Thesis for the Degree of Master of Agiculture

Panax ginseng leaf extracts exert anti-obesity effects in high fat diet-induced obese rats

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Major of Food Application Technology
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1. Introduction

Obesity is one of the major public health problems and is associated with increased metabolic diseases worldwide (8, 16). Obesity is caused by an imbalance between energy intake and energy expenditure, generated excessive adipose tissue (5, 13). Adipose tissue mass can be regulated by adipogenesis process or adipocyte differentiation that has been extensively studied for the treatment of prevention of obesity (7, 12). However, As of September 2013, drug approved by the U.S. Food and Drug Administration (FDA) for the long-term treatment of obesity was only one, a gastrointestinal lipase inhibitor (2). Therefore, the strategy for the prevention of obesity using health functional food is considered to be important, as much as treatment.

The root of ginseng has been traditionally used for the prevention and treatment of various diseases for over 2000 years, however, aerial parts of ginseng did not be effectively utilized. Previous studies reported that the leaf of ginseng is rich in bioactive compounds and contents of some ginsenosides are higher than in the roots (10). Also, stem, leaf, and berry of American ginseng (*Panax quinquefolium)* had anti-obesity activities in vivo (15, 17, 20). However, the anti-obesity effect of leaf extracts of Korean ginseng (*Panax ginseng* CA Meyer) has not yet been investigated.

In the present study, we show that leaf extracts of Korean ginseng were significantly reduce body weight in high-fat diet induced obese rats and inhibit the differentiation of 3T3-L1 adipocytes through regulating the

expression of key transcriptional factors.

2. Materials and Methods

2.1 Preparation of ginseng leaf extracts

Leaf extracts of ginseng were provided by Punggi Ginseng Experimental Station (Yeongju, Korea). Two different types of leaf extract from ginseng were prepared with green leaf (GL) and dried leaf (DL) of ginseng. GL and DL were extracted with distilled water and stored at 4 °C for further use.

2.2. Animals

Five-week old, male Sprague-Dawley (SD) rat were purchased from HyoChang Animal (Seoul, Korea). After 1 week adaptation period, animals were randomly grouped as follows: 1) normal diet (ND) 2) high fat diet (HFD) 3) HFD + GL supplementation (3.3 mg/kg) 4) HFD + DL supplementation (3.3 mg/kg) groups. The composition of high-fat diet is shown in Table 1 and all rats were maintained under a 12 h light-dark cycle. GL and DL were orally administered to the rat everyday throughout the experiment period. Each experimental group consisted of 7 animals. After finishing treatment, blood and various adipose tissues for further analysis.

Ingredients (g/kg of diet)	ND	HFD
Corn starch	400	200
Casein	200	200
Sucrose	200	140
Cellulose	50	50
Mineral mix	35	35
Vitamin mix	10	10
D,L-methionine	3.5	3
Choline	1.5	2
Corn oil	60	210
Lard	40	150

Table 1. The composition of experimental diets.

2.3. Biochemical analysis

On the final day of the experiments, the blood was collected into the serum collection tubes and centrifuged at 3000 rpm for 15 min to obtain the plasma supernatant. Plasma marker levels for nephrotoxicity, hepatotoxicity test, and lipid profiling were performed in Food Analysis Research Center in Suwon Women's University (Korea).

2.4 Cell culture and differentiation

The 3T3-L1 mouse preadipocyte cells were purchased from the Korea Cell Line Bank (KCLB, Seoul, Korea) and cultured in Dulbecco's modified Eagle medium (DMEM; GIBCO, NY, USA) supplemented with 10% bovine calf serum (BCS; GIBCO). To differentiation the 3T3-L1 adipocytes were cultured to post-confluence (designated as day 0) for 2 days and the media was replaced with DMEM containing 10% fetal bovine serum (FBS; GIBCO) and MDI solution (0.5 mM IBMX, 1 μM DEXA, 0.125 mM indomethacin, and 10 μg/mL insulin) for 2 days. After, the cells were maintained in media supplemented with 10% FBS and 10 μg/mL insulin for 4 days. To examine the anti-adipogenic effect of leaf extracts of ginseng, the cells undergoing differentiation were treated with GL and DL during all stages of differentiation.

2.5. Oil Red O staining and triglyceride (TG) assay

Oil Red O solution (ORO) and TG assay were performed as previously described (submitted for publication). Briefly, for staining of lipid droplets, the cells undergo differentiation into mature adipocytes were washed, fixed and stained with Oil red O solution to detect lipid droplets. All images were obtained on microscope (Leica, Germany). TG assay was performed according to the manufacturer's instructions.

2.6. Reverse transcription-polymerase chain reaction (RT-PCR)

RNA was isolated using RNAiso Plus reagent (TaKaRa Bio, Japan). Complementary DNA was synthesized using the Prime-Script RT reagent Kit (TaKaRa Bio) according to the manufacturer's protocol. The specific rat and mouse primers were designed as shown in Table 2. The expressions of mRNA in 3T3-L1 adipocytes and epididymis adipose tissues were normalized to b-actin or GAPDH using Image J software, respectively (National Institutes of Health, Bethesda, MD, USA). The sequences of the designed primers are shown in Table 2.

Gene name	Accession no.	Forward primer	Reverse primer
aP2 (M)	NM_024406	AACACCGAGATTTCCTTCAA	TCACGCCTTTCATAACACAT
Adiponectin (M)	NM_009605	ACCTACGACCAGTATCAGGAAAAG	ACTAAGCTGAAAGTGTGTCGACTG
C/EBPα (M)	NM_001287523	TTACAACAGGCCAGGTTTCC	GGCTGGCGACATACAGATCA
C/EBPα (R)	NM_001287579	GCCAAGAAGTCGGTGGATAA	CCTTGACCAAGGAGCTCTCA
C/EBPβ (M)	NM_009883	GACTACGCAACACACGTGTAACT	CAAAACCAAAAAACATCAACAACCC
C/EBPδ (M)	NM_007679	GATCTGCACGGCCTGTTGTA	CTCCACTGCCCACCTGTC
LPL (M)	NM_008509	TCCTCTGACATTTGCAGGTCTATC	TCACGCCTTTCATAACACAT
PPAR γ (M)	AB644275	TTTTCAAGGGTGCCAGTTTC	AATCCTTGGCCCTCTGAGAT
PPAR γ (R)	NM_013124	CTTGGCCATATTTATAGCTGTCATTATT	TGTCCTCGATGGGCTTCAC
β-actin (M)	EF095208	GACAACGGCTCCGGCATGTGCAAAG	TTCACGGTTGGCCTTAGGGTTCAG
GAPDH (R)	NM_017008	TCTGACATGCCGCCTGGAGAA	TGGAGGCCATGTAGGCCATGA

M, Mouse; R, Rat

Table 2. Sequences and accession numbers for primers used in RT-PCR.

2.7. Western blot

Total proteins were extracted using with pro-prep lysis buffer (iNtRON BIOTECHNOLOGY) containing phosphatase inhibitors and protease inhibitor cocktail. Lysates were centrifuged at 13,000 rpm for 15 min. Protein samples were separated by SDS-PAGE, transferred onto nitrocellulose membranes, blocked using 5% non-fat skim milk and incubated with antibodies against PPARγ, LPL, Ap2, Adiponectin (Abcam plc, Cambridge, UK), C/EBPα (Cell Signaling Technology, Beverly, MA, USA), and β-actin (Santa Cruz Biotechnology, CA, USA) overnight at 4°C.

2.8. Statistical analysis

Data are expressed as mean ± SD. Statistical comparisons were performed using 1-way ANOVA. Values of $P<0.05$ were considered statistically significant (*P < 0.05, **P < 0. 01).

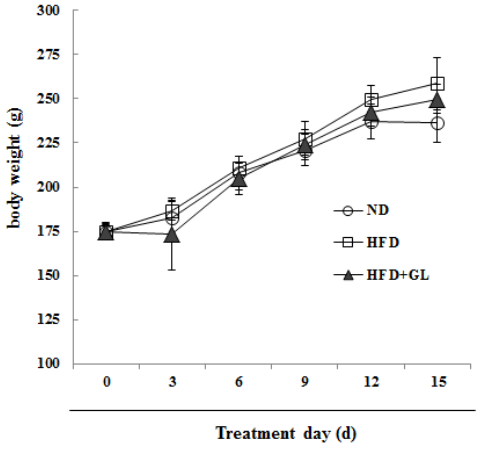
3. Results

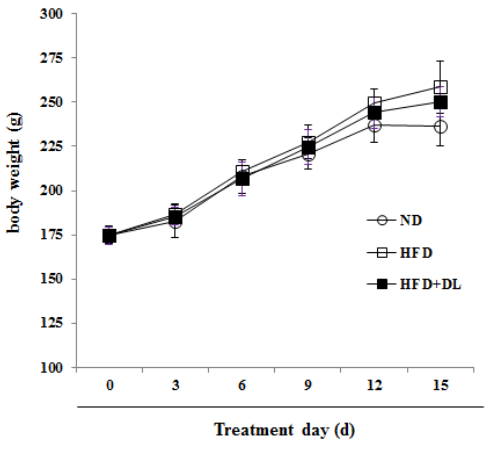
3.1. Effects of GD and DL on body weight, adipose tissue mass, and food intake in high-fat diet induced obese rats

In order to examine the biological effectiveness of GL and DL, we confirmed the anti-obesity effects of GL and DL in diet induced obese rats. Final body weights were slightly lower in GL and DL supplementation rats (HFD+GL and HFD+DL rats), compared to the HFD rats without supplementation (HFD-fed rats), although there was no significant difference (Fig. 1A). Adipose tissues mass (epididymis, abdominal) were significantly decreased in the HFD+GL and HFD+DL rats, compared to the HFD-fed rats (Fig. 1B).

Also, the HFD+GL and HFD+DL rats were decreased the food efficiency ratio, although there was no significant difference (Fig. 1C).

(A)





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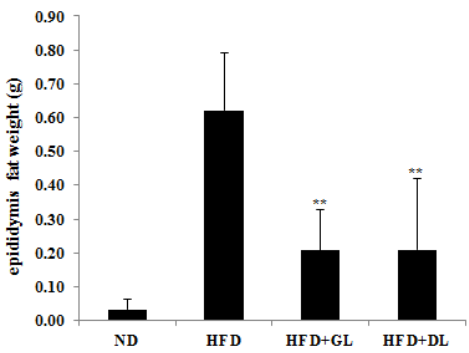

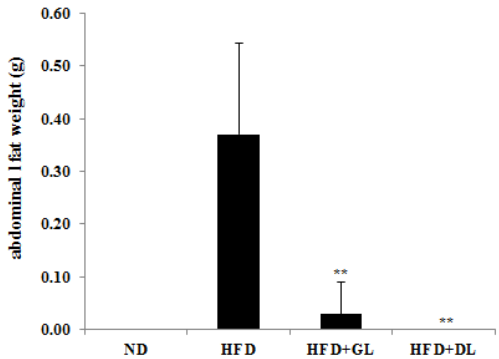

(C)

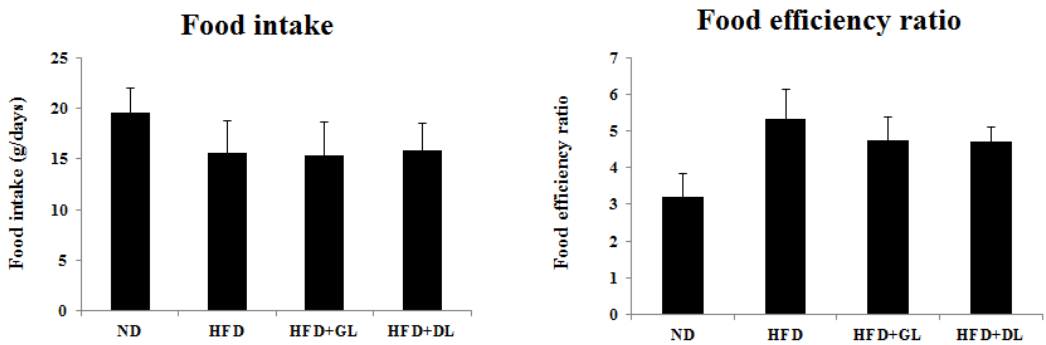

Fig. 1. Effects of GD and DL on body weight, adipose tissue mass, and food intake in high-fat diet induced obese rats. **(A)**The body weight of rat was measured every 3 days (n=7 rat/group). Rats were fed with ND or HFD. The HFD-fed rats were treated with or without GL or DL at a dose of 3.3mg/kg/day.**(B)**Total epididymal WAT and abdominal WAT weight. **(C)** Food intake an d Food efficiency ratio. Significantly different from the high-fat diet group (HFD), P values of <0.01(**) or<0.05(*).

3.2. Effects of GL and DL on activity levels of serum marker enzymes

To evaluate the safety of GL and DL, We confirmed the nephrotoxic and hepatotoxic effects of GL and DL. The levels of ALB, TBIL, BUN, and CRE were no significant differences between HFD-fed rats and HFD+GL or HFD+DL rats (Table 3). As expected, GL and DL did not affect to the other organs mass, compared to the HFD rats (Table 4). Furthermore, GL and DL reduces HDL-cholesterol and increases LDL-cholesterol and triglyceride levels.

	ND	HFD	HFD+GL	HFD+DL
ALB (g/dL)	2.2±0.06	2.3±0.18	2.1±0.28	2.2±0.29
Tbil (mg/dl)	0.2±0.02	0.2±0.02	0.2±0.02	0.2±0.02
BUN (mg/dl)	28.0±3.80	20.5±4.91	19.2±5.59	22.9±4.77
CRE (mg/dl)	0.3±0.07	0.3±0.06	0.3±0.01	0.3±0.03
TG (mg/dl)	43.8±27.13	72.2±24.13	24.1±12.04**	32.1±17.28*
HDL (mg/dl)	31.0±4.05	34.3±1.83	33.5±5.31	41.3±6.41 *
LDL (mg/dl)	10.3±1.51	19.2±3.65	12.5±1.83**	12.6±0.94 **

Table 3. Plasma biochemical values in male rats. Significantly different from the high-fat diet group (HFD), P values of <0.01(**) or <0.05(*).

		ND	HFD	HFD+GL	HFD+DL
Liver weight	(g)	4.9±0.51	7.3±1.40	4.7±0.67*	5.2±0.49*
Kidney weight	(g)	1.7±0.07	1.9±0.12	1.7±0.11	1.8±0.18
Heart weight	(g)	0.6±0.03	0.8±0.07	0.7±0.13	0.7±0.05
Spleen weight	(g)	0.3±0.04	0.4±0.03	0.3±0.08	0.3±0.06
Pancreas	(g)	0.6±0.10	0.4±0.17	0.8±0.16	1.0±0.10
Muscle weight					
Gastrocnemius	(g)	0.1±0.02	0.2±0.01	0.1±0.01	0.2±0.01
Soleus	(g)	2.0±0.13	2.0±0.28	1.9±0.32	1.8±0.21

Table 4. Effects of GL and DL on the weights of liver, kidney, heart, spleen, pancreas, and muscle tissue in male rats. Significantly different from the high-fat diet group (HFD), P values of <0.01(**) or <0.05(*).

3.3. Effects of GD and DL on the mRNA and protein expression of adipogenic-related genes in adipose tissues

To investigate the mechanisms underlying the anti-obesity effects of GL and DL, we measured the mRNA and protein expression of adipogenesis-related genes, such as key adipogenic regulators (C/EBP α and PPARγ). The mRNA and protein expression of C/EBPα and PPARγ were significantly lower in the epididymal adipose tissues of HFD+GL and HFD+DL rats compared to the HFD-fed rats (Fig. 2A-B). Also, the protein expression of downstream target genes of C/EBPα and PPARγ, including aP2 and adiponectin, was decreased in both HFD+GL and HFD+DL rats (Fig. 2B).

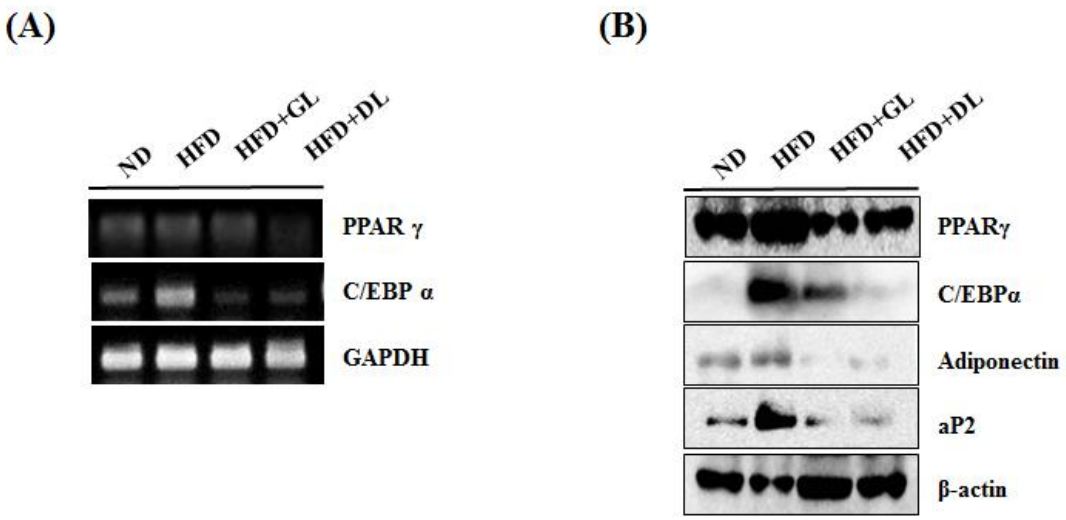

Fig. 2. Effects of GD and DL on the mRNA and protein expression of adipogenic-related genes in adipose tissues. Rats were fed with a ND or HFD. The HFD-fed rats were treated with or without GL or DL at a dose of 3.3 mg/kg/day. Epididymal WAT was acquired at the final day of the experiments. **(A)** The mRNA expression of PPARγ, C/EBPα, and GAPDH in epididymal WAT. **(B)** The protein expression of the indicated adipogenic genes in epididymal WAT.

3.4. Effects of GD and DL on proliferation of 3T3-L1adipocytes

In order to investigate whether the anti-obesity effects of the GL and DL in vivo were occurred by inhibiting adipogenesis of adipocytes, we conducted an in vitro experiment using 3T3-L1 adipocytes.

First, we determine the cytotoxicity effects of GL and DL. 3T3-L1 adipocytes were treated with GL or DL in preadipocytes or fully differentiated adipocyte (mature adipocytes). GL and DL did not affect to viability of 3T3-L1 preadipocytes and fully differentiated adipocyte (mature adipocytes) (Fig. 3).

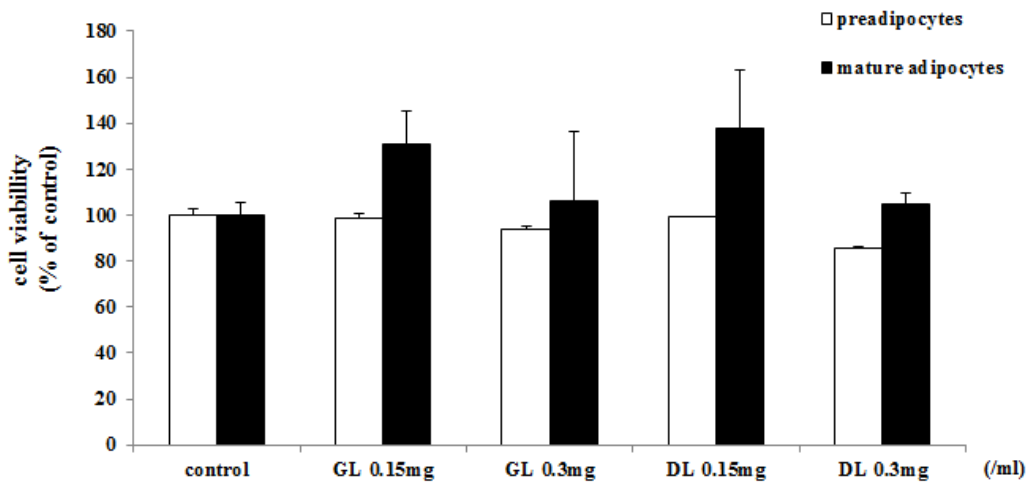

Fig. 3. Effects of GL and DL on cell viability of 3T3-L1 preadipocytes and differentiated adipocytes. 3T3-L1 preadiocytes were treated with GL or DL for 48 h or were left untreated. 3T3-L1 preadipocytes were differentiated into adipocytes and treated with GL or DL for entire differentiation period or after induction of differentiation. Cell viability was determined by MTT assay and measured as absorbance at 495 nm.

3.5. Effects of GD and DL on differentiation and lipid accumulation of 3T3-L1 adipocytes

3T3-L1 adipoyctes were treated with GL or DL throughout the period of differentiation. DL significantly decreases the lipid accumulation in a dose-dependent manner (Fig. 4A-B). Furthermore, DL at a dose of 0.15 and 0.3 mg/ml markedly decreased the intracellular TG levels by 15.0% and 30.1%, respectively (Fig. 4C). However, GL had little effect on the differentiation of 3T3-L1 adipocytes, suggesting that anti-obesity effects of GL in vivo were caused by specific factor released other organs or increased energy expenditure.

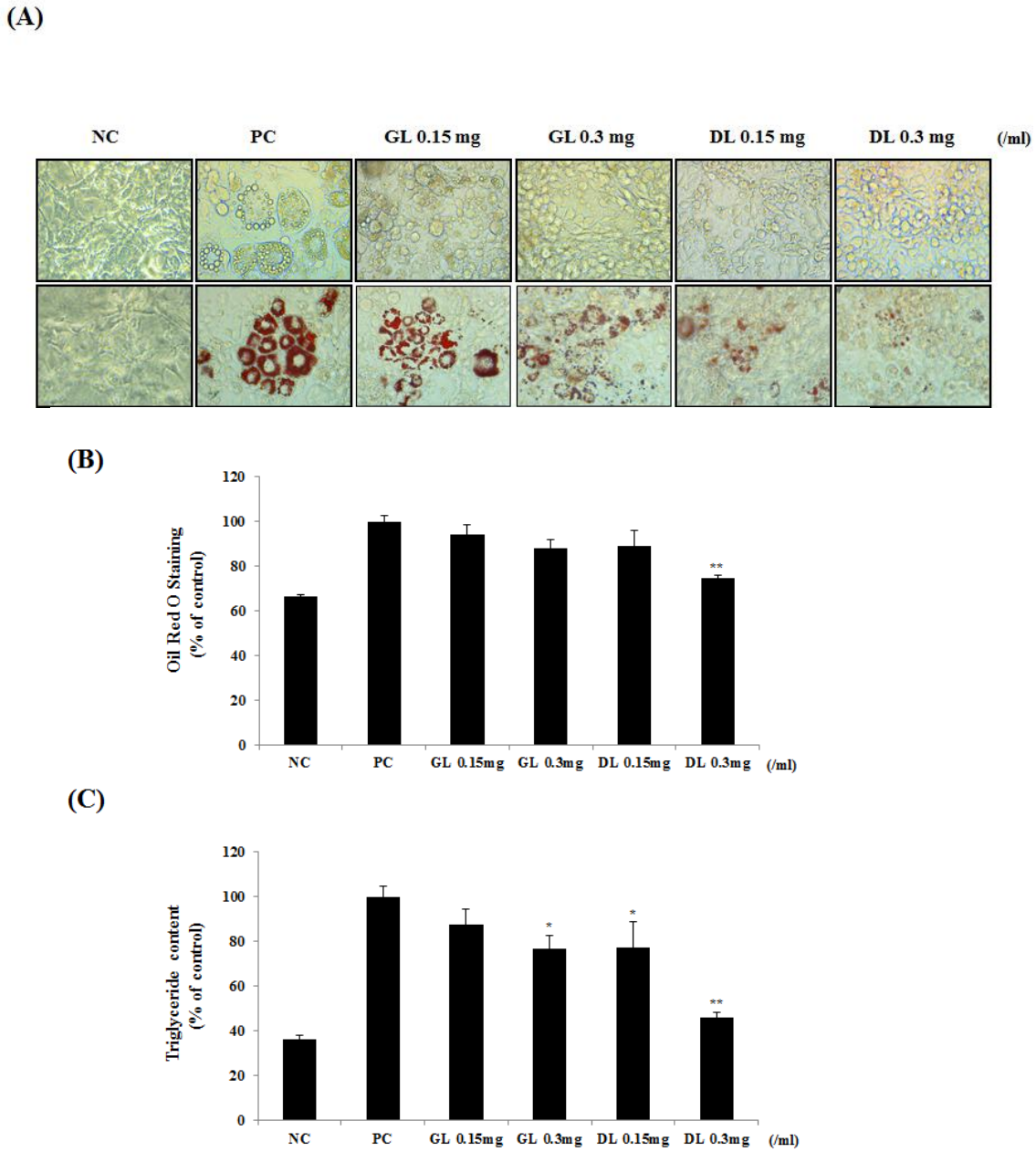

Fig. 4. Effects of GL and DL on the differentiation and lipid accumulation of 3T3-L1. 3T3-L1 preadiocytes were treated with GL and DL for 8 days or were left untreated. (A) The differentiated cells were stained by Oil Red O

and photographed with a microscope at 400 × magnification. (B) The stained cells by Oil Red O measured as absorbance at 495 nm. (C) Intracellular triglyceride concentrations measured as absorbance at 570 nm. Significantly different from the high-fat diet group (HFD), P values of <0.01(**) or <0.05(*).

3.6. Effect of GD and DL on the mRNA and protein expression of adipogenic-related genes during 3T3-L1 adipocytes

To investigate the mechanisms underlying the anti-differentiation effects of GL and DL, we measured the mRNA and protein expression levels of adipogenic-related genes, such as key adipogenic regulators (C/EBPs and PPARγ). Treatment of DL significantly decreased the mRNA and protein expression levels of C/EBPα, PPARγ, and adiponectin and also decreased the mRNA expression of fatty acid-binding protein 4 (aP2), lipoprotein lipase (LPL), adiponectin, and C/EPBs (β and δ) (Fig. 5A-B). GL only slightly reduced the mRNA and protein expressions of adipogenesis-related genes. Remarkably, GL greatly increased the protein expression level of PPARγ than untreated cells.

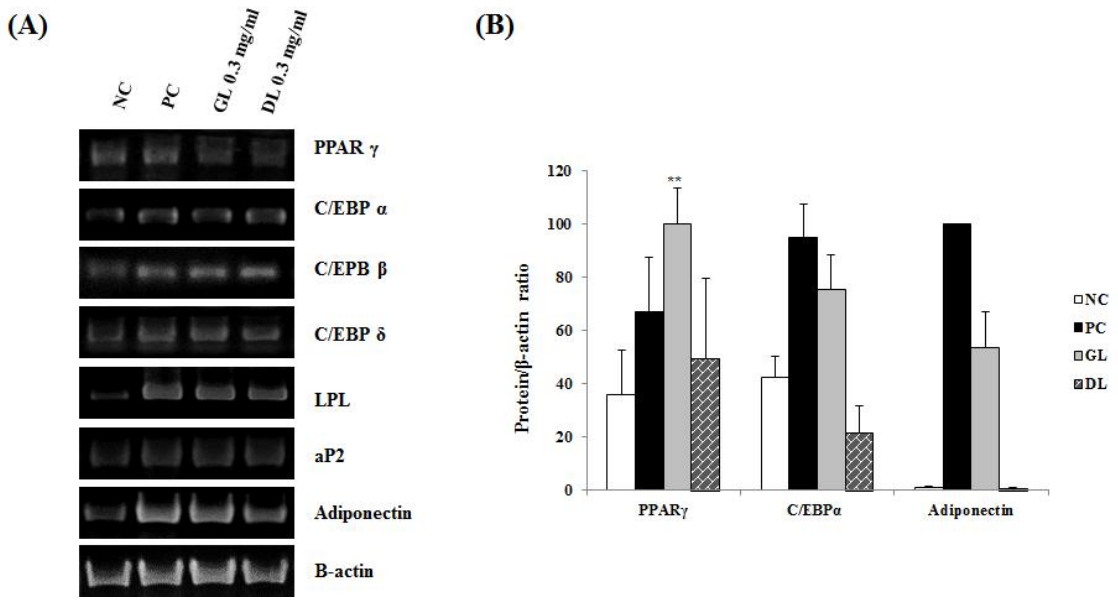

Fig. 5. Effects of GL and DL on mRNA and protein expressions of adipogenesis-related genes in 3T3-L1 adipocytes. Cells were treated with GL and DL for 8 days during differentiation and total RNA and protein was isolated. **(A)** The mRNA expression of the adipogenic genes in 3T3-L1 cells. **(B)** The protein expression of PPARγ, C/EBPα, anda diponectin in 3T3-L1 cells.

4. Discussion

A previous review indicated that the root and leaf-stem extracts of ginseng have similar pharmacological effects (15). Also, a number of studies found that the berry extracts of ginseng could also be used to treat diabetes in obese mice (3, 4, 18, 19).

In this study, we found that two kinds of leaf extracts from ginseng have anti-obesity effects in vivo through different mechanisms. GL and DL similarly reduced the body weight gain and adipose tissue mass in high-fat induced obese rats. In the case of DL, these effects were thought to be caused by a decreased differentiation of adipocytes. The expression of mRNA and protein analyses revealed that DL regulated adipogenesis process in 3T3-L1 adipocytes by modulating adipogenic gene expression. On the other hands, GL had little effects on the differentiation of adipocytes. Interestingly, cells treated GL were significantly increased the protein expression of PPARγ than untreated cells.

PPARγ is mainly expressed in adipose tissues (WAT, BAT) and a master regulator of adipogenesis as well as a potent metabolic modulator of whole-body lipid metabolism and insulin sensitivity (1, 6, 14). Previous studies report that the mice, a lack of skeletal-muscle PPARγ, developed excess adiposity and hepatic insulin resistance (1, 11). Another study indicates that PPARγ hypomorphic mice have mild lipodystrophy because of an efficient compensation by other organs such as, muscle (9). Therefore, from a dynamic point of view, we hypotheses that anti-obesity

effects of GL may be caused by improved glucose homeostasis or energy expenditure through greatly increased expression of PPARr. However, further experiments such as, glucose homeostasis changes by supplement with GL are required to establish this hypothesis.

In summary, our results demonstrate that the leaf extracts of korean ginseng have anti-osbesity effects by selectively reducing the adipose tissue mass. DL suppressed the adipogenesis of 3T3-L1 adipocytes by modulating the expression of central transcription factors, but not GL. These results were probably due to metabolism modulation or increased energy expenditure. However, additional experimentation is needed to further clarify this issue. To our knowledge, these finding are the first to provide anti-obesity effects of the leaf extracts of Korean ginseng, it can be used to a potential therapeutic agent or dietary supplement against obesity.

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고려인삼 잎 추출물에 대한 항비만 효과

이슬기

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초록

최근 한 연구는 인삼의 지상부 (열매, 잎, 줄기) 가 뿌리만큼 다양하고 많은 사포닌을 포함하고 있다고 보고한 바 있으며 이러한 사포닌의 항산화, 항염증 및 항비만 효과는 밝혀진 바 있다. 그러나, 고려인삼 잎 추출물의 항비만효과는 아직 밝혀진 바 없다. 그러므로, 본 연구에서는 고려인삼의 생잎 (Green leaf of ginseng, GL) 및 건조된 잎 (Dried leaf of ginseng, DL) 열수 추출물의 항비만 효과를 생체 내, 외에서 조사하였다. GL 및 DL의 섭취는 고지방식이에 의한 비만유도 쥐의 몸무게 및 부고환 지방, 복강 후 지방 조직을 감소시켰다. 또한, 생체 외에서 DL은 PPARγ 및 C/EBPα와 같은 지방생성 주요 조절인자의 발현을 조절함으로써 3T3-L1 지방전구세포의 지방형성과정 (adipogenesis) 을 억제하였다. 반면에, GL은 3T3-L1 세포의 지방형성과정에 미미한 영향을 미쳤으며, 또한 대조군에 비해 PPARγ의 발현을 확실하게 증가시켰다. 이러한 결과는 생체 내에서의 GL의 항비만 효과와는 일치하지 않다. 그러므로, GL의 항비만 효과는 지방조직이 아닌 다른 조직에서 분비된 특정 인자로 인한 혹은 생체 에너지 소비율 증가 유도를 통해 도출된 결과라고

사료된다. 본 연구에서는, 고려인삼 잎 추출물의 항비만 효과를 처음으로 검증하였으며, 이를 활용한 비만 치료제 및 기능성식품 개발 가능성을 제공한다.