Three New Benzophenone Glucosides from the Leaves of *Planchonella obovata*

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Chemical investigation of the EtOH extract of the leaves of *Planchonella obovata* resulted in the isolation of four benzophenone glucosides, **1**–**4**, of which three, **2**–**4**, are new chemical entities together with five known flavonol glycosides. Their structures were elucidated by spectroscopic analysis. Among the isolated compounds, iriflophenone 2-*O*-(2,6-di-*O*-galloyl)-*β*-D-glucopyranoside (**4**) showed some activity (91.4 and 15.0% inhibition at 100 and 10 μg/ml, resp.) against the *α*-glucosidase of *Bacillus stearothermophilus*.

Introduction. – Inhibitors of *α*-glucosidases located in the small intestine brush border reduce the release of glucose from dietary carbohydrates and improve the glycemic control [1]. Several *α*-glucosidase inhibitors, *e.g.*, acarbose and miglitol, are currently used in clinic for the treatment of type-II diabetes [2]. In this context, we had set up an enzymatic screening system to find new *α*-glucosidase inhibitors from higher plants [3]. A preliminary study indicated that the AcOEt- and BuOH-soluble fractions of the MeOH extract of the leaves of *Planchonella obovata* (R. Br.) PIERRE (Sapotaceae) were active against *α*-glucosidase, both showing more than 85% inhibition at 100 μg/ml, and 15 to 20% inhibition at 10 μg/ml. *P. obovata* is an evergreen large tree, widely distributed in Tropical Asia such as India, Malaysia, Philippines, and Taiwan, serving as street and garden tree [4]. Previous studies on the chemical constituents of the *Planchonella* plants resulted in the isolation of pyrrolizidine alkaloids [5], triterpenoids [6], steroids, and saccharides [7]. However, no chemical investigation of the species *P. obovata* has been reported so far. Bioassay-guided fractionation and separation of these two active fractions *via Sephadex LH-20*, centrifugal partition chromatography, semi-preparative and preparative HPLC on *RP-18* columns led to the isolation of nine compounds. Here, we describe the structural characterization and *α*-glucosidase inhibitory activity of these compounds.

Results and Discussion. – Compounds **1**–**4** (*Fig. 1*) possessed a common 2,4,4′,6-tetraoxygenated benzophenone skeleton characteristic of iriflophenone derivatives. This conclusion was supported by their ^{1}H-NMR spectra which exhibited characteristic signals for a *meta*-coupled *AX* system (δ(H) 6.06 and 6.24 ($J = 2.0$) for **1**) and an *AA′XX′* system (δ(H) 7.68 and 6.78 ($J = 8.7$) for **1**), and by their ^{13}C-NMR spectra, which displayed signals for a conjugated CO group (δ(C) 197.5 (C(7)) for **1**), four O-bearing aryl C-atoms (164 ppm > δ(C) > 157 ppm), six aromatic CH groups, of which two (for C(3) and C(5)) appeared between 95 and 99 ppm, and two non-O-bearing

quaternary C-atoms, one around δ(C) 110 (for C(1)) and the other around δ(C) 132 (for C(1′)) [8]. These data also revealed an asymmetric moiety in ring *A*.

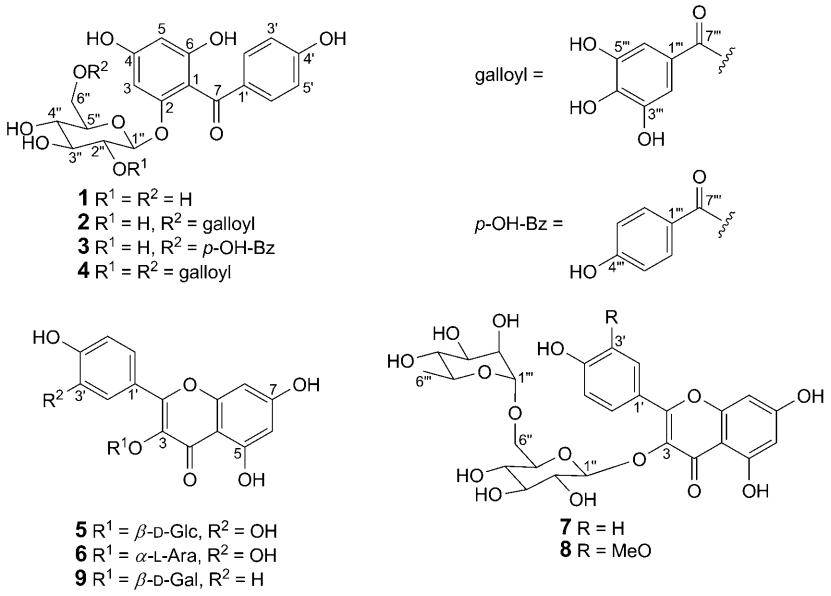

Fig. 1. *Structures of compounds* **1**–**9**

Compound **1** had a molecular formula $C_{19}H_{20}O_{10}$, as deduced from ESI-MS, and ^{1}H- and ^{13}C-NMR data. It contained a β-*O*-D-glucoypyranosyl residue as evidenced by the characteristic signals for the anomeric H-atom (δ(H) 4.82 (*d*, $J = 7.6$)) and anomeric C-atom (δ(C) 102.2 (*d*)), and C(6) (δ(C) 62.4 (*t*)). The structure of **1** was established as iriflophenone 2-*O*-β-D-glucopyranoside by comparison with literature data [9]. The ^{13}C-NMR data of **1** (CD_3OD) were almost identical to those reported except for the data for C(1) and C(1′) [9]. The reported chemical shifts for C(1) (δ(C) 99.8) and C(1′) (δ(C) 124.4) should be revised to δ(C) 110.3 (*s*) and δ(C) 132.1 (*s*), respectively. The revised assignments were confirmed by the correlations in the HMBC spectrum: C(1) (δ(C) 110.3, *s*)/H–C(3) (δ(H) 6.24, *d*) and H–C(5) (δ(H) 6.06, *d*), and C(1′) (δ(C) 132.1, *s*)/H–C(3′)/H–C(5′) (δ(H) 6.78, *d*). During our literature survey, we also found that the reported ^{1}H- and ^{13}C-NMR data (D_2O) for iriflophenone 4-*O*-β-D-glucopyranoside [8] were identical to those of **1** (D_2O). To clarify the glycosylated position, an NOESY experiment for **1** was carried out. This spectrum showed that the anomeric H-atom had NOE correlation with one aryl H-atom (δ(H) 6.24) only, instead of two as expected for the 4-*O*-β-D-glucopyranoside (for a typical NOESY depiction, see *Fig. 2* for compound **4**). Likewise, the HMBC spectrum of **1** (for a typical HMBC, see *Fig. 2* for compound **4**) revealed that the signal of the *O*-glucosylated C-atom (δ(C) 158.7, C(2)), assigned by its correlation to the anomeric H-atom (δ(H) 4.82), correlated to one aryl H-atom *singlet* only (δ(H) 6.24, H–C(3)). Hence, the structure of iriflophenone 4-*O*-β-D-glucopyranoside reported in [8] should be revised as **1**.

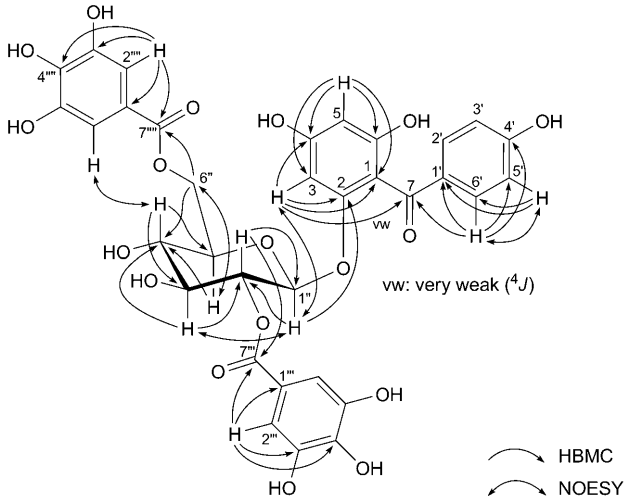

Fig. 2. *NOESY Correlations and HMBCs of compound* **4** (CD_3OD, 400 MHz)

Compound **2** had a molecular formula $C_{26}H_{24}O_{14}$, as deduced from HR-ESI-MS. Its ^{1}H- and ^{13}C-NMR spectra were highly similar to those of **1** except for the presence of additional signals typical for a galloyl moiety (δ(H) 7.07 (*s*, H−C(2‴), H−C(6‴)); δ(C) 168.3 (*s*, C(7‴)), 146.4 (*s*, C(3‴), C(5‴)), 139.8 (*s*, C(1‴)), and 110.2 (*d*, C(2‴), C(6‴))) [10]. Hence, **2** is a galloyl derivative of **1**. The ^{1}H-NMR assignment of the glucosyl moiety in **2** (*Table 1*) was accomplished by analysis of a COSY spectrum. These data revealed that the signals of the Glc CH_2(6) (*i.e.*, CH_2(6″)) in **2** were downfield-shifted relative to those in **1** (δ(H) 4.46/4.38 *vs.* 3.86/3.67) (*Table 1*). Accordingly, the structure of **2** was established as iriflophenone 2-*O*-(6-*O*-galloyl)-β-D-glucopyranoside. This structure was supported by the correlation of the ester C-atom (*i.e.*, C(7‴)) to the Glc CH_2(6) (*i.e.*, CH_2(6″)) and H−C(2)/H−C(6) of the galloyl residue (*i.e.*, H−C(2‴)/H−C(6‴)) observed in the HMBC spectrum.

Compound **3** had a molecular formula $C_{26}H_{24}O_{12}$, as deduced from HR-ESI-MS. Its ^{1}H- and ^{13}C-NMR spectra were also very similar to those of **1** except for the presence of additional signals typical for a 4-hydroxybenzoyl moiety, an *AA′XX′* system at δ(H) 7.90 (*d*, H−C(2‴)/H−C(6‴)) and 6.83 (*d*, H−C(3‴)/H−C(5‴)) (J_{AX} = 8.8 Hz) in the ^{1}H-NMR spectrum, and δ(C) 168.1 (*s*, C(7‴)), 163.5 (*s*, C(4‴)), 133.0 (*d*, C(2‴), C(6‴)), and 116.2 (*d*, C(3‴), C(5‴)) in the ^{13}C-NMR spectrum [11]. Since the signals of the Glc CH_2(6) (*i.e.*, CH_2(6″)) in **3**, assignable on the basis of the characteristic coupling pattern, were also downfield-shifted relative to those in **1** (δ(H) 4.60/4.25 in **3** *vs.* 3.86/3.67 in **1**), this arylcarboxyl residue was *O*-linked to the Glc C(6). Accordingly, the structure of **3** was established as iriflophenone 2-*O*-[6-*O*-(4-hydroxybenzoyl]-β-D-glucopyranoside.

Compound **4** had a molecular formula $C_{33}H_{28}O_{18}$, as deduced from HR-ESI-MS. Its ^{1}H- and ^{13}C-NMR spectra were similar to those of **2** except for the presence of additional signals typical for another galloyl moiety at δ(H) 6.92 (*s*, 2 H) in the ^{1}H-NMR spectrum, and the corresponding C-atom signals in the ^{13}C-NMR spectrum

Table 1. *^{1}H- and ^{13}C-NMR Data of Compounds* **1**–**4** (^{1}H at 400 MHz; ^{13}C at 100 MHz, in CD_3OD)

	δ(H)				δ(C)			
	1	**2**	**3**	**4**[a])	**1**	**2**	**3**	**4**[b])
C(1)					110.3 (*s*)	110.7 (*s*)	110.6 (*s*)	111.3 (*s*)
C(2)					158.7 (*s*)	158.6 (*s*)	158.6 (*s*)	157.9 (*s*)
H–C(3)	6.24 (*d*, *J* = 1.8)	6.22 (*d*, *J* = 2.0)	6.25 (*d*, *J* = 2.0)	6.27 (*d*, *J* = 2.2)	95.8 (*d*)	96.2 (*d*)	95.9 (*d*)	96.8 (*d*)
C(4)					162.4 (*s*)	162.2 (*s*)	160.1 (*s*)	158.9 (*s*)
H–C(5)	6.06 (*d*, *J* = 1.8)	6.07 (*d*, *J* = 2.0)	6.08 (*d*, *J* = 2.0)	6.06 (*d*, *J* = 2.2)	98.1 (*d*)	98.4 (*d*)	98.4 (*d*)	98.7 (*d*)
C(6)					159.6 (*s*)	159.7 (*s*)	162.7 (*s*)	161.6 (*s*)
C(7)					197.5 (*s*)	197.4 (*s*)	197.6 (*s*)	196.7 (*s*)
C(1′)					132.1 (*s*)	132.3 (*s*)	132.4 (*s*)	131.6 (*s*)
H–C(2′/6′)	7.68 (*d*, *J* = 8.8)	7.65 (*d*, *J* = 8.8)	7.64 (*d*, *J* = 8.7)	7.41 (*d*, *J* = 8.7)	133.5 (*d*)	133.4 (*d*)	133.4 (*d*)	133.2 (*d*)
H–C(3′/5′)	6.78 (*d*, *J* = 8.8)	6.74 (*d*, *J* = 8.8)	6.73 (*d*, *J* = 8.7)	6.56 (*d*, *J* = 8.7)	115.8 (*d*)	115.8 (*d*)	155.7 (*d*)	115.6 (*d*)
C(4′)					163.6 (*s*)	163.4 (*s*)	163.4 (*s*)	163.4 (*s*)
H–C(1″)	4.82 (*d*, *J* = 7.6)	*ca.* 4.80[c])	4.89 (*d*, *J* = 7.8)	5.09 (*d*, *J* = 8.0)	102.2 (*d*)	102.4 (*d*)	101.1 (*d*)	100.8 (*d*)
H–C(2″)	3.09 (*dd*, *J* = 7.6, 8.8)	3.11 (*dd*, *J* = 7.7, 9.3)	3.08 (*dd*, *J* = 9.0, 7.8)	*ca.* 4.80[c])	74.6 (*d*)	74.6 (*d*)	74.5 (*d*)	74.6 (*d*)
H–C(3″)	3.37 (*dd*, *J* = 8.8, 9.3)	3.38–3.44 (*m*)	3.39 (*t*, *J* = 9.0)	3.63 (*dd*, *J* = 8.0, 9.0)	77.7 (*d*)	77.6 (*d*)	77.7 (*d*)	76.3 (*d*)
H–C(4″)	3.27 (*t*, *J* = 9.3)	3.36–3.42 (*m*)	*ca.* 3.31[d])	3.58 (*dd*, *J* = 9.0, 9.2)	71.0 (*d*)	71.1 (*d*)	71.6 (*d*)	71.2 (*d*)
H–C(5″)	3.33–3.37 (*m*)	3.61 (*ddd*, *J* = 2.2, 4.8, 9.5)	3.60–3.64 (*m*)	3.71 (*ddd*, *J* = 2.2, 4.4, 9.5)	78.1 (*d*)	75.5 (*d*)	75.5 (*d*)	75.6 (*d*)
CH_2(6″)	3.86 (*dd*, *J* = 12.3, 2.3), 3.67 (*dd*, *J* = 12.3, 5.3)	4.46 (*dd*, *J* = 2.2, 12.1), 4.38 (*dd*, *J* = 4.8, 12.1)	4.60 (*dd*, *J* = 2.1, 11.8), 4.25 (*dd*, *J* = 7.0, 11.8)	4.51 (*dd*, *J* = 2.2, 12.0), 4.40 (*dd*, *J* = 12.0, 4.4)	62.4 (*t*)	64.4 (*t*)	65.0 (*t*)	64.2 (*t*)
C(1‴)						121.3 (*s*)	122.0 (*s*)	121.4 (*s*)
H–C(2‴/6‴)		7.07 (*s*)	7.90 (*d*, *J* = 8.8)	6.92 (*s*)		110.2 (*d*)	133.0 (*d*)	110.6 (*d*)
H–C(3‴/5‴)			6.83 (*d*, *J* = 8.8)			146.4 (*s*)	116.2 (*d*)	146.1 (*s*)
C(4‴)						139.8 (*s*)	163.5 (*s*)	139.7 (*s*)
C(7‴)						168.3 (*s*)	168.1 (*s*)	166.9 (*s*)

[a]) ^{1}H-NMR Data for galloyl-2 (R^2): δ(H) 7.11 (*s*, H–C(2⁗/6⁗)). [b]) ^{13}C-NMR data for galloyl-2 (R^2): δ(C) 121.3 (*s*, C(1⁗)), 110.3 (*d*, C(2⁗/6⁗)), 146.5 (*s*, C(3⁗/5⁗)), 139.8 (*s*, C(4⁗)), 168.3 (*s*, C(7⁗)). [c]) Signals overlapped with DOH. [d]) Signals overlapped with CD_2HOD.

(*Table 1*). Hence, **4** is a galloyl derivative of **2**. The ^{1}H-NMR data of the glucosyl moiety in **4**, assigned based on the analysis of a COSY spectrum, revealed that the signals of the Glc H–C(1), H–C(2), and CH_2(6) in **4** were downfield-shifted relative to those in **1** (δ(H) 5.09 *vs.* 4.82 (H–C(1)); 4.80 *vs.* 3.09 (H–C(2)); 4.51/4.40 *vs.* 3.86/3.67 (CH_2(6))). Accordingly, the two galloyl residues were *O*-linked to the Glc C(2) and C(6), respectively. The structure of **4** was established as iriflophenone 2-*O*-(2,6-di-*O*-galloyl)-β-D-glucopyranoside. This structure was confirmed by the observation of the correlation of the galloyl-1 (R′) C(7) (*i.e.*, C(7‴), an ester C-atom) to the Glc H–C(2) (*i.e.*, H–C(2″)), and the galloyl-2 (R′) C(7) (*i.e.*, C(7⁗)) to the Glc CH_2(6) (*i.e.*, CH_2(6″)) in the HMBC spectrum (*Fig. 2*).

Compounds **5**–**9** were flavonol 3-*O*-glycosides, and were identified as quercetin 3-*O*-β-D-glucopyranoside (**5**) [12], quercetin 3-*O*-α-L-arabinopyranoside (**6**) [13], kaempferol 3-*O*-robinobioside (**7**) [14], isorhamnetin 3-*O*-robinobioside (**8**) [15], and kaempferol 3-*O*-β-D-galactopyranoside (**9**) [16], upon comparing their spectroscopic data to those reported.

To the best of our knowledge, the benzophenone glucosides **2**–**4** are new chemical entities. The complete ^{1}H- and ^{13}C-NMR spectral assignments for these compounds (*Table 1*) were accomplished based on detailed 1D- and 2D-NMR spectral analysis as indicated above. The NOESY and HMBC correlations for **4** are depicted in *Fig. 2* as typical examples for compounds **1**–**4**.

The inhibitory activities of compounds **1**–**9** against α-glucosidase type IV from *Bacillus stearothermophilus* [3] were assayed (*Table 2*). The flavonol glycosides **7**–**9** with kaempferol or 3′-methoxykaempferol as aglycon were inactive at a concentration of 100 μg/ml. The flavonol glycoside **6** with quercetin as aglycon showed some activity with 40.1% inhibition at the same concentration. Hence, the presence of more phenolic functions in the aglycon, such as quercetin in **5** *vs.* kaempferol in **7**, and a less-polar glycosyl residue, such as L-arabinosyl in **6** *vs.* D-glucosyl in **5**, appear to increase the activity (*Table 2*). As for benzophenone glucosides **1**–**4**, the inhibition order is as follows: **4** (91.4%), **1** (33.0%), **2** (9.6%), **3** (3.5%). Thus, more galloyl residues (**4** *vs.* **2**) and more phenolic functions (**2** *vs.* **3**) provide better activity as observed for stilbenoids

Table 2. *Inhibitory Effects of Compounds* **1**–**9** *against* α*-Glucosidase*

Compound	Inhibition [%][a])[b])	
	100 μg/ml	10 μg/ml
1	33.0 ± 3.1	− 1.6 ± 5.7
2	9.6 ± 7.1	0.2 ± 5.5
3	3.5 ± 2.4	1.4 ± 2.8
4	91.4 ± 6.3	15.0 ± 6.9
5	9.7 ± 1.4	2.2 ± 0.6
6	40.1 ± 7.8	− 0.6 ± 0.7
7	− 13.3 ± 1.2	− 4.5 ± 1.5
8	− 14.2 ± 3.4	− 5.6 ± 2.3
9	− 8.7 ± 3.8	− 6.5 ± 1.6

[a]) The % inhibition at two concentrations of each tested compound in triplicate experiments was expressed as mean ± SD. [b]) The % inhibition of the positive control acarbose: 70.5 ± 5.2 at 40 ng/ml.

[3]. At 10 μg/ml, only compound **4** showed weak activity, 15.0% inhibition, which was even weaker than the BuOH-soluble fraction (97 and 20% inhibition at 100 and 10 μg/ml, resp.) of the MeOH extract of the leaves. This may be due to synergistic effects or the presence of additional active constituents in the extract.

Experimental Part

General. HPLC: *Agilent 1100* system, *Phenomenex Prodigy ODS-3* (250 × 10 mm, 5 μm; semi-prep.), *Lichrospher RT RP-18e* (250 × 25 mm, 5 μm; prep. use) (*Merck*, D-Darmstadt), detection at 280 nm, MeCN and MeOH (*Mallinckrodt Baker Inc.*, USA), deionized H_2O (*Barnstead water purification system*, Dubuque, IA, USA). Centrifugal partition chromatography (CPC): *Sanki CPC* (*model LLB-M*, 110 ml; *Sanki Engineering Ltd.*, Kyoto, Japan). TLC (System *1*): silica gel *60* F_{254} (*Merck*, D-Darmstadt); $CHCl_3$/MeOH/H_2O 7:4:1. Bioassay system: *SPECTRAmax_ PLUS* (*Molecular Devices*, California, USA); *p*-nitrophenyl α-D-glucopyranoside, α-glucosidase type IV from *B. stearothermophilus* (*Sigma–Aldrich Co.*, Germany), K_2HPO_4 and KH_2PO_4 (*Merck*, D-Darmstadt). M.p.: DSC. Optical rotations: *Jasco DIP-370* polarimeter. UV Spectra (MeOH): λ_{max} nm (log ε), *Hitachi 150-20 Double Beam* spectrophotometer. CD Spectra (MeOH): λ in nm, *Jasco J-720* spectropolarimeter. IR Spectra (KBr): *Jasco FT/IR-410* spectrometer; in cm^{-1}. ^1H-, ^{13}C-, and 2D-NMR spectra: *Bruker AV-400* spectrometer (δ in ppm, in CD_3OD, residual non-deuterated solvent peaks as reference: δ(H) 3.30 and δ(C) 49.0 ppm, *J* in Hz). MS: *MicrOTOF orthogonal ESI-TOF* (HR-ESI-MS) mass spectrometer (*Bruker*, *Daltonik*, D-Bremen), in *m*/*z* (rel. %).

Plant Material. The leaves of *Planchonella obovata* (R. Br.) PIERRE were collected in September 2006, in Lanyu Island, Taitung County, Taiwan. A specimen was authenticated by Prof. *Ih-Sheng Chen*, College of Pharmacy, Kaohsiung Medical University, Kaohsiung, Taiwan. A voucher specimen (NTUSP200609A) was deposited with the Herbarium of School of Pharmacy, National Taiwan University.

Extraction and Isolation. The powdered, dried leaves (342.6 g) were percolated with MeOH (2.5 l × 3) at r.t. and then extracted with warm MeOH (2 l, 50°) to give the MeOH extract (50.2 g) upon evaporation under reduced pressure. This extract suspended in H_2O (500 ml) was partitioned against CH_2Cl_2 (3 × 300 ml), AcOEt (1 × 300 ml), and BuOH (sat. with H_2O, 3 × 300 ml) to give fractions soluble in CH_2Cl_2 (13.1 g), AcOEt (6.8 g), BuOH (9.1 g), and H_2O (24.4 g). A portion of the AcOEt-soluble fraction (*E.I*, 2.6 g) was fractionated on a CPC (× 2, 1.35 g each run) with the upper phase of $CHCl_3$/MeOH/H_2O 10:10:5 as mobile phase, to give four fractions (*E.I-1* – *E.I-4*). *Fr. E.I-2* (147.7 mg) yielded **1** (25.6 mg) and **2** (49.4 mg) after separation over a *Sephadex LH-20* column (329 ml, 50% $MeOH_{aq}$). *Fr. E.I-3* (75.6 mg) yielded **3** (4.5 mg) after separation over a *Sephadex LH-20* (329 ml, 75% $MeOH_{aq}$), followed by semi-prep. HPLC on an *RP-18* column (four runs, 18% $MeCN_{aq}$, flow rate 2.5 ml/min), and two successive *Sephadex LH-20* columns (106 ml, 50% $MeOH_{aq}$; 85 ml, MeOH). Another portion of the AcOEt-soluble fraction (*E.II*, 2.7 g) was fractionated on a *Sephadex LH-20* column (740 ml, MeOH) to give three fractions (*E.II-1* – *E.II-3*). *Fr. E.II-2* (966 mg) was sonicated with CH_2Cl_2 and AcOEt, 20 ml each, in sequence to give fractions soluble in CH_2Cl_2 and AcOEt (*E.II-2E*, 399 mg). *Fr. E.II-2E* was separated over a *Sephadex LH-20* column (353 ml, CH_2Cl_2/MeOH 1:1) to give four fractions (*E.II-2E-1* – *E.II-2E-4*). *Fr. E.II-2E-4* (6.0 mg) was pure **4**. *Fr. E.II-2E-2* (21.4 mg) dissolved in 50% $MeOH_{aq}$ was separated by semi-prep. HPLC on an *RP-18* column (4 runs), delivered with 17% $MeCN_{aq}$, yielded **5** (4.7 mg) and **6** (1.4 mg). The BuOH-soluble fraction (*B*, 9.1 g) was fractionated on a *Sephadex LH-20* column (3.31 l, MeOH) to give seven fractions (*B-1* – *B-7*), combined based on silica-gel TLC analysis. Part of *Fr. B-2* (222 mg out of 1.30 g) was further purified by CC on *Sephadex LH-20* (328 ml, MeOH/H_2O 3:1) to give additional crop of compound **1** (146 mg). Most of *Fr. B-3* (140 mg out of 175 mg) dissolved in 9% $MeCN_{aq}$ was separated by prep. HPLC on a *RP-18* column (four runs), delivered with 9% to 32% $MeCN_{aq}$ in 60 min by a linear gradient mode, flow rate 8.2 ml/min, to give three fractions (*B-3-1* – *B-3-3*). *Fr. B-3-2* (t_R 42–55 min; 36.7 mg) was further separated by semi-prep. HPLC on a *RP-18* column (five runs), eluted by 37 to 41% $MeOH_{aq}$, flow rate 2.4 ml/min, to give three fractions (*Frs. B-3-2-1* – *B-3-2-3*). *Fr. B-3-2-2* (3.4 mg; t_R 45.7 min) yielded **7** (2.0 mg) after separation

over a *Sephadex LH-20* column (97 ml, MeOH). *Fr. B-3-2-3* (6.7 mg) yielded **8** (1.6 mg; t_R 21.2 min) after separation by semi-prep. HPLC on a *RP-18* column (two runs), eluted by 20% $MeCN_{aq}$, flow rate 2.5 ml/min with UV detection at 280 nm. Most of *Fr. B-5* (150 mg out of 197 mg) dissolved in 50% $MeOH_{aq}$ was separated by prep. HPLC on a *RP-18* column (four runs), with 19% $MeCN_{aq}$, flow rate 8.0 ml/min, detection at 280 nm, to give **9** (t_R 54.2 min; 1.2 mg) and additional crop of **3** (t_R 57 min; 10.8 mg). *Fr. B-6* (461 mg) was separated on a *Sephadex LH-20* column (329 ml, 75% $MeOH_{aq}$), followed by recrystallization (H_2O), to yield additional crop of **2** (53.9 mg).

Assay for α-Glucosidase Activity. Tests were performed by the method of *Pistia-Brueggemann* and *Hollingsworth* [17], slightly modified according to [3]. Compounds **1**–**9** were dissolved in 10% $MeOH_{aq}$ and assayed against α-glucosidase (type IV from *Bacillus stearothermophilus*). Acarbose (*Bayer*) was chosen as the pos. control.

*Iriflophenone 2-*O*-β-*D*-Glucopyranoside* (= *3,5-Dihydroxy-2-[(4-hydroxyphenyl)carbonyl]phenyl β-*D*-Glucopyranoside*; **1**). TLC (System *1*): R_f 0.57. $[\alpha]_D^{25} = -30.0$ ($c = 0.1$, MeOH) ([10]: −24 ($c = 1.0$, MeOH)). CD: $[\theta]_{231}$ +5822, $[\theta]_{240}$ +3533, $[\theta]_{277}$ +5985, $[\theta]_{298}$ +3156. ^{1}H- and ^{13}C-NMR: *Table 1* (CD_3OD).

*Iriflophenone 2-*O*-(6-*O*-Galloyl)-β-*D*-glucopyranoside* (= *3,5-Dihydroxy-2-[(4-hydroxyphenyl)carbonyl]phenyl 6-*O*-[(3,4,5-Trihydroxyphenyl)carbonyl]-β-*D*-glucopyranoside*; **2**). TLC (System *1*): R_f 0.43. M.p. 220.9°. $[\alpha]_D^{25} = -10.0$ ($c = 0.1$, MeOH). UV: 276 (4.48), 219 (4.88). CD (MeOH): $[\theta]_{230}$ +9603, $[\theta]_{243}$ +5960, $[\theta]_{269}$ +7047. IR: 3522, 3274, 1682, 1595, 1510, 1455, 1325, 1264, 1192, 1177, 1081, 1048, 925, 833, 772. ^{1}H- and ^{13}C-NMR: *Table 1*. ESI-MS (neg.): 559.0 ($[M-H]^-$). ESI-MS (pos.): 583.1 ($[M+Na]^+$). HR-ESI-MS (neg.): 559.1094 ($[M-H]^-$, $C_{26}H_{23}O_{14}^-$; calc. 559.1088).

*Iriflophenone 2-*O*-[6-*O*-(4-Hydroxybenzoyl)]-β-*D*-glucopyranoside* (= *3,5-Dihydroxy-2-[(4-hydroxyphenyl)carbonyl]phenyl 6-*O*-[(4-Hydroxyphenyl)carbonyl]-β-*D*-glucopyranoside*; **3**). TLC (System *1*): R_f 0.67. $[\alpha]_D^{24} = -5.0$ ($c = 0.1$ MeOH). UV: 273 (4.42), 214 (4.87). CD (MeOH): $[\theta]_{227}$ +10271, $[\theta]_{257}$ +9498. IR: 3336, 2925, 1698, 1604, 1511, 1455, 1275, 1167, 1072, 769. ^{1}H- and ^{13}C-NMR: *Table 1*. ESI-MS (neg.): 527.1 ($[M-H]^-$). ESI-MS (pos.): 551.2 ($[M+Na]^+$). HR-ESI-MS (neg.): 527.1174 ($[M-H]^-$, $C_{26}H_{23}O_{12}^-$; calc. 527.1190).

*Iriflophenone 2-*O*-(2,6-Di-*O*-galloyl)-β-*D*-glucopyranoside* (= *3,5-Dihydroxy-2-[(4-hydroxyphenyl)-carbonyl]phenyl 2,6-Bis-*O*-[(3,4,5-trihydroxyphenyl)carbonyl]-β-*D*-glucopyranoside*; **4**). TLC (System *1*): R_f 0.13. $[\alpha]_D^{25} = -30.0$ ($c = 0.1$, MeOH). UV: 274 (4.63), 217 (5.01). CD (MeOH): $[\theta]_{215}$ +3517, $[\theta]_{233}$ +3282, $[\theta]_{258}$ +12016, $[\theta]_{265}$ +13089, $[\theta]_{273}$ +13375, $[\theta]_{313}$ +73 $[\theta]_{366}$ +6981. IR: 3355, 1704, 1609, 1450, 1317, 1228, 1034, 930, 762. ^{1}H- and ^{13}C-NMR: *Table 1*. HMBC and NOESY: *Fig. 2*. ESI-MS (neg.): 710.7 ($[M-H]^-$). ESI-MS (pos.): 734.8 ($[M+Na]^+$). HR-ESI-MS (neg.): 711.1220 ($[M-H]^-$, $C_{33}H_{27}O_{18}^-$; calc. 711.1197).

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