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# Apoptosis, antimicrobial and antioxidant activities of phytochemicals from *Garcinia malaccensis* Hk.f

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ABSTRACT

**Objective:** To study the chemical constituents of stembark of *Garcinia malaccensis* (*G. malaccensis*) together with apoptotic, antimicrobial and antioxidant activities. **Methods**: Purification and structure elucidation were carried out by chromatographic and spectroscopic techniques, respectively. MTT and trypan blue exclusion methods were performed to study the cytotoxic activity. Antibacterial activity was conducted by disc diffusion and microdilution methods, whereas antioxidant activities were done by ferric thiocyanate method and DPPH radical scavenging. **Results:** The phytochemical study led to the isolation of α, β-mangostin and cycloart-24-en-3 β-ol. α-Mangostin exhibited cytotoxic activity against HSC-3 cells with an $IC_{50}$ of 0.33 μM. β- and α-mangostin showed activity against K562 cells with $IC_{50}$ of 0.40 μM and 0.48 μM, respectively. α-Mangostin was active against Gram-positive bacteria, *Staphylococcus aureus* (*S. aureus*) and *Bacilus anthracis* (*B. anthracis*) with inhibition zone and MIC value of (19 mm; 0.025 mg/mL) and (20 mm; 0.013 mg/mL), respectively. In antioxidant assay, α-mangostin exhibited activity as an inhibitor of lipid peroxidation. **Conclusions:** *G. malaccensis* presence α- and β-mangostin and cycloart-24-en-3 β-ol. β-Mangostin was found very active against HSC-3 cells and K562. The results suggest that mangostins derivatives have the potential to inhibit the growth of cancer cells by inducing apoptosis. In addition, α-and β-mangostin was found inhibit the growth of Gram-positive pathogenic bacteria and also showed the activity as an inhibitor of lipid peroxidation.

## 1. Introduction

The *Garcinia* species are known to be rich in phytochemical contents such as flavonoids, phenolic acids and xanthones. Mangosteen fruits from *Garcinia malaccensis* (*G. mangostana*) Linn also known as the queen of fruit is a most popular xanthone-rich and edible fruit that has been widely used and commercialized for its medicinal properties and is considered to be one of the best of all tropical fruits in Southeast Asia[1]. Some cytotoxic activities have been reported on the compounds isolated from *Garcinia*. α-Mangostin from *G. mangostana* has shown to protect mitochondria from peroxidative damage[2], apoptotic effects[3] and reducing oxidative damages[4]. *G. malaccensis* Hk.f (Guttiferae), locally known as "manggis burung" is a member of the Guttiferae family. Its fruit much resembles that of *G. mangostana* and *G. hombroniana*. Its mature fruit is similar in size to *G. hombroniana* but smaller than *G. mangostana*.

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This paper describes the first report on chemical constituents of the species of *G. malaccensis*. α-Mangostin (1) was found to be a major compound along with β-mangostin (2). In addition, isolation of a cycloart-24-en-3 β-ol (3) has never been reported from the genus before. α-Mangostin was focused in the bioactivity study due to the abundant amount of the sample. The compounds were tested for their activity as cytotoxic, antibacterial and antioxidant. The finding could provide interesting bioactivity and characteristics of the distribution of the compounds in the genus of *Garcinia*.

## 2. Materials and methods

### 2.1. General experimental procedures

The melting point was measured on a Buchi B545 melting point apparatus and reported uncorrected. The $^{13}$C-NMR and $^{1}$H-NMR were recorded on a Bruker 400 MHz in $CDCl_3$. The ultraviolet (UV) spectrum was recorded using a Secomam Uvi light XT2 spectrophotometer. The infrared (IR) spectrum was obtained from a Perkin Elmer infrared spectrophotometer. Optical rotation was measured on a Polax-2L polarimeter. Scanning electron microscope was conducted on a Carl Zeiss AG Evo 50. Fetal bovine serum, DMEM and RPMI medium, trypsin-EDTA, 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT), and penicillin-streptomycin were purchased from Invitrogen (USA). Organic solvents, silica gels and TLC plate were obtained from Merck (Germany). Paclitaxel was obtained from Sigma Aldrich (USA).

### 2.2. Plant material

The stembarks of *G. malaccensis* were collected from Agricultural Garden, Kuantan in November 2009. A voucher specimen (MT27) was deposited in the herbarium of Kulliyyah of Pharmacy the International Islamic University Malaysia.

### 2.3. Cell lines and bacteria

Human chronic myelogenous leukemia (K562) was purchased from American Type Tissue Culture (ATCC, USA). Meanwhile, human oral cancer (HSC-3) and human non-small lung carcinoma (H1299) cells were courtesy from Prof. Masa-Aki Ikeda of Tokyo Medical and Dental University. The antibacterial testing was carried out using two strains bacteria; Gram-positive *Staphylococcus aureus (S. aureus)* (ATCC 25923) and *Bacilus anthracis* (*B. anthracis*) (ATCC 14578); Gram-negative, *Pseudomonas aeruginosa* (*P. aeruginosa*) (ATCC 27853) and *Escherichia coli* (*E. coli*) (ATCC 35218). All bacteria were obtained from American Type Culture Collection (ATCC, USA).

### 2.4. Extraction

Dried and powdered stembark of *G. malaccensis* (580 g) was extracted by soxhlet extractor with *n*-hexane (hex), dichloromethane (DCM) and methanol (MeOH) successively. Evaporation of the respective solvent gave amount the hex, DCM and MeOH extracts as much as 9.0 g (1.6%), 27.8 g (4.8%) and 184 g (31.7%), respectively.

### 2.5. Cytotoxicity assay

Cytotoxicity assay were carried out using the MTT assay according to Mosmann[5] for monolayer cells (HSC-3 and H1299) and trypan blue exclusion method for suspension cells (K562). Monolayer and suspension cells were cultured in DMEM and RPMI 1640, respectively, supplemented with 10% (v/v) foetal bovine serum and 1% penicillin-streptomycin in a 96 well plate at a density of $6\times10^4$ cells/mL. After reaching confluence ($2\times10^5$ cell/mL), the cells were treated with the samples. The sample was dissolved in absolute ethanol (1 mg/mL). The cells were treated in triplicate with serial dilution of sample (0.01-1 mg/mL) for 24 h. The suspension cells were counted directly using trypan blue dye. Paclitaxel was used as a commercialized standard against K562. To minimize the interference of residue of supernatant, the monolayer cell were washed twice with phosphate buffer saline (PBS), the 20 μL of MTT stock solution (5 mg/mL) was added to each well and the plates were further incubated overnight at 37 ℃. DMSO (100 μL) was added to each well to solubilize the water insoluble purple formazan crystal. After 1 h, the absorbance was measured at 570 and 630 nm (reference) with a microplate reader. The 50% reduction in cell number relative to the control or $IC_{50}$ was established by extrapolation from linear regression of experimental data.

### 2.6. Antibacterial activity

#### 2.6.1. Disc diffusion method

The agar disc diffusion method was employed to determine antibacterial activities of the extracts. Briefly, inoculums containing $10^7$ CFU/mL were spread on Mueller-Hinton agar plates for bacteria. Using sterile forceps, the sterile filter papers (6 mm diameter) containing the 2 mg/mL for each compound (200 μg/disc) and standard antibiotics, streptomycin sulphate (10 μg/disc) or negative control (methanol) were laid down on the surface of inoculated agar plate. The plates were incubated at 37 ℃ for 24 h. Each compound was tested in triplicate and the zone of inhibition was measured in millimeter diameter.

#### 2.6.2. Microdilution method

Minimum inhibitory concentration (MIC) was performed as previously described with slight modification. MIC was

measured by determining the smallest amount of compound or standard antibiotic needed to inhibit the growth of tested bacteria. This was done using 96-well plates and performed on Versa Max™ Tunable microplate reader. The assay plates were filled with Mueller-Hinton broth medium containing different concentrations of compounds and the tested bacteria ($10^7$ CFU/mL). After 24 h incubation at 37 ℃, the turbidity in each well was measured at 600 nm.

### 2.7. Antioxidant activity

#### 2.7.1. Ferric thiocyanate (FTC) method

The detection of lipid peroxidation and preparation of solutions were carried out according to method described by Kikuzaki and Nakatani[6]. A mixture of 4.0 mg of sample in 4.0 mL of 99.5% ethanol, 4.1 mL of 2.5% linoleic acid in 99.5% ethanol, 8.0 mL of 0.02 M phosphate buffer (pH 7.0) and 3.9 mL of water in a screw-cap vial (38 mm×75 mm) was placed in an oven at 40 ℃ in the dark. A 9.7 mL of 75% (v/v) ethanol and 0.1 mL of 30% ammonium thiocyanate was added to 0.1 mL of the mixture. Three minutes after the addition of 0.1 mL of $2.0\times10^{-2}$ M ferrous chloride in 3.5% hydrochloric acid to the reaction mixture, the absorbance was measured at 500 nm at every 24 h until the absorbance of the control reached a maximum value.

#### 2.7.2. Dot-blot 2, 2-diphenyl-1-picrylhydrazyl (DPPH) staining

A quick screening of antioxidant activity was done using Dot-Blot DPPH staining methods[7] with slight modification. A drop of compound in methanol (1 mg/mL) was loaded on TLC plate and left to dry for a few minutes. The silica gel plate was sprayed with 0.4 mM DPPH solution in methanol until it was evenly covered and the formation of white yellow or yellow spot was observed on the plate.

#### 2.7.3. DPPH free radical scavenging activity method

The established method[8] was used with slight modification. Stock solution of sample was prepared in methanol (1 mg/mL). A test sample solution (200 μL) was added to 3.8 mL of 50 μM DPPH methanolic solution (to give a final concentration of 500, 250, 125, 62.5 μg/mL). After vortexing, the mixture was incubated for 30 min at room temperature. The color changes of DPPH (from deep violet to light yellow) were measured at 517 nm. The difference in absorbance between a test sample and a control (methanol) was expressed as % inhibition. The activity was shown as $IC_{50}$ value (50% inhibitory concentration). The percentage inhibition was calculated by the following formula:

Percentage of inhibition=Abs. DPPH-Abs. DPPH and Sample/Abs. DPPH×100%

### 2.8. Statistical analysis

All experiments were conducted in triplicate manner and the data obtained were represented as mean ± standard deviations that were interpreted using Microsoft Office Excel 2007®.

## 3. Results

### 3.1. Phytochemical investigation

The dried and powdered stembark of *G. malaccensis* (580 g) were extracted (soxhlet) for 18 h with *n*-hexane, DCM and MeOH successively. The DCM extract (25 g) was fractionated by vacuum liquid chromatography (VLC) on silica gel 230-400 mesh using hex, hex-DCM (1:1), DCM, acetone, and MeOH to afford five fractions (DF1-DF5). Fraction DF3 was washed with acetone to yield compound 1 (7.3 g, 1.3%) (Figure 1).

α-Mangostin (1) was obtained as a yellow amorphous powder; mp. 175-176 ℃, lit[9]. 180-182 ℃; $R_f$ 0.27 in DCM; UV $\lambda_{max}$ (EtOH) nm: 317.4; IR $\upsilon_{max}$ (KBr) $cm^{-1}$: 3 422, 2 925, 1 612, 1 458, 1 281; ESI-MS [M+1] m/z : 411.18, (calcd $C_{15}H_{26}O$, 410.1729); $^1$H NMR (400 MHz, Acetone-d6): δ 13.79 (s, 1H, H-1), 9.59 (s, 1H, H-6), 9.48 (s, 1H, H-3), 6.83 (s, 1H, H-5), 6.41 (s, 1H, H-4), 5.28 (mt, *J*= 6.0 Hz, 2H, H-12 and H-17), 4.16 (dd, *J*=18.0 and 10.0 Hz, 1H, H-11), 3.81 (s, 3H, OMe-21), 3.38 (d, *J*= 6.0 Hz, 2H, H-16), 1.85 (s, 3H, H-14), 1.80 (s, 3H, H-15), 1.65 (s, 3H, H-20). $^{13}$C NMR (100 MHz, Acetone-d6): δ 181.8 (C-9), 162.3 (C-3), 160.3 (C-1), 156.6 (C-6), 155.4 (C-10a), 154.9 (C-4a), 5 (C-7), 137.2 (C-8), 130.6 (C-13), 130.4 (C-18), 123.9 (C-17), 122.7 (C-12), 111.0 (C-8a), 110.2 (C-2), 102.6 (C-9a), 101.6 (C-5), 91.9 (C-4), 60.4 (OMe-21), 26.0 (C-14), 25.0 (C-16), 21.2 (C-11), 17.3 (C-20), 16.9 (C-15).

*n*-hexane extract (9 g) was fractionated by VLC on silica gel 60 (230-400 mesh) and eluted with petroleum ether (PE), PE:DCM (1:1), DCM and acetone to give four fraction (MF1-MF4). Fraction MF2 was purified by column chromatography (CC) on silica gel 60 (70-3230 mesh) using hex, hex: DCM (9:1), (8:2), (7:3), (6:4) and (5:5) to give new fraction (1-100). Fraction 54-60 and 60-100 were purified by washing with DCM to yield compound 2 (406 mg, 0.076%) and 3 (93 mg, 0.02%). β-Mangostin (2) was obtained as a yellow crystal; mp 165-166 ℃; lit[9]. 160-165 ℃; $R_f$ 0.77 in DCM; UV $\lambda_{max}$ (EtOH) nm: 217.6; IR $\upsilon_{max}$ (KBr) $cm^{-1}$: 3 400, 2 933, 1 603, 1 640, 1 204; ESI-MS [M+1] m/z: 425.20, (calcd $C_{25}H_{28}O_6$, 424.1886); $^1$H NMR (400 MHz, Acetone-d6): δ 13.7 (s, 1H, H-1), 6.88 (s, $^1$H, H-5), 6.53 (s, 1H, H-4), 5.29 (tt, *J*= 6.6 and 1.4 Hz, 1H, H-12), 5.22 (tt, *J*=6.6 and 1.4 Hz, 1 H, H-17), 4.14 (d, *J*=6.6 Hz, 1H, H-11), 3.98 (s, 3H, OMe-21), 3.82 (s, 3H, OMe-22), 3.33 (d, *J*=6.6 Hz, 1H, H-16), 1.84 (s, 3H, H-14), 1.79 (s, 3H, H-15), 1.67 (s, 3H, H-19), 1.65 (s, 3H, H-20). $^{13}$C NMR (100 MHz, Acetone-d6): δ 182.0 (C-9), 163.6 (C-3), 159.6 (C-1), 155.4 (C-6), 156.7 (C-10a), 155.3 (C-4a), 143.7 (C-7), 137.2

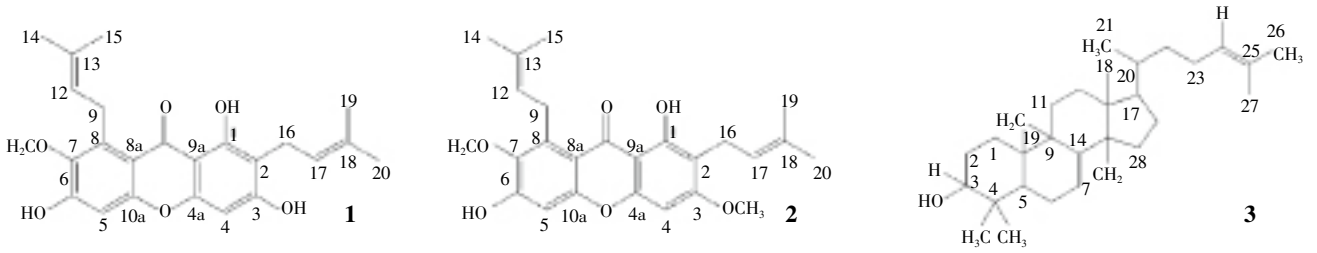

**Figure 1.** Structure of compound 1–3.

(C–8), 130.5 (C–13), 130.5 (C–18), 123.8 (C–17), 122.4 (C–12), 111.2 (C–8a), 110.9 (C–2), 103.2 (C–9a), 101.8 (C–5), 88.9 (C–4), 60.5 (OMe–21), 55.6 (OMe–22), 25.0 (C–14), 21.0 (C–16), 26.0 (C–11), 17.4 (C–20), 16.9 (C–15).

Cycloart–24–en–3 β –ol (3) was obtained as a white needle; = +68.1 (MeOH, c 0.0088); mp 90.5–92 ℃; lit[10]. 100–101 ℃; $R_f$ 0.61 in DCM; UV $\lambda_{max}$ (EtOH) nm: 315.0; IR $\upsilon_{max}$ (KBr) $cm^{-1}$ : 3 311, 2 932, 1 450, 1 376, 1 098, 1 047, 1 024; ESI–MS [M+1] m/z : 427.39, (calcd $C_{30}H_{50}O$, 426. 3862); $^1$H NMR (400 MHz, $CDCl_3$): δ 5.21 (brtt, *J*=4.6 Hz, 1H, H–24), 3.31 (p, *J*= 4.5, 1H, H–3), 2.05–1.97 (m, 2H, H–23), 1.92–1.88 (m, 2H, H–16), 1.78–176 (m, 2H, H–2), 1.71 (s, 3H, H–26), 1.66–1.56 (m, 10H, H–1(2), H–6(2), H–15(2), H–17(1), H–18(3)), 1.52 (dd, *J*=12.4 and 4.8 Hz, 1H, H–5), 1.48–1.38 (m, 2H, H–22), 1.36–1.30 (m, 5H, H–7(2), H–12(2), H–20(1), 1.27 (dd, *J*=6.4 and 2.9 Hz, 1H, H–8), 1.17–1.09 (m, 2H, H–11), 0.99 (s, 6H, H–27 and H–29), 0.91 (s, 3H, H–28), 0.89 (s, 3H, H–21), 0.83 (s, 3H, H–30), 0.57 and 0.35 (ABq, *J*=3.5, 2H, H–19). $^{13}$C NMR (100 MHz, $CDCl_3$): δ 130.9 (C–25), 125.3 (C–24), 78.9 (C–3), 52.3 (C–17), 48.8 (C–14), 48.0 (C–5), 47.1 (C–8), 45.3 (C–13), 40.5 (C–4), 36.4 (C–22), 35.9 (C–20), 32.9 (C–15), 32.0 (C–1), 31.0 (C–12), 30.4 (C–2), 28.2 (C–7), 26.5 (C–16), 26.1 (C–9), 26.1 (C–23), 25.6 (C–19), 25.6 (C–26), 25.4 (C–29), 24.9 (C–11), 21.1 (C–6), 20.0 (C–10), 19.3 (C–28), 18.2 (C–21), 18.1 (C–27),17.7 (C–18), 14.0 (C–30).

### 3.2. Cytotoxic activity

α –Mangostin showed a significant antiproliferation

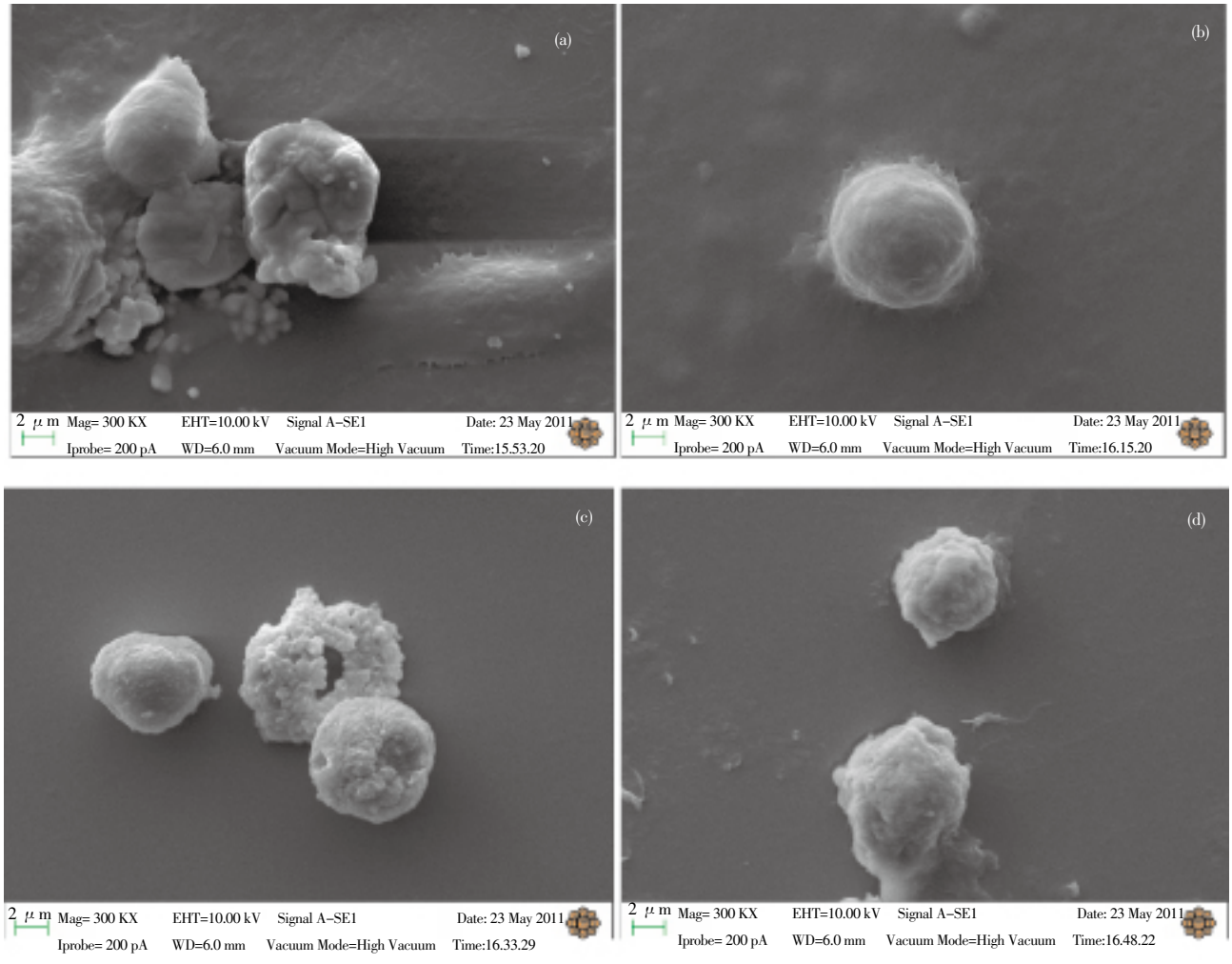


**Figure 2.** Cell membrane blebbing during apoptosis after treated with β –mangostin.
(a) HSC–3 (treated with 0.33 μM); (b) HSC–3 (untreated); (c) K562 (treated with 0.40 μM) and (d) K562 (untreated).

effect against K562 by affecting cell proliferation and was found to be active in a dose dependent manner with $IC_{50}$ value at 0.48 μM. The compound was not active against HSC3 and H1299 cell lines ($IC_{50}$> 10 μM). β-Mangostin also exhibited significant activity against HSC-3 and K562 with $IC_{50}$ values of 0.33 and 0.40 μM, respectively and not active against H1299. Cycloart-24-en-3 β-ol showed no activity against all tested cell lines. In order to investigate whether β-mangostin induce apoptosis to the K562 cells, the cell lysates was investigated for the occurrence of DNA fragmentation. The results of the gel electrophoresis showed that DNA fragmentation occurred in the K562 cells exposed to β-mangostin at 55 μg/mL (data not shown). Scanning electron microscope has been done on the cells after treatment with β-mangostin (Figure 2). It showed that β-mangostin induce apoptosis on HSC-3 and K562 indicated by blebbing phenomenon which is a specific pattern of apoptosis.

### 3.3. Antimicrobial activity

α and β-mangostin exhibited antibacterial activity only against two Gram-positive, *S. aureus* and *B. anthracis* at concentration of 200 μg/disc. The diameter of inhibition zones and MIC values were in range of 9.5-20 mm and 0.013- >3 mg/mL, respectively. Cycloart-24-en-3 β-ol showed inhibitory effect only to Gram-negative, *P. aeruginosa* with diameter of inhibition zone (MIC value) at 8.5 mm (>3 mg/mL). *B. athracis* and *S. aureus* were the most sensitive strain to α, β-mangostin and streptomycin sulphate. α mangostin showed diameter of inhibition zone (MIC value) at 19 mm (0.025 mg/mL) and 20 mm (0.013 mg/mL), respectively and β-mangostin exhibited inhibition zone (MIC value) at 10.5 mm (>3 mg/mL) and 9.5 mm (>3 mg/mL), respectively and streptomycin sulphate showed zone inhibition at 13 mm and 15 mm, respectively. The results showed that Gram-positive bacteria appear to be the most sensitive strain than Gram-negative bacteria to the isolated compounds.

### 3.4. Antioxidant activity

Figure 3 shows the results of antioxidant activity of α-mangostin that exhibited stronger antioxidant activity than α-tocopherol in FTC method. The percentages of inhibition of α-mangostin and α-tocopherol were 96.9 % and 84.94%, respectively. When α-mangostin was screened for dot blot DPPH staining, the yellow spots appeared quickly at the location of the drops which showed radical scavenging activity. α-Mangostin exhibited a moderate intensity of yellow spots compared to vitamin C that showed stronger intensities of white yellow spots (data not shown). The percentage of inhibition of α-mangostin, ascorbic acid and α-tocopherol at concentration of 0.25 mg/mL were 3.8, 54.8 and 25.4, respectively.

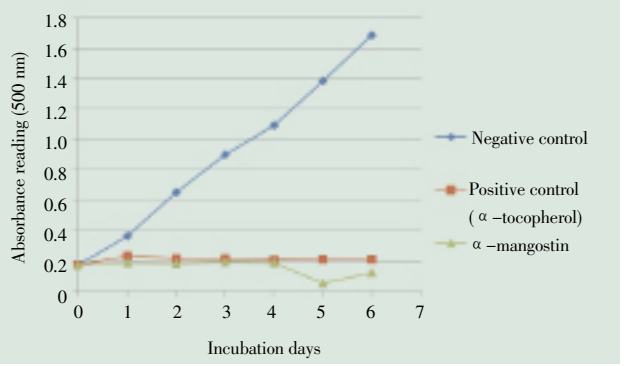


**Figure 3.** The antioxidant activity of α-mangostin by FTC method. Data represent three independent experiments performed in triplicate.

## 4. Discussion

α-Mangostin was found as an orange powder with a melting point of 175-176 ℃. UV spectrum showed absorbance at 317 nm characteristic of xanthone moiety. IR of α-mangostin revealed a hydroxyl group at 3 422 $cm^{-1}$, a carbonyl group with C=O stretching at 1 612 $cm^{-1}$ and O-H bending at 1 281 $cm^{-1}$. The structure of α-mangostin was characterized by comparison the NMR of the compound with literature data[18]. The *n*-hexane extract was partitioned with PE, DCM and acetone. A repeated chromatography of the MF2 fraction yielded β-mangostin and cycloart-24-en-3 β-ol. β-Mangostin was found as a yellow crystal with a melting point of 165-166 ℃. The $^{1}H$ and $^{13}C$ NMR spectra of β-mangostin resembled to those of α-mangostin, except that it included the signal of a methoxyl group at C-3. The $^{1}H$ and $^{13}C$ spectroscopic data agreed with previous published report[9]. Cycloart-24-en-3 β-ol was obtained as a white needle with a melting point of 90.5-92 ℃. The UV spectrum of cycloart-24-en-3 β-ol showed absorbance at 315 nm. The IR spectrum revealed a hydroxyl group at 3 311 $cm^{-1}$ and 2 932 $cm^{-1}$ for a double bond in the olefinic group. Its ESI-MS spectrum showed a molecular ion of m/z [M+1] 427 was in agreement with the molecular formula of $C_{30}H_{50}O$. Its structure had five double bonds equivalent which suggested five cyclics and one double bond suggesting that four of them were assigned in the tetracyclic carbon framework of the steroidal nucleus and one in olefinic group. The $^{1}H$ and $^{13}C$ NMR of cycloart-24-en-3 β-ol agreed with literature data[10-17].

The use of antineoplastic drug is based on their selective toxicity toward malignant cells. α-mangostin selectively exhibited a moderate activity against K562 cells. Previously, Nakagawa *et al*[3] reported the apoptotic effect of α-mangostin on human colon cancer (DLD-1) cell at 20 μM. β-mangsotin was the most active compound to inhibit cell cancer growth by inducing apoptosis. However, the activity of the compounds could not beat the commercial anticancer agent, paclitaxel with $IC_{50}$ value of 0.1 μM.

The antibacterial activity of α and β-mangostin and cycloart-24-en-3 β-ol against the employed bacteria was qualitatively and quantitatively assayed by the presence of inhibition zones, diameter of inhibition zone and MIC

values. The Gram-negative were not sensitive to the isolated because of the barrier in the bacteria that consist of specific cell wall structure, outer membrane and a unique periplasmic space that were not found in the Gram-positive bacteria. The outer membrane of Gram-negative bacteria is rich in lipopolysaccharide molecules almost impermeable to hydrophilic compounds. α and β-mangostin are the hydrophilic compounds that could not penetrate to the barrier. The absence of this barrier in the Gram-positive bacteria allows the direct contact of α and β-mangostin with the phospholipid bilayer of the cell membrane, where they bring about their effects, causing either an increase of ion permeability and leakage of vital intracellular constituent or impairment of the bacterial enzyme system[19]. Cycloart-24-en-3 β-ol is a lipophilic compound and can penetrate to the barrier in Gram-negative outer membrane and only showed the activity on *P. aeruginosa*.

Antioxidant assay using FTC method was carried out on α-mangostin only, this due to its availability in good amount. In this method, the linoleic acid (RCOOH) was reduced by $Fe^{2+}$ to free radical ($RO^{\cdot}$), while the ferrous ion itself underwent oxidation process to $Fe^{3+}$. The $Fe^{3+}$ ion reacted with thiocyanate ion ($SCN^-$) to give complex $Fe(SCN)_3$ was measured by UV spectrophotometer at 500 nm. The low absorbance value corresponding to a high percent of inhibition thus indicate that the sample could inhibit lipid peroxidation. The percentage of inhibition value for α-mangostin very low compared to the vitamin C in DPPH radical scavenging test. The percentage of inhibition depends on the amount and the nature of radical scavenger present in the compound. Based on these results, α-mangostin showed activity as an inhibition of lipid peroxidation rather than radical scavenger.

In conclusion, the stembark of *G. malaccensis* showed the presence of xanthones, namely α- and β-mangostin together with a triterpenoid, cycloart-24-en-3 β-ol. β-Mangostin was found very active against HSC-3 cells and K562. However, cycloart-24-en-3 β-ol was not active against the cell lines tested. None of the compounds showed activity against H1299. The results suggest that mangostins derivatives have the potential to inhibit the growth of cancer cells by inducing apoptosis. In addition, α-and β-mangostin was found inhibit the growth of Gram-positive pathogenic bacteria and also showed the activity as an inhibitor of lipid peroxidation.

## Conflict of interest statement

We declare that we have no conflict of interest.

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