行政院國家科學委員會專題研究計畫 成果報告

輔酵素 Q10 與冠狀動脈心臟病人脂質過氧化、抗氧化酵素活性及血漿同半胱胺酸濃度關係之探討(第 2 年)
研究成果報告(完整版)

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中文摘要

許多文獻指出心血管疾病患者有輔酵素 Q10 缺乏之情形，而輔酵素 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及預防心血管疾病之功效。然而，冠狀動脈心臟病人輔酵素 Q10 濃度與心血管疾病的相關性，目前結果仍未一致。本研究目的為探討使用輔酵素 Q10 補充劑對冠狀動脈心臟病（CAD）人脂質過氧化指標（丙二醛）及抗氧化酵素活性（過氧化氫酵素、超氧化物歧化酵素及麩胱甘肽過氧化酵素）之影響。CAD 受試者經心導管檢查冠狀動脈狹窄程度大於或等於 50 %者（共 51 位），後隨機分派至安慰劑組（n = 14）及輔酵素 Q10 補充劑組別（Q10-60 mg/d，n = 19； Q10-150 mg/d，n = 18），介入十二週。結果：總共有 43 位受試者完成試驗。試驗介入第 4 週，輔酵素 Q10-150 mg/d 組之丙二醛濃度顯著降低（p = 0.03）。在抗氧化酵素方面，介入第 12 週後，輔酵素 Q10-150 mg/d 組之受試者的過氧化氫酵素、超氧化物歧化酵素活性顯著上升（p = 0.03, p = 0.04）；然而，麩胱甘肽過氧化酵素活性卻無顯著之影響。此外，發現血漿輔酵素 Q10 濃度與丙二醛濃度有顯著之負相關（β = -0.21, p < 0.01）。因此本研究推論冠狀動脈心臟病人使用輔酵素 Q10 補充劑應可改善其脂質過氧化壓力及抗氧化酵素活性。

關鍵字：輔酵素 Q10 補充劑、脂質過氧化、抗氧化酵素、冠狀動脈心臟病、安慰劑控制

Abstract

Background & aims: Coenzyme Q10 (ubiquinone) is a lipid-soluble component of the mitochondrial respiratory chain for adenosine triphosphate (ATP) synthesis. Many studies have documented a deficiency of coenzyme Q10 in patients with cardiovascular disease. The purpose of this study was to investigate the effect of coenzyme Q10 supplementation on oxidative stress and antioxidant enzyme activities in patients with coronary artery disease (CAD).

Methods: This study was an intervention study. Patients who were identified by cardiac catheterization as having at least 50% stenosis of one major coronary artery or receiving percutaneous transluminal coronary angioplasty (n = 51) were randomly assigned to either the placebo group (n = 14) or one of the two coenzyme Q10 supplement groups [60 mg/day (Q10-60 group), n = 19; 150 mg/day (Q10-150 group), n = 18]. Intervention was administered for 12 weeks. Patients' blood samples were analyzed every 4 weeks for plasma coenzyme Q10 concentrations, malondialdehyde (MDA) and antioxidant enzyme activities [catalase (CAT), superoxide dismutase (SOD), glutathione peroxidase (GPx)].

Results: Forty-three subjects with CAD completed the intervention study. The MDA levels were reduced significantly at week 4 in the Q10-150 group (p = 0.03) and these patients had lower MDA levels than the patients in the placebo group at week 8 (p = 0.03). In respect to antioxidant enzyme activities, CAT and SOD activities were significantly increased in the Q10-150 group at week 12 (p = 0.03, 0.04, respectively). GPx activity was unchanged after coenzyme Q10 intervention. There was a significant correlation between plasma coenzyme Q10 and MDA levels (β = -0.21, p < 0.01), even after lipid adjustment, but there was no significant correlation with antioxidant enzyme activities.

Conclusion: Coenzyme Q10 supplements at a dose of 150 mg reduced oxidative stress and increased the activities of antioxidant enzymes in CAD patients. We suggest a higher dose of coenzyme Q10 supplements might promote rapid and sustainable antioxidation in CAD patients.

Keywords: Coenzyme Q10, lipid peroxidation, antioxidant enzyme activity, coronary artery disease, placebo-controlled study

This study was registered under www.clinicaltrials.gov/ Identifier no. NCT01163500

1. Introduction

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.[1, 2] Coenzyme Q10 can be synthesized in tissue from farnesyl diphosphate and tyrosine and also can be obtained from consumption of meat, poultry, fish, vegetables and fruits. Coenzyme Q10 is obtained from the diet in an oxidized form of which 75-95% is then converted into a reduced form in our body, but total absorption of coenzyme Q10 is thought to be less than 10%.[3,4] Tissues with high energy requirements, such as the heart, kidney, liver, and skeletal muscle cells, need a higher amount of coenzyme Q10 to synthesize ATP.[2] Coenzyme Q10 is recognized as an intracellular antioxidant that protects the membrane phospholipids, mitochondrial membrane protein and low-density lipoprotein (LDL) from free radical-induced oxidative damage.[5, 6]

Cardiovascular disease (CVD) is the leading cause of death worldwide.[7] In the past, many studies[8, 9,10] have documented a deficiency of coenzyme Q10 in patients with CVD and the benefits of treating these patients with coenzyme Q10 supplementation.[11-15] A recent study[16] indicated a relation existed between low plasma coenzyme Q10 concentration and coronary artery disease (CAD), which may contribute to the higher susceptibility of some individuals to CVD, especially Asian Indians and Chinese.[17] A double-blind, randomized, controlled study conducted by Tiano et al.[18] treated 35 ischemic heart disease patients with coenzyme Q10 at a dose of 100 mg three times daily (300 mg/day) for 1 month. The results showed a significant increase in the activities of endothelium-bound extracellular superoxide dismutase (ecSOD) and endothelium-dependent relaxation. However, in some clinical trials coenzyme Q10 supplements only produced a slight improvement or none at all in the patients with congestive heart failure.[19, 20, 21] The Department of Health (DOH) in Taiwan recommends a daily intake of no more than 30 mg of coenzyme Q10 for healthy adults. However, the DOH in Taiwan does not provide any information on the use of coenzyme Q10 to prevent CAD. Therefore, in this study we investigated the effect of coenzyme Q10 supplementation (60 mg/day and 150 mg/day) on oxidative stress and antioxidant enzyme activities in patients with CAD.

2. Materials and Methods

2.1 Subjects

This study was designed as a randomized, parallel, placebo-controlled study. CAD patients were recruited from the cardiology clinic of Taichung Veterans General Hospital, which is a teaching hospital in central Taiwan. Patients identified by cardiac catheterization as having at least 50% stenosis of one major coronary artery or receiving percutaneous transluminal coronary angioplasty (PTCA) were enrolled in this study. Subjects with diabetes, liver, or renal diseases were excluded to minimize the influence of other cardiovascular risk factors. Patients under statin therapy or currently taking vitamin supplements were also excluded. None of our subjects had experienced acute myocardial infarction within the previous 6 months. Informed consent was obtained from each subject. This study was approved by the Institutional Review Board of Taichung Veterans General Hospital, Taiwan.

2.2 Study protocol

We enrolled 59 CAD patients in this study, but 8 subjects declined to participate. The remaining 51 patients were randomly assigned to one of three groups: a placebo group (n = 14) or either one of two coenzyme Q10 groups [60 mg/day (Q10-60 group), n = 19; 150 mg/day (Q10-150 group), n = 18] (Fig. 1). The coenzyme Q10 capsules were commercially available preparations (New Health Taiwan Co., Ltd.). Intervention was administered for 3 months (12 weeks). Patients were instructed to take one capsule daily and complete a 24-h diet recall at week 0 and week 12 to ensure they were maintaining their usual dietary intake during the study period. To monitor compliance, the researchers reminded patients to check the capsules bag every 4 weeks to confirm the bag was empty.

The age, gender, smoking habits, alcohol use, family history, and exercise frequency of all subjects were recorded. Body weight and height were measured; the body mass index (BMI; kg/m^2) was then calculated. Blood pressure was measured after a resting period of at least 5 min. Hypertension was defined as blood pressure ≥ 140/90 mmHg or currently taking antihypertensive drugs. All subjects were instructed how to complete a 24-h diet recall, and the recall data was obtained on the day blood samples were drawn. Nutrient composition was calculated with the use of Nutritionist Professional software (E-Kitchen Business Corp., Taiwan), and the nutrient database was based on the Taiwan food composition table (Department of Health, 1998).

2.3 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) containing EDTA as an anticoagulant or no anticoagulant as required. Serum and plasma were prepared and then stored frozen (-80℃) until analysis. Hematological entities (i.e., BUN, serum creatinine, total cholesterol, triacylglycerol, LDL, HDL) were measured by using an automated biochemical analyzer. Automated high sensitivity C-reactive protein (hs-CRP) measurements concentration was performed with particle-enhanced Immunonephelometry with an Immage analyzer.

2.4 Plasma coenzyme Q10 and homocysteine measurement

Plasma coenzyme Q10 was measured by using high-performance liquid chromatography (HPLC) according to the method of Chu et al.[22] The intra- and inter-assays of fasting plasma coenzyme Q10 variability were 1.8% (n = 6) and 4.4% (n = 21), respectively. Plasma homocysteine was also determined by HPLC, as previously described.[23, 24] The intra- and inter-assays of fasting plasma homocysteine variability were 1.0% (n = 3) and 4.3% (n = 6), respectively. All analyses were performed in duplicate.

2.5 Lipid peroxidation and antioxidant enzyme activity measurement

Plasma malondialdehyde (MDA) was determined using the TBARS (thiobarbituric acid reactive substances) method, as described by Botsoglou[25] and Chung et al.[26] Red blood cells (RBC) were diluted with 25x sodium phosphate buffer for superoxide dismutase (SOD) and glutathione peroxidase (GPx) measurement and 250x sodium phosphate buffer for catalase (CAT) measurement. The methods for

measuring RBC, CAT, SOD and GPx were previously described[26], and measurements were performed spectrophotometrically at 240, 325 and 340 nm, respectively. Protein contents of plasma and RBC were determined based on the Biuret reaction of the BCA kit (Thermo, Rockford, IL, USA). The MDA levels were expressed as nmol/mg protein and the antioxidant enzyme activity levels were expressed as unit/mg of protein. All analyses were performed in duplicate and completed within 7 days.

2.6 Statistical analyses

Data were analyzed with SigmaStat statistical software (version 2.03; Jandel Scientific, San Rafael, CA). Differences in subjects' demographic data and the hematological measurement data among the three intervention groups were analyzed by one-way analysis of variance (ANOVA) or Kruskal-Wallis ANOVA on ranks, Friedman repeated measures ANOVA on ranks within each group. Tukey's post hoc test was used to assess the statistically significant differences among the groups. To examine the relation of coenzyme Q10 concentration with oxidative stress (MDA) and antioxidant enzyme activities (CAT, SOD, GPx), Spearman rank-order correlation was used. Results were considered statistically significant at $P < 0.05$. Values presented in the text are means ± standard deviation (SD).

3. Results

A total of 43 CAD subjects completed the study. There were no significant differences among groups with respect to age, BMI, blood pressure, anthropometric measurements, hematological entities (i.e., BUN, serum creatinine, lipid profiles, hs-CRP), plasma homocysteine concentration and the frequency of smoking and alcohol consumption at baseline (Table 1).

Figure 2 shows the effect of coenzyme Q10 supplementation on lipid peroxidation and antioxidant enzyme activities. Plasma coenzyme Q10 concentration was significantly increased after coenzyme Q10 supplementation (Q10-150 group) at week 4 ($p < 0.01$) and the levels rose significantly with intervention. The MDA levels were reduced significantly at week 4 in the Q10-150 group ($p = 0.03$). The Q10-150 group had significantly lower MDA levels than the placebo group at week 8 ($p = 0.03$). In respect to antioxidant enzyme activities, patients in the Q10 -150 group had a slight increase of CAT activity at week 8 ($p = 0.07$) and significantly higher levels of CAT ($p = 0.03$) and SOD ($p = 0.04$) than the placebo group at week 12, but GPx activity was unchanged after coenzyme Q10 intervention. In the placebo group, antioxidant enzyme activities (CAT and SOD) were decreased after 12 weeks ($p < 0.01$, $p = 0.02$). In addition, plasma homocysteine and hs-CRP concentrations were unchanged after coenzyme Q10 intervention (data not shown).

The correlations between coenzyme Q10 concentration, MDA and antioxidant enzyme activities are assessed in Table 2. There was a significantly negative relation between plasma coenzyme Q10 and MDA levels ($\beta = -0.21$, $p < 0.01$), even after lipid adjustment, but there was no significant correlation with antioxidant enzyme activities. The MDA level was significantly lower in patients with a higher plasma Q10 concentration ($p = 0.01$) after we used the median level of plasma coenzyme Q10 concentration (0.11 μmol/L) as a cut-off point (Table 3).

4. Discussion

The results showed a significantly negative correlation between plasma coenzyme Q10 concentration and the lipid peroxidation marker (MDA) in this study. Coenzyme Q10 at a dose of 150 mg reduced MDA levels by approximately 17% at week 4 and MDA levels were significantly lower in the Q10-150 group than the placebo group at week 8 ($p = 0.03$). Based on our results, it seems clear that coenzyme Q10 has a protective effect against CAD, which can be ascribed to its antioxidant function. Coenzyme Q10 can provide rapid protective effects against lipid peroxide (MDA), which is an indicator of free radical-induced damage during myocardial ischemia.[27, 28] A coenzyme Q10 supplement at a dose of 150 mg compared with a dose of 60 mg showed a significant reduction of lipid peroxidation in this study. Antioxidant enzymes like CAT, SOD and GPx are the first line of defense against reactive oxygen species (ROS) and a decrease in their activities contributes to the oxidant attack on cells. The activities of CAT and SOD, but not GPx, were significantly increased after 12 weeks of coenzyme Q10 supplementation at a dose of 150 mg. However, there was no significant correlation between plasma coenzyme Q10 concentration and antioxidant enzyme activities (Table 2). Notably, the activities of CAT and SOD were significantly decreased in the placebo group compared with baseline. Some factors such as disease and age may affect antioxidant enzyme activities. The mean age of our CAD patients in this study was 75 years and the protective effects of endogenous enzymatic antioxidant and coenzyme Q10 levels may decline with age and CAD.[29, 30] Endogenous coenzyme Q10 synthesis might be impaired or insufficient in subjects with CAD and individuals taking statin medications, but we have excluded the patients who were being treated with statin. In this study, we treated CAD patients with coenzyme Q10 in doses up to 150 mg (equivalent to five times the daily intake recommended by the DOH in Taiwan) for 3 months, but the levels of MDA did not decrease significantly until week 4, and there was no further reduction during the study period. In a study on healthy subjects, a plateau in absorption of coenzyme Q10 occurred at a dose of 200 mg and better plasma absorption was achieved at a dose of 300 mg.[31] As a result, we consider supplementation of coenzyme Q10 in CAD patients at a higher dosage might provide better absorption and sustainable antioxidation.

The concentration of plasma coenzyme Q10 in healthy individuals generally ranges from 0.87 – 1.16 μmol/L[4]. Littarru et al.[8] were the first authors to point out that 75% of heart disease patients had coenzyme Q10 deficiency in blood and myocardium with disease severity. In this study, the levels of plasma coenzyme Q10 were low at baseline (0.08 ± 0.04 μmol/L) in our stable CAD subjects. The levels of plasma coenzyme Q10 increased significantly, especially in the Q10-150 group (0.10 ± 0.05 to 0.30 ± 0.16 μmol/L), but the levels were still not as high as those in healthy subjects. The results of this study showed that a lower MDA level in a higher plasma coenzyme Q10 concentration stratified with the median level (0.11 μmol/L) (Table 4). After 4 weeks of coenzyme Q10 supplementation (both 60 mg/day and 150 mg/day), the mean values of plasma coenzyme Q10 exceeded the median level (0.11 μmol/L). An increase in the concentration of coenzyme Q10 may somehow affect the mitochondrial respiratory function[32], and early supplementation should be administrated in cases of deficiency.[33] Additionally, most of the subjects in the coenzyme Q10

supplement groups told a clinical physician that they felt better and experienced progressive recovery after coenzyme Q10 supplementation. Many studies[34-37] reported remarkable clinical benefits such as improved tolerance of work in stable angina pectoris patients after administration of coenzyme Q10 at doses of 30 mg to 150 mg per day for a short period (1 or 4 weeks). There was no adverse effect from or complaint about coenzyme Q10 supplements in this study. The DOH in Taiwan recommends a coenzyme Q10 intake of not more than 30 mg a day for healthy adults, but the International Coenzyme Q10 Association suggested 300 mg a day for healthy adults. Ikematsu et al.[38] suggested that coenzyme Q10 was well-tolerated and safe for healthy adults even at an intake of up to 900 mg/day. Compared with other antioxidant vitamins, coenzyme Q10 is an expensive supplement, but it has a more powerful antioxidant activity than other antioxidant vitamins.[39, 27, 28] Coenzyme Q10 also has a greater synergistic effect than other antioxidant vitamins such as vitamin A, C, and E. Although we did not examine the levels of plasma vitamin A, C and E in this study, Singh et al.[27, 28] have documented that coenzyme Q10 supplements increase the levels of vitamin A, C, and E. As a result, coenzyme Q10 supplementation might be beneficial in CAD patients.

The relation between coenzyme Q10 and inflammation has been reported in cell[40, 41] and animal models.[42, 43] In clinical studies, levels of high sensitivity lipoprotein (hs-CRP) and homocysteine are commonly used as inflammatory markers. Coenzyme Q10 may provide an anti-inflammatory effect and mediated by its antioxidant activity.[41] In this study, we have determined the levels of hs-CRP and homocysteine. It is not surprising that patients with CAD had higher concentrations of hs-CRP (> 0.3 mg/dL) and homocysteine (> 12 μmol/L) (Table 1). However, the levels of hs-CRP and homocysteine were unchanged after coenzyme Q10 supplementation (data not shown). In a recent study[44], patients with multiple cardiovascular risk factors were treated with antioxidants (vitamin C, E, selenium and coenzyme Q10) for 6 months, but no significant difference in homocysteine and hs-CRP levels was observed. Further clinical study is needed to confirm the anti-inflammatory effect of coenzyme Q10 supplements.

In conclusion, coenzyme Q10 supplements at a dose of 150 mg can reduce oxidative stress (MDA) and increase antioxidant enzyme activities in CAD patients. We consider a higher dose of coenzyme Q10 supplements might provide rapid and sustainable antioxidation in CAD patients. Further study is needed to demonstrate whether a high dose of coenzyme Q10 correlates with clinical benefits. The findings of this study provide information that may be helpful to clinical dietitians and physicians when they are advising CAD patients about coenzyme Q10 supplementation.

Conflict of Interest

The authors have no conflict of interest. All authors have read and approved the final manuscript.

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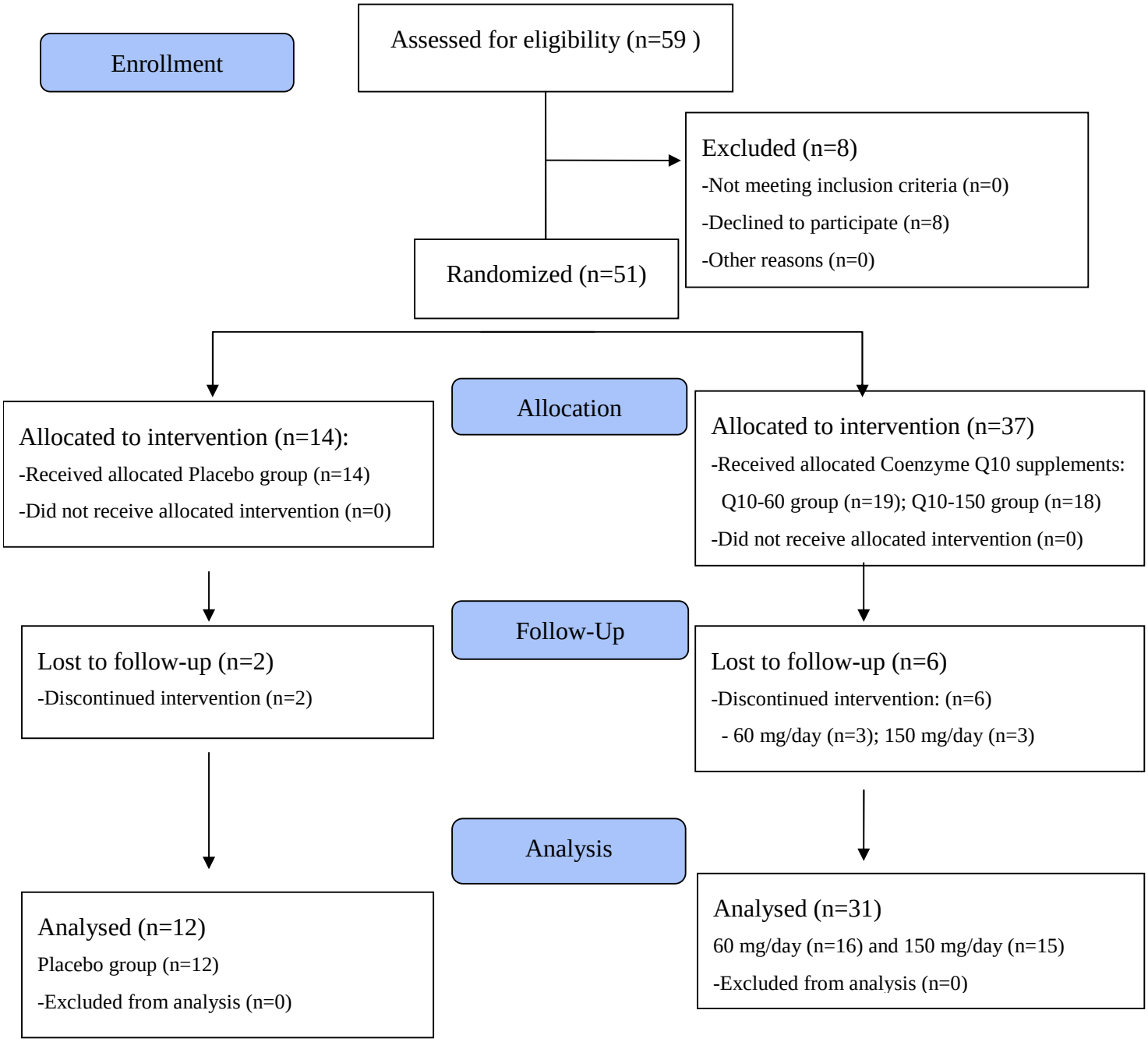
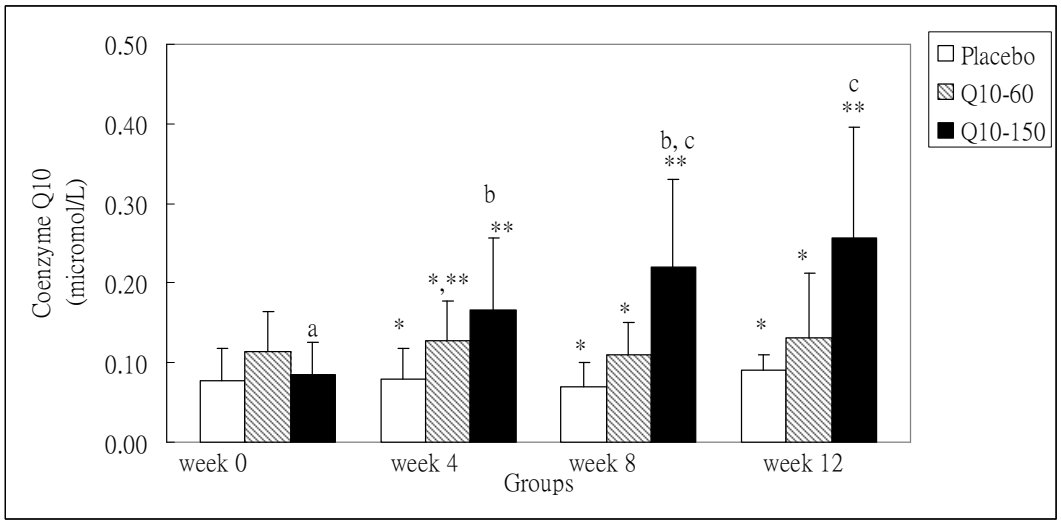
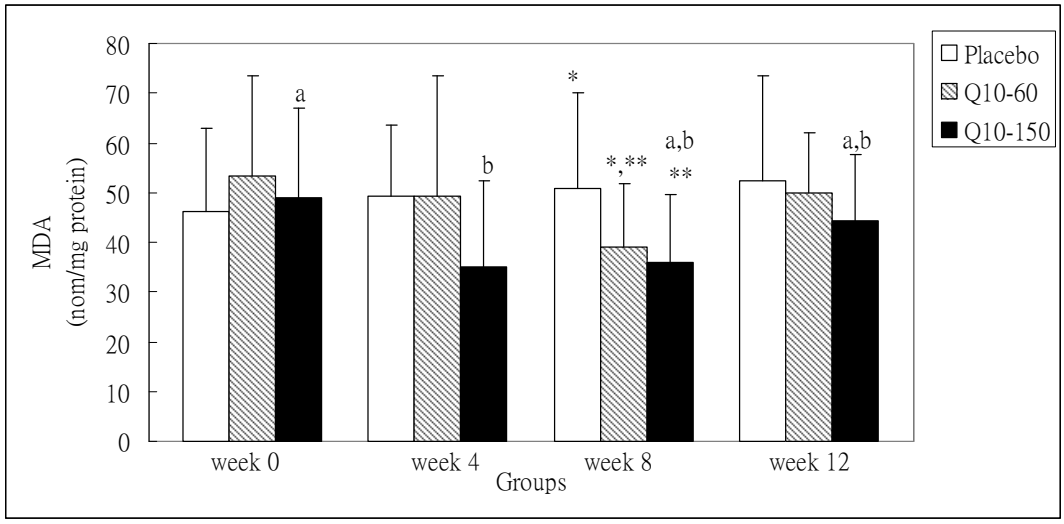
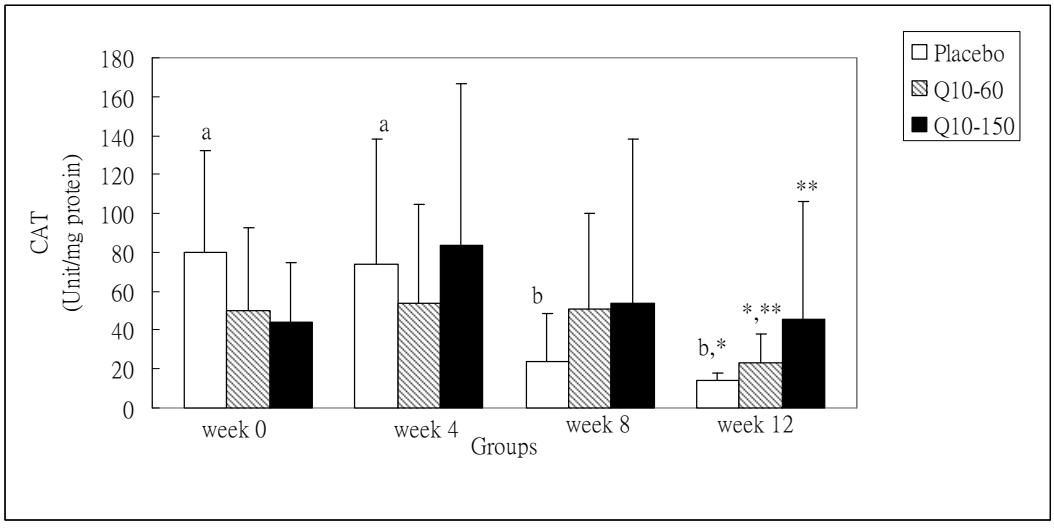
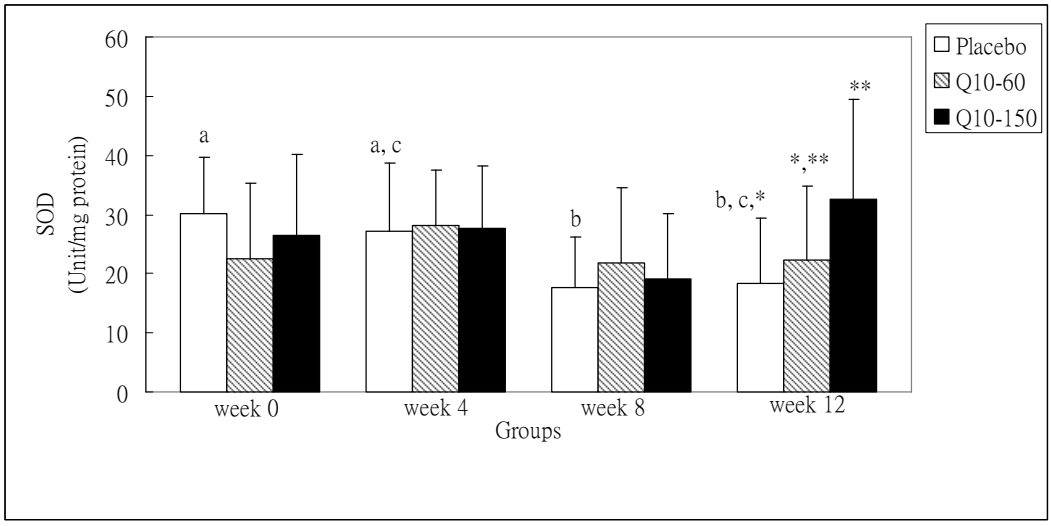


Figure 1 Flow Diagram


Coenzyme Q10 (micromol/L)
0.50
0.40
0.30
0.20
0.10
0.00
Placebo
Q10-60
Q10-150
a
b
b, c
c
week 0
week 4
week 8
week 12
Groups


MDA (nom/mg protein)
80
70
60
50
40
30
20
10
0
Placebo
Q10-60
Q10-150
a
b
a,b
a,b
week 0
week 4
week 8
week 12
Groups


CAT (Unit/mg protein)
180
160
140
120
100
80
60
40
20
0
Placebo
Q10-60
Q10-150
a
a
b
b,*
week 0
week 4
week 8
week 12
Groups



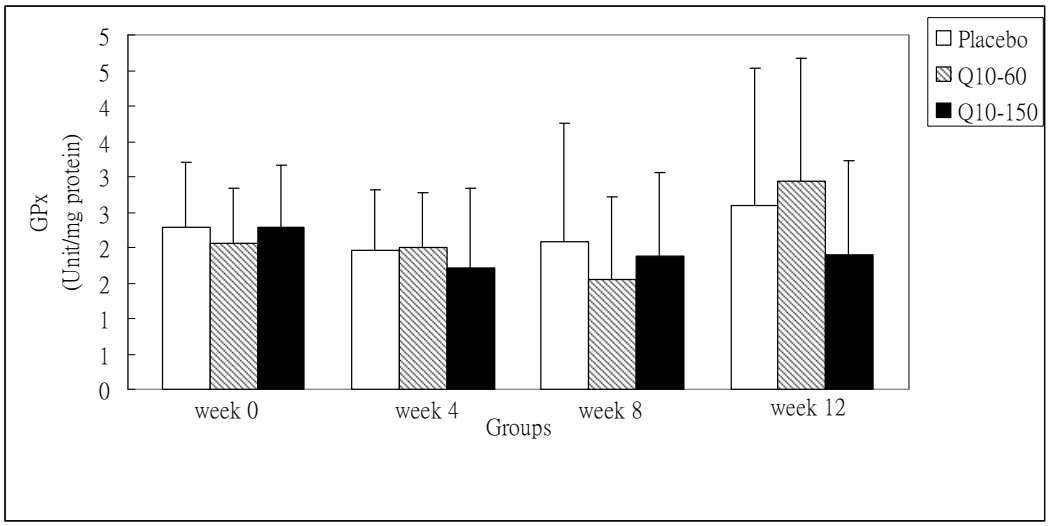


Figure 2 Concentration of plasma coenzyme Q10, lipid peroxidation and antioxidant enzymes activities after intervention.

(*,** Values are significantly different among three intervention groups at the same period. ;

$^{a, b, c}$ Values are significantly different after intervention in the same group; $P < 0.05$).

CAD, coronary artery disease; CAT, catalase activity; MDA, malondialdehyde; GPx, glutathione peroxidase.

Table 1 General baseline characteristics of subjects[1]

groups	Placebo （n = 12）	Q10-60 （n = 16）	Q10-150 （n = 15）
Male / Female (n)	12 / 0	14 / 2	14 / 1
Age (y)	75.6 ± 7.9	73.0 ± 7.7	77.1 ± 9.9
Systolic blood pressure (mmHg)	133.6 ± 14.7	132.8 ± 12.5	133.7 ± 14.0
Diastolic blood pressure (mmHg)	72.1 ± 7.0	75.0 ± 12.9	74.7 ± 8.6
Body mass Index (BMI, kg/m^2)	26.2 ± 3.4	26.3 ± 3.0	24.7 ± 3.1
Waist hip ratio (W/H)	0.9 ± 0.1	0.9 ± 0.1	0.9 ± 0.1
Mid-arm circumference (Cm)	26.1 ± 2.7	29.4 ± 3.6	26.4 ± 2.1
Triceps skin fold (TSF, mm)	6.4 ± 3.1	8.4 ± 3.7	6.9 ± 4.3
BUN (mg/dL)	21.1 ± 6.1	22.8 ± 7.4	21.7 ± 8.4
Serum creatinine (mg/dL)	1.3 ± 0.3	1.3 ± 0.4	1.4 ± 0.3
TC (mg/dL)	177.4 ± 34.7	186.2 ± 30.3	204.1 ± 37.1
TG (mg/dL)	126.2 ± 54.9	144.7 ± 100.1	133.9 ± 81.3
LDL-C (mg/dL)	113.9 ± 34.2	120.3 ± 25.2	132.3 ± 33.9
HDL-C (mg/dL)	37.2 ± 12.6	37.6 ± 9.1	38.0 ± 11.3
hs-CRP (mg/dL)	0.6 ± 1.8	0.4 ± 0.6	0.3 ± 0.3
Plasma homocysteine (μmol/L)	20.1 ± 10.3	18.2 ± 8.0	18.2 ± 7.1
Smoking frequency (%)	28.6	57.9	38.9
Alcohol consumption (%)	42.9	31.6	27.8

[1]Mean ± SD.

BUN, blood urea nitrogen; HDL-C, high-density lipoprotein; hs-CRP, high sensitivity C-reactive protein; LDL-C, low density lipoprotein; TC, total cholesterol; TG, triglyceride.

Table 2 Correlation between coenzyme Q10 concentration with lipid peroxidation and antioxidant enzymes activities

	MDA (nmol/ mg protein)	CAT (unit/mg protein)	SOD (unit/mg protein)	GPx (unit/mg protein)
	Correlation coefficient (*P value*)			
Q10 (μmol/L)	-0.21 (< 0.01)	0.03 (0.66)	0.04 (0.59)	-0.08 (0.31)
Q10/TC (μmol/mmol)	-0.22 (< 0.01)	0.02 (0.76)	0.04 (0.59)	-0.02 (0.77)
Q10/TG (μmol/mmol)	-0.24 (< 0.01)	0.00 (0.91)	-0.09 (0.26)	-0.03 (0.65)
Q10/LDL (μmol/mmol)	-0.23 (< 0.01)	0.00 (0.93)	0.04 (0.64)	-0.04 (0.60)
Q10/HDL (μmol/mmol)	-0.17 (0.03)	0.04 (0.60)	0.09 (0.26)	-0.03 (0.70)

CAT, catalase activity; MDA, malondialdehyde; GPx, glutathione peroxidase; HDL, high-density lipoprotein; LD low-density lipoprotein; SOD, superoxide dismutase; TC, total cholesterol; TG, triglyceride.

Table 3 Lipid peroxidation and antioxidant enzymes activities after stratification by plasma coenzyme Q10 concentration

Plasma Coenzyme Q10	MDA (nmol/ mg protein)	CAT (unit/mg protein)	SOD (unit/mg protein)	GPx (unit/mg protein)
< 0.11 μmol/L	49.7 ± 18.0[1,a]	49.3 ± 53.0	25.3 ± 11.8	2.3 ± 1.4
≥ 0.11 μmol/L	42.1 ± 16.8[b]	54.2 ± 62.3	25.4 ± 12.9	2.0 ± 1.1

[1]Mean ± SD. [a, b] $P < 0.05$

CAT, catalase activity; MDA, malondialdehyde; GPx, glutathione peroxidase.

中華民國九十九年八月三日

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）

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及預防心血管疾病之功效。然而，冠狀動脈心臟病人輔酵素 Q10 濃度與心血管疾病的相關性，目前結果仍未一致。因此，本研究目的是要探討輔酵素 Q10 濃度與冠狀動脈心臟病人血脂質濃度、脂質過氧化、抗氧化酵素活性與血漿同半胱胺酸濃度之關係。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心臟科醫生全力支持與協助。但每組介入成功完成人數仍有流失之情形。本研究結果推論冠狀動脈心臟病人使用輔酵素 Q10 補充劑應可改善其脂質過氧化壓力及抗氧化酵素活性，但對於發炎指標:同半胱胺酸濃度似乎無顯著之影響。我們期望此研究結果可提供營養師及臨床醫師在心臟人營養上關於輔酵素 Q10 補充之建議，期達預防醫學之目的。另外本研究計畫亦做為碩博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。

無研發成果推廣資料

97 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：97-2320-B-040-034-MY2
計畫名稱：輔酵素 Q10 與冠狀動脈心臟病人脂質過氧化、抗氧化酵素活性及血漿同半胱胺酸濃度關係之探討	

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	0	100%	篇	
		研究報告/技術報告	1	1	100%		國科會計畫成果報告
		研討會論文	1	1	100%		第 36 屆台灣營養學年會
		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	1	1	100%	人次	
		博士生	0	0	100%		
		博士後研究員	0	0	100%		
		專任助理	1	1	100%		
國外	論文著作	期刊論文	0	1	100%	篇	預期發表 1 篇國際 SCI 期刊
		研究報告/技術報告	0	0	100%		
		研討會論文	0	1	100%		預期發表 1 篇國際研討會論文
		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。)	本計畫之初步研究成果發表於第三十六屆營養學年會海報題目’’輔酵素 Q10 補充劑對冠狀動脈心臟病人脂質過氧化及抗氧化酵素活性之影響’’，以及目前將結果撰寫 manuscript 投稿於國際 SCI 期刊中。

	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

□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)

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