Monoterpenes from *Paeonia albiflora* and Their Inhibitory Activity on Nitric Oxide Production by Lipopolysaccharide-Activated Microglia

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Eleven new monoterpenes, paeonidangenin (**1**), paeonidanin A (**2**), paeonidanin B (**3**), paeonidanin C (**4**), paeonidanin D (**5**), paeonidanin E (**6**), paeoniflorone (**7**), 4-*O*-methylbenzoylpaeoniflorin (**8**), 4-*O*-methylgalloylpaeoniflorin (**9**), 4-*O*-methyldebenzoylpaeoniflorin (**10**), and 4-*O*-methylalbiflorin (**11**), were isolated from the 60% ethanol extract of the roots of *Paeonia albiflora*. Their structures were determined primarily on the basis of 1D and 2D NMR techniques and MS studies. Paeonidanins D (**5**) and E (**6**) are unprecedented examples of "cage-like" monoterpene dimers. The inhibitory effects of the isolated compounds on nitric oxide production by lipopolysaccharide (LPS)-activated N9 microglia were evaluated.

The genus *Paeonia* consists of 35 species, distributed mainly in temperate areas of Eurasia. The roots of *Paeonia albiflora* Pall. (Chishao) are one of the most important crude drugs in Chinese traditional medicine. It has a variety of activities, such as inhibiting platelet aggregation[1] and antihepatic fibrosis[2] and anti-inflammatory[3] activities, and the ability to improve learning and spatial resolution and delay senility.[4] Paeonia species are rich in "cage-like" monoterpenes, which are the major active substances of *P. albiflora*, and a number of monoterpenes, such as paeoniflorin, oxypaeoniflorin, and benzoylpaeoniflorin, have been isolated from the plant.[5] In the course of searching for biologically active substances from *P. albifloria*, 25 monoterpenes were isolated and identified. Eleven of these were identified as new compounds and were named paeonidangenin (**1**), paeonidanin A (**2**), paeonidanin B (**3**), paeonidanin C (**4**), paeonidanin D (**5**), paeonidanin E (**6**), paeoniflorone (**7**), 4-*O*-methylbenzoylpaeoniflorin (**8**), 4-*O*-methylgalloylpaeoniflorin (**9**), 4-*O*-methyldebenzoylpaeoniflorin (**10**), and 4-*O*-methylalbiflorin (**11**). The other 14 known compounds were paeoniflorin (**12**),[6,7] benzoylpaeoniflorin (**13**),[6] galloylpaeoniflorin (**14**),[8,9] mudanpioside C (**15**),[10] 6′-*O*-vanillylpaeoniflorin (**16**),[5] 6′-*O*-α-glucopyranosylpaeoniflorin (**17**),[5] oxypaeoniflorin (**18**),[10] benzoyloxypaeoniflorin (**19**),[11] mudanpioside E (**20**),[10] lactiflorin (**21**),[12] paeonivayin (**22**),[13] paeoniflorigenone (**23**),[14] 4-*O*-methylpaeoniflorin (**24**),[6] and paeonidanin (**25**).[15] Their structures were elucidated using spectroscopic data, mainly 1D NMR, 2D NMR, and mass spectra. All of these compounds were screened for their inhibitory activities on nitric oxide production by lipopolysaccharide (LPS)-activated N9 microglia.

Results and Discussion

Compound **1** was obtained as a colorless oil. The molecular formula of **1** was determined as $C_{18}H_{20}O_6$ by HRESIMS at *m/z* 355.1156 $[M + Na]^+$ (calcd 355.1158). The ^1H NMR spectrum of **1** displayed a methyl group at δ_H 1.41 (3H, s), a methoxy group at δ_H 3.38 (3H, s), and an acetal proton at δ_H 5.10 (1H, s). The ^{13}C NMR spectrum of **1** displayed a benzoyl moiety, an acetal carbon at δ_C 104.7, and a carbonyl carbon at δ_C 205.6. The ^{13}C NMR spectrum of **1** was similar to that of paeonidanin,[15] except for the absence of a glucose moiety, indicating that **1** was the aglycone of paeonidanin. This deduction was further supported by HSQC and HMBC spectra. In the HMBC spectrum, correlations of H-3 to C-2, C-4, and C-5, H-5 to C-3, C-6, C-8, and C-9, H-7 to C-2, C-4, and C-6, H-9 to C-2, C-5, and C-8, and H-10 to C-1, C-2, and C-3 could be observed. The HMBC correlations from H-8 to C-7′ and from the methoxy protons to C-9 confirmed that the benzoyl moiety was connected to C-8 and the methoxy group was connected to C-9. The relative configuration of **1** was determined by the NOE correlations between H-8 and H-9 and between the methoxy protons and H-3. From these spectroscopic data, compound **1** was deduced to be the aglycone of paeonidanin, and it was named paeonidangenin.

The molecular formula of compound **2**, $C_{31}H_{34}O_{12}$, was established by HRESIMS (*m/z* 621.1947 $[M + Na]^+$). The ^1H NMR spectrum of **2** displayed a methyl group at δ_H1.33 (3H, s), a methoxy group at δ_H 3.26 (3H, s), an acetal proton at δ_H 5.08 (1H, s), and an anomeric proton at δ_H 4.63 (1H, d, $J = 7.8$ Hz). The ^1H and ^{13}C NMR data as well as acid hydrolysis and GC comparison with an authentic sample indicated the presence of a D-glucopyranosyl moiety. The β-configuration of the glycosidic linkage was determined from the coupling constant of the anomeric proton. The ^{13}C NMR spectrum displayed 31 carbons including two benzoate moieties, an acetal carbon at δ_C 107.5, and a carbonyl carbon at δ_C 208.9. The ^{13}C NMR spectrum was similar to that of paeonidanin,[15] except for the presence of a benzoyl moiety, indicating that they had the same molecular skeleton. The chemical shift of C-6′ was downfield to δ_C 65.0, suggesting that the benzoyl moiety was attached to C-6′. This deduction was further supported by HSQC and HMBC spectra. The HMBC correlations from H-3 to C-1, C-4, and C-5, from H-5 to C-1, C-4, C-8, and C-9, from H-7 to C-2, C-4, and C-6, from H-9 to C-2, C-4, and C-6, and from H-10 to C-1, C-2, and C-3 were observed. The HMBC correlations from H-1′ to C-1, from H-8 to C-7″, from H-6′ to C-7‴, and from the methoxy protons to C-9 confirmed that the sugar was connected to C-1, the benzoyl moiety (carbonyl carbon at δ_C 167.8) was connected to C-8, another benzoyl moiety (carbonyl carbon at δ_C 167.6) was connected to C-6′, and the methoxy group was connected to C-9. The relative configuration of **2** was determined by the NOE correlations between H-9 and H-8 and between the methoxy protons and H-3. On the basis of the ^1H, ^{13}C, and 2D NMR (HSQC, HMBC, NOESY) data, the structure of **2** was unambiguously established as 6′-*O*-benzoylpaeonidanin, and it was named paeonidanin A.

The HRESIMS of compound **3** showed a quasimolecular ion at *m/z* 669.1795, corresponding to the molecular formula $[C_{31}H_{34}O_{15}Na]^+$. The ^{13}C NMR spectrum was similar to that of **2** except for the presence of a galloyl moiety in **3** instead of a benzoyl

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moiety in **2**. The HMBC correlation from H-6′ to C-7‴ confirmed that the galloyl moiety was connected to C-6′. On the basis of the ^{1}H, ^{13}C, and 2D NMR (HSQC, HMBC, NOESY) data, the structure of **3** was established as 6′-*O*-galloylpaeonidanin, and it was named paeonidanin B.

The molecular formula of compound **4**, $C_{31}H_{34}O_{13}$, was established by HRESIMS (*m*/*z* 637.1895 [M + Na]$^+$). The ^{13}C NMR spectrum was also similar to that of **2** except for the presence of a *p*-hydroxybenzoyl moiety in **4** instead of a benzoyl moiety in **2**. The HMBC cross-peak from H-8 to C-7″ confirmed that the *p*-hydroxybenzoyl moiety was connected to C-8. On the basis of the ^{1}H, ^{13}C, and 2D NMR (HSQC, HMBC, NOESY) data, the structure of **4** was identified as 6′-*O*- benzoyl-4″- hydroxypaeonidanin, and it was named paeonidanin C.

The HRESIMS of compound **5** showed a quasimolecular ion peak at *m*/*z* 979.3214 [M + Na]$^+$, corresponding to the molecular formula $C_{47}H_{56}O_{21}$. The ^{1}H NMR spectrum displayed two methyl groups at δ_H 1.36 (3H, s) and 1.42 (3H, s), a methoxy group at δ_H 3.27 (3H, s), two acetal protons at δ_H 5.05 (1H, s) and 5.34 (1H, s), and two anomeric protons at δ_H 4.11 (1H, d, J = 7.2 Hz) and 4.58 (1H, d, J = 7.2 Hz). The ^{13}C NMR spectrum displayed 47 carbons including two benzoyl moieties and two carbonyl carbons at δ_C 208.2 and 210.1. The sugar unit was identified as β-D-glucopyranose as in **2**. The ^{13}C NMR spectrum suggested that compound **5** was a dimer comprising two paeonidanin units. This deduction was further supported by HSQC and HMBC spectra. In the HMBC spectrum, the methoxy protons had a correlation with C-9‴, which confirmed that the methoxy group was connected to C-9‴. The HMBC correlation from H-9 to C-6⁗ confirmed that C-9 was connected to C-6⁗ through an oxygen atom. The relative configuration of **5** was established by the NOE correlations observed between H-9 and H-8, between H-9‴ and H-8‴, between the methoxy protons and H-3‴, and between H-6⁗ and H-3. On the basis of the ^{1}H, ^{13}C, and 2D NMR (HSQC, HMBC, NOESY) data, the structure of **5** was unambiguously identified as a dimer comprising two paeonidanin units, and it was named paeonidanin D.

The molecular weight of compound **6** was deduced from the HRESIMS, which displayed an [M + Na]$^+$ ion at *m*/*z* 965.3058 corresponding to an elemental composition $C_{46}H_{54}O_{21}Na$. The ^{1}H NMR spectrum displayed two methyl groups at δ_H 1.29 (3H, s) and 1.43 (3H, s), two acetal protons at δ_H 5.33 (1H, s) and 5.39 (1H, s), and two anomeric protons at δ_H 4.21 (1H, d, J = 7.5 Hz) and 4.58 (1H, d, J = 7.5 Hz). The ^{13}C NMR spectrum displayed 46 carbons including two benzoyl moieties, a carbonyl carbon at δ_C 208.4, and three acetal carbons at δ_C 102.3, 106.5, and 107.4. The sugar unit was identified as β-D-glucopyranose as in **2**. The ^{13}C NMR data suggested that compound **6** was a dimer comprising paeonidanin and paeoniflorin structural moieties. This deduction was further supported by HSQC and HMBC spectra. The HMBC correlations from H-9 to C-6⁗ confirmed that C-6⁗ of paeoniflorin was connected to C-9 of paeonidanin through an oxygen atom. The relative configuration of C-9 in **6** was established by the NOE correlation between H-9 and H-8. On the basis of the ^{1}H, ^{13}C, and 2D NMR (HSQC, HMBC, NOESY) data, the structure of **6** was unambiguously identified as a dimer comprising paeonidanin and paeoniflorin structural moieties, and it was named paeonidanin E.

HRESIMS data (*m*/*z* 359.1108 [M + Na]$^+$) of compound **7** established the molecular formula as $C_{17}H_{20}O_7$. The ^{1}H NMR spectrum displayed two methyl groups at δ_H 1.16 (3H, d, J = 6.5 Hz) and 1.34 (3H, s). The ^{13}C NMR spectrum displayed 17 carbons including a galloyl moiety and a carbonyl carbon at δ_C 216.6. The ^{13}C NMR spectrum of **7** was similar to that of mudanpioside-F,[14] except for the absence of a glucose moiety and two olefinic carbons and the presence of a galloyl group, which indicated that they had the same molecular skeleton. This deduction was further supported by HSQC and HMBC spectra. The HMBC correlations from H-2 to C-1, C-3, and C-7, from H-3 to C-1, C-2, C-4, and C-10, from H-7 to C-2, C-4, and C-6, from H-8 to C-1, C-6, and C-9, and from H-10 to C-1, C-2, and C-3 were observed. The HMBC correlation from H-8 to C-7′ confirmed that the galloyl moiety was connected to C-8. The NOE correlations between H-8 and both H-3β and H-2, between H-9 and H-7β, and between H-10 and H-3α established the relative configuration of compound **7**. On the basis of the ^{1}H, ^{13}C, and 2D NMR (HSQC, HMBC, NOESY) data, the structure of compound **7** was established and named paeoniflorone.

The ESIMS of **8** showed a quasimolecular ion [M + Na]$^+$ at *m*/*z* 621. Taking into account the ^{1}H and ^{13}C NMR spectra, the molecular formula was established as $C_{31}H_{34}O_{12}$. The ^{1}H NMR spectrum displayed a methyl group at δ_H 1.26 (3H, s), a methoxy group at δ_H 3.29 (3H, s), an acetal proton at δ_H 5.42 (1H, s), and an anomeric proton at δ_H 4.57 (1H, d, J = 7.5 Hz). The sugar unit was identified as β-D-glucopyranose as in **2**. The ^{13}C NMR spectrum displayed 31 carbons including two benzoyl moieties, and two acetal carbons at δ_C 102.2 and 109.2. The ^{13}C NMR spectrum was similar to that of benzoylpaeoniflorin,[6] except for the presence of a methoxy group. The HMBC correlations from the methoxy protons to C-4 confirmed that the methoxy group was connected to C-4. From these spectroscopic data, compound **8** was identified as 4-*O*-methylbenzoylpaeoniflorin.

The ESIMS of **9** showed a quasimolecular ion [M + Na]$^+$ at *m*/*z* 669. Taking into account the ^{1}H and ^{13}C NMR spectroscopic data, the molecular formula was established as $C_{31}H_{34}O_{15}$. The ^{13}C NMR spectrum was similar to that of galloylpaeoniflorin,[9] except for the appearance of a methoxy group in **9**. The HMBC correlations from the methoxy protons to C-4 confirmed that the methoxy group was connected to C-4. Thus, compound **9** was identified as 4-*O*-methylgalloylpaeoniflorin.

The ESIMS of compound **10** showed a quasimolecular ion [M + Na]$^+$ at *m*/*z* 413, which indicated a molecular formula of $C_{17}H_{26}O_{10}$, in combination with the ^{1}H and ^{13}C NMR spectroscopic data. The ^{13}C NMR spectrum was similar to that of debenzoylpaeoniflorin,[16] except for the presence of a methoxy group. The HMBC correlations from the methoxy protons to C-4 confirmed that the methoxy group was connected to C-4. Therefore, compound **10** was identified as 4-*O*-methyldebenzoylpaeoniflorin.

The ESIMS of compound **11** showed a quasimolecular ion [M + Na]$^+$ at *m*/*z* 517, which indicated a molecular formula of $C_{24}H_{30}O_{11}$, in combination with the ^{1}H and ^{13}C NMR spectroscopic data. The ^{1}H NMR spectrum displayed a methyl group at δ_H 1.53 (3H, s), a methoxy group at δ_H 3.36 (3H, s), and an anomeric proton at δ_H 4.54 (1H, d, J = 7.2 Hz). The sugar unit was identified as β-D-glucopyrannose as in **2**. The ^{13}C NMR spectrum displayed 24 carbons including a benzoate moiety and a carbonyl carbon at δ_C 178.0. The ^{13}C NMR spectrum of **11** was similar to that of albiflorin,[17] except for the presence of a methoxy group. The HMBC correlation from the methoxy protons to C-4 confirmed that the methoxy group was connected to C-4. From these spectroscopic data, compound **11** was identified as 4-*O*-methylalbiflorin.

We evaluated the inhibitory activities of compounds **1**−**25** on NO production in LPS-activated N9 microglia. The results showed that most of the isolated compounds significantly suppressed NO production. As shown in Table 6, compounds **2**, **3**, **8**, **12**, and **15** displayed strong activities similar to the positive control, curcumin. However, compounds **1**, **4**, **6**, **7**, **18**, **20**−**22**, and **25** exhibited only weak activities. In addition, the cell viability in the present experiment was determined by the MTT method in order to discover whether the inhibitory effects on NO production were due to the cytotoxicity of the test compounds. The results showed that compounds **5**, **13**, **14**, and **23** were cytotoxic to N9 microglial cells.

Experimental Section

General Experimental Procedures. Optical rotations were measured on a Perkin-Elmer 241 MC polarimeter. UV spectra were measured with a Perkin-Elmer 241 polarimeter, and NMR experiments were

Table 1. ^{1}H NMR Data of **1−4**

position	**1** (*J* in Hz)[a]	**2** (*J* in Hz)[b]	**3** (*J* in Hz)[b]	**4** (*J* in Hz)[b]
3	2.63 d (15.7)	2.40 d (18.0)	2.38 d (18.0)	2.35 d (18.0)
	2.67 d (15.7)	2.51 d (18.0)	2.57 d (18.0)	2.48 d (18.0)
5	2.85 d (7.2)	2.90 m	2.89 m	2.84 m
7	2.00 d (10.8)	1.89 d (10.2,)	1.91 d (9.6)	1.82 d (10.4)
	2.38 dd (10.8, 7.2)	2.92 m	2.91 m	2.86 m
8	4.61 d (12.3)	4.70 d (11.7)	4.67 d (11.7)	4.60 d (11.9)
	4.71 d (12.3)	4.81 d (11.7)	4.78 d (11.7)	4.71 d (11.9)
9	5.10 s	5.08 s	5.05 s	5.00 s
10	1.41 s	1.33 s	1.31 s	1.29 s
1′		4.63 d (7.8)	4.57 d (7.5)	4.58 d (7.8)
2′	8.03 d (7.5)	3.33 dd (9.6, 7.8)	3.27 m	3.30 m
3′	7.46t (7.5)	3.45 t (9.6)	3.38 m	3.40 m
4′	7.61 t (7.5)	3.40 t (9.6)	3.35 m	3.35 m
5′	7.46 t (7.5)	3.66 m	3.55 m	3.62 m
6′	8.03 d (7.5)	4.56 dd (11.7, 7.0)	4.48 dd (11.7, 7.0)	4.51 dd (11.7, 7.0)
		4.68 dd (11.7,2.5)	4.51 dd (11.7, 2.5)	4.63 dd (11.7,2.3)
2″,6″		8.02 d (7.5)	8.03 d (7.5)	7.82 d (8.3)
3″,5″		7.47 t (7.5)	7.47 t (7.5)	6.82 d (8.3)
4″		7.60 t (7.5)	7.60 t (7.5)	
2‴,6‴		8.02 d (7.5)	7.05 s	8.02 d (7.2)
3‴,5‴		7.47 t (7.5)		7.44 t (7.2)
4‴		7.60 t (7.5)		7.56 t (7.5)
OMe	3.38 s	3.26 s	3.26 s	3.23 s

[a] $CDCl_3$ was used as solvent. [b] Methanol-d_4 was used as solvent. The data of **1−3** were obtained at 600 MHz, while the data of **4** were obtained at 300 MHz.

Table 2. ^{1}H NMR Data of **5** and **6** in Methanol-d_4[a]

5 (*J* in Hz)				**6** (*J* in Hz)			
position		position		position		position	
3	2.50 d (18.2)	3‴	2.42 d (18.2)	3	2.59 d (18.3)	3‴	1.75 d (12.3)
	2.96 m		3.16 m		2.86 d (18.3)		2.54 d (12.3)
5	2.86 d (4.2)	5‴	2.73 d (4.2)	5	2.93 m	5‴	2.43 m
7	2.16 m	7‴	2.14 m	7	2.20 m	7‴	1.98 m
8	4.83 s	8‴	4.34 d (12.1)	8	4.81 s	8‴	4.58 d (12.0)
			4.58 d (12.1)				4.63 d (12.0)
9	5.34 s	9‴	5.05 s	9	5.33 s	9‴	5.39 s
10	1.42 s	10‴	1.36 s	10	1.43 s	10‴	1.29 s
1′	4.58 d (7.2)	1⁗	4.11 d (7.2)	1′	4.58 d (7.5)	1⁗	4.21 d (7.5)
2′	3.17 m	2⁗	3.21 m	2′	3.14 m	2⁗	3.14 m
3′	3.31 m	3⁗	3.31 m	3′	3.22 m	3⁗	3.24 m
4′	3.15 m	4⁗	3.18 m	4′	3.17 m	4⁗	3.22 m
5′	3.20 m	5⁗	3.22 m	5′	3.16 m	5⁗	3.23 m
6′	3.60 m	6⁗	3.63 m	6′	3.65 m	6⁗	3.70 m
	3.88 d (11.4)		3.77 d (11.1)		3.88 d (11.7)		3.77 d (11.7)
2″,6″	8.03 m	2′′′′′,6′′′′′	8.08 m	2″,6″	8.05 m	2′′′′′,6′′′′′	8.05 m
3″,5″	7.48 m	3′′′′′,5′′′′′	7.52 m	3″,5″	7.49 m	3′′′′′,5′′′′′	7.49 m
4″	7.60 m	4′′′′′	7.63 m	4″	7.62 m	4′′′′′	7.62 m
OMe			3.27 s	OMe			

[a] The data of **5** and **6** were obtained at 300 MHz.

performed on Bruker ARX-300 and -600 spectrometers, using TMS as an internal standard. HRESIMS spectra were obtained on a Bruker APEX-II mass spectrometer. Preparative HPLC was carried out on a Waters 600 chromatograph with an ODS C-18 column (250 mm × 20 mm, 10 μm, Inertsil ODS-3, GL Sciences Inc.) and Waters 490 UV detector. Silica gel (200−300 mesh) used for column chromatography and silica gel GF_{254} (10−40 μm) used for TLC were supplied by Qingdao Marine Chemical Factory, Qingdao, P. R. China. ODS (40−75 μm) used for column chromatography was supplied by Fuji Silysia Chemical, Ltd., Japan. Spots were detected on TLC plates under UV light or by heating after spraying with 5% H_2SO_4 in C_2H_5OH (v/v).

Plant Material. The roots of *P. albiflora* were purchased from Liaoning Yaocai Co., China, and were authenticated by Prof. Qishi Sun, Department of Pharmaceutical Botany, School of Traditional Chinese Materia Medica, Shenyang Pharmaceutical University. This plant originated from the Neimenggu Province of China. A voucher specimen (20050920) was deposited in the hebarium of the Department of Natural Products Chemistry, Shenyang Pharmaceutical University.

Biological Materials. Fetal bovine serum (FBS) and Iscove's modified Dulbecco's medium (IMDM) were purchased from Gibco BRL (Grand Island, NY). LPS (E5:055) and curcumin were purchased from Sigma Chemical Co. (St. Louis, Mo). Thiazoyl blue (MTT) was purchased from Sino-American Biotechnology (Beijing, China). Compounds **1−25** and curcumin were dissolved initially in DMSO and then diluted with PBS (0.01 M, pH = 7.4) for subsequent experiments. DMSO at the highest concentration possibly present under the experimental conditions used (0.1%) was not toxic to cells.

Microglial Cell Culture. The murine microglial cell line N9 was a kind gift from Dr. P. Ricciardi-Castagnoli (Universita Degli Studi di Milano-Bicocca, Milan, Italy). The cells were grown in IMDM supplemented with 5% heat-inactivated FBS, 2 mM L-glutamine, 100U/mL penicillin, 100 Ag/mL streptomycin, and 5 × 10^{-5} M 2-mercaptoethanol.

Nitrite Production Assay. Accumulated nitrite in the culture supernatants, an indicator of NO synthase activity, was measured by the Griess reaction.[18] The N9 microglial cells were cultured for 24 h in 96-well plates (1 × 10^5 cell/well) in IMDM media (100 μL/well) at 37 °C in 5% CO_2. The cells were treated with LPS (1 μL/mL) and the test compounds for 48 h. Curcumin was used as a positive control. Then, 50 μL culture supernatants were mixed with 50 μL of Griess reagent (part I: 1% sulfanilamide; part II: 0.1% naphthylethylene diamide dihydrochloride and 2% phosphoric acid) at room temperature. Fifteen minutes later, the absorbance was determined at 540 nm using the Spectra Shell reader (Tecan, Grödig, Austria). The results were

Table 3. ^{1}H NMR Data of **7−11** in Methanol-d_4[a]

position	**7** (*J* in Hz)	**8** (*J* in Hz)	**9** (*J* in Hz)	**10** (*J* in Hz)	**11** (*J* in Hz)
2	2.79 (m)				
3	1.86 dd (18.3, 8.8)	1.64 d (12.5)	1.68 d (12.0)	1.83 d (10.8)	2.02 d (10.4)
	2.25 dd (18.3, 9.2)	1.81 d (12.5)	1.77 d (12.0)	2.39 d (10.8)	2.82 d (10.4)
4					4.27 d (5.1)
5	2.02 d (6.9)	2.67d (6.5)	2.60 d (6.5)	2.61 d (5.4)	2.93 m
7	1.27 m	1.70 d (12.5)	1.61 d (10.8)	1.90 d (12.3)	2.05 d (15.3)
	1.58 dd (9.2, 4.9)	2.46 dd (12.0, 6.6)	2.40 dd (10.8, 6.5)	2.11 d (12.3)	2.39 d (15.3)
8	4.24 d (11.4)	4.72 s	4.67 s	3.92 d (12.6)	4.69 d (12.3)
	4.34 d (11.4)			4.01 d (12.6)	4.81 d (12.3)
9	1.34 s	5.42 s	5.38 s	5.30 s	
10	1.16 d (6.5)	1.26 s	1.24 s	1.36 s	1.53 s
1′		4.57 d (7.5)	4.53 d (7.5)	4.55 d (7.8)	4.54 d (7.2)
2′	7.06 s	3.24 m	3.26 m	3.20 m	3.24 m
3′		3.37 m	3.36 m	3.35 m	3.25 m
4′		3.35 m	3.35 m	3.26 m	3.26 m
5′		3.59 m	3.51 m	3.27 m	3.31 m
6′	7.06 s	4.52 dd (12.0, 7.2)	4.45 dd (11.7, 2.4)	3.62 dd (12.0, 4.8)	3.63 dd (11.7, 5.8)
		4.64 dd (12.0, 2.4)	4.47 dd (11.7, 7.5)	3.84 br d (12.0)	3.86 dd (11.7, 1.7)
2″,6″		8.03 m	8.00 d (7.5)		8.08 d (7.2)
3″,5″		7.49 m	7.46 t (7.5)		7.50 t (7.2)
4″		7.63 m	7.58 t (7.5)		7.61 t (7.2)
2‴,6‴		8.05 m	7.06 s		
3‴,5‴		7.49 m			
4‴		7.63 m			
OMe		3.29 s	3.28 s	3.41 s	3.36 s

[a] The data of **7** were obtained at 300 MHz, while the data of **8−11** were obtained at 600 MHz.

Table 4. ^{13}C NMR Data of **1−5**

position	**1**[a]	**2**[b]	**3**[b]	**4**[b]		position	**5**[b]
1	85.7	88.5	88.6	88.5	88.7	1‴	88.2
2	82.2	87.3	87.5	87.3	87.5	2‴	87.5
3	48.2	49.5	49.4	49.5	49.8	3‴	49.8
4	205.6	208.9	209.1	209.1	208.2	4‴	210.1
5	46.6	48.2	48.2	48.3	48.6	5‴	48.4
6	63.6	64.7	64.6	64.9	65.2	6‴	64.9
7	30.2	27.2	27.5	27.3	27.6	7‴	26.8
8	61.8	63.8	63.8	63.3	63.7	8‴	63.9
9	104.7	107.5	107.6	107.6	107.7	9‴	107.7
10	19.3	20.6	20.7	20.7	20.7	10‴	20.5
1′	129.8	99.8	99.9	99.9	99.9	1⁗	99.7
2′	129.1	74.9	75.0	75.0	75.0	2⁗	74.7
3′	128.6	77.8	77.9	78.2	78.0	3⁗	78.0
4′	133.6	72.0	72.1	72.0	71.7	4⁗	71.7
5′	128.6	75.2	75.2	75.3	77.8	5⁗	76.8
6′	129.1	65.0	64.8	65.1	62.8	6⁗	69.8
7′	167.3						
1″		131.3	131.6	121.9	131.2	1′′′′′	131.2
2″,6″		130.6	130.6	132.9	130.7	2′′′′′,6′′′′′	130.9
3″,5″		129.6	129.6	116.2	129.6	3′′′′′,5′′′′′	129.9
4″		134.5	134.4	163.7	134.4	4′′′′′	134.8
7″		167.8	167.8	167.8	167.8	7′′′′′	167.9
1‴		131.1	121.3	131.5			
2‴,6‴		130.5	110.1	130.5			
3‴,5‴		129.6	146.6	129.7			
4‴		134.4	139.9	134.5			
7‴		167.6	167.9	167.6			
OMe	55.7	55.7	55.8	55.7			55.8

[a] $CDCl_3$ was used as solvent. [b] Methanol-d_4 was used as solvent. The data of **1**, **2**, and **5** were obtained at 75 MHz, while the data of **3** and **4** were obtained at 150 MHz.

Table 5. ^{13}C NMR Data of **6−11** in Methanol-d_4

position	**6**		**7**	**8**	**9**	**10**	**11**
1	88.7	89.1	72.0	89.1	89.1	89.2	93.4
2	87.6	87.3	31.2	86.9	87.1	87.2	86.9
3	49.5	44.5	43.4	42.0	41.6	42.2	41.7
4	208.4	106.5	216.6	109.2	109.5	109.5	68.4
5	47.9	43.8	35.2	42.0	41.6	40.5	41.6
6	65.2	72.1	45.6	71.5	72.0	71.7	56.8
7	27.6	22.9	14.4	22.9	22.7	23.3	28.4
8	63.7	61.7	70.7	61.5	61.5	59.0	62.0
9	107.4	102.3	24.4	102.2	102.5	102.4	178.0
10	20.8	19.5	18.5	19.5	19.6	19.6	20.5
1′	100.0	100.0	121.2	100.1	100.0	99.9	100.1
2′	75.0	74.8	110.2	74.9	74.8	74.9	74.8
3′	78.1	78.1	146.5	77.8	77.9	78.1	78.1
4′	75.1	71.7	140.0	72.1	71.5	73.4	71.6
5′	77.9	76.7	146.5	75.1	75.1	77.9	78.0
6′	62.9	69.5	110.2	65.0	64.7	62.8	62.8
7′			168.2				
1″	131.2	131.2		131.2[a]	131.1		131.2
2″,6″	130.7	130.9		130.6	130.6		130.7
3″,5″	129.6	129.9		129.7	129.6		129.6
4″	134.4	134.7		134.5	134.4		134.4
7″	167.8	168.0		167.9	167.9		167.9
1‴				131.4[a]	121.3		
2‴,6‴				130.6	110.2		
3‴,5‴				129.7	146.6		
4‴				134.5	139.9		
7‴				167.6	167.9		
OMe				51.5	51.8	51.4	49.5

[a] Assignments may be exchanged. The data of **6**, **7**, and **9−11** were obtained at 75 MHz, while the data of **8** were obtained at 150 MHz.

expressed as a percentage of the response of the related LPS-treated groups that were designated as 100%. IC_{50} values for the inhibition of NO release were determined on the basis of linear or nonlinear regression analysis of the concentration−response data curves (n = 3). Dunnet's *t*-test was used for statistical analysis.

Cell Viability Assay. Cell viability was evaluated in microglial cells by the MTT (3-(4,5-dimethylthiazole-2-yl)-2,5-diphenyltetrazolium bromide) assay.[19] In brief, microglial cells at 5×10^4 cells/well were seeded into 96-well microtiter plates and treated with various test sample solutions with LPS (1 μg/mL) for 48 h. The treated cells were incubated with MTT (0.25 mg/mL) at 37 °C for 3 h. After various treatments, the medium was removed and the cells were incubated with MTT (0.25 mg/mL) for 3 h at 37 °C. The formazan crystals in the cells were solubilized with DMSO. The level of MTT formazan was determined by measuring its absorbance at 490 nm with the Spectra Shell reader.

Extraction and Isolation. The roots of *P. albiflora* (6 kg) were repeatedly (×3) extracted with EtOH−H_2O (6:4, v/v) for 2 h. The combined extracts were concentrated under vacuum. The residue was suspended in H_2O and partitioned with EtOAc (×3). The EtOAc layer was subjected to silica gel column chromatography with $CHCl_3$−MeOH (100:1 to 1:1) to obtain 13 fractions (A−M). Fraction E (5 g) was further subjected to silica gel column chromatography using a $CHCl_3$−acetone gradient system to obtain compound **24** (120 mg), and fraction G (12 g) was further subjected to silica gel column chromatography with a $CHCl_3$−acetone gradient system to obtain 13 fractions

Table 6. Effect of Compounds **1**−**25** on NO Production by LPS-Activated Microglia[c]

compound	cont.[a]	conc. 0[b]	1.0 μM	3.0 μM	10.0 μM	30.0 μM	IC_{50} (μM)
1	7.8 ± 0.5	100.0 ± 2.5[d]	89.9 ± 5.4	91.5 ± 2.1	85.0 ± 6.9[e]	66.2 ± 6.4[f]	>100
2	9.4 ± 0.6	100.0 ± 2.2[d]	86.6 ± 14.1	74.9 ± 4.3[e]	59.1 ± 7.7[f]	57.2 ± 7.6[f]	36.2
3	9.2 ± 0.4	100.0 ± 2.3[d]	63.0 ± 16.9[e]	64.1 ± 8.9[f]	52.2 ± 3.4[f]	43.5 ± 6.0[f]	13.9
4	10.4 ± 0.5	100.0 ± 6.6[d]	106.5 ± 6.3	111.0 ± 4.4	94.3 ± 5.8	80.3 ± 4.0[f]	>100
5	10.8 ± 0.6	100.0 ± 6.2[d]	111.5 ± 0.8	96.6 ± 6.2	83.5 ± 4.5[f]	77.1 ± 4.6[f]	—[h]
6	8.7 ± 0.8	100.0 ± 3.5[d]	101.9 ± 0.5	101.8 ± 2.0	88.8 ± 2.7[e]	69.2 ± 0.6[g]	64.7
7	9.7 ± 0.4	100.0 ± 3.3[d]	91.3 ± 1.5	83.8 ± 4.6[e]	67.5 ± 0.3[g]	64.1 ± 3.4[g]	61.8
8	7.6 ± 0.2	100.0 ± 5.8[d]	91.9 ± 1.2	95.5 ± 3.3	70.4 ± 1.5[f]	34.9 ± 2.6[g]	22.6
9	10.5 ± 0.2	100.0 ± 0.6[d]	80.8 ± 8.8	113.4 ± 7.6	108.4 ± 10.1	101.5 ± 6.85	>100
10	19.3 ± 2.4	100.0 ± 1.3[d]	101.3 ± 3.9	103.5 ± 6.5	97.4 ± 8.4	78.9 ± 4.9	>100
11	18.5 ± 0.6	100.0 ± 6.5[d]	91.5 ± 2.1	91.6 ± 9.0	95.5 ± 9.0	102.4 ± 1.9	>100
12	8.8 ± 0.8	100.0 ± 2.9[d]	100.3 ± 3.1	87.8 ± 4.8	79.5 ± 4.5[e]	66.7 ± 7.7[e]	27.8
13	8.5 ± 0.7	100.0 ± 2.7[d]	86.3 ± 1.1[e]	81.4 ± 6.2[e]	79.5 ± 9.5[e]	68.6 ± 8.1[f]	—[h]
14	7.0 ± 0.2	100.0 ± 5.8[d]	82.1 ± 0.1[e]	84.4 ± 1.2[e]	62.1 ± 12.5[e]	69.5 ± 5.5[g]	—[h]
15	10.5 ± 0.2	100.0 ± 0.6[d]	71.8 ± 1.3[f]	66.7 ± 4.6[f]	57.5 ± 2.2[f]	38.5 ± 1.1[g]	12.8
16	19.8 ± 3.9	100.0 ± 1.3[d]	107.1 ± 4.3	106.3 ± 4.5	99.8 ± 1.2	90.1 ± 2.1	>100
17	18.7 ± 0.2	100.0 ± 2.2[d]	90.1 ± 5.7	91.5 ± 6.7	85.1 ± 1.8[e]	70.5 ± 0.8	>100
18	19.6 ± 0.4	100.0 ± 2.6[d]	71.7 ± 2.3[f]	83.0 ± 4.2[e]	76.6 ± 5.6[g]	76.6 ± 1.6[g]	>100
19	20.8 ± 0.7	100.0 ± 4.5[d]	97.1 ± 4.3	96.4 ± 4.5	91.8 ± 1.2	90.1 ± 2.1	>100
20	19.6 ± 0.4	100.0 ± 2.5[d]	86.2 ± 3.1[e]	83.9 ± 5.8[e]	80.9 ± 5.1[e]	67.4 ± 5.5[g]	>100
21	18.2 ± 0.2	100.0 ± 2.2[d]	89.4 ± 1.3	91.5 ± 4.4	86.7 ± 3.8[e]	81.2 ± 8.7[e]	>100
22	18.5 ± 0.8	100.0 ± 6.3[d]	86.7 ± 4.5[e]	88.3 ± 1.8[e]	82.0 ± 2.3[e]	82.6 ± 0.3[f]	>100
23	22.7 ± 0.5	100.0 ± 4.1[d]	92.6 ± 3.9	71.9 ± 0.2	24.5 ± 0.7	26.7 ± 1.1	—[h]
24	21.8 ± 0.6	100.0 ± 4.0[d]	111.2 ± 2.3	108.6 ± 1.8	107.2 ± 3.3	90.4 ± 6.5	>100
25	7.9 ± 0.5	100.0 ± 2.5[d]	93.8 ± 1.5	92.2 ± 4.2	82.3 ± 0.7[f]	70.5 ± 1.7[g]	>100
curcumin	22.8 ± 0.6	100.0 ± 2.2[d]	86.6 ± 2.1	75.6 ± 3.2[e]	56.1 ± 6.8[f]	35.2 ± 5.9[f]	13.5

[a] In unstimulated microglial cells, only small amounts of NO_2^- (5.8 ± 1.3 μM) could be detected in the medium. [b] Stimulation of microglial cells with LPS resulted in a marked increase in NO_2^- production (18.7 ± 0.5 μM). Data are represented as mean ± SEM of triplicate cultures. [c] Primary microglial cells were treated with serial dilutions of the fraction or compounds in the presence of LPS (1 μg/mL) and then incubated for 48 h. Absorbance of 540 nm was determined after mixing the culture supernatants with Griess reagent as described in the Experimental Section. [d] $p < 0.001$ vs control group (cultured in medium alone). [e] $p < 0.05$. [f] $p < 0.01$. [g] $p < 0.001$ vs the groups treated with LPS alone. Curcumin was used as a positive control. [h] "—" means this compound exhibited toxicity against microglial cells at the test concentrations.

(G1−G13). Fraction G4 was further chromatographed on a C-18 reversed-phase open column to yield subfractions G44 and G47; then subfraction G47 was purified by TLC ($CHCl_3$−MeOH, 10:1) to obtain compounds **8** (48 mg) and **14** (72 mg), and subfraction G44 was purified by TLC ($CHCl_3$−MeOH, 8:1) to obtain compounds **3** (35 mg) and **9** (13 mg). Fraction G7 was further chromatographed on a C-18 reversed-phase open column to obtain subfractions G73 and G75; then subfraction G75 was purified by HPLC (MeOH−H_2O, 7:3) to obtain compounds **1** (12 mg), **22** (18 mg), and **25** (26 mg), and subfraction G73 was purified by HPLC (MeOH−H_2O, 6:4) to obtain compounds **5** (22 mg), **16** (42 mg), and **17** (12 mg). Fraction H (14 g) was further subjected to silica gel column chromatography with a $CHCl_3$−MeCOMe gradient system to obtain 10 fractions (H1−H10), and fraction H7 was further chromatographed on a C-18 reversed-phase open column to obtain subfraction H74. Then H74 was purified by HPLC (MeOH−H_2O, 6:4) to obtain compounds **2** (18 mg) and **20** (25 mg). Fraction I (16 g) was further subjected to silica gel column chromatography with a $CHCl_3$−MeOH gradient system to give nine fractions (I1−I9), and fraction I2 was further chromatographed on a C-18 reversed-phase open column to obtain subfractions I21 and I24. Then subfraction I21 was purified by HPLC (MeOH−H_2O, 5:5) to obtain compounds **10** (42 mg) and **11** (17 mg), and subfraction I24 was purified by HPLC (MeOH−H_2O, 5:5) to obtain compounds **4** (33 mg), **23** (12 mg), and **12** (220 mg). Fraction I5 was further chromatographed on a C-18 reversed-phase open column to obtain subfractions I54 and I57. Then subfraction I54 was purified by HPLC (MeOH−H_2O, 4:6) to obtain compounds **13** (120 mg), **15** (29 mg), and **19** (35 mg), and subfraction I57 was purified by HPLC (MeOH−H_2O, 4:6) to obtain compounds **6** (54 mg) and **7** (19 mg). Fraction I6 was further chromatographed on a C-18 reversed-phase open column to obtain subfraction I63, which was then purified by HPLC (MeOH−H_2O, 6:4) to obtain compounds **21** (39 mg) and **18** (16 mg).

Hydrolysis of 2−6 and 8−11 and Determination of the Absolute Configuration of Sugars. A solution of each compound (3.0 mg) in 1 N HCl (1 mL) was stirred at 90 °C in a stoppered vial for 2 h. After cooling, the solution was evaporated under a stream of N_2. Anhydrous pyridine solutions (0.1 mL) of each residue and L-cysteine methyl ester hydrochloride (0.06 N) were mixed and warmed at 60 °C for 1 h. The trimethylsilylation reagent trimethylsilylimidazole (0.15 mL) was added, followed by warming at 60 °C for another 30 min. After drying the solution, the residue was partitioned between H_2O and cyclohexane. The cyclohexane layer was concentrated, then dissolved in 200 μL of acetone, and analyzed by GC using a DB-1701 column. The temperatures of the injector and detector were 270 and 280 °C, respectively. A temperature gradient system was used for the oven, starting at 160 °C for 1 min and increasing up to 230 °C at a rate of 5 °C /min. The peaks of authentic samples of D-glucose and L-glucose after treatment in the same manner were detected at 24.16 and 25.56 min.

Paeonidangenin (1): colorless oil; $[\alpha]^{25}_D$ −20.1(c 0.12, $CHCl_3$); UV (MeOH) λ_{max} (log ε) 230 (4.21) nm; IR (KBr) ν_{max} 3434, 2933, 1722, 1278, 1097, 1029, 715 cm^{-1}; ^1H NMR ($CDCl_3$, 600 MHz) Table 1 and ^{13}C NMR ($CDCl_3$, 75 MHz) Table 4; ESIMS m/z 355 $[M + Na]^+$; HRESIMS m/z 355.1156 $[M + Na]^+$ (calcd for $C_{18}H_{20}O_6Na$, 355.1158).

Paeonidanin A (2): white, amorphous powder; $[\alpha]^{25}_D$ −41.1 (c 0.25, MeOH); UV (MeOH) λ_{max} (log ε) 230 (4.25) nm; IR (KBr) ν_{max} 3418, 2968, 2843, 1718, 1279, 1054, 1031, 1014, 714 cm^{-1}; ^1H NMR (CD_3OD, 600 MHz) Table 1 and ^{13}C NMR (CD_3OD, 75 MHz) Table 4; ESIMS m/z 621 $[M + Na]^+$; HRESIMS m/z 621.1947 $[M + Na]^+$ (calcd for $C_{31}H_{34}O_{12}Na$, 621.1948).

Paeonidanin B (3): white, amorphous powder; $[\alpha]^{25}_D$ −31.6 (c 0.14, MeOH); UV (MeOH) λ_{max} (log ε) 224 (4.25) nm; IR (KBr) ν_{max} 3382, 2946, 2838, 1711, 1279, 1029, 716 cm^{-1}; ^1H NMR (CD_3OD, 600 MHz) Table 1 and ^{13}C NMR (CD_3OD, 150 MHz) Table 4; ESIMS m/z 669 $[M + Na]^+$; HRESIMS m/z 669.1792 $[M + Na]^+$ (calcd for $C_{31}H_{34}O_{15}Na$, 669.1795).

Paeonidanin C (4): white, amorphous powder; $[\alpha]^{25}_D$ −42.6 (c 0.10, MeOH); UV (MeOH) λ_{max} (log ε) 226 (4.25) nm; IR (KBr) ν_{max} 3380, 2934, 1711, 1278, 1073, 1024, 715 cm^{-1}; ^1H NMR (CD_3OD, 300 MHz) Table 1 and ^{13}C NMR (CD_3OD, 150 MHz) Table 4; ESIMS m/z 637 $[M + Na]^+$; HRESIMS m/z 637.1895 $[M + Na]^+$ (calcd for $C_{31}H_{34}O_{13}Na$, 637.1897).

Paeonidanin D (5): white, amorphous powder; $[\alpha]^{25}_D$ −65.4 (c 0.14, MeOH); UV (MeOH) λ_{max} (log ε) 230 (4.21) nm; IR (KBr) ν_{max} 3436, 2630, 1715, 1384, 1278, 1099, 1071, 715 cm^{-1}; ^1H NMR (CD_3OD, 300 MHz) Table 2 and ^{13}C NMR (CD_3OD, 75 MHz) Table 4; ESIMS m/z 979 $[M + Na]^+$; HRESIMS m/z 979.3214 $[M + Na]^+$ (calcd for $C_{47}H_{56}O_{21}Na$, 979.3212).

Paeonidanin E (6): white, amorphous powder; $[\alpha]^{25}_D$ −65.4 (c 0.10, MeOH); UV (MeOH) λ_{max} (log ε) 230 (4.20) nm; IR (KBr) ν_{max} 3382,

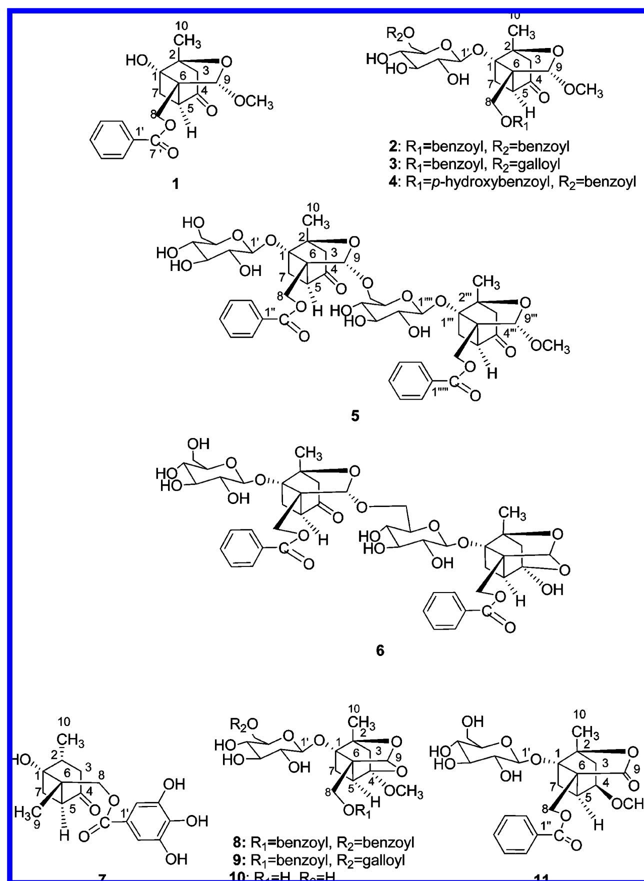

Figure 1. Structures of compounds **1**−**11**.

2948, 2838, 1654, 1452, 1118, 1029, 667 cm^{-1}; ^{1}H NMR (CD_3OD, 300 MHz) Table 2 and ^{13}C NMR (CD_3OD, 75 MHz) Table 4; ESIMS *m/z* 965 [M + Na]$^+$; HRESIMS *m/z* 965.3058 [M + Na]$^+$ (calcd for $C_{46}H_{54}O_{21}Na$, 965.3055).

Paeoniflorone (7): white, amorphous powder; $[\alpha]^{25}_{D}$ −32.8 (*c* 0.12, MeOH); UV (MeOH) λ_{max} (log ε) 220 (4.25) nm; IR (KBr) ν_{max} 3377, 29347, 2841, 1705, 1217, 1050, 1030, 768 cm^{-1}; ^{1}H NMR (CD_3OD, 300 MHz) Table 2 and ^{13}C NMR (CD_3OD, 75 MHz) Table 5; ESIMS *m/z* 359 [M + Na]$^+$; HRESIMS *m/z* 359.1108 [M + Na]$^+$ (calcd for $C_{17}H_{20}O_7Na$, 359.1107).

4-*O*-Methylbenzoylpaeoniflorin (8): white, amorphous powder; $[\alpha]^{25}_{D}$ −31.6 (*c* 0.15, MeOH); UV (MeOH) λ_{max} (log ε) 230 (4.25) nm; IR (KBr) ν_{max} 3389, 2944, 1721, 1278, 1073, 1027, 714 cm^{-1}; ^{1}H NMR (CD_3OD, 600 MHz) Table 3 and ^{13}C NMR (CD_3OD, 150 MHz) Table 5; ESIMS *m/z* 621[M + Na]$^+$; HRESIMS *m/z* 621.1952 [M + Na]$^+$ (calcd for $C_{31}H_{34}O_{12}Na$, 621.1948).

4-*O*-Methylgalloylpaeoniflorin (9): white, amorphous powder; $[\alpha]^{25}_{D}$ −32.2 (*c* 0.12, MeOH); UV (MeOH) λ_{max} (log ε) 220 (4.25) nm; IR (KBr) ν_{max} 3348, 2945, 2832, 1451, 1114, 1030, 668 cm^{-1}; ^{1}H NMR (CD_3OD, 600 MHz) Table 3 and ^{13}C NMR (CD_3OD, 75 MHz) Table 5; ESIMS *m/z* 669 [M + Na]$^+$; HRESIMS *m/z* 669.1799 [M + Na]$^+$ (calcd for $C_{31}H_{34}O_{15}Na$, 669.1795).

4-*O*-Methyldebenzoylpaeoniflorin (10): white, amorphous powder; $[\alpha]^{25}_{D}$ −33.9 (*c* 0.13, MeOH); UV (MeOH) λ_{max} (log ε) 205 (0.42) nm; IR (KBr) ν_{max} 3372, 2945, 2833, 1030, 635 cm^{-1}; ^{1}H NMR (CD_3OD, 600 MHz) Table 3 and ^{13}C NMR (CD_3OD, 75 MHz) Table 5; ESIMS *m/z* 413[M + Na]$^+$; HRESIMS *m/z* 413.1431 [M + Na]$^+$ (calcd for $C_{17}H_{26}O_{10}Na$, 413.1424).

4-*O*-Methylalbiflorin (11): white, amorphous powder; $[\alpha]^{25}_{D}$ −31.7 (*c* 0.11, MeOH); UV (MeOH) λ_{max} (log ε) 230 (4.25) nm; IR (KBr) ν_{max} 3397, 2517, 1751, 1452, 1118, 1021, 601 cm^{-1}; ^{1}H NMR (CD_3OD, 600 MHz) Table 3 and ^{13}C NMR (CD_3OD, 75 MHz) Table 5; ESIMS *m/z* 517[M + Na]$^+$; HRESIMS *m/z* 517.1690 [M + Na]$^+$ (calcd for $C_{24}H_{30}O_{11}Na$, 517.1686).

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