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Studies on the Constituents of *Solanum* Plants. V.[1] The Constituents of *S. lyratum* THUNB. II

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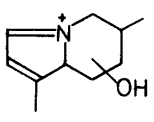
Two new steroidal alkaloid glycosides, tentatively named SL-c (**1**) and SL-d (**2**), were obtained from the stems of *Solanum lyratum* THUNB. The chemical structures of **1** and **2** were characterized respectively as β-lycotrioside and β-lycotetraoside, both having a mixture of (25ξ)-solanidan-3β,23β-diol and (25ξ)-Δ^5-solaniden-3β,23β-diol as the aglycone moiety. These compounds markedly inhibited the growth of a human cervical cancer cell line, JTC-26.

Keywords——*Solanum lyratum*; Solanaceae; steroidal alkaloid glycoside; solanidan-3β,23β-diol; JTC-26

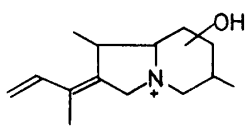
An oriental medicine, Ryuda-yōsentō (竜蛇羊泉湯),[2] consisting of three herbs, the whole plants of *Solanum lyratum* THUNB., *S. nigrum* L. and *Duchesnea indica* (Andr.) FOCKE, has traditionally been used as a remedy for various cancers in the Shanghai region of China. In addition, European *Solanum dulcamara* L. ("bittersweet" or "woody nightshade") in the same genus as *S. lyratum* has been used for the treatment of cancers and warts[3] from the time of Galen, and references to its use have appeared in the literature in many countries.[3] In 1965, Kupchan *et al.*[3] isolated β-solamarine[4] as an anti-tumor substance from *S. dulcamara*.

We have now obtained two steroidal alkaloid glycosides, tentatively named SL-c (**1**) and SL-d (**2**) from the stems of *S. lyratum*, in addition to two neutral steroid glycosides, SL-a and SL-b,[5] which were reported in the preceding paper.[6] This paper deals with the structural characterization of the above two substances. Extraction and separation were achieved as described in Chart 1 of the previous paper,[6] and recrystallization of fr. 8 afforded SL-c (3.4 g, 0.31%), while further separation of fr. 11 gave SL-d (1.8 g, 0.16%).

SL-c (**1**), colorless needles from MeOH, mp 222—225 °C (dec.), $[\alpha]_D$ −51.5° (pyridine), was positive to the Dragendorff reagent. Measurement of the electron-impact mass spectrum (EI-MS) of **1** without[7] obtaining a volatile derivative showed a fragmentation pattern with peaks at *m/z* 415 ($C_{27}H_{45}NO_2^+$), 413 ($C_{27}H_{43}NO_2^+$), 220 ($C_{14}H_{22}NO^+$), 166 ($C_{10}H_{16}NO^+$), which are characteristic[8] of the solanidane skeleton and furthermore indicated the presence of a hydroxyl group in ring E or F (Chart 1). The field desorption mass spectrum (FD-MS)



m/z 166 ($C_{10}H_{16}NO^+$)



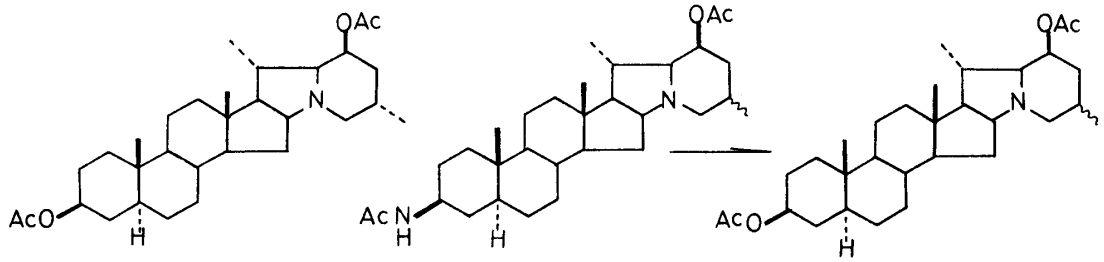
m/z 220 ($C_{14}H_{22}NO^+$)

Chart 1

showed molecular ions at *m/z* 901 and 899 (intensity 901 > 899) suggesting that SL-c (**1**) was composed of an aglycone of the solanidane type and 3 mol of hexose, and that the aglycone part consisted of at least two substances. Acid hydrolysis of SL-c (**1**) gave the aglycone and sugar fractions, the latter of which was identified as a mixture of glucose and galactose on thin layer chromatography (TLC). The aglycone (**3**), colorless needles, mp 216—219 °C, $[\alpha]_D$ +37.1° ($CHCl_3$), showed a signal at δ 5.40 characteristic of 6-H on a Δ^5-steroid framework, but it integrated at less than one proton (*ca.* 1/4H) in the proton nuclear magnetic resonance (^{1}H-NMR) spectrum. The carbon-13 nuclear magnetic resonance (^{13}C-NMR) spectrum[9] of **3** indicated the presence of a mixture of Δ^5 and the corresponding dihydro compound (Table II), in a ratio of approximately 1 : 3 based on their intensities. Catalytic reduction (Pd-black/H_2) of **1** followed by hydrolysis with mineral acid yielded **4**, colorless needles, mp 222—224 °C, $[\alpha]_D$ +38.9° ($CHCl_3$), whose EI-MS exhibited a molecular ion at *m/z* 415, suggesting that **3** was completely hydrogenated. Acetylation of **4** in the usual way gave the diacetate (**4a**), whose EI-MS showed a molecular ion at *m/z* 499. The ^{1}H-NMR spectrum of **4a** showed a signal (br s, $W_{h/2}$ = 8 Hz) at δ 5.01 adjacent to the acetoxyl group together with other signals assignable to 10-Me (3H, s, δ 0.83), 13-Me (3H, s, δ 0.90), 20-Me (3H, d, δ 0.93), 25-Me (3H, d, δ 1.10), two acetoxyl groups (6H, s, δ 2.01) and 3-H (1H, m, δ 4.66) as listed in Table I. The

Table I. ^{1}H-NMR Chemical Shifts of **4a**, **5a** and **6a** (in $CDCl_3$)

	4a	Dihydroleptinidine diacetate (**5a**)	Solanogantamine diacetate (**6a**)
10-Me	0.83 (s)	0.83 (s)	0.80 (s)
13-Me	0.90 (s)	0.86 (s)	0.90 (s)
20-Me	0.93 (d, J = 6 Hz)	—	0.93 (d, J = 6 Hz)
25-Me	1.10 (d, J = 6 Hz)	—	1.13 (d, J = 6 Hz)
3-H	4.66 (m)	4.68 (hept, J = 5, 10 Hz, $W_{h/2}$ = 23 Hz)	3.70 (m)
23-H	5.01 ($W_{h/2}$ = 8 Hz)	5.07 (m, $W_{h/2}$ = 6 Hz)	5.03 ($W_{h/2}$ = 7 Hz)
26-H(β)	2.68 (d, J = 10 Hz)	2.5—3.0 (m)	2.67 (d, J = 11 Hz)
OAc	2.01 (s) × 2	2.00 (s), 2.04 (s)	2.02 (s)
NAc			1.93 (s)



dihydroleptinidin diacetate (**5a**) — solanogantamine diacetate (**6a**) — **7a**

Chart 2

signal pattern at δ 5.01 closely resembled that assigned to 23α-H of dihydroleptinidine diacetate[10] (**5a**), suggesting that **4** possesses a *sec*-hydroxyl group at C-23β. Furthermore, although the whole signal pattern of **4a** was analogous to that of **5a**, a significant difference between the two spectra was apparent. Pakrashi *et al.*[11] obtained solanogantamine (**6**) from *Solanum giganteum* JACQ. and transformed into the corresponding 3β-hydroxyl compound (**7**), whose diacetate (**7a**) was not identical with **5a** in terms of the infrared (IR) spectrum.

Thus, they[11] concluded that the configuration at C-25 in **6** might be different from that of **5**. Since the ^{1}H-NMR spectrum of **4a** was found to exhibit a good coincidence with that of solanogantamine diacetate (**6a**) as shown in Table I, the diacetate **4a** was considered to be probably identical with **7a**. However, authentic specimens of **5**, **5a**, **7** and **7a** were not available for measurement of the NMR (^{1}H and ^{13}C) spectra, and comparison of the physical constants of **4** and **4a** with those of **5**, **5a**, **7** and **7a** did not allow us to arrive at a definite conclusion. The ^{13}C-NMR spectra of **3** and **4** were assigned[9] as shown in Table II. As regards the sugar moiety, all the signals attributable to the sugar residue of **1** were superimposable on those of SL-b as listed in Table III, and thus it was concluded that **1** possessed the same sugar residue as SL-b.

Consequently, the chemical structure of SL-c (**1**) was shown to be the 3-*O*-*β*-D-glucopyranosyl-(1→2)-*β*-D-glucopyranosyl-(1→4)-*β*-D-galactopyranoside of a mixture of (25*ξ*)-solanidan-3*β*,23*β*-diol and (25*ξ*)-Δ^5-solaniden-3*β*,23*β*-diol.

Next, SL-d (**2**), mp 242—246 °C (dec.), $[\alpha]_D$ −62.3° (pyridine) was hydrolyzed with 2 N HCl–MeOH to furnish the same aglycone as obtained from SL-c (**1**), and glucose, galactose and xylose as sugar components. The methyl ether (**8**) was prepared from **2** by the Kuhn method.[12] Its EI-MS showed peaks due to terminal permethylated-hexosyl and -pentosyl cations at *m/z* 219 and 175, respectively. The methyl ester (**8**) was then methanolyzed with 1 N HCl–MeOH to yield a mixture of methylated sugars consisting of methyl 2,3,4,6-tetra-*O*-

Table II. ^{13}C-NMR Chemical Shifts of **3** and **4**

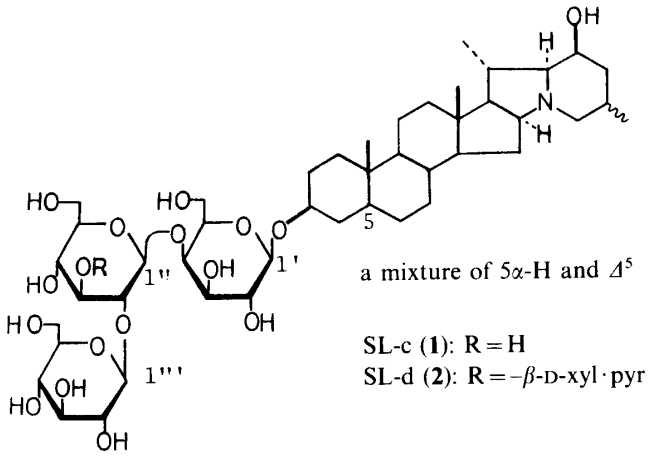
	3		**4**
	5α-H	Δ^5	
C-1	37.1	37.3	37.0
C-2	31.5	31.5	31.5
C-3	71.2	71.6	71.2
C-4	38.2	42.3	38.2
C-5	45.0	141.0	45.0
C-6	28.7	121.3	28.7
C-7	32.3	32.1	32.3
C-8	35.4	31.7	35.4
C-9	54.5	50.2	54.5
C-10	35.6	36.6	35.6
C-11	21.0	20.8	21.0
C-12	39.6	39.4	39.6
C-13	41.4	41.1	41.4
C-14	57.4	57.7	57.4
C-15	31.5	31.5	31.5
C-16	69.6	69.6	69.6
C-17	62.2	62.2	62.2
C-18	16.8	16.6	16.8
C-19	12.4	19.4	12.4
C-20	30.6	30.6	30.6
C-21	18.9	18.9	18.9
C-22	78.9	78.9	78.9
C-23	67.0	67.0	67.0
C-24	37.1	37.1	37.0
C-25	26.9	26.9	26.9
C-26	58.7	58.7	58.7
C-27	22.4	22.4	22.4

In $CDCl_3$.

TABLE III. ^{13}C-NMR Chemical Shifts of the Sugar Moieties of SL-c (**1**) and SL-b

		SL-c (**1**)	SL-b
Galactose	C-1′	102.3	102.3
	C-2′	73.1	73.1
	C-3′	75.4	75.4
	C-4′	80.8	80.6
	C-5′	76.5	76.4
	C-6′	60.4	60.5
Glucose (inner)	C-1″	105.0	104.8
	C-2″	85.8	85.6
	C-3″	78.0	78.1
	C-5″	71.7	71.7
	C-5″	77.4	77.9
	C-6″	61.4	61.5
Glucose	C-1‴	106.7	106.6
	C-2‴	74.9	74.9
	C-3‴	78.2	78.7
	C-4‴	70.2	70.3
	C-5‴	77.4	77.4
	C-6‴	63.1	62.9

In d_5-pyridine.



a mixture of 5α-H and Δ^5

SL-c (**1**): R = H
SL-d (**2**): R = -β-D-xyl·pyr

Chart 3

methyl α-D-glucopyranoside, methyl 2,3,4-tri-*O*-methyl α,β-D-xylopyranoside, methyl 2,3,6-tri-*O*-methyl α,β-D-galactopyranoside and methyl 4,6-di-*O*-methyl α,β-D-glucopyranoside on TLC. The appearance of four anomeric carbon signals at δ 103.2, 105.4, 2 × 105.5 in the ^{13}C-NMR spectrum of **2** (Table IV) indicated **2** to be a tetraglycoside. Taking into consideration the result of methanolysis, it seems most probable that **2** possesses a β-lycotetraosyl residue. The ^{13}C-NMR spectrum of **2** was therefore compared with that of desgalactotigonin,[13] having a β-lycotetraoside as the sugar moiety, and the ^{13}C-NMR chemical shifts due to the sugar part were found to be superimposable on each other (Table IV).

Consequently, the structure of SL-d (**2**) was characterized as the 3-*O*-β-D-glucopyranosyl-(1→2)-[β-D-xylopyranosyl-(1→3)]-β-D-glucopyranosyl-(1→4)-β-D-galactopyranoside of a mixture of (25ξ)-solanidan-3β,23β-diol and (25ξ)-Δ^5-solaniden-3β,23β-diol.

The inhibitory activities of SL-a, SL-b, SL-c (**1**) and SL-d (**2**) against JTC-26[13b,14] (cells

Table IV. ^{13}C-NMR Chemical Shifts of the Sugar Moieties of SL-d (**2**) and Desgalactotigonin

		SL-d (**2**)	Desgalactotigonin[13]
Galactose	C-1′	102.5	102.4
	C-2′	73.1	73.1
	C-3′	75.0	75.0
	C-4′	79.8	79.7
	C-5′	76.0	76.0
	C-6′	60.6	60.6
Glucose (inner)	C-1″	104.7	104.8
	C-2″	81.2	81.1
	C-3″	87.0	87.0
	C-4″	70.4	70.6
	C-5″	77.5	77.5
	C-6″	62.4	63.1
Glucose	C-1‴	104.8	104.8
	C-2‴	75.3	75.2
	C-3‴	78.3	78.5
	C-4‴	70.7	71.1
	C-5‴	77.8	77.8
	C-6‴	63.0	63.1
Xylose	C-1⁗	104.8	104.8
	C-2⁗	75.5	75.5
	C-3⁗	78.5	78.5
	C-4⁗	70.4	70.4
	C-5⁗	67.2	67.2

In d_5-pyridine.

Table V. Inhibitory Activity against JTC-26 Cell Growth

(μg/ml)	SL-a (%)	SL-b (%)	SL-c (**1**) (%)	SL-d (**2**) (%)
0.5	—	—	−20.0—0.0	−15.0—0.0
1.0	0.0	—	−43.0——30.0	−31.0——20.0
3.0	−4.0	—	−114.0——40.0	−82.0——35.0
5.0	−8.0	25.0	−85.0——71.4	−65.0——55.0
8.0	−4.0	—	100.0	100.0
10.0	−4.0	75.0	100.0	100.0
15.0	−4.0	100.0	100.0	100.0
20.0	−4.0	100.0	100.0	100.0

originating from human cervical cancer) were studied and the results are listed in Table V. The steroidal alkaloids, SL-c (**1**) and SL-d (**2**), exhibited significant inhibition of cell growth. The spirostanol glycoside, SL-b, also exhibited inhibitory activity. It is quite interesting that the furostanol glycoside corresponding to SL-b, *i.e.*, SL-a, did not exhibit inhibitory activity. Various *in vivo* antitumor tests and an examination of toxicity to normal cells are currently under study.

Experimental

Melting points were determined on a Yanagimoto micromelting point apparatus (hot-stage type) and are uncorrected. The optical rotations were measured with a Union PM-201 automatic polarimeter. The IR spectra were

recorded on a Hitachi 215 spectrometer. The NMR spectra were run on a JEOL PS-100 spectrometer and a JEOL FX-200 spectrometer (100 and 200 MHz for ^{1}H-NMR and 50.1 MHz for ^{13}C-NMR). Chemical shifts are given on a δ (ppm) scale with tetramethylsilane as an internal standard. The EI-MS and FD-MS were recorded on a JEOL JMS D-300 spectrometer. TLC was performed on precoated Kieselgel 60 plates (Merck) and detection was achieved by spraying 10% H_2SO_4 followed by heating.

SL-c (1)——Colorless needles from MeOH, mp 222—225 C (dec.), $[\alpha]_D^{22}$ −51.5° (c=0.54, pyridine). Dragendorff reagent: positive. IR ν_{max}^{KBr} cm^{-1}: 3400 (OH). EI-MS m/z: 415 ($C_{27}H_{45}NO_2^+$), 413 ($C_{27}H_{43}NO_2^+$), 220 ($C_{14}H_{22}NO^+$), 166 ($C_{10}H_{16}NO^+$, base peak). FD-MS m/z: 901, 899 (M^+, intensity 901>899). ^{13}C-NMR (d_5-pyridine): Table III.

Acid Hydrolysis of 1——A solution of **1** (260 mg) in 2 N HCl–MeOH (10 ml) was refluxed for 2 h, and water (30 ml) was added. The deposited precipitates were collected by filtration and recrystallized from dil. MeOH to afford **3** as colorless needles (52 mg). mp 216—219 °C, $[\alpha]_D^{22}$ +37.1° (c=0.89, $CHCl_3$). IR ν_{max}^{KBr} cm^{-1}: 3455 (OH), EI-MS m/z: 415 ($C_{27}H_{45}NO_2^+$), 413 ($C_{27}H_{43}NO_2^+$), 371 ($C_{25}H_{41}NO^+$), 344 ($C_{23}H_{38}NO^+$), 342 ($C_{23}H_{36}NO^+$), 220 ($C_{14}H_{22}NO^+$), 166 ($C_{10}H_{16}NO^+$), 148 ($C_{10}H_{14}N^+$). ^{1}H-NMR ($CDCl_3$) δ: 0.84 (3H, s, 10-Me), 0.89 (3H, s, 13-Me), 1.02 (3H, d, J=7 Hz, 20-Me), 1.23 (3H, d, J=7 Hz, 25-Me), 5.40 (*ca.* 1/4H, m, 6-H). ^{13}C-NMR ($CDCl_3$): Table II. The filtrate was concentrated to 1/4 of the initial volume, heated on a hot bath and passed through Amberlite IRA-400 to give the sugar solution, which was examined by TLC (solv. $CHCl_3$–MeOH–acetone–H_2O=3:3:3:1) to detect glucose (Rf 0.34) and galactose (Rf 0.30).

Catalytic Hydrogenation Followed by Acidic Hydrolysis of 1——A solution of **1** (110 mg) in EtOH (25 mg) was hydrogenated over Pd-black (30 mg) in an H_2 gas flow for 2.5 h at room temperature. The catalyst was filtered off and the filtrate was evaporated to dryness *in vacuo* to give the residue (105 mg), which was hydrolyzed with 2 N HCl–MeOH (10 ml), neutralized with 5% KOH–MeOH and concentrated to a small volume. This concentrate was shaken with $CHCl_3$–H_2O, and the organic layer was evaporated to dryness *in vacuo* to give the residue, which was recrystallized from dil. MeOH to give **4** as colorless needles (22 mg). mp 222—224 °C, $[\alpha]_D^{22}$ +38.9° (c=0.94, $CHCl_3$) [in lit. dihydroleptinidine[10] (**5**), mp 215 °C, $[\alpha]_D$ +32° ($CHCl_3$), **7**,[11] mp 215—216 °C, $[\alpha]_D$ +40.4° ($CHCl_3$)], EI-MS m/z: 415 ($C_{27}H_{45}NO_2^+$), 371 ($C_{25}H_{41}NO^+$), 344 ($C_{23}H_{38}NO^+$), 220 ($C_{14}H_{22}NO^+$), 166 ($C_{10}H_{16}NO^+$), 148 ($C_{10}H_{14}N^+$, base peak). ^{13}C-NMR ($CDCl_3$): Table II. *Anal.* Calcd for $C_{27}H_{45}NO_2$: C, 78.02; H, 10.91. Found: C, 78.23; H, 10.97.

Acetylation of 4——**4** (18 mg) was dissolved in Ac_2O–pyridine (each 3 ml), and the solution was heated at 70 °C for 1 h then evaporated to dryness *in vacuo* to give the residue, which was purified by silica gel column chromatography (solv. *n*-hexane–acetone=3:1). The product was recrystallized from dil. MeOH to yield the diacetate (**4a**) as colorless needles (12 mg). mp 209—213 °C, $[\alpha]_D^{22}$ +13.1° (c=0.76, $CHCl_3$) [in lit. **5a**,[10] mp 222—223 °C, $[\alpha]_D$ +2.95° ($CHCl_3$), **7a**,[11] mp 212—214 °C, $[\alpha]_D$ +8.5° ($CHCl_3$)]. ^{1}H-NMR ($CDCl_3$): Table I. *Anal.* Calcd for $C_{31}H_{49}NO_4$: C, 74.51; H, 9.88. Found: C, 74.62; H, 9.81.

SL-d (2)——Colorless needles from dil. MeOH. mp 242—246 °C (dec.). $[\alpha]_D^{23}$ −62.3° (c=0.72, pyridine). Dragendorff reagent: positive. IR ν_{max}^{KBr} cm^{-1}: 3400 (OH). ^{13}C-NMR (d_5-pyridine): Table IV.

Acid Hydrolysis of 2——A solution of **2** (37 mg) in 2 N HCl–MeOH (5 ml) was refluxed for 1.5 h on a hot bath, then neutralized with 3% KOH–MeOH. The deposited salt was filtered off and the filtrate was passed through Sephadex LH-20 with MeOH to furnish the aglycone (11 mg), which was identical with **3** in terms of mp, EI-MS and ^{13}C-NMR ($CDCl_3$), and methyl α-D-glucopyranoside (Rf 0.26), methyl α-D-galactopyranoside (Rf 0.25) and methyl α,β-D-xylopyranoside (Rf 0.42, 0.47), which were identical with authentic specimens on TLC (solv. $CHCl_3$–MeOH–H_2O=7:3:0.5).

Methylation of 2——A suspension of **2** (19 mg) in *N*, *N*-dimethylformamide (DMF) (1 ml), Ag_2O (300 mg) and CH_3I (5 ml) was stirred overnight at room temperature. The reaction mixture was filtered and washed with acetone, and the filtrate was evaporated to dryness *in vacuo* to give the residue, which was subjected to silica gel column chromatography (solv. the lower phase of $CHCl_3$–MeOH–AcOEt–H_2O=2:2:5:1) to afford the methyl ether (**8**) as a syrup (9 mg). EI-MS m/z: 219 (hexose 4×Me), 187 (m/z 219−MeOH), 175 (pentose 3×Me), 143 (m/z 175−MeOH).

Methanolysis of 8——A solution of **9** (5 mg) in 2 N HCl–MeOH (1 ml) was refluxed for 2 h and neutralized with 3% KOH–MeOH. The products were examined by TLC (solv. *n*-hexane–AcOEt=2:3) to detect methyl 2,3,4-tri-*O*-methyl α,β-D-xylopyranoside (Rf 0.57, 0.42), methyl 2,3,4,6-tetra-*O*-methyl α-D-glucopyranoside (Rf 0.43), methyl 2,3,6-tri-*O*-methyl α,β-D-galactopyranosidepyranoside (Rf 0.11, 0.09) and methyl 4,6-di-*O*-methyl α,β-D-glucopyranoside (Rf 0.06).

Test of the Activity of SL-a, SL-b, SL-c (1) and SL-d (2) against JTC-26——Mixtures of 1×10^5 JTC-26 cells (originating from human cervical cancer) per ml (90% Eagle's MEM, 10% Feta-1 calf serum (Microbiological) with 0.5, 1.0, 3.0, 5.0, 8.0, 10.0, 15.0 or 20.0 μg/ml of SL-a, SL-b, SL-c (**1**) and SL-d (**2**) were prepared, and four samples of each (total volume 20 ml each) were placed in Petri dishes (100×15 mm) (Lux Co.). Blank samples which did not contain the above-mentioned compounds were also prepared. The samples were cultured in a CO_2 incubator at 37 °C for 144 h. The number of live cells was counted and the average was compared with the blank to determine the growth inhibition ratio. The results are summarized in Table V.

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