Two New Phenolic Compounds from *Artemisia iwayomogi*

by **Xi-Tao Yan**[a]), **Yan Ding**[a])[b]), **Wei Li**[a]), **Ya-Nan Sun**[a]), **Seo-Young Yang**[a]), **Young-Sang Koh**[c]), and **Young-Ho Kim***[a])

[a]) College of Pharmacy, Chungnam National University, Daejeon 305-764, Korea (phone: + 82-42-821-5933; fax: +82-42-823-6566; e-mail: yhk@cnu.ac.kr)
[b]) School of Food Science and Technology, Dalian Polytechnic University, Dalian 116-034, P. R. China
[c]) School of Medicine, Jeju National University, Jeju 690-756, Korea

Two new phenolic compounds, (*Z*)-5′-hydroxyjasmone 5′-*O*-{6″-*O*-[(*E*)-caffeoyl]-*β*-D-glucopyranoside} (**1**) and quercetin-7-*O*-*β*-D-glucuronide methyl ester (**2**), along with ten known phenolic compounds, **3**–**12**, were isolated from the aerial parts of *Artemisia iwayomogi*. Their structures were elucidated by spectroscopic methods, including 1D- and 2D-NMR, and HR-ESI-TOF-MS techniques. The inhibitory effects of compounds **1**–**12** on the LPS-stimulated production of IL-12 p40, IL-6, and TNF-*α* in bone marrow-derived dendritic cells were evaluated.

Introduction. – Since the discovery of artemisinin in the leaves of *Artemisia annua* in the early 1970s, plants belonging to the *Artemisia* genus have attracted considerable attention for their chemical constituents. *Artemisia iwayomogi* (Compositae), a member of the *Artemisia* genus, is a perennial herb widely distributed in Northeast Asian, especially Korea. The aerial parts of *A. iwayomogi* have long been used in traditional Korean medicine (called '*Han In Jin*') to cure various infectious diseases such as carbuncle, sores, cholecystitis, and hepatitis, and to treat fever, inflammation, and jaundice [1][2]. Previous phytochemical investigations on this plant led to the isolation of phenolic compounds, terpenes, and coumarins as major constituents [3–6].

In the course of our ongoing search for novel anti-inflammatory compounds from medicinal plants, twelve compounds were isolated from a MeOH extract of the aerial parts of *A. iwayomogi*, including two new phenolic compounds, (*Z*)-5′-hydroxyjasmone 5′-*O*-{6″-*O*-[(*E*)-caffeoyl]-*β*-D-glucopyranoside} (**1**) and quercetin-7-*O*-*β*-D-glucuronide methyl ester (**2**), and ten known phenolic compounds: patuletin 3-*O*-*β*-D-glucopyranoside (**3**) [7], quercetin (**4**) [8], citrusin C (**5**) [9], myrciaphenone A (**6**) [10], annphenone (**7**) [11], *erythro*-1,2-bis(4-hydroxy-3-methoxyphenyl)propane-1,3-diol (**8**) [12][13], *threo*-1,2-bis(4-hydroxy-3-methoxyphenyl)propane-1,3-diol (**9**) [13], protocatechuic aldehyde (**10**) [14], chlorogenic acid methyl ester (**11**) [15], and 3,4-di(*O*-caffeoyl)isoquinic acid (**12**) [16] (*Fig. 1*). Among them, compounds **3** and **8**–**12** were isolated from this plant for the first time. Herein, we describe the isolation and structure elucidation of the two new phenolic compounds, as well as the anti-inflammatory activities of the isolates in LPS-stimulated bone marrow-derived dendritic cells.

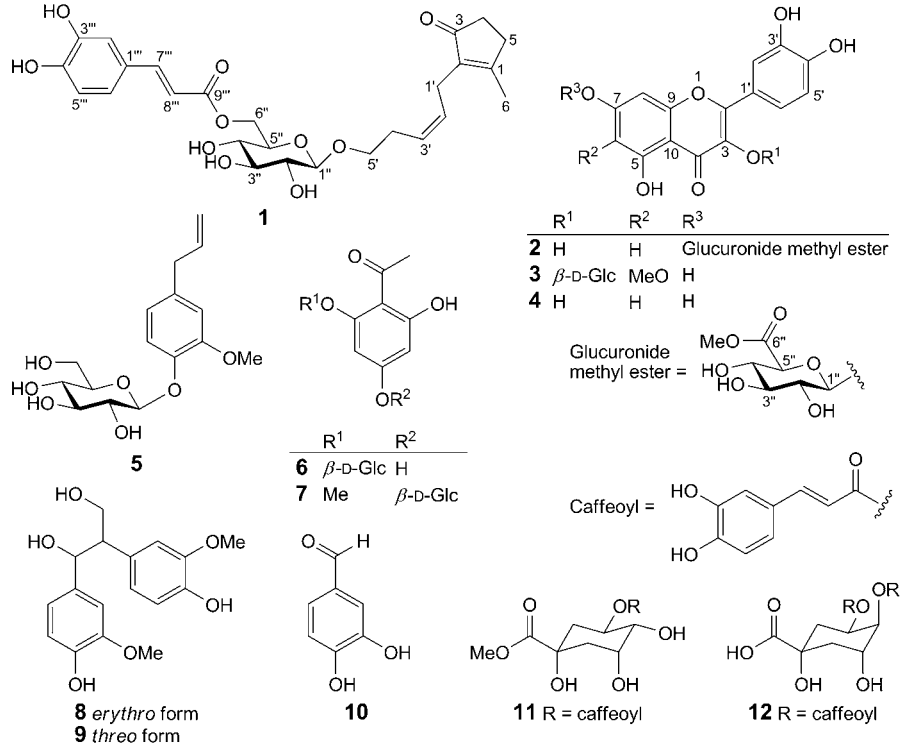

Fig. 1. *The structures of compounds* **1**–**12**

Results and Discussion. – Compound **1** was obtained as a white amorphous powder. The molecular formula was determined as $C_{26}H_{32}O_{10}$ by high-resolution electrospray-ionization time-of-flight mass spectrometry (HR-ESI-TOF-MS) (*m*/*z* 503.1923 ([*M* – H]$^-$)). The IR spectrum showed strong absorption bands for OH (3365 cm^{-1}) and conjugated CO groups (1684 cm^{-1}), and conjugated olefinic bonds (1634 cm^{-1}). The ^{1}H-NMR spectrum of **1** (*Table*) exhibited downfield signals of three aromatic H-atoms at δ(H) 6.99 (*d*, *J* = 2.1, H–C(2‴)), 6.89 (*dd*, *J* = 8.3, 2.1, H–C(6‴)), and 6.74 (*d*, *J* = 8.3, H–C(5‴)), revealing one typical *ABX* coupling system, and of four olefinic H-atoms at δ(H) 7.52 (*d*, *J* = 15.8, H–C(7‴)), 6.24 (*d*, *J* = 15.8, H–C(8‴)), 5.38 (*dtt*, *J* = 10.9, 7.6, 1.4, H–C(3′)), and 5.27 (*dtt*, *J* = 10.9, 6.9, 1.4, H–C(2′)), suggesting the presence of both (*E*)- and (*Z*)-form C=C bonds. Moreover, the ^{1}H-NMR spectrum of **1** showed signals corresponding to five O-bearing CH groups at δ(H) 4.31 (*d*, *J* = 8.3, H–C(1″)) and 3.19–3.54 (*m*, H–C(2″,3″,4″,5″)), two pairs of O-bearing CH_2 groups at δ(H) 4.48 (*dd*, *J* = 11.6, 2.1, H_a–C(6″)) and 4.30 (*dd*, *J* = 11.6, 6.2, H_b–C(6″)), and 3.78 (*dt*, *J* = 9.6, 7.6, H_a–C(5′)) and 3.61 (*dt*, *J* = 9.6, 6.9, H_b–C(5′)), which were supported by DEPT-135 and HMQC data, four CH_2 groups in the range of δ(H) 2.91–2.24 (*m*), and one Me group at δ(H) 2.01 (*s*, Me(6)). Analyses of ^{13}C-NMR, DEPT, and HMQC data revealed that **1** contains 26 C-atoms comprising one Me, six CH_2, and twelve CH groups, and seven quaternary C-atoms (*Table*). These spectral data implied the presence of one caffeoyl,

Table. *[1]H- and [13]C-NMR Data* (600 and 150 MHz, resp.) *of* **1** (CD_3OD) *and* **2** ((D_6)DMSO). δ in ppm, J in Hz. Atom numbering as indicated in *Fig. 1*.

Position	**1**		Position	**2**	
	δ(H)	δ(C)		δ(H)	δ(C)
1		174.8	2		147.7
2		139.9	3		136.1
3		212.2	4		176.0
4	2.24–2.26 (*m*)	35.3	5		160.5
5	2.42–2.44 (*m*)	32.7	6	6.44 (*d*, *J* = 1.8)	98.6
6	2.01 (*s*)	17.6	7		162.2
1′	2.88–2.91 (*m*)	22.3	8	6.81 (*d*, *J* = 1.8)	94.0
2′	5.27 (*dtt*, *J* = 10.9, 6.9, 1.4)	129.0	9		155.8
3′	5.38 (*dtt*, *J* = 10.9, 7.6, 1.4)	127.5	10		104.8
4′	2.46–2.49 (*m*)	29.4	1′		121.8
5′	3.61 (*dt*, *J* = 9.6, 6.9), 3.78 (*dt*, *J* = 9.6, 7.6)	70.8	2′	7.72 (*d*, *J* = 1.8)	115.4
1″	4.31 (*d*, *J* = 8.3)	104.8	3′		145.1
2″	3.19 (*dd*, *J* = 8.3, 7.6)	75.2	4′		148.0
3″	3.34–3.39 (*m*)	78.1	5′	6.90 (*d*, *J* = 8.2)	115.6
4″	3.31–3.36 (*m*)	72.0	6′	7.56 (*dd*, *J* = 8.2, 1.8)	120.1
5″	3.51–3.54 (*m*)	75.5	1″	5.33 (*d*, *J* = 7.3)	99.0
6″	4.30 (*dd*, *J* = 11.6, 6.2), 4.48 (*dd*, *J* = 11.6, 2.1)	64.9	2″	3.28–3.35 (*m*)	72.8
1‴		127.8	3″	3.29–3.37 (*m*)	75.4
2‴	6.99 (*d*, *J* = 2.1)	115.2	4″	3.35–3.42 (*m*)	71.3
3‴		147.0	5″	4.21 (*d*, *J* = 10.1)	75.2
4‴		149.8	6″		169.3
5‴	6.74 (*d*, *J* = 8.3)	116.6	MeO	3.67 (*s*)	52.0
6‴	6.89 (*dd*, *J* = 8.3, 2.1)	123.2			
7‴	7.52 (*d*, *J* = 15.8)	147.2			
8‴	6.24 (*d*, *J* = 15.8)	115.0			
9‴		169.2			

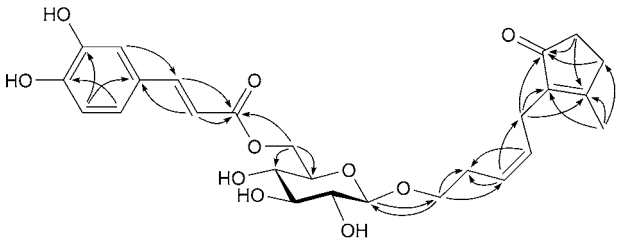

Fig. 2. *Key HMBCs* (H → C) *of* **1**

one 5′-hydroxyjasmone, and one glucosyl moiety in the molecule of **1**, which was confirmed by HMBCs and comparison of the NMR data with those reported in [17][18]. The C=C bond in the caffeoyl moiety was determined to be (*E*)-configured based on its coupling constant (J = 15.8 Hz) between the H–C(7‴) and H–C(8‴), while the C=C bond in the 5′-hydroxyjasmone moiety was determined to be (*Z*)-configured due to the smaller coupling constant (J = 10.9 Hz) between the H–C(2′) and

H–C(3′). Correlations in the HMBC spectrum (*Fig. 2*) observed from the signal at δ(H) 4.31(H–C(1″)) to that at δ(C) 70.8 (CH_2(5′)), and from the signals at δ(H) 3.61 and 3.78 (CH_2(5′)) to that at δ(C) 104.8 (CH(1″)), indicated that the O–CH_2(5′) group of the (*Z*)-5′-hydroxyjasmone moiety was connected to the anomeric C-atom (H–C(1″)) of the glucosyl moiety. Correlations from the signals at δ(H) 4.30 and 4.48 (CH_2(6″)) to that at δ(C) 169.2 (C(9‴)) suggested that the caffeoyl moiety was linked to CH_2(6″) of the glucosyl moiety. The β-configuration at the anomeric center of the glucosyl moiety was supported by the relatively large *J* value ($J = 8.3$ Hz). The absolute D-configuration of the glucosyl moiety was determined by GC analysis. Consequently, the structure of **1** was elucidated as (*Z*)-5′-hydroxyjasmone 5′-*O*-{6″-*O*-[(*E*)-caffeoyl]-β-D-glucopyranoside}.

Compound **2** was obtained as a yellow amorphous powder. Its molecular formula was determined as $C_{22}H_{20}O_{13}$ on the basis of HR-ESI-TOF-MS data (*m*/*z* 491.0828 ([*M* – H]$^-$)). The IR spectrum showed absorption bands for OH groups (3308 cm^{-1}), an ester CO group (1740 cm^{-1}), an α,β-unsaturated CO group (1654 cm^{-1}), and a conjugated olefinic bond (1612 cm^{-1}). The ^{1}H-NMR spectrum of **2** (*Table*) exhibited signals for three *ABX* coupling aromatic H-atoms at δ(H) 7.72 (*d*, $J = 1.8$, H–C(2′)), 7.56 (*dd*, $J = 8.2$, 1.8, H–C(6′)), and 6.90 (*d*, $J = 8.2$, H–C(5′)), two *meta*-coupling aromatic H-atoms at δ(H) 6.81 (*d*, $J = 1.8$, H–C(8)) and 6.44 (*d*, $J = 1.8$, H–C(6)), for five H-atoms in the range of δ(H) 3.28 – 5.33, and one MeO group at δ(H) 3.67 (*s*). The ^{13}C-NMR spectrum of **2**, combined with the HMQC spectrum, showed 22 C-atom signals, including those of one Me group and ten CH groups, and eleven quaternary C-atoms. The quaternary C-atom signals at δ(C) 176.0, 147.7, 136.1, and 104.8 were typical of C(4), C(2), C(3), and C(10) of a 3-*O*-substituted flavonoid moiety. Additional signals of five *O*-bearing aromatic C-atoms at δ(C) 162.2 (C(7)), 160.5 (C(5)), 155.8 (C(9)), 148.0 (C(4′)), and 145.1 (C(3′)) suggested that this 3-*O*-substituted flavonoid moiety corresponded to a quercetin (2-(3′,4′-dihydroxyphenyl)-3,5,7-trihydroxy-4*H*-chromen-4-one) group [8]. Based on comparison of the NMR spectral data with those reported in literature [19][20], the signals of seven *O*-bearing C-atoms at δ(C) 99.0 (C(1″)), 72.8 (C(2″)), 75.4 (C(3″)), 71.3 (C(4″)), 75.2 (C(5″)), 169.3 (C(6″)), and 52.0 (Me(7″)) were consistent with the β-D-glucuronide methyl ester group. The larger coupling constant of the anomeric H-atom signal at δ(H) 5.33 (*d*, $J = 7.3$) indicated the β-configuration of the glucuronide methyl ester group. This group was also evidenced by the HMBCs (*Fig. 3*), especially the key HMBCs H–C(5″)/C(6″) and Me(7″)/C(6″). The HMBC from the signal at δ(H) 5.33 (*d*, $J = 7.3$, H–C(1″)) to that at δ(C) 162.2 (C(7)) indicated that the β-D-glucuronide methyl ester group was linked to the quercetin group through C(1″)–O–C(7). Thus, the structure of **2** was determined as quercetin-7-*O*-β-D-glucuronide methyl ester.

Inhibitory effects of the isolated compounds, **1** – **12**, on proinflammatory cytokines were evaluated by evaluating the production of IL-12 p40, IL-6, and TNF-α in LPS-stimulated bone marrow-derived dendritic cells. *SB203580*, an inhibitor of cytokine suppressive binding protein/p38 kinase, was used as a positive control, which inhibited IL-12 p40, IL-6, and TNF-α production with IC_{50} values of 5.0 ± 0.2, 3.5 ± 0.1, and 7.2 ± 0.3 μM, respectively. Among the compounds, a mixture of compounds **8** and **9** (a pair of diastereoisomers) and compound **4** exhibited potent inhibitory activities against IL-12 p40 production with IC_{50} values of 0.03 ± 0.001 and 5.1 ± 0.2 μM, respectively.

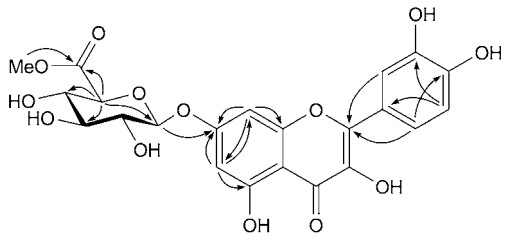

Fig. 3. *Key HMBCs* (H → C) *of* **2**

Moreover, compound **7** showed moderate inhibitory activity against IL-12 p40 with an IC_{50} value of 21.6 ± 1.5 μM. However, only compound **4** inhibited IL-6 and TNF-α production with IC_{50} values of 52.93 ± 2.2 μM and 50.11 ± 2.1 μM, respectively. The remaining compounds did not show any significant activity against the production of IL-12 p40, IL-6, and TNF-α in LPS-stimulated bone marrow-derived dendritic cells ($IC_{50} > 60$ μM).

This work was financially supported by the *Technology Development Program for Agriculture and Forestry* (No. 108079-3), the *Ministry for Agriculture, Forestry and Fisheries*, Korea, and the *Priority Research Centers Program* through the *National Research Foundation of Korea* (NRF) funded by the *Ministry of Education, Science and Technology* (2009-0093815), Korea.

Experimental Part

General. Column chromatography (CC): silica gel (SiO_2; 40–75 and 75–200 μm particle size; *Fuji Silysia Chemical Ltd.*, Japan) and *YMC RP-18* resins (30–50 μm particle size; *Fuji Silysia Chemical Ltd.*, Japan). TLC: Silica gel *60* F_{254} and *RP-18* F_{254S} plates (*Merck*, DE-Darmstadt). GC: *Shimadzu GC-2010* spectrometer. Optical rotations: *Jasco P-2000* digital polarimeter. UV Spectra: *Shimadzu UV-1800* spectrophotometer; λ_{max} (log ε) in nm. IR Spectra: *Nicolet 380 FT-IR* spectrometer; KBr pellets; $\tilde{\nu}$ in cm^{-1}. NMR Spectra: *Jeol ECA 600* spectrometer (^{1}H- and ^{13}C-NMR at 600 and 125 MHz, resp.); δ in ppm rel. to Me_4Si as an internal standard, J in Hz. ESI-MS: *Agilent 1100 LC-MSD trap* spectrometer. HR-ESI-TOF-MS: *Agilent 6530 Accurate-Mass Q-TOF* LC/MS system; in m/z.

Plant Material. The aerial parts of *Artemisia iwayomogi* were collected on Jeju Island in June 2007, and taxonomically identified by *Y.-H. K.* at the College of Pharmacy, Chungnam National University, Daejeon, Korea. A voucher specimen (CNU07105) has been deposited with the herbarium of the above college.

Extraction and Isolation. The dried aerial parts of *Artemisia iwayomogi* (3 kg) were extracted with MeOH under reflux (3×18 l, 12 h each). The combined extract was concentrated under reduced pressure to yield a residue (465 g), which was suspended in H_2O (3 l), and partitioned successively with $CHCl_3$ (4×3 l) and AcOEt (4×3 l) to yield a $CHCl_3$-soluble fraction (94 g), an AcOEt-soluble fraction (42 g), and a H_2O fraction, resp. The AcOEt-soluble fraction was subjected to column chromatography (CC; SiO_2; $CHCl_3$/MeOH 50:1 to 10:1) to afford six subfractions, *Frs. 1–6*. *Fr. 2* (31 g) was further subjected to repeated CC (SiO_2; hexane/acetone 5:1 to 0:1), and then to CC (*YMC* C-18; H_2O/MeOH 2:1 to 1:1) to yield compounds **1** (26 mg), **4** (45 mg), **5** (13 mg), **6** (10 mg), **7** (30 mg), a mixture **8**/**9** (8 mg), **10** (24 mg), and **12** (420 mg). The H_2O fraction was submitted to CC (*Diaion HP-20*; H_2O/MeOH 100:0, 75:25, 50:50, 25:75, 0:100) to yield five fractions, *Frs. A–E*. *Fr. C* (eluted with H_2O/MeOH 1:1, 56 g) was subjected to CC (SiO_2; CH_2Cl_2/MeOH/H_2O 12:1:0.05 to 1:1:0.1) to afford four subfractions, *Frs. C1–C4*. *Fr. C2* was purified by repeated CC (*YMC C-18*; H_2O/MeOH 2.5:1 to 1:1) to

yield compounds **2** (22 mg) and **11** (26 mg). *Fr. C3* was further purified by CC (*YMC C-18*; H_2O/MeOH 1.5:1 to 1:1) to yield compound **3** (10 mg).

(*Z*)-*5′-Hydroxyjasmone 5′*-O-*{6″*-O-*[(*E*)-caffeoyl]-β-D-glucopyranoside}* (=*2-[(2Z)-5-[[6*-O-*[(2*E*)-3-(3,4-Dihydroxyphenyl)-1-oxoprop-2-en-1-yl]-β-D-glucopyranosyl]oxy]pent-2-en-1-yl]-3-methyl-2-cyclopenten-1-one*; **1**). White powder. $[\alpha]_D^{18} = -69.9$ ($c = 1.0$, MeOH). UV (MeOH): 234 (2.76), 330 (1.72). IR (KBr): 3365, 1684, 1634, 1605, 1525, 1440, 1386, 1283, 1164, 1039, 851, 803. ^{1}H- and ^{13}C-NMR: see the *Table*. HR-ESI-MS (neg.): 503.1923 ($[M-H]^-$, $C_{26}H_{31}O_{10}^-$; calc. 503.1917).

Quercetin-7-O-*β*-D-*glucuronide methyl ester* (=*2-(3,4-Dihydroxyphenyl)-3,5-dihydroxy-7-[(6-methyl-β-D-glucopyranuronosyl)oxy]-4*H-*1-benzopyran-4-one*; **2**): Yellow powder. $[\alpha]_D^{18} = -189.1$ ($c = 1.0$, MeOH). UV (MeOH): 256 (1.86), 372 (1.60). IR (KBr): 3308, 1740, 1654, 1612, 1597, 1497, 1317, 1247, 1212, 1173, 1088, 1044, 1024, 1000. ^{1}H- and ^{13}C-NMR: see the *Table*. HR-ESI-MS (neg.): 491.0828 ($[M-H]^-$, $C_{22}H_{19}O_{13}^-$; calc. 491.0826).

Acid Hydrolysis. Compound **1** (2.0 mg) was dissolved in 1N HCl (dioxane/H_2O 1:1, *v*/*v*, 1 ml) and then heated to 80° in a water bath for 3 h. The cooled mixture was diluted with H_2O (4 ml) and extracted with AcOEt (3 × 5 ml). The aq. layer was thoroughly dried under N_2 after neutralization with Ag_2CO_3. The residues were dissolved in 0.1 ml of dry pyridine, and then L-cysteine methyl ester hydrochloride in pyridine (0.06M, 0.1 ml) was added to the solns. The mixture was heated at 60° for 2 h, and 0.1 ml of TMSCl (Me_3SiCl) soln. was added, followed by heating at 60° for 1.5 h. The dried product were partitioned with hexane and H_2O (0.1 ml each), and the org. layer was analyzed by GC (column: *SPB-1*, 0.25 mm × 30 m; detector, FID; detector temp., 300°; column temp., 210°; injector temp., 270°; carrier gas, He, 2 ml/min). The monosaccharide was confirmed as D-glucose by comparison of the retention time of the monosaccharide derivative (t_R 14.09 min) with that of authentic sugar derivatives (D-glucose derivative: t_R 14.11 min and L-glucose derivative: t_R 14.26 min), which were prepared by the same reaction from the standard glucoses.

Biological Assay. Bone marrow-derived dendritic cells (BMDCs) were grown from wild-type *C57BL/6* mice (*Taconic Farm*, NY, USA). Briefly, the mouse tibia and femur were obtained by flushing with *Dulbecco*'s modified *Eagle*'s medium to yield bone marrow cells. The cells were cultured in *RPMI 1640* medium containing 10% heat-inactivated fetal bovine serum (FBS) (*Gibco*, NY, USA), 50 μM 2-sulfanylethanol, and 2 mM glutamine supplemented with a 3% *J558L* hybridoma cell culture supernatant containing granulocyte-macrophage colony-stimulating factor. The culture medium was replaced with fresh medium every second day. At day 6 of culture, nonadherent cells and loosely adherent DC aggregates were harvested, washed, and resuspended in *RPMI 1640* supplemented with 5% FBS. The BMDCs were incubated in 48-well plates at a density of 2×10^5 cells/ml, and then treated with the test compounds in DMSO (2, 10, 25, and 50 μM) for 1 h before stimulation with 10 ng/ml LPS from *Salmonella minnesota* (*Alexis*, NY, USA). Supernatants were harvested 16 h after stimulation. Concentrations of murine IL-12 p40, IL-6, and TNF-α in the culture supernatant fraction were determined by enzyme-linked immune-sorbent assay (*BD Pharmingen*, CA, USA) according to the manufacturer's instructions. The data are presented as mean ± SD of at least three independent experiments performed in triplicate.

REFERENCES

[1] J. K. Kim, 'Illustrated Natural Drugs Encyclopedia', Namsandang Publishers, Seoul, 1989, p. 79.
[2] J. H. Park, 'Korean Folk Medicine', Busan National University Publishers, Busan, 1999, p. 68.
[3] Y. Ding, C. Liang, S. Y. Yang, J. C. Ra, E. M. Choi, J.-A. Kim, Y. H. Kim, *Biol. Pharm. Bull.* **2010**, *33*, 1448.
[4] A. R. Kim, Y. N. Zou, T. H. Park, K. H. Shim, M. S. Kim, N. D. Kim, J. D. Kim, S. J. Bae, J. S. Choi, H. Y. Chung, *Phytother. Res.* **2004**, *18*, 1.
[5] H.-H. Yu, Y.-H. Kim, B.-S. Kil, K.-J. Kim, S.-I. Jeong, Y.-O. You, *Planta Med.* **2003**, *69*, 1159.
[6] H. Greger, C. Zdero, F. Bohlmann, *Phytochemistry* **1986**, *25*, 891.
[7] W. Bylka, *Acta Physiol. Plant.* **2004**, *26*, 393.
[8] H. Wagner, V. M. Chari, J. Sonnenbichler, *Tetrahedron Lett.* **1976**, *17*, 1799.

[9] M.-R. Kim, S.-K. Lee, C.-S. Kim, K.-S. Kim, D.-C. Moon, *Arch. Pharmacal Res.* **2004**, *27*, 1029.
[10] M. Yoshikawa, H. Shimada, N. Nishida, Y. Li, I. Toguchida, J. Yamahara, H. Matsuda, *Chem. Pharm. Bull*. **1998**, *46*, 113.
[11] A. K. Singh, V. Pathak, P. K. Agrawal, *Phytochemistry* **1997**, *44*, 555.
[12] K. Lundquist, *Acta Chem. Scand., B* **1979**, *33*, 418.
[13] S. G. Guan, W. B. Yu, S. H. Guan, *Shizhen Guoyi Guoyao* **2010**, *21*, 905.
[14] E. T. Kolehmainen, K. P. Laihia, J. M. I. Hyötyläinen, R. T. Kauppinen, *Spectrochim. Acta, Part A* **1995**, *51*, 419.
[15] X. Zhu, X. Dong, Y. Wang, P. Ju, S. Luo, *Helv. Chim. Acta* **2005**, *88*, 339.
[16] M. Ono, C. Masuoka, Y. Odake, S. Ikegashira, Y. Ito, T. Nohara, *Food Sci. Technol. Res.* **2000**, *6*, 106.
[17] J. Kitajima, T. Ishikawa, A. Urabe, *Chem. Pharm. Bull.* **2004**, *52*, 1013.
[18] P. Zhao, T. Tanaka, K. Hirabayashi, Y.-J. Zhang, C.-R. Yang, I. Kouno, *Phytochemistry* **2008**, *69*, 3087.
[19] E.-R. Woo, M. S. Piao, *Arch. Pharmacal Res.* **2004**, *27*, 173.
[20] D. P. Allais, A. Simon, B. Bennini, A. J. Chulia, M. Kaouadji, C. Delage, *Phytochemistry* **1991**, *30*, 3099.

Received April 23, 2013