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Purunusides A-C, α-Glucosidase Inhibitory Homoisoflavone Glucosides from *Prunus domestica*

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Purunusides A-C (**1-3**), new homoisoflavone glucosides together with the known compounds β-sitosterol (**4**) and 6,7-methylenedioxy-8-methoxycoumarin (**5**) have been isolated from *n*-butanol and ethyl acetate soluble fractions of *Prunus domestica*. Their structures were assigned on the basis of spectral studies. The compounds **1-3** showed potent inhibitory activity against the enzyme α-glucosidase.

Key words: *Prunus domestica*, Rosaceae, Purunusides A-C, α-Glucosidase inhibition

INTRODUCTION

The family Rosaceae comprises around 120 genera and about 4,000 species. The Prunus itself is a large genus comprising about 100 species with common occurrence all over the world. *Prunus domestica* is a deciduous tree, which is commonly cultivated in Pakistan and India not only for its edible fruit but also as a remedy for a variety of ailments including lowering of the level of blood sugar (Chopra et al., 1956; Parajapati et al., 2003; Zaidi et al., 2009; Baquar et al., 1989; Cavender et al., 2006; Fujii et al., 2006; Stacewicz-Sapuntzakis et al., 2001). Previously many compounds have been reported from this species (Nakatani et al., 2000; Nagarajan and Parmar, 1977; Nagarajan and Seshadri, 1964; Kayano et al., 2004; Deineka et al., 2005). The ethnopharmacological and chemotaxonomic importance of this species prompted us to carry out further phytochemical studies on *P. domestica*. An ethanolic extract of the shoots of *P. domestica* showed strong toxicity in brine shrimp lethality test (Meyer et al., 1982). Further pharmacological screening of the ethanolic extract and its subsequent sub-fractions showed inhibitory activity against the enzyme α-glucosidase which was most pronounced in the *n*-BuOH soluble fraction. Bioassay directed isolation studies on this fraction have now resulted in the isolation of three new homoisoflavone glucosides named as purunuside A (**1**), B (**2**) and C (**3**), respectively.

Studies on the ethyl acetate soluble fraction provided β-sitosterol (**4**) (Rubinstein et al., 1976) and 6,7-methylenedioxy-8-methoxycoumarin (**5**) (Sarker et al., 1994) isolated for the first time from this species. The compounds **1-3** showed significant inhibitory activity against the enzyme α-glucosidase.

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MATERIALS AND METHODS

General experimental procedures

Optical rotations were measured on an ATAGO AP-300 digital polarimeter using a 200 mm tube. IR spectra were measured on JASCO 302-A spectrophotometer in KBr. UV spectra were recorded on Hitachi-UV-3200 spectrophotometer. EI and HR-FAB-MS were recorded on Variant MAT 311 A and JEOL HX 110 mass spectrometers (*m/z*, rel int). The ^{1}H-NMR and ^{13}C-NMR, HMQC and HMBC spectra were recorded on Bruker AMX spectrometer operating at 400 MHz for ^{1}H- and 100 MHz for ^{13}C-NMR, respectively. The chemical shift values are reported in ppm (δ), relative

to tetramethylsilane as an internal standard and the coupling constant (*J*) are in Hz. Aluminium sheets precoated with silica gel 60-F_{254} (20 × 20 cm, 0.2 mm thick; E-Merck) were used for TLC. Silica gels 70-230 mesh and 250-400 mesh (E-Merck) were used for column and flash chromatography, respectively. Reverse phase column chromatography was performed on silica gel RP-18 (40-63 µm, E-Merck). Visualization of the TLC plates was carried out under UV at 254 and 366 nm and by spraying with ceric sulphate solution (with heating).

Materials

The shoots of *Prunus domestica* Linn. (12 kg) were collected from Swat Valley of North Western Frontier of Pakistan in December, 2007 and identified by Plant Taxonomist, Department of Botany, University of Karachi. A voucher specimen has been deposited in the herbarium of the University (No. 162007).

Extraction and isolation

The shade dried shoots of *Prunus domestica* (12 kg) were extracted with ethanol (3 × 15 L) at room temperature. The combined ethanolic extract was evaporated under reduced pressure to obtain a dark brown dense liquid (150 g). It was suspended in water and successively extracted with *n*-hexane, ethyl acetate, and *n*-butanol. The BuOH-soluble fraction (35 g) was subjected to column chromatography using $CHCl_3$-MeOH in increasing order of polarity. The fraction which eluted with $CHCl_3$-MeOH (7:3) (2.0 g) was subjected to flash chromatography eluting with $CHCl_3$-MeOH in increasing order of polarity. The major fraction which eluted with $CHCl_3$-MeOH (1:1) (1.3 g) was subjected to reverse phase column chromatography eluting with MeOH-H_2O in increasing order of polarity to obtain purunuside A (**1**) (MeOH-H_2O 2:1) (25 mg), purunuside B (**2**) (MeOH-H_2O 1:1) (28 mg) and purunuside C (**3**) (MeOH-H_2O 1:2) (32 mg), respectively. The EtOAc soluble fraction (40 g) was subjected to column chromatography eluting with *n*-hexane-EtOAc (0-100%) to obtain sub-fractions: E_A-E_F. The sub-fraction E_C (60 mg) obtained from *n*-hexane-EtOAc (4:6) crystallized from methanol to furnish β-sitosterol (**4**) (40 mg). The fraction E_F (1.1 g) which eluted with EtOAc was rechromatographed using *n*-hexane-EtOAc and EtOAc-MeOH in increasing order of polarity. The fraction which eluted with EtOAc-MeOH (9.5:0.5) (30 mg) showed one major spot on TLC and on further purification through preparative TLC in the same solvent system afforded 6,7-methylenedioxy-8-methoxycoumarin (**5**) (15 mg).

Purunuside A (1)

Light yellow gummy solid; UV $\lambda_{max}^{(MeOH)}$ nm (log ε): 298 (4.07), 262 (3.25), 226 (4.12), NaOAc; 312, 273, 231; IR (KBr) ν_{max} cm^{-1}: 3450, 2700-2800, 1690, 1670, 1600-1610, 1225; $[\alpha]_D^{23}$ +16.0 (c 0.12, MeOH). HRFAB-MS (neg.) *m/z*: 603.1713 $[M-H]^-$ (calcd. 603.1705 for $C_{29}H_{31}O_{14}$); EIMS *m/z*: 442 , 372, 236, 206, 167. ^{1}H-NMR (400 MHz, CD_3OD); *δ*: 7.91 (1H, d, *J* = 15.1 Hz, H-1'''), 6.82 (1H, s, H-8), 6.44 (2H, s, H-2', H-6'), 6.23 (1H, d, *J* = 15.1 Hz, H-2'''), 5.02 (1H, d, *J* = 7.6 Hz, H-1''), 4.53 (1H, dd, *J* = 11.7, 5.0 Hz, H-6''), 3.90 (3H, s, 5-OMe), 3.86 (1H, m, H-4''), 3.82 (1H, m, H-3''), 3.81 (1H, m, H-5''), 3.72 (6H, s, 3'-OMe, 5'-OMe), 3.31 (1H, m, H-2''), 3.25 (2H, s, H-9), 3.15 (1H, dd, *J* = 11.7, 2.1 Hz, H-6''), 1.81 (3H, s, Me); ^{13}C-NMR (100 MHz, CD_3OD); *δ*: 180.0 (C-4), 167.6 (C-3'''), 163.4 (C-7), 162.6 (C-8a), 159.1 (C-2), 157.1 (C-5), 156.2 (C-3', C-5'), 147.2 (C-1'''), 139.6 (C-4'), 134.1 (C-1'), 120.4 (C-3), 116.2 (C-2'''), 110.2 (C-6), 109.4 (C-4a), 105.6 (C-2', C-6'), 105.0 (C-1''), 97.9 (C-8), 78.4 (C-5''), 77.8 (C-3''), 75.5 (C-2''), 71.2 (C-4''), 62.3 (6''), 61.5 (5-OMe), 57.0 (3'-OMe, 5'-OMe), 27.0 (C-9), 17.8 (Me).

Purunuside B (2)

Light yellow gummy solid; UV $\lambda_{max}^{(MeOH)}$ nm (log ε): 259 (3.12), 230 (4.17), NaOAc; 270, 242; IR (KBr) ν_{max} cm^{-1}: 3450, 1690, 1670, 1600-1610, 1225; $[\alpha]_D^{23}$ +14.5 (c 0.12, MeOH). HRFAB-MS (neg.) *m/z*: 645.2183 $[M-H]^-$ (calcd. 645.2176 for $C_{32}H_{37}H_{14}$); EIMS *m/z*: 484, 372, 278, 206, 167. ^{1}H-NMR (400 MHz, CD_3OD); *δ*: 7.91 (1H, d, *J* = 15.3 Hz, H-1'''), 6.67 (1H, s, H-8), 6.33 (1H, d, *J* = 15.3 Hz, H-2'''), 6.31 (2H, s, H-2', H-6'), 5.02 (1H, d, *J* = 7.6 Hz, H-1''), 4.53 (1H, dd, *J* = 11.6, 5.1 Hz, H-6''), 4.20 (1H, dd, *J* = 8.9, 3.0 Hz, H-4'''), 4.13 (1H, dd, *J* = 8.9, 5.3 Hz, H-4'''), 3.91 (3H, s, 5-OMe), 3.86 (1H, m, H-4''), 3.82 (1H, m, H-3''), 3.82 (1H, m, H-5''), 3.72 (6H, s, 3'-OMe, 5'-OMe), 3.32 (1H, m, H-2''), 3.25 (2H, s, H-9), 3.18 (1H, dd, *J* = 11.6, 2.0 Hz, H-6''), 1.81 (3H, s, Me), 1.45 (2H, m, H-5'''), 1.02 (3H, t, *J* = 6.4 Hz, H-6'''); ^{13}C-NMR (100 MHz, CD_3OD); *δ*: 180.0 (C-4), 165.0 (C-3'''), 163.2 (C-7), 162.5 (C-8a), 159.1 (C-2), 157.0 (C-5), 156.2 (C-3', C-5'), 143.4 (C-1'''), 139.6 (C-4'), 134.0 (C-1'), 120.3 (C-3), 116.3 (C-2'''), 110.9 (C-6), 109.2 (C-4a), 105.6 (C-2', C-6'), 105.0 (C-1''), 97.8 (C-8), 78.1 (C-5''), 77.8 (C-3''), 75.4 (C-2''), 71.4 (C-4''), 66.4 (C-4'''), 62.3 (6''), 61.7 (5-OMe), 57.0 (3'-OMe, 5'-OMe). 27.0 (C-9), 21.5 (C-5'''), 17.8 (Me), 11.6 (C-6''').

Purunuside C (3)

Light yellow gummy solid; UV $\lambda_{max}^{(MeOH)}$ nm (log ε): 291 (4.05), 262 (3.25), 226 (4.12), NaOAc; 306, 273, 231; IR (KBr) ν_{max} cm^{-1}: 3450, 1690, 1670, 1600-1610, 1225; $[\alpha]_D^{23}$ +24.0 (c 0.12, MeOH). HRFAB-MS (neg.) *m/z*:

695.1976 $[M-H]^-$ (calcd. 695.1966 for $C_{35}H_{35}H_{15}$); EIMS *m/z*: 534, 442, 372, 328, 206, 167. ^{1}H-NMR (400 MHz, CD_3OD); δ: 7.92 (1H, d, J = 15.1 Hz, H-1'''), 7.91 (2H, d, J = 8.6 Hz, H-5''', H-9'''), 6.94 (2H, d, J = 8.6 Hz, H-6''', H-8'''), 6.67 (1H, s, H-8), 3.25 (2H, s, H-9), 6.44 (2H, s, H-2'- H-6'), 6.23 (1H, d, J = 15.1 Hz, H-2'''), 5.02 (1H, d, J = 7.5 Hz, H-1''), 4.53 (1H, dd, J = 11.7, 5.0 Hz, H-6''), 3.90 (3H, s, 5-OMe), 3.87 (1H, m, H-4''), 3.82 (1H, m, H-3''), 3.79 (1H, m, H-5''), 3.61 (6H, s, 3'-OMe, 5'-OMe), 3.33 (1H, m, H-2''), 3.18 (1H, dd, J = 11.7, 2.1 Hz, H-6''), 1.81 (3H, s, Me), ^{13}C-NMR (100 MHz, CD_3OD); δ: 180.0 (C-4), 163.4 (C-7), 163.1 (C-3'''), 162.5 (C-8a), 159.4 (C-2), 157.0 (C-5), 156.1 (C-3', C-5'), 155.9 (C- 7'''), 146.6 (C-4'''), 143.0 (C-1'''), 139.6 (C-4'), 134.1 (C-1'), 123.6 (C-5''', C-9'''), 120.0 (C-3), 118.2 (C-6''', C-8'''), 116.0 (C-2'''), 111.0 (C-6), 109.4 (C-4a), 105.6 (C-2', C-6'), 105.0 (C-1''), 97.8 (C-8), 78.2 (C-5''), 77.6 (C-3''), 75.4 (C-2''), 71.2 (C-4''), 62.3 (6''), 61.5 (5-OMe), 57.1 (3'-OMe, 5'-OMe), 27.0 (C-9), 17.8 (Me).

Acid hydrolysis of compounds 1-3

A solution of compounds **1**, **2** and **3** (each 4 mg) in MeOH (5 mL) containing 1N HCl (2 mL) was refluxed for 4h, concentrated under reduced pressure diluted with H_2O and extracted with EtOAc. The aqueous phase in each case was concentrated to obtain the glycone which could be identified as D-glucose by the sign of its optical rotation ($[\alpha]_D^{23}$ +51.8, c = 0.02 MeOH) from **1**, ($[\alpha]_D^{23}$ +51.5, c = 0.02 MeOH) from **2** and ($[\alpha]_D^{23}$ +51.4, c = 0.02 MeOH) from **3**, respectively. It was further confirmed through co-TLC with an authentic sample of D-glucose. The aglycone in each case was a mixture of products and could not be worked up due to paucity of material.

Enzyme inhibition assay

The inhibitory activity of the compounds has been determined against α-glucosidase (E.C. 3.2.1.20), from saccharomyces sp. purchased from Wako Pure Chemical Industries Ltd. (Wako 076-02841). The inhibition has been measured spectrophotometrically at pH 6.9 at 37°C using 1 mM *p*-nitrophenyl α-D-glucopyranoside (PNP-G) as a substrate and 0.69 units/mL enzyme, in 50 mM sodium phosphate buffer containing 100 mM NaCl. 1-Deoxynojirimycin (0.425 mM) was used as a positive control (Ali et al., 2002). The increment in absorption at 400 nm due to the hydrolysis of PNP-G by α-glucosidase was monitored continuously with the spectrophotometer (Molecular Devices U.S.A.) (Matsui et al., 1996).

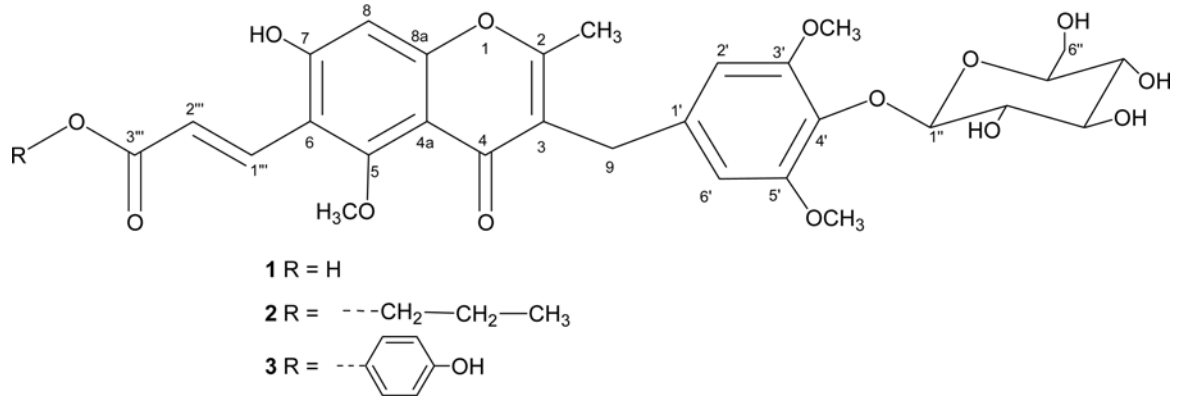
Estimation of IC_{50} values

The concentration of the test compounds which inhibited the hydrolysis of PNP-G by α-glucosidase by 50% (IC_{50}) were determined by monitoring the effect of increasing the concentration of these compounds in the assays of the inhibition values, the IC_{50} values were then calculated using EZ-Fit Enzyme Kinetics program (Perrella Scientific Inc.).

RESULTS AND DISCUSSION

The *n*-BuOH and EtOAc-soluble fractions of the ethanolic extract of the shoots of *P. domestica* were subjected to a series of column chromatographic techniques to obtain compounds **1**-**5** and their structures established by UV, IR, MS and NMR spectroscopy. The compounds **1**-**3** gave violet coloration with ferric chloride and recognized as flavonoidal glucosides from their positive reactions with *Molisch* and *Shinoda* reagents (Shinoda, 1928). All of them on acid hydrolysis provided D-glucose which could be identified through sign of its optical rotation and co-TLC with an authentic sample.

Purunuside A (**1**) (Fig. 1) was isolated as light yellow gummy solid. It gave brisk effervescence with dilute



1 R = H

2 R = ---CH_2—CH_2—CH_3

3 R = ---

Fig. 1. Structures of compounds **1-3**

sodium bicarbonate solution revealing the presence of free carboxylic group. The molecular formula $C_{29}H_{32}O_{14}$ was established by HRFABMS showing $[M-H]^-$ peak at *m/z* 603.1713 (calcd. for $C_{29}H_{31}O_{14}$, 603.1705) having fourteen degrees of unsaturation. The IR spectrum revealed the presence of OH groups (3450 cm^{-1}), carboxylic group (2700-2800 cm^{-1}), conjugated acid carbonyl (1690 cm^{-1}), conjugated ketone (1670 cm^{-1}), conjugate double bond (1600-1610 cm^{-1}) and methoxyl group (1225 cm^{-1}). It showed UV absorption maxima at 298, 262, 226 nm. The UV spectrum did not show any bathochromic shift on addition of $AlCl_3$/HCl showing the absence of hydroxyl groups at C-3 and C-5, respectively. On the other hand, a bathochromic shift was observed on addition of NaOAc revealing the presence of phenolic group at C-7. The ^{1}H-NMR spectrum showed trans olefinic protons at δ 7.91 and 6.23 (1H each, d, J = 15.1 Hz). The two aromatic protons were observed as singlet at δ 6.44. The three methoxyl groups gave singlets at δ 3.90 and 3.72, respectively. The benzylic protons were observed as singlet at δ 3.25. The methyl group appeared as a singlet at δ 1.81. The anomeric proton of the glucose moiety was observed as doublet at δ 5.02 and its larger coupling constant (7.6 Hz) allowed us to assign β-configuration. The methine protons of the glucose moiety were observed in the range of δ 3.81-3.31 while the oxymethylene protons resonated at δ 4.53 (1H, dd, J = 11.7, 5.0 Hz) and 3.15 (1H, dd, J = 11.7, 2.1 Hz) respectively. The ^{13}C-NMR (BB and DEPT) spectra showed twentyone signals comprising four methyl, two methylene, ten methine and thirteen quaternary carbons. The carbonyl carbon of the chromone moiety was observed at δ 180.0 followed by the carbonyl carbon of the carboxylic group at δ 167.6. The olefinic carbons were observed at δ 159.1, 120.4, 147.2 and 116.2, respectively. The benzylic carbon appeared at δ 27.0 and the methoxyl groups gave signals at δ 57.0 and 61.5, whereas the methyl group resonated at δ 17.8. The anomeric carbon resonated at δ 105.0, the oxymethine carbons of the glucose moiety appeared at δ 71.2-78.4 and the oxymethylene carbon was observed at δ 62.3. The data indicated homoisoflavone skeleton which was confirmed by EIMS showing a peak at *m/z* 442 resulting from the loss of sugar unit from the molecular ion peak. A characteristic fragment of homoisoflavonoids at *m/z* 167 revealed the presence of a dimethoxyhydroxybenzyl group (Liu et al., 2009). The EIMS also showed fragments at *m/z* 206 and 236 due to retro Diels-Alder fragmentation, the later fragment revealing the presence of one methoxyl, one hydroxyl and propenoic acid moieties in ring A (Ahmed et al., 2006). The relative positions of various substituents could be ascertained through HMBC correlations illustrated in (Fig. 2). On the basis of these evidences, the structure of purunuside A (**1**) could be assigned as (*E*)-3-[3-(3,5-dimethoxy-4-{[3,4,5-trihydroxy-6-(hydroxymethyl) tetrahydro-2*H*-pyran-2-yl] oxy}benzyl)-7-hydroxy-5-methoxy-2-methyl-4-oxo-4*H*-chromen-6-yl]-2-propenoic acid.

Purunuside B (**2**) was obtained as light yellow gummy solid. The molecular formula was established as $C_{32}H_{38}O_{14}$ by HRFABMS showing $[M-H]^-$ peak at *m/z* 645.2183 (calcd. for $C_{32}H_{37}O_{14}$, 645.2176). The UV and IR spectra were similar to those of **1**. The ^{1}H- and ^{13}C-NMR spectra also showed the common features except the presence of additional signals due to a propyl group [δ 1.02 (3H, t, J = 6.4 Hz), 1.45 (2H, m), 4.20 (1H, dd, J = 8.9, 3.0 Hz) and 4.13 (1H, dd, J = 8.9, 5.3 Hz); δ 66.4 (C-4'''), 21.5 (C-5''') and 11.6 (C-6''')]. Thus compound **2** is a propyl ester of **1**. This could be confirmed through HMBC correlations; the oxymethylene protons showing H-C long range correlations with carbonyl carbon at δ 165.0 and the methyl group at δ 11.6. The rest of the HMBC correlations were similar to those of **1**, in conformity to the assigned structure of purunuside B (**2**) as propyl (*E*)-3-[3-(3,5-dimethoxy-4-{[3,4,5-trihydroxy-6-(hydroxymethyl)tetra-

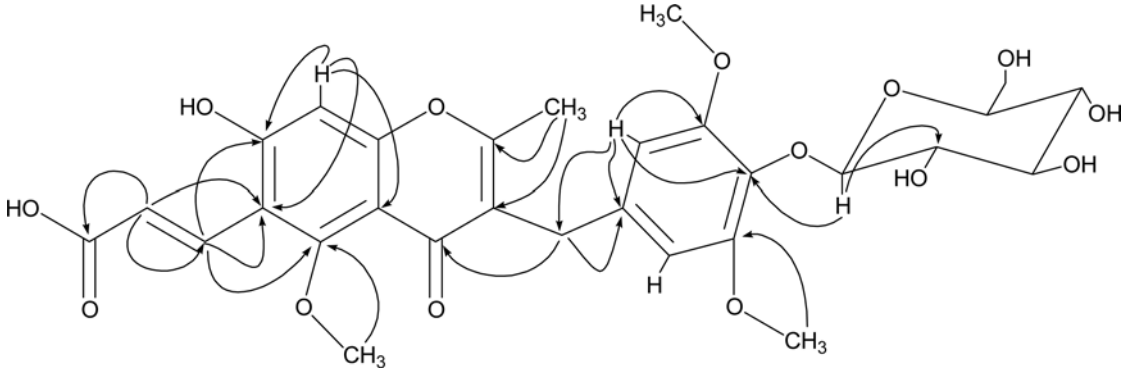


Fig. 2. Important HMBC correlations of compound **1**

hydro-2*H*-pyran-2-yl]oxy}benzyl)-7-hydroxy-5-methoxy-2-methyl-4-oxo-4*H*-chromen-6-yl]-2-propenoate.

Purunuside C (**3**) was obtained as light yellow gummy solid. The molecular formula was established as $C_{35}H_{36}O_{15}$ by HRFABMS showing $[M-H]^-$ peak at *m/z* 695.1976 (calcd. for $C_{35}H_{35}O_{15}$, 695.1966). The UV and IR spectra were similar to those of **1**. The ^{1}H-NMR spectra showed common features to those of **1** except the presence of 4-hydroxyphenyl moiety [δ 7.52 (2H, d, J = 8.6 Hz), 6.94 (2H, d, J = 8.6 Hz); δ 146.6 (C-4'''), 155.9 (C-7'''), 123.6 (C-5''' & C-9'''), 118.2 (C-6''' & C-8''')]. Thus purunuside C (**3**) is 4-hydroxyphenyl ester of **1**. The HMBC correlations were again similar to those of **1** allowing us to assign its structure as 4-hydroxyphenyl (*E*)-3-[3,5-dimethoxy-4-{[3,4,5-trihydroxy-6-(hydroxymethyl) tetrahydro-2H-pyran-2-yl] oxy}benzyl)-7-hydroxy-5-methoxy-2-methyl-4-oxo-4*H*-chromen-6-yl}-2-propenoate.

Non insulin dependent diabetes mellitus (NIDDM) is caused by a secretory decrease in insulin from pancreatic Langerhans β cells or lowering of the insulin resistance. In addition, the long term manifestation of this disease can result in the development of retinopathy, neuropathy, and cataracts and so on. Among the therapeutic drugs to prevent a high blood glucose level, the inhibitors of α-glucosidase (AGH, EC 3.2.1.20), which is a membrane-bound enzyme at the epithelium of the small intestine that catalyzes the cleavage of glucose from disaccharide, are effective for delaying glucose absorption (Matsui et al., 1996). The inhibitors of α-glucosidase could retard the use of dietary carbohydrates to suppress postprandial hyperglycemia, such as acarbose, miglitol and voglibose are well known (Matsuura et al., 2002). It has been demonstrated in an animal model of chronic BHV that glucosidase inhibitors can alter glycosylation and have anti-viral activity. As the mechanism of action of α-glucosidase inhibitors is the induction of misfold or otherwise defective glycoproteins such inhibitors may be useful therapeutics for many viruses, especially those which bud from the endoplasmic reticulum (where protein folding takes place). For example bovine viral diarrhea virus, a pestivirus akin to hepatitis C virus, is also extremely sensitive to glucosidase inhibition (Mehta et al., 1998). α-Glucosidase inhibitors, such as DNJ (deoxynojirimycin), NB-DNJ, are potent inhibitors of human immunodeficiency virus (HIV) replication and HIV-mediated syncytium formation *in vitro* (Fischer et al., 1995). Inhibition of α-glucosidase causes abnormal functionality of glycoproteins because of incomplete modification of glycans. Suppressions of this processing is to be expected for antiviral activity and decreasing of growth rate of tumor (Kurihara et al., 1995). The compounds **1-3** showed potent inhibitory activity against α-glucosidase (Table I). The enhanced activity is obviously due to the presence of glucose moiety at C-4' of the homoisoflavone skeleton. Although the activities were comparable but the compound 3 was the most potent and it may be attributed to 4-hydroxy phenyl ester moiety which probably acts as a pharmacophoric group.

Table I. IC_{50} (μM) values of compounds **1-3** against α-glucosidase

Compound	IC_{50} ± S.E.M. (μM)
1	216.6 ± 0.027
2	268.4 ± 0.047
3	203.6 ± 1.700
Deoxynojirimycin*	281.3 ± 2.800

S.E.M. = standard error of the mean.
*Positive control

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