A new phenolic derivative with soluble epoxide hydrolase and nuclear factor-kappaB inhibitory activity from the aqueous extract of *Acacia catechu*

Ya Nan Sun\(^a\), Wei Li\(^b\), Seok Bean Song\(^c\), Xi Tao Yan\(^d\), Yan Zhao\(^a\), A. Reum Jo\(^a\), Jong Seong Kang\(^a\) and Kim Young Ho\(^a\)

\(^a\)College of Pharmacy, Chungnam National University, Daejeon, Korea; \(^b\)School of Biotechnology, Yeungnam University, Gyeongsan, Korea; \(^c\)Gyeongbuk Institute for Bio-industry (GIB), Andong City, Korea; \(^d\)Shaanxi Key Laboratory of Natural Products & Chemical Biology, College of Science, Northwest A & F University, Yangling, China

**ABSTRACT**

One novel phenolic compound, \((4S,5R)-4-(3,4-dihydroxyphenyl)-5-(3-oxobutyl)dihydrofuran-2(3H)-one (1)\), as well as 12 known compounds (2–13) was obtained from the aqueous extract of *Acacia catechu* and their chemical structures were determined by spectroscopic analysis. Compounds 8 and 9 exhibited significant soluble epoxide hydrolase (sEH) inhibitory activities with IC\(_{50}\) values of 26.6 ± 0.5 and 24.4 ± 5.6 μM, respectively. Compounds 7–10 showed significant inhibitory effects on TNFα-induced nuclear factor kappa B (NF-κB) transcriptional activity in a dose-dependent manner, with IC\(_{50}\) values ranging from 11.15 to 19.45 μM.

**1. Introduction**

*Acacia catechu* (Leguminosae) locally known as *khair* is a medium-sized deciduous tree with a wavy and lodged trunk that is primarily distributed in India, Africa and most of China (Li et al. 2011). This tree produces nutrients and therapeutic ingredients that prevent, mitigate and treat many diseases, such as antipyretics, anthelmintics and sore throat remedies in Traditional Chinese Medicine (Mutai et al. 2007; Singh et al. 2009; Gedara et al. 2015). For these treatments, a decoction of bark mixed with milk is taken to treat colds and coughs, while
it is administered alone or in combination with opium to treat severe diarrhoea (Clement et al. 1999). Primary studies have reported that *A. catechu* contains a variety of bioactive components such as phenolic acids, alkaloids, terpenes, tannins and flavonoids (Fourie et al. 1974; Mujoo et al. 2011; Readel et al. 2001), which are responsible for numerous biological and pharmacological properties including hypoglycaemic, anti-inflammatory, antibacterial, antiplatelet aggregation, antihypertensive, analgesic, anticancer, chemoprotective and antiatherosclerotic activities (Croft 1998; Pietta 2000; Karoune et al. 2015; Ndidi et al. 2015; Stohs et al. 2015). In this study, evaluation of the soluble epoxide hydrolase (sEH) and nuclear factor kappa B (NF-κB) transcriptional inhibitory properties resulted in isolation of 13 phenolic compounds from the aqueous extract of *A. catechu*.

2. Results and discussion

Compound 1 was obtained as a yellow solid with the molecular formula C_{14}H_{16}O_{5} as deduced from the [M + H]+ peak at m/z 265.1065 (calcd. for C_{14}H_{17}O_{5}, 265.1071) by HRESIMS. The
The 1H NMR spectrum of 1 presented three aromatic signals at $\delta_H$ 7.19 (1H, d, $J_H = 2.2$ Hz, H-2'), 7.24 (1H, d, $J_H = 8.0$ Hz, H-5') and $\delta_H$ 6.75 (1H, dd, $J_H = 8.0, 2.2$ Hz, H-6'), belonging to an ABX system, suggesting the presence of a 1,2,4-trisubstituted benzene ring. In addition, one carbonyl-bearing methyl $\delta_H$ 1.88 (s, 3H) and several aliphatic proton signals were observed in the high field. The 13C NMR spectrum of 1 confirmed the presence of a carbonyl carbon ($\delta_C$ 206.9), a carbonyl ester group ($\delta_C$ 176.8), an aromatic ring ($\delta_C$ 147.2, 146.4, 129.7, 119.3, 116.4, 115.9), an oxygenated carbon ($\delta_C$ 83.3) and five aliphatic carbons ($\delta_C$ 43.9, 39.4, 35.8, 29.4, 25.5). Accounting for the degree of unsaturation calculation, compound 1 was inferred to have two rings, including a benzene ring. In the HMBC spectrum, the correlations from H-9 ($\delta_H$ 1.88) to C-8 ($\delta_C$ 206.9) and C-7 ($\delta_C$ 39.4) suggested the position of an oxobutyl moiety. Furolactone was deduced based on the correlations between H-3 ($\delta_H$ 2.92, 2.75), H-4 ($\delta_H$ 3.59) and C-2 ($\delta_C$ 176.9). The cross-peaks between H-4/C-1', H-4/C-2' and H-4/C-6' indicate

### Table 1. The sEH inhibitory activities of compounds 1–13.

| Compounds | Inhibition of compounds on sEH$^b$ |
|-----------|-----------------------------------|
|           | 100 μM (%) | IC$_{50}$ (μM) |
| 1         | 57.9 ± 0.4 | N.T. |
| 2         | 20.2 ± 1.7 | N.T. |
| 3         | 11.9 ± 0.1 | N.T. |
| 4         | 4.9 ± 4.6  | N.T. |
| 5         | 3.4 ± 2.3  | N.T. |
| 6         | 26.8 ± 2.0 | N.T. |
| 7         | 67.8 ± 0.2 | N.T. |
| 8         | 90.8 ± 0.5 | 26.6 ± 0.5 |
| 9         | 85.2 ± 1.5 | 24.4 ± 5.6 |
| 10        | 73.6 ± 0.8 | 62.4 ± 0.5 |
| 11        | 20.9 ± 5.4 | N.T. |
| 12        | 60.5 ± 5.1 | N.T. |
| 13        | 40.7 ± 1.5 | N.T. |
| AUDA$^a$  |         | 10.9 ± 1.2 nM |

Note: N. T-not tested.

$^a$AUDA: 12-(3-adamantan-1-yl-ureido)-dodecanoic acid butyl ester, positive control.

$^b$Tested compounds were examined in a set of experiments repeated three times.

### Table 2. Inhibitory effects of compounds 1–13 on the TNFα-induced NF-κB transcriptional activity.

| Compounds | IC$_{50}$ $^a$ (μM) |
|-----------|---------------------|
| 1         | 27.94 ± 2.35        |
| 2         | 31.57 ± 2.51        |
| 3         | 32.07 ± 2.01        |
| 4         | 27.75 ± 2.70        |
| 5         | 25.47 ± 2.75        |
| 6         | 24.22 ± 2.53        |
| 7         | 15.01 ± 2.68        |
| 8         | 19.45 ± 2.24        |
| 9         | 15.24 ± 2.34        |
| 10        | 11.15 ± 2.31        |
| 11        | 49.60 ± 2.26        |
| 12        | > 50                |
| 13        | 40.48 ± 2.34        |
| Apigenin$^b$ | 1.64 ± 0.19        |

$^a$Values are mean ± SD ($n = 3$).

$^b$Apigenin was used as a positive control.

$^1$H NMR spectrum of 1 presented three aromatic signals at $\delta_H$ 7.19 (1H, d, $J_H = 2.2$ Hz, H-2'), 7.24 (1H, d, $J_H = 8.0$ Hz, H-5') and $\delta_H$ 6.75 (1H, dd, $J_H = 8.0, 2.2$ Hz, H-6'), belonging to an ABX system, suggesting the presence of a 1,2,4-trisubstituted benzene ring. In addition, one carbonyl-bearing methyl $\delta_H$ 1.88 (s, 3H) and several aliphatic proton signals were observed in the high field. The 13C NMR spectrum of 1 confirmed the presence of a carbonyl carbon ($\delta_C$ 206.9), a carbonyl ester group ($\delta_C$ 176.8), an aromatic ring ($\delta_C$ 147.2, 146.4, 129.7, 119.3, 116.4, 115.9), an oxygenated carbon ($\delta_C$ 83.3) and five aliphatic carbons ($\delta_C$ 43.9, 39.4, 35.8, 29.4, 25.5). Accounting for the degree of unsaturation calculation, compound 1 was inferred to have two rings, including a benzene ring. In the HMBC spectrum, the correlations from H-9 ($\delta_H$ 1.88) to C-8 ($\delta_C$ 206.9) and C-7 ($\delta_C$ 39.4) suggested the position of an oxobutyl moiety. Furolactone was deduced based on the correlations between H-3 ($\delta_H$ 2.92, 2.75), H-4 ($\delta_H$ 3.59) and C-2 ($\delta_C$ 176.9). The cross-peaks between H-4/C-1', H-4/C-2' and H-4/C-6' indicate
that the aromatic ring was attached to C-4 (δ C 43.9) of furolactone. Correlations of H-6 (δ H 1.61) and H-7 (δ H 2.38) with C-5 (δ C 83.3) suggested that the oxobutyl group was linked to C-5 of furolactone. These findings led to the conclusion that 1 has the structure 4-(3,4-dihydroxyphenyl)-5-(3-oxobutyl)dihydrofuran-2(3H)-one. The absolute configuration of 1 was determined based on a CD experiment. The observed positive cotton effect at 280 nm of 1 led to assignment of a 4S,5R-configuration (Li et al. 2011). Therefore, the structure of 1 was characterised as (4S,5R)-4-(3,4-dihydroxyphenyl)-5-(3-oxobutyl)dihydrofuran-2(3H)-one.

The other known compounds were identified as (5R)-5-(1-(3,4-dihydrophenyl)-3-oxobutyl)dihydrofuran-2(3H)-one (2; Li et al. 2011), (5R)-5-(3,4-dihydrophenyl)-γ-valerolactone (3; Lambert et al. 2005), 4-hydroxybenzoic acid (4; Rukachaisirikul et al. 2010), isovanillic acid (5; Ding et al. 2000), 4-methyl pyrocatechol (6; Huang et al. 2011), pyrocatechol (7; Fujita et al. 1982), (+)-aromadendrin (8; Fossen et al. 1998), (+)-taxifolin (9; Fossen et al. 1998), (+)-catechin (10; Du et al. 2005), planchol A (11; Luo et al. 2013), (+)-isorariciresinol (12; Erdemoglu et al. 2003) and (+)-lyoniresinol (13; Li et al. 2010), by comparing their physical and spectroscopic data with those reported in the literature (Figure 1).

The inhibitory activity of isolated compounds against sEH was evaluated. Specifically, the amount of 6-methoxy-2-naphthaldehyde produced from the substrate (PHOME) was quantified in the presence or absence of compounds 1–13 using a fluorescence photometer at wavelengths of 330 and 465 nm. (Moser et al. 2012; Lee et al. 2014). AUDA was used as a positive control (IC50 = 10.9 ± 1.2 nM).

The inhibitory activity of isolated compounds was first evaluated against sEH at 100 μM. Compounds 8–10 showed significant inhibitory effects, with inhibition rates of 73.6–90.8%. Various concentrations (12.5, 25, 50 and 100 μM) of these compounds were then tested, and compounds 8 and 9 were found to exert potent activities, with IC50 values of 24.4 ± 5.6 μM and 26.6 ± 0.5 μM, respectively (Table 1). Compound 10 showed moderate effects with an IC50 value of 62.4 ± 0.5 μM.

All compounds were tested for cytotoxic activity against HepG2 cells. And no significant cytotoxicity was observed at the tested concentrations (data shown in supporting information). Compounds 1–13 were pretreated with transfected HepG2 cells at various concentrations (0.1, 1 and 10 μM), stimulating with TNF-α. The results showed that compounds 1–10 significantly inhibited TNF-α-induced NF-κB transcriptional activity in a dose-dependent manner, with IC50 values ranging from 11.15 to 32.07 μM. Compounds 11 and 13 had weak effects, with IC50 values of 40.48 and 49.60 μM, respectively (Table 2). However, compound 12 was inactive (IC50 > 50 μM) at the tested concentrations.

Based on the structure–activity relationship of the isolated compounds, the results showed that flavonoids (8–10), especially flavonols (8 and 9) are primary bioactive constituents against sEH. Conversely, phenols and flavonoids (1–10) exerted significant NF-κB inhibitory activities. These results indicated that phenols and flavonoid derivative components of A. catechu may play key functional roles in anti-inflammatory activity.

### 3. Experimental

#### 3.1. General experimental procedures

The NMR spectra were recorded using a JEOL ECA 600 spectrometer (1H, 600 MHz; 13C, 150 MHz), with tetramethylsilane (TMS) as an internal standard. Heteronuclear
multiple quantum correlation (HMQC), heteronuclear multiple bond correlation (HMBC) and $^1$H–$^1$H correlation spectroscopy (COSY) spectra were recorded using a pulsed field gradient. HR-ESI-MS was carried out using an Agilent 6530 LC-MSd Trap spectrometer. Column chromatography was performed using a silica gel (Kieselgel 60, 70–230 and 230–400 mesh, Merck, Darmstadt, Germany), YMC RP-18 resins, and thin layer chromatography (TLC) was performed using precoated silica gel 60 F$_{254}$ and RP-18 F$_{254S}$ plates (both 0.25 mm, Merck, Darmstadt, Germany).

### 3.2. Plant material

The aqueous extracts of *A. catechu* were purchased from the herbal company, Naemome Dah, Ulsan, Korea in 2013, and were identified by Prof. Young Ho Kim, Chungnam National University. A voucher specimen (CNU13107) was deposited at herbarium, College of Pharmacy, Chungnam National University, Korea.

### 3.3. Extraction and isolation

The aqueous extracts of *A. catechu* (2.5 kg) were dissolved three times with MeOH under reflux. The MeOH extract (1.5 kg) was suspended in $H_2$O (2.0 L) and partitioned with EtOAc and $n$-BuOH. The EtOAc extract (600 g) was subjected to silica gel column chromatography with a gradient of CHCl$_3$–MeOH–$H_2$O (1:0:0–2:1:0.1), to yield seven fractions (Fr. A1–A7). Fraction A2 was further purified by silica gel column chromatography with $n$-hexane–EtOAc (20:1–1:1) elution solvent to yield eight sub-fractions (Fr. A2.1–A2.8). Further purification of Fr. A2.3 was conducted by chromatography in an RP column with an eluent gradient of MeOH–$H_2$O (1:10–2:1) to yield compounds 1 (10.0 mg), 4 (44.0 mg), 5 (59.0 mg), 6 (9.0 mg), 7 (16.0 mg) and 11 (26.0 mg). Fr. A2.5 was further chromatographed in an RP column with MeOH–$H_2$O (1:2–1:1) to yield compound 2 (110.0 mg). Fr. A2.7 was chromatographed in a silica gel column with an eluent gradient of n-hexane–EtOAc (3:1:0.05) to yield compound 8 (40.0 mg). Fr. A3 was subjected to silica gel column chromatography with a gradient of $n$-hexane–EtOAc (15:1–2:1) to yield six fractions (Fr. A3.1–A3.6). Fr. A3.5 was separated using an RP column with a MeOH–$H_2$O (1:2–2:1) elution solvent to yield compound 12 (10.0 mg) and 13 (8.0 mg). Fr. A5 was separated by silica gel column chromatography with a gradient of CHCl$_3$–MeOH–$H_2$O (10:1:0–5:1:0.1) to yield four sub-fractions (Fr. A5.1–A5.4). Fraction A5.2 was further chromatographed on an RP chromatography column with MeOH–$H_2$O (1:10) to yield compound 10 (390.0 mg).

#### 3.3.1. (4S,5R)-4-(3,4-dihydroxyphenyl)-5-(3-oxobutyl)dihydrofuran-2(3H)-one (1)

Yellow amorphous powder; C$_{14}$H$_{16}$O$_5$; [α]$_{D}^{25}$D: +13.02 (c 0.1, MeOH); $^1$H NMR (pyridine-$d_5$, 600 MHz): 2.75 (1H, m, H-3), 2.92 (1H, m, H-3), 3.59 (1H, dd, $J=13.2$, 5.4 Hz, H-4), 4.66 (1H, m, H-5), 1.61 (2H, m, H-6), 2.38 (2H, m, H-7), 1.88 (3H, s, H-9), 7.19 (1H, d, $J=2.2$ Hz, H-2’), 7.24 (1H, d, $J=8.0$ Hz, H-5’), 6.75 (1H, dd, $J=8.0$, 2.2 Hz, H-6’). $^{13}$C NMR (pyridine-$d_5$, 150 MHz): 176.8 (C-2), 35.8 (C-3), 43.9 (C-4), 83.3 (C-5), 25.5 (C-6), 39.4 (C-7), 206.9 (C-8), 29.4 (C-9), 129.7 (C-1’), 116.4 (C-2’), 146.4 (C-3’), 147.2 (C-4’), 115.9 (C-5’), 119.3 (C-6’). HR-ESI-MS: m/z 265.1065 [M + H]$^+$ (calcd. for C$_{14}$H$_{17}$O$_5$, 265.1071).
3.4. sEH assay

The sEH inhibition assays were performed as described in the previous study. A solution of recombinant sEH from human (the enzymes were purchased from Cayman Chemical Company) in buffer (Bis-Tris-HCl, 25 mM, pH 7.0, containing 0.1 mg/ml BSA) was incubated with an inhibitor at room temperature for 30 min. To the resultant solution, cyano(6-methoxynaphthalen-2-yl)methyl trans-[(3-phenyloxiran-2-yl)methyl] carbonate (purchased from Cayman Chemical Company) was added and incubated at room temperature for 20–45 min. ZnSO₄ was added and the resultant solution of fluorescence intensity (excitation filter 330 nm, emission filter 465 nm) was measured.

Briefly, 50 μL of sEH and 20 μL of various concentrations of the compounds dissolved in MeOH were added in 96-well plate containing 80 μL of 25 mM bis-tris-HCl buffer, mixed with 50 μL of 40 μM PHOME. After starting the enzyme reaction at 37 °C, products by hydrolysis of the substrate were monitored at excitation and emission of 330 and 465 nm during 1 h.

Enzyme activity (%) = \(\left(\frac{S_{60} - S_0}{C_{60} - C_0}\right) \times 100\)

where \(C_{60}\) and \(S_{60}\) were the fluorescence of control and test samples after 60 min, \(S_0\) and \(C_0\) is the fluorescence of control at 0 min.

3.5. Cell culture

Human hepatocarcinoma HepG2 cells were maintained in Dulbecco’s modified Eagles’ medium (DMEM) (Invitrogen, Carlsbad, CA) containing 5% heat-inactivated foetal bovine serum (FBS), 1X penicillin at 37 °C and 5% CO₂. Human TNF-α was purchased from ATgen (Seoul, Korea).

3.6. Cell viability assay

A CCK-8 cell-counting kit (Dojindo, Kumamoto, Japan) was used to analyse the cell toxicity of the compounds according to the manufacturer’s instructions. HepG2 cells were cultured overnight in a 96-well plate (about 1 × 10⁴ cells/well). After 24 h, each compound was added in a dose-dependent manner. After 24 h of treatment, 10 μL of the CCK-8 solution was added to triplicate wells and incubated for 1 h. The absorbance at 450 nm was measured to determine the viable cell numbers.

3.7. NF-κB luciferase assay

The luciferase vector was first transfected into human hepatocarcinoma HepG2 cells. HepG2 cells were transfected with NF-κB luciferase reporter (3Enhancer-ConA) with the Neon® Transfection system (Invitrogen) according to the manufacturer’s instructions. After a limited amount of time, the cells were lysed, and luciferin, the substrate of luciferase, was introduced into the cellular extract along with Mg²⁺ and an excess of ATP. Under these conditions, luciferase enzymes expressed by the reporter vector could catalyse the oxidative carboxylation of luciferin. Cells were seeded at 2 × 10⁵ cells per well in 12-well plates and grown. After 24 h, cells were transfected with inducible NF-κB luciferase reporter and constitutively expressing Renilla reporter. After 24 h of transfection, the medium was changed to assay medium (Opti-MEM + 0.5% FBS + 0.1 mM NEAA + 1 mM sodium pyruvate + 100 units/mL penicillin + 10 μg/mL streptomycin) and cells were pretreated for 1 h with either
vehicle (DMSO) and compounds, followed by 1 h of treatment with 10 ng/mL TNF-α for 20 h. Unstimulated cells were used as a negative control (−), apigenin was used as a positive control. Dual luciferase assay was performed 48 h after transfection, and promoter activity values are expressed as arbitrary units using a Renilla reporter for internal normalisation.

4. Conclusion

In this study, 13 phenolic compounds (1–13) were isolated from the aqueous extract of A. catechu. Compounds 1, 3, 6, 11 and 13 were isolated from the Leguminosae family for the first time. Moreover, this study is the first to show that phenolic components from A. catechu inhibit sEH and TNFα-induced NF-κB transcriptional activity. The results presented herein suggest that the phenolic components in A. catechu contribute to its observed anti-inflammatory effects.

Disclosure statement

No potential conflict of interest was reported by the authors.

Funding

This work was financially supported by the Priority Research Centers Program through the National Research Foundation of Korea (NRF) funded by the Ministry of Education, Science and Technology [grant number 2009-0093815] of the Republic of Korea.

References

Clement BA, Goff CM, Forbes TDA. 1999. Toxic amines and alkaloids from Acacia berlandieri. Phytochemistry 46:249–254.
Croft KD. 1998. The chemistry and biological effects of flavonoids and phenolic acids. Ann NY Acad Sci. 854:435–442.
Ding HY, Lin HC, Teng CM, Wu YC. 2000. Phytochemical and pharmacological studies on Chinese Paeonia species. J Chin Chem Soc. 47:381–388.
Du ZZ, Zhao PJ, He HP, Zhu N, Hao XJ, Shen YM. 2005. The chemical constituents from the callus culture of Trewia nudiflora. Helv Chim Acta. 88:2424–2429.
Erdemoglu N, Sener B, Ozcan Y, Ide S. 2003. Structural and spectroscopic characteristics of two new dibenzylybutane type lignans from Taxus baccata L. J Mol Struct. 655:459–466.
Fossen T, Pedersen AT, Andersen ØM. 1998. Flavonoids from red onion (Allium cepa). Phytochemistry 47:281–285.
Fourie TG, Ferreira D, Roux DG. 1974. 8-O-Methyl and the first 3-O-methyl flavan-3,4-diol from Acacia saxatilis. Phytochemistry 13:2573–2581.
Fujita M, Nagai M, Inoue T. 1982. Carbon-13 nuclear magnetic resonance spectral study. Effect of o-methylation of ortho-substituted phenols on the aryl carbon shielding and its application to interpretation of the spectra of some flavonoids. Chem Pharm Bull. 30:1151–1156.
Gedara SR, Galala AA. 2015. New cytotoxic spirostane saponin and biflavonoid glycoside from the leaves of Acacia saligna (Labili) H.L Wendl. Nat Prod Res. 28:324–329.
Huang C, Ghavtadze N, Chattopadhay Y, Gevorgyan V. 2011. Synthesis of catechols from phenols via pd-catalyzed silanol-directed CH oxygenation. J Am Chem Soc. 133:17630–17633.
Karoune S, Falleh H, Kechbar MS, Halis Y, Mkadmini K, Belhama M, Rahmoune C, Ksouri R. 2015. Evaluation of antioxidant activities of the edible and medicinal Acacia albida organs related to phenolic compounds. Nat Prod Res. 29:452–454.
Lambert JD, Rice JE, Hong J, Hou Z, Yang CS. 2005. Synthesis and biological activity of the tea catechin metabolites, M4 and M6 and their methoxy-derivatives. Bioorg Med Chem Lett. 15:873–876.
Lee GH, Oh SJ, Lee SY, Lee JY, Ma JY, Kim YH, Kim SK. 2014. Discovery of soluble epoxide hydrolase inhibitors from natural products. Food Chem Toxicol. 64:225–230.
Li L, Seeram NP. 2010. Maple syrup phytochemicals include lignans, coumarins, stilbene, and other previously unreported antioxidant phenolic compounds. J Agr Food Chem. 58:11673–11679.
Li XC, Yang LX, Wang HQ, Chen RY. 2011. Phenolic compounds from the aqueous extract of Acacia catechu. Chin Chem Lett. 22:1331–1334.
Luo ZM, Du LX, Li LX, An MQ, Zhang ZZ, Wan XC, Bao GH, Zhang L, Ling TJ. 2013. Fuzhuanins A and B: the B-ring fission lactones of flavan-3-ols from fuzhuan brick-tea. J Agr Food Chem. 61:6982–6990.
Mutai C, Abatis d, Vagias C, Moreau D, Roussakis C, Roussis V. 2007. Lupane triterpenoids from Acacia mellifera with cytotoxic activity. Molecules 12:1035–1044.
Mujoo K, Haridas V, Hoffmann JJ, Waochter GA, Hutter L. 2011. Triterpenoid saponins from Acacia victoriae (Bentham) decrease tumor cell proliferation and induce apoptosis. Cancer Res. 61:5486–5490.
Moser d, Achenbach J, Klingler FM, Estella B, Hahn S, Proschak E. 2012. Evaluation of structure-derived pharmacophore of soluble epoxide hydrolase inhibitors by virtual screening. Bioorg Med Chem Lett. 22:6762–6765.
Ndidi US, Umar IA, Mohammed A, Samuel C, Oladeru AO, Yakubu RN. 2015. Effects of aqueous extracts of Acacia albida stem bark on Wistar albino rats infected with Trypanosoma evansi. Nat Prod Res. 29:1153–1156.
Pietta PG. 2000. Flavonoids as antioxidants. J Nat Prod. 63:1035–1042.
Readel K, Seigler D, Hwang K, Keesy J, Seilheimer S. 2001. Tannins from mimosoid legumes of Texas and México. Econ Bot. 55:212–222.
Rukachaisirikul V, Khamthong N, Sukpondma Y, Phongpaichit S, Towatana NH, Graidist P, Sakayaroj J, Kirtikara K. 2010. Cyclohexene, diketopiperazine, lactone and phenol derivatives from the sea fanned-derived fungi Nigrospora sp. PSU-F11 and PSU-F12. Arch Pharm Res. 33:375–380.
Singh BN, Singh BR, Singh RL, Prakash D, Dhakarey R, Upadhyay G, Sarma BK, Singh HB. 2009. Oxidative DNA damage protective activity, antioxidant and anti-quorum sensing potentials of Moringa oleifera. Food Chem Toxicol. 47:1109–1116.
Stohs SJ, Bagchi D. 2015. Antioxidant, anti-inflammatory, and chemoprotective properties of Acacia catechu heartwood extracts. Phytother Res. 29:818–824.