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山竹果皮中异戊烯基双苯吡酮类成分*

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摘　要：从山竹（*Garcinia mangostana*）的果皮中分离得到22个双苯吡酮类化合物，经波谱数据分析分别鉴定为：1，3，7-三羟基呫酮（**1**）、1，3，6，7-四羟基-8-异戊烯基呫酮（**2**）、1，3，5-三羟基-4-异戊烯基呫酮（**3**）、8-deoxygartanin（**4**）、cudraxanthone G（**5**）、gartanin（**6**）、6-脱氧-γ-倒捻子素（**7**）、γ-倒捻子素（**8**）、α-倒捻子素（**9**）、1，3-二羟基-6，7-二甲氧基-2，8-二异戊烯基呫酮（**10**）、β-倒捻子素（**11**）、garcinone D（**12**）、garcinone B（**13**）、mangostenone D（**14**）、3-*O*-methylmangostenone D（**15**）、9-hydroxycalabaxanthone（**16**）、11-羟基-1-异倒捻子素（**17**）、brasilixanthone B（**18**）、garcimangosxanthone D（**19**）、BR-xanthone A（**20**）、tovophyllin A（**21**）和1，3，6-trihydroxy-2，5-bis（3-methylbut-2-enyl）-6′，6′-dimethyl-4′，5′-dihydropyrano［2，3′：7，8］xanthone（**22**）。其中化合物**2**~**22**为异戊烯基双苯吡酮类，且化合物**3**和**15**属首次从山竹中分离。在小鼠海马神经元HT22细胞上测试了所有化合物对谷氨酸诱导的细胞死亡的保护活性。

关键词：藤黄科（Guttiferae）；山竹（*Garcinia mangostana*）；异戊烯基双苯吡酮；神经保护活性
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Prenylated xanthones from the pericarps of *Garcinia mangostana*

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Abstract：Phytochemical investigation on the pericarps of *Garcinia mangostana* resulted in the isolation of 22 xanthones. On the basis of the spectroscopic data，the structures of these known compounds were identified as 1，3，7-trihydroxyxanthone（**1**），1，3，6，7-tetrahydroxy-8-prenylxanthone（**2**），1，3，5-trihydroxy-4-prenylxanthone（**3**），8-deoxygartanin（**4**），cudraxanthone G（**5**），gartanin（**6**），6-deoxy-γ-mangostin（**7**），γ-mangostin（**8**），α-mangostin（**9**），1，3-dihydroxy-6，7-dimethoxy-2，8-diprenylxanthone（**10**），β-mangostin（**11**），garcinone D（**12**），garcinone B（**13**），mangostenone D（**14**），3-*O*-methylmangostenone D（**15**），9-hydroxycalabaxanthone（**16**），11-hydroxy-1-isomangostin（**17**），brasilixanthone B（**18**），garcimangosxanthone D（**19**），BR-xanthone A（**20**），tovophyllin A（**21**），and 1，3，6-trihydroxy-2，5-bis（3-methylbut-2-enyl）-6′，6′-dimethyl-4′，5′-dihydropyrano［2，3′：7，8］xanthone（**22**）. Compounds **2**-**22** were prenylated xanthones，and **3** and **15** were isolated from this plant

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for the first time. The neuroprotective effects of all compounds against glutamate-induced cell death were tested in murine hippocampal neuronal cell line HT22.

Key words: Guttiferae; *Garcinia mangostana*; prenylated xanthones; neuroprotective activity

Alzheimer's disease (AD) is a chronic neurodegenerative disease characterized by progressive memory loss and cognitive impairment. Oxidative stress caused by the generation of reactive oxygen species (ROS) could lead to neuronal damage inducing cell death, playing an important role in the initiation and progression of AD [1]. Therefore, the development of neuroprotective drugs is considered to be one of the attractive therapeutic strategies for AD patients. In the studies of AD drugs, a growing number of bioactive natural products have been found to be capable of providing neuroprotection against various insults and damage [2]. Flavonoids are a large group of natural polyphenolic compounds with a variety of bioactivities especially anti-inflammatory, anticancer, neuroprotective, and cardiovascular properties [3-5]. They are ubiquitous in plants including herbs, fruits, and vegetables.

Garcinia mangostana L. (Clusiaceae) is a highly valuable plant widely cultivated in tropical regions of Africa and Asia [6]. Its fruits are well-known as pleasant-tasting mangosteen in normal diets, and its pericarps, stem and root barks, and leaves are used as folk medicine for treatment of various diseases such as diarrhea, gonorrhea, and skin rashes [7]. Xanthones, a subclass of flavonoids, are the most characteristic constituents of *G. mangostana*, which exhibit anti-inflammatory, antioxidant, cytotoxic, antimicrobial, and antimalarial activities [7]. Recently, several xanthones isolated from mangosteen shown multifunctional activities against AD [8]. In our continuing search for natural multi-targeted agents for AD [9-10], 22 known xanthones including 21 prenylated ones were isolated from the pericarps of *G. mangostana*. The neuroprotection of these xanthones were evaluated in glutamate-induced HT22 cells, and two compounds exhibited significant neuroprotective activity at a concentration of 1 μmol/L. Herein, the isolation, structural elucidation, and neuroprotection of these xanthones are described.

1 Materials and methods

1.1 Instruments and reagents

NMR spectra were recorded on Bruker AM-400 and AM-500 spectrometers using TMS as an internal standard. MS were recorded on a Finnigan LC Q^{DECA} instrument. A Shimadzu LC-20AT equipped with a SPD-M20A PDA detector was used for HPLC, and a YMC pack ODS-A column (250 mm × 10 mm, S-5 μm, 12 nm) was used for semipreparative HPLC separation. Silica gel (300-400 mesh, Qingdao Haiyang Chemical Co., Ltd.), reversed phase C_{18} ($Rp-C_{18}$) silica gel (12 nm, S-50 μm, YMC Co. Ltd.), MCI gel (CHP20P, 75 - 150 μm, Mitsubishi Chemical Industries Ltd.), and Sephadex LH-20 (Amersham Biosciences) were used for column chromatography (CC). Glutamate was purchased from Research Biochemicals International (Natick, MA, USA). Trypsin, DMSO, and 3-(4, 5-dimethylthiazol-2-yl) -2, 5-diphenyltetrazolium bromide (MTT) were purchased from Sigma-Aldrich (St. Louis, MO, USA). Dulbecco's modified Eagle's medium (DMEM) and fetal bovine serum (FBS) were obtained from Gibco-BRL (Grand Island, NY, USA).

1.2 Plant material

The fruits of *G. mangostana* were purchased from Guangzhou, Guangdong Province, China, in August 2015. The plant was identified by one of the authors (Guihua Tang), and a voucher specimen (H20150801) was deposited at the School of Pharmaceutical Sciences, Sun Yat-sen University.

1.3 Extraction and isolation

After removing the pulps, the air-dried pericarps of *G. mangostana* (5.0 kg) were powdered and extracted with 95% EtOH (15 L) for three times at room temperature. The combined 95% EtOH extracts were concentrated in vacuo to a black residue (500 g), which was suspended in water and then partitioned successively with petroleum ether and EtOAc to give two corresponding portions. The EtOAc extract

（200 g） was subjected to CC over MCI gel eluting with a gradient of increasing MeOH in H_2O （20% – 100%） to yield four fractions （A–D）. Fr. A was subjected to polyamide CC eluting with a gradient of increasing MeOH in H_2O （20%–100%） to gain three fractions （A_1–A_3）. Subsequently， each fraction was subjected to CC over silica gel （CH_2Cl_2–MeOH，15∶1），Sephadex LH–20 （MeOH），Rp–C_{18} column （MeOH–H_2O，40%–100%），and then further purified by semipreparative HPLC （MeOH–H_2O，50%–100%） to yield pure compounds. Compounds **2** （20 mg，t_R 7.0 min） and **1** （33 mg，t_R 8.0 min） were obtained from Fr. A_1. Fr. A_2 gave compounds **8** （360 mg），**13** （30 mg），and **7** （13 mg）. Fr. A_3 gave compounds **12** （80 mg），**17** （36 mg），and **3** （8 mg）. Fr. C was subjected to CC over silica gel using petroleum ether–EtOAc（50∶1 → 0∶1） to gain four fractions （C_1–C_4）. Subsequently， each fraction was subjected to CC over Rp–C_{18} column， silica gel， Sephadex LH–20， and HPLC to yield pure compounds. Compounds **6** （120 mg），**16** （20 mg），and **14** （15 mg） were obtained from Fr. C_1. Compound **10** （35 mg） was yielded from Fr. C_2. Fr. C_3 gave compounds **11** （85 mg），**22** （40 mg），and **21** （23 mg）. Fr. D was chromatographed on a Rp–C_{18} column （MeOH–H_2O，30%–100%） to gain three subfractions （D_1–D_3）. Subsequently， each fraction was subjected to CC over Rp–C_{18} column， silica gel， Sephadex LH–20， and HPLC to yield pure compounds. Fr. D_1 gave compounds **9** （620 mg），**20** （21 mg），**19** （7.5 mg），**15** （16 mg），and **18** （5 mg）. Compounds **4** （40 mg） and **5** （15.4 mg） were yielded from Fr. C_2. The purity of all isolated compounds was estimated to be greater than 95%， as determined by ^{1}H NMR spectra.

1.4　Bioactivity assay

The HT22 cells were maintained in DMEM supplemented with 10% （*V*/*V*） FBS and incubated at 37 ℃ under 5% CO_2. Compounds **1**–**22** were tested for their cytotoxicity in HT22 cells and their protective effects on glutamate induced neuronal death in HT22 cells by using MTT Assay. Briefly， cells were seeded in 96–well plates （1×10^4 cells/well），and six wells were used for each treatment group. The group treated with 0.1% （v/v） DMSO was the vehicle control. After 24 h incubation， HT22 cells were pretreated with different concentrations of compounds for 30 min before exposure to glutamate （2 mmol/L）. Following incubation for 24 h， cell growth was measured at indicated time points by addition of 10 μL of MTT （5 mg/mL） at 37 ℃ for 2 h， and DMSO （100 μL） was added to dissolve the formazan crystals. Optical density was measured using a microplate reader （Bio–Tek， USA） at 570 nm， and all data were represented as fold increase relative to the untreated control.

2　Results and discussion

2.1　Structural identification

The air–dried pericarps of *G. mangostana* were extracted with 95% EtOH. After concentrating the EtOH in vacuo， the black residue was suspended in water and then partitioned with petroleum ether and EtOAc， respectively. The EtOAc fraction was subjected to column chromatography over MCI gel， silica gel， Rp–C_{18}， Sephadex LH–20， and semipreparative HPLC to obtain compounds **1**–**22**.

The structures **1**–**22** （Fig. 1） were determined by comparison of their spectroscopic data with literature data. All compounds （**1**–**22**） were identified as known xanthones. They were 1，3，7–trihydroxyxanthone （**1**），1，3，6，7–tetrahydroxy–8–prenylxanthone （**2**），1，3，5–trihydroxy–4–prenylxanthone （**3**），8–deoxygartanin （**4**），cudraxanthone G （**5**），gartanin （**6**），6–deoxy–γ–mangostin （**7**），γ–mangostin （**8**），α–mangostin （**9**），1，3–dihydroxy–6，7–dimethoxy–2，8–diprenylxanthone （**10**），β–mangostin （**11**），garcinone D （**12**），garcinone B （**13**），mangostenone D （**14**），3–*O*–methylmangostenone D （**15**），9–hydroxycalabaxanthone （**16**），11–hydroxy–1–isomangostin （**17**），brasilixanthone B （**18**），garcimangosxanthone D （**19**），BR–xanthone A （**20**），tovophyllin A （**21**），and 1，3，6–trihydroxy–2，5–bis（3–methylbut–2–enyl）–6′，6′–dimethyl–4′，5′–dihydropyrano［2，3′∶7，8］xanthone （**22**），respectively. Compound **1** is a simple xantone and **2**–**22** repre-

sent the type of prenylated xanthones.

2.1.1 1,3,7-Trihydroxyxanthone (1)

Yellow solid; $C_{13}H_8O_5$; M_r = 244.0; ^{1}H NMR (400 MHz, Methanol-d_4) δ: 7.46 (1H, s, H-8), 7.33 (1H, d, J = 8.0 Hz, H-5), 7.22 (1H, d, J = 8.0 Hz, H-6), 6.29 (1H, s, H-4), 6.15 (1H, s, H-2); ^{13}C NMR (100 MHz, Methanol-d_4) δ: 181.7 (C, C-9), 167.4 (C, C-3), 164.6 (C, C-1), 159.5 (C, C-4a), 155.3 (C, C-7), 151.2 (C, C-10a), 125.2 (CH, C-6), 122.1 (C, C-8a), 119.8 (CH, C-5), 109.4 (CH, C-8), 103.6 (C, C-9a), 99.0 (CH, C-2), 94.8 (CH, C-4). The NMR and MS data were in consistent with those reported in the literature [11-12]. Thus, compound **1** was determined as 1, 3, 7-trihy-

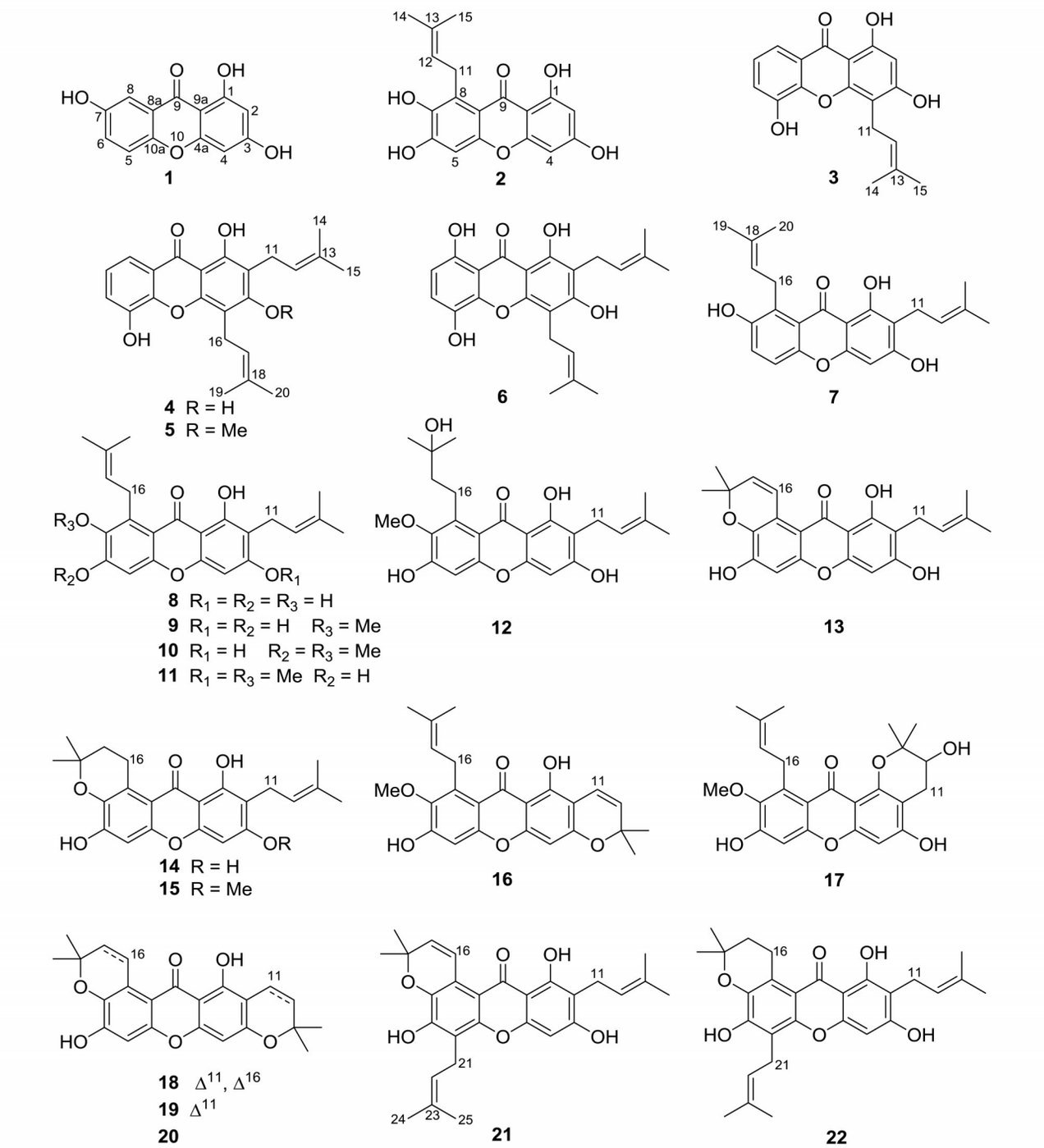

Fig. 1 The chemical structures of compounds **1**-**22**

droxyxanthone.

2.1.2　1，3，6，7-Tetrahydroxy-8-prenylxanthone（2）　Yellow powder；$C_{18}H_{16}O_6$；M_r= 328.1；^{1}H NMR（400 MHz，Methanol-d_4）δ：6.60（1H，s，H-5），6.11（1H，br. s，H-4），6.03（1H，br. s，H-2），5.19（1H，t，J = 6.7 Hz，H-12），4.02（2H，d，J = 6.7 Hz，H-11），1.76（3H，s，H-14），1.59（3H，s，H-15）；^{13}C NMR（100 MHz，Methanol-d_4）δ：183.4（C，C-9），165.5（C，C-3），164.6（C，C-1），158.4（C，C-4a），154.1（C，C-10a），153.5（C，C-6），142.2（C，C-7），131.8（C，C-13），129.4（C，C-8），124.7（CH，C-12），112.0（C，C-8a），104.0（C，C-9a），101.0（CH，C-5），98.5（CH，C-2），93.8（CH，C-4），26.6（CH_2，C-11），26.1（CH_3，C-15），18.3（CH_3，C-14）. Its NMR and MS data were identical with those reported in the literature [13]. Therefore，it was determined to be 1，3，6，7-tetrahydroxy-8-prenylxanthone.

2.1.3　1，3，5-Trihydroxy-4-prenylxanthone（3）　Yellow solid；$C_{18}H_{16}O_5$；M_r= 312.1；^{1}H NMR（400 MHz，Methanol-d_4）δ：7.61（1H，d，J = 7.7 Hz，H-8），7.23（1H，d，J = 7.7 Hz，H-6），7.17（1H，t，J = 7.7 Hz，H-7），6.24（1H，s，H-2），5.34（1H，t，J = 6.7 Hz，H-12），3.53（2H，d，J = 6.7 Hz，H-11），1.85（3H，s，H-14），1.66（3H，s，H-15）；^{13}C NMR（100 MHz，Methanol-d_4）δ 182.5（C，C-9），165.1（C，C-1），162.3（C，C-3），156.0（C，C-4a），147.8（C，C-5），147.1（C，C-10a），132.3（C，C-13），124.6（CH，C-7），123.6（CH，C-6），122.4（C，C-8a），121.2（CH，C-12），116.1（CH，C-8），108.5（C，C-4），103.8（C，C-9a），98.6（CH，C-2），26.0（CH_3，C-15），22.4（CH_2，C-11），18.0（CH_3，C-14）. Its NMR and MS data were in accordance with those of 1，3，5-trihydroxy-4-（3-methylbut-2-enyl）-9*H*-xanthen-9-one [14]. Thus，compound **3** was determined as shown and named 1，3，5-trihydroxy-4-prenylxanthone.

2.1.4　8-Deoxygartanin（4）　Yellow solid；$C_{23}H_{24}O_5$；M_r=380.2；^{1}H NMR（400 MHz，Methanol-d_4）δ：7.57（1H，dd，J = 7.8 and 1.5 Hz，H-8），7.17（1H，dd，J = 7.8 and 1.5 Hz，H-6），7.11（1H，t，J = 7.8 Hz，H-7），5.28（1H，t，J = 7.1 Hz，H-12），5.20（1H，t，J = 7.0 Hz，H-17），3.58（2H，d，J = 7.1 Hz，H-11），3.35（2H，d，J = 7.0 Hz，H-16），1.87 and 1.79（each 3H，s，H-15 and H-20），1.67（6H，s，H-14 and H-19）；^{13}C NMR（100 MHz，Methanol-d_4）δ：182.5（C，C-9），162.3（C，C-3），159.3（C，C-1），153.9（C，C-4a），147.7（C，C-5），146.9（C，C-10a），132.9 and 132.7（each C，C-13 and C-18），124.4（CH，C-7），123.45 and 123.37（each CH，C-12 and C-17），122.4（C，C-8a），120.9（CH，C-6），116.2（CH，C-8），111.7（C，C-2），108.0（C，C-4），103.9（C，C-9a），26.0 and 25.9（each CH_3，C-14 and C-19），22.6 and 22.4（each CH_2，C-11 and C-16），18.1 and 18.0（each CH_3，C-15 and C-20）. Its NMR and MS data were identical with those reported in the literature [15-16]. Hence，it was defined as 8-deoxygartanin.

2.1.5　Cudraxanthone G（5）　Yellow prisms；$C_{24}H_{26}O_5$；M_r=394.2；^{1}H NMR（400 MHz，Methanol-d_4）δ：7.63（1H，dd，J = 7.8 and 1.7 Hz，H-8），7.24（1H，dd，J = 7.8 and 1.7 Hz，H-6），7.18（1H，t，J = 7.8 Hz，H-7），5.31（1H，t，J = 6.9 Hz，H-17），5.24（1H，t，J = 6.8 Hz，H-12），3.80（3H，s，3-OCH_3），3.59（2H，d，J = 6.9 Hz，H-16），3.36（2H，d，J = 6.8 Hz，H-11），1.85 and 1.80（each 3H，s，H-15 and H-20），1.68（6H，s，H-14 and H-19）；^{13}C NMR（100 MHz，Methanol-d_4）δ：183.4（C，C-9），165.1（C，C-3），159.8（C，C-1），154.2（C，C-4a），148.1（C，C-5），147.2（C，C-10a），132.6 and 132.3（each C，C-13 and C-18），124.8（CH，C-7），124.0 and 123.9（each CH，C-12 and C-17），122.4（C，C-8a），121.4（CH，C-6），118.1（C，C-2），116.2（CH，C-8），114.9（C，C-4），106.8（C，C-9a），62.4（CH_3，3-OCH_3），25.94 and

25.87 (each CH_3, C-14 and C-19), 23.5 and 23.3 (each CH_2, C-11 and C-16), 18.1 and 18.0 (each CH_3, C-15 and C-20). The NMR and MS data were in consistent with those reported in the literature [17]. So **5** was elucidated as cudraxanthone G.

2.1.6 Gartanin (**6**) Yellow needles; $C_{23}H_{24}O_6$; M_r=396.2; ^{1}H NMR (400 MHz, Methanol-d_4) δ: 7.14 (1H, d, J = 8.8 Hz, H-7), 6.52 (1H, d, J = 8.8 Hz, H-6), 5.27 (1H, t, J = 6.8 Hz, H-12), 5.20 (1H, t, J = 7.0 Hz, H-17), 3.57 (2H, d, J = 7.0 Hz, H-16), 3.35 (2H, d, J = 6.8 Hz, H-11), 1.87 and 1.81 (each 3H, s, H-15 and H-20), 1.69 (6H, s, H-14 and H-19); ^{13}C NMR (100 MHz, Methanol-d_4) δ: 186.0 (C, C-9), 163.2 (C, C-3), 158.9 (C, C-1), 154.2 (C, C-8), 154.0 (C, C-4a), 145.6 (C, C-10a), 138.6 (C, C-5), 133.0 and 132.7 (each C, C-13 and C-18), 124.2 (CH, C-6), 123.3 and 123.2 (each CH, C-12 and C-17), 112.1 (CH, C-7), 109.7 (C, C-2), 108.6 (C, C-8a), 108.3 (C, C-4), 102.8 (C, C-9a), 26.0 and 25.9 (each CH_3, C-14 and C-19), 22.6 and 22.4 (each CH_2, C-11 and C-16), 18.1 and 18.0 (each CH_3, C-15 and C-20). Its NMR and MS data were identical with those reported in the literature [16]. Therefore, compound **6** was determined to be gartanin.

2.1.7 6-Deoxy-γ-mangostin (**7**) Yellow solid; $C_{23}H_{24}O_5$; M_r=380.2; ^{1}H NMR (400 MHz, Methanol-d_4) δ: 7.18 (1H, d, J = 8.9 Hz, H-6), 7.11 (1H, d, J = 8.9 Hz, H-5), 6.24 (1H, s, H-4), 5.26 (2H, m, H-12 and H-17), 3.30 (4H, m, H-11 and H-16), 1.85 and 1.80 (each 3H, s, H-15 and H-20), 1.68 (6H, s, H-14 and H-19); ^{13}C NMR (100 MHz, Methanol-d_4) δ: 184.4 (C, C-9), 164.2 (C, C-3), 161.6 (C, C-1), 156.5 (C, C-4a), 152.5 (C, C-7), 152.4 (C, C-10a), 131.73 and 131.71 (each C, C-13 and C-18), 129.5 (C, C-8), 124.8 and 123.9 (each CH, C-12 and C-17), 123.5 (CH, C-6), 119.8 (C, C-2), 116.6 (CH, C-5), 111.2 (C, C-8a), 104.2 (C, C-9a), 93.1 (CH, C-4), 26.5 (CH_2, C-16), 26.1 and 26.0 (each CH_3, C-14 and C-19), 22.2 (CH_2, C-11), 18.3 and 17.9 (each CH_3, C-15 and C-20). The NMR and MS data of **7** were in consistent with those reported in the literature [18]. Thus, this compound was assigned as 6-deoxy-γ-mangostin.

2.1.8 γ-Mangostin (**8**) Yellow crystals; $C_{23}H_{24}O_6$; M_r= 396.2; ^{1}H NMR (400 MHz, Methanol-d_4) δ: 6.68 (1H, s, H-5), 6.25 (1H, s, H-4), 5.27 (2H, m, H-12 and H-17), 4.13 (2H, d, J = 6.8 Hz, H-16), 3.32 (2H, d, J = 6.8 Hz, H-11), 1.86 and 1.80 (each 3H, s, H-15 and H-20), 1.68 (6H, s, H-14 and H-19); ^{13}C NMR (100 MHz, Methanol-d_4) δ: 183.5 (C, C-9), 163.3 (C, C-3), 161.5 (C, C-1), 156.3 (C, C-4a), 154.2 (C, C-6), 153.7 (C, C-10a), 142.2 (C, C-7), 131.7 and 131.6 (each C, C-13 and C-18), 129.3 (C, C-8), 124.9 and 124.0 (each CH, C-12 and C-17), 112.0 (C, C-2), 111.1 (C, C-8a), 103.9 (C, C-9a), 100.9 (CH, C-5), 92.9 (CH, C-4), 26.6 (CH_2, C-16), 26.1 and 26.0 (each CH_3, C-14 and C-19), 22.2 (CH_2, C-11), 18.3 and 17.9 (each CH_3, C-15 and C-20). Its NMR and MS data were in accordance with those reported in the literature [16]. Therefore, compound **8** was determined to be γ-mangostin.

2.1.9 α-Mangostin (**9**) Yellow needles; $C_{24}H_{26}O_6$; M_r=410.2; ^{1}H NMR (400 MHz, Methanol-d_4) δ: 6.58 (1H, s, H-5), 6.14 (1H, s, H-4), 5.13-5.26 (2H, m, H-12 and H-17), 3.98 (2H, d, J = 6.3 Hz, H-16), 3.72 (3H, s, 7-OCH_3), 3.23 (2H, d, J = 7.1 Hz, H-11), 1.79 and 1.76 (each 3H, s, H-15 and H-20), 1.64 (6H, s, H-14 and H-19); ^{13}C NMR (100 MHz, Methanol-d_4) δ: 182.9 (C, C-9), 163.3 (C, C-3), 161.4 (C, C-1), 157.5 (C, C-10a), 156.5 (C, C-6), 156.0 (C, C-4a), 144.5 (C, C-7), 138.3 (C, C-8), 131.55 and 131.51 (each C, C-13 and C-18), 125.1 (CH, C-17),

123.9 (CH, C-12), 112.2 (C, C-8a), 111.3 (C, C-2), 103.7 (C, C-9a), 102.7 (CH, C-5), 93.1 (CH, C-4), 61.3 (CH_3, 7-OCH_3), 27.1 (CH_2, C-16), 25.9 (CH_3 × 2, C-14 and C-19), 22.2 (CH_2, C-11), 18.3 and 17.9 (each CH_3, C-15 and C-20). Its NMR and MS data were identical with those reported in the literature [16, 19]. Thus, the structure of **9** was defined as α-mangostin.

2.1.10 1, 3-Dihydroxy-6, 7-dimethoxy-2, 8-diprenylxanthone (**10**) Yellow powder; $C_{25}H_{28}O_6$; M_r=424.2; ^{1}H NMR (400 MHz, Acetone-d_6) δ 13.73 (1H, s, 1-OH), 6.92 (1H, s, H-5), 6.42 (1H, s, H-4), 5.27 (2H, m, H-12 and H-17), 4.12 (2H, d, J = 6.8 Hz, H-16), 4.02 and 3.77 (each 3H, s, 6-OCH_3 and 7-OCH_3), 3.35 (2H, d, J = 7.2 Hz, H-11), 1.82 and 1.78 (each 3H, s, H-15 and H-20), 1.65 (6H, s, H-14 and H-19); ^{13}C NMR (100 MHz, Acetone-d_6) δ: 182.9 (C, C-9), 163.2 (C, C-3), 161.6 (C, C-1), 159.4 (C, C-4a), 156.2 (C, C-10a), 155.7 (C, C-6), 145.1 (C, C-7), 137.3 (C, C-8), 131.4 and 131.3 (each C, C-13 and 18), 124.8 and 123.5 (each CH, C-12 and C-17), 112.2 (C, C-8a), 111.2 (C, C-2), 103.7 (C, C-9a), 99.6 (CH, C-5), 93.2 (CH, C-4), 60.9 and 56.7 (each CH_3, 6-OCH_3 and 7-OCH_3), 26.7 (CH_2, C-16), 26.0 and 25.9 (each CH_3, C-14 and C-19), 22.0 (CH_2, C-11), 18.3 and 17.9 (each CH_3, C-15 and C-20). The NMR and MS data were in consistent with those reported in the literature [20]. Hence, its structure was determined to be 1, 3-dihydroxy-6, 7-dimethoxy-2, 8-diprenylxanthone.

2.1.11 β-Mangostin (**11**) Yellow solid; $C_{25}H_{28}O_6$; M_r=424.2; ^{1}H NMR (400 MHz, CD-Cl_3) δ: 13.44 (1H, s, 1-OH), 7.28 (1H, s, H-5), 6.34 (1H, s, H-4), 5.21-5.30 (2H, m, H-12 and H-17), 4.11 (2H, d, J = 6.1 Hz, H-16), 3.92 and 3.83 (each 3H, s, 3-OCH_3 and 7-OCH_3), 3.37 (2H, d, J = 7.1 Hz, H-11), 1.85 and 1.82 (each 3H, s, H-15 and H-20), 1.714 and 1.705 (each 3H, s, H-14 and H-19); ^{13}C NMR (100 MHz, CD-Cl_3) δ: 181.9 (C, C-9), 163.5 (C, C-3), 159.7 (C, C-1), 155.7 (C, C-4a), 155.2 (C, C-10a), 154.5 (C, C-6), 142.6 (C, C-7), 137.0 (C, C-8), 132.0 and 131.6 (each C, C-13 and C-18), 123.2 and 122.3 (each CH, C-12 and C-17), 112.3 (C, C-8a), 111.5 (C, C-2), 103.8 (C, C-9a), 101.5 (CH, C-5), 88.8 (CH, C-4), 62.0 and 55.8 (each CH_3, 3-OCH_3 and 7-OCH_3), 26.5 (CH_2, C-16), 25.8 (CH_3 × 2, C-14 and C-19), 21.3 (CH_2, C-11), 18.2 and 17.7 (each CH_3, C-15 and C-20). Its NMR and MS data were identical with those reported in the literature [16, 19]. So compound **11** was elucidated as β-mangostin.

2.1.12 Garcinone D (**12**) Yellow solid; $C_{24}H_{28}O_7$; M_r=428.2; ^{1}H NMR (400 MHz, Methanol-d_4) δ: 6.55 (1H, s, H-5), 6.14 (1H, s, H-4), 5.22 (1H, t, J = 7.4 Hz, H-12), 3.80 (3H, s, 7-OCH_3), 3.17-3.31 (4H, m, H-11 and H-16), 1.77 (3H, s, H-15), 1.71 (2H, m, H-17), 1.66 (3H, s, H-20), 1.33 (6H, s, H-14 and H-19); ^{13}C NMR (100 MHz, Methanol-d_4) δ: 182.8 (C, C-9), 163.3 (C, C-3), 161.3 (C, C-1), 157.5 (C, C-6), 156.5 (C, C-10a), 155.9 (C, C-4a), 144.4 (C, C-7), 139.4 (C, C-8), 131.5 (C, C-13), 123.9 (CH, C-12), 112.0 (C, C-8a), 111.2 (C, C-2), 103.6 (C, C-9a), 102.7 (CH, C-5), 93.2 (CH, C-4), 72.1 (C, C-18), 61.5 (CH_3, 7-OCH_3), 45.4 (CH_2, C-17), 29.0 (CH_3 × 2, C-19 and C-20), 26.0 (CH_3, C-14), 23.5 (CH_2, C-16), 22.2 (CH_2, C-11), 17.9 (CH_3, C-15). The NMR and MS data of **12** were in consistent with those reported in the literature [16]. Therefore, its structure was defined as garcinone D.

2.1.13 Garcinone B (**13**) Yellow needles; $C_{23}H_{22}O_6$; M_r=394.1; ^{1}H NMR (400 MHz, Methanol-d_4) δ: 7.99 (1H, d, J = 10.2 Hz, H-16), 6.69 (1H, s, H-5), 6.26 (1H, s,

H-4），5.83（1H，d，J = 10.2 Hz，H-17），5.24（1H，t，J = 7.2 Hz，H-12），3.29（2H，d，J = 7.2 Hz，H-11），1.80（3H，s，H-15），1.68（3H，s，H-14），1.48（6H，s，H-19 and H-20）；^{13}C NMR（100 MHz，Methanol-d_4）δ：182.1（C，C-9），162.4（C，C-3），160.0（C，C-1），155.0（C，C-6），153.0（C，C-10a），152.7（C，C-4a），138.0（C，C-7），131.9（CH，C-17），130.3（C，C-13），122.5（C，C-8），120.8（CH，C-12），120.0（CH，C-16），110.1（C，C-8a），107.4（C，C-2），102.5（C，C-9a），102.2（CH，C-5），91.9（CH，C-4），75.4（C，C-18），25.8（CH_3 × 2，C-19 and 20），24.6（CH_3，C-14），20.8（CH_2，C-11），16.5（CH_3，C-15）. Its NMR and MS data were in accordance with those reported in the literature [21]，suggesting that **12** was identical to garcinone B.

2.1.14 Mangostenone D（**14**） Yellow solid；$C_{23}H_{24}O_6$；M_r=396.2；^{1}H NMR（400 MHz，Methanol-d_4）δ：6.64（1H，s，H-5），6.24（1H，s，H-4），5.25（1H，t，J = 7.1 Hz，H-12），3.45（2H，t，J = 6.7 Hz，H-16），3.29（2H，d，J = 7.1 Hz，H-11），1.89-1.83（2H，m，H-17），1.79（3H，s，H-15），1.68（3H，s，H-14），1.38（6H，s，H-19 and H-20）；^{13}C NMR（100 MHz，Methanol-d_4）δ：183.6（C，C-9），163.4（C，C-3），161.4（C，C-1），156.3（C，C-6），154.4（C，C-10a），154.3（C，C-4a），140.4（C，C-7），131.6（C，C-13），124.0（CH，C-12），122.7（C，C-8），111.7（C，C-8a），111.2（C，C-2），103.9（C，C-9a），101.7（CH，C-5），93.1（CH，C-4），75.4（C，C-18），33.8（CH_2，C-17），26.6（CH_3 × 2，C-19 and C-20），26.0（CH_3，C-14），23.7（CH_2，C-16），22.2（CH_2，C-11），17.9（CH_3，C-15）. Its NMR and MS data were identical with those reported in the literature [22]，indicating that the structure of **14** was the same as mangostenone D.

2.1.15 3-*O*-Methylmangostenone D（**15**） Yellow crystals；$C_{24}H_{26}O_6$；M_r=410.2；^{1}H NMR（400 MHz，$CDCl_3$）δ：13.46（1H，s，1-OH），6.81（1H，s，H-5），6.36（1H，s，H-4），5.25（1H，m，H-12），3.92（3H，s，3-OCH_3），3.53（2H，d，J = 6.8 Hz，H-16），3.37（2H，d，J = 7.1 Hz，H-11），1.90（2H，t，J = 6.8 Hz，H-17），1.81（3H，s，H-15），1.70（3H，s，H-14），1.41（6H，s，H-19 and H-20）；^{13}C NMR（100 MHz，$CDCl_3$）δ：182.5（C，C-9），163.3（C，C-3），159.6（C，C-1），155.4（C，C-4a），153.1（C，C-10a），151.5（C，C-6），138.0（C，C-7），131.6（C，C-13），122.4（CH，C-12），121.4（C，C-8），111.5（C，C-8a），111.3（C，C-2），104.0（C，C-9a），100.3（CH，C-5），88.8（CH，C-4），75.5（C，C-18），55.8（CH_3，3-OCH_3），32.9（CH_2，C-17），26.5（CH_3 × 2，C-19 and C-20），25.8（CH_3，C-14），22.3（CH_2，C-16），21.3（CH_2，C-11），17.8（CH_3，C-15）. The NMR and MS data were in consistent with those reported in the literature [23]. Thus，the structure of **15** was determined to be 3-*O*-methylmangostenone D.

2.1.16 9-Hydroxycalabaxanthone（**16**） Yellow solid；$C_{24}H_{24}O_6$；M_r=408.2；^{1}H NMR（400 MHz，Methanol-d_4）δ：6.54（1H，d，J = 10.0 Hz，H-12），6.53（1H，s，H-5），5.98（1H，s，H-4），5.53（1H，d，J = 10.0 Hz，H-11），5.21（1H，t，J = 6.3 Hz，H-17），3.95（2H，d，J = 6.3 Hz，H-16），3.74（3H，s，7-OCH_3），1.80（3H，s，H-20），1.68（3H，s，H-19），1.42（6H，s，H-14 and H-15）；^{13}C NMR（100 MHz，Methanol-d_4）δ：182.8（C，C-9），160.7（C，C-3），158.7（C，C-1），157.9（C，C-6），157.3（C，C-10a），156.4（C，C-4a），144.9（C，C-7），138.3（C，C-8），131.5（C，C-18），127.9（CH，C-12），125.1（CH，C-17），116.5（CH，C-11），112.0（C，C-8a），105.2（C，C-2），104.3（C，C-9a），102.9（CH，C-5），94.9（CH，C-4），78.8（C，C-13），61.3（CH_3，7-OCH_3），28.7（CH_3 × 2，C-14 and C-15），27.2（CH_2，C-16），26.0（CH_3，C-19），18.4（CH_3，C-20）. Its NMR and MS data were in accordance with those re-

ported in the literature [16], which indicated that **16** have the same structure as 9-hydroxycalabaxanthone.

2.1.17　11-Hydroxy-1-isomangostin (**17**)　Yellow solid; $C_{24}H_{26}O_7$; M_r=426.2; ^{1}H NMR (400 MHz, Methanol-d_4) δ: 6.65 (1H, s, H-5), 6.31 (1H, s, H-4), 5.30 (1H, t, J = 6.2 Hz, H-17), 4.05 (2H, d, J = 6.2 Hz, H-16), 3.79 (1H, dd, J = 7.4 and 5.6 Hz, H-12), 3.75 (3H, s, 7-OCH_3), 2.91 (1H, dd, J = 17.0 and 5.6 Hz, H-11a), 2.56 (1H, dd, J = 17.0 and 7.4 Hz, H-11b), 1.82 (3H, s, H-20), 1.67 (3H, s, H-19), 1.47 (3H, s, H-15), 1.35 (3H, s, H-14); ^{13}C NMR (100 MHz, Methanol-d_4) δ: 178.9 (C, C-9), 162.1 (C, C-3), 158.3 (C, C-4a), 156.7 (C, C-1), 156.1 (C, C-6), 155.6 (C, C-10a), 144.7 (C, C-7), 138.3 (C, C-8), 131.3 (C, C-18), 125.7 (CH, C-17), 114.9 (C, C-8a), 107.5 (C, C-9a), 105.3 (C, C-2), 102.3 (CH, C-5), 94.4 (CH, C-4), 79.5 (C, C-13), 69.6 (CH, C-12), 61.2 (CH_3, 7-OCH_3), 27.1 (CH_2, C-16), 27.0 (CH_2, C-11), 26.0 (CH_3, C-19), 25.6 (CH_3, C-14), 20.6 (CH_3, C-15), 18.3 (CH_3, C-20). The NMR and MS data were in consistent with those reported in the literature [24], which suggested that the structure of **17** was identical to 11-hydroxy-1-isomangostin.

2.1.18　Brasilixanthone B (**18**)　Yellow powder; $C_{23}H_{20}O_6$; M_r=392.1; ^{1}H NMR (400 MHz, $CDCl_3$) δ: 13.61 (1H, s, 1-OH), 8.01 (1H, d, J = 10.2 Hz, H-16), 6.82 (1H, s, H-5), 6.72 (1H, d, J = 10.1 Hz, H-11), 6.25 (1H, s, H-4), 5.82 (1H, d, J = 10.2 Hz, H-17), 5.57 (1H, d, J = 10.1 Hz, H-12), 1.49 (6H, s, H-19 and H-20), 1.47 (6H, s, H-14 and H-15); ^{13}C NMR (100 MHz, $CDCl_3$) δ: 182.4 (C, C-9), 160.0 (C, C-3), 157.8 (C, C-1), 156.5 (C, C-4a), 153.1 (C, C-6), 150.9 (C, C-10a), 136.9 (C, C-7), 132.3 (CH, C-17), 127.2 (CH, C-12), 121.0 (CH, C-16), 119.7 (C, C-8), 115.7 (CH, C-11), 108.6 (C, C-8a), 104.4 (C, C-2), 103.9 (C, C-9a), 102.4 (CH, C-5), 94.3 (CH, C-4), 78.0 (C × 2, C-13 and C-18), 28.3 (CH_3 × 2, C-14 and C-15), 27.3 (CH_3 × 2, C-19 and C-20). Its NMR and MS data were identical with those reported in the literature [25]. Thus, compound **18** was assigned to be brasilixanthone B.

2.1.19　Garcimangosxanthone D (**19**)　Yellow powder; $C_{23}H_{22}O_6$; M_r= 394.1; ^{1}H NMR (400 MHz, $CDCl_3$) δ: 13.72 (1H, s, 1-OH), 6.80 (1H, s, H-5), 6.73 (1H, d, J = 10.0 Hz, H-11), 6.25 (1H, s, H-4), 5.56 (1H, d, J = 10.0 Hz, H-12), 3.49 (2H, t, J = 6.8 Hz, H-16), 1.88 (2H, t, J = 6.8 Hz, H-17), 1.46 (6H, s, H-14 and H-15), 1.39 (6H, s, H-19 and H-20); ^{13}C NMR (100 MHz, $CDCl_3$) δ: 182.6 (C, C-9), 159.6 (C, C-3), 157.8 (C, C-1), 156.5 (C, C-4a), 153.1 (C, C-6), 151.7 (C, C-10a), 138.0 (C, C-7), 127.1 (CH, C-12), 121.3 (C, C-8), 115.7 (CH, C-11), 111.3 (C, C-8a), 104.3 (C, C-2), 103.9 (C, C-9a), 100.5 (CH, C-5), 94.1 (CH, C-4), 77.8 (C, C-13), 75.6 (C, C-18), 32.9 (CH_2, C-17), 28.3 (CH_3 × 2, C-14 and C-15), 26.5 (CH_3 × 2, C-19 and C-20), 22.3 (CH_2, C-16). The NMR and MS data were in consistent with those reported in the literature [26]. Therefore, the structure of **19** was determined as garcimangosxanthone D.

2.1.20　BR-xanthone A (**20**)　Yellow needles; $C_{23}H_{24}O_6$; M_r= 396.2; ^{1}H NMR (400 MHz, $CDCl_3$) δ: 13.73 (1H, s, 1-OH), 6.79 (1H, s, H-5), 6.24 (1H, s, H-4), 3.50 (2H, t, J = 6.7 Hz, H-16), 2.71 (2H, t, J = 6.8 Hz, H-11), 1.88 (2H, t, J = 6.7 Hz, H-17), 1.83 (2H, t, J = 6.8 Hz, H-12), 1.39 (6H, s, H-19 and H-20), 1.37 (6H, s, H-14 and H-15); ^{13}C NMR (100 MHz, $CDCl_3$) δ: 182.6 (C, C-9), 160.5 (C, C-1), 160.4 (C, C-6), 154.9 (C, C-10a), 153.3 (C, C-3), 151.5 (C, C-4a), 137.8 (C, C-7), 121.3 (C, C-8), 111.2 (C, C-2), 103.5 (C, C-8a), 103.0 (C, C-9a), 100.5 (CH, C-5), 94.0 (CH, C-4), 75.9 (C,

C-13), 75.5 (C, C-18), 32.9 (CH_2, C-16), 31.9 (CH_2, C-11), 26.8 and 26.5 (each CH_3 × 2, C-14, C-15, C-19, and C-20), 22.4 (CH_2, C-17), 16.1 (CH_2, C-12). Its NMR and MS data were in accordance with those reported in the literature [27], suggesting that **20** had the same structure as BR-xanthone A.

2.1.21 Tovophyllin A (**21**) Yellow solid; $C_{28}H_{30}O_6$; M_r=462.2; ^{1}H NMR (400 MHz, Acetone-d_6) δ: 13.72 (1H, s, 1-OH), 8.05 (1H, d, J = 10.2 Hz, H-16), 6.51 (1H, s, H-4), 5.88 (1H, d, J = 10.2 Hz, H-17), 5.47-5.18 (2H, m, H-12 and H-22), 3.59 (2H, d, J = 7.4 Hz, H-21), 3.37 (2H, d, J = 7.2 Hz, H-11), 1.89 (3H, s, H-25), 1.80 (3H, s, H-15), 1.66 (6H, s, H-14 and H-24), 1.47 (6H, s, H-19 and H-20). ^{13}C NMR (100 MHz, Acetone-d_6) δ: 183.4 (C, C-9), 163.1 (C, C-3), 161.4 (C, C-1), 155.9 (C, C-4a), 151.8 (C, C-10a), 150.7 (C, C-6), 138.2 (C, C-7), 132.6 (CH, C-17), 132.4 (C, C-23), 131.4 (C, C-13), 123.5 (CH, C-12), 122.3 (CH, C-22), 121.6 (CH, C-16), 118.2 (C, C-8), 116.2 (C, C-5), 111.0 (C, C-2), 108.4 (C, C-8a), 103.7 (C, C-9a), 93.3 (CH, C-4), 76.8 (C, C-18), 27.2 (CH_3 × 2, C-19 and C-20), 25.9 (CH_3 × 2, C-14 and C-24), 23.2 (CH_2, C-21), 22.0 (CH_2, C-11), 18.1 (CH_3, C-25), 17.9 (CH_3, C-15). The NMR and MS data were in consistent with those reported in the literature [28]. Hence, the structure of **21** was defined as tovophyllin A.

2.1.22 1,3,6-Trihydroxy-2,5-bis(3-methylbut-2-enyl)-6′,6′-dimethyl-4′,5′-dihydropyrano[2,3′:7,8]xanthone (**22**) Yellow solid; $C_{28}H_{32}O_6$; M_r=464.2; ^{1}H NMR (400 MHz, Methanol-d_4) δ: 6.29 (1H, s, H-4), 5.24 (2H, m, H-12 and H-22), 3.52 (2H, d, J = 6.6 Hz, H-21), 3.43 (2H, m, H-16), 3.28 (2H, m, H-11), 1.89 (3H, s, H-25), 1.85 (2H, m, H-17), 1.78 (3H, s, H-15), 1.66 (6H, s, H-14 and H-24), 1.38 (6H, s, H-19 and H-20); ^{13}C NMR (100 MHz, Methanol-d_4) δ: 184.0 (C, C-9), 163.4 (C, C-3), 161.3 (C, C-1), 156.3 (C, C-4a), 152.3 (C, C-10a), 151.7 (C, C-6), 139.8 (C, C-7), 132.5 (C, C-13), 131.6 (C, C-23), 124.0 (CH, C-12), 123.1 (CH, C-22), 119.8 (C, C-8), 114.3 (C, C-5), 111.5 (C, C-8a), 111.2 (C, C-2), 103.9 (C, C-9a), 93.1 (CH, C-4), 75.6 (C, C-18), 34.0 (CH_2, C-17), 26.6 (CH_3 × 2, C-19 and C-20), 26.0 (CH_3 × 2, C-14 and C-24), 23.5 (CH_2, C-16), 23.2 (CH_2, C-21), 22.2 (CH_2, C-11), 18.2 (CH_3, C-25), 17.9 (CH_3, C-15). Its NMR and MS data were identical with those reported in the literature [29]. Therefore, compound **22** was elucidated to be 1,3,6-trihydroxy-2,5-bis(3-methylbut-2-enyl)-6′,6′-dimethyl-4′,5′-dihydropyrano[2,3′:7,8]xanthone.

2.2 The results of preliminary screening for potential neuroprotective natural products in glutamate-induced HT22 cells

The isolated compounds were evaluated for their ability to protect neuronal damage in the model of glutamate-induced HT22. Initially, the results of cytotoxicity assay showed that most of the compounds had no obvious cytotoxicity to HT22 cells at a concentration of 30 μmol/L. At a sub-concentration of 10 μmol/L, 11 compounds (**2**, **5-10**, **14**, **16**, **19**, and **22**) exhibited well protective effects on glutamate-induced cell death in HT22 cells (Fig. 2A). Furthermore, these active compounds were subjected to evaluate their activities at the lowest tested concentration of 1 μmol/L, and compounds **6** and **8** still showed neuroprotection (Fig. 2B).

In conclusion, the chemical constituents of *G. mangostana* were studied, and **22** xanthones including **21** prenylated ones were isolated from the pericarps extracts. Compounds **3** and **15** were isolated from this plant for the first time. The neuroprotective effects of all compounds against glutamate-induced cell death were investigated in murine hippocampal neuronal cell line HT22. Among them, compounds **6** and **8** were the most active natural products. These findings sug-

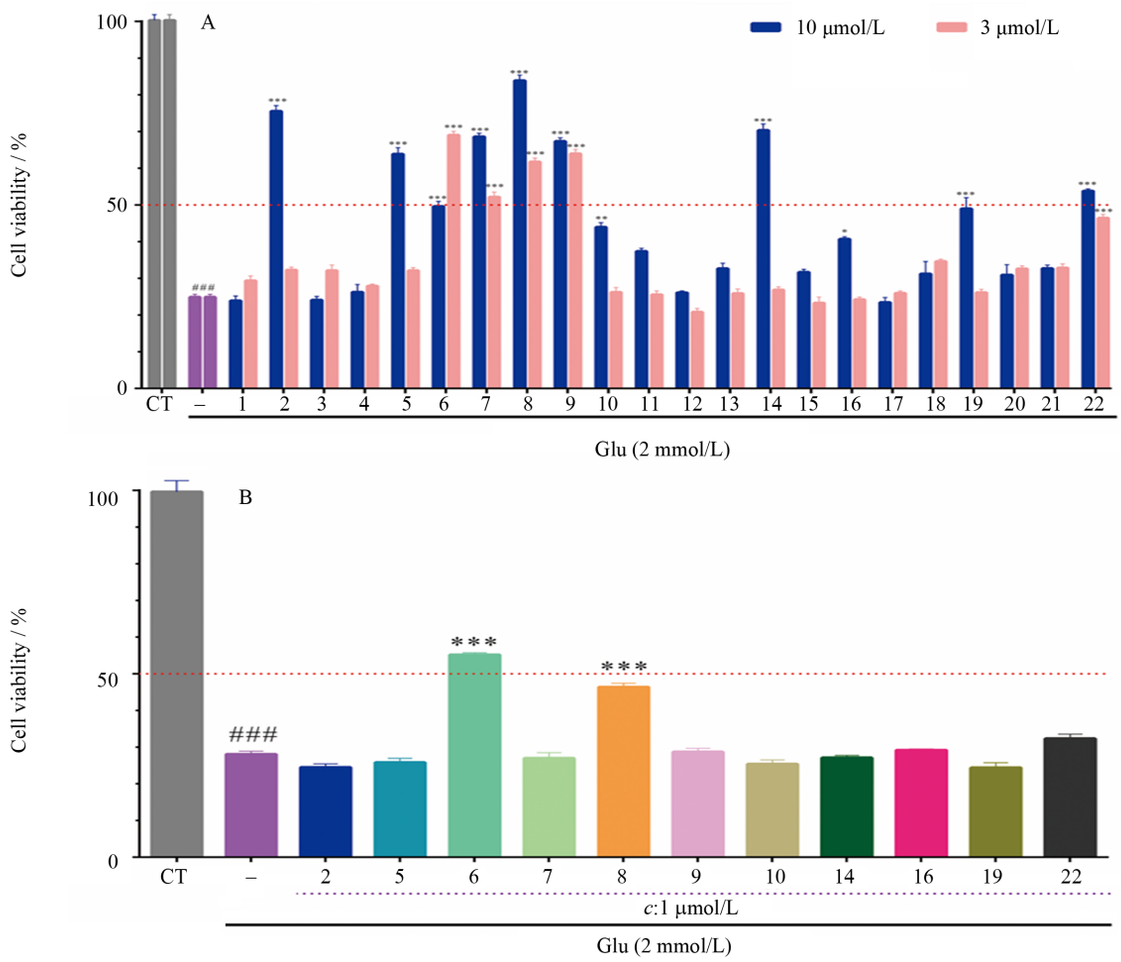

Fig. 2　The neuroprotective effects of the isolated compounds on glutamate-induced cytotoxicity in HT22 cells. Cells were pretreated with or without different compounds at indicated concentrations （A： 10 and 3 μmol/L； B： 1 μmol/L） for 30 min and then incubated with 2 mmol/L glutamate for 24 h. Cell viability was determined by MTT assay. Glu： glutamate. Data are presented as means ± S. D. One-way ANOVA followed by Tukey's test. ###P < 0. 001 vs. control group；
***P < 0. 001， **P < 0. 01， *P < 0. 05 vs. glutamate-treated group

gested that active xanthones might be promising agents for anti-AD drugs development， and also expanded the potential usage of this medicinal-edible plant.

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