# 科技部補助專題研究計畫成果報告
# 期末報告

## 甘草酸衍生物抑制不同血癌細胞增生分化能力及誘導細胞凋亡的機制探討

計 畫 類 別 ： 個別型計畫
計 畫 編 號 ： MOST 104-2314-B-040-016-
執 行 期 間 ： 104年08月01日至105年07月31日
執 行 單 位 ： 中山醫學大學醫學系

計 畫 主 持 人： 蕭培靜
共 同 主 持 人： 楊順發
計畫參與人員： 碩士級-專任助理人員：林怡伶
碩士班研究生-兼任助理人員：林雅萩

中 華 民 國 105 年 10 月 20 日

中 文 摘 要 ： 甘草(Licorice)是傳統中藥材之一。甘草中的黃酮類化合物主要為chalcones，而chalcones中的licochalcone A在先前研究已經證實具有抗癌的特性。然而licochalcone A對於人類急性骨髓性白血病(acute myeloid leukemia, AML)的影響仍尚未釐清。我們利用licochalcone A，處理人類三株人類急性骨髓性白血病細胞株(MV4-11, U937 and HL-60)，並分析其細胞存活率。結果發現licochalcone A會抑制三株骨髓性白血病細胞株的生長，我們也利用flow cytometer 分析方式發現licochalcone A會誘導HL-60的細胞凋亡。此外，licochalcone A會經由活化caspase-3、8、9及裂解PARP來誘發HL-60細胞凋亡。而我們也發現licochalcone A會增加了Erk1/2, JNK1/2及P38 MAPK磷酸化的程度。而利用了JNK1/2的專一性抑制劑也顯著地降低了licochalcone A所誘發的caspase-3的活化。我們的結果顯示licochalcone A是經由JNK1/2路徑來誘發HL-60細胞產生凋亡，這也暗示著licochalcone A適合作為另一種有效的人類急性骨髓性白血病的抗癌輔助藥物。

中 文 關 鍵 詞 ： 甘草酸、血癌細胞、細胞凋亡

英 文 摘 要 ： Chalcones, a major phenolic constituent of licorice (Glycyrrhiza inflate), has been reported to exhibit anti-tumor properties in various cancer cells and animal model. Numerous investigations demonstrated that licochalcone A can modulate many cancer targets, suppressing cancer cell growth and/or inducing apoptosis. However, the effect of licochalcone A on acute myeloid leukemia (AML) remains unclear. Here, the molecular mechanism by which licochalcone A-induced apoptosis effects in human AML cells was investigated. The results showed that licochalcone A significantly inhibited cell proliferation of three AML cell lines (MV4-11, U937 and HL-60). Furthermore, licochalcone A induced apoptosis of HL-60 cells through caspases-3, -8, and -9 activations and PARP cleavage in dose-dependent manner. Inhibition of JNK1/2 by specific inhibitors significantly abolished the licochalcone A -induced activation of the caspase-3. Taken together, our results suggest that licochalcone A induced HL-60 cell apoptosis and could serve as a potential additional chemotherapeutic agent for treating AML.

英 文 關 鍵 詞 ： licochalcone A, acute myeloid leukemia, apoptosis

# Licochalcone A induces apoptosis in human promyelocytic leukemia cells

**Shih-Chung Wang [1], Shun-Fa Yang[2,3], Chiao-Wen Lin[4], Pei-Ching Hsiao[5,6,*]**

[1]Pediatric Hematology/Oncology, Changhua Christian Hospital, Changhua , Taiwan

[2]Institute of Medicine, Chung Shan Medical University, Taichung, Taiwan

[3]Department of Medical Research, Chung Shan Medical University Hospital, Taichung, Taiwan

[4]Institute of Oral Sciences, Chung Shan Medical University, Taichung, Taiwan

[5]School of Medicine, Chung Shan Medical University, Taichung, Taiwan

[6]Department of Internal Medicine, Chung Shan Medical University Hospital, Taichung, Taiwan

*Corresponding author: Pei-Ching Hsiao

School of Medicine, Chung Shan Medical University, 110, Section 1, Chien-Kuo N. Road, Taichung 40201, Taiwan, ROC

Tel: +886-4-24730022#34253; Fax: +886-4-23248171; Email: cshy046@csh.org.tw

## Abstract

Chalcones, a major phenolic constituent of licorice (Glycyrrhiza inflate), has been reported to exhibit anti-tumor properties in various cancer cells and animal model. Numerous investigations demonstrated that licochalcone A can modulate many cancer targets, suppressing cancer cell growth and/or inducing apoptosis. However, the effect of licochalcone A on acute myeloid leukemia (AML) remains unclear. Here, the molecular mechanism by which licochalcone A-induced apoptosis effects in human AML cells was investigated. The results showed that licochalcone A significantly inhibited cell proliferation of three AML cell lines (MV4-11, U937 and HL-60). Furthermore, licochalcone A induced apoptosis of HL-60 cells through caspases-3, -8, and -9 activations and PARP cleavage in dose-dependent manner. Inhibition of JNK1/2 by specific inhibitors significantly abolished the licochalcone A -induced activation of the caspase-3. Taken together, our results suggest that licochalcone A induced HL-60 cell apoptosis and could serve as a potential additional chemotherapeutic agent for treating AML.

## Introduction

Acute myeloid leukemia (AML) is a lethal malignant disease characterized by the rapid growth of abnormal white blood cells (WBCs). Chemotherapy is currently the primary treatment for AML [1], and enhancing apoptosis of AML cells is a promising target as a final cure for this disease. Although conventional chemotherapy of AML with either cytarabine or daunorubicin given as a single agent induces complete remission in ca. 30%~40% of patients and combination treatment with both agents induces complete remission in more than 50% of patients [2], only 20%~30% of patients enjoy long-term disease-free survival [2], and these chemotherapeutic drugs can also affect normal cells causing unpleasant side effects such as anemia, bleeding, and infection. Thus, there is a need for new agents to treat AML.

Licochalcone A (Lico A) (Figure 1A), a flavonoid found in licorice root (Glycyrrhiza glabra), has been reported to have anti-angiogenesis, anti-inflammatory and anti-cancer properties [3-12]. For example, hu et al. found that Licochalcone A attenuates lipopolysaccharide-induced acute kidney injury by Inhibiting NF-κB activation [6]. Moreover, Tasi et al. also shown that Licochalcone A induces autophagy through PI3K/Akt/mTOR inactivation and autophagy suppression enhances Licochalcone A-induced apoptosis of human cervical cancer cells[10]. However, there are no reports concerning the anticancer effects of licochalcone A on human AML cells. Although it is quite clear that licochalcone A can inhibit the growth or induce apoptosis of various solid tumor cells, the precise impact of licochalcone A on non-solid tumors is still not fully understood. In the present study, we investigated the cytotoxic effects of licochalcone A on three AML cell lines (MV4-11, U937 and HL-60), and its underlying mechanisms in vitro.

## Materials and Methods

### Materials

Licochalcone A of 98% purity was purchased from Sigma-Aldrich (St. Louis, MO). A 50-mM stock solution of licochalcone A was made in dimethyl sulfoxide (DMSO; Sigma) and stored at -20 ℃. The final concentration of DMSO for all treatments was < 0.5%. Antibodies specific for cleaved caspase-3, caspase-8, caspase-9, poly(ADP-ribose) polymerase (PARP), phosphorylated (p)-extracellular signal-regulated kinase (ERK)1/2, p-p38, p-c-Jun N-terminal kinase (JNK) and β-actin (for the Western blot analysis) were purchased from Santa Cruz Biotechnology (Santa Cruz, CA). Unless otherwise specified, other chemicals used in this study were purchased from Sigma.

### Cell Culture

Human MV4-11 AML cell lines were kindly provided by Dr. L.-I. Lin (National Taiwan University, Taipei, Taiwan), while the HL-60, and U937 cell lines were purchased from the American Type Culture Collection (ATCC; Manassas, VA). All cells were cultured in the recommended conditions, supplemented with 10% heat-inactivated fetal bovine serum (FBS; Gibco, Grand Island, NY), 0.1 mM non-essential amino acids, 2 mM L-glutamine, 100 U/ml penicillin, and 100 μg/ml streptomycin.

### In Vitro Cytotoxicity Assay

AML cells (MV4-11, HL-60, and U937) were plated in 96-well microtiter plates and treated with various concentrations of licochalcone A for 24 h, and cell viabilities were assessed using an MTS (Promega, Madison, WI) assay. The absorbance (A) was read at 490 nm using an enzyme-linked immunosorbent assay (ELISA) reader

(MQX200; Bio-Tek Instruments, Winooski, VT). The cell viability rate (multiples) was determined by: $A_{490,\ licochalcone\ A}/A_{490,\ vehicle}$.

**Flow Cytometric Analysis**

HL-60 cells ($2 \times 10^6$/ml) were treated with vehicle (0.5% DMSO) or 12.5, 25 and 50 μM licochalcone A, and the mixture was allowed to incubate for 24 h. At the end of incubation, cells were collected and fixed with 70% ethanol. Cells were stained with propidium iodide (PI) buffer (4 μg/ml PI, 1% Triton X-100, and 0.5 mg/ml RNase A in phosphate-buffered saline (PBS) for 30 min in the dark at room temperature and then filtered through a 40-μm nylon filter (Falcon, San Jose, CA). The cell-cycle distribution was analyzed for $10^4$ collected cells by a FACS Vantage flow cytometer that uses the Cellquest acquisition and analysis program (Becton-Dickinson FACS Calibur, San Jose, CA). Apoptotic cells with hypodiploid DNA content were detected in the sub-$G_1$ region. All results were obtained from three independent experiments.

**Annexin-V/PI Staining Assay**

Apoptosis-mediated death of tumor cells was examined using a double-staining method with an FITC-labeled Annexin-V/PI Apoptosis Detection kit (BD Biosciences, San Jose, CA). For PI and Annexin-V double-staining, cells were suspended in 100 μl of binding buffer (10 mM HEPES/NaOH, 140 mM NaCl, and 2.5 mM $CaCl_2$ at pH 7.4) and stained with 5 μl of FITC-conjugated Annexin-V and 5 μl of PI (50 μg/ml) for 30 min at room temperature in the dark, and then 400 μl of binding buffer was added. Apoptotic cells were analyzed via flow cytometry, by a FACScan system flow cytometric analysis. Data acquisition and analysis were performed in a Becton-Dickinson FACS Calibur flow cytometer using Cell Quest software (BD

Biosciences).

**Western Blot Analysis**

Cell lysates were prepared as previously described . Equal amounts of protein extracts (20 μg) were subjected to 10% or 12% sodium dodecylsulfate polyacrylamide gel electrophoresis (SDS-PAGE) and blotted onto polyvinylidene fluoride membranes (Millipore, Belford, MA). After blocking, the membrane was incubated with primary antibodies for caspases-9, -3, and -8, PARP, ERK1/2, p-ERK1/2, p38, JNK1/2, p-JNK1/2, Bax, Bcl-2, Bid, α-tubulin, and β-actin. Blots were then incubated with a horseradish peroxidase (HRP)-conjugated anti-mouse or anti-rabbit antibody. Signals were detected via enhanced chemiluminescence using Immobilon Western HRP Substrate (Millipore, Billerica, MA).

**Statistical Analysis**

Values are shown as the mean ± SE. Statistical analyses were performed using the Statistical Package for Social Science software, vers. 16 (SPSS, Chicago, IL). Data comparisons were performed with Student's *t*-test when two groups were compared. A one-way analysis of variance (ANOVA) followed by Tukey's post-hoc test was used when three or more groups were analyzed. Differences were considered significant at the 95% confidence level when $p < 0.05$.

## Results

### Cytotoxic effects of licochalcone A-treated AML Cell Lines

The chemical structure of licochalcone A was shown in Figure 1A. To determine the cytotoxicity and the effect on cell proliferation of licochalcone A on three AML cell lines (HL-60, MV4-11, and U937), cells were treated with different concentrations of licochalcone A (0-50 μM) for 24h. As shown in Figures 1B-1D, after treatment for 24 h, licochalcone A significantly reduced the cell viability in a concentration-dependent manner for the three AML cell lines.

### Licochalcone A-induced cell apoptosis in HL-60 cells

To determine whether the inhibitory effect of cell viability of licochalcone A is associated with induction of cell apoptosis, HL-60 cells were treated with different concentrations (0-50 μM) of licochalcone A for 24 h. Cell cycle analysis by flow cytometry was showed a dose-dependent increased accumulation of cell population in sub-G1 phase after a 24-hour treatment with licochalcone A (Figure 2). Meanwhile, Annexin-V and PI double-staining displayed an increased percentage of apoptotic cells after a 24h treatment of licochalcone A (Figure 3), respectively.

### Licochalcone A induced activation of caspase-3, -8 and -9 in HL-60 cells

To further confirm the involvement of caspase activation in licochalcone A-induced apoptosis, activation of caspases-3, -8, and -9 and cleavage of PARP were detected. Figure 4 shows that exposure of HL-60 cells to licochalcone A (0-50 μM)

for 24 h, caused concentration-dependent increased of the cleaved fragments of caspases-9, -8, and -3. Furthermore, cleaved PARP was also significantly increased in licochalcone A treated HL-60 cells.

**The apoptosis induction by licochalcone A is dependent on the regulation of JNK1/2 signaling pathways in HL-60 cells**

In a further investigation for the underlying molecular mechanisms, we determined whether MAPKs were activated in licochalcone A-treated HL-60 cells by a Western blot analysis. Results showed that the phosphorylation of ERK1/2, JNK1/2 and p38 MAPK were increased in cells treated with licochalcone A in a dose-dependent manner (Figure 5). Next, we further investigated relationships among licochalcone A induced activation of caspases-3 and MAPKs. HL-60 cells were pretreated with 10 μM U0126 (an ERK inhibitor), JNK-In-8 (a JNK inhibitor), or SB202190 (a p38 inhibitor) for 1 h, treated with 25 μM licochalcone A for another 24 h, and then analyzed by Western blotting. As shown in Figure 6, treatment JNK-In-8 significantly attenuated licochalcone A-induced caspase-3 activation. These findings suggest that activation of JNK1/2 might play a critical upstream role in licochalcone A-mediated caspase activation in HL-60 cells.

**References:**


[1] Bishop JF. The treatment of adult acute myeloid leukemia. Semin Oncol 1997; 24: 57-69.

[2] Tallman MS, Gilliland DG and Rowe JM. Drug therapy for acute myeloid leukemia. Blood 2005; 106: 1154-1163.

[3] Fu B, Li H, Wang X, Lee FS and Cui S. Isolation and identification of flavonoids in licorice and a study of their inhibitory effects on tyrosinase. J Agric Food Chem 2005; 53: 7408-7414.

[4] Tsai JP, Hsiao PC, Yang SF, Hsieh SC, Bau DT, Ling CL, Pai CL and Hsieh YH. Licochalcone A suppresses migration and invasion of human hepatocellular carcinoma cells through downregulation of MKK4/JNK via NF-kappaB mediated urokinase plasminogen activator expression. PLoS One 2014; 9: e86537.

[5] He W, Wu JJ, Ning J, Hou J, Xin H, He YQ, Ge GB and Xu W. Inhibition of human cytochrome P450 enzymes by licochalcone A, a naturally occurring constituent of licorice. Toxicol In Vitro 2015; 29: 1569-1576.

[6] Hu J and Liu J. Licochalcone A Attenuates Lipopolysaccharide-Induced Acute Kidney Injury by Inhibiting NF-kappaB Activation. Inflammation 2016; 39: 569-574.

[7] Huang HC, Tsai LL, Tsai JP, Hsieh SC, Yang SF, Hsueh JT and Hsieh YH. Licochalcone A inhibits the migration and invasion of human lung cancer cells via inactivation of the Akt signaling pathway with downregulation of MMP-1/-3 expression. Tumour Biol 2014; 35: 12139-12149.

[8] Kim KH, Yoon G, Cho JJ, Cho JH, Cho YS, Chae JI and Shim JH. Licochalcone A induces apoptosis in malignant pleural mesothelioma through downregulation of Sp1 and subsequent activation of mitochondria-related apoptotic pathway. Int J Oncol 2015; 46: 1385-1392.

[9] Park MR, Kim SG, Cho IA, Oh D, Kang KR, Lee SY, Moon SM, Cho SS, Yoon G, Kim CS, Oh JS, You JS, Kim DK, Seo YS, Im HJ and Kim JS. Licochalcone-A induces intrinsic and extrinsic apoptosis via ERK1/2 and p38 phosphorylation-mediated TRAIL expression in head and neck squamous carcinoma FaDu cells. Food Chem Toxicol 2015; 77: 34-43.

[10] Tsai JP, Lee CH, Ying TH, Lin CL, Lin CL, Hsueh JT and Hsieh YH. Licochalcone A induces autophagy through PI3K/Akt/mTOR inactivation and autophagy suppression enhances Licochalcone A-induced apoptosis of human cervical cancer cells. Oncotarget 2015; 6: 28851-28866.

[11] Xin H, Qi XY, Wu JJ, Wang XX, Li Y, Hong JY, He W, Xu W, Ge GB and Yang L. Assessment of the inhibition potential of Licochalcone A against

human UDP-glucuronosyltransferases. Food Chem Toxicol 2016; 90: 112-122.

[12] Yang X, Jiang J, Yang X, Han J and Zheng Q. Licochalcone A induces T24 bladder cancer cell apoptosis by increasing intracellular calcium levels. Mol Med Rep 2016; 14: 911-919.

**Figure 1**

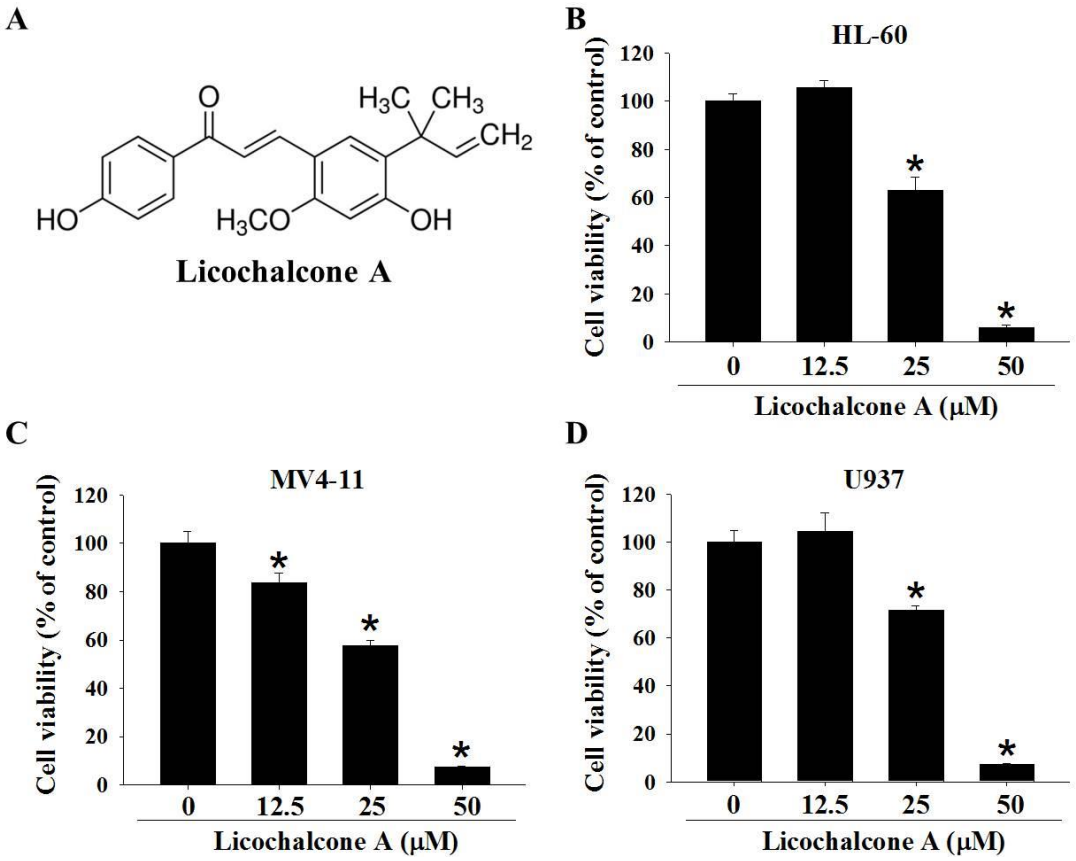


**Figure 1. Cytotoxic effect of licochalcone A in three AML cell lines (HL-60, U937 and MV4-11).** Cells were treated with different concentrations of licochalcone A (0, 12.5, 25, and 50 μM) for 24 h and then observed under microscopes to reveal cell death in a dose-dependent manner. Data represent mean of 3 determinations per condition repeated 3 times. Results are shown as mean ± SE.

**Figure 2**

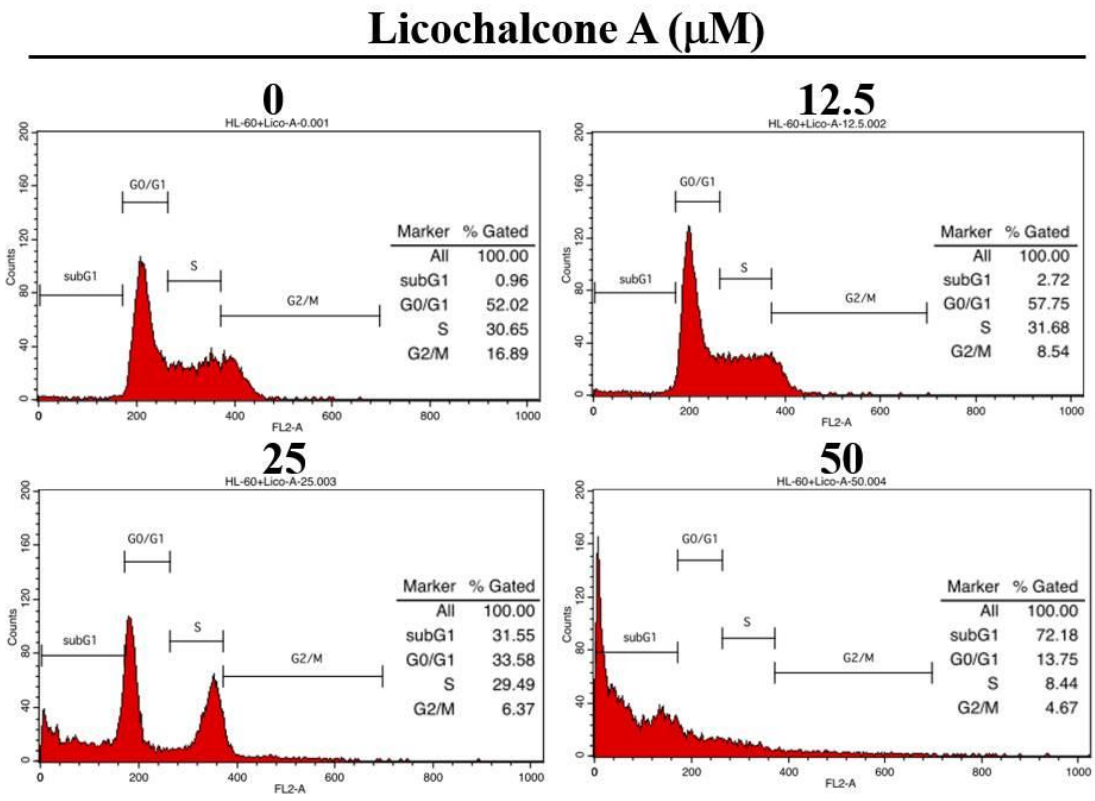


**Figure 2. Licochalcone A induced cell apoptosis in HL-60 cells.** HL-60 cells were incubated for 18 h in the absence of serum and then treated with indicated concentrations of licochalcone A (0, 12.5, 25, and 50 μM) for 24 h, after which the cells were stained with PI, and analyzed for DNA content by flow cytometry.

**Figure 3**

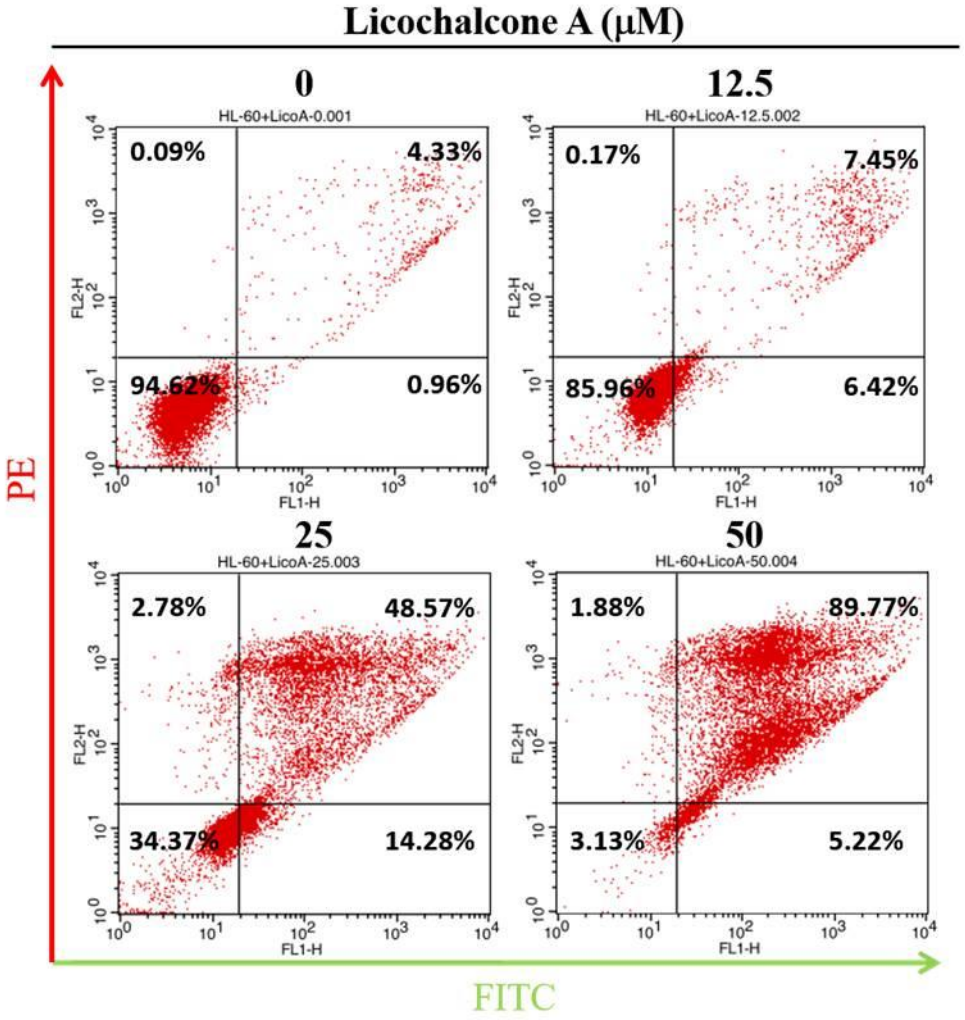


**Figure 3. Licochalcone A induced cell apoptosis in HL-60 cells by Annexin-V and PI double-stained flow cytometry.** HL-60 cells were incubated for 18 h in the absence of serum and then treated with indicated concentrations of licochalcone A (0, 12.5, 25, and 50 μM) for 24 h, cells were harvested and then subjected to quantitative analysis of cell apoptosis by Annexin-V and PI double-stained flow cytometry.

**Figure 4**

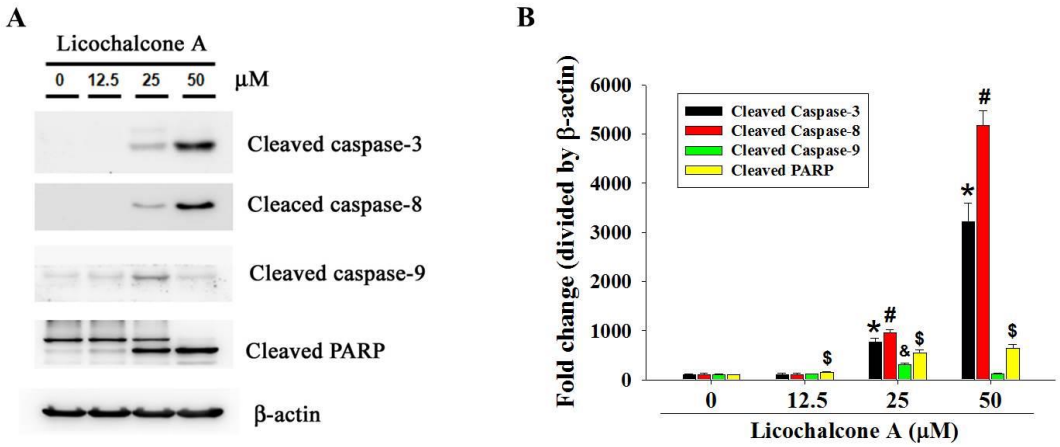


**Figure 4. Activation of caspase 3, -8, -9 and PARP were increased in Licochalcone A-treated HL-60 cells.** HL-60 cells were treated with 0, 12.5, 25 and 50 μM licochalcone A for 24 h, subjected to western blotting with an antibody against PARP or cleaved caspase-3, -8 and -9 antibodies.

**Figure 5**

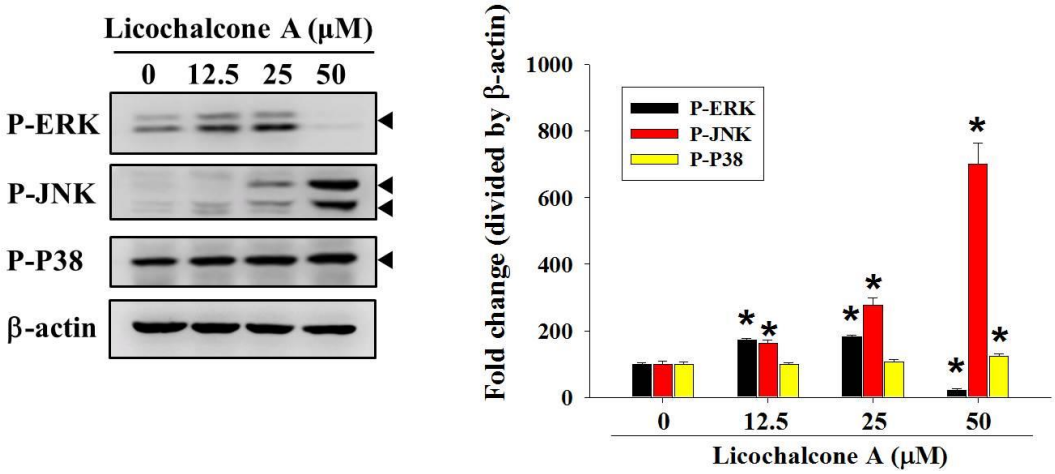


**Figure 5**. **Licochalcone A activates the phosphorylation of ERK1/2, p38 MAPK and JNK1/2 in HL-60 cells.** Cells were treated with different concentrations of Licochalcone A (0-50 μM) for 24 h and then subjected to western blotting with an antibody against ERK1/2, JNK1/2, and p38 MAPK. Values represent the mean ± SE of three independent experiments. (*) $p < 0.05$ compared to the vehicle control group.

**Figure 6**

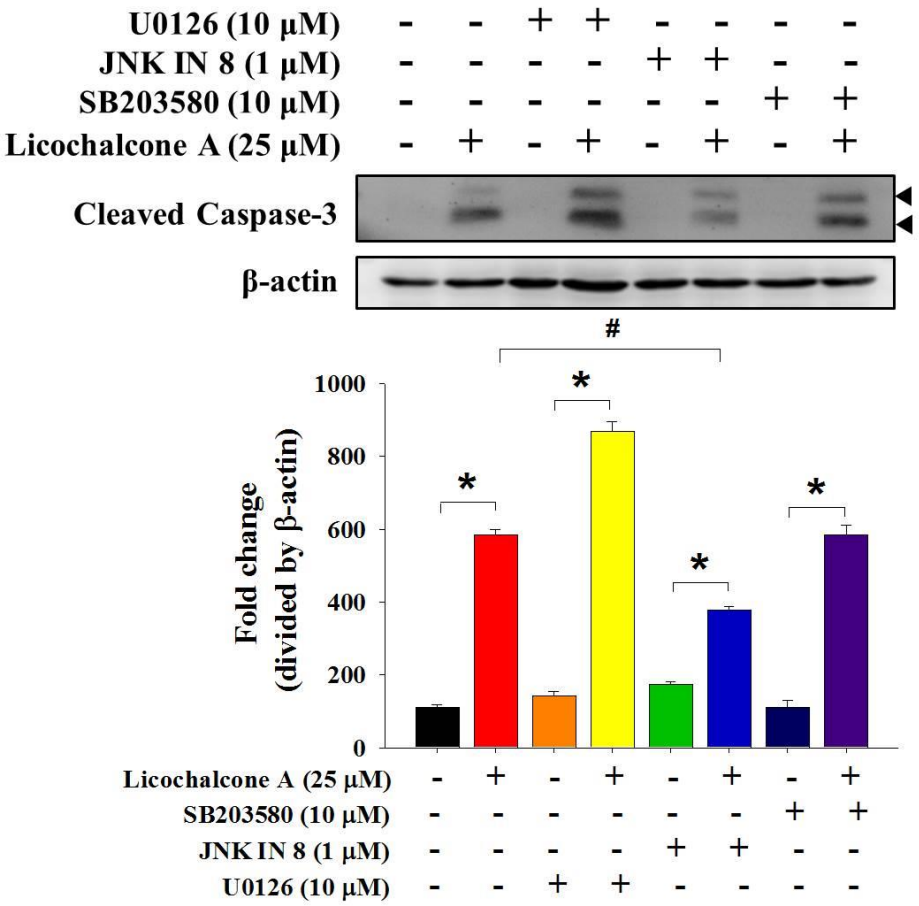


**Figure 6. JNK1/2 are essential for caspase activation induced by Licochalcone A**. HL-60 cells were treated for 24 hours with 20 μM Licochalcone A with or without a 1-hour pretreatment of 10 μM U0126, JNK-IN-8, or SB203580. The expression of cleaved caspase-3 was detected by western blotting. Quantitative results of cleaved caspase-3 protein levels, which were adjusted to the β-actin protein level and expressed as multiples of induction beyond each respective control. Values represent the mean ± SE of three independent experiments.

# 科技部補助計畫衍生研發成果推廣資料表

日期:2016/10/20

| | |
|---|---|
| 科技部補助計畫 | 計畫名稱: 甘草酸衍生物抑制不同血癌細胞增生分化能力及誘導細胞凋亡的機制探討 |
| | 計畫主持人: 蕭培靜 |
| | 計畫編號: 104-2314-B-040-016- 學門領域: 血液 |

無研發成果推廣資料

# 104年度專題研究計畫成果彙整表

| | |
|---|---|
| **計畫主持人：**蕭培靜 | **計畫編號：**104-2314-B-040-016- |
| **計畫名稱：**甘草酸衍生物抑制不同血癌細胞增生分化能力及誘導細胞凋亡的機制探討 | |

| 成果項目 | | | | | 量化 | 單位 | 質化<br>（說明：各成果項目請附佐證資料或細項說明，如期刊名稱、年份、卷期、起訖頁數、證號...等） |
|---|---|---|---|---|---|---|---|
| 國內 | 學術性論文 | 期刊論文 | | | 0 | 篇 | |
| | | 研討會論文 | | | 1 | | |
| | | 專書 | | | 0 | 本 | |
| | | 專書論文 | | | 0 | 章 | |
| | | 技術報告 | | | 0 | 篇 | |
| | | 其他 | | | 0 | 篇 | |
| | 智慧財產權及成果 | 專利權 | 發明專利 | 申請中 | 0 | 件 | |
| | | | | 已獲得 | 0 | | |
| | | | 新型/設計專利 | | 0 | | |
| | | 商標權 | | | 0 | | |
| | | 營業秘密 | | | 0 | | |
| | | 積體電路電路布局權 | | | 0 | | |
| | | 著作權 | | | 0 | | |
| | | 品種權 | | | 0 | | |
| | | 其他 | | | 0 | | |
| | 技術移轉 | 件數 | | | 0 | 件 | |
| | | 收入 | | | 0 | 千元 | |
| 國外 | 學術性論文 | 期刊論文 | | | 0 | 篇 | |
| | | 研討會論文 | | | 0 | | |
| | | 專書 | | | 0 | 本 | |
| | | 專書論文 | | | 0 | 章 | |
| | | 技術報告 | | | 0 | 篇 | |
| | | 其他 | | | 0 | 篇 | |
| | 智慧財產權及成果 | 專利權 | 發明專利 | 申請中 | 0 | 件 | |
| | | | | 已獲得 | 0 | | |
| | | | 新型/設計專利 | | 0 | | |
| | | 商標權 | | | 0 | | |
| | | 營業秘密 | | | 0 | | |
| | | 積體電路電路布局權 | | | 0 | | |
| | | 著作權 | | | 0 | | |
| | | 品種權 | | | 0 | | |
| | | 其他 | | | 0 | | |

| | | | | | |
|---|---|---|---|---|---|
| | 技術移轉 | 件數 | 0 | 件 | |
| | | 收入 | 0 | 千元 | |
| 參與計畫人力 | 本國籍 | 大專生 | 0 | 人次 | |
| | | 碩士生 | 1 | | |
| | | 博士生 | 0 | | |
| | | 博士後研究員 | 0 | | |
| | | 專任助理 | 1 | | |
| | 非本國籍 | 大專生 | 0 | | |
| | | 碩士生 | 0 | | |
| | | 博士生 | 0 | | |
| | | 博士後研究員 | 0 | | |
| | | 專任助理 | 0 | | |
| 其他成果<br>（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。） | | | | | |

# 科技部補助專題研究計畫成果自評表

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（簡要敘述成果是否具有政策應用參考價值及具影響公共利益之重大發現）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（請於其他欄註明專利及技轉之證號、合約、申請及洽談等詳細資訊）
   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200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）
   甘草(Licorice)是傳統中藥材之一。甘草中的黃酮類化合物主要為chalcones，而chalcones中的licochalcone A在先前研究已經證實具有抗癌的特性。然而licochalcone A對於人類急性骨髓性白血病 (acute myeloid leukemia, AML)的影響仍尚未釐清。我們利用licochalcone A，處理人類三株人類急性骨髓性白血病細胞株並分析其細胞存活率。結果發現licochalcone A會抑制三株骨髓性白血病細胞株的生長，我們也利用flow cytometer 分析方式發現licochalcone A會誘導HL-60的細胞凋亡。此外，licochalcone A會經由活化caspase-3、8、9及裂解PARP來誘發HL-60細胞凋亡。這也暗示著licochalcone A適合作為另一種有效的人類急性骨髓性白血病的抗癌輔助藥物。

4. 主要發現
   本研究具有政策應用參考價值：■否　□是，建議提供機關
   （勾選「是」者，請列舉建議可提供施政參考之業務主管機關）
   本研究具影響公共利益之重大發現：■否　□是
   說明：（以150字為限）