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Polyacetylenes from the Roots of Cultivated-Wild Ginseng and Their Cytotoxicity *In Vitro*

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Column chromatographic separation of the roots of cultivated-wild ginseng (Jangnoisam) led to the isolation of seven polyacetylenes (**1**-**7**). Their structures were determined by spectroscopic methods to be panaxynol (**1**), ginsenoyne-A (**2**), panaxydol (**3**), 10-methoxy heptadeca-1-ene-4, 6-dyne-3, 9-diol (**4**) (3*R*, 9*R*, 10*R*)-panaxytriol (**5**), panaxyne (**6**), and ginsenoyne-C (**7**). These compounds were isolated from this source for the first time. The compounds were tested for their cytotoxic activity against four human cancer cell lines (A549, SK-OV-3, SK-MEL-2, and HCT-15) *in vitro* using the SRB method. Panaxydol (**3**) and panaxyne (**6**) showed significant and selective cytotoxicity against SK-OV-3 with ED_{50} values 2.93 and 1.40 μM, respectively.

Key words: Korean cultivated-wild ginseng, Polyacetylene, Cytotoxicity

INTRODUCTION

The roots of *Panax ginseng* (Araliaceae) have been used for centuries as a tonic and as a remedy for a variety of pathological conditions. There have been many reports of the biological activities of *P. ginseng*, which has been shown to be antitumor (Ahn *et al*., 2006), anti-inflammatory (Lee *et al*., 2006), and antioxidant (Kang *et al*., 2007). Most studies of *P. ginseng* investigated the ginsenoside saponins (Baek *et al*., 1995; Kitagawa *et al*., 1989; Park *et al*., 1996). Recently, other constituents of the *Panax* species apart from saponin have been reported, in particular polyacetylenes (Matsunaga *et al*., 1990; Park *et al*., 1996). These polyacetylenes were shown to be cytotoxic (Matsunaga *et al*., 1990; Fujimoto *et al*., 1991), and anti-inflammatory (Lee *et al*., 2004; Ryu *et al*., 1998). Although ginsenosides and phenolic compounds have been isolated from Korean cultivated-wild ginseng (Jangnoisam) (Choi *et al.,* 2007; Kim *et al*., 2006), there have been no studies of the non-saponin constituents of Korean cultivated-wild ginseng (Jangnoisam). Therefore, we isolated seven polyacetylenes (**1**-**7**) by chromatography from the 70% EtOH extract of the roots of Korean cultivated-wild ginseng and tested their cytotoxic activity against four human cancer cell lines (A549, SK-OV-3, SK-MEL-2, and HCT-15) *in vitro* using the sulforhodamin B bioassay (SRB).

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MATERIALS AND METHODS

General experimental procedures

The optical rotations were determined using a Jasco P-1020 polarimeter (Jasco Co. Japan). The UV spectra were recorded on a Cary 5000 UV/Vis/NIR spectrometer (Varian Co., U.S.A.). The NMR spectra were recorded on a Bruker Biospin Advance 500 (Bruker Co., Germany). The ESI-MS data were obtained using an HP-1100 High Performance Liquid Chromatography/Quattro LC Triple Quadrupole Tandem Mass Spectrometer (Agilent Co., U.S.A.). The preparative HPLC was carried out over a Gemini® RP-C_{18} column (5 μ, 10×250 mm, Phenomenex Co., U.S.A.) using an RI detector (Shodex Co., Japan). Open column chromatography was carried out over silica gel (Silica gel 60, 70-230 mesh, Merck Co., Germany). Thin-layer chromatography (TLC) was performed on silica gel 60 F_{254} and RP-18 F_{254s} (Merck Co., Germany). The packing material

for molecular sieve column chromatography was Sephadex LH-20 (Pharmacia Co., Sweden). The packing material of open column chromatography was silica gel 60 RP C_{18} (40-63 μm, Merck Co., Germany). Low pressure liquid chromatography was carried out over a Lobar®-A glass prepacked column (Lichroprep® Si 60, Lichroprep® RP-18, 240×10 mm, 40-63 μm, Merck Co., Germany), an FMI QSY-0 pump (Fluid metering, Inc., U.S.A.), and a Duramat® 80 pump (CFG Prominent Co., Germany)

Plant material

Korean cultivated-wild ginseng (35.3 g) was supplied from the Korean Insam Association (Seoul, Korea) in July 2005.

In vitro cytotoxicity test

SRB was used to determine the cytotoxicity of the compounds. The cytotoxic activity of each compound against four cultured human tumor cell lines was examined at the Korea Research Institute of Chemical Technology. The cell lines were A549 (non small cell lung adenocarcinoma), SK-OV-3 (ovarian cancer cells), SK-MEL-2 (skin melanoma), and HCT-15 (colon cancer cells) (Skehan *et al.*, 1990).

Extraction and isolation

The dried roots of Korean cultivated-wild ginseng (35.3 g) were refluxed three times with 70% EtOH. The resulting 70% EtOH extract (8.0 g) was partitioned by solvent to give n-hexane (1.0 g), $CHCl_3$ (400 mg) and n-BuOH (1.6 g) soluble fractions. The n-hexane fraction (1.0 g) was chromatographed over Sephadex LH-20 (methylene chrolide:MeOH = 1:1) to give four fractions (H1-H4). The H3 fraction (600.0 mg) was subjected to silica gel column chromatography (n-hexane:EtOAc = 4:1) to give six fraction (H31-H36). The H33 fraction (200.0 mg) was applied to LiChroprep Lobar®-A Si 60 column chromatography (chloroform:EtOAc = 20:1) to give six fractions (H331-H336). The H331 fraction (80.0 mg) was purified using preparative HPLC (MeOH:H_2O = 85:15) to afford **1** (22.3 mg). The H332 fraction (35.0 mg) was purified using preparative HPLC (MeOH:H_2O = 75:25) to afford **2** (4.8 mg) and **3** (10.3 mg). The H34 fraction (100.0 mg) was purified using LiChroprep Lobar®-A Si 60 column chromatography (chloroform:acetone = 10:1) and preparative HPLC (MeOH :H_2O = 75:25) to afford **4** (3.1 mg). The H35 fraction (100.0 mg) was purified using LiChroprep Lobar®-A Si 60 column chromatography (chloroform:MeOH = 20:1) and preparative HPLC (MeOH:H_2O = 50:50) to give **5** (3.0 mg). The H4 fraction (50.0 mg) was purified using LiChroprep Lobar®-A Si 60 column chromatography (n-hexane:chloroform:EtOAc = 3:2:1) and preparative HPLC (C_{18} column, MeOH:H_2O = 70:30, 2.5 mL/min) to afford **6** (13.0 mg). The $CHCl_3$ fraction (400.0 mg) was chromatographed over a Sephadex LH-20 (methylene chrolide:MeOH = 1:1) to give four fractions (C1-C4). The C3 fraction (70.0 mg) was purified using LiChroprep Lobar®-A RP-18 column chromatography (MeOH:H_2O = 70:30) and preparative HPLC (MeOH:H_2O = 70:30) to afford **7** (5.0 mg).

Panaxynol (1)

Colorless oil, $[\alpha]_D$: -33.0 (c=2.1, MeOH), UV λ_{max} (MeOH) nm: 231, 243, 257, ESI-MS *m/z*: 267 $[M+Na]^+$, ^{1}H-NMR ($CDCl_3$, 500 MHz): δ 5.24 (1H, d, *J* = 10.1 Hz, H-1a), 5.46 (1H, d, *J* = 17.1 Hz, H-1b), 5.94 (1H, ddd, *J* = 17.1, 10.1, 5.1 Hz, H-2), 4.91 (1H, t, *J* = 5.1 Hz, H-3), 3.03 (2H, d, *J* = 6.9 Hz, H-8), 5.37 (1H, dt, *J* = 10.6, 6.9 Hz, H-9), 5.51 (1H, dt, *J* = 10.6, 7.1 Hz, H-10), 2.02 (1H, q, *J* = 7.1 Hz, H-11), 1.25-1.33 (10H, m, H-12-16), 0.88 (3H, t, *J* = 7.0 Hz, H-17), ^{13}C-NMR ($CDCl_3$, 125 MHz): δ 117.2 (C-1), 136.4 (C-2), 63.7 (C-3), 74.5 (C-4), 71.5 (C-5), 64.2 (C-6), 80.5 (C-7), 17.9 (C-8), 122.1 (C-9), 133.3 (C-10), 27.4 (C-11), 29.4 (C-12), 29.4 (C-13), 29.3 (C-14), 32.0 (C-15), 22.8 (C-16), 14.3 (C-17).

Ginsenoyne-A (2)

Colorless oil, $[\alpha]_D$: -80.8 (c=0.24, MeOH), UV λ_{max} (MeOH) nm: 216, 230, 242, 255, 268, 284, ESI-MS *m/z*: 281 $[M+Na]^+$, ^{1}H-NMR ($CDCl_3$, 500 MHz): δ 5.26 (1H, d, *J* = 10.2 Hz, H-1a), 5.47 (1H, d, *J* = 17.0 Hz, H-1b), 5.95 (1H, ddd, *J* = 17.0, 10.2, 5.6 Hz, H-2), 4.92 (1H, t, *J* = 5.6 Hz H-3), 2.38 (1H, dd, *J* = 17.7, 7.2 Hz, H-8a), 2.71 (1H, dd, *J* = 17.7, 5.4 Hz, H-8b), 3.15 (1H, ddd, *J* = 7.2, 5.4, 4.1 Hz, H-9), 2.98 (1H, dd, *J* = 6.0, 4.1 Hz, H-10), 1.40 (4H, m, H-11, 12), 1.49 (4H, m, H-13, 14), 2.07 (2H, q, 6.0 Hz, H-15), 5.81 (1H, ddd, *J* = 17.1, 10.0, 6.0 Hz, H-16), 4.94 (1H, d, *J* = 10.0 Hz, H-17a), 5.03 (1H, dd, *J* = 17.1 Hz, H-17b), ^{13}C-NMR ($CDCl_3$, 125 MHz): δ 117.4 (C-1), 136.2 (C-2), 63.7 (C-3), 75.1 (C-4), 66.5 (C-5), 71.1 (C-6), 76.9 (C-7), 19.7 (C-8), 54.5 (C-9), 57.1 (C-10), 27.7 (C-11), 26.5 (C-12), 29.1 (C-13), 29.0 (C-14), 33.8 (C-15), 139.1 (C-16), 114.6 (C-17).

Panaxydol (3)

Colorless oil, $[\alpha]_D$: -79.3 (c=1.01, MeOH), UV λ_{max} (MeOH) nm : 216, 230, 242, 255, 268, 284, ESI-MS *m/z*: 283 $[M+Na]^+$, ^{1}H-NMR ($CDCl_3$, 500 MHz): δ 5.25 (1H, d, *J* = 10.1 Hz, H-1a), 5.47 (1H, d, *J* = 17.0 Hz, H-1b), 5.94 (1H, ddd, *J* = 17.0, 10.1, 5.6 Hz, H-2), 4.92 (1H, br s, H-3), 2.38 (1H, dd, *J* = 17.7, 7.0 Hz, H-8a), 2.70 (1H, dd, *J* = 17.7, 5.4 Hz, H-8b), 3.15 (1H, ddd, *J* = 7.0, 5.4, 4.1 Hz, H-9), 2.97 (1H, dd, *J* = 6.0, 4.1 Hz, H-10), 1.52 (4H, m, H-11, 12), 1.34 (8H, m, H-13 - 16), 0.88 (3H, t, *J* = 7.0 Hz, H-17), ^{13}C-NMR ($CDCl_3$, 125 MHz): δ 117.4 (C-1), 136.2 (C-2), 63.7 (C-3), 75.1 (C-4), 71.1 (C-5), 66.5 (C-6), 76.9 (C-7), 19.6 (C-8), 54.5 (C-9), 57.2 (C-10), 27.7 (C-11), 26.7

(C-12), 29.6 (C-13), 29.4 (C-14), 31.9 (C-15), 22.8 (C-16), 14.3 (C-17).

10-Methoxy heptadeca-1-ene-4, 6-dyne-3, 9-diol (4)
Colorless oil, $[\alpha]_D$: +4.9 (c=0.16, MeOH), UV λ_{max} (MeOH) nm : 231, 243, 257, ESI-MS *m/z*: 315 $[M+Na]^+$, ^{1}H-NMR ($CDCl_3$, 500 MHz): δ 5.26 (1H, d, *J* = 10.1 Hz, H-1a), 5.47 (1H, d, *J* = 17.0 Hz, H-1b), 5.94 (1H, ddd, *J* = 17.0, 10.1, 5.8 Hz, H-2), 4.92 (1H, t, *J* = 5.8 Hz, H-3), 2.58 (1H, dd, *J* = 17.7, 7.0 Hz, H-8a), 2.56 (1H, dd, *J* = 17.7, 5.4 Hz, H-8b), 3.25 (1H, ddd, *J* = 7.0, 5.4, 4.1 Hz, H-9), 3.72 (1H, dd, *J* = 6.0, 4.1 Hz, H-10), 1.56 (2H, m, H-11), 1.30 (10H, m, H-12 - 16), 0.88 (3H, t, *J* = 7.0 Hz, H-17), 3.44 (3H, s, OCH_3), ^{13}C-NMR ($CDCl_3$, 125 MHz): δ 117.4 (C-1), 136.3 (C-2), 63.8 (C-3), 71.3 (C-4), 74.6 (C-5), 66.2 (C-6), 78.7 (C-7), 24.9 (C-8), 82.2 (C-9), 71.1 (C-10), 30.0 (C-11), 25.4 (C-12), 29.4 (C-13), 32.0 (C-14), 22.9 (C-15), 25.4 (C-16), 14.3 (C-17), 58.6 (OCH_3).

(3*R*, 9*R*, 10*R*)-Panaxytriol (5)
Colorless oil, $[\alpha]_D$: +12.9 (c=0.095, MeOH), UV λ_{max} (MeOH) nm : 231, 243, 257, ESI-MS *m/z*: 301 $[M+Na]^+$, ^{1}H-NMR ($CDCl_3$, 500 MHz): δ 5.26 (1H, d, *J* = 10.2 Hz, H-1a), 5.47 (1H, d, *J* = 17.0 Hz, H-1b), 5.94 (1H, ddd, *J* = 17.0, 10.1, 5.3 Hz, H-2), 4.92 (1H, t, *J* = 5.3 Hz, H-3), 2.59 (2H, br d, *J* = 5.4 Hz, H-8), 3.65 (1H, dd, *J* = 5.4, 4.6 Hz, H-9), 3.60 (1H, dd, *J* = 5.3, 4.6 Hz, H-10), 1.47 (2H, br t, *J* = 5.3 Hz, H-11), 1.30 (10H, m, H-12 - 16), 0.89 (3H, t, *J* = 7.0 Hz, H-17), ^{13}C-NMR ($CDCl_3$, 125 MHz): δ 117.4 (C-1), 136.2 (C-2), 63.7 (C-3), 74.9 (C-4), 71.1 (C-5), 66.7 (C-6), 78.3 (C-7), 25.8 (C-8), 72.3 (C-9), 73.3 (C-10), 33.8 (C-11), 25.2 (C-12), 29.7 (C-13), 29.4 (C-14), 32.0 (C-15), 22.9 (C-16), 14.3 (C-17).

Panaxyne (6)
Colorless oil, $[\alpha]_D$: +21.9 (c=0.63, MeOH), UV λ_{max} (MeOH) nm: 227, 238, 252, ESI-MS *m/z*: 243 $[M+Na]^+$, ^{1}H-NMR ($CDCl_3$, 500 MHz): δ 2.02 (1H, s, H-1), 2.56 (2H, br s, H-5), 3.66 (1H, s, H-6), 3.60 (1H, s, H-7), 1.51 (2H, m H-8), 1.36 (6H, m, H-9 - 11), 2.07 (2H, m, H-12), 5.82 (1H, ddd, *J* = 17.0, 10.0, 6.7 Hz, H-13), 5.00 (1H, dd, *J* = 17.0, 1.6 Hz, H-14a), 4.93 (1H, dd, *J* = 10.0, 0.73 Hz, H-14b), ^{13}C-NMR ($CDCl_3$, 125 MHz): δ 65.6 (C-1), 68.2 (C-2), 67.1 (C-3), 74.6 (C-4), 25.0 (C-5), 72.3 (C-6), 73.2 (C-7), 33.7 (C-8), 25.6 (C-9), 29.2 (C-10), 29.0 (C-11), 33.9 (C-12), 139.2 (C-13), 114.6 (C-14).

Ginsenoyne-C (7)
Colorless oil, $[\alpha]_D$: -20.0 (c=0.1, MeOH), UV λ_{max} (MeOH) nm : 219, 231, 243, 257, ESI-MS *m/z* : 299 $[M+Na]^+$, ^{1}H-NMR ($CDCl_3$, 500 MHz): δ 5.26 (1H, d, *J* = 10.2 Hz, H-1a), 5.47 (1H, d, *J* = 17.0 Hz, H-1b), 5.94 (1H, ddd, *J* = 17.0, 10.2, 5.6 Hz, H-2), 4.92 (1H, br t, *J* = 5.6 Hz H-3), 2.59 (2H, brs, H-8), 3.65 (1H, dd, *J* = 5.8, 4.3 Hz, H-9), 3.60 (1H, dd, *J* = 5.6, 4.6 Hz, H-10), 1.51 (2H, m, H-11), 1.41 (6H, m, H-12-14), 2.05 (2H, q, *J* = 6.0 Hz, H-15), 5.81 (1H, ddd, *J* = 17.2, 10.0, 6.6 Hz, H-16), 4.94 (1H, d, *J* = 10.0 Hz, H-17a), 5.01 (1H, d, *J* = 17.2 Hz, H-17b), ^{13}C-NMR ($CDCl_3$, 125 MHz): δ 117.2 (C-1), 136.2 (C-2), 63.5 (C-3), 74.8 (C-4), 70.9 (C-5), 66.5 (C-6), 78.1 (C-7), 25.0 (C-8), 72.2 (C-9), 73.1 (C-10), 33.5 (C-11), 25.4 (C-12), 28.8 (C-13), 29.0 (C-14), 33.7 (C-15), 139.1 (C-16), 114.6 (C-17).

RESULTS AND DISCUSSION

The structures of the polyacetylenes (**1**-**7**) were identified by comparison of their spectral data (^{1}H-, ^{13}C-NMR, and MS) with those reported in the literature. These compounds were isolated from Korean cultivated-wild ginseng for the first time.

Compound **1** was obtained as a colorless oil. The UV spectrum of **1** showed typical absorption bands for a enediyne chromophore (λ_{max} at 231, 243, and 257 nm) (Hirakura *et al*., 1992; Fujimoto *et al*., 1994). The ESI-MS spectrum of **1** showed a quasimolecular ion peak at *m/z* 267 $[M+Na]^+$. The ^{1}H-NMR spectrum of **1** showed the presence of ABX system protons [δ 5.24 (1H, d, *J* = 10.1 Hz, H-1a), 5.46 (1H, d, *J* = 17.1 Hz, H-1b), 5.94 (1H, ddd, *J* = 17.1, 10.1, 5.1 Hz, H-2)], AB system *cis* olefinic protons [δ 5.37 (1H, dt, *J* = 10.6, 6.9 Hz, H-9), 5.51 (1H, dt, *J* = 10.6, 7.1 Hz, H-10)], and an oxygenated proton [δ 4.91 (1H, t, *J* = 5.1 Hz, H-3)]. Four diyne carbons were exhibited in the ^{13}C-NMR spectrum at δ 74.5 (C-4), 71.5 (C-5), 64.2 (C-6), and 80.5 (C-7). Based on the above considerations and on a comparison with data in the literature (Hirakura *et al*., 1992), the structure of **1** was identified as panaxynol.

Compound **2** was obtained as a colorless oil. The ESI-MS spectrum of **2** showed a quasimolecular ion peak at *m/z* 281 $[M+Na]^+$. The main differences in the ^{1}H-NMR spectrum of **2** compared to **1** were the presence of two pairs of ABX system protons [δ 5.26 (1H, d, *J* = 10.2 Hz, H-1a), 5.47 (1H, d, *J* = 17.0 Hz, H-1b), 5.95 (1H, ddd, *J* = 17.0, 10.2, 5.6 Hz, H-2), 5.81 (1H, ddt, *J* = 17.1, 10.0, 6.0 Hz, H-16), 4.94 (1H, d, *J* = 10.0 Hz, H-17a), and 5.03 (1H, d, *J* = 17.1 Hz, H-17b)], and two protons of epoxide ring [δ 3.15 (1H, ddd, *J* = 7.2, 5.4, 4.1 Hz, H-9), 2.98 (1H, dd, *J* = 6.0, 4.1 Hz, H-10)]. Based on the above considerations and a comparison with the data in the literature (Hirakura *et al*., 1991), the structure of **2** was identified as ginsenoyne A.

Compound **3** was obtained as a colorless oil. The ESI-MS spectrum of **3** showed a quasimolecular ion peak at *m/z* 283 $[M+Na]^+$. The ^{1}H- and ^{13}C-NMR spectra of **3** were similar to those of **2**, except for the replacement of a vinyl group [δ_H 5.81 (1H, ddd, *J* = 17.1, 10.0, 6.0 Hz, H-16), 4.94

(1H, d, J = 10.0 Hz, H-17a), and 5.03 (1H, d, J = 17.1 Hz, H-17b), δ_C 139.1 (C-16), 114.6 (C-17)] with a methyl group [δ_H 0.88 (3H, t, J = 7.0 Hz, H-17), δ_C 14.3 (C-17)] in **3**. Based on the above considerations and a comparison with the data in the literature (Hirakura *et al*., 1994), the structure of **3** was identified as panaxydol.

Compound **4** was obtained as a colorless oil. The ESI-MS spectrum of **4** showed a quasimolecular ion peak at m/z 315 $[M+Na]^+$. The ^{1}H- and ^{13}C-NMR spectra of **4** were similar to those of **3**, except for the replacement of the epoxide ring with a hydroxyl and an methoxy group [δ_H 3.44 (3H, s), δ_C 58.6]. Based on the above considerations and a comparison with the data in the literature (Fujimoto *et al*., 1991, Ahn *et al*., 1988), the structure of **4** was identified as 10-methoxy heptadeca-1-ene-4, 6-dyne-3, 9-diol.

Compound **5** was obtained as a colorless oil. The ESI-MS spectrum of **5** showed a quasimolecular ion peak at m/z 301 $[M+Na]^+$. The ^{1}H- and ^{13}C-NMR spectra of **5** were

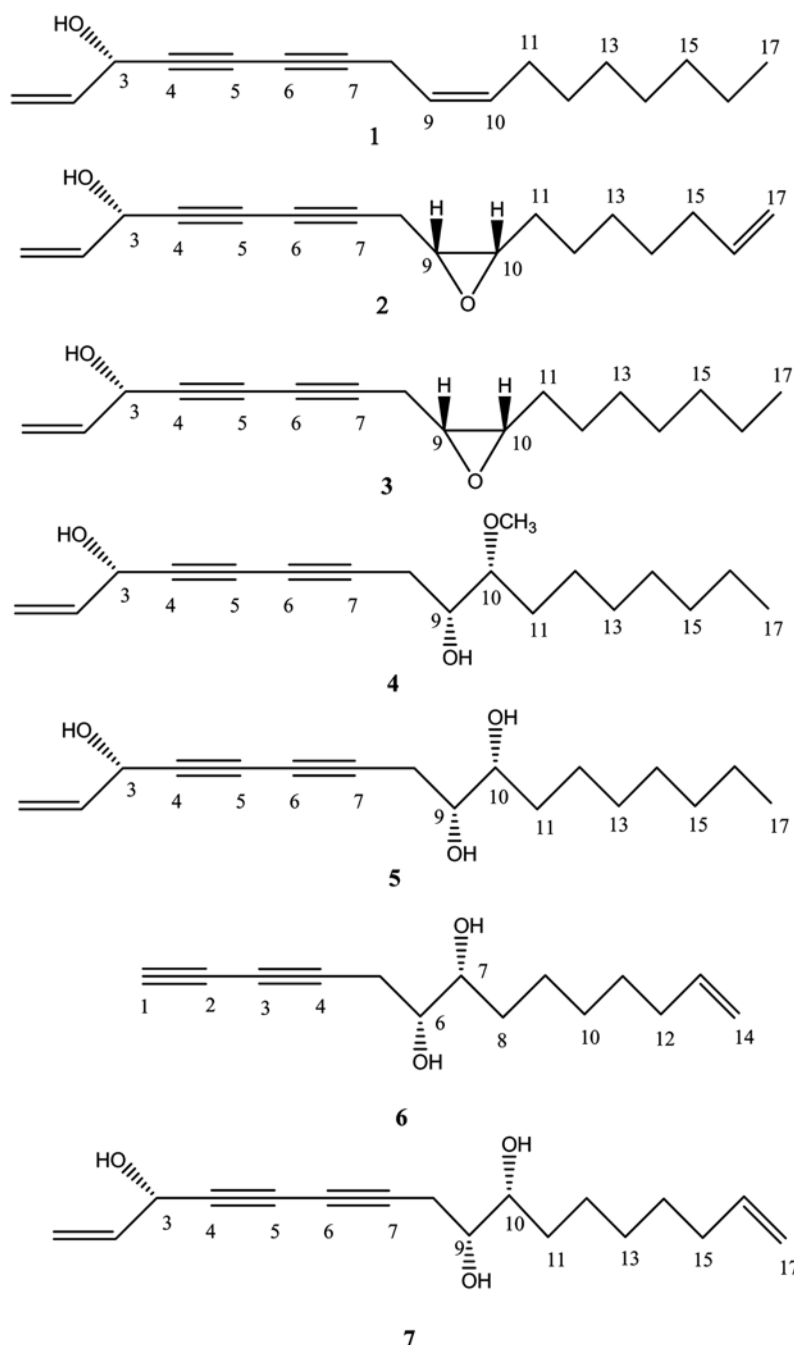

Fig. 1. Structures of polyacetylenes (**1**-**7**) from cultivated-wild ginseng

Table I. Cytotoxic activity of polyacetylenes (**1-7**)*

Compound	A549	SK-OV-3	SK-MEL-2	HCT-15
panaxynol (**1**)	6.04	2.38	4.06	4.04
ginsenoyne-A (**2**)	5.95	2.14	3.94	4.11
panaxydol (**3**)	10.40	2.93	4.05	3.94
10-methoxy heptadeca-1-ene-4, 6-dyne-3, 9-diol (**4**)	17.59	14.36	13.85	14.09
(3R, 9R, 10R)-panaxytriol (**5**)	22.84	19.04	22.61	22.70
panaxyne (**6**)	13.92	1.40	11.84	6.59
ginsenoyne-C (**7**)	13.64	14.07	13.16	13.52

*ED_{50} values of compounds against each cancer cell line, which was defined as the concentration (μM) that caused 50% inhibition of cell growth in vitro.

similar to those of **4**, except for the replacement of a methoxy group with a hydroxyl group. Based on the above considerations and a comparison with the data in the literature (Mayer *et al*., 2002), the structure of **5** was identified as (3*R*, 9*R*, 10*R*)-panaxytriol.

Compound **6** was obtained as a colorless oil. The ESI-MS spectrum of **6** showed a quasimolecular ion peak at *m/z* 243 $[M+Na]^+$. The ^{1}H-NMR spectrum showed the presence of ABX system protons [δ 5.82 (1H, ddd, *J* = 17.0, 10.0, 6.7 Hz, H-13), 5.00 (1H, dd, *J* = 17.0, 1.6 Hz, H-14a), 4.93 (1H, dd, *J* = 10.0, 0.73 Hz, H-14b)], six methylene protons [δ 2.56 (2H, br s, H-5), 1.51 (2H, m H-8), 1.36 (6H, m, H-9 - 11), 2.07 (2H, m, H-12)], two oxygenated protons [δ 3.66 (1H, s, H-6), 3.60 (1H, s, H-7)], and one terminal methyne proton [δ 2.02 (1H, s, H-1)]. Four diyne carbons were exhibited in the ^{13}C-NMR spectrum at δ 65.6 (C-1), 68.2 (C-1), 67.1 (C-3), 74.6 (C-4). Based on the above considerations and a comparison with the data in the literature (Kim *et al*., 1989), the structure of **6** was identified as panaxyne.

Compound **7** was obtained as a colorless oil. The ESI-MS spectrum of **7** showed a quasimolecular ion peak at *m/z* 299 $[M+Na]^+$. The ^{1}H- and ^{13}C-NMR spectra of **7** were similar to those of **2**, except for the replacement of an epoxide ring [δ_H 3.15 (1H, ddd, *J* = 7.2, 5.5, 4.1 Hz, H-9), 2.98 (1H, dd, *J* = 6.0, 4.1 Hz, H-10), δ_C 54.5 (C-9), 57.1 (C-10)] with two hydroxyl group [δ_H 3.65 (1H, ddd, *J* = 5.8, 4.3 Hz, H-9), 3.60 (1H, ddd, *J* = 5.6, 4.6 Hz, H-10), δ_C 72.2 (C-9), 73.1 (C-10)]. Based on the above considerations and a comparison with the data in the literature (Hirakura *et al*., 1991), the structure of **7** was identified as ginsenoyne-C.

Compounds **1-7** were evaluated for their cytotoxic activity against four human cancer cell lines (A549, SK-OV-3, SK-MEL-2, and HCT-15) by SRB (Table I). Panaxynol (**1**) and ginsenoyne-A (**2**) were considerably cytotoxic against the four cancer cell lines tested. In addition, panaxydol (**3**) and panaxyne (**6**) showed significant selective activity against SK-OV-3 with ED_{50} values of 2.93 and 1.40 μM, respectively.

Panaxynol and panaxydol isolated from the roots of *Panax ginseng* have previously been shown to have a good cytotoxic effect against human gastric adenocarcinoma cells (MK-1), mouse malignant melanoma cells (B-16) and mouse fibroblast-derived tumor cells (L-929) with ED_{50} values of 0.027, 1.23, and 2.50 μg/mL, respectively (Matsunaga *et al*., 1990). Panaxyne isolated from *Panax ginseng* was reported to be cytotoxic against L1210 cells with an ED_{50} value of 11.0 μg/mL (Kim *et al*., 1989).

Based on the above results, the polyacetylenes in the Korean cultivated-wild ginseng (Jangnoisam) could be main constituent group which posess the antitumoral effect.

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