符程度、達成預期目標情況、研究成果之學術或應用價值（簡要敘述成果所代表之意義、價值、影響或進一步發展之可能性）、是否適合在學術期刊發表或申請專利、主要發現或其他有關價值等，作一綜合評估。

1. 請就研究內容與原計畫相符程度、達成預期目標情況作一綜合評估

■ 達成目標

□ 未達成目標（請說明，以 100 字為限）

□ 實驗失敗

□ 因故實驗中斷

□ 其他原因

說明：

2. 研究成果在學術期刊發表或申請專利等情形：

論文：□已發表 ■未發表之文稿 □撰寫中 □無

專利：□已獲得 □申請中 □無

技轉：□已技轉 □洽談中 □無

其他：（以 100 字為限）

3. 請依學術成就、技術創新、社會影響等方面，評估研究成果之學術或應用價值（簡要敘述成果所代表之意義、價值、影響或進一步發展之可能性）（以 500 字為限）

行政院衛生署公佈民國 99 年國人十大死因，心臟疾病及腦血管疾病分別占十大死亡原因的第 2 位及第 3 位。而代謝症候群聚集數種心血管疾病的危險因子於一身，包括血脂異常、血壓過高、血糖上升與腹部肥胖，這些都增加了心血管疾病的發生率與死亡率。輔酵素 Q10 為一種內生性親脂溶性的抗氧化物質，主要存在於心肌及肝臟的粒線體細胞膜上，其被認為具有抗氧化之功效。本研究目的是要了解代謝症候群之受試者輔酵素 Q10 與脂質過氧化、抗氧化酵素活性與發炎之相關性。本研究計畫內容與原先計畫相評達 80 %。受試者的取得尚稱順利，原因為參與本研究計畫的家醫科醫生全力支持與協助。本研究結果推論代謝症候群受試者血漿輔酵素 Q10 濃度與健康受試者有顯著之差異。輔酵素 Q10 濃度與脂質過氧化壓力及抗氧化酵素活性有顯著的相關性。本研究計畫亦做為碩士班學生之研究論文，故此研究計畫成果未來將發表於 SCI 期刊。

國科會補助計畫衍生研發成果推廣資料表

日期:2011/07/27

國科會補助計畫	計畫名稱: 輔?Q10與維生素A，C，E對代謝症候群受試者之氧化壓力，抗氧化酵素活性與發炎相關性之探討
	計畫主持人: 林娉婷
	計畫編號: 99-2320-B-040-011- 學門領域: 保健營養

無研發成果推廣資料

99 年度專題研究計畫研究成果彙整表

計畫主持人：林娉婷	計畫編號：99-2320-B-040-011-
計畫名稱：輔?Q10 與維生素 A，C，E 對代謝症候群受試者之氧化壓力，抗氧化酵素活性與發炎相關性之探討	

成果項目			量化			單位	備註（質化說明：如數個計畫共同成果、成果列為該期刊之封面故事...等）
			實際已達成數（被接受或已發表）	預期總達成數(含實際已達成數)	本計畫實際貢獻百分比		
國內	論文著作	期刊論文	0	1	100%	篇	
		研究報告/技術報告	1	1	100%		
		研討會論文	0	1	100%		
		專書	0	0	100%		
	專利	申請中件數	0	0	100%	件	
		已獲得件數	0	0	100%		
	技術移轉	件數	0	0	100%	件	
		權利金	0	0	100%	千元	
	參與計畫人力（本國籍）	碩士生	3	2	100%	人次	
		博士生	0	0	100%		
		博士後研究員	0	0	100%		
		專任助理	0	0	100%		
國外	論文著作	期刊論文	0	1	100%	篇	
		研究報告/技術報告	0	0	100%		
		研討會論文	0	0	100%		
		專書	0	0	100%	章/本	
	專利	申請中件數	0	0	100%	件	
		已獲得件數	0	0	100%		
	技術移轉	件數	0	0	100%	件	
		權利金	0	0	100%	千元	
	參與計畫人力（外國籍）	碩士生	0	0	100%	人次	
		博士生	0	0	100%		
		博士後研究員	0	0	100%		
		專任助理	0	0	100%		

其他成果 (無法以量化表達之成果如辦理學術活動、獲得獎項、重要國際合作、研究成果國際影響力及其他協助產業技術發展之具體效益事項等，請以文字敘述填列。)	無。本研究計畫做為碩士班學生之研究論文，故此研究計畫成果未來將發表於SCI 期刊。

	成果項目	量化	名稱或內容性質簡述
科教處計畫加填項目	測驗工具(含質性與量性)	0	
	課程/模組	0	
	電腦及網路系統或工具	0	
	教材	0	
	舉辦之活動/競賽	0	
	研討會/工作坊	0	
	電子報、網站	0	
	計畫成果推廣之參與（閱聽）人數	0	

國科會補助專題研究計畫成果報告自評表

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■達成目標

□未達成目標（請說明，以 100 字為限）

□實驗失敗

□因故實驗中斷

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