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# Cytotoxic ceramides and glycerides from the roots of *Livistona chinensis*

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ABSTRACT

A 70% ethanol extract of the roots of *Livistona chinensis* has been investigated, led to the isolation of 13 compounds, including a new ceramide, (2*S*,3*S*,4*R*,9*Z*)-2-[(2*R*)-2-hydroxytricosanoylamino]-9-octadecene-1,3,4-triol (**2**), a new glycosyl ceramide, 1-*O*-β-D-glucopyranosyl-(2*S*,3*S*,4*R*,9*Z*)-2-[(2*R*)-2-hydroxydocosanoylamino]-9-octadecene-1,3,4-triol (**3**), three new monoacylglycerols, 1-(34-hydroxytetratriacontanoyl)-*sn*-glycerol (**9**), 1-[nonadeca-(9*Z*,12*Z*)-dienoyl]-*sn*-glycerol (**10**), and 1-[12-hydroxypentatriaconta-(13*E*,15*Z*)-dienoyl]-*sn*-glycerol (**11**), a new diacylglycerol, 1-(heptadeca-6*Z*,9*Z*-dienoyl)-3-(octadeca-6*Z*,9*Z*,12*Z*-trienoyl)-*sn*-glycerol (**12**), as well as a new diacylglycerol aminoglycoside, 1-octadecanoyl-2-nonadecanoyl-3-*O*-(6-amino-6-deoxy)-β-D-glucopyranosyl-*sn*-glycerol (**13**). The structures of new compounds were elucidated, based on spectroscopic, zymologic and chemical methods. Among the compounds tested, compounds **3**, **4** and **13** showed significantly antiproliferative effects against the human tumor cell lines (K562, HL-60, HepG2, and CNE-1) with the $IC_{50}$ of 10–65 μM. To our knowledge, this is first report of the occurrence of ceramides and acylglycerols in the genus *Livistona*.



## 1. Introduction

The genus *Livistona* is widely distributed throughout the tropical zone of Asia and Australia. There are three species of this genus growing in South China [1]. Their fruits have traditionally been used for analgesic and hemostatic purposes, and to treat nasopharyngeal carcinoma, choriocarcinoma, esophageal cancer, and leukemia [2,3]. Phytochemically, it has been reported to contain some of flavonoids, steroids, phenolics, fatty acids, amino acids, and vitamins in the fruits of *L. chinensis* [4–9]. The extract from the roots of *L. chinensis* showed significantly antiproliferative effects against human myeloid leukemia cells (L1210), Lymphoid neoplasm (P388D1), gastric cancer cells (SGC7901), cervical cancer cells (HeLa), human liver cancer cells (Hele7404), melanoma cells (B16), and mouse neuroblastomax rat glioma hybrid cells (T24) as well [10]. However, there is no phytochemical study on the roots of *L. chinensis*. In our continuing search for natural bioactive agents from high plants, we fractionated and isolated 13 compounds from the roots of *L. chinensis*, including a known ceramide, (2*S*,3*S*,4*R*)-2-[(2*R*)-2-hydroxytetracosanoylamino]-1,3,4-octadecanetriol (**1**), a known glycosyl ceramide, 1-*O*-β-D-glucopyranosyl-(2*S*,3*S*,4*R*,9*Z*)-2-[(2*R*)-2-hydroxytetracosanoylamino]-1,3,4-octadecanetriol (**4**), a new ceramide (**2**), a new glycosyl ceramide (**3**), four known monoacylglycerols, 1-hexadecanoyl-*sn*-glycerol (**5**), 1-

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octacosanoyl-*sn*-glycerol (**6**), 1-[octadec-(9*Z*)-enoyl]-*sn*-glycerol (**7**) and 1-(26-hydroxyhexacosanoyl)-*sn*-glycerol (**8**), three new monoacylglycerols (**9**–**11**), a new diacylglycerol (**12**), as well as a new diacylglycerol aminoglycoside (**13**), from the chloroform and the ethyl acetate soluble fraction of a 70% EtOH extract. Their chemical structures were given in Fig. 1. Moreover, their cytotoxic activities against four human tumor cell lines (HL-60, K562, HepG2 and CNE-1) were also evaluated. Herein, we report the isolation and structural elucidation of the new compounds (**2**–**3**, **9**–**13**), the identification of known compounds (**1**, **4**–**8**), and the cytotoxic activities of compounds **1**–**13**.

## 2. Experimental

### 2.1. General

Optical rotations were measured using a JASCO P-1030 automatic digital polarimeter (Tokyo, Japan). NMR spectra were recorded on a Bruker DPX-400 spectrometer using standard Bruker pulse programs. Chemical shifts were shown as $\delta$-values with reference to tetramethylsilane (TMS) as an internal standard. GC-MS analysis was done using a Shimadzu GC-14A unit coupled with a GCMS-QP 2000 instrument. ESI-MS data were obtained on an Agilent 1200 HPLC/6410B TripleQuad

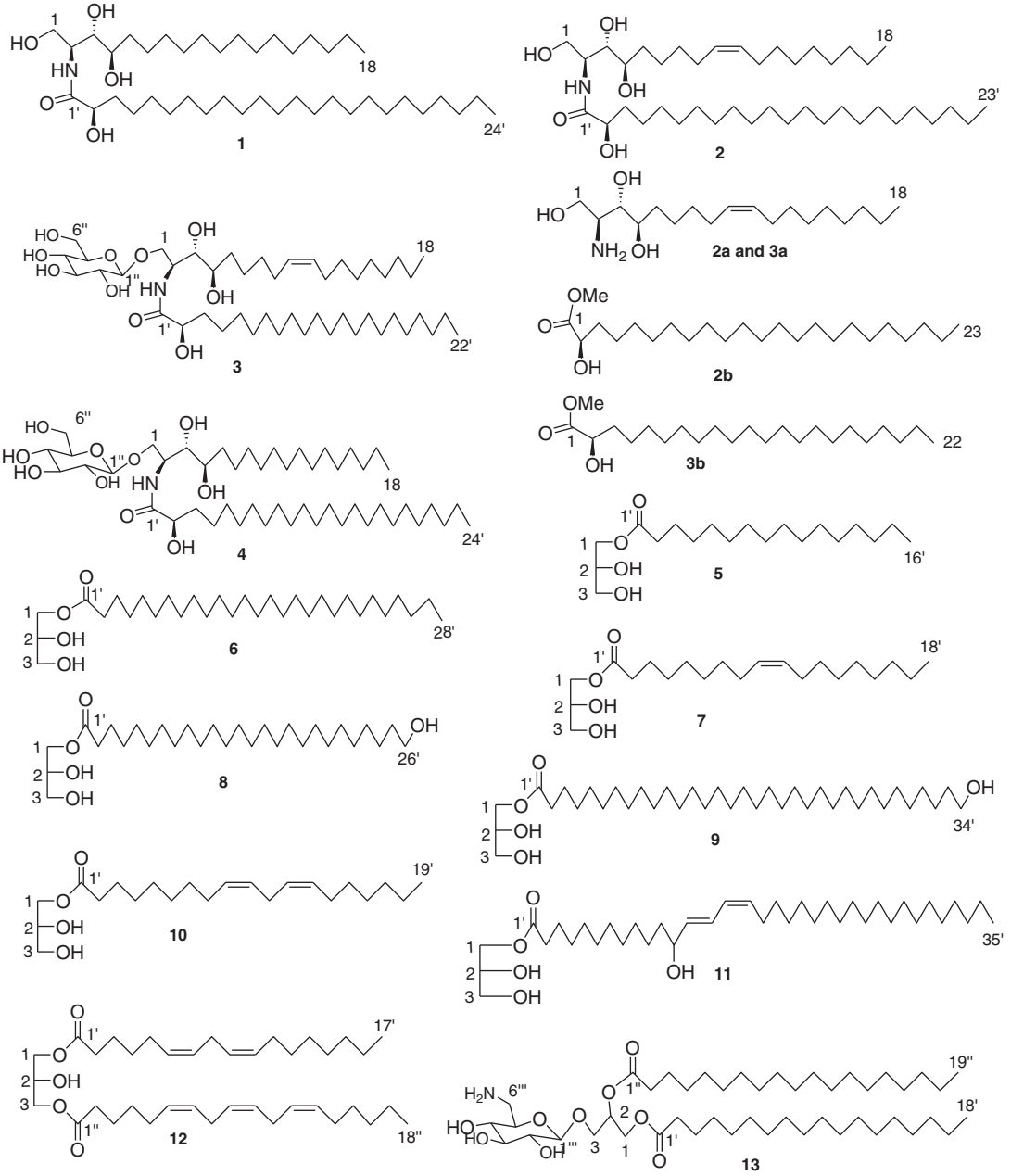


**Fig. 1.** Chemical structures of compounds **1**–**13**.

mass spectrometer (Santa Clara, CA), and HRESIMS were measured on a Bruker APEX II mass spectrometer. TLC was carried out on preparative Silica gel $60F_{254}$ plates (Merck, Darmstadt, Germany), and spots were visualized by spraying the plates with 15% $H_2SO_4$, and then heating them at 105 °C. Sephadex LH-20 (Pharmacia, Sweden), and silica gel (Qingdao Ocean Chemical Co., Ltd, Qingdao, China) were used for column chromatography.

### 2.2. Plant material

The fresh roots of *Livistona chinensis* (Jacq.) R.Br. were collected in Jiangmen, Guangdong Province, China, in September 2009, and were identified by Prof. Xiangjiu He, School of Pharmaceutical Sciences at Wuhan University. A voucher specimen (No. 20100115) is available at the School of Pharmaceutical Sciences, Wuhan University in Wuhan (430071), China.

### 2.3. Extraction and isolation

The air-dried roots of *L. chinensis* (1.5 kg) were extracted with 70% EtOH (25 L×3) at refluxed. Evaporation of the organic solvent under a vacuum at 55 °C yielded a crude extract (560 g). The concentrated brown syrup was resuspended in water (3 L) and partitioned with chloroform (3 L×3), ethyl acetate (3 L×3) and water-saturated n-butanol (3 L×3) gradually to afford 33.0 g, 35.3 g and 230.3 g of dried organic extracts, respectively. The $CHCl_3$ fraction (32.0 g) was fractionated over a silica gel column (800 g, 80×8 cm) using a stepwise gradient elution of cyclohexane-EtOAc (100:1, 8 L), (50:1, 5 L), (33:1, 9 L), (25:1, 15 L), (15:1, 10 L), (10:1, 13 L), (5:1, 8 L), (2:1, 5 L), (1:1, 5 L), (0:1, 5 L). This process yielded 19 fractions (LCC.1-LCC.19). LCC.7 (0.55 g) was separated on a silica gel column (25 g, 28×2 cm), using cyclohexane-ethyl acetate (100:1, 500 mL), (50:1, 300 mL), (30:1, 500 mL), (20:1, 600 mL) to yield compound **5** (66 mg). LCC.8 (1.34 g) was further purified by a silica gel column (50 g, 43×3 cm) and eluted with cyclohexane-ethyl acetate (100:1, 500 mL), (50:1, 600 mL), (30:1, 600 mL), (20:1, 300 mL), (10:1, 300 mL) to obtain compound **7** (50 mg). LCC.9 (2.05 g) was subjected to a silica gel column (65 g, 48×3 cm) and eluted with cyclohexane-ethyl acetate (100:1, 500 mL), (50:1, 1000 mL), (30:1, 1000 mL), (25:1, 500 mL), (20:1, 800 mL), (15:1, 800 mL), (10:1, 800 mL), (5:1, 500 mL), (2:1, 300 mL), (1:1, 200 mL), yielding compounds **1** (30 mg), **8** (250 mg) and **10** (350 mg), respectively. LCC.10 (2.15 g) was further purified with a silica gel column (70 g, 43×3 cm) eluted with $CHCl_3$/MeOH (100:1, 2000 mL), (50:1, 1000 mL), (30:1, 800 mL), followed by a Sephadex LH-20 column (40 g, 60×2 cm) eluted with $CHCl_3$–MeOH (7:3, 500 mL), yielding compounds **6** (65 mg) and **12** (35 mg), respectively. LCC.12 (0.45 g) was subjected to a silica gel column (25 g, 24×2 cm) and eluted with $CHCl_3$–MeOH (100:1, 1000 mL), (50:1, 800 mL), (30:1, 500 mL), (25:1, 600 mL), (20:1, 300 mL), yielding compound **4** (250 mg). LCC.15 (1.25 g) was subjected to a silica gel column (45 g, 30×3 cm) eluted with $CHCl_3$–MeOH (100:1, 1000 mL), (50:1, 1000 mL), (30:1, 800 mL), (25:1, 800 mL), (15:1, 500 mL), (10:1, 500 mL), (5:1, 200 mL), followed by a Sephadex LH-20 column (40 g, 60×2 cm) eluted with $CHCl_3$–MeOH (7:3, 500 mL), yielding compound **13** (100 mg).

The EtOAc fraction (34.3 g) was fractionated over a silica gel column (800 g, 65×8 cm) eluted with $CHCl_3$–MeOH (100:1, 10 L), (50:1, 8 L), (33:1, 9 L), (25:1, 5 L), (20:1, 8 L), (15:1, 5 L), (10:1, 8 L), (5:1, 10 L), (2:1, 7 L), (1:1, 5 L). This process yielded 23 fractions (LCE.1–LCE.23). LCE.3 (0.35 g) was separated on a silica gel column (25 g, 30×1.5 cm), eluted with cyclohexane-ethyl acetate (100:1, 500 mL), (50:1, 300 mL), (25:1, 400 mL), (20:1, 300 mL), (15:1, 300 mL), (10:1, 300 mL) to yield compound **11** (68 mg). LCE.5 (1.35 g) was further purified by a silica gel column (40 g, 38×2 cm) and eluted with $CHCl_3$–MeOH (100:1, 1000 mL), (50:1, 500 mL), (25:1, 500 mL), (15:1, 500 mL), (10:1, 500 mL), (5:1, 500 mL) to obtain compound **9** (150 mg). LCE.6 (1.76 g) was subjected to a silica gel column (60 g, 38×3 cm) and eluted with $CHCl_3$–MeOH (100:1, 1000 mL), (50:1, 800 mL), (25:1, 800 mL), (15:1, 800 mL), (10:1, 500 mL), (5:1, 500 mL), yielding compounds **2** (25 mg) and **3** (120 mg).

*(2S,3S,4R,9Z)-2-[(2R)-2-hydroxytricosanoylamino]-9-octadecene-1,3,4-triol* (**2**) was a white amorphous powder; $[\alpha]^{25}{}_D$ + 10.1 (c 0.20, pyridine); $^1$H NMR ($C_5D_5N$, 400 MHz) δ 8.64 (1H, d, $J$ = 9.2 Hz, NH), 5.53 (2H, m, H-9 and H-10), 5.17 (1H, m, H-2), 4.67 (1H, dd, $J$ = 7.8, 3.6 Hz, H-2′), 4.56 (1H, dd, $J$ = 10.8, 4.4 Hz, H-1a), 4.48 (1H, m, H-1b), 4.41 (1H, dd, $J$ = 6.6, 4.2 Hz, H-3), 4.29 (1H, m, H-4), 2.23–2.38 (4H, m, H-5a, H-8a, H-9a and H-3′a),1.96–2.09 (5H, m, H-5b, H-8b, H-9b, H-3′b and H-4′a), 1.66–1.82 (3H, m, H-6a, H-6b and H-4′b), 1.27–1.33 (methylene band), 0.88 (6H, br t, $J$ = 6.6 Hz, H-18 and H-23′); $^{13}$C NMR data ($C_5D_5N$, 100 MHz) δ 175.4 (C, C-1′), 128.8 (CH CH, C-9 and C-10), 76.8 (CH, C-3), 73.1 (CH, C-4), 72.6 (CH, C-2′), 62.2 ($CH_2$, C-1), 53.1 (CH, C-2), 35.8 ($CH_2$, C-3′), 34.3 ($CH_2$, C-5), 32.3 ($CH_2$, C-16 and C-21′), 29.8–30.5 (methylenes), 26.9 ($CH_2$, C-8 and C-11), 23.1 ($CH_2$, C-17 and C-22′), 14.4 ($CH_3$, C-18 and C-23′); ESI-MS m/z 666 $[M-H]^-$; HR-ESI-MS m/z 668.6570 (calcd. for $C_{41}H_{82}NO_5$, 668.6556).

*1-O-β-D-glucopyranosyl-(2S,3S,4R,9Z)-2-[(2R)-2-hydroxydocosanoylamino]-9-octadecene-1,3,4-triol* (**3**) was a white amorphous powder; $[\alpha]^{25}{}_D$ + 2.3 (c 0.20, pyridine); $^1$H NMR ($C_5D_5N$, 400 MHz) δ 8.56 (1H, d, $J$ = 9.2 Hz, NH), 5.45 (2H, m, H-9 and H-10), 5.27 (1H, br s, H-2), 4.94 (1H, d, $J$ = 7.6 Hz, H-1″), 4.70 (1H, dd, $J$ = 10.6, 6.6 Hz, H-1), 4.57 (1H, dd, $J$ = 7.8, 3.8 Hz, H-2′), 4.56 (1H, m, H-6″), 4.48 (1H, m, H-4″), 4.32 (1H, dd, $J$ = 11.8, 5.4 Hz, H-6″), 4.28 (1H, m, H-2″), 4.15–4.21 (3H, m, H-1, 4 and 3″), 3.99 (1H, t, $J$ = 7.8 Hz, H-2″), 3.85 (1H, br s, H-5″) 2.13–2.28 (4H, m, H-5a, H-8a, H-11a and H-3′a), 1.86–2.08 (5H, m, H-5b, H-8b, H-11b, H-3′b and H-4′a), 1.61–1.81 (3H, m, H-6a, H-6b and H-4′b), 1.23–1.29 (methylene band), 0.88 (6H, t, $J$ = 6.4 Hz, H-18 and H-22′); $^{13}$C NMR data ($C_5D_5N$, 100 MHz) δ 176.1 (C, C-1′), 130.9 (CH CH, C-9 or C-10), 130.7 (CH CH, C-9 or C-10), 106.0 (CH, C-1″), 79.0 (CH, C-3″), 78.9 (CH, C-5″), 76.6 (CH, C-3), 76.3 (CH, C-2″), 72.9 (CH, C-2′),72.9 (CH, C-4), 71.9 (CH, C-4″), 70.8 ($CH_2$, C-1), 63.1 ($CH_2$, C-6″), 52.2 (CH, C-2), 36.0 ($CH_2$, C-3′), 34.4 ($CH_2$, C-5), 32.6 ($CH_2$, C-16 and C-20′), 30.1–30.6 (methylenes), 28.4 ($CH_2$, C-8 or C-11), 28.0 ($CH_2$,C-8 or C-11), 23.4 ($CH_2$, C-17 and C-21′), 14.8 ($CH_3$, C-18 and C-22′); ESI-MS m/z 838 $[M+Na]^+$; HR-ESI-MS m/z 838.6534 (calcd. for $C_{46}H_{89}NO_{10}Na$, 838.6576).

*1-(34-hydroxytetratriacontanoyl)-sn-glycerol* (**9**) was a colorless oil; $C_{37}H_{74}O_5$; $[\alpha]^{25}{}_D$ + 7.1 (c 0.20, pyridine); $^1$H NMR ($C_5D_5N$, 400 MHz) δ 4.75 (1H, dd, $J$ = 11.2, 4.4 Hz, H-1a), 4.67

(1H, dd, $J$ = 11.2, 6.4 Hz, H-1b), 4.47 (1H, m, H-2), 4.15 (2H, br d, $J$ = 5.6 Hz, H-3), 3.90 (2H, t, $J$ = 6.4 Hz, H-34′), 2.37 (2H, t, $J$ = 7.6 Hz, H-2′),1.77 (2H, m, H-3′), 1.65 (2H, m, H-33′), 1.53 (2H, m, H-32′), 1.22–1.34 (methylene band); $^{13}$C NMR data ($C_5D_5N$, 100 MHz) δ 175.0 (C, C-1′), 72.2 (CH, C-2), 68.0 ($CH_2$, C-1), 65.6 ($CH_2$, C-3), 63.3 ($CH_2$, C-34′), 35.7 ($CH_2$, C-2′), 35.1 ($CH_2$, C-33′), 30.7–31.3 (methylenes), 27.8 ($CH_2$, C-32′), 26.6 ($CH_2$, C-3′); ESI-MS m/z 597 $[M–H]^-$; HR-ESI-MS m/z 599.5617 (calcd. for $C_{37}H_{75}O_5$, 599.5589).

*1-[nonadeca-(9Z,12Z)-dienoyl]-sn-glycerol* (**10**) was a colorless oil; $[\alpha]^{25}_D$ + 3.1 (c 0.20, $CHCl_3$); $^1$H NMR ($CDCl_3$, 400 MHz) δ 5.28 (4H, m, H-9′, H-10′, H-12′ and H-13′), 4.11 (2H, m, H-1), 3.98 (1H, t, $J$ = 7.0 Hz, H-2), 3.57 (2H, t, $J$ = 6.6 Hz, H-3), 2.70 (2H, t, $J$ = 6.6 Hz, H-11′), 1.99 (2H, m, H-2′), 1.93–2.01 (4H, m, H-8′ and H-14′), 1.47–1.57 (6H, m, H-3′, H-18′ and H-17′), 1.18–1.23 (methylene band), 0.81 (3H, t, $J$ = 6.8 Hz, H-19′); $^{13}$C NMR data ($CDCl_3$, 100 MHz) δ 174.0 (C, C-1′), 130.2 (CH CH, C-9′, C-10′, C-12′ or C-13′), 130.0 (CH CH, C-9′, C-10′, C-12′ or C-13′), 128.1 (CH CH, C-9′, C-10′, C-12′ or C-13′), 127.9 (CH CH, C-9′, C-10′, C-12′ or C-13′), 68.3 (CH, C-2), 64.9 ($CH_2$, C-1), 63.0 ($CH_2$, C-3), 34.1 ($CH_2$, C-2′), 31.9 ($CH_2$, C-17′), 29.1–29.7 (methylenes), 28.6 ($CH_2$, C-11′), 27.2 ($CH_2$, C-8′and C-14′), 24.9 ($CH_2$, C-3′), 22.7 ($CH_2$, C-18′), 14.1 ($CH_2$, C-19′); ESI-MS m/z 391 $[M + Na]^+$; HR-ESI-MS m/z 391.3589 (calcd. for $C_{22}H_{40}O_4Na$, 391.3513).

*1-[12-hydroxypentatriaconta-(13E,15Z)-dienoyl]-sn-glycerol* (**11**) was a colorless oil; $[\alpha]^{25}_D$ + 10.1 (c 0.20, $CHCl_3$); $^1$H NMR ($CDCl_3$, 400 MHz) δ 6.42 (1H, dd, $J$ = 15.0, 11.0 Hz, H-14′), 5.90 (1H, t, $J$ = 10.8 Hz, H-15′), 5.59 (1H, dd, $J$ = 15.8, 6.6 Hz, H-13′), 5.39 (1H, dd, $J$ = 18.6, 7.8 Hz, H-16′), 4.12 (2H, m, H-1), 4.09 (1H, m, H-12′), 3.87 (1H, m, H-2), 3.61 (1H, m, H-3), 3.55 (1H, m, H-3), 2.25–2.33 (2H, m, H-2′), 2.11 (2H, m, H-17′), 1.97 (2H, m, H-3′), 1.47–1.57 (4H, m, H-11′ and H-34′), 1.18–1.25 (methylene band), 0.81 (3H, t, $J$ = 6.8 Hz, H-35′); $^{13}$C NMR data ($CDCl_3$, 100 MHz) δ 173.4 (C, C-1′), 134.7 (CH CH, C-13′), 132.1 (CH CH, C-16′), 126.7 (CH CH, C-15′), 124.9 (CH CH, C-14′), 71.8 (CH, C-12′), 69.3 (CH, C-2), 64.1 ($CH_2$, C-1), 62.1 ($CH_2$, C-3), 36.3 ($CH_2$, C-11′), 33.2 ($CH_2$, C-2′), 32.7 ($CH_2$, C-18′), 30.9 ($CH_2$, C-19′), 30.5 ($CH_2$, C-20′), 27.9–28.7 (methylenes), 26.7 ($CH_2$, C-17′), 24.3 ($CH_2$, C-3′), 23.9 ($CH_2$, C-10′), 21.5 ($CH_2$, C-34′), 13.1 ($CH_3$, C-35′); ESI-MS m/z 607 $[M–H]^-$; HR-ESI-MS m/z 631.4552 (calcd. for $C_{38}H_{72}O_5Na$, 631.4603).

*1-(heptadeca-6Z,9Z-dienoyl)-3-(octadeca-6Z,9Z,12Z-trienoyl)-sn-glycerol* (**12**) was a colorless oil; $[\alpha]^{25}_D$ + 1.3 (c 0.20, $CHCl_3$); $^1$H NMR ($CDCl_3$, 400 MHz) δ 5.23–5.33 (10H, m, H-6′, H-7′, H-9′, H-10′, H-6″, H-7″, H-9″, H-10″, H-12″ and H-13″), 5.19 (1H, m, H-2), 4.22 (2H, dd, $J$ = 8.2, 3.8 Hz, H-1), 4.08 (2H, dd, $J$ = 11.8, 5.8 Hz, H-3), 2.72 (6H, m, H-8′, H-8″ and H-11″), 2.25 (4H, m, H-2′ and H-2″), 1.93 (8H, m, H-5′, H-11′, H-5″ and H-14″), 1.54 (8H, m, H-3′, H-14′, H-3″ and H-18″), 1.18–1.23 (methylene band), 0.81 (6H, m, H-15′ and H-18′); $^{13}$C NMR data ($CDCl_3$, 100 MHz) δ 173.2 (C, C-1′ or C-1″), 172.8 (C, C-1′ or C-1″), 127.1–132.0 (CH CH, C-6′, C-7′, C-9′, C-10′, C-6′, C-7″, C-9″, C-10″, C-12″ and C-13″), 68.9 (CH, C-2), 62.1 ($CH_2$, C-1 and C-3), 34.2 ($CH_2$, C-2′ or C-2″), 34.0 ($CH_2$, C-2′ or C-2″), 31.5 ($CH_2$, C-13′ and C-16″), 27.9–28.7 (methylenes), 27.2 ($CH_2$, C-5′, C-11′, C-5″ and C-14″), 25.6 ($CH_2$, C-8′, C-8″ and C-11″), 24.8 ($CH_2$, C-3′ and C-3″), 22.6 ($CH_2$, C-16′), 20.6 ($CH_2$, C-17″), 14.2 ($CH_3$, C-17′ or C-18″), 14.1 ($CH_3$, C-17′ or C-18″); ESI-MS m/z 601 $[M + H]^+$; HR-ESI-MS m/z 623.3705 (calcd. for $C_{38}H_{64}O_5Na$, 623.3761).

*1-octadecanoyl-2-nonadecanoyl-3-O-(6-amino-6-deoxy)-β-D-glucopyranosyl-sn-glycerol* (**13**) was a white amorphous powder; $C_{46}H_{89}NO_9$; $[\alpha]^{25}_D$ + 35.1 (c 0.20, $CHCl_3$); $^1$H NMR (DMSO-$d_6$, 400 MHz) δ 5.42 (1H, d, $J$ = 4.0 Hz, -NH), 5.13 (1H, m, H-2), 4.77 (1H, d, $J$ = 4.4 Hz, –OH), 4.66 (1H, d, $J$ = 6.4 Hz, –OH), 4.57 (1H, d, $J$ = 3.2 Hz, H-1‴), 4.35 (1H, dd, $J$ = 12, 2.8 Hz, H-1a), 4.14 (1H, dd, $J$ = 11.8, 7.4 Hz, H-1b), 3.89 (1H, dd, $J$ = 10.4, 6.0 Hz, H-3a), 3.77 (1H, m, H-5‴), 3.39 (2H, m, H-3b and H-3‴), 3.18 (1H, m, H-2‴), 2.91 (2H, m, H-6‴a and H-4‴), 2.53 (1H, m, H-6‴b), 2.27 (4H, m, H-2′ and H-2″), 1.50 (4H, m, H-3′ and H-3″), 1.23 (methylene band), 0.85 (6H, t, $J$ = 6.8 Hz, H-18′ and H-19″); $^{13}$C NMR data (DMSO-$d_6$, 100 MHz) δ 173.0 (C, C-1′ or C-1″), 172.8 (C, C-1′ or C-1″), 98.8 (CH, C-1‴), 74.9 (CH, C-4‴), 73.4 (CH, C-3‴), 72.1 (CH, C-2‴), 70.2 (CH, C-2), 69.0 (CH, C-5‴), 65.2 ($CH_2$, C-3), 63.0 ($CH_2$, C-1), 55.2 ($CH_2$, C-6‴), 34.0 ($CH_2$, C-2′ or C-2″), 33.9 ($CH_2$, C-2′ or C-2″), 31.8 ($CH_2$, C-16′ and C-17″), 28.9–29.5 (methylenes), 24.9 ($CH_2$, C-3′ and C-3″), 22.6 ($CH_2$, C-17′ and C-18″), 14.4 ($CH_3$, C-18′ and C-19″); ESI-MS m/z 834 $[M + Cl]^-$; HR-ESI-MS m/z 834.6171 (calcd. for $C_{46}H_{89}NO_9Cl$, 834.6143).

### 2.4. Methanolysis of compounds (**1**–**4** and **13**)

Each compound (10 mg) was added to a mixture of HCl (10 mL, 1 N) and MeOH (30 mL), with this solution refluxed for 10 h under magnetic stirring. TLC was developed in order to judge whether the reaction was complete. Then, 50 mL $H_2O$ were added to the refluxed mixture, which was extracted with *n*-hexane (3 × 50 mL). One fatty acid methyl ester or two were afforded from the *n*-hexane extract. The MeOH/$H_2O$ phase was evaporated under reduced pressure to yield a sphingosine or a mixture of glycerol and 1-[(6-amino-6-deoxy)-β-D-glucopyranosyl]-methyl ester.

### 2.5. Epoxidation of compouds (**2a**, **3a**, **7**, **10**, **11** and **12**)

A solution of substrate (10 mg) in 3 mL $CH_2Cl_2$ was stirred and cooled in an ice bath as a solution of *m*-CPBA (50 mg) in $CH_2Cl_2$ (5 mL) was added dropwise. The resulting mixture was stirred in the ice bath for an additional 30 min. TLC was developed to judge whether the reaction was complete. The mixture was washed with 10% $Na_2CO_3$ (5 × 6 mL) and saturated NaCl solution (10 mL). The organic layer was dried ($Na_2SO_4$) and the solvent was removed on a rotary evaporator to give a viscous oil, and GC–MS was performed to determine the position of the double bond.

### 2.6. Enzymatic hydrolysis of **13**

Compound **13** (10 mg) were dissolved in 10 mL of dioxane-$H_2O$ (1:1) and treated with lipase enzyme type III (2 mg, 46 U, from a *Pseudomonas* species, lot 093 K0698, Sigma-Aldrich) at 37 °C, with shaking for 8 h. TLC was developed in order to judge whether the reaction was complete. The reaction mixtures were quenched with 5% acetic acid (2.5 mL), and the product was dried under reduced pressure. The crude residues were dissolved in water and extracted with $CHCl_3$, concentrated under reduced pressure, and analyzed by ESI-MS.

### 2.7. Antiproliferative assay

The antiproliferative assay was performed on four human tumor cell lines, namely human myeloid leukemia HL-60, human myeloid leukemia K562, human liver cancer HepG2 and human nasopharyngeal carcinoma CNE-1, performed according to a reported protocol [11]. All the cells were cultured in RPMI-1640 medium (Hyclone, Logan, UT), and supplemented with 10% fetal bovine serum (Hyclone) and antibiotic (100 units/mL penicillin and 100 μg/mL streptomycin) in 5% $CO_2$ at 37 °C. The cytotoxicity assay was performed according to the MTT method in 96-well microplates. Briefly, 200 μL of adherent cells were seeded into each well of 96-well cell culture plates and allowed to adhere for 12 h before drug addition, while suspended cells were seeded just before drug addition with an initial density of $5\times10^4$ cells/mL. Each tumor cell line was exposed to the test compound at concentrations of 100, 50, 25, 12.5, 6.25 μg/mL in quadruples for 48 h.

## 3. Results and discussion

### 3.1. Structure elucidation of new compounds 2–3, 9–13

Compound **2** was isolated as a white powder. The $^1$H NMR spectrum of **2** displayed a downfield doublet at $\delta_H$ 8.64 (1H, d, $J=9.2$ Hz, NH), eight olefinic, oxygenated or other heteroatomized protons between $\delta_H$ 4.29 to 5.53, as well as the signals of a very strong aliphatic methylene band at 1.27-2.38 and six methyl protons at $\delta_H$ 0.88 (6H, m, H-18 and H-23′). The $^{13}$C NMR and HMQC spectral data of **2**, showing a carbonyl group at $\delta_C$ 175.4 (C-1′), one double bond at $\delta_C$ 128.8 (C-9 and C-10), five oxygenated or other heteroatomized carbons at $\delta_C$ 72.6 (C-4), 76.8 (C-3), 53.1 (C-2), 62.2 (C-1) and 73.1 (C-2′), aliphatic methyllenes between $\delta_C$ 23.1–35.8, and two methyl groups at $\delta_C$ 14.4 (C-18 and C-23′), supporting the above analyzed. The downfield doublet at $\delta_C$ 8.64 (NH) had no correlation to any carbon in the HMQC spectrum. On the other hand, a correlation from $\delta_H$ 8.64 (NH) to $\delta_H$ 5.17 (1H, m, H-2), and the correlations from $\delta_H$ 8.64 (NH) to $\delta_C$ 175.4 and 53.1 were observed in the $^1$H-$^1$H COSY and HMBC spectra, respectively. These 1D NMR data and 2D NMR correlations established the presence of an amide function in the molecule, and suggested **2** is a ceramide. The chemical shifts of the allylic methylene carbons in this compound were assigned at $\delta_C$ 26.9 (C-8 and C-11), based on the clearly observed HMBC correlations from the olefinic signals at $\delta_H$ 5.53 (2H, m, H-9 and H-10) to these two carbon signals. Since the chemical shifts of allylic methylene carbons are different when alkene double bonds are *trans*-oriented ($\delta_C>30$ ppm) compared with *cis*-oriented ($\delta_C<28$ ppm) [12], the double bond in **2** was assigned as *Z*-configuration. The negative ESI-MS spectrum of **2** showed molecular ion $[M-H]^-$ at m/z 666. In order to determine the position of the double bond, and the absolute configuration of **2**, the acid methanolysis method [13] was used. A sphingosine (**2a**) and a fatty acid methyl ester (**2b**) were obtained by methanolysis of **2**. Oxidation of the sphingosine (**2a**) with *m*-chloroperoxybenzoic acid (*m*-CPBA) afforded the epoxy derivative **2′a** (Fig. 2) [14]. After the usual workup, the compound **2′a** was analyzed by GC-MS. It found some fragments at 331, 177, 156, 114, 219 and 44. From the data above, the position of the double bone in the long chain base was determined at C-9. By comparison with the literature value of synthetic sphingamines [13], the obtained optical

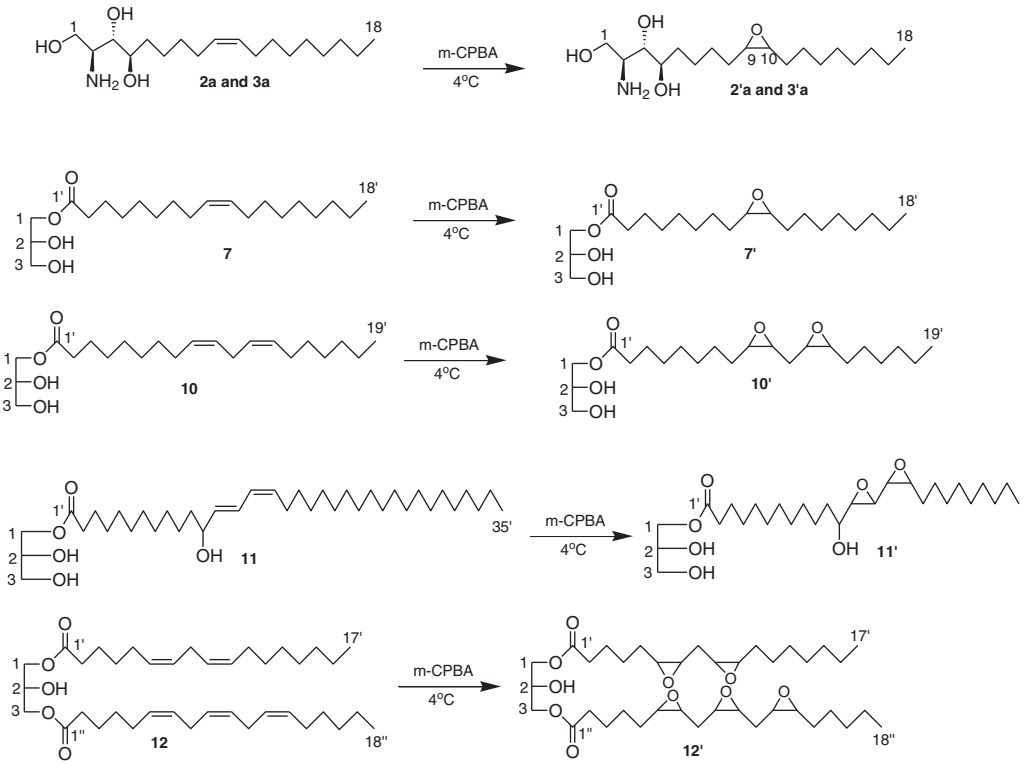


**Fig. 2.** Oxidative reaction of compounds **2a**, **3a**, **7**, **10**, **11** and **12**.

rotation value of **2a** {$[\alpha]_D^{20} = +18.5°$ (c 0.25, $CHCl_3$)} indicated an absolute configuration of 2*S*, 3*S*, and 4*R* for **2**. In the same manner, the absolute configuration at C-2 of the 2-hydroxy fatty acid was presumed to be *R*-configuration from the specific rotation of fatty acid methyl ester (**2b**) {$[\alpha]_D^{20} = -6.2°$ (c 0.25, $CHCl_3$)} [13]. In light of the above evidence, the structure of **2** was deduced to be (2*S*,3*S*,4*R*,9*Z*)-2-[(2*R*)-2-hydroxytricosanoylamino]-9-octadecene-1,3,4-triol.

Compound **3** was isolated as white powder. The HR-ESI-MS suggested a molecular formula of $C_{46}H_{89}NO_{10}$. A comparison of the NMR data between compounds **3** and **2** indicated that the proton signals of the fatty acid portion were similar in these two structures, while the $^1H$ NMR spectrum of **3** displayed fifteen olefinic, oxygenated or other heteroatomized protons between $\delta_H$ 3.8 to 5.5, more than that of **2**. And from the $^{13}C$ NMR of compounds **2** and **3**, it found that there were six more carbon signals between 52.2 and 106.0 in compound **3** than that of **2**. These data suggested the presence of glucopyranose in compound **3**. And the signal of the anomeric proton appeared at 4.94 as a doublet (1H, d, $J_{1,2} = 7.6$ Hz, diaxial) and other glucose protons assigned suggested this glucopyranose was a β-D-glucopyranose. The positive ESI-MS spectrum of **3** showed molecular ion $[M + Na]^+$ peaks m/z 838 and fragment ion m/z 500 [glucosyl-long chain base + Na]$^+$. The presence of a fragment ion at m/z 500 and the identification of a fatty acid residue indicated that **3** is composed of a common long-chain base [glucosyl-(2-amino-octadecene-1,3,4-triol) + Na]$^+$, linked to a fatty acid residue (2-hydroxydocosanoyl). The obtained optical rotation value of **3a** {$[\alpha]_D^{20} = +17.9°$ (c 0.25, $CHCl_3$)} indicated an absolute configuration of 2*S*, 3*S*, and 4*R* for **3**. In the same manner, the absolute configuration at C-2 of the 2-hydroxy fatty acid was presumed to be *R*-configuration from the specific rotation of fatty acid methyl ester (**3b**) {$[\alpha]_D^{20} = -7.2°$ (c 0.25, $CHCl_3$)}. Oxidation of the sphingosine (**3a**) with *m*-chloroperoxybenzoic acid (*m*-CPBA) afforded the epoxy derivative **3′a** (Fugure 2). After the usual workup, the compound **3′a** was analyzed by GC-MS. The fragments at 331, 177, 156, 114, 219 and 44 were also found. Hence, the position of the double bone in the long chain base was determined at C-9. The foregoing evidence led to the determination of **3** as 1-*O*-β-D-glucopyranosyl-(2*S*,3*S*,4*R*,9*Z*)-2-[(2*R*)-2-hydroxydocosanoylamino]- 9-octadecene-1,3,4-triol.

Compound **9** was isolated as colorless oil. The HR-ESI-MS suggested a molecular formula of $C_{37}H_{74}O_5$. Comparing the NMR data with compound **8**, it found that the proton signals of the fatty acid portion were similar in these two structures, with the exception the number of proton signals at 1.22–1.77 ppm. Hence, the structure of **9** was established as 1-(34-hydroxytetratriacontanoyl)-*sn*-glycerol.

Compound **10** was isolated as colorless oil. The HR-ESI-MS suggested a molecular formula of $C_{22}H_{40}O_4$. A comparison of the NMR data between compounds **10** and **6** indicated that the proton signals of the fatty acid portion were similar in these two structures, with the exception of the number and assignments of proton signals 1.18–2.70 and the signals 5.28 (4H, m, CH CH) in these two compounds. The chemical shifts of the allylic methylene carbons were assigned at $\delta_C$ 27.2 (C-8′ and C-14′) and 28.6 (C-11′) based on the clearly observed HMBC correlations from the olefinic signals at $\delta_H$ 5.28 (4H, m, H-9′, H-10′, H-12′ and H-13′) to these three carbon signals. Therefore, the double bonds were assigned in the *Z*-configuration. Oxidation of the sphingosine (**10**) with *m*-chloroperoxybenzoic acid (*m*-CPBA) afforded the epoxy derivative **10′** (Fig. 2). After the usual workup, the compound **10′** was analyzed by GC-MS. Some fragments at 400, 316, 86, 260,142, 274, 128, 100 and 44 were found from GC-MS. From the data above, **10** was identified as 1-[nonadeca-(9*Z*,12*Z*)-dienoyl]-*sn*-glycerol.

Compound **11** was isolated as colorless oil and the negative ESI-MS gave an ion peaks at m/z 607 $[M-H]^-$, corresponding to a molecular formula $C_{38}H_{72}O_5$, which was supported by its NMR data and HR-ESI-MS. Assignment of all the $^1H$ and $^{13}C$ NMR signals was taken for two spin systems. The first spin system was assigned to be a glycerol moiety [$\delta_H$ 3.55 and 3.61 ($\delta_C$ 62.1); $\delta_H$ 3.87 ($\delta_C$ 69.3); $\delta_H$ 4.09 and 4.12 ($\delta_C$ 64.1)]. And, the second group of signals was attributable to one long unsaturated fatty acid ester chain, where one terminal methyl characteristic signal was ascribed $\delta$ 0.81 (3H, m) and the carbonyl carbon signal of the fatty ester group was clearly observed [$\delta$ 173.4 (C-1′)]. The correlations from 5.59 (1H, dd, $J = 15.8$, 6.6 Hz, H-13′) to 6.42 (1H, dd, $J = 15.0$, 11.0 Hz, H-14′) and 4.09 (1H, m, H-12′), and from 5.90 (1H, t, $J = 10.8$ Hz, H-15′) to 6.42 (1H, dd, $J = 15.0$, 11.0 Hz, H-14′) and 5.39 (1H, dd, $J = 18.6$, 7.8 Hz, H-16′), were observed in the $^1H$-$^1H$ COSY spectrum. Hence, one partial structure (−CHOHCH CHCH CH–) was feasibly deduced. To determine the position of the double bonds, Oxidation of the sphingosine (**11**) with *m*-chloroperoxybenzoic acid (*m*-CPBA) afforded the epoxy derivative **11′** (Fig. 2). After the usual workup, the compound **11′** was analyzed by GC–MS. From GC–MS, it found some fragments at 640, 374, 352, 332, 310, 290, 268, 86 and 44. The chemical shifts of the allylic methylene carbons were assigned at $\delta_C$ 27.9 (C-17′) and 71.8 (C-12′) based on the clearly observed $^1H$-$^1H$ COSY and HMBC correlations from the olefinic signals at $\delta_H$ 5.39 (1H, dd, $J = 18.6$, 7.8 Hz, H-16′) and 5.59 (1H, dd, $J = 15.8$, 6.6 Hz, H-13′) to these two carbon signals. The double bond in C-15′ was assigned as *Z*-configuration. Since the coupling constant of alkene proton (−COHCH = C-) is different when alkene double bond is *trans*-oriented ($J \approx 13.6$ Hz) compared with *cis*-oriented ($J \approx 6.2$ Hz) [15]. And, the coupling constant of the alkene methylene proton (H-13′) of **11** was 15.6 Hz. Hence, the double bond in C-13′ was assigned in the *E*-configuration. From the data above, **11** was identified as 1-[12-hydroxypentatriaconta-(13*E*,15*Z*)-dienoyl]-*sn*-glycerol.

Compound **12** was isolated as colorless oil and gave a quasi molecular ion peak at m/z 623 $[M + Na]^+$ in the positive ESI-MS, and two fragment ions at m/z 222 [$C_{17}H_{30}O$–CO] and 234 [$C_{18}H_{30}O$–CO] in GC–MS, corresponding to a molecular formula of $C_{38}H_{64}O_5$, which was supported from its NMR and HR-ESI-MS data. Assignment of all the $^1H$ and $^{13}C$ NMR signals taken in $CDCl_3$ for two spin systems were shown. The first spin system was assigned to be a glycerol moiety [$\delta_H$ 4.08 and 4.22 ($\delta_C$ 62.1); $\delta_H$ 4.08 and 4.22 ($\delta_C$ 62.1); $\delta_H$ 5.19 ($\delta_C$ 68.9)]. And, the second group of signals was attributable to two long unsaturated fatty acid ester chains, where two terminal methyl characteristic signals were ascribed at $\delta$ 0.81 (6H, m) and the carbonyl carbon signals of the two fatty ester groups were clearly observed [$\delta$ 172.8 (C-1′ or C-1″) and $\delta$ 173.2 (C-1′ or C-1″)]. In this compound, the glyceroyl group was also deduced by analyzed its $^1H$-$^1H$ COSY and HMQC spectral data. The two fatty acid ester groups were assigned to C-1 and C-3, respectively, based on the presence of HMBC correlations of H-1 and H-3 to the carbonyl carbons (C-1′ and 1″) of the fatty acid

ester groups. The chemical shifts of the allylic methylene carbons were assigned at $\delta_C$ 25.6 and 27.2, based on the clearly observed HMBC correlations from the olefinic signals at $\delta_H$ 5.23–5.33 (10 H, m) to these seven carbon signals. Therefore, the double bonds in **12** were assigned in the *Z*-configuration. In order to determine the position of the double bond, Oxidation of the sphingosine (**12**) with *m*-chloroperoxybenzoic acid (*m*-CPBA) afforded the epoxy derivative **12′** (Fig. 2). After the usual workup, the compound **12′** was analyzed by GC–MS. It found a series of fragments at 680, 610, 582, 568, 554, 540, 526, 512, 498, 484, 456, 226, 198, 184, 170, 156, 142, 128, 114, 100 and 72 from GC–MS spectrum. Therefore, it can be concluded these two fatty acids were named heptadeca-(6*Z*,9*Z*)-dienoic acid and octadeca-(6*Z*,9*Z*,12*Z*)-trienoic acid, respectively. From the data above, compound **12** was identified as 1-(heptadeca-6*Z*,9*Z*-dienoyl)-3-(octadeca-6*Z*,9*Z*,12*Z*-trienoyl)-*sn*-glycerol.

Compound **13** was isolated as a white amorphous powder and the molecular formula of $C_{46}H_{89}NO_9$ was drawn from its HR-ESI-MS. Assigned all the $^1H$ and $^{13}C$ NMR signals taken in DMSO-$d_6$, three spin systems were shown. The first spin system was assigned to be a glycerol moiety [$\delta_H$ 4.14 and 4.35 ($\delta_C$ 63.0); $\delta_H$ 3.89 and 3.39 ($\delta_C$ 65.2); $\delta_H$ 5.13 ($\delta_C$ 70.2)]. The second group of signals was attributable to two long saturated fatty acid ester chains, where two terminal methyl characteristic signals were ascribed at $\delta$ 0.85 (6H, t, $J = 6.8$ Hz) and the carbonyl carbon signals of the two fatty ester groups were clearly observed [$\delta$ 172.8 (C-1′ or C-1″) and $\delta$ 173.0 (C-1′ or C-1″)]. The third spin system indicated the presence of a glycosyl moiety. The glyceroyl and glycosyl moieties in **13** were also deduced by analysis of the $^1H$-$^1H$ COSY, HMQC and HMBC spectra. **13** was therefore deduced to be a glyceroglycolipid. The glycosyl moiety was shown to be a six-carbon aminosugar unit according to its NMR data. Compared with the reported value [16], the glycosyl moiety in **13** had identical $^{13}C$ NMR data as those reported for a 6-amino-6-deoxy-β-glucopyranosyl moiety. This finding was confirmed by a HMBC experiment. The HMBC spectrum showed the aminosugar unit was attached to C-3, due to the presence of a long-range correlation between H-3 and the anomeric carbon signal of the aminosugar unit. The two fatty acid ester groups were assigned to C-1 and C-2, respectively, based on the presence of HMBC correlations of H-1 and H-2 to the carbonyl carbons (C-1′ and 1″) of the fatty acid ester groups. **13** was thus determined to be a diacylglyceryl aminoglycoside. To deduce the exact structures of the fatty acids, it was hydrolyzed in methanol/NaOMe (2 h). After the usual workup, the nonpolar organic extract was analyzed by ESI-MS, and two ion peaks $[M+H]^+$ at m/z 299 and 313 were observed, corresponding to stearic acid methyl ester and nonadecanoic acid methyl ester, respectively. The MeOH phase was subjected to acid methanolysis, and after HPLC purification, methyl 6-deoxy-6-amino-glucopyranoside was obtained. The D-configuration of the sugar was deduced by comparing its optical rotation ($[a]_D^{25} + 113.0°$ in $H_2O$) with the reported data of an authentic methyl 6-deoxy-6-amino-β-D-glucopyranoside ($[a]_D^{25} + 147.0°$ in $H_2O$) [17]. In order to determine the location of the two fatty acid ester chains, a selective deacylation experiment was performed. Treatment with lipase type III [18], only octadecanoic acid was obtained analyzed by ESI-MS. Therefore, the octadecanoyl residue was attached to the C-1 position of the glycerol moiety in **13**. Accordingly, **13** was determined to be 1-octadecanoyl-2-nonadecanoyl-3-*O*-(6-amino-6-deoxy)-β-D-glucopyranosyl-*sn*-glycerol.

Compared their ESI-MS and NMR data with the literature, the known compounds were identified as (2*S*,3*S*,4*R*)-2-[(2*R*)-2-Hydroxytetracosanoylamino]-1,3,4-octadecanetriol (**1**) [13,19], 1-*O*-β-D-glucopyranosyl-(2*S*,3*S*,4*R*,9*Z*)-2-[(2*R*)-2-hydroxytetracosanoylamino]- 1,3,4-octadecanetriol (**4**) [20], 1-hexadecanoyl-*sn*-glycerol (**5**) [21], 1-octacosanoyl-*sn*-glycerol (**6**) [22], 1-[octadec-(9*Z*)-enoyl]-*sn*-glycerol (**7**) [23] and 1-(26-hydroxyhexacosanoyl)-*sn*-glycerol (**8**) [24,25].

### 3.2. Antiproliferative activity of the compounds 1–13

All the isolated compounds **1**–**13** from *L. chinensis* were tested for their potential antiproliferative capacity *in vitro* on HepG 2 (human hepatocellular carcinoma), HL-60 (human myeloid leukemia), K562 (human myeloid leukemia) and CNE-1 (human nasopharyngeal carcinoma). Cell proliferation was evaluated by determining the viable cells with the MTT assay, based on the intracellular reduction of tetrazolium salts into a blue product called formazan by the mitochondrial enzyme succinate dehydrogenase. The potential of all the isolates (**1**–**4**, **13**) to inhibit the proliferation of above four cell lines were summarized in Table 1. Compounds **3**, **4**, and **13** exhibited significant inhibition of proliferation in the four cell lines with $IC_{50}$ values in the range of 10–65 μM. It has been noted that the analogs (AGLs) of compounds **3** and **4** have been reported to exhibit *in vitro* antitumor activity against murine B16 melanoma, and the *in vitro* cytotoxicity of these analogs (AGLs) was weak and no activity was observed against B16 melanoma cells at 20 μg/mL [26]. This type compounds may show different activity against different cell lines, or other reasons. Glycoglycerolipids, as the analogs of compound **13**, exhibit several interesting activities; in particular, *in vitro* and *in vivo* tumor inhibitory activity [27–29]. It is evident that compounds **1** and **2**, soluble in medium, showed 10–20-fold weaker than that of

**Table 1**
Antiproliferative activities of the compounds (**1**–**4**, **13**) against HL-60, K562, HepG2 and CNE-1 cell lines.

| Compounds | $IC_{50}$ (μM) | | | |
|---|---|---|---|---|
| | HL-60 | K562 | HepG2 | CNE-1 |
| **1** | 184.46 ± 17.02 | 130.25 ± 12.02 | > 200 | > 200 |
| **2** | 176.25 ± 15.02 | 110.25 ± 9.46 | > 200 | > 200 |
| **3** | 14.20 ± 1.02 | 12.38 ± 1.02 | 62.76 ± 5.93 | 19.73 ± 1.25 |
| **4** | 18.20 ± 1.29 | 14.68 ± 0.82 | 74.76 ± 7.13 | 22.73 ± 0.35 |
| **13** | 20.26 ± 1.59 | 18.70 ± 1.23 | 30.50 ± 2.21 | 12.54 ± 0.86 |
| Cisplatin | 12.60 ± 0.89 | 15.70 ± 0.63 | 15.50 ± 1.21 | 14.54 ± 1.06 |

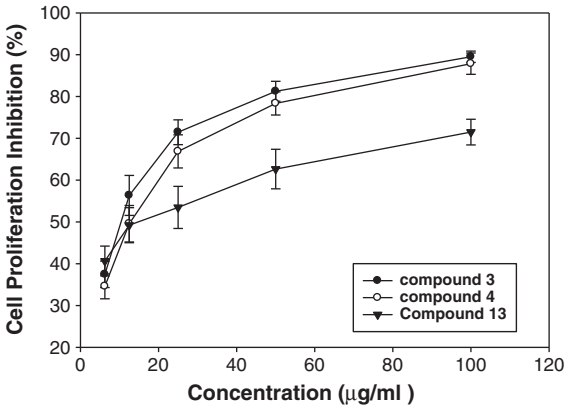


**Fig. 3.** Dose–response curve of antiproliferative activity against K562 of compounds **3, 4** and **13** (mean ± SD, $n = 3$).

compounds **3** and **4**, which suggests that the presence of a glucose moiety seems to improve the antiproliferative activity greatly. The isolated compounds **5**–**12** exhibited ineffectively. Perhaps, the solubility of compounds **5**–**12** was very limited in medium. The compounds **3** and **4** seemed to cause complete inhibition of the viability target cell line (K562), resulting in an antiproliferative efficacy of about 90.0% at the concentration 100 μg/mL. Also, compound **13** showed complete antiproliferation of human nasopharyngeal carcinoma (CNE-1) at the highest concentration tested with the antiproliferative efficacy of 91.3%. Good reducing capacity for the tetrazolium salt was also observed for **13** against HepG 2 and K562, which showed an antiproliferative efficacy of 67.3% and 71.5% at the concentration of 100 μg/mL. From the dose–response curve of antiproliferative activity against K562 cells among **3**, **4** and **13** (Fig. 3), we found that the inhibitive activity of compounds **3**, **4** and **13** changed moderately with the dose. So, these compounds (**3**, **4**, **13**) may be worth of further investigation.

## 4. Conclusions

In current research, the phytochemical analysis of the 70% EtOH extract of the roots of *L. chinensis* led to the isolation of a new ceramide (**2**), a new glycosyl ceramide (**3**), three new monoacylglycerols (**9**–**11**), a new diacylglycerol (**12**), and a new diacylglycerol aminoglycoside (**13**), along with a known ceramide (**1**), a known glycosyl ceramide (**4**), and four known monoacylglycerols (**5**–**8**). This is first report of the occurrence of ceramides and acylglycerols in the genus *Livistona*. Also, Several compounds from the roots of *L. chinensis* have been shown to possess strong antiproliferative activity.

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