行政院國家科學委員會專題研究計畫 期末報告

探討香菜酒精萃出物對脂肪細胞分化及發炎反應之影響(第3年)

計 畫 類 別 ： 個別型
計 畫 編 號 ： NSC 99-2320-B-040-005-MY3
執 行 期 間 ： 101年08月01日至102年07月31日
執 行 單 位 ： 中山醫學大學營養學系（所）

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公 開 資 訊 ： 本計畫涉及專利或其他智慧財產權，2年後可公開查詢

中 華 民 國 102年11月03日

中文摘要： 肥胖可增加糖尿病、心血管疾病、高血壓、膽囊疾病及癌症等疾病發病率，因預防肥胖，已成為重要身體保健議題(reviewed in Kopelman, 2000)。本研究計畫研究成果證實香菜酒精萃出物可減少脂肪細胞脂肪合成，減少 adipogenesis 過程中重要轉錄因子表現與增加磷酸化 AMPK 蛋白質表現，具有抑制前脂肪細胞分化為成熟脂肪細胞之功效。現今已知肥胖與慢性發炎反應有關，肥胖會增加吞噬細胞侵入脂肪組織數目，而吞噬細胞分泌物質(macrophage-secreted factors)導致脂肪細胞不正常分泌 adipokines，誘發發炎反應及胰島素阻抗(Gutierrez et al., 2009； Weisberg et al., 2003； Xu et al., 2003)。研究發現，肥胖引起的脂肪細胞發炎與第 2
型糖尿病、心血管疾病及代謝症候群等與肥胖有關的病症發生有密切關係 (reviewed in Kopelman,2000)。本研究計畫研究成果證實香菜酒精萃出物可調控 LPS 誘發 RAW 264.7 macrophage 發炎反應。

中文關鍵詞： 香菜, 發炎, 脂肪細胞

英文摘要： BACKGROUND: Coriandrum sativum is used not only as spice to aid flavor and taste values in food, but also as folklore medicine in many countries. Since little is known about the anti-inflammatory ability of aerial parts (stem and leaf) of Coriandrum sativum, the present study investigated the effect of aerial parts of Coriandrum sativum on lipopolysaccharide (LPS)-stimulated RAW 264.7 macrophages. We further explored the molecular mechanism underlying these pharmacological properties of Coriandrum sativum.
RESULTS: Both stem and leaf of Coriandrum sativum ethanolic extracts (CSEE) significantly decreased LPS-induced nitric oxide and prostaglandin E2 production as well as inducible nitric oxide synthase, cyclooxygenase-2, and pro-interleukin-1 刍 expression. Moreover, LPS-induced I 岂 B-脉 phosphorylation and nuclear p65 protein expression as well as nuclear factor-岂 B (NF-岂 B) nuclear protein-DNA binding affinity and reporter gene activity were dramatically inhibited by aerial parts of CSEE. Exogenous addition of CSEE stem and leaf

significantly reduced LPS-induced expression of phosphorylated mitogen-activated protein kinases (MAPKs).
CONCLUSION: Our data demonstrated that aerial parts of CSEE have a strong anti-inflammatory property which inhibits pro-inflammatory mediator expression by suppressing NF-岂B activation and MAPK signal transduction pathway in LPS-induced macrophages.

英文關鍵詞： Coriandrum sativum, inflammation, adipocytes

第一、二年以 3T3-L1 前脂肪細胞株 (3T3-L1 preadipocyte)分化為脂肪細胞之研究模式探討香菜酒精萃出物對調控 adipogenesis 之影響。結果發現香菜萃出物可抑制 3T3-L1 前脂肪細胞分化為脂 肪細胞過程經由可增加 ERK1/2 磷酸化和抑制 CCAAT/enhancer Binding Protein β (C/EBP β) 蛋白質表現使減少脂肪細胞內油滴的累積及抑制 Peroxisome-Proliferator Activated Receptor γ (PPAR γ)、C/EBP α mRNA 和蛋 白質的表現，降低 Fatty Acid Synthase mRNA 和蛋白質表現及 ap2 mRNA。 此外於脂肪細胞分化過程中添加香菜萃出物，會顯著增加脂肪細胞中 AMP-activated protein kinase (AMPK) 磷酸化及抑制肪細胞激素 (adipokine) resistin mRNA 的表現 (參期中報告)。

第三年探討香萃出物對抗發炎的影響 (**Suppressive effects of aerial part extracts of *Coriandrum sativum* L. on LPS-induced inflammatory responses in murine RAW 264.7 macrophages)**

Abstract

BACKGROUND: *Coriandrum sativum* is used not only as spice to aid flavor and taste values in food, but also as folklore medicine in many countries. Since little is known about the anti-inflammatory ability of aerial parts (stem and leaf) of *Coriandrum sativum*, the present study investigated the effect of aerial parts of *Coriandrum sativum* on lipopolysaccharide (LPS)-stimulated RAW 264.7 macrophages. We further explored the molecular mechanism underlying these pharmacological properties of *Coriandrum sativum*.

RESULTS: Both stem and leaf of *Coriandrum sativum* ethanolic extracts (CSEE) significantly decreased LPS-induced nitric oxide and prostaglandin E_2 production as well as inducible nitric oxide synthase, cyclooxygenase-2, and pro-interleukin-1β expression. Moreover, LPS-induced IκB-α phosphorylation and nuclear p65 protein expression as well as nuclear factor-κB (NF-κB) nuclear protein-DNA binding affinity and reporter gene activity were dramatically inhibited by aerial parts of CSEE. Exogenous addition of CSEE stem and leaf significantly reduced LPS-induced expression of phosphorylated mitogen-activated protein kinases (MAPKs).

CONCLUSION: Our data demonstrated that aerial parts of CSEE have a strong anti-inflammatory property which inhibits pro-inflammatory mediator expression by suppressing NF-κB activation and MAPK signal transduction pathway in LPS-induced macrophages.

Introduction

Spices are used not only to aid flavor, color and nutrition values in food, but also to treat various physical problems in traditional medicines.[10] *Coriandrum sativum,* commonly known as coriander or Chinese parsley, belongs to the family *Apiaceae*, which is widely cultivated all over the world. The seeds and aerial parts (stem and leaf) of *Coriandrum sativum* are commonly used as spices in Middle Eastern, Mediterranean, Indian, Latin American, African, Southeast Asian and Taiwanese cuisines. Data from numerous researchers have shown the therapeutic values of the seeds and seed oil of *Coriandrum sativum* due to its hypoglycemic, hypolipidemic, hepatoprotective, antimutagenic, antihypertensive, antioxidant, anxiolytic, antimicrobial, and post-coital antifertility activity.[11-19] The aerial parts of *Coriandrum sativum* have antioxidant and free radical scavenging activities, suppressive activity on lead and mercury deposition and bactericidal and anti-adhesive effects on *Helicobacter pylori*.[20-23] Recent studies reported that *Coriandrum sativum* seed oil reduced UV-induced erythema test of human skin and leaves of *Coriandrum sativum* water extract decreased LPS-induced NO production and had scavenging effects on NO.[24,25] Based on previous studies of *Coriandrum sativum* presented herein, it is worth conducting a detailed investigation on the anti-inflammatory property of aerial parts of *Coriandrum sativum*. The objective of the present study is to assess the regulatory efficacy of aerial parts of *Coriandrum sativum* on the LPS-induced inflammatory responses in RAW 264.7 macrophages as well as to explore the possible molecular mechanism behind these actions.

Result

RESULTS

The extraction yields of leaf and stem of CSEE were 11.76% and 11.97%, respectively. In addition, the phenolic contents, expressed as GAE, of leaf and stem extracts were 15.5±1.9 and 17±3.8 mg g^{-1} dry

extract, respectively. The amount of flavonoids, expressed as QE, of the leaf extract was 16.14± 1.17 mg g^{-1} dry extract which was 5.7 times higher than that of stem extract.

At the test concentrations, cell viability of the LPS-activated cells treated with leaf or stem of CSEE was more than 90% of that of cells treated with LPS alone, as assessed by mitochondrial reduction of MTT after 18 h challenge (Table 1, and 2).

As shown in Tables 1 and 2, stimulation of macrophages with LPS resulted in a strong increase in NO and PGE_2 production. A dose-dependent decrease in NO and PGE_2 production was noted in cells treated with leaf or stem of CSEE in the presence of LPS. At a concentration of 150 μg mL^{-1}, 80% and 75% reduction in nitrite production were noted in cells treated with leaf and stem of CSEE, respectively. The PGE_2 levels in cells treated with 150 μg mL^{-1} of the leaf and stem of CSEE were 25.2±2.4% and 52.5±7.7%, respectively, of the level of cells treated with LPS alone.

The immunoblot assay showed that the protein expression of iNOS, COX-2, and proIL-1β was undetectable in resting RAW 264.7 macrophages and was highly induced in the presence of LPS. The addition of exogenous leaf or stem of CSEE significantly reduced LPS-induced protein expression of iNOS, COX-2, and pro-IL-1β ($P < 0.05$, Fig. 1). As noted for the changes in protein expression, real-time RT-PCR further showed that LPS-induced mRNA expression of iNOS, COX-2, and pro-IL-1β was significantly decreased by leaf and stem of CSEE (Table 3 and 4).

Upon LPS treatment, the amounts of cytoplasmic phosphorylated IκB-α protein and nuclear p65 protein were tremendously increased compared with those of the control (Fig. 2). Addition of 150 μg mL^{-1} leaf or stem of CSEE significantly abolished the level of LPS-induced protein expression of phosphorylated IκB-α and nuclear p65 ($P < 0.05$).

To test whether the mitogen-activated protein kinase (MAPK) signaling pathway was involved in the anti-inflammatory property of *Coriandrum sativum*, we examined the effect of leaf and stem of CSEE on LPS-induced MAPK activation. LPS treatment resulted in strong increases in the amounts of phosphorylated ERK1/2, p38 and JNK-1 expression ($P < 0.05$, Fig. 3). Addition of stem extracts significantly reduced LPS-induced phosphorylated JNK and p38. However, LPS-induced activation of ERK1/2 was diminished only by high doses of CSEE stem. Addition of high doses of leaf extracts significantly inhibited LPS-induced activation of MAPKs. The amount of the unphosphorylated form of MAPKs was not influenced by the LPS treatment or LPS plus stem or leaf of CSEE.

EMSA experiments were used to evaluate the effect of *Coriandrum sativum* on activation of NF-κB. As shown in Fig. 4A, the nuclear extract from LPS-stimulated macrophages showed a marked increase in NF-κB nuclear protein DNA-binding activity compared with that in unstimulated macrophages. Pretreatment of cells with leaf or stem of CSEE suppressed the activation of NF-κB binding to its consensus DNA sequences. The specificity of the NF-κB nuclear protein–DNA binding was verified by competition assay with a 50-fold excess of unlabeled NF-κB probe and unlabeled mutant NF-κB probe.

To investigate the transcriptional activity of NF-κB, the expression of reporter genes in cells transfected with pNF-κB-Luc and the internal control pSV-β-galactosidas were analyzed. Consistent with the EMSA assay result, the expression of LPS-induced NF-κB-Luc activity was significantly inhibited in cultures treated with 150 $\mu g\ mL^{-1}$ leaf or stem of CSEE (Fig. 4B, $P < 0.05$).

LC/MS analysis of the testing samples and several authentic standards revealed that only rutin is identified in both the stem and leaf of CSEE. Rutin showed the $[M-H]^-$ ion at m/z 609 and its retention time was 31.9 min. Based on the peak area of mass spectral peak, the rutin concentrations in stem and leaf extracts

were 130.5 and 42.0 μg g^{-1}, respectively. Two unknown large UV peaks presented at 48.3 min ($[M-H]^-$ ion at m/z 253) and 52.6 min ($[M-H]^-$ ion at m/z 221) were not identified (Fig. 5).

DISCUSSION

Numerous studies have focused on herbal remedies and botanicals because they offer much promise in health benefits and disease treatments without excessive side effects and cytotoxicity.[33] A wide variety of plant-derived products especially spices have shown the anti-inflammatory effect but only few of them have been examined for the molecular mechanism of this inhibitory action.[34] In this study, we investigated the anti-inflammatory properties of aerial part of CSEE and dissected the possible molecular mechanism of action of *Coriandrum sativum*. The data presented herein showed that both the leaf and stem of CSEE significantly decreased NO and PGE_2 production. The inhibition of NO and PGE_2 was due to the inhibition of iNOS and COX-2 expression respectively, at mRNA and protein levels as shown by real-time RT-PCR and Western blot. Moreover, we demonstrated that the aerial part of CSEE suppressed iNOS, COX-2 and IL-1β expression acting at the transcriptional level possibly via inhibition of LPS-induced MAPK pathway and transcription factor NF-κB activation.

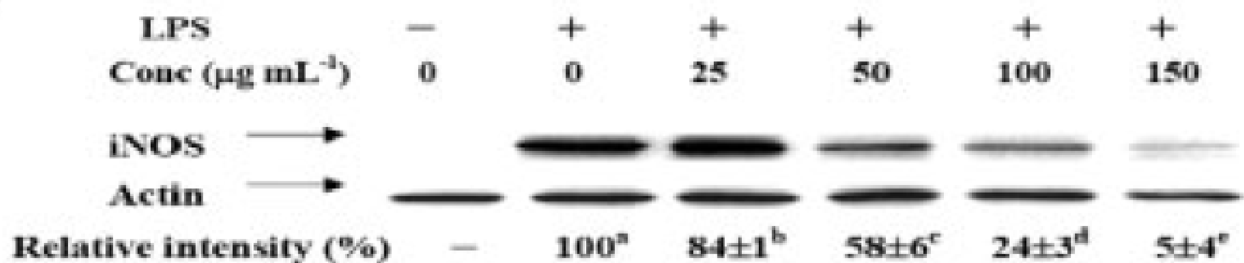
Considerable interest in immunomodulation therapy is now focused on blocking the activation of NF-κB in macrophages, which results in suppressing a range of inflammatory mediator expression such as iNOS, COX-2, and IL-1β.[35,36,2] Our data clearly showed that CSEE effectively inhibited the NF-κB pathway by blocking LPS-induced IκB-α phosphorylation, nuclear p65 expression and subsequent DNA binding affinity and transcriptional activation**.** These results suggested that stem and leaf of CSEE decreased the expression of pro-inflammatory mediators via down-regulating the NF-κB pathway in stimulated macrophages.

MAPKs, one of the most important intracellular signaling pathways, are a family of serine/threonine protein kinases, which include JNK, ERK, and p38 kinase subgroups at least in mammalian cells. MAPK pathways were involved in a battery of cellular events, including cell proliferation and growth, cell differentiation, cell movement, cellular senescence and apoptosis.[37] Although the exact signal pathways of MAPKs are still unclear, LPS-induced phosphorylation and activation of MAPKs in macrophages lead to the production of pro-inflammatory mediators as a result of the activation of transcription factors including NF-κB.[38] In the present study, the aerial part of CSEE significantly decreased LPS-induced phosphorylation of the three MAPKs, which implies that the inflammatory signal transduction by MAPKs pathway could be impeded by *Coriandrum sativum* in LPS-induced macrophages.Numerous studies have demonstrated that phenolic compounds in spices contribute to the health benefits of spices.[39] A previous study indicated that luteolin, vicenin, ferulic acid and arbutin were the main components in the aerial part of CSEE.[32] However, in this study, only rutin was identified in the stem and leaf of CSEE and the rutin concentration in stem extracts (130.5 μg g^{-1}) was higher than that in the leaf extracts (42.0 μg g^{-1}). It was interesting to note that the total amount of flavonoids in the stem extracts was also lower than that in the leaf extracts, although there was no significant difference in the amount of total phenolics between stem and leaf of CSEE. Furthermore, the amount of rutin in the IC50 values of the stem and leaf of CSEE against LPS-induced NO production in RAW264.7 macrophage was 8.2 μg mL^{-1} and 2.7 μg mL^{-1}, much lower than the reported IC50 value of rutin on LPS-induced NO production (25.3 μg mL^{-1}).[40] These results indicated that it is not only rutin which provides contributions to the anti-inflammatory properties of *Coriandrum sativum*, but other yet unknown compounds in the stem and leaf of CSEE may present further contributions as well. It is noteworthy that the antimutagenicity of *Coriandrum sativum* was dependant on the chlorophyll content in *Coriandrum sativum*

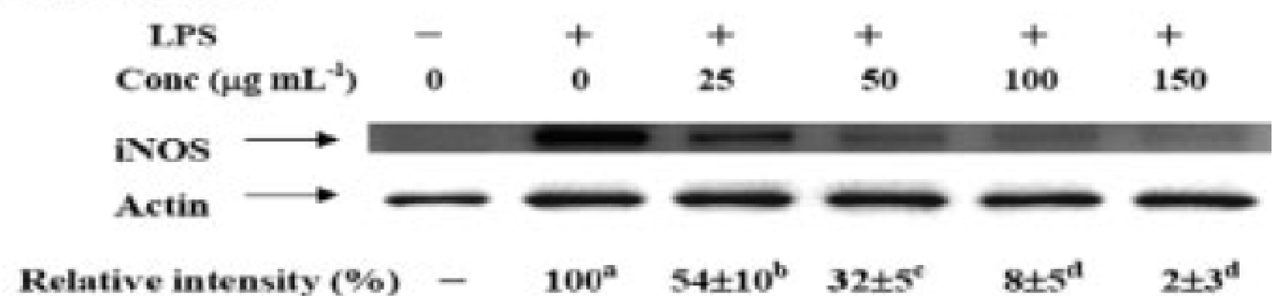
juice. [14] Moreover, the chlorophyllin, a water-soluble derivative of chlorophyll, inhibited NO production and iNOS expression by modulating LPS-induced NF-κB activation in RAW264.7 macrophages.[41] Therefore, the amount of chlorophyll in CSEE is likely to play an important in its anti-inflammatory property.

In conclusion, we demonstrated that both the leaf and the stem of CSEE modulate LPS-induced inflammatory events in RAW 264.7 macrophages. This inhibitory activity of *Coriandrum sativum*, at least in part, occurs through *Coriandrum sativum* modulating the NF-κB activation and MAPK pathway. According to these experiment results supporting the anti-inflammatory property of the leaf and stem of CSEE, it would be worthwhile to explore the biomedical importance of the aerial parts of CSEE in the treatment and prevention of chronic inflammatory related diseases.

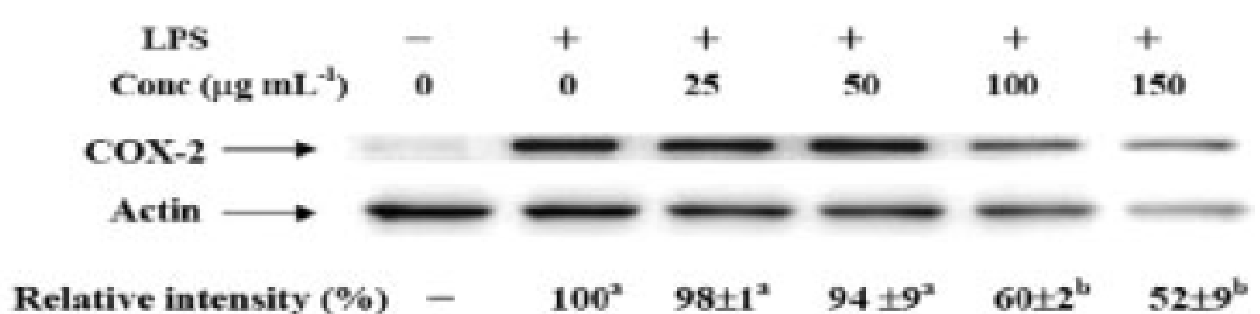
(A) Stem of CSEE



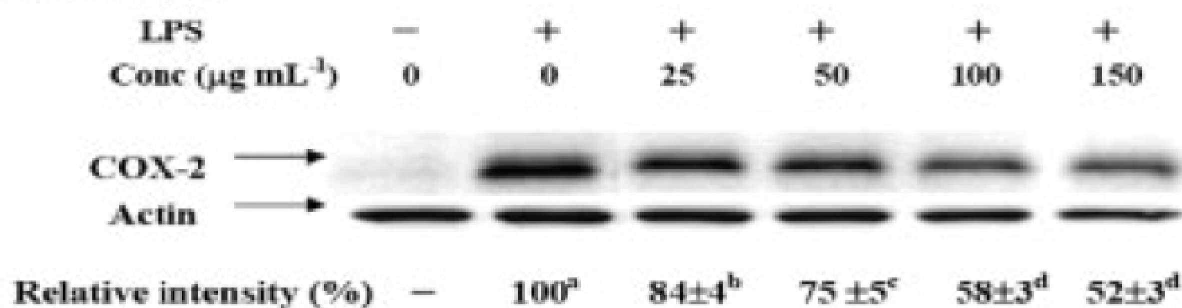
(B) Leaf of CSEE



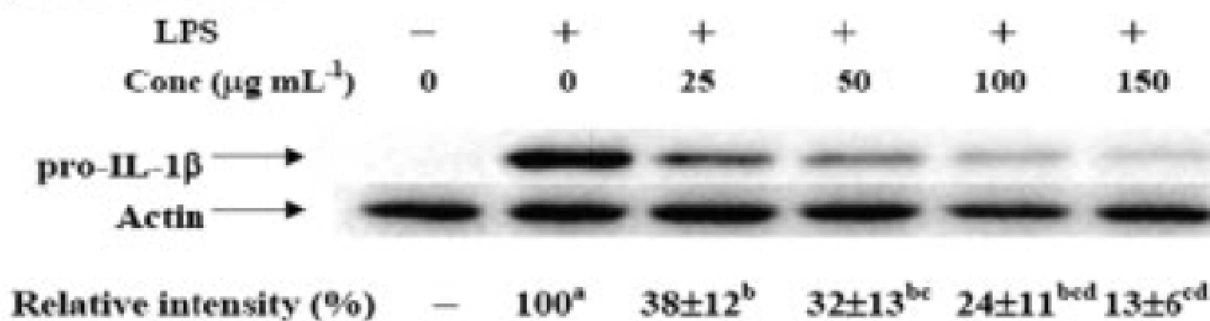
(C) Stem of CSEE



(D) Leaf of CSEE



(E) Stem of CSEE



(F) Leaf of CSEE

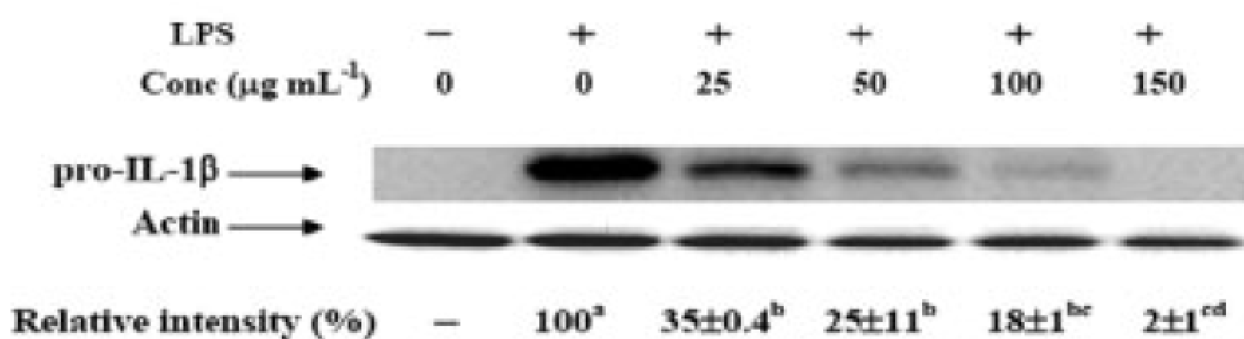


Figure 1. Effects of stem and leaf of CSEE on LPS-induced iNO, COX-2. and Pro-IL-1β protein expressions in RAW 264.7 macrophages. (A) iNOS protein expression was measured by RAW 264.7 macrophages treated with or without LPS (10 ng mL^{-1}) plus DMSO vehicle control and 25 to 150 μg mL^{-1} stem or leaf of CSEE for

24 h. (B) COX-2 and (C) Pro-IL-1β protein expression were measured in cells preincubated with 25 to 150 μg mL^{-1} stem or leaf of CSEE for 1h and then treated with either DMSO vehicle control or 10 ng mL^{-1} LPS for 6 h. Data are the mean ± SD of at least four separate experiments and are expressed as the percentage of the culture treated with LPS alone. Values not sharing the same letter are significantly different ($P < 0.05$).

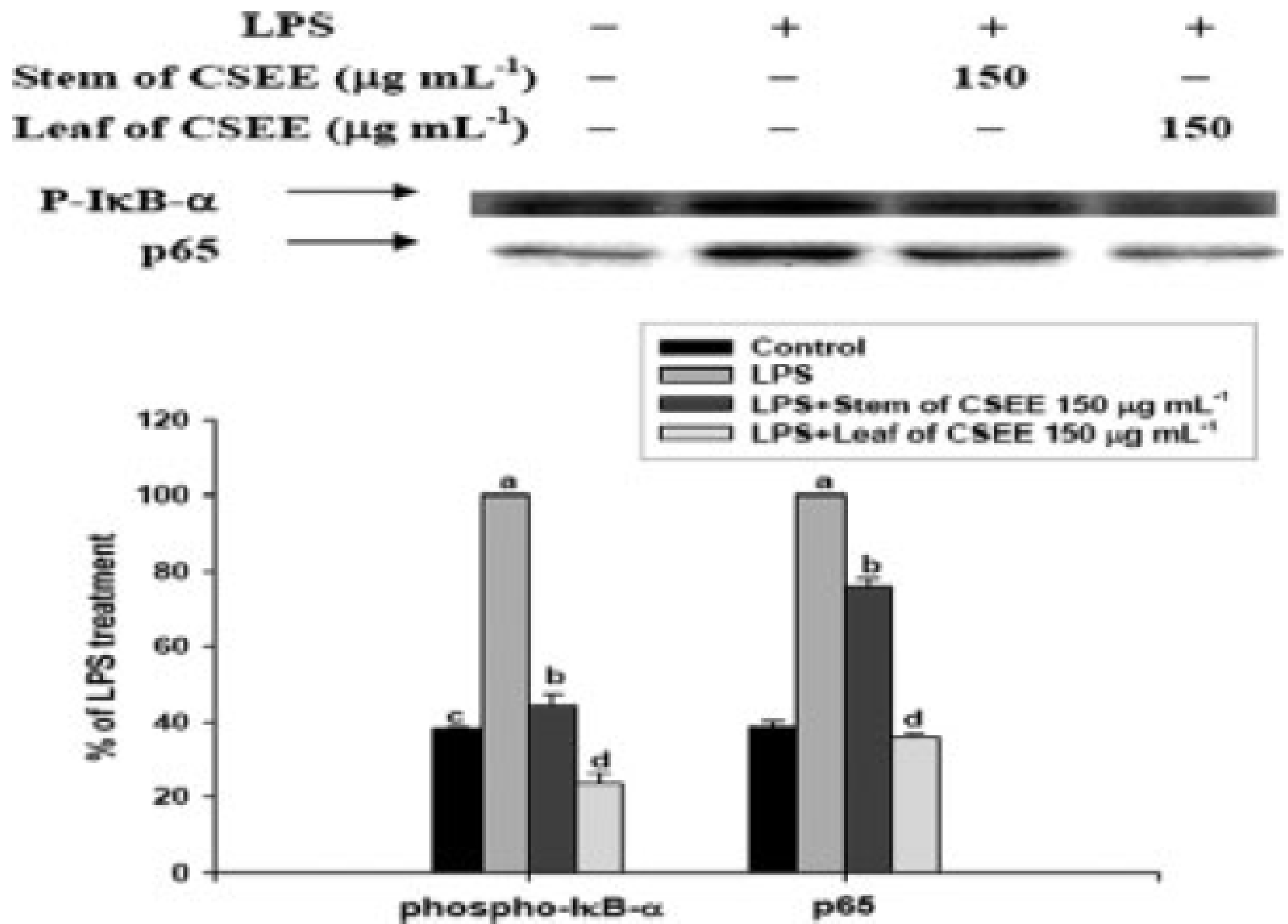
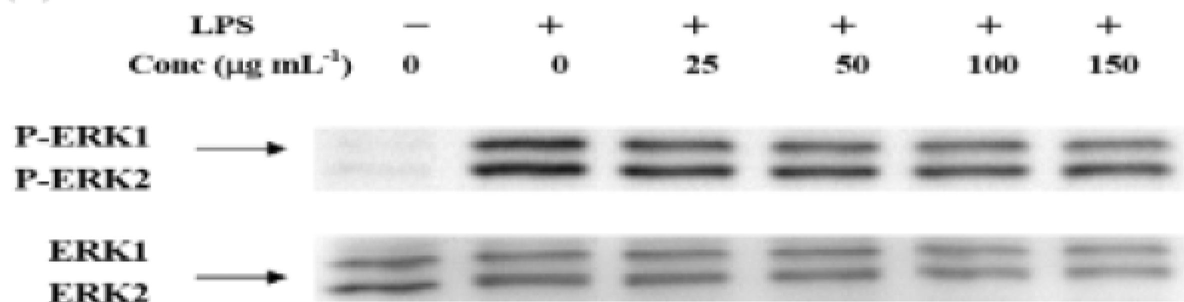


Figure 2. Effects of stem and leaf of CSEE on LPS-induced of expressions of cytoplasmic phosphorylated IκBα and nuclear p65. RAW 264.7 macrophages were preincubated with 150 μg mL^{-1} stem or leaf of CSEE for 14 h and then treated with either DMSO vehicle control or 10 ng mL^{-1} LPS for 30 min. Western blot analysis was used to measure the protein content of phosphorylated IκB-α in the cytosolic factions and to measure p65 protein content in the nuclear protein fractions of RAW 264.7 macrophages.

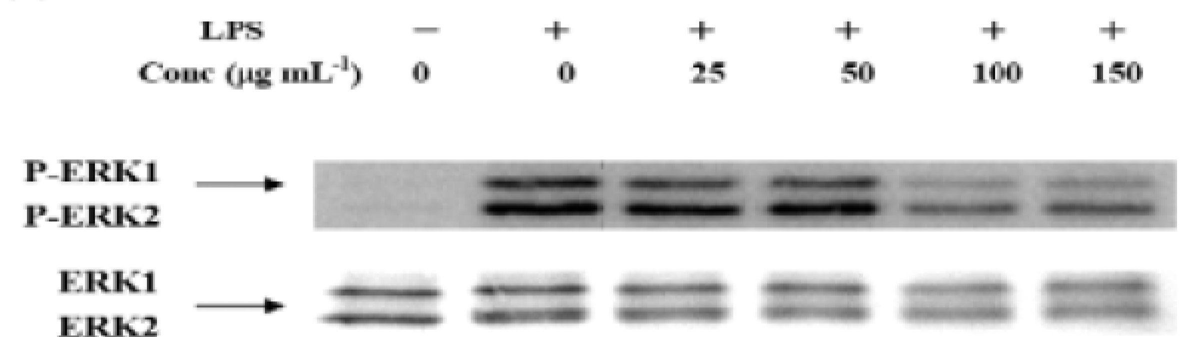
(A) Stem of CSEE



Relative intensity (%)

p-ERK1	−	100^{a}	$83±4^{b}$	$83±4^{b}$	$76±9^{b}$	$62±5^{c}$
p-ERK2	−	100^{a}	$89±1^{bc}$	$90±3^{b}$	$80±6^{cd}$	$76±8^{d}$

(B) Leaf of CSEE



Relative intensity (%)

p-ERK1	−	100^{a}	$94±3^{a}$	$95±7^{a}$	$53±19^{b}$	$38±4^{b}$
p-ERK2	−	100^{a}	$99±7^{a}$	$97±7^{a}$	$37±7^{b}$	$46±11^{b}$

(C) Stem of CSEE

LPS	−	+	+	+	+	+
Conc (μg mL^{-1})	0	0	25	50	100	150

P-p38 →
p38 →

Relative intensity (%)	−	100^{a}	$86±7^{b}$	$79±11^{c}$	$12±4^{d}$	$8±2^{d}$

(D) Leaf of CSEE

LPS	−	+	+	+	+	+
Conc (μg mL^{-1})	0	0	25	50	100	150

P-p38 →
p38 →

Relative intensity (%)	−	100^{a}	$104±9^{a}$	$90±25^{a}$	$61±1^{ab}$	$23±0.3^{b}$

(E) Stem of CSEE

LPS	−	+	+	+	+	+
Conc (μg mL^{-1})	0	0	25	50	100	150

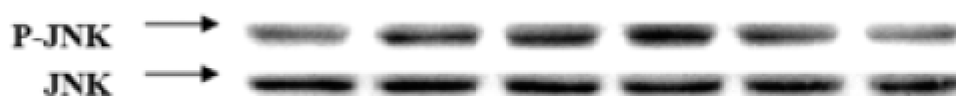
P-JNK →
JNK →

Relative intensity (%)	$41±7^{c}$	100^{a}	$82±4^{b}$	$76±3^{b}$	$30±9^{c}$	$34±1^{c}$

(F) Leaf of CSEE

LPS	−	+	+	+	+	+
Conc (μg mL^{-1})	0	0	25	50	100	150

P-JNK →
JNK →



Relative intensity (%)	$28±30^{c}$	100^{b}	$100±6^{b}$	$118±3^{a}$	$87±13^{b}$	$29±8^{c}$

Figure 3. Effects of stem and leaf of CSEE on LPS-induced of activation of MAPKs. RAW 264.7 macrophages were preincubated with 25 to 150 μg mL^{-1} stem or leaf of CSEE for 1 h and then treated with either DMSO vehicle control or 10 ng mL^{-1} LPS for 30 min. Cells were lysed and Western blotting was performed with the antibodies for phosphorylated (A) ERK 1/2, (B) p38 and (C) JNK and the cells were then reprobed with antibodies against the corresponding MAPKs. The ratios of immunointensity between the MAPKs and the phosphorylated MAPKs are shown and are expressed as the percentage of the culture treated

with LPS alone.

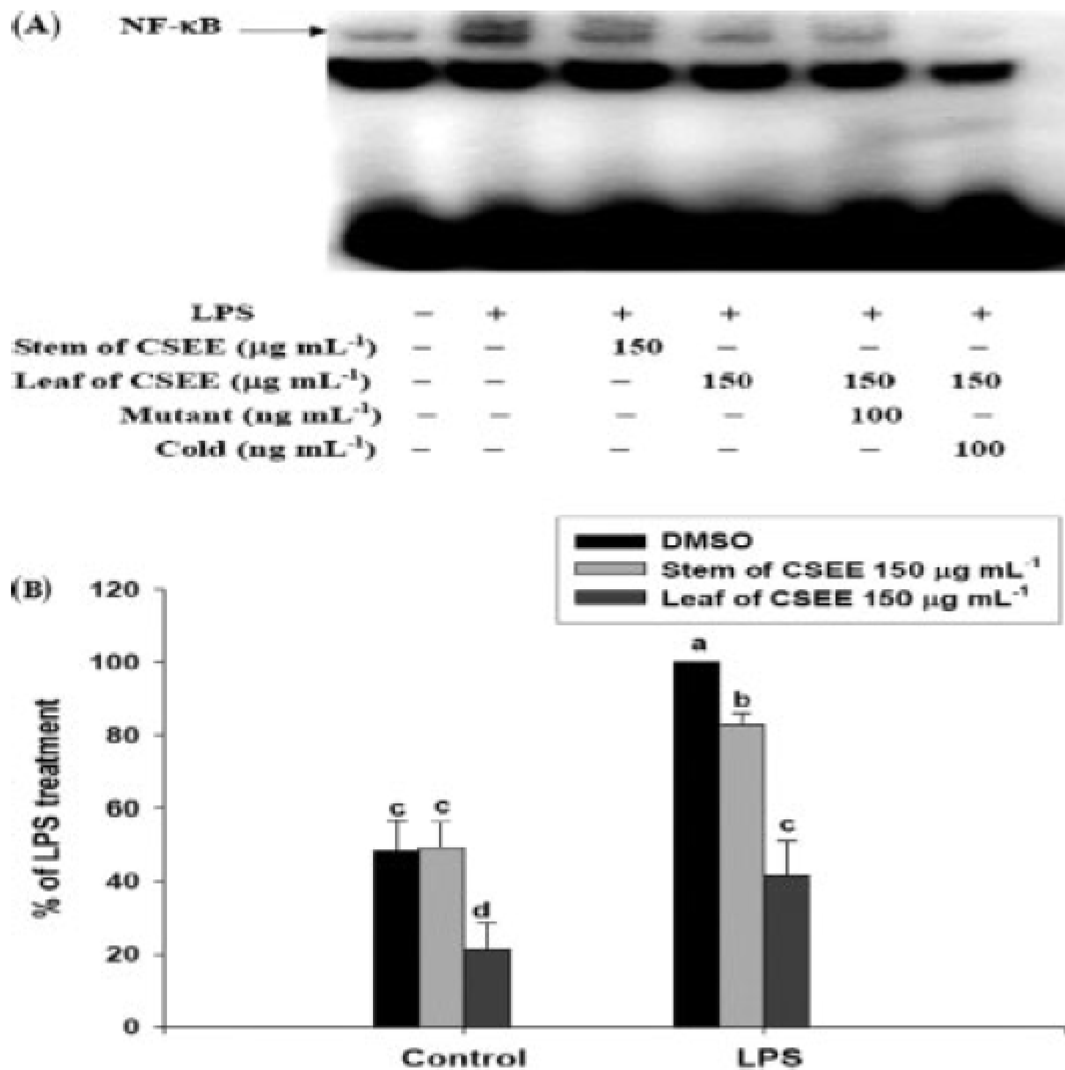


Figure 4. Effects of stem and leaf of CSEE on activation of NF-κB. (A) RAW 264.7 macrophages were preincubated with 150 μg mL^{-1} stem or leaf of CSEE for 14 h and then treated with either DMSO vehicle control or 10 ng mL^{-1} LPS for 30 min. EMSA experiments were carried out by using the LightShift Chemiluminescent EMSA Kit from Pierce Chemical Co. The unlabeled double-stranded oligonucleotides of NF-κB and the unlabeled double-stranded mutant NF-κB oligonucleotide were added for the competition assay and specificity assay, respectively. Bands were detected by using streptavidin-horseradish peroxidase and were developed by using a SuperSignal West Pico kit from Pierce Chemical Co. (B) Cells were transiently transfected with pSV-β-galactosidase and pNF-κB-Luc reporter gene for 6 h and cells were treated with either vehicle control or 10 ng mL^{-1} LPS plus 150 μg mL^{-1} stem or leaf of CSEE for 8 h. Cells were harvested and the level of luciferase and β-galactosidase activity were measured by Luciferase Assay System and β-Galactosidase Enzyme Assay System with Reporter Lysis Buffer from Promega Co., respectively.

附件一

行政院國家科學委員會補助專題研究計畫 □期中進度報告 ■期末報告

（計畫名稱）

探討香菜酒精萃出物對脂肪細胞分化及發炎反應之影響

計畫類別：■個別型計畫 □整合型計畫

計畫編號：NSC 99 - 2320 - B - 040 - 005 - MY3

執行期間：99 年 08 月 01 日起至 102 年 07 月 31 日

執行機構及系所：中山醫學大學營養系

計畫主持人：劉凱莉

共同主持人：

計畫參與人員：陳姵吟，巫玉琳，黃馨瑤，彭佳琳，許雅嵐，詹承儒，柯雅羚，陳慶文，陳雅芳。

本計畫除繳交成果報告外，另含下列出國報告，共 _0__ 份：

□移地研究心得報告

□出席國際學術會議心得報告

□國際合作研究計畫國外研究報告

處理方式：除列管計畫及下列情形者外，得立即公開查詢

■涉及專利或其他智慧財產權，□一年□二年後可公開查詢

中 華 民 國 年 月 日

國科會補助計畫衍生研發成果推廣資料表

日期:2013/11/03

<table>
<tr><td rowspan="3">國科會補助計畫</td><td>計畫名稱: 探討香菜酒精萃出物對脂肪細胞分化及發炎反應之影響</td></tr>
<tr><td>計畫主持人: 劉凱莉</td></tr>
<tr><td>計畫編號: 99-2320-B-040-005-MY3　　學門領域: 保健營養</td></tr>
<tr><td colspan="2">無研發成果推廣資料</td></tr>
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99 年度專題研究計畫研究成果彙整表

計畫主持人：劉凱莉			計畫編號：99-2320-B-040-005-MY3				
計畫名稱：探討香菜酒精萃出物對脂肪細胞分化及發炎反應之影響							
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	1	2	100%	篇	以發表一篇，還有1~2 篇在撰寫
		研究報告/技術報告	0	0	100%		
		研討會論文	11	11	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	14	14	100%	人次	
		博士生	6	6	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	0	100%	篇	
		研究報告/技術報告	0	0	100%		
		研討會論文	2	2	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

	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

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）

肥胖可增加糖尿病、心血管疾病、高血壓、膽囊疾病及癌症等疾病發病率，因預防肥胖，已成為重要身體保健議題(reviewed in Kopelman, 2000)。本研究計畫研究成果證實香菜酒精萃出物可減少脂肪細胞脂肪合成，減少 adipogenesis 過程中重要轉錄因子表現與增加磷酸化 AMPK 蛋白質表現，具有抑制前脂肪細胞分化為成熟脂肪細胞之功效。現今已知肥胖與慢性發炎反應有關，肥胖會增加吞噬細胞侵入脂肪組織數目，而吞噬細胞分泌物質(macrophage-secreted factors)導致脂肪細胞不正常分泌 adipokines，誘發發炎反應及胰島素阻抗(Gutierrez et al., 2009; Weisberg et al., 2003; Xu et al., 2003)。研究發現，肥胖引起的脂肪細胞發炎與第 2

型糖尿病、心血管疾病及代謝症候群等與肥胖有關的病症發生有密切關係 (reviewed in Kopelman,2000)。本研究計畫研究成果證實香菜酒精萃出物可調控 LPS 誘發 RAW 264.7 macrophage 發炎反應。