Supporting Information

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Bioactive Alkaloids from the Beibu Gulf Coral-associated Fungus *Acremonium sclerotigenum* GXIMD 02501

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General experimental procedures

The NMR spectra were obtained on a Bruker Avance III 700 spectrometer (Bruker BioSpin, Fällanden, Switzerland) using TMS as an internal standard. HR-ESIMS spectra were collected on a Waters Xevo G2-S TOF mass spectrometer (Waters Corporation, USA). TLC and column chromatography (CC) were performed on plates precoated with silica gel GF254 (10–40 μm) and over silica gel (200–300 mesh) (Qingdao Marine Chemical Factory, China), respectively. All solvents employed were of analytical grade (Tianjin Damao Chemical and Industry Factory, China). Semi-preparative high-performance liquid chromatography (Semiprep HPLC) was performed on a Shimadzu Prominence-I LC 2030 system (Shimadzu, Tokyo, Japan), equipping with an ODS column (YMC-pack ODS-A, YMC Co. Ltd., Japan, 10 × 250 mm, 5 μm, 2.5 mL/min). The artificial sea salt was a commercial product (Guangzhou Haili Aquarium Technology Company, China).

Fungal fermentation and isolation

A large-scale fermentation of the strain GXIMD 02501 was further carried out in the Czapek medium (saccharose 3.0%, KCl 0.05%, $FeSO_4 \cdot 7H_2O$ 0.05%，$NaNO_3$ 0.3%，KH_2PO_4 0.1%，NaBr 3.0%, pH 7.4) employing with 300 mL × 100 Erlenmeyer flasks (1

L) at room temperature for 60 days. Then all the cultures were overlaid and extracted with EtOAc to yield a brown extract (32 g), which was further separated into 18 subfractions (Frs. L1~L18) via ODS silica gel chromatography eluting with MeOH/H_2O (10~100%). Fr. L8 was purified by semipreparative HPLC (58% MeOH/H_2O, 2 mL/min, 220 nm) to provide **4** (6 mg, t_R 22 min). Fr. L12 was purified by semipreparative HPLC (75% MeCN/H_2O, 2 mL/min, 220 nm) to provide **2** (2.4 mg, t_R 12 min). Fr. L16 was purified by semipreparative HPLC (73% CH_3CN/H_2O, 2 mL/min, 220 nm) to provide **1** (2.5 mg, t_R 21 min). Fr. L17 was purified by semipreparative HPLC (75% MeCN/H_2O, 2 mL/min, 220 nm) to provide **3** (42 mg, t_R23 min).

*Campyridone D (**1**)*: light yellow oil. ^{1}H NMR (700 MHz, $CDCl_3$) δ_H 12.53 (1H, s, H-1), 8.19 (1H, s, H-6), 7.40 (2H, d, J = 8.4 Hz, H-2′, 6′), 6.91 (2H, d, J = 8.4 Hz, H-3′, 5′), 5.57 (1H, dq, J = 15.5, 6.5 Hz, H-21), 5.37 (1H, dd, J = 15.5, 8.4 Hz, H-20), 4.05 (1H, d, J = 13.2 Hz, H-17), 3.17 (1H, dd, J = 13.2, 10.7 Hz, H-8), 2.02 (1H, m, H-11a), 0.40 (1H, m, H-11b), 1.97 (1H, m, H-9), 1.92 (1H, m, H-14a), 1.37 (1H, m, H-14b), 1.80 (1H, m, H-13a), 0.85 (1H, m, H-13b), 1.74 (3H, dd, J = 6.5, 1.8 Hz, H_3-22), 1.48 (3H, s, H_3-18), 1.45 (1H, m, H-10), 1.32 (1H, m, H-12), 0.93 (1H, m, H-15), 0.89 (3H, d, J = 6.5 Hz, H_3-19). ^{13}C NMR (175 MHz, $CDCl_3$) δ_C 202.2 (C, C-7), 166.3 (C, C-2), 164.5 (C, C-4), 155.7 (C, C-4′), 154.0 (CH, C-6), 133.0 (CH, C-20), 130.4 (CH, C-2′, 6′), 126.6 (CH, C-21), 125.6 (C, C-1′), 121.1 (C, C-5), 115.7 (CH, C-3′, 5′), 102.1 (C, C-3), 84.6 (CH, C-17), 71.9 (C, C-16), 48.5 (C, C-8), 46.9 (CH, C-15), 43.2 (CH, C-9), 39.6 (CH, C-11), 39.2 (CH_2, C-10), 35.0 (CH_2, C-13), 32.1 (CH, C-12), 24.9 (CH_2, C-14), 23.0 (CH_3, C-18), 22.7 (CH_3, C-19), 18.3 (CH_3, C-22). HR-ESI-MS *m/z* 450.2297 $[M + H]^+$ (calcd for $C_{27}H_{32}NO_5$, 450.2280), 472.2108 $[M + Na]^+$ (calcd for $C_{27}H_{31}NNaO_5$, 472.2100).

*Campyridone A (**2**)*: light yellow oil. ^{1}H NMR (700 MHz, CD_3OD) δ_H 7.77 (1H, s, H-6), 7.39 (2H, d, J = 8.6 Hz, H-2′, 6′), 6.88 (2H, d, J = 8.7 Hz, H-3′,5′), 5.45 (1H, dq, J = 15.3, 6.5 Hz, H-21), 5.18 (1H, s, H-17), 5.09 (1H, dd, J = 15.3, 9.4 Hz, H-20), 2.46 (1H, dd, J = 11.9, 9.4 Hz, H-9), 2.15 (1H, m, H-14a), 1.83 (1H, m, H-13a), 1.79 (3H, s, H_3-18), 1.76 (1H, m, H-15), 1.74 (1H, m, H-11a), 1.55 (1H, m, H-10), 1.51 (3H, d, J = 6.5 Hz,

H_3-22), 1.45 (1H, m, H-12), 1.12 (1H, m, H-14b), 1.03 (1H, m, H-13b), 0.92 (3H, d, *J* = 6.6 Hz, H_3-19), 0.59 (1H, m, H-11b). ^{13}C NMR (175 MHz, CD_3OD) δ_C 201.2 (C, C-7), 182.6 (C, C-4), 160.2 (C, C-2), 158.8 (C, C-4′), 150.1 (C, C-16), 143.2 (C, C-6), 131.7 (CH, C-21), 130.1 (CH, C-2′, 6′), 127.3 (CH, C-20), 123.6 (C, C-1′), 117.1 (CH, C-17), 116.7 (CH, C-3′ ,5′), 112.2 (C, C-5), 108.3 (C, C-3), 94.9 (C, C-8), 51.2 (CH, C-9), 46.0 (CH, C-15), 41.1 (CH, C-10), 40.1 (CH_2, C-11), 36.4 (CH_2, C-13), 33.7 (CH, C-12), 30.3 (CH_2, C-14), 22.9 (CH_3, C-19), 21.2 (CH_3, C-18), 18.1 (CH_3, C-22). HR-ESI-MS *m/z* 432.2177 $[M + H]^+$ (calcd for $C_{27}H_{32}NO_5$, 432.2175), 454.1993 $[M + Na]^+$ (calcd for $C_{27}H_{29}NNaO_4$, 454.1994), 470.1733 $[M + K]^+$ (calcd for $C_{27}H_{29}NNaO_4$, 470.1734).

*Ilicicolin H (**3**)*: pale yellow solid; ^{1}H NMR (700 MHz, CD_3OD): δ_H 7.44 (1H, s, H-6), 4.93 (1H, m, H-8), 2.49 (1H, q, *J* = 10.5 Hz, H-9), 1.15 (1H, m, H-10), 0.51 (1H, q, *J* = 11.8 Hz, H-11a), 1.74 (1H, m, H-11b), 1.30 (1H, m, H-12), 0.90 (1H, m, H-13a), 1.71 (1H, m, H-13b), 1.97 (1H, m, H-14a), 0.90 (1H, m, H-14b), 1.60 (1H, overlapped, H-15), 5.24 (1H, br s, H-17), 1.57 (3H, s, H_3-18), 0.86 (3H, d, *J* = 6.5 Hz, H_3-19), 5.15 (1H, m, H-20), 5.33 (1H, m, H-21), 1.51 (3H, d, *J* = 6.5 Hz, H_3-22), 6.81 (2H, d, *J* = 8.4 Hz, H-3′/5′), 7.24 (2H, d, *J* = 8.4 Hz, H-2′/6′); ^{13}C NMR (175 MHz, CD_3OD): δ_C 164.1 (qC, C-2), 108.1 (qC, C-3), 176.9 (qC, C-4), 115.8 (qC, C-5), 140.6 (CH, C-6), 211.0 (qC, C-7), 54.8 (CH, C-8), 46.3 (CH, C-9), 44.6 (CH, C-10), 40.6 (CH_2, C-11), 33.8 (CH, C-12), 36.6 (CH_2, C-13), 30.9 (CH_2, C-14), 45.6 (CH, C-15), 139.2 (qC, C-16), 120.6 (CH, C-17), 21.3 (CH_3, C-18), 23.2 (CH_3, C-19), 134.4 (CH, C-20), 127.3 (CH, C-21), 18.2 (CH_3, C-22), 125.0 (qC, C-1′), 116.1 (CH, C-3′/5′), 131.3 (CH, C-2′/6′), 158.1 (qC, C-4′). HR-ESI-MS *m/z* 434.2329 $[M + H]^+$ (calcd for $C_{27}H_{32}NO_5$, 434.2331), 456.2140 $[M + Na]^+$ (calcd for $C_{27}H_{29}NNaO_4$, 456.2151).

*Phenazine-1-carboxylic acid (**4**)*:light yellow solid. ^{1}H NMR (700 MHz, $CDCl_3$) δ_H 15.60 (1H, s, 1-COOH), 8.99 (1H, dd, *J* = 7.0, 1.4 Hz, H-2), 8.54 (1H, dd, *J* = 8.7, 1.4 Hz, H-4), 8.36 (1H, dd, *J* = 8.2, 1.1 Hz, H-9), 8.30 (1H, dd, *J* = 8.6, 0.8 Hz, H-6), 8.07–7.97 (3H, overlapped, H-3, H-7, H-8). ^{13}C NMR (175 MHz, $CDCl_3$) δ_C 166.1 (C, 1-COOH), 144.3 (C, C-10a), 143.6 (C, C-9a), 140.2 (C, C-5a), 140.0 (C, C-4a), 137.6 (CH, C-3),

135.3 (CH, C-4), 133.4 (CH, C-9), 131.9 (CH, C-6), 130.4 (CH, C-2), 130.2 (CH, C-8), 128.1 (CH, C-7), 125.1 (C, C-1). HR-ESI-MS *m/z* 207.0556 $[M - H_2O + H]^+$ (calcd for $C_{13}H_7N_2O$, 207.0558), 225.0664 $[M + H]^+$ (calcd for $C_{13}H_9N_2NaO_2$, 225.0664).

Bioassay

NF-*κ*B luciferase reporter gene assay

The isolated compounds (**1**−**4**) were tested for their inhibitory activities of LPS-induced NF-*κ*B activation in RAW264.7 cells by NF-*κ*B luciferase reporter gene assay [1, 2]. RAW264.7 cells stably transfected with a NF-κB luciferase reporter gene are kindly provided by Professor Xu (University of Western Australia, Nedlands, Australia), which were pretreated with these compounds (10 μM) and BAY11-7082 (NF-*κ*B inhibitor, 5 μM, Sigma-Aldrich) as positive control in 96-well plates for 30 min, followed by 5 μg/mL LPS stimulation for 8 h. Data were expressed as the mean ± SD and analyzed using GraphPad Prism 7.0 software (San Diego, CA, USA). Statistical differences among groups were performed using one-way analysis of variance (ANOVA) with Bonferroni *post-hoc* test. A level of $p < 0.05$ was considered statistically significant.

Cytotoxicity Assay

The cytotoxicity of compounds **1**–**4** against PC-3 and 22Rv1 cell lines was tested by MTT assay [3, 4]. In brief, PC-3 and 22Rv1 cells were provided from national collection of authenticated cell cultures, which were cultured in RPMI1640 and DMEM media, respectively, supplemented with 10% fetal bovine serum (FBS) and 1 × penicillin/streptomycin (Gibco). Cells were cultured at 37 °C in a humidified incubator containing 5% CO_2, which were then seeded in 96-well plates at 500–1000 cells per well (optimum density for growth) in a total volume of 100 μL of media. Serially diluted compounds (50 μL) in 250 μL of media were added to the cells. After 4 days of incubation, Cell-Titer GLO reagents (Promega) were added, and luminescence was measured on GLOMAX microplate luminometer (Promega), according to the manufacturer´s instructions. Docetaxel was used as a positive control with the IC_{50} values of 0.12 and

0.030 μM for PC-3 and 22Rv1 cells, respectively.

Anti-*Vibrio* Assay

Anti-*Vibrio* effects against a panel of marine biofilm-forming bacterial strains, including *Vibrio parahemolyticus*, *V. alginolyticus*, *V. owensii*, and *V. coralliilyticus*, were tested by using a K–B disc agar diffusion method [1, 5]. Chloramphenicol was used as a positive control with the same MIC value of 0.67 μg/mL.

Molecular Docking

The Schrödinger 2019-4 suite (Schrödinger Inc., New York, NY, USA) was employed to perform the docking study as reported previously [1]. The crystal structure of human NF-κB p65 was obtained from Protein Data Bank (http://www.pdb.org) (PDB code: 3GUT, chain A). The initial structure of protein was first automatically corrected by "Protein Preparation" module. Then, the binding site was putatively similar to the pocket of HIV-1 LTR, which was included in the crystal structure. The ligands were then flexibly docked to the pocket by the Glide module with standard precision mode. The docking pose with best glide score was chosen for presenting the bind mode of molecule. The PyMOL software (DeLano Scientific, Palo Alto, CA, USA) was used to obtain the 3D structures of the binding models.

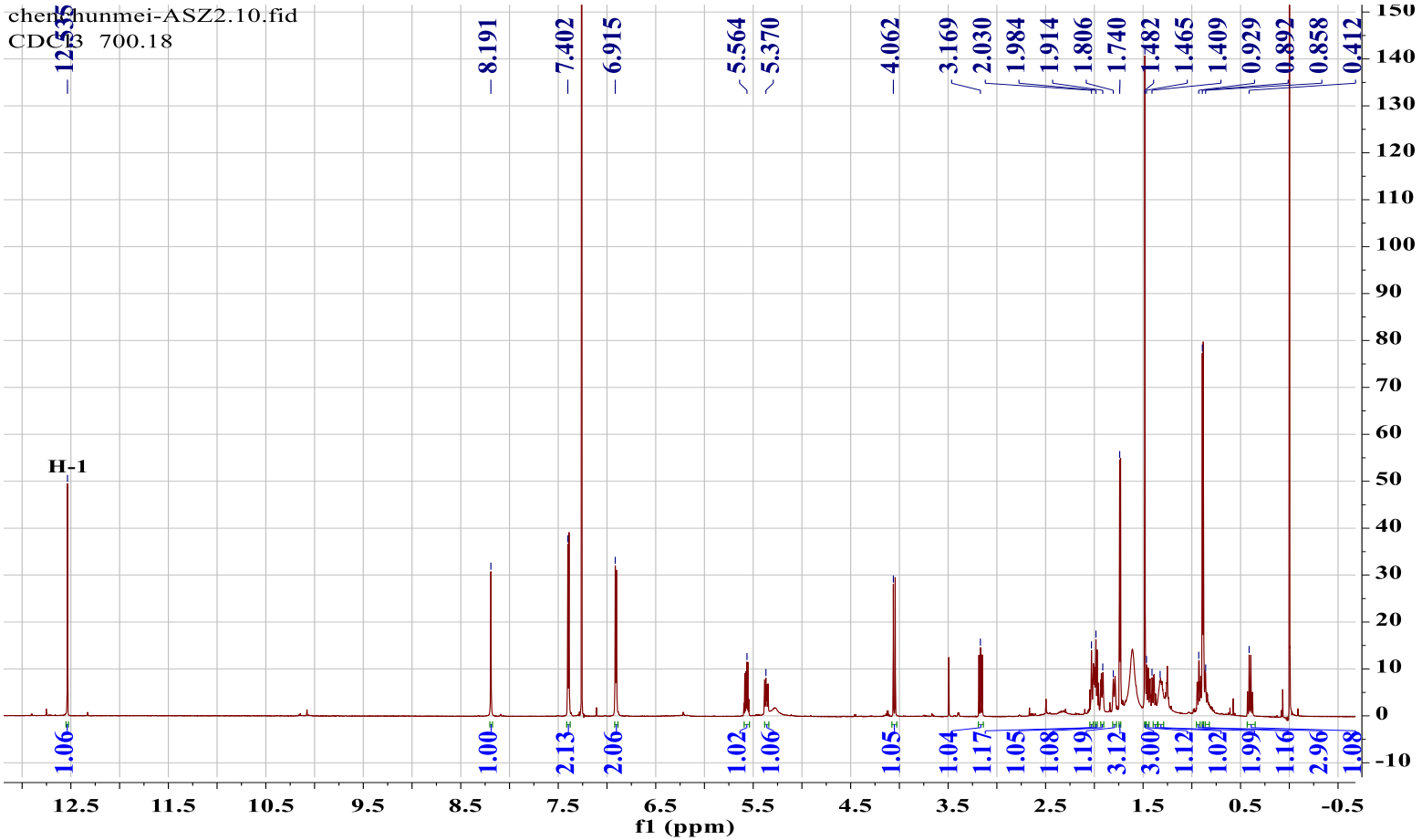


Figure S1: ^{1}H NMR spectrum of campyridone D (**1**) ($CDCl_3$, 700 MHz).

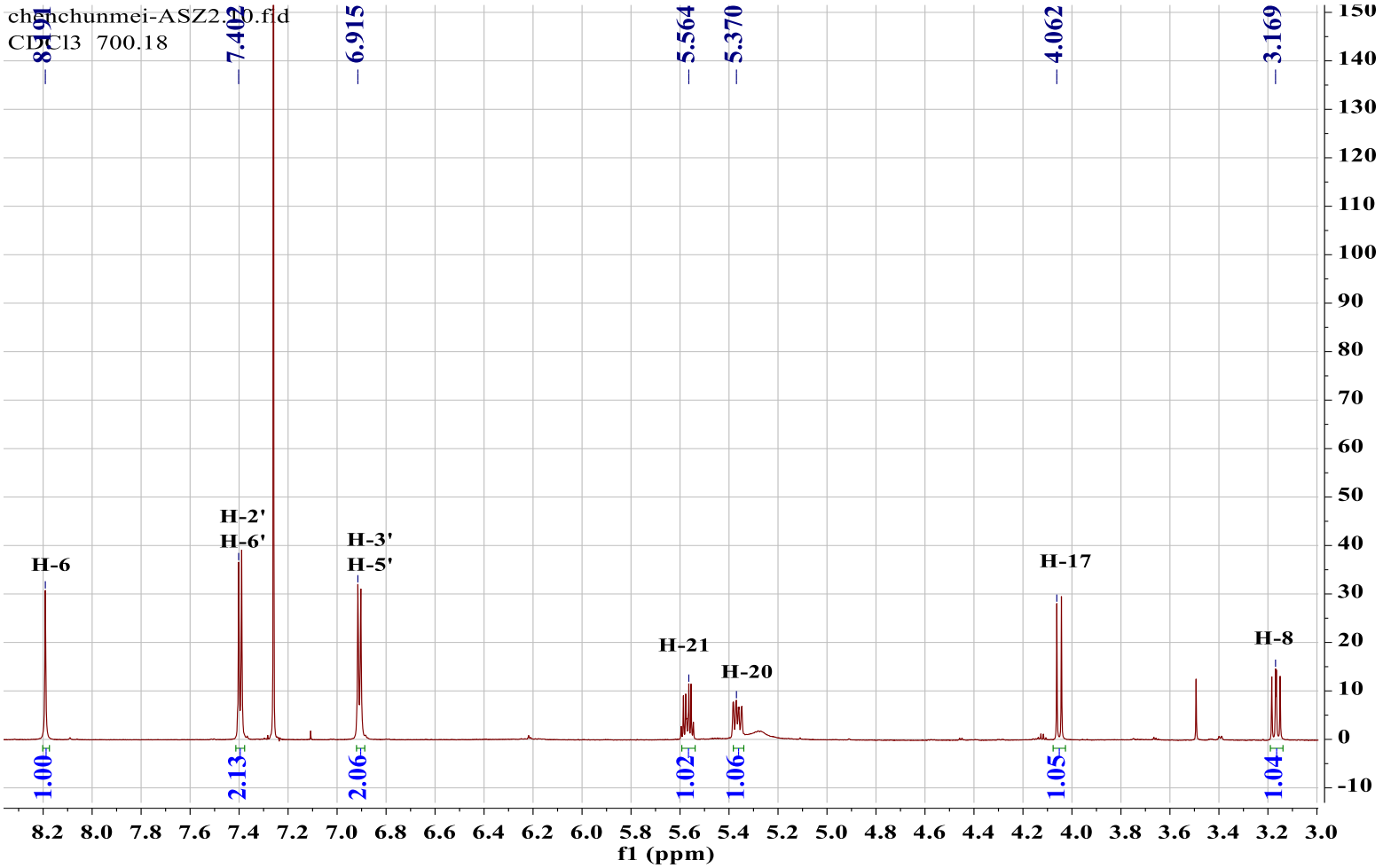


Figure S2: ^{1}H NMR spectrum of campyridone D (**1**) ($CDCl_3$, 700 MHz) (From δ_H 3.0 ppm to 8.3 ppm)

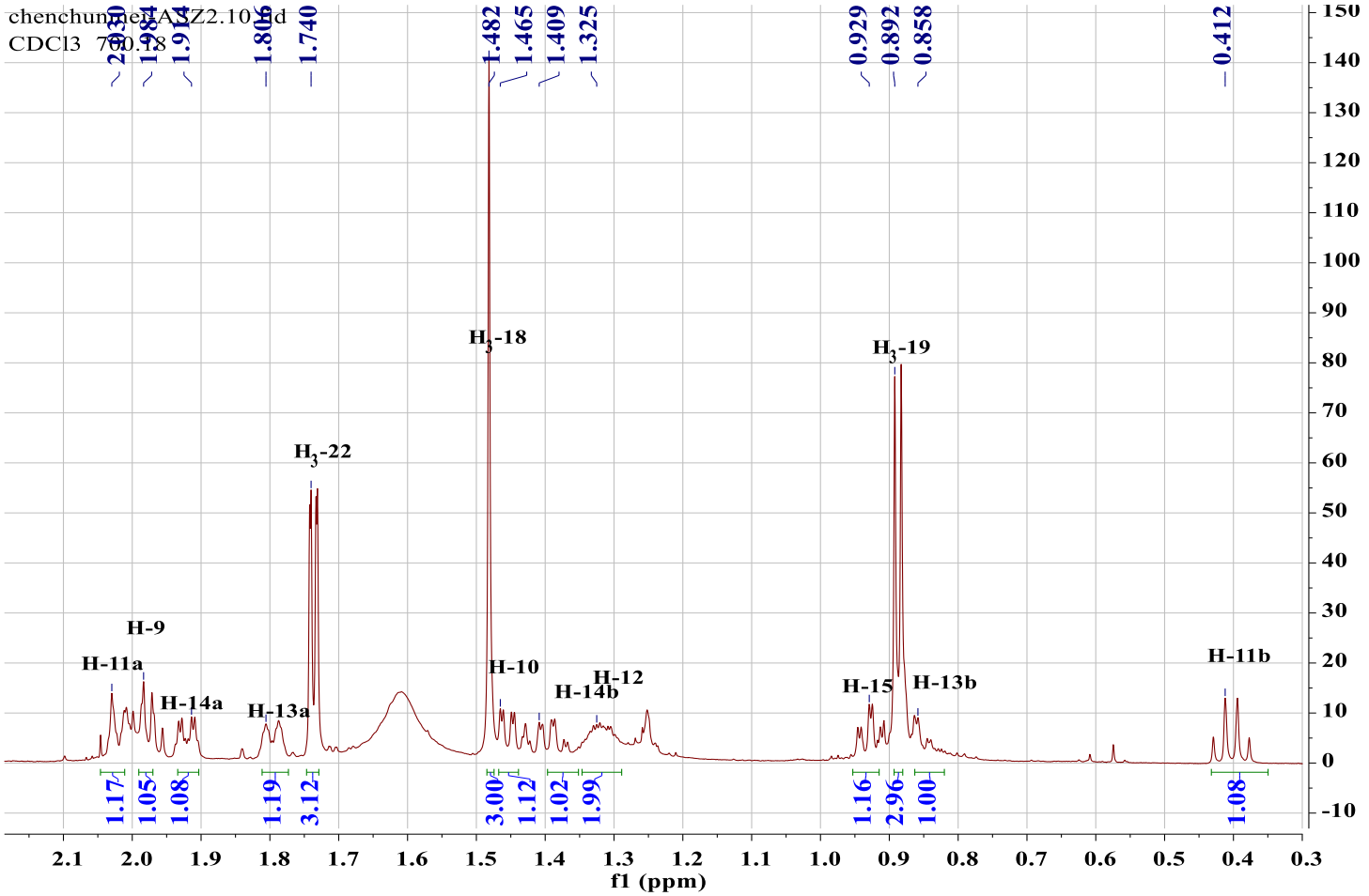


Figure S3: ^{1}H NMR spectrum of campyridone D (**1**) ($CDCl_3$, 700 MHz) (From δ_H 0.3 ppm to 2.2 ppm).

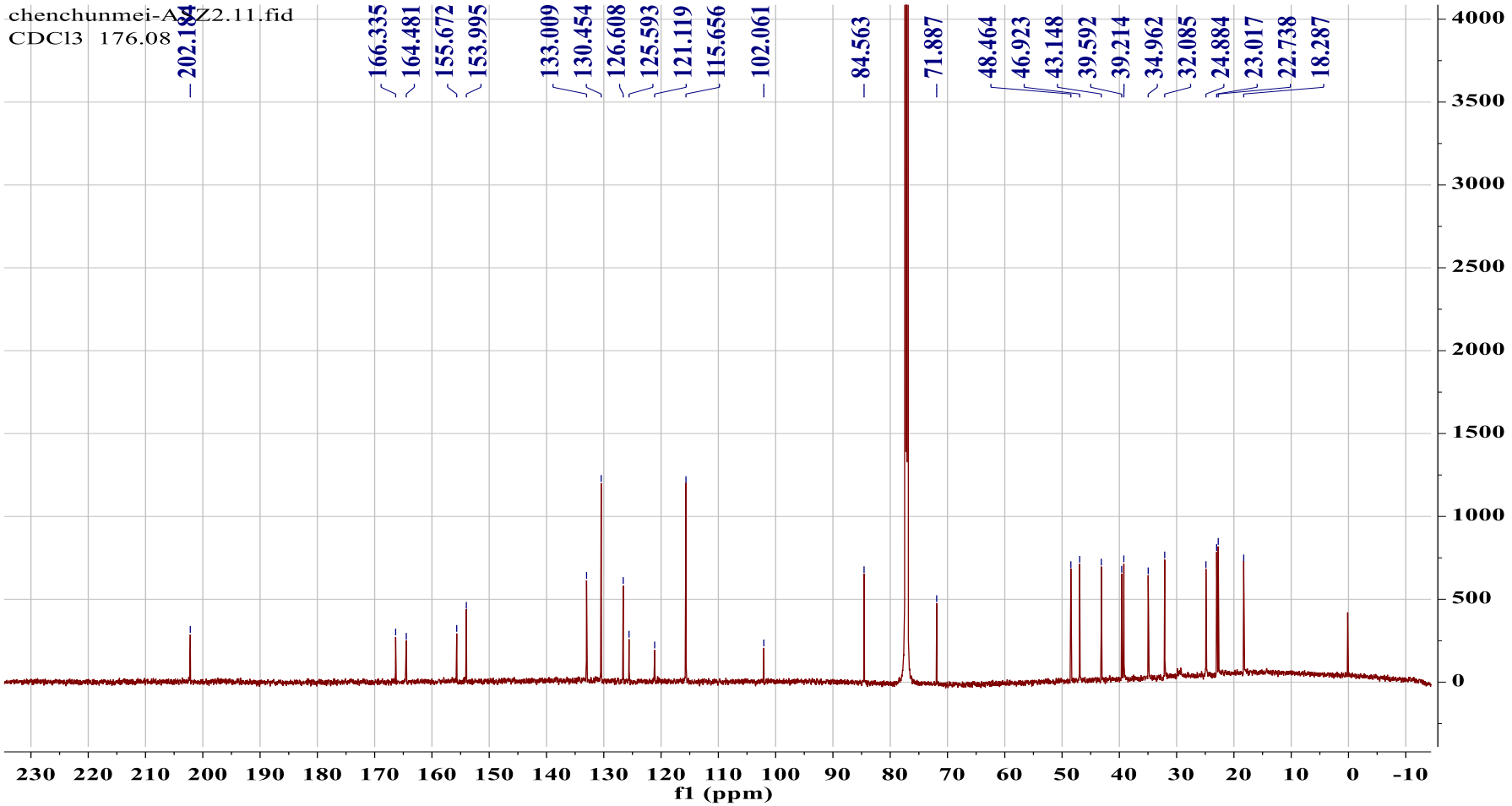


Figure S4: ^{13}C NMR spectrum of campyridone D (**1**) ($CDCl_3$, 175 MHz).

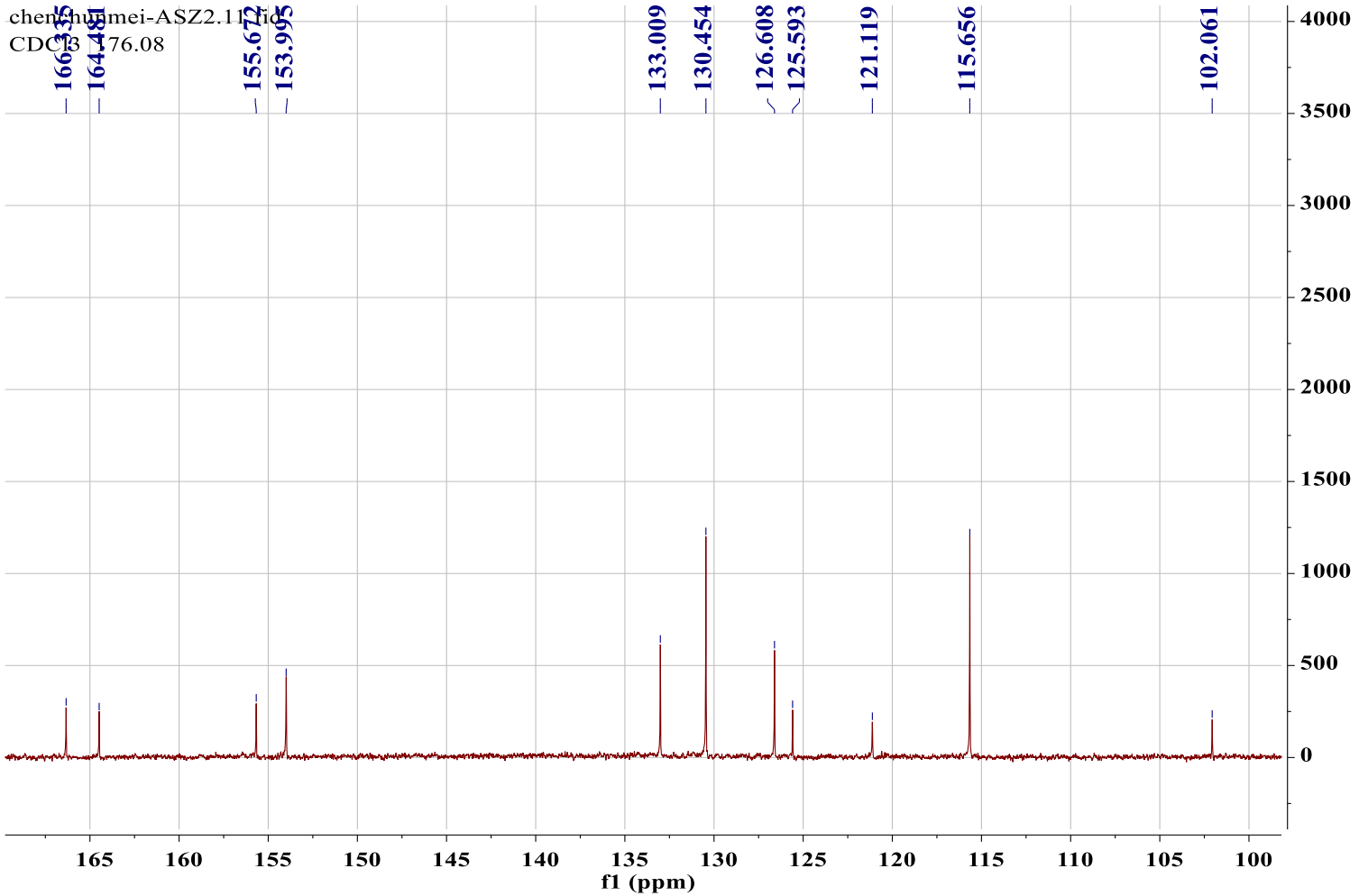


Figure S5: ^{13}C NMR spectrum of campyridone D (**1**) ($CDCl_3$, 175 MHz) (From δ_C 100 ppm to 170 ppm).

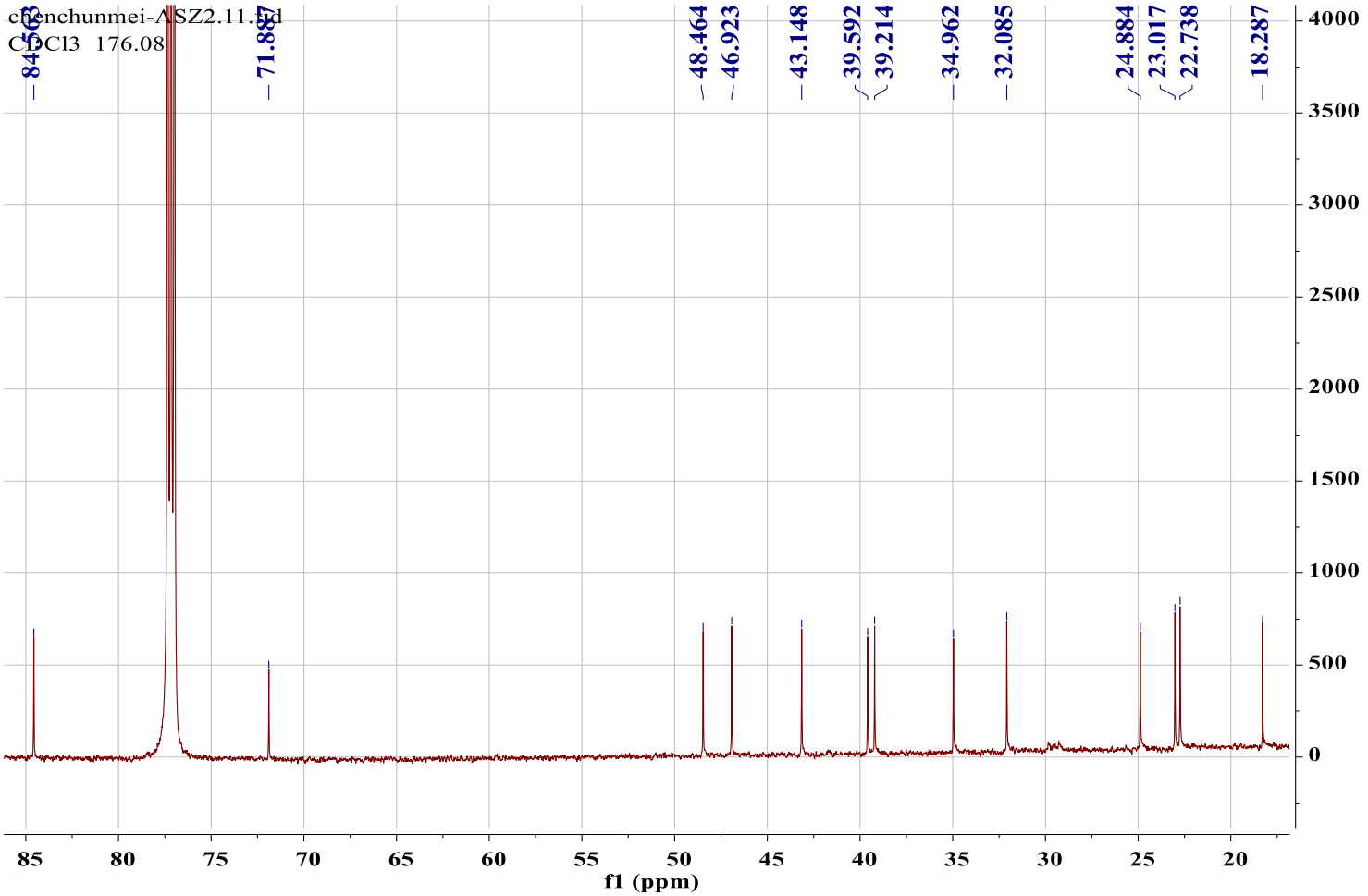


Figure S6: ^{13}C NMR spectrum of campyridone D (**1**) ($CDCl_3$, 175 MHz) (From δ_C 15 ppm to 85 ppm).

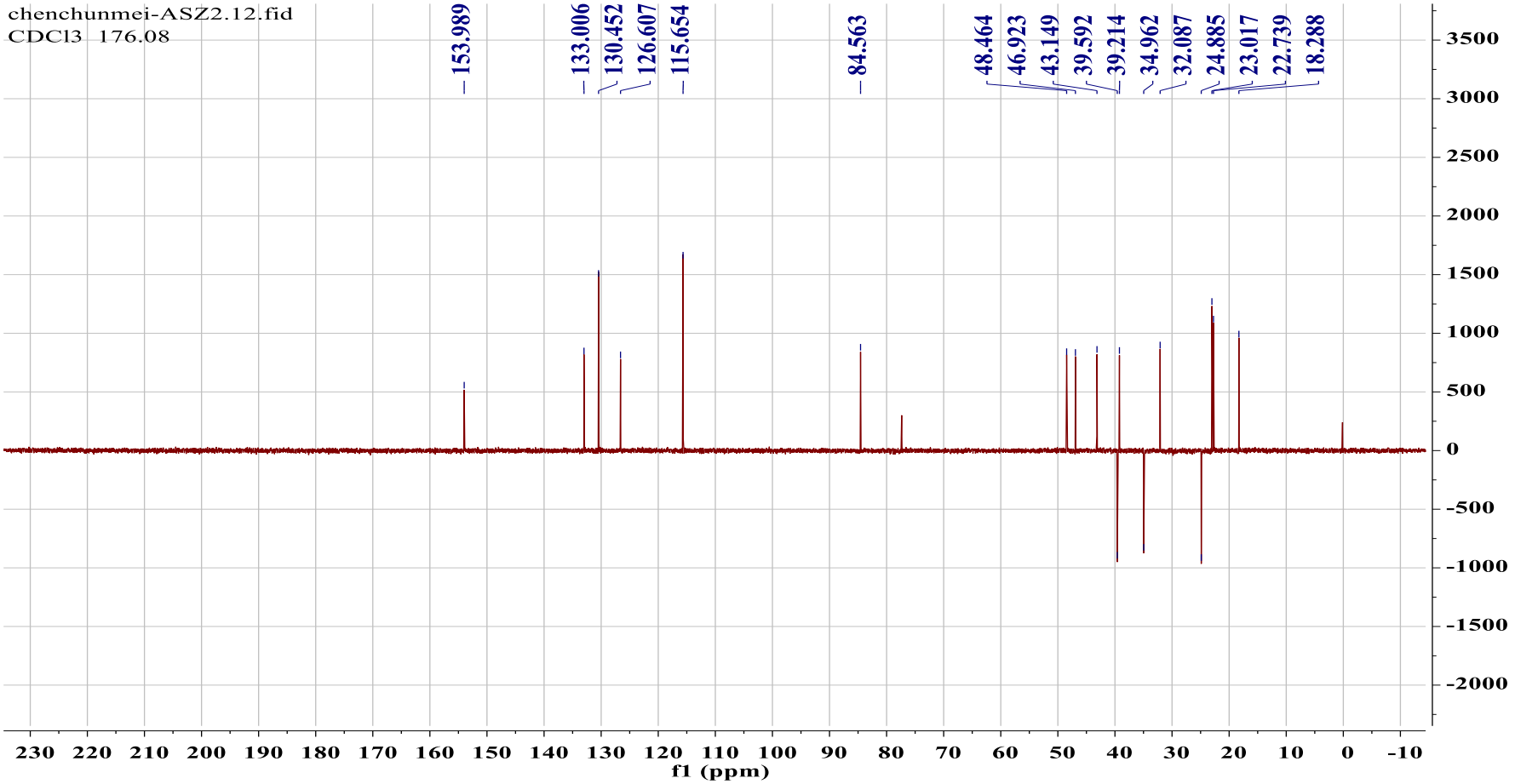


Figure S7: DEPT 135 NMR spectrum of campyridone D (**1**) ($CDCl_3$, 175 MHz).

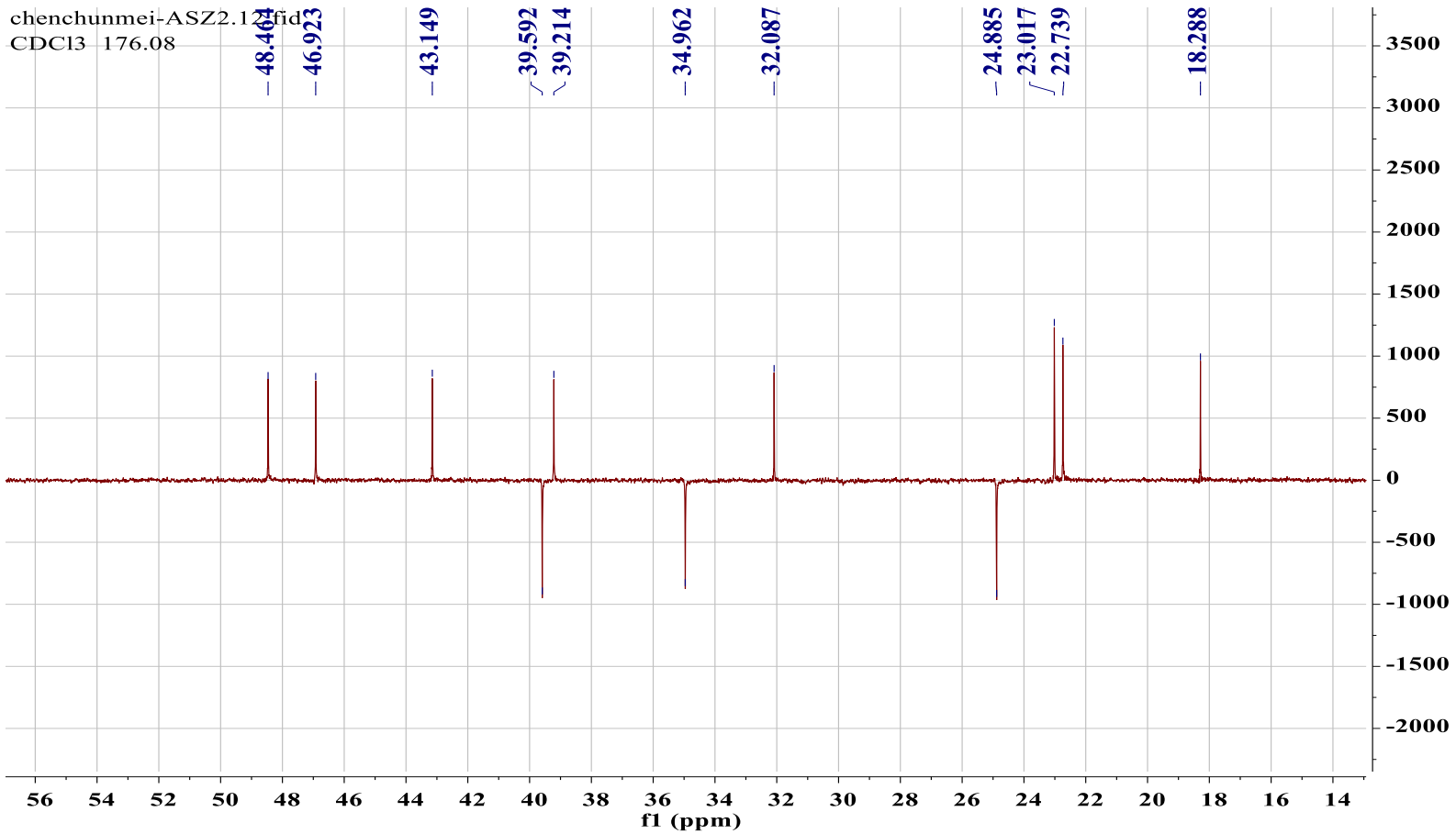


Figure S8: DEPT 135 NMR spectrum of campyridone D (**1**) ($CDCl_3$, 175 MHz) (From δ_C 14 ppm to 56 ppm).

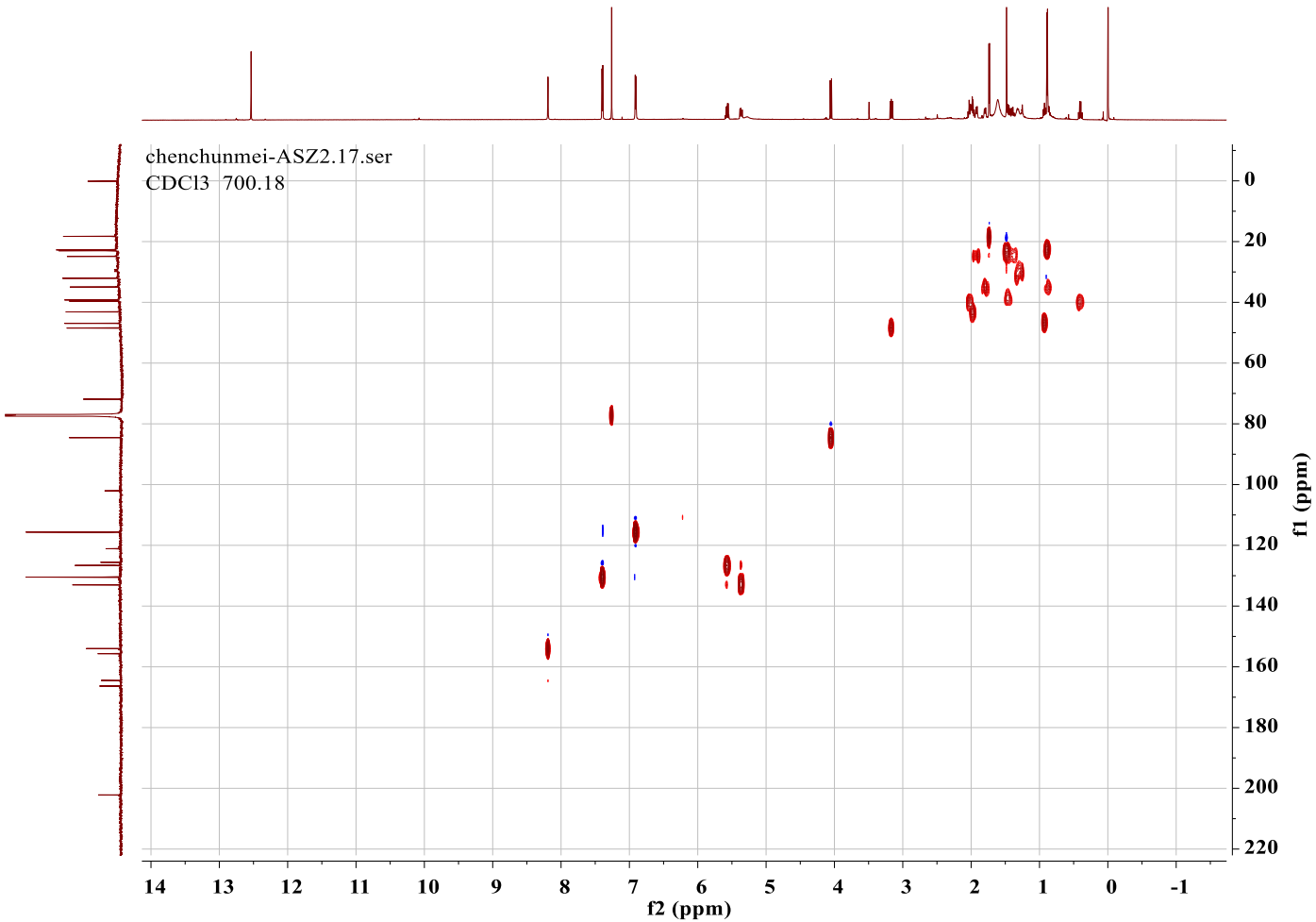


Figure S9: HSQC spectrum of campyridone D (**1**) ($CDCl_3$).

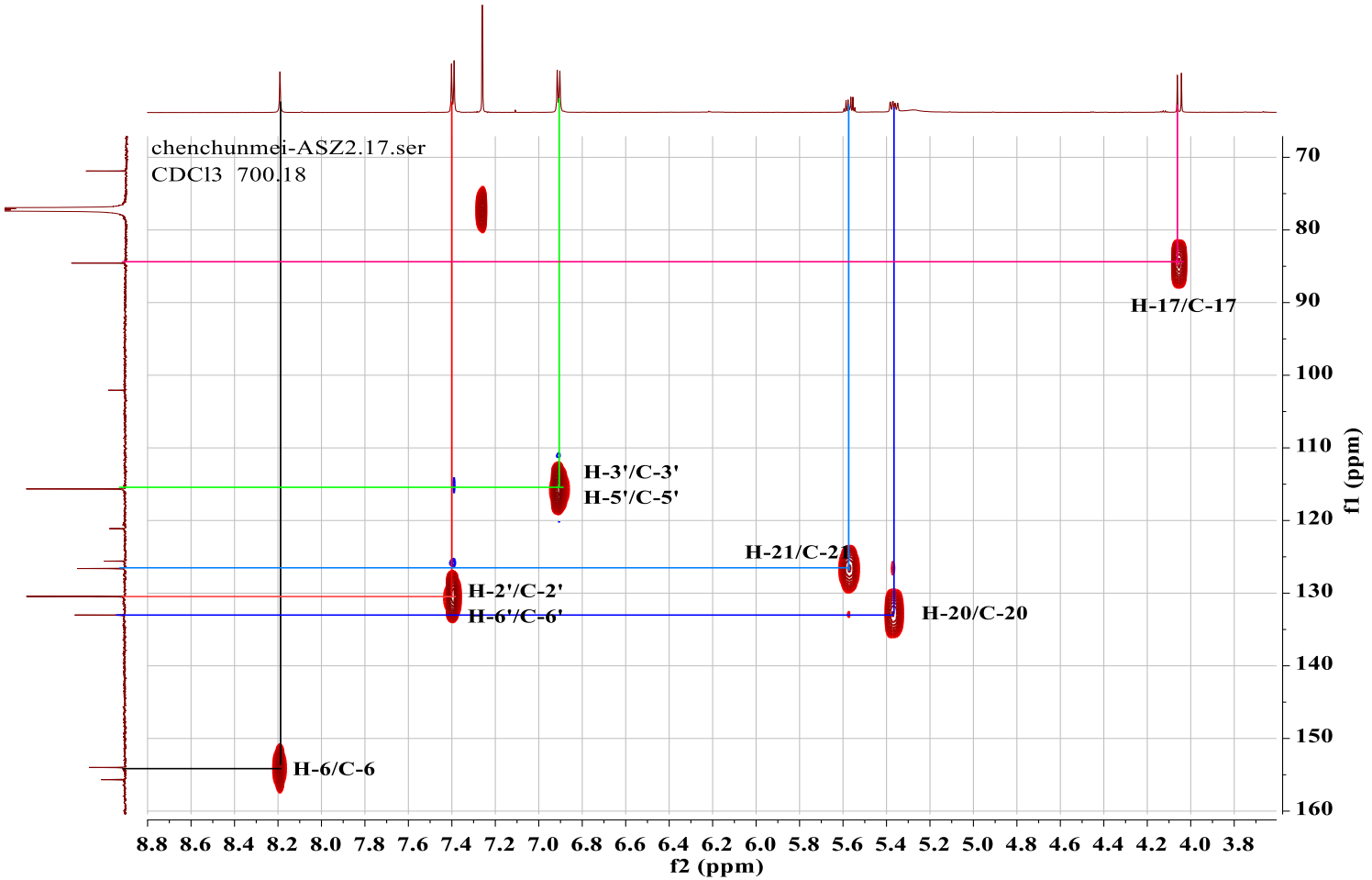


Figure S10: HSQC spectrum of campyridone D (**1**) ($CDCl_3$) (From δ_H 3.6 ppm to 8.8 ppm).

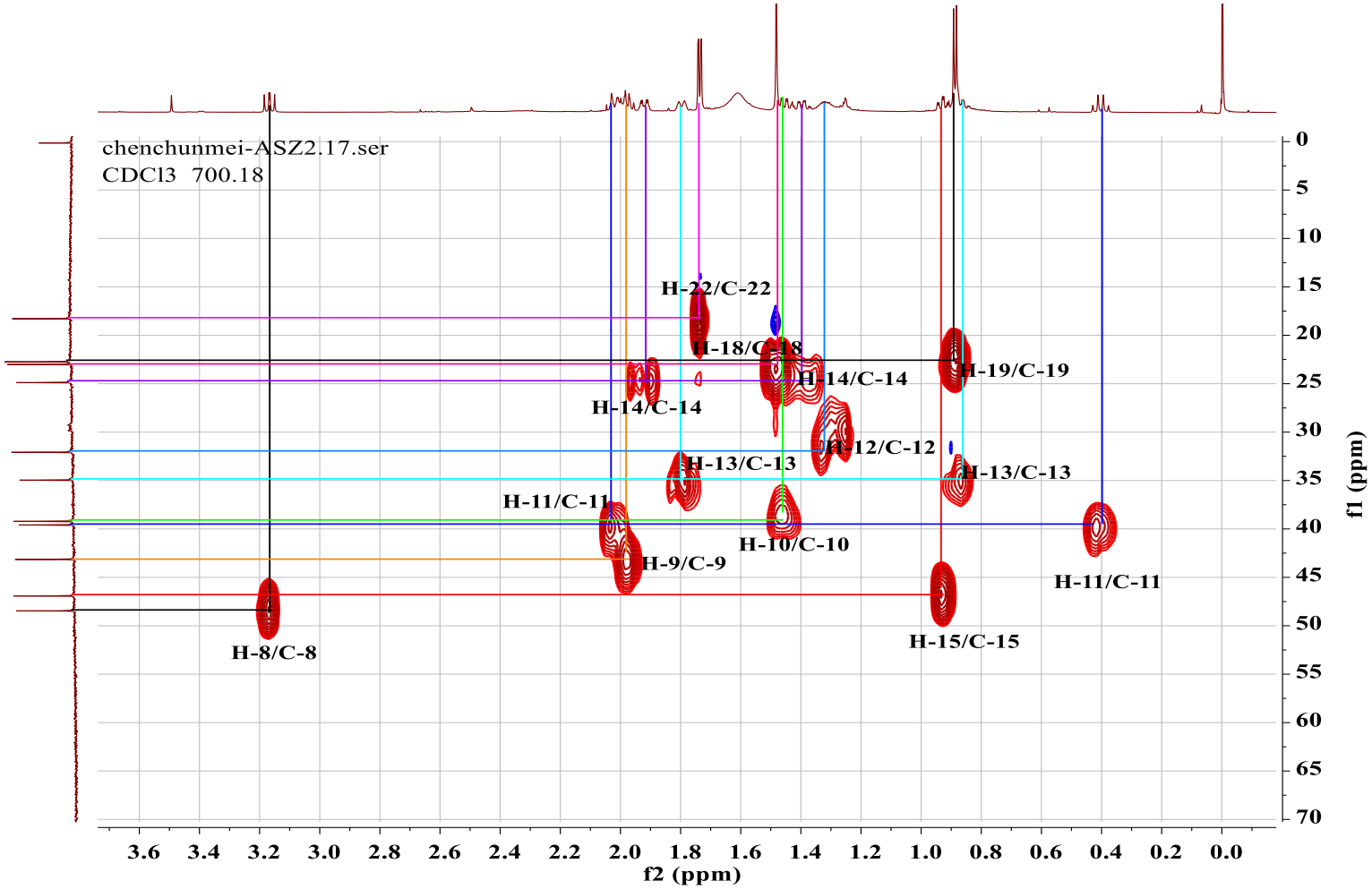


Figure S11: HSQC spectrum of campyridone D (**1**) ($CDCl_3$) (From δ_H 0 ppm to 3.6 ppm).

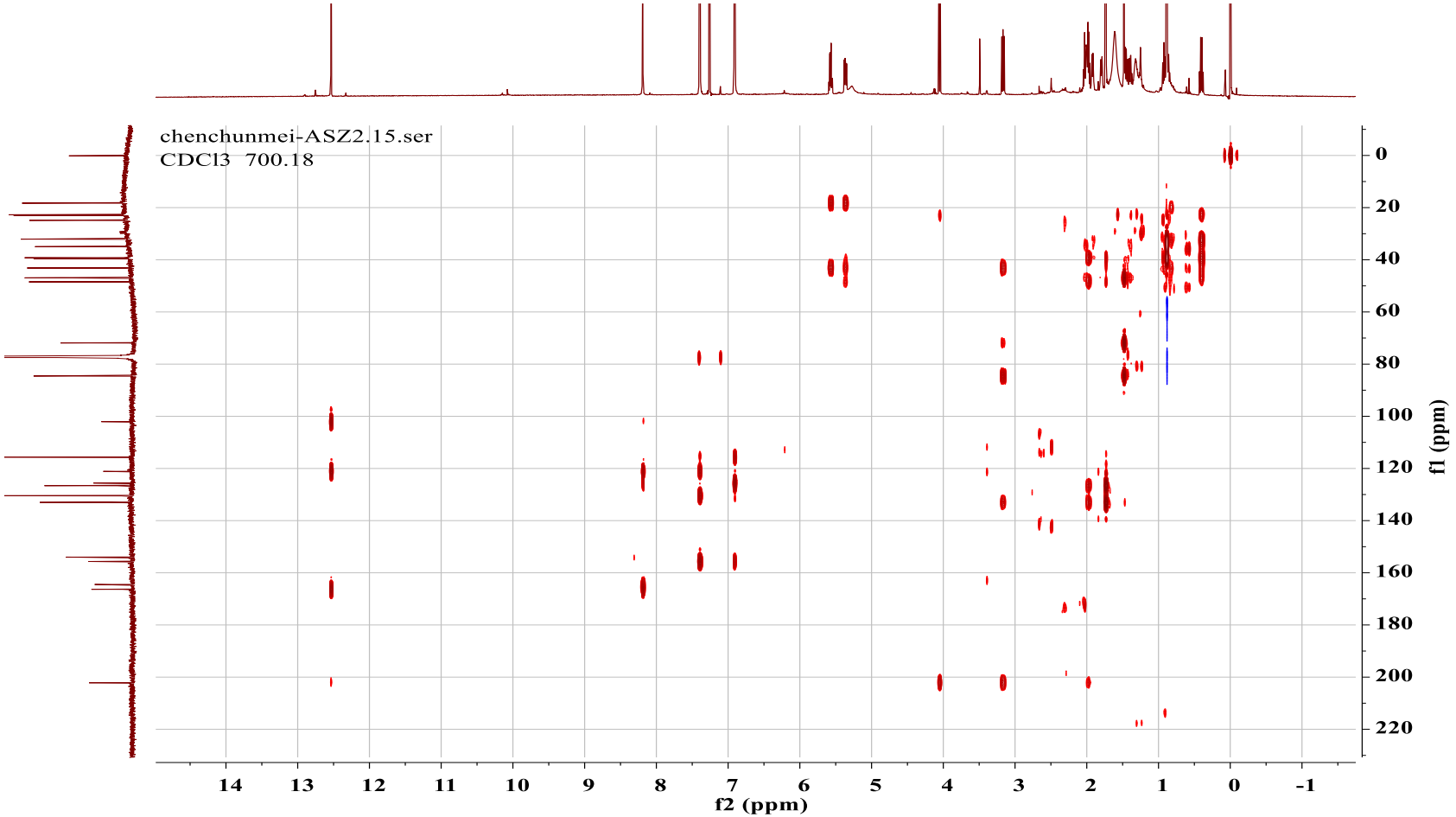


Figure S12: HMBC spectrum of campyridone D (**1**) ($CDCl_3$).

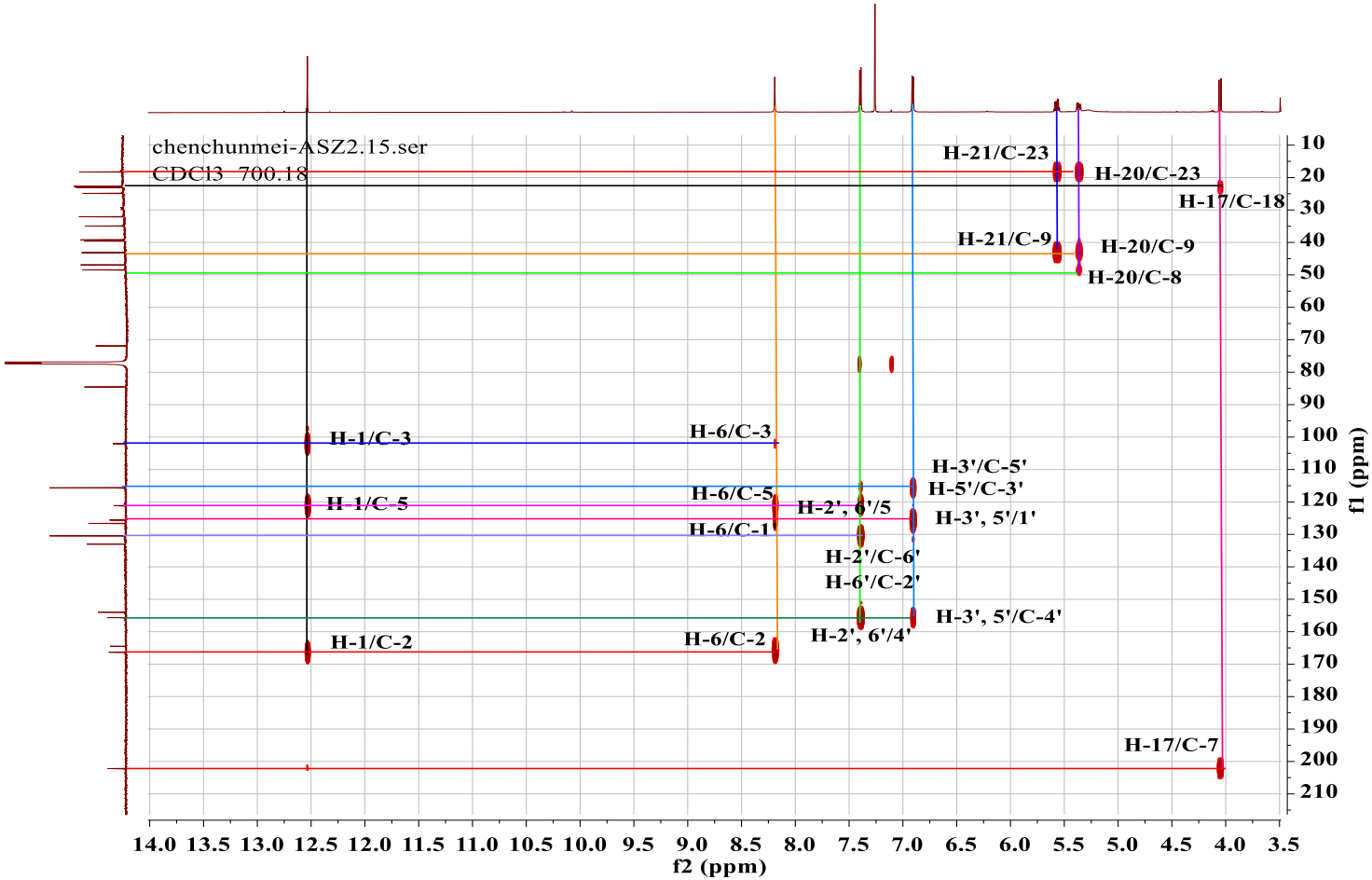


Figure S13: HMBC spectrum of campyridone D (**1**) ($CDCl_3$) (From δ_H 3.5 ppm to 14.0 ppm).

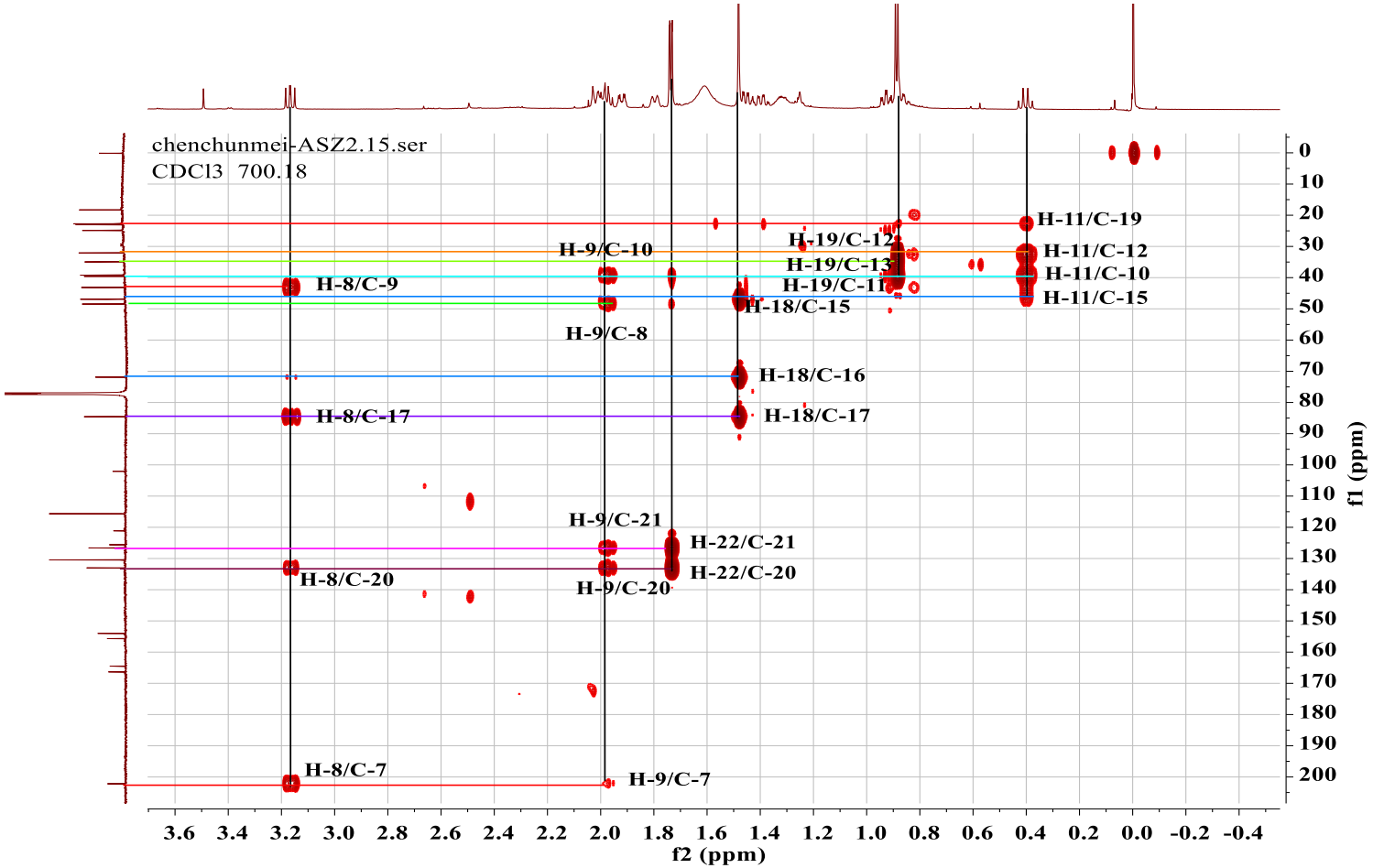


Figure S14: HMBC spectrum of campyridone D (**1**) ($CDCl_3$) (From δ_H 0 ppm to 3.6 ppm).

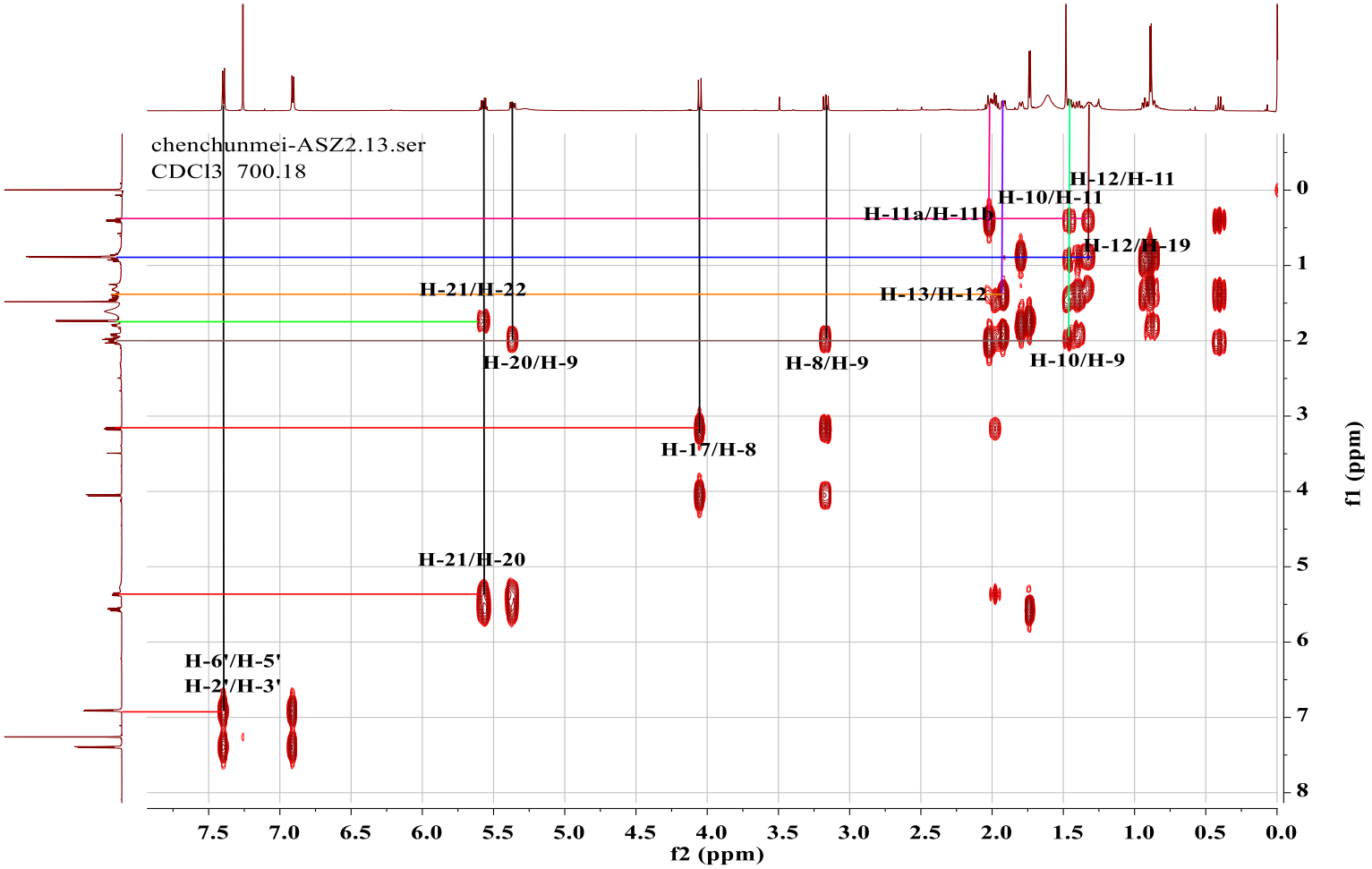


Figure S15: ^{1}H-^{1}H COSY spectrum of campyridone D (**1**) ($CDCl_3$).

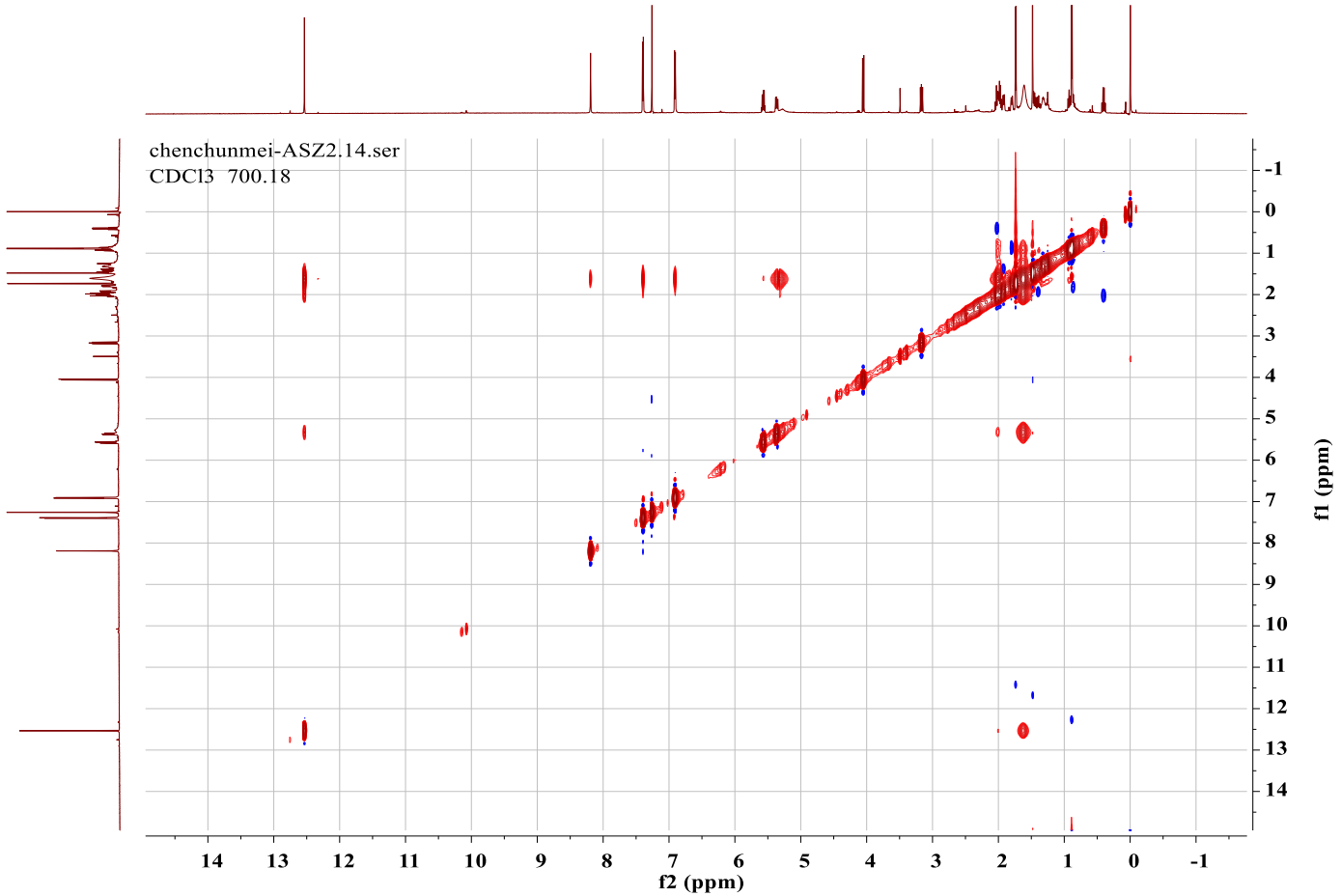
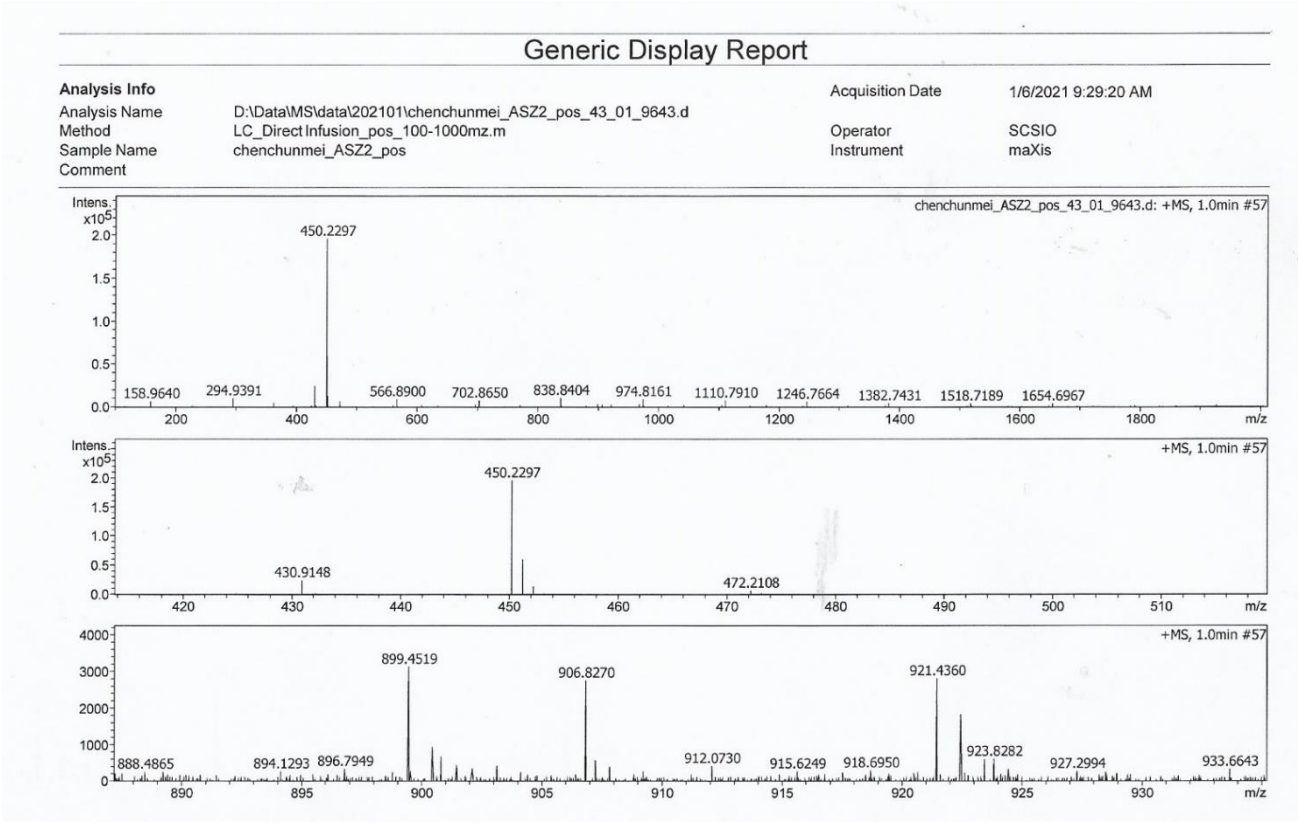


Figure S16: NOESY spectrum of campyridone D (**1**) ($CDCl_3$).



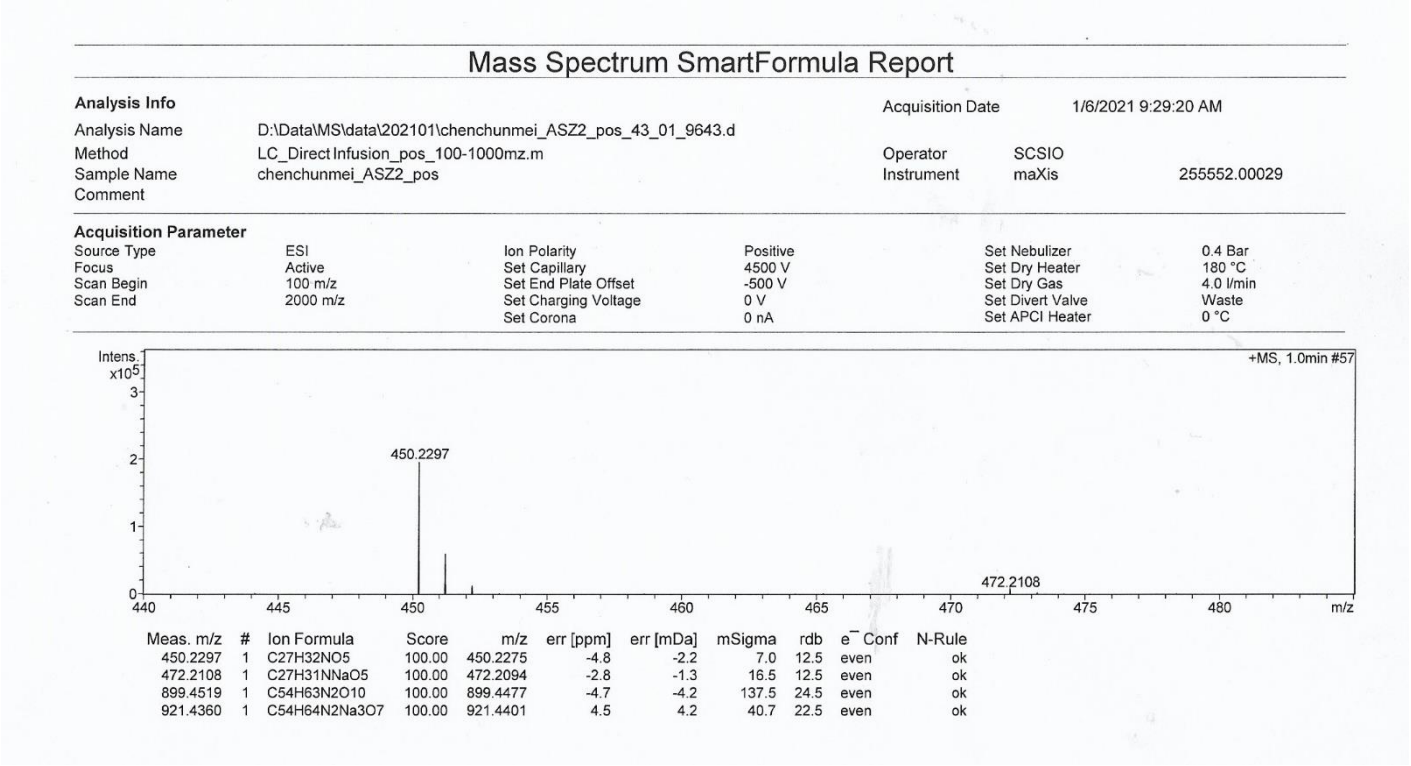


Figure S17: HR-ESIMS spectrum of campyridone D (**1**).

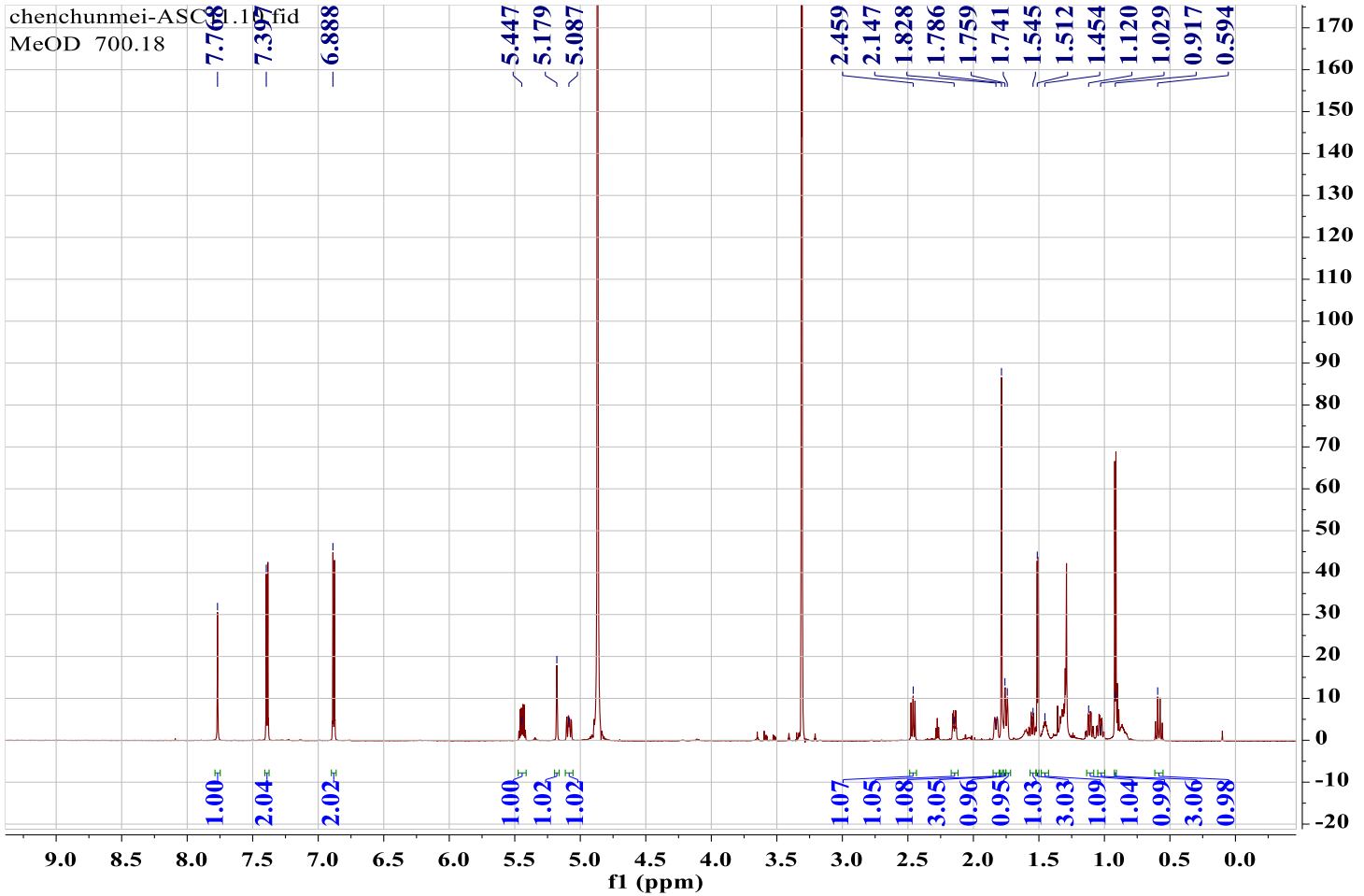


Figure S18: ^{1}H NMR spectrum of campyridone A (**2**) (CD_3OD, 700 MHz).

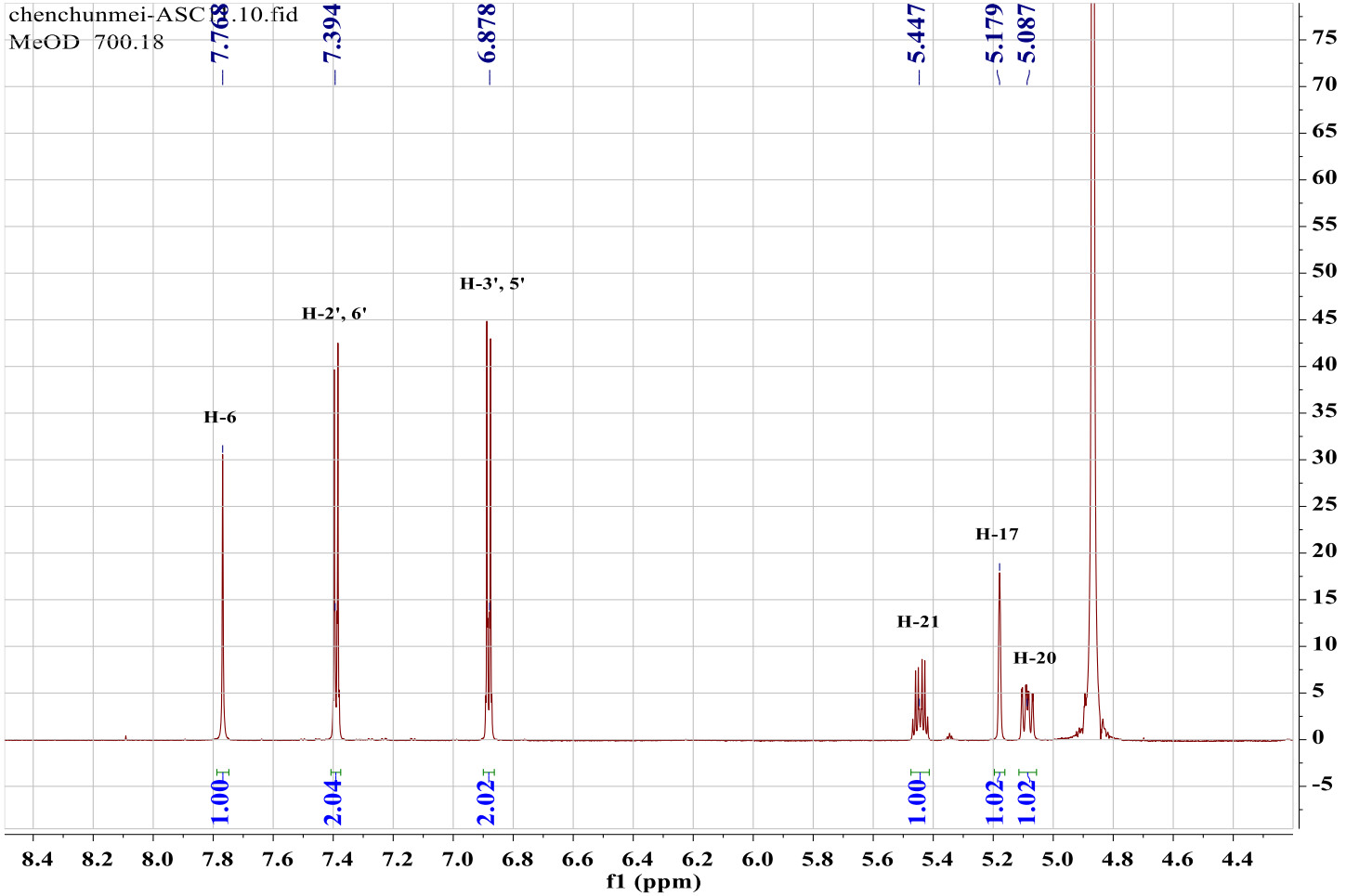


Figure S19: ^{1}H NMR spectrum of campyridone A (**2**) (CD_3OD, 700 MHz) (From δ_H 4.4 ppm to 8.4 ppm).

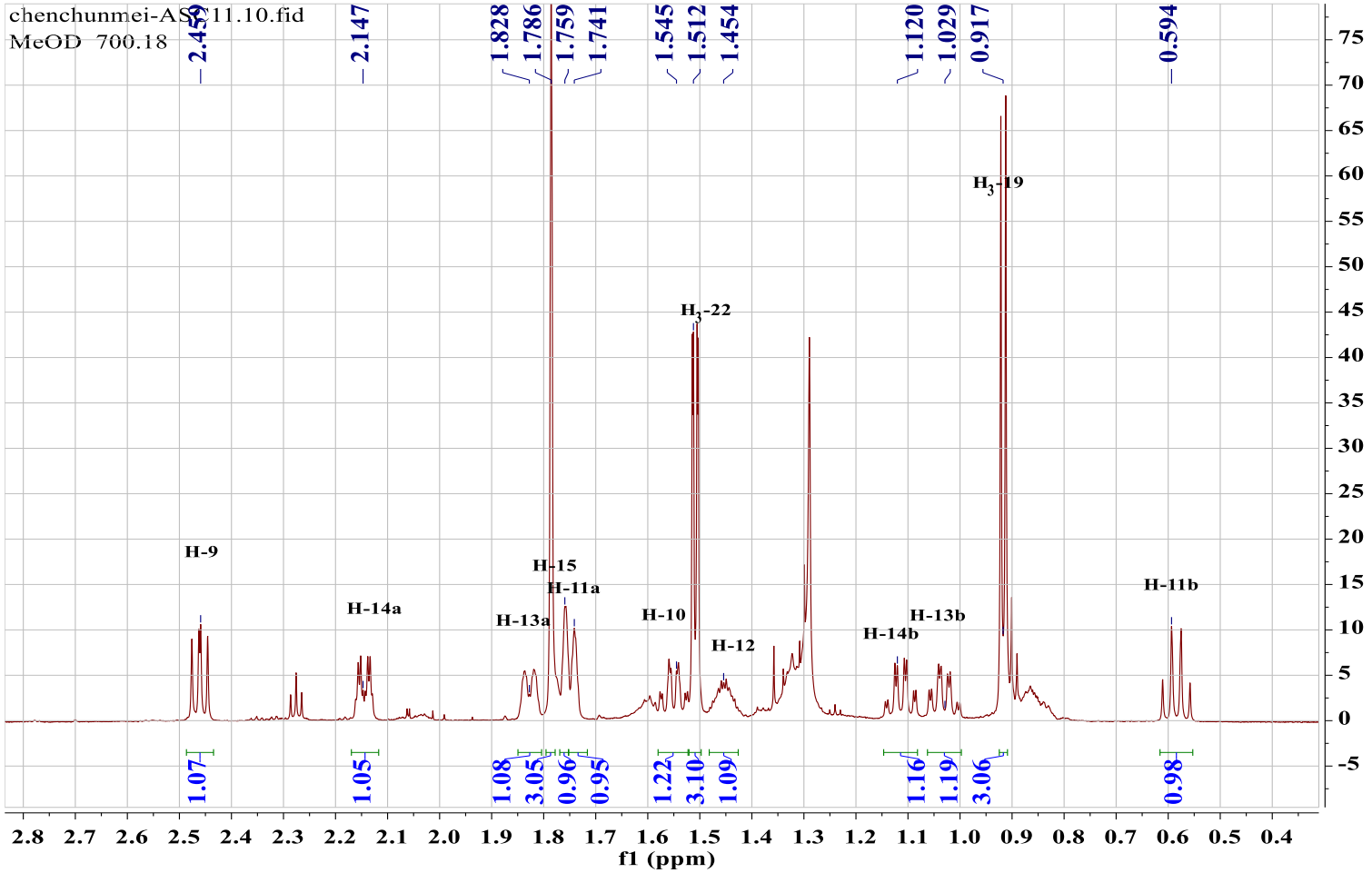


Figure S20: ^{1}H NMR spectrum of campyridone A (**2**) (CD_3OD, 700 MHz) (From δ_H 0.4 ppm to 2.8 ppm).

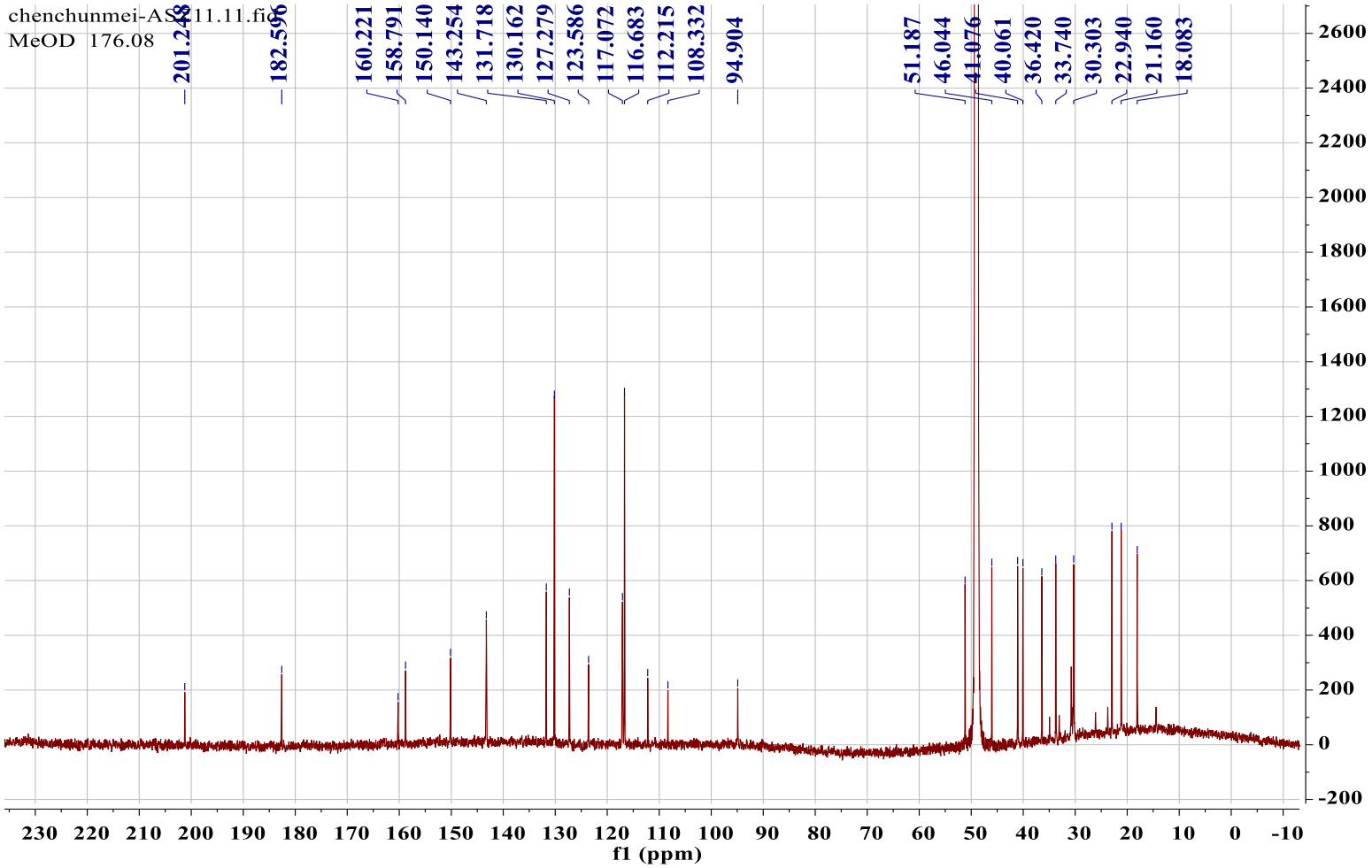


Figure S21: ^{13}C NMR spectrum of campyridone A (**2**) (CD_3OD, 175 MHz).

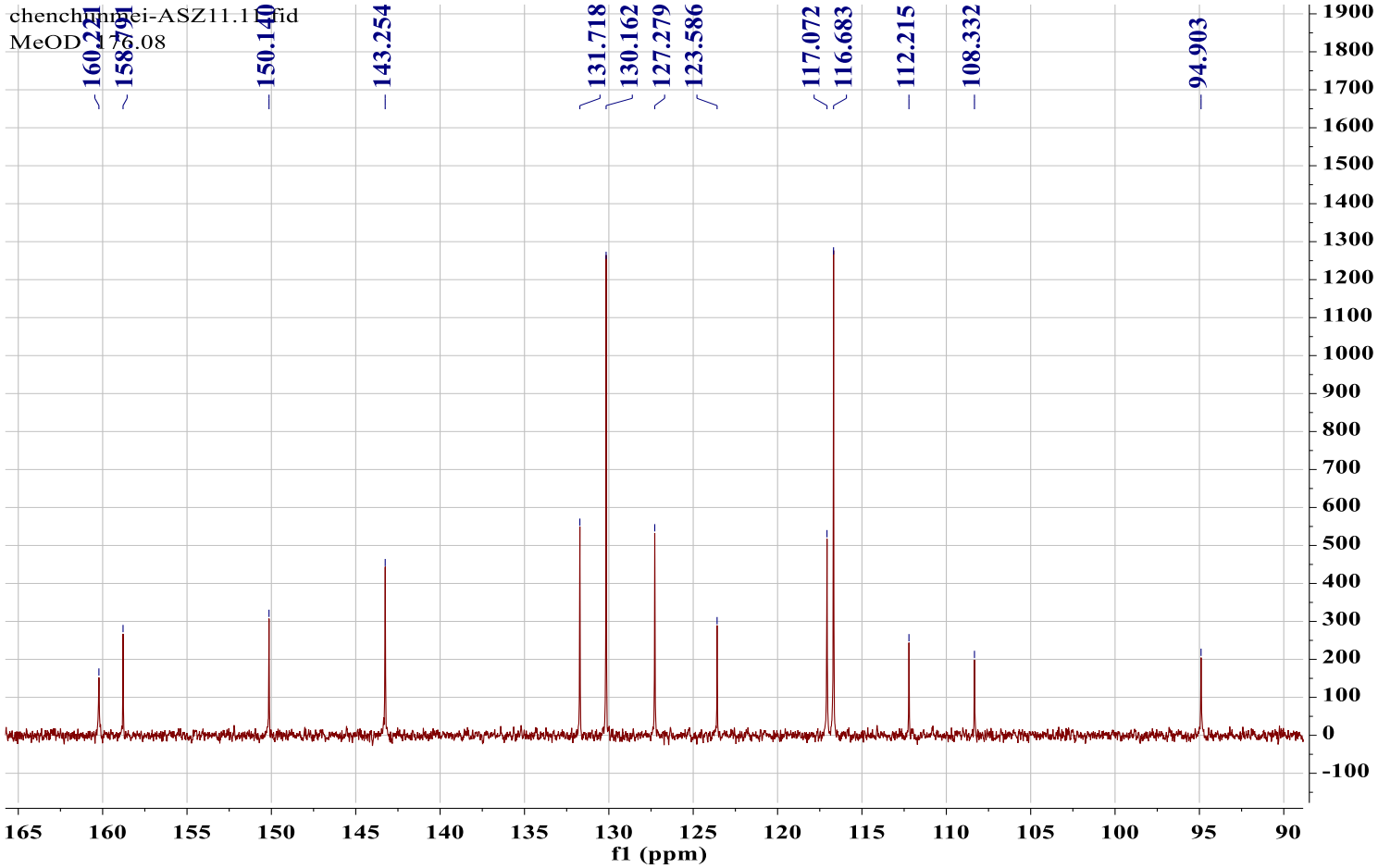


Figure S22: ^{13}C NMR spectrum of campyridone A (**2**) (CD_3OD, 175 MHz) (From δ_C 90 ppm to 165 ppm).

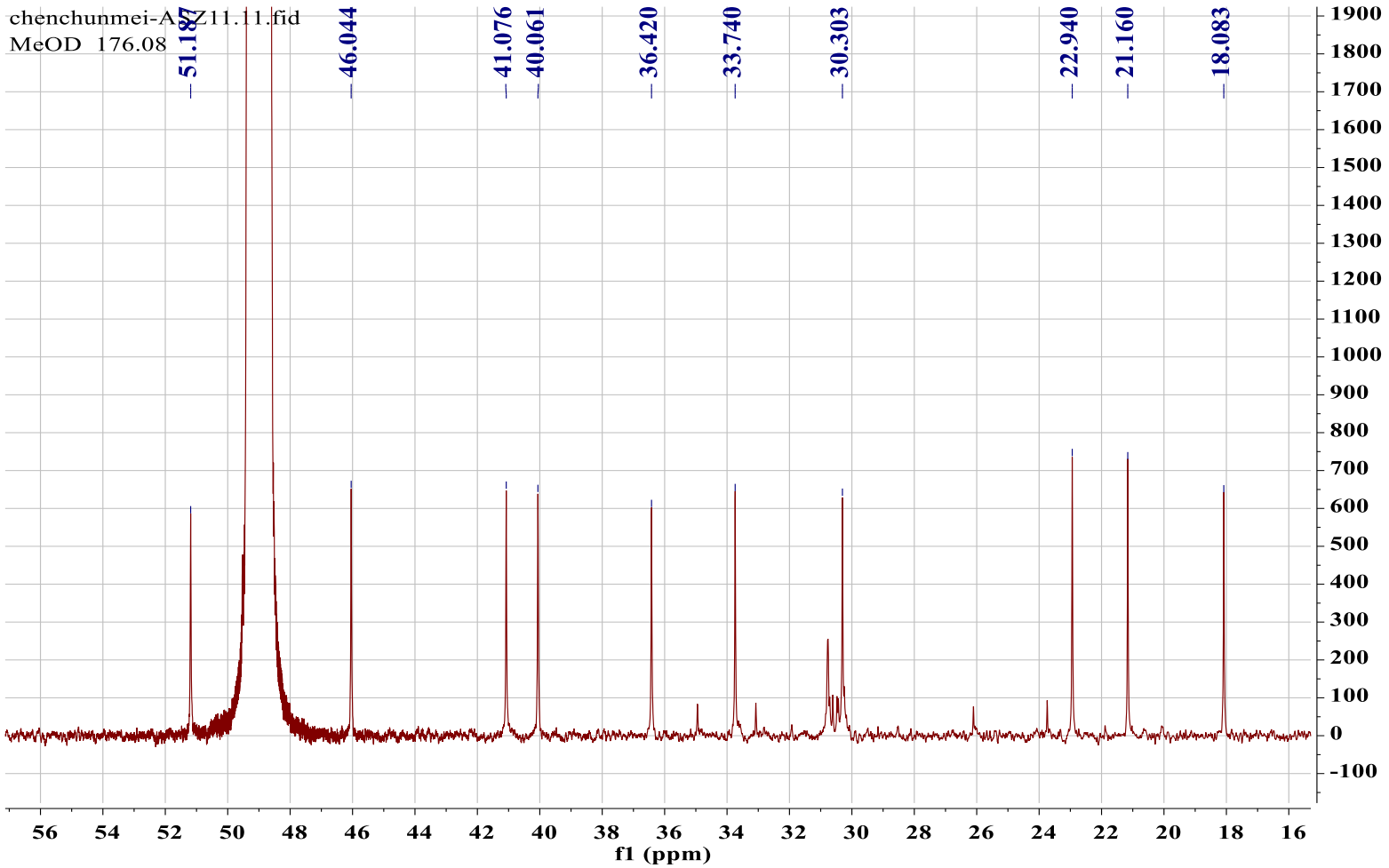


Figure S23: ^{13}C NMR spectrum of campyridone A (**2**) (CD_3OD, 175 MHz) (From δ_C 16 ppm to 56 ppm).

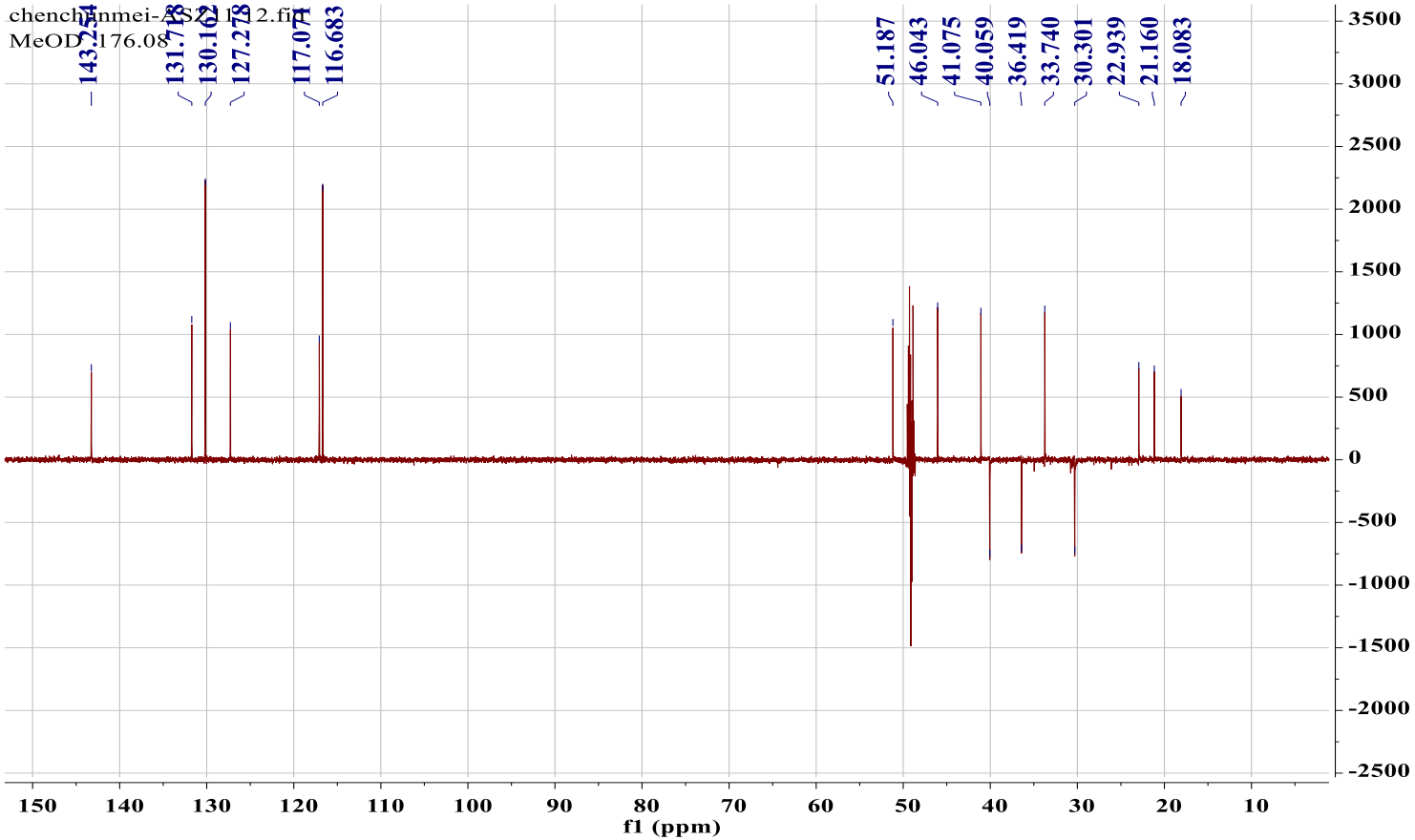


Figure S24: DEPT 135 NMR spectrum of campyridone A (**2**) (CD_3OD, 175 MHz).

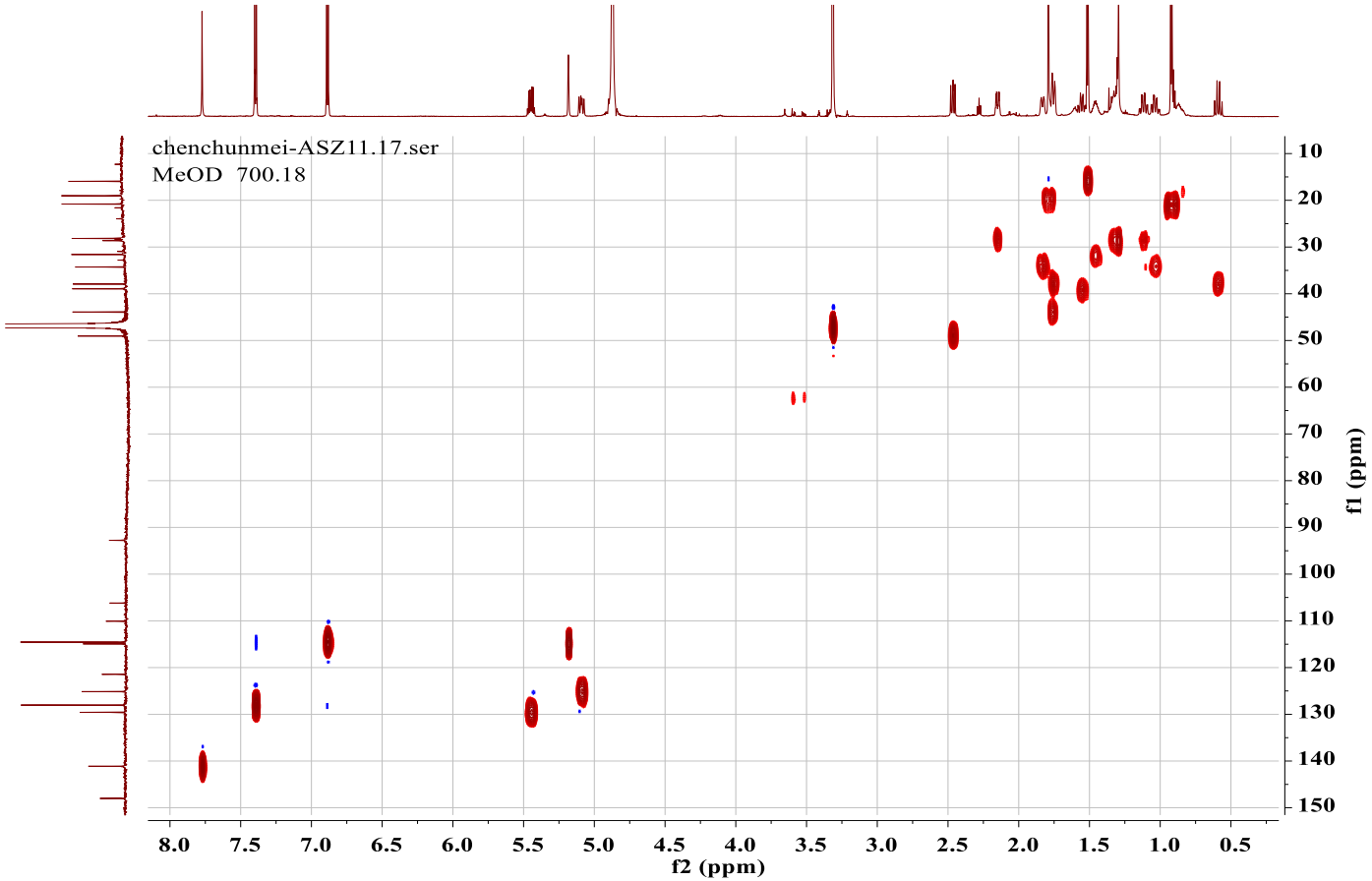


Figure S25: HSQC spectrum of campyridone A (**2**) (CD_3OD).

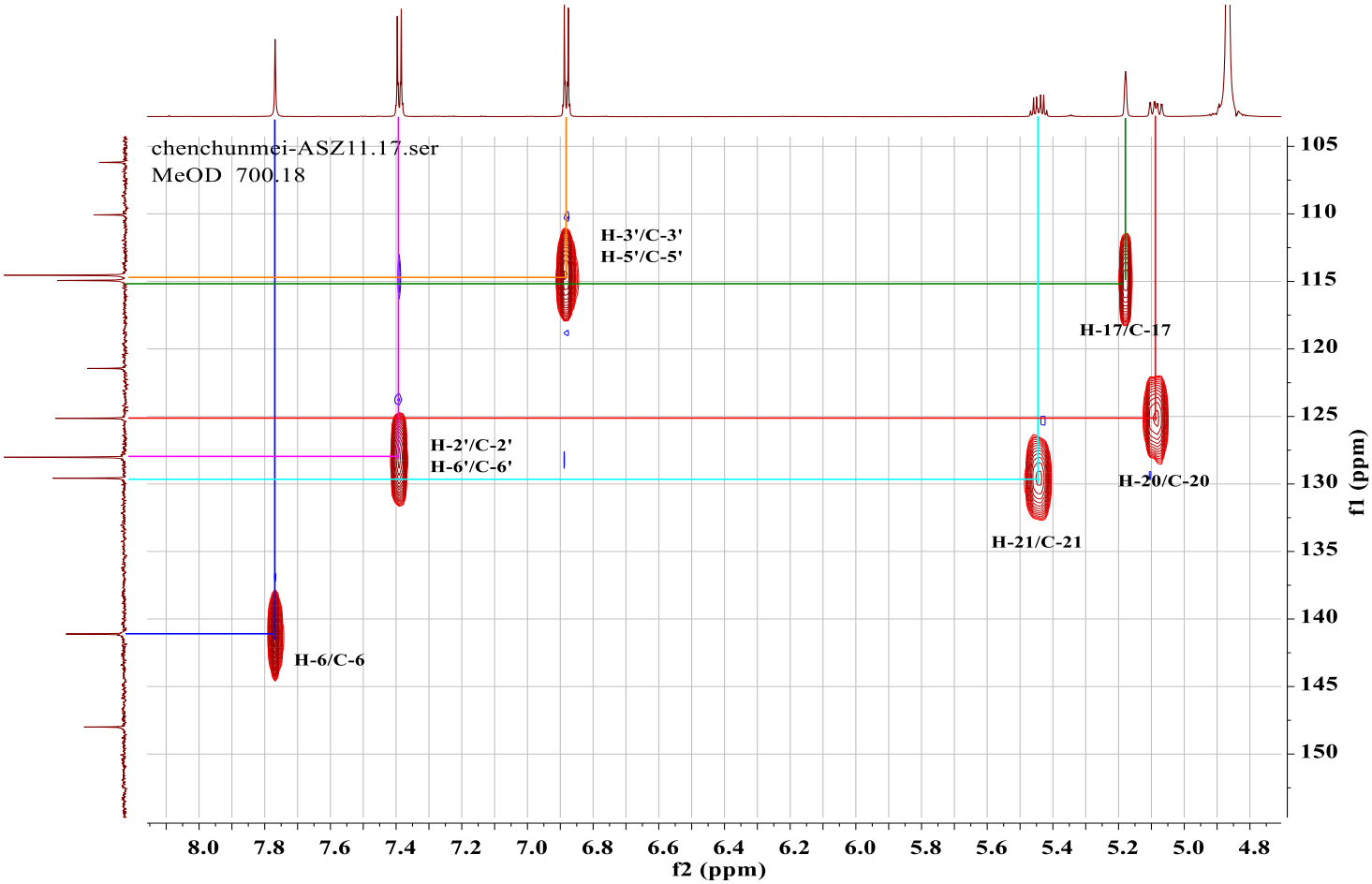


Figure S26: HSQC spectrum of campyridone A (**2**) (CD_3OD) (From δ_H 4.8 ppm to 8.0 ppm).

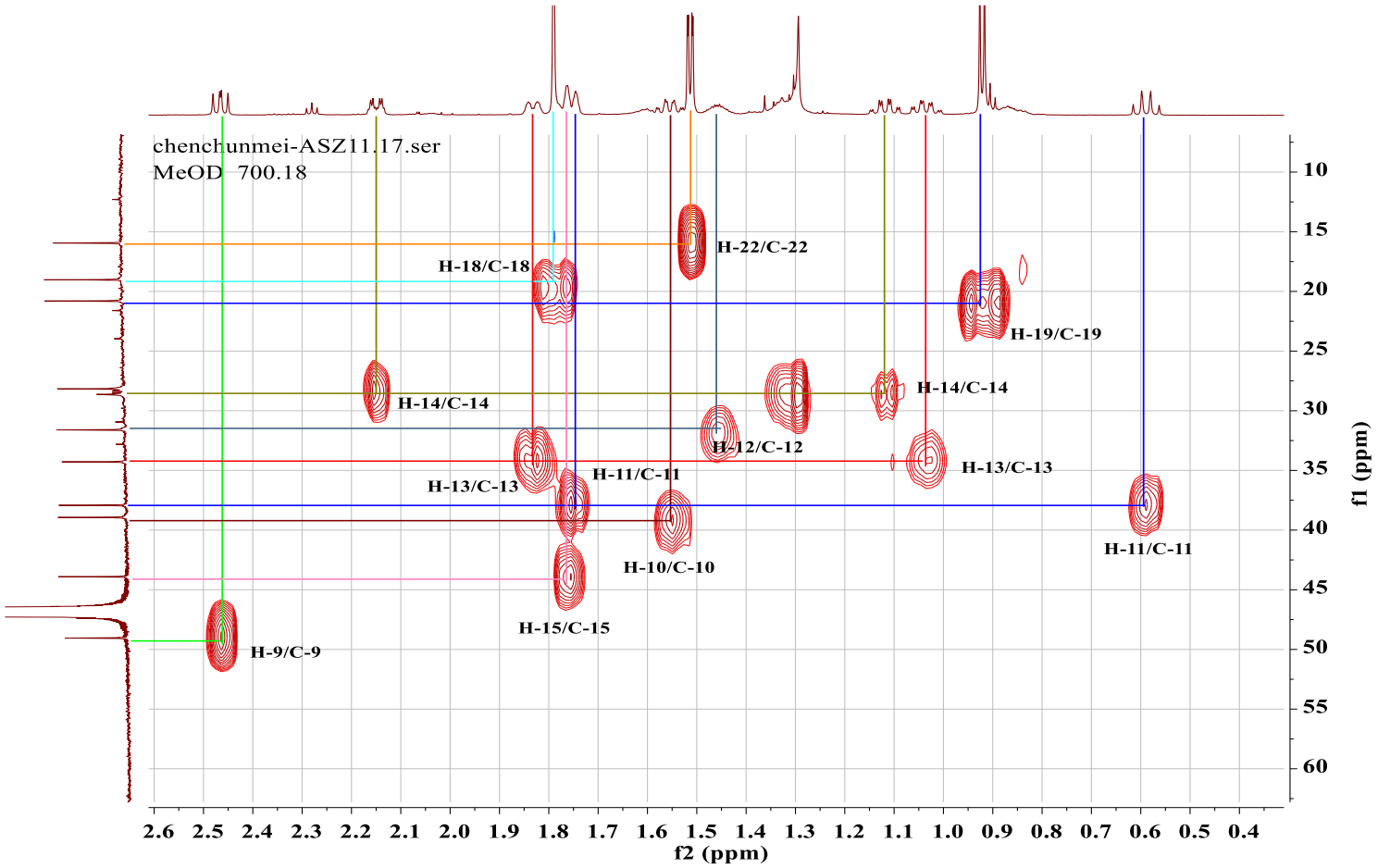


Figure S27: HSQC spectrum of campyridone A (**2**) (CD_3OD) (From δ_H 0.4 ppm to 2.6 ppm).

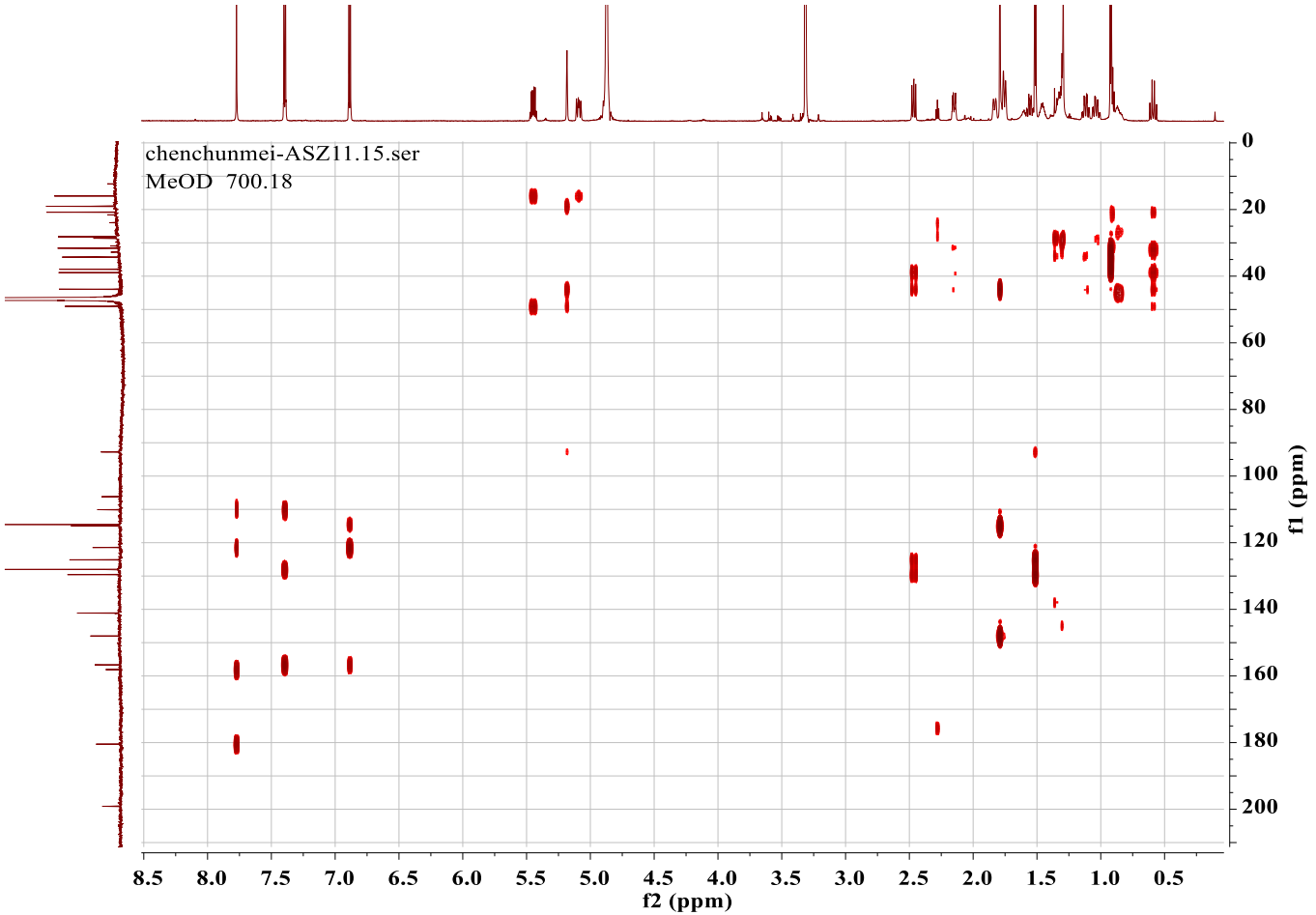


Figure S28: HMBC spectrum of campyridone A (**2**) (CD_3OD).

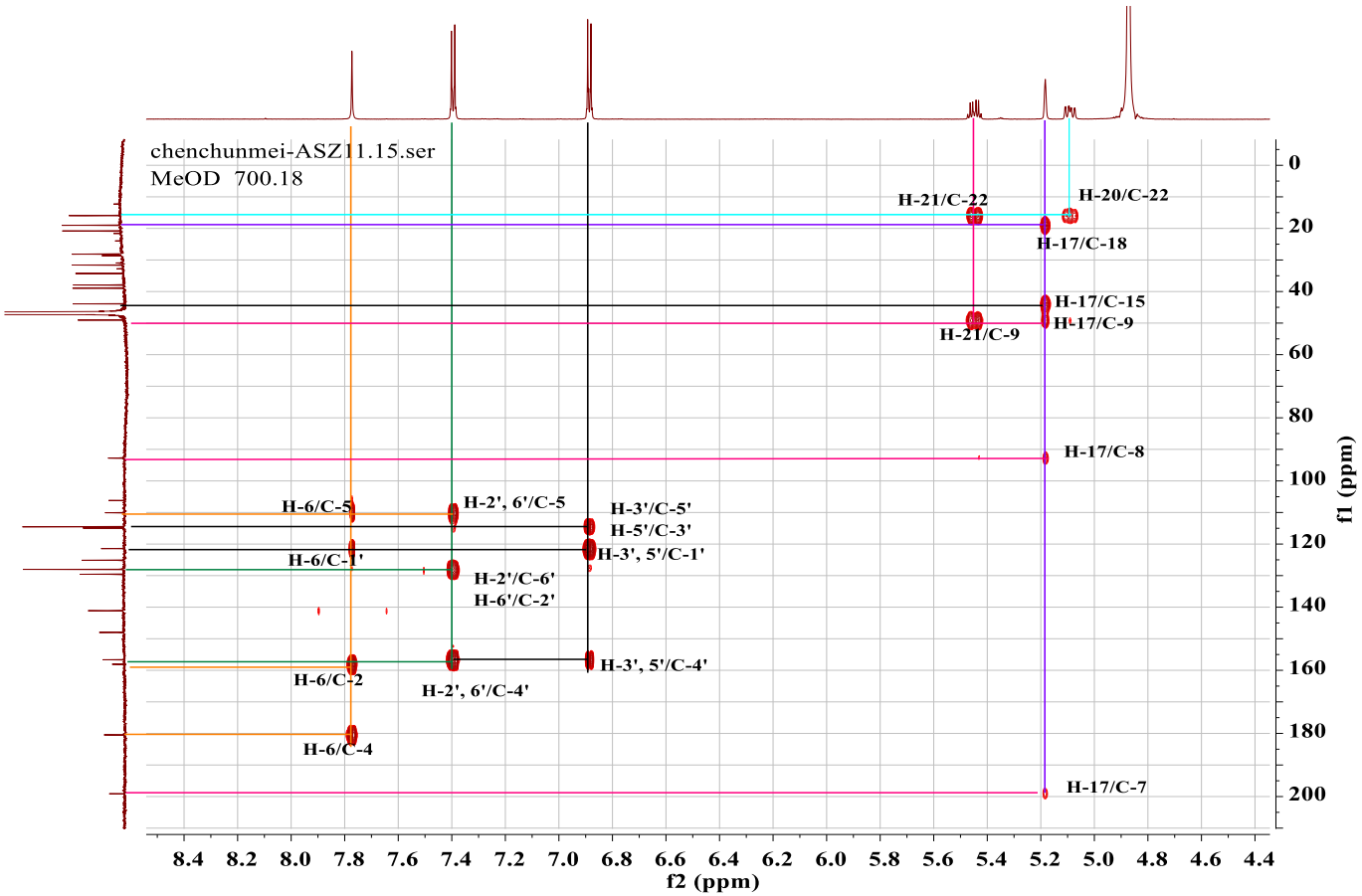


Figure S29: HMBC spectrum of campyridone A (**2**) (CD_3OD) (From δ_H 4.4 ppm to 8.5 ppm).

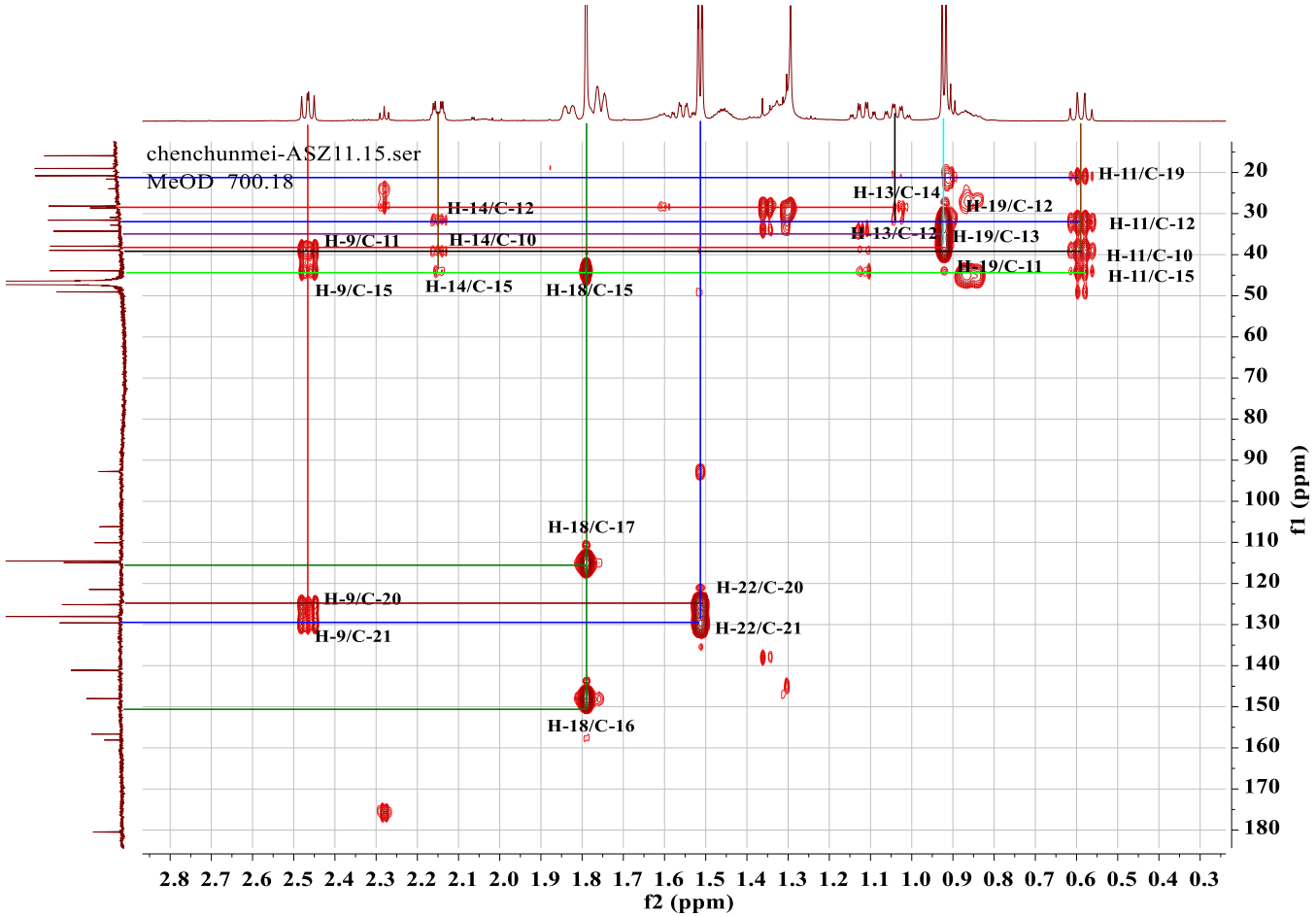


Figure S30: HMBC spectrum of campyridone A (**2**) (CD_3OD) (From δ_H 0.3 ppm to 2.8 ppm).

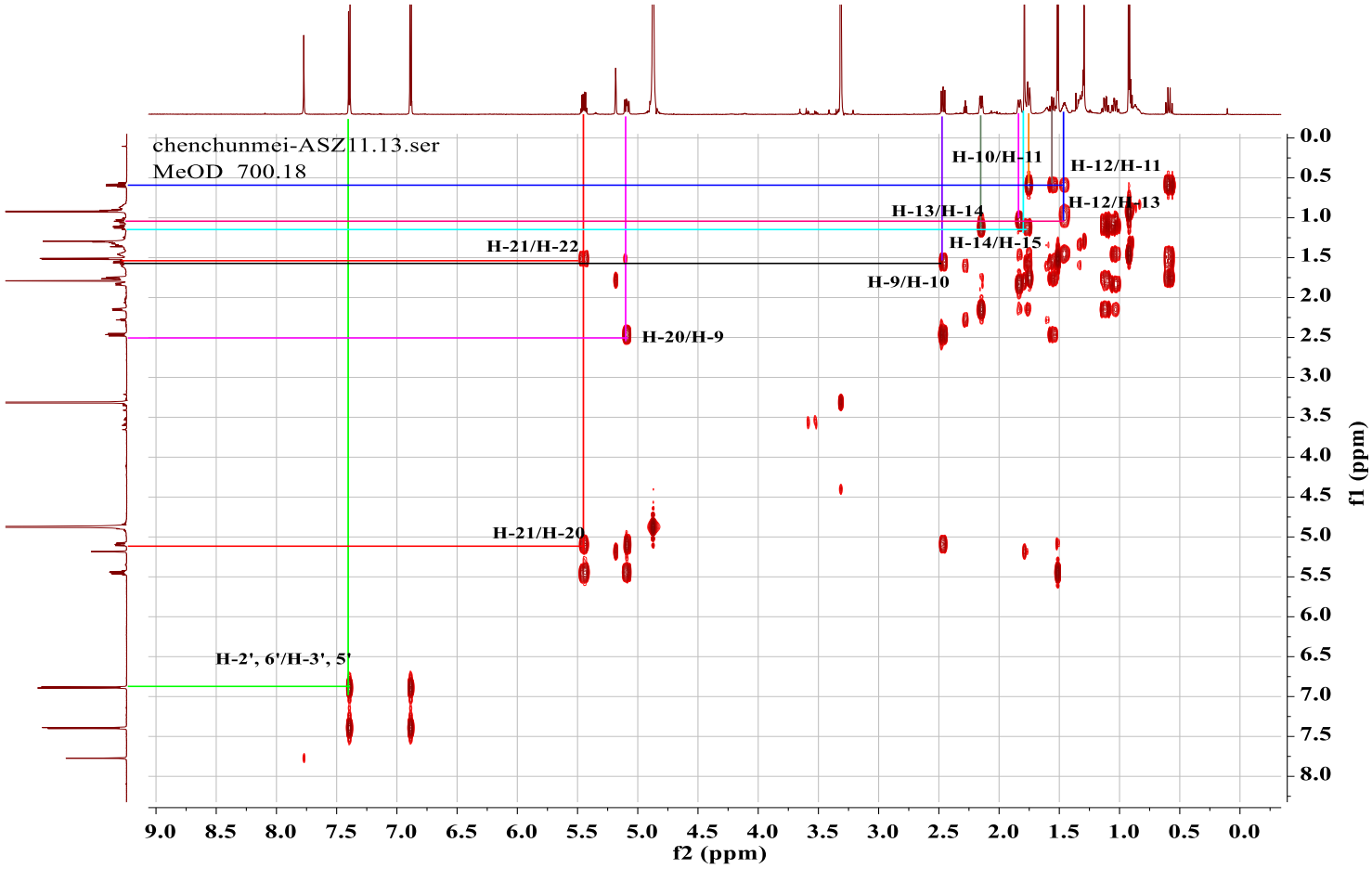


Figure S31: ^{1}H-^{1}H COSY spectrum of campyridone A (**2**) (CD_3OD).

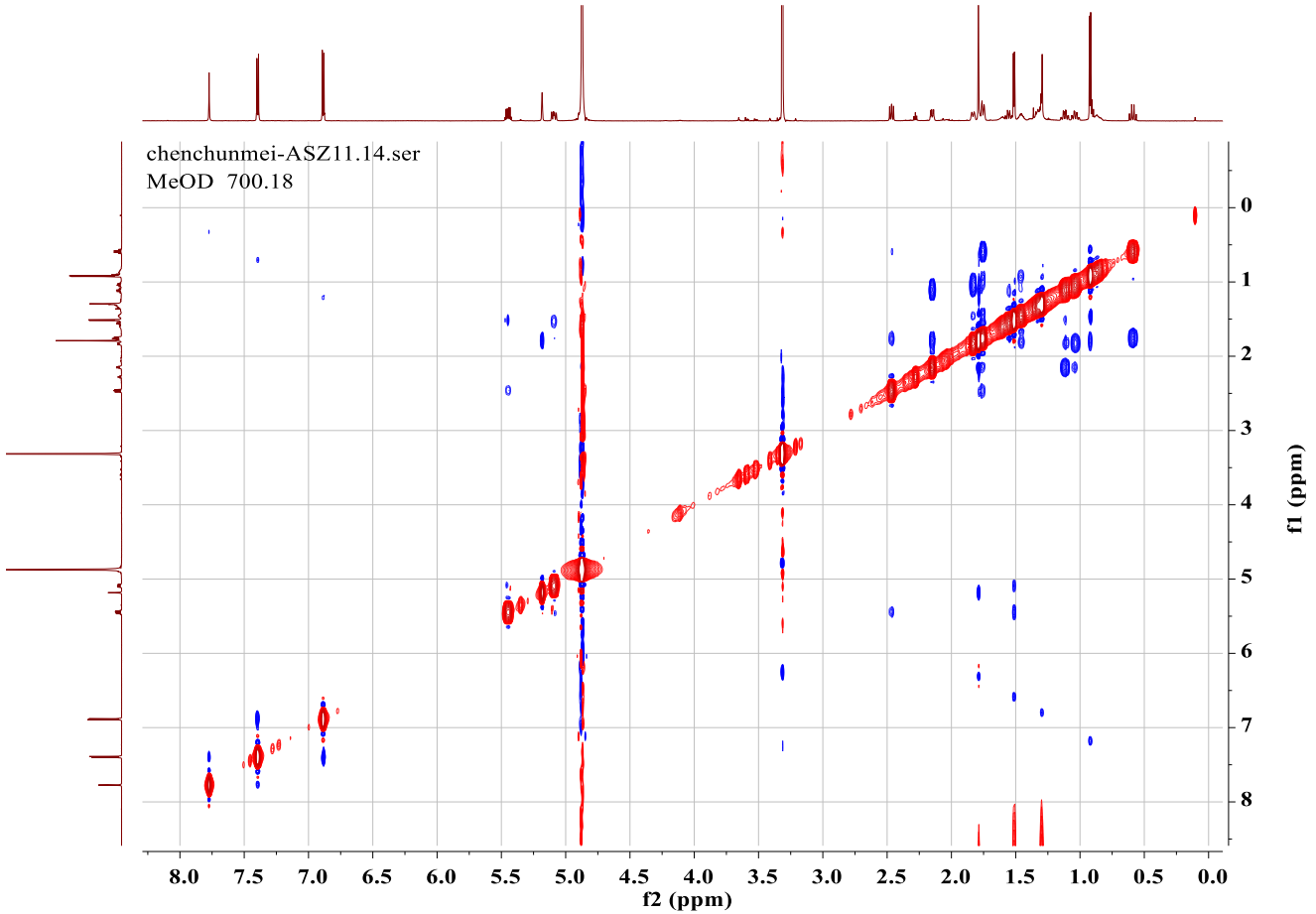


Figure S32: NOESY spectrum of campyridone A (**2**) (CD_3OD).

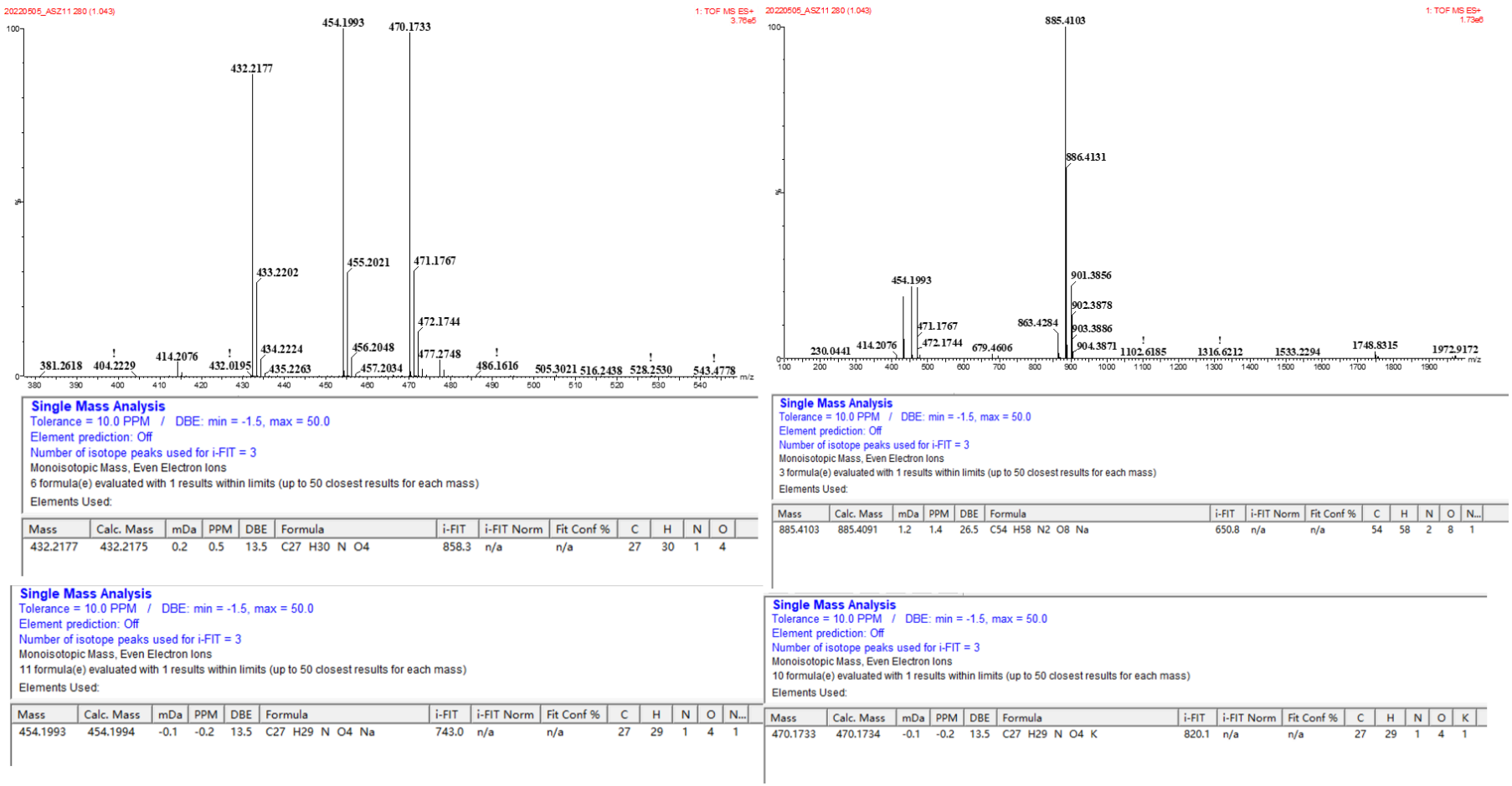


Figure S33: HR-ESIMS spectrum of campyridone A (**2**).

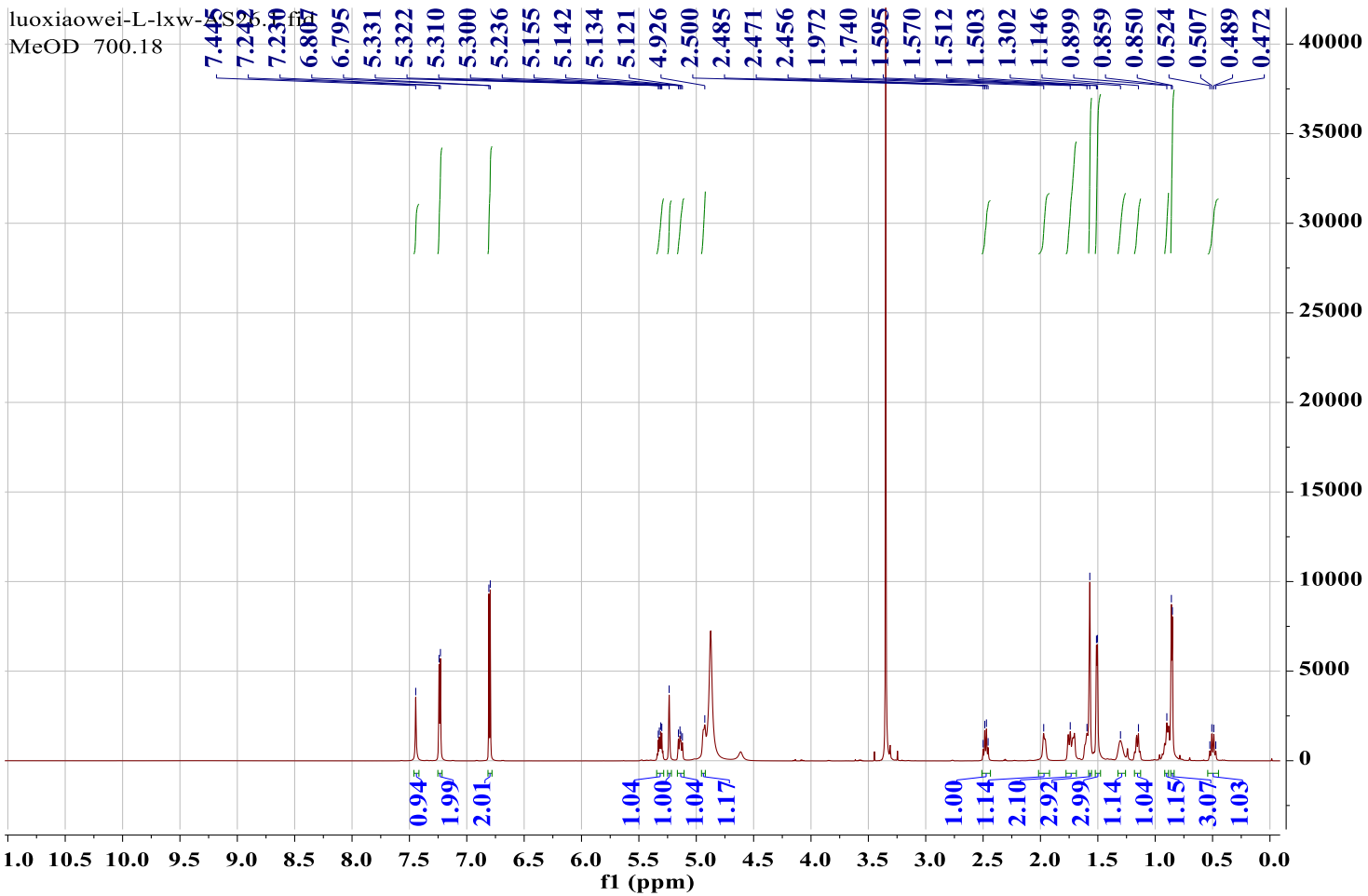


Figure S34: ^{1}H NMR spectrum of ilicicolin H (**3**) (CD_3OD, 700 MHz).

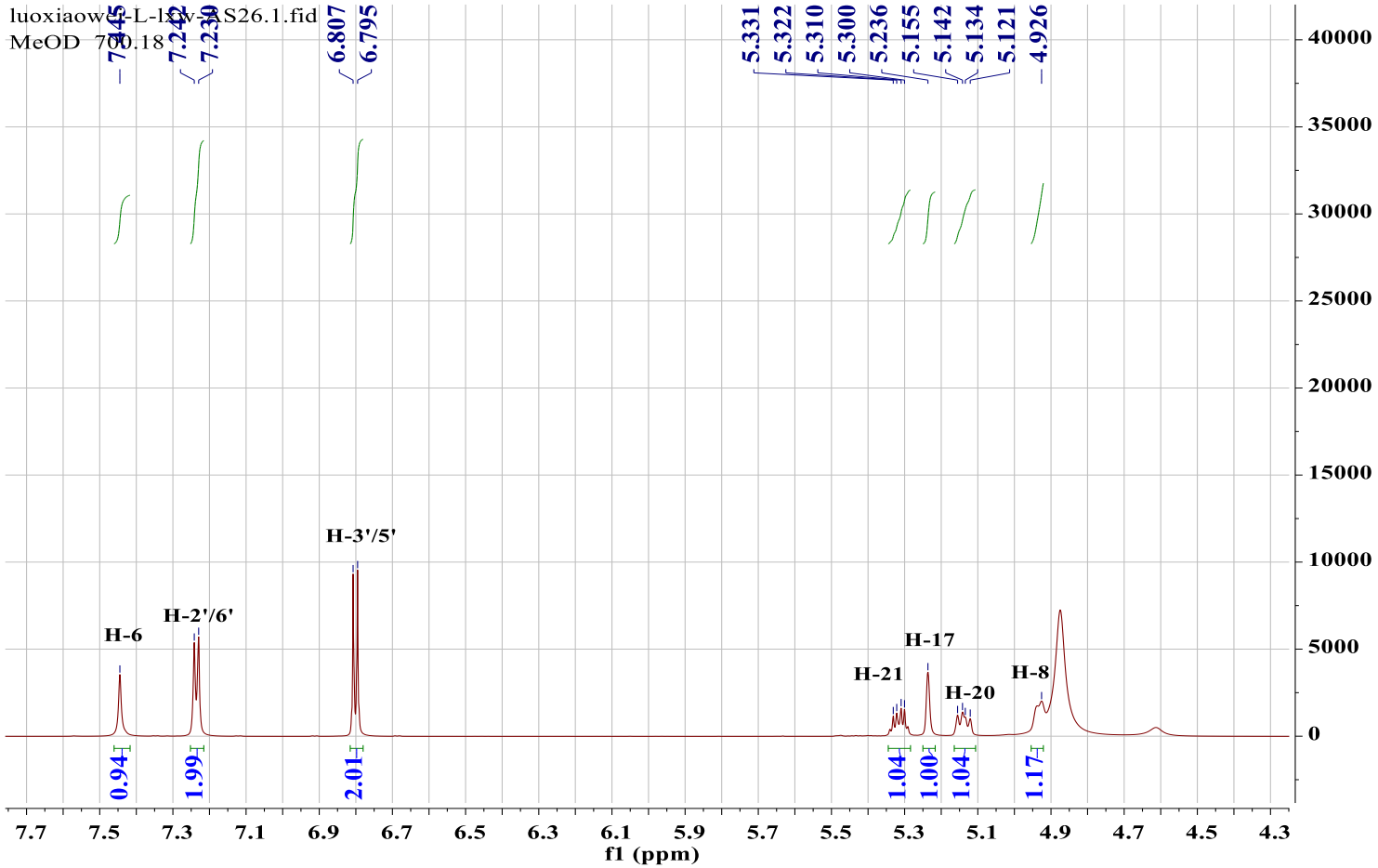


Figure S35: ^{1}H NMR spectrum of ilicicolin H (**3**) (CD_3OD, 700 MHz) (From δ_H 4.3 ppm to 7.7 ppm).

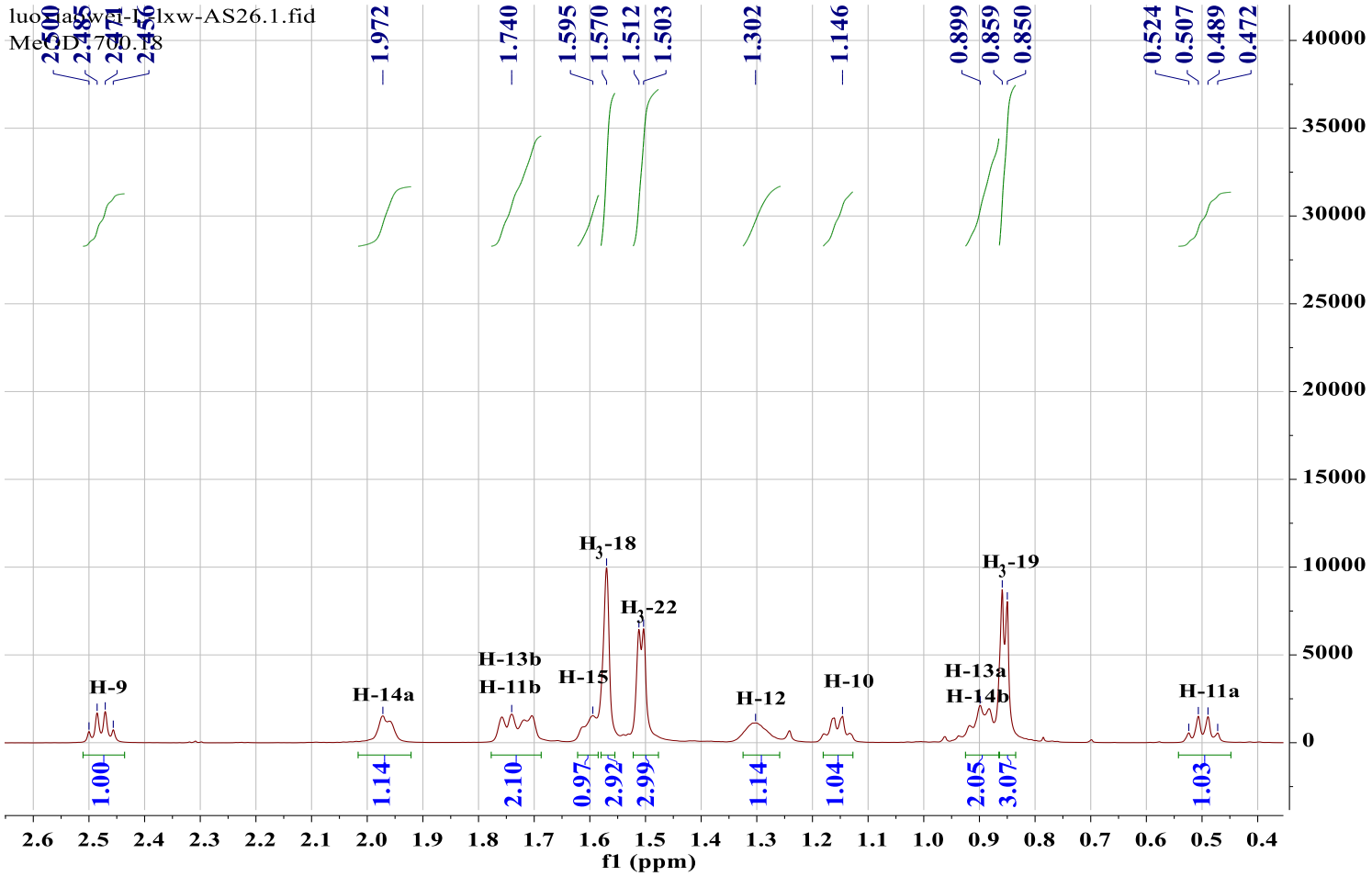


Figure S36: ^{1}H NMR spectrum of ilicicolin H (**3**) (CD_3OD, 700 MHz) (From δ_H 0.4 ppm to 2.6 ppm).

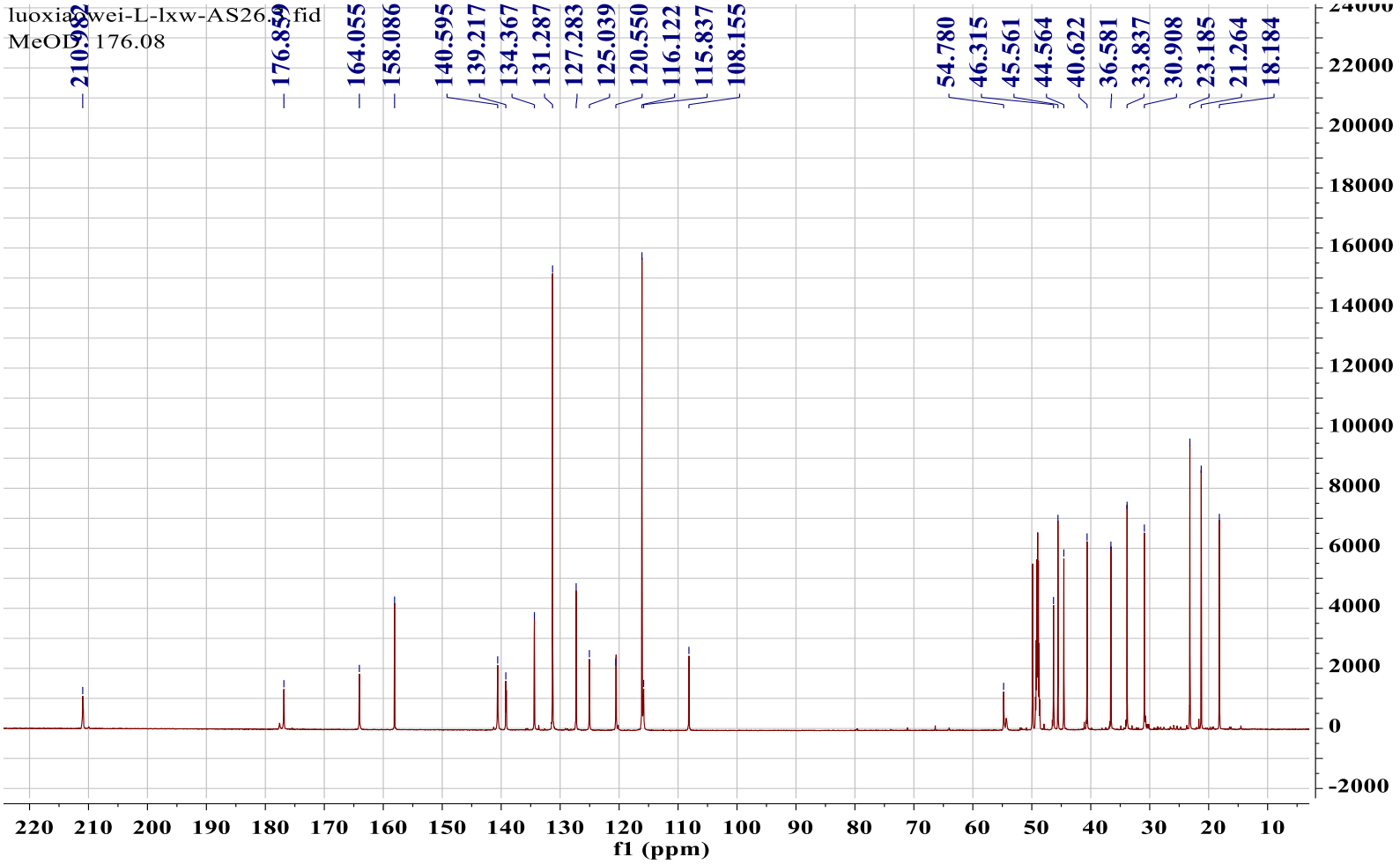


Figure S37: ^{13}C NMR spectrum of ilicicolin H (**3**) (CD_3OD, 175 MHz).

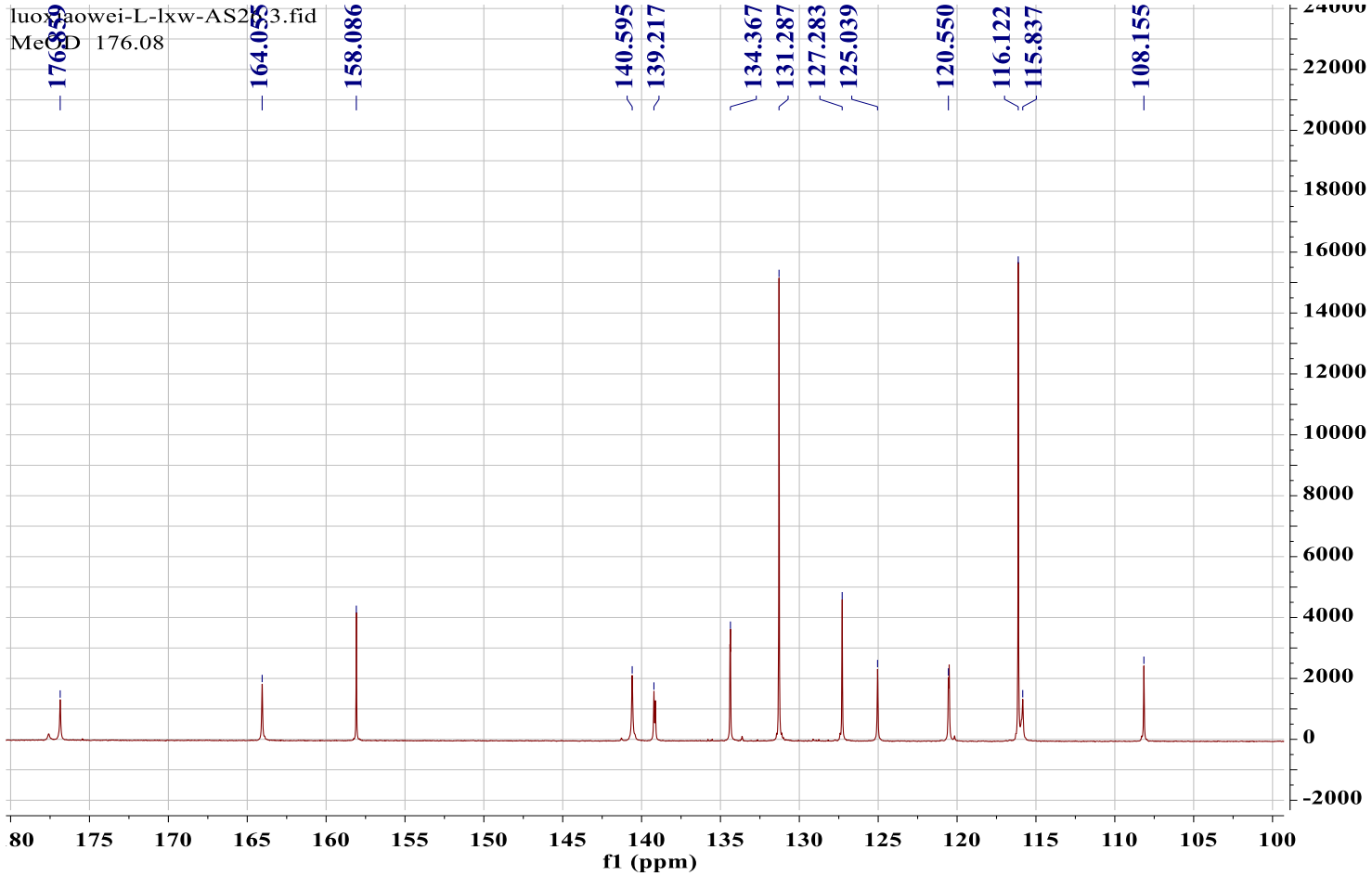


Figure S38: ^{13}C NMR spectrum of ilicicolin H (**3**) (CD_3OD, 175 MHz) (From δ_C 100 ppm to 180 ppm).

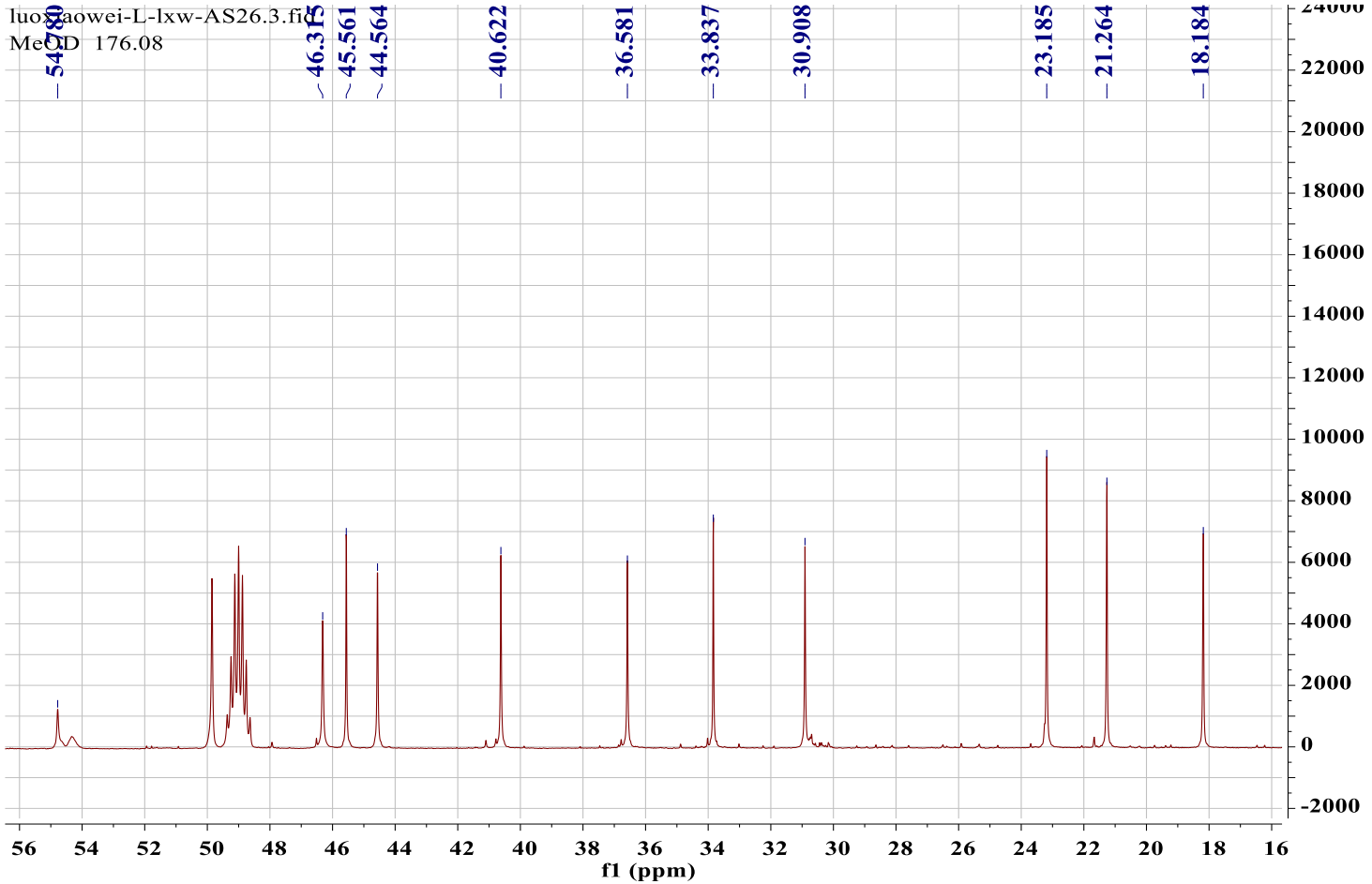


Figure S39: ^{13}C NMR spectrum of ilicicolin H (**3**) (CD_3OD, 175 MHz) (From δ_C 16 ppm to 56 ppm).

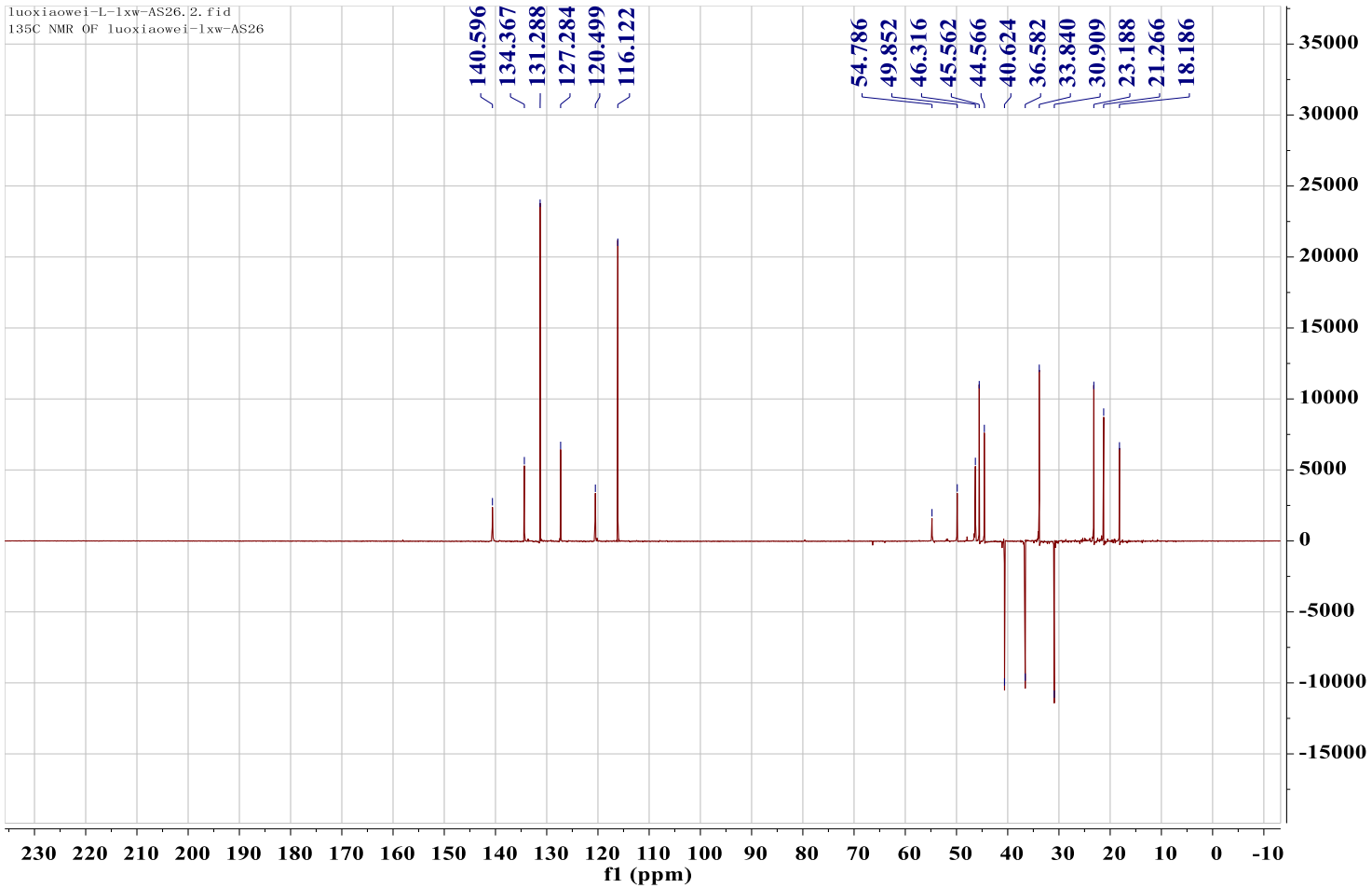


Figure S40: DEPT 135 NMR spectrum of ilicicolin H (**3**) (CD_3OD, 175 MHz).

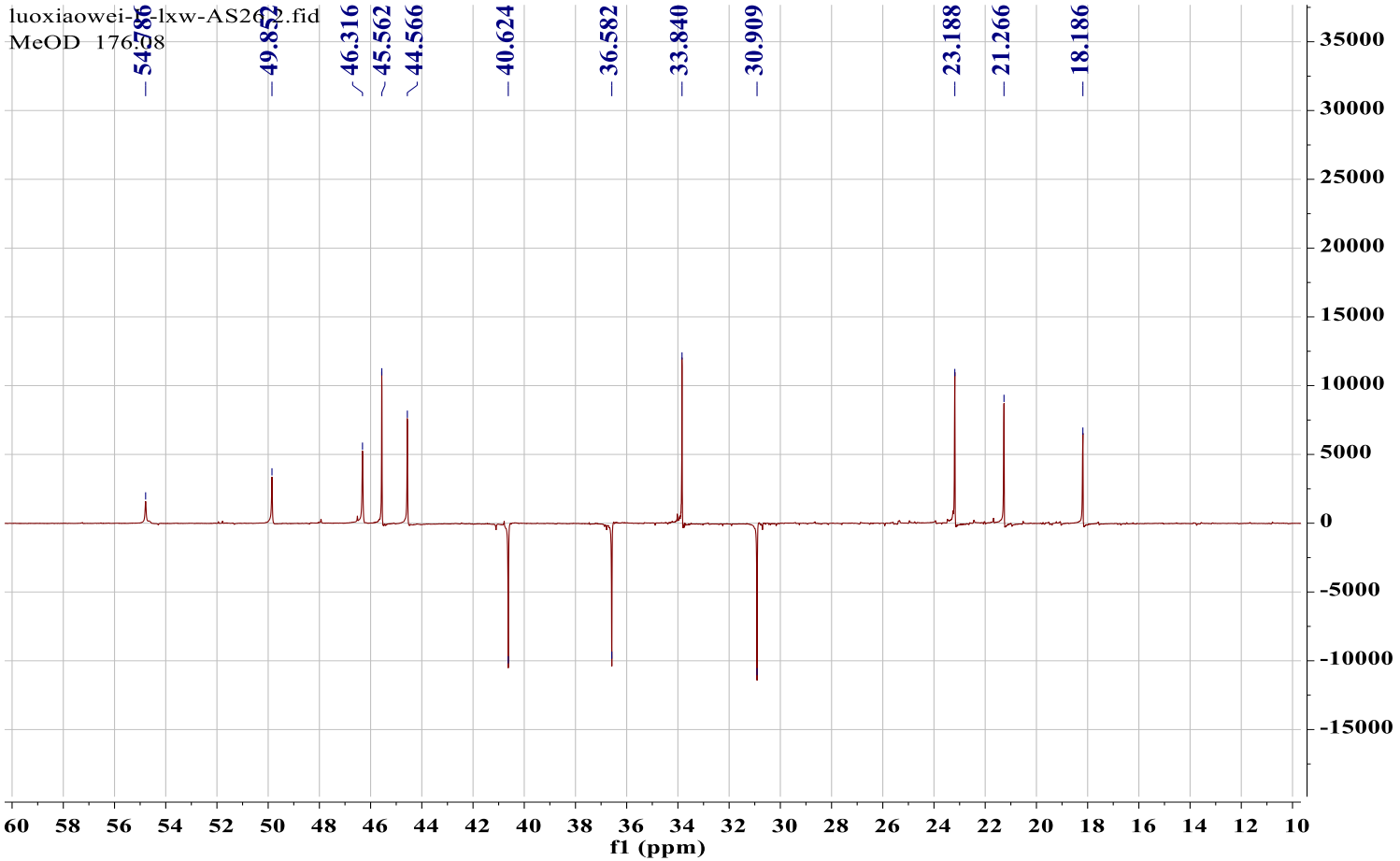
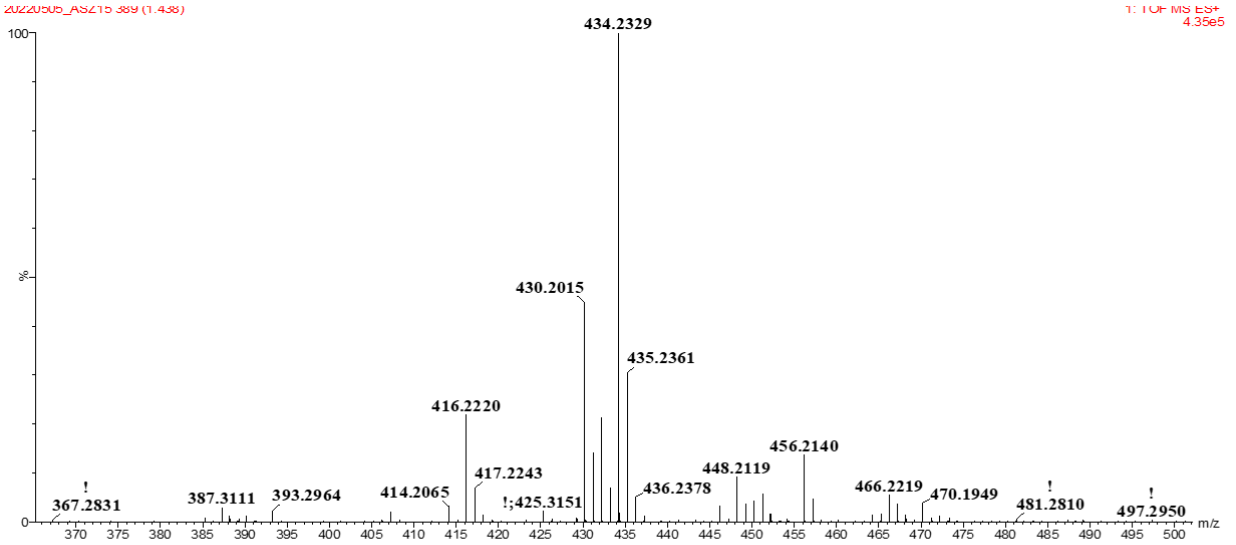


Figure S41: DEPT 135 NMR spectrum of ilicicolin H (**3**) (CD_3OD, 175 MHz) (From δ_C 10 ppm to 60 ppm).



Single Mass Analysis

Tolerance = 10.0 PPM / DBE: min = -1.5, max = 50.0

Element prediction: Off

Number of isotope peaks used for i-FIT = 3

Monoisotopic Mass, Even Electron Ions

6 formula(e) evaluated with 1 results within limits (up to 50 closest results for each mass)

Elements Used:

Mass	Calc. Mass	mDa	PPM	DBE	Formula	i-FIT	i-FIT Norm	Fit Conf %	C	H	N	O
434.2329	434.2331	-0.2	-0.5	12.5	C27 H32 N O4	942.4	n/a	n/a	27	32	1	4

Mass	Calc. Mass	mDa	PPM	DBE	Formula	i-FIT	i-FIT Norm	Fit Conf %	C	H	N	O	N...
456.2140	456.2151	-1.1	-2.4	12.5	C27 H31 N O4 Na	628.5	n/a	n/a	27	31	1	4	1

Figure S42: HR-ESIMS spectrum of ilicicolin H (**3**).

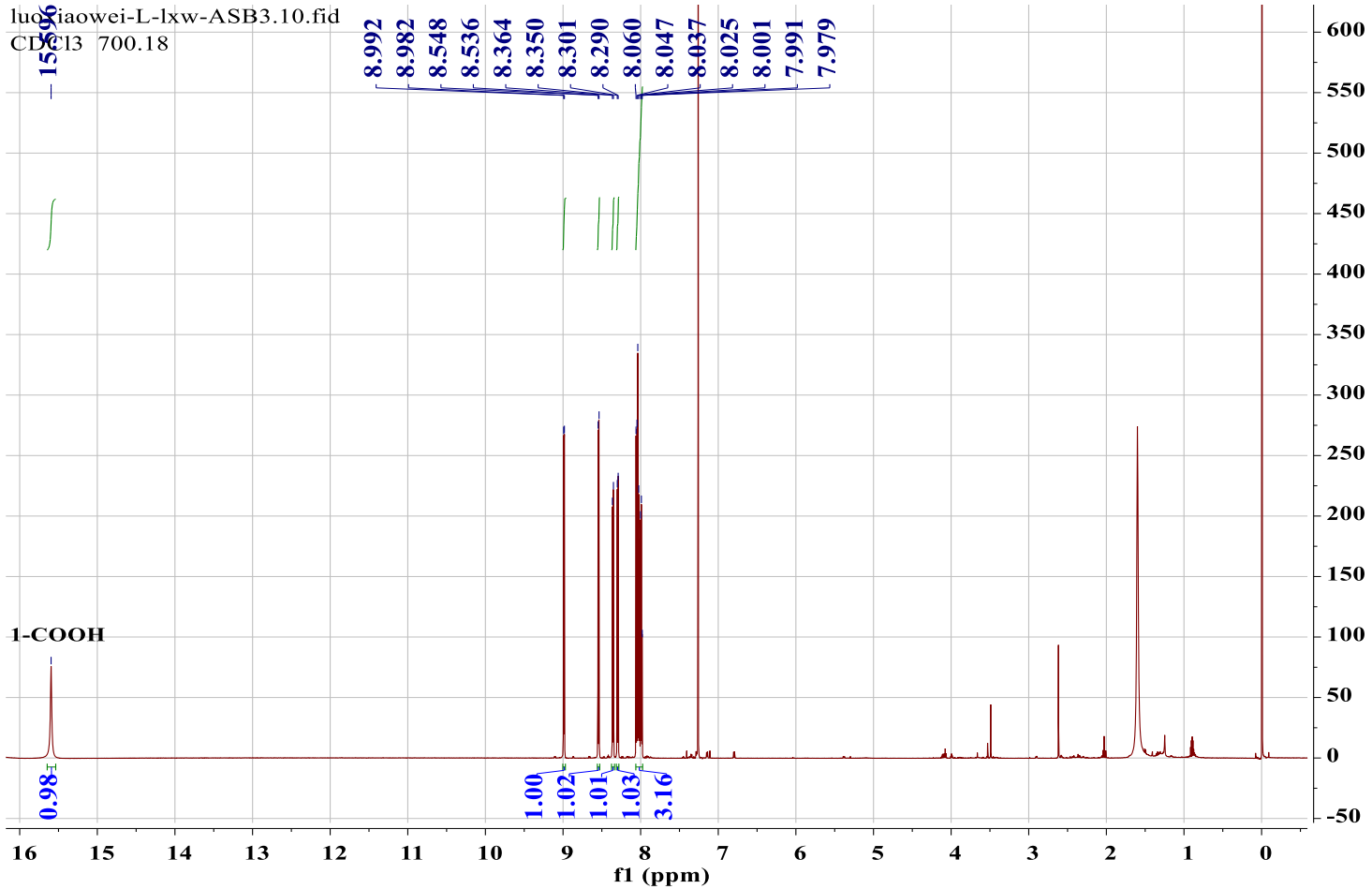


Figure S43: ^{1}H NMR spectrum of phenazine-1-carboxylic acid (**4**) ($CDCl_3$, 700 MHz).

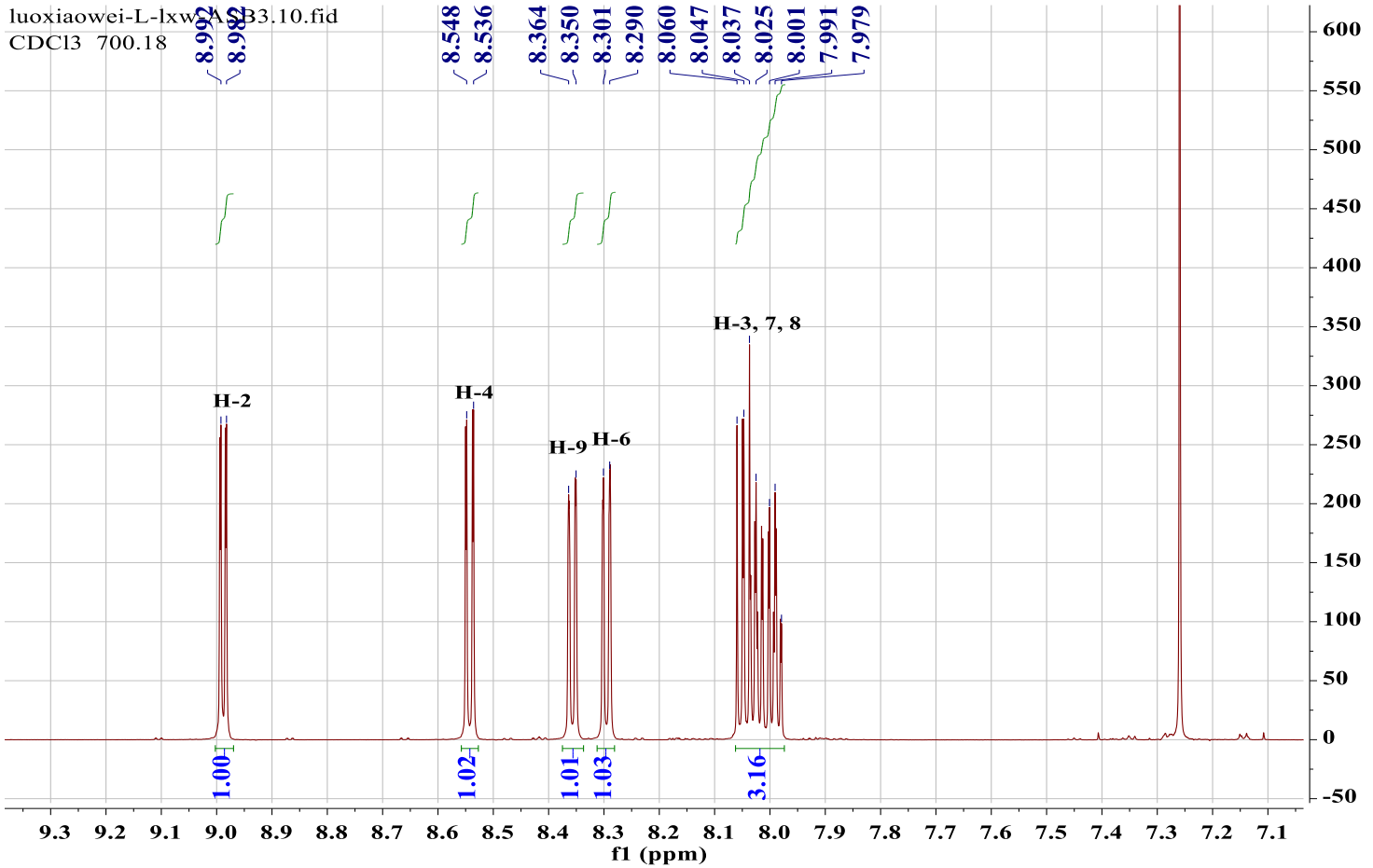


Figure S44: ^{1}H NMR spectrum of phenazine-1-carboxylic acid (**4**) ($CDCl_3$, 700 MHz) (From δ_H 7.1ppm to 9.3 ppm).

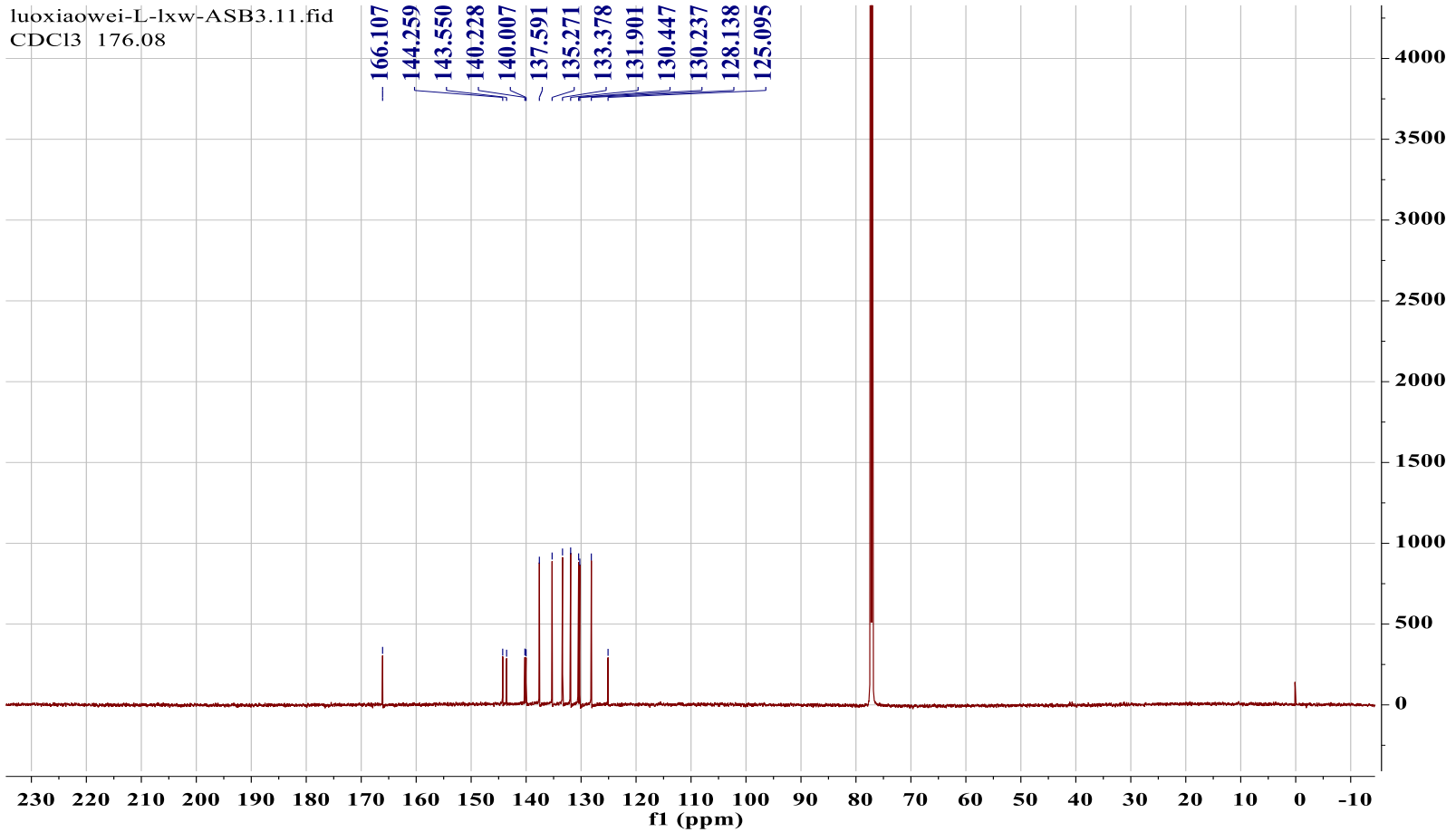


Figure S45: ^{13}C NMR spectrum of phenazine-1-carboxylic acid (**4**) ($CDCl_3$, 175 MHz).

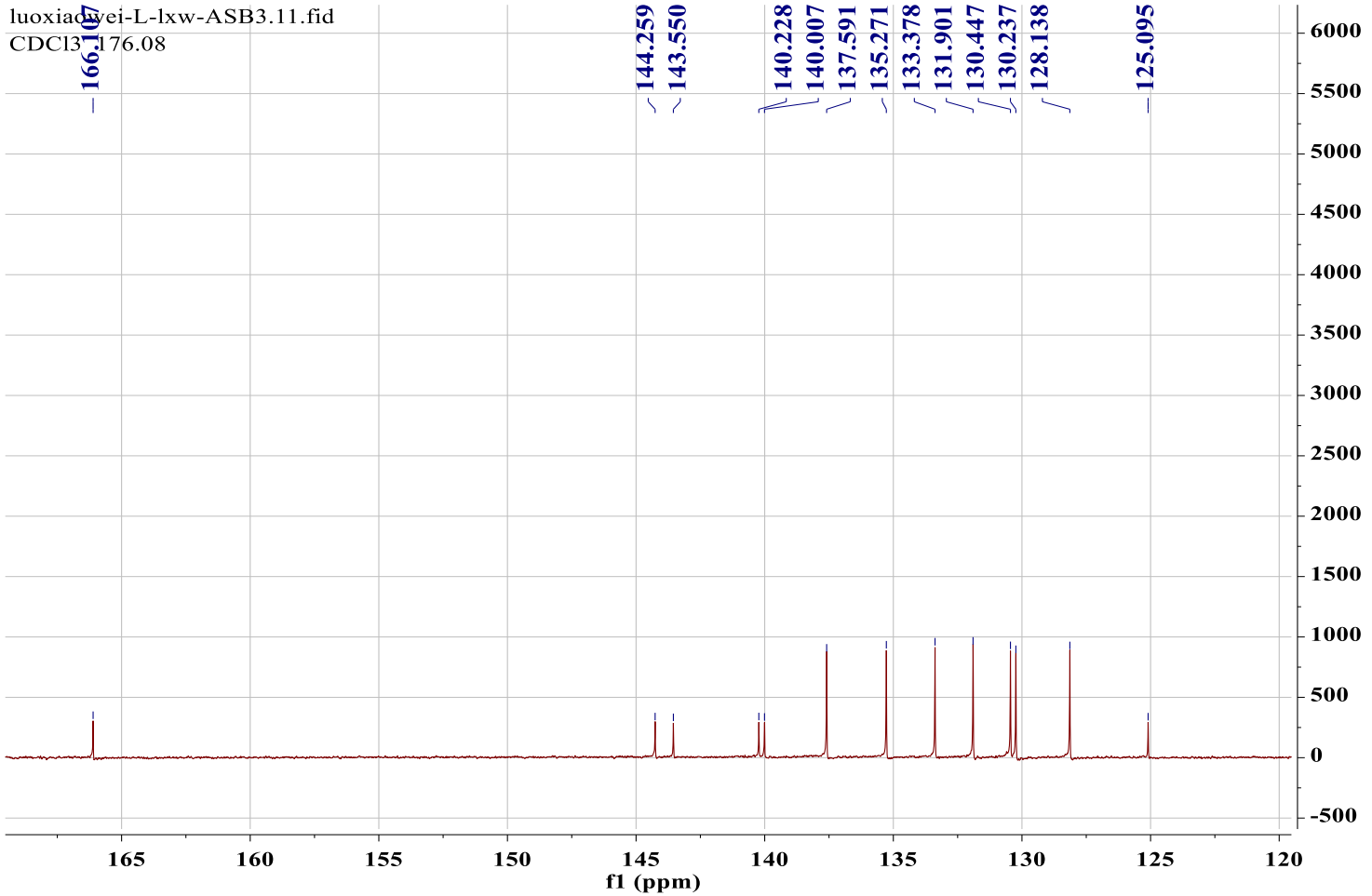


Figure S46: ^{13}C NMR spectrum of phenazine-1-carboxylic acid (**4**) ($CDCl_3$, 175 MHz) (From δ_C 120 ppm to 170 ppm).

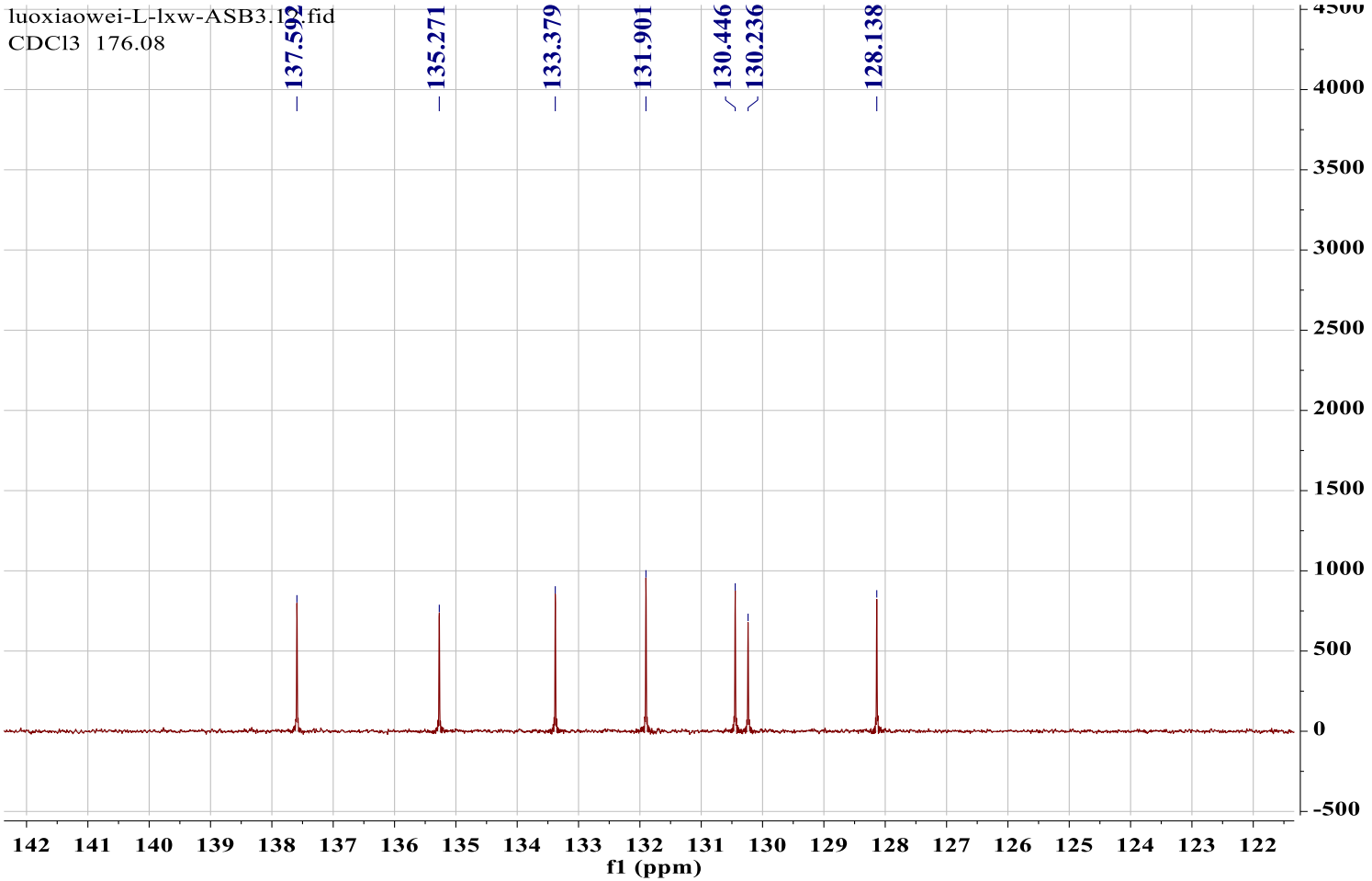
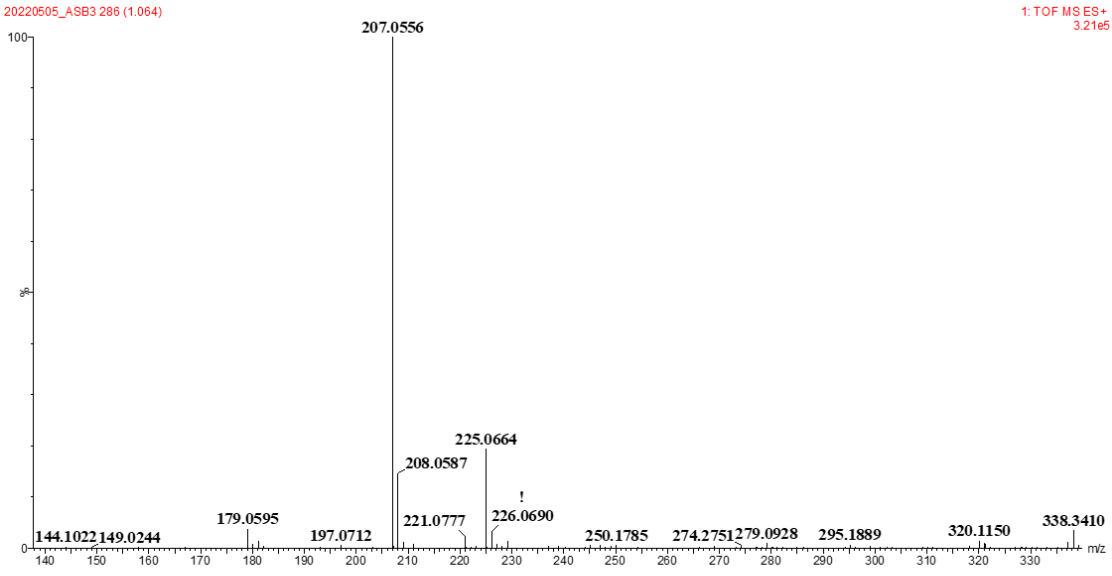


Figure S47: DEPT 135 NMR spectrum of phenazine-1-carboxylic acid (**4**) ($CDCl_3$, 175 MHz).



Single Mass Analysis

Tolerance = 10.0 PPM / DBE: min = -1.5, max = 50.0

Element prediction: Off

Number of isotope peaks used for i-FIT = 3

Monoisotopic Mass, Even Electron Ions

7 formula(e) evaluated with 1 results within limits (up to 50 closest results for each mass)

Elements Used:

Mass	Calc. Mass	mDa	PPM	DBE	Formula	i-FIT	i-FIT Norm	Fit Conf %	C	H	N	O
225.0664	225.0664	0.0	0.0	10.5	C13 H9 N2 ...	563.6	n/a	n/a	13	9	2	2

Mass	Calc. Mass	mDa	PPM	DBE	Formula	i-FIT	i-FIT Norm	Fit Conf %	C	H	N	O
207.0556	207.0558	-0.2	-1.0	11.5	C13 H7 N2...	830.5	n/a	n/a	13	7	2	1

Figure S48: HR-ESIMS spectrum of phenazine-1-carboxylic acid (**4**).

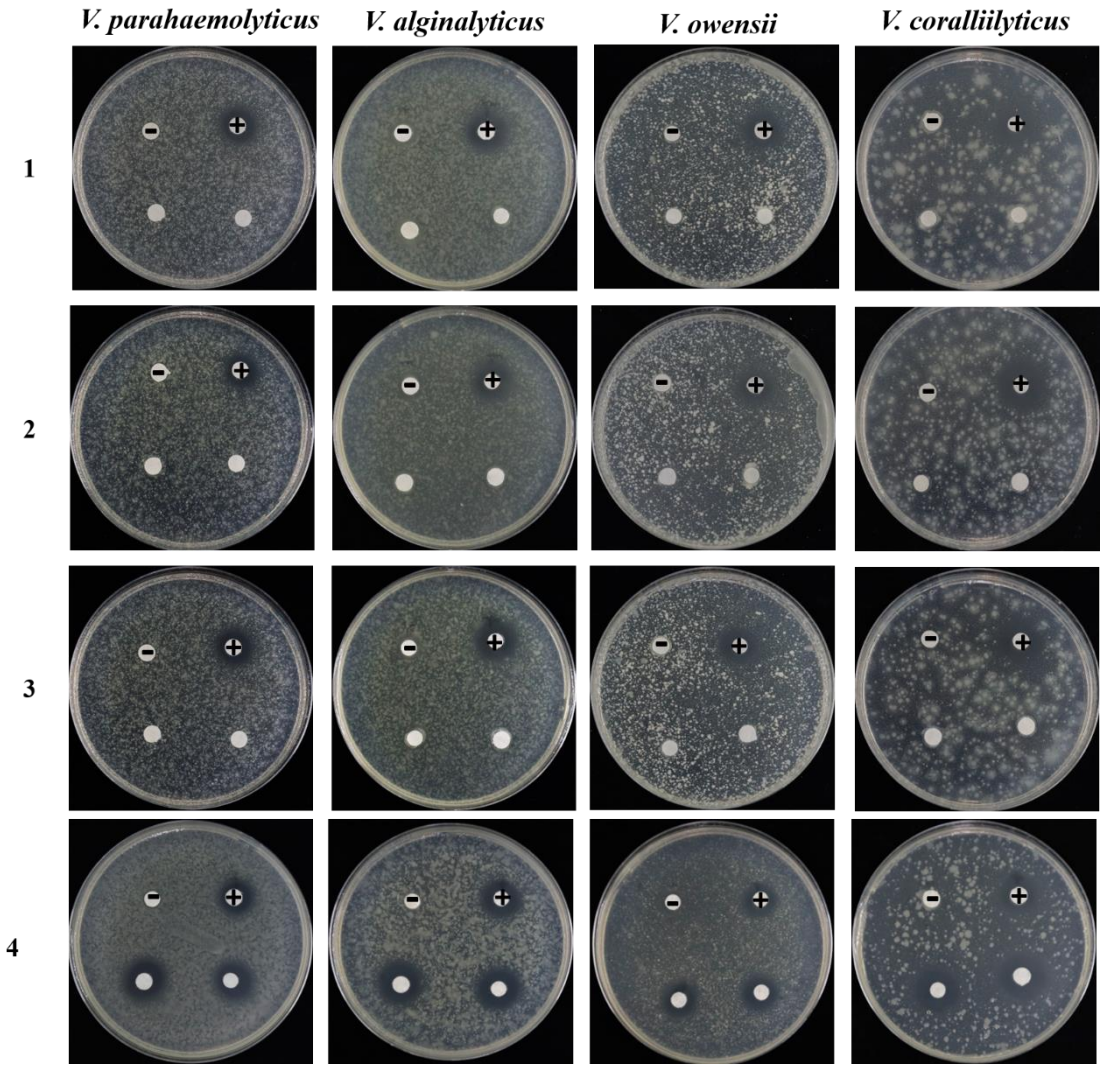


Figure S49: Anti-*Vibrio* activity of compounds **1**–**4** (3 μL 10 mg/mL) by agar diffusion method. The radius of zone of inhibition was measured in mm. (+: 0.1 mg/mL chloramphenicol, –: negative)

Table S1: The radius of zone (mm) of inhibition of compound **4** and chloramphenicol.

Compds	*V. parahaemolyticus*	*V. alginalyticus*	*V. owensii*	*V. coralliilyticus*
4	13.8	15.9	13.4	16.0
chloramphenicol	11.7	13.9	12.7	14.2

References

[1] Y.-T. Zhang, Z.-C. Li, B.-Y. Huang, K. Liu, S. Peng, X.-M. Liu, C.-H. Gao, Y.-H. Liu, Y.-H. Tan, and X.-W. Luo (2022). Anti-osteoclastogenic and antibacterial effects of chlorinated

polyketides from the Beibu Gulf coral-derived fungus *Aspergillus unguis* GXIMD 02505, *Mar. Drugs* **20**, 178.

[2] H.-M. Lu, Y.-H. Tan, Y.-T. Zhang, Z.-C. Li, J.-Y. Chen, C.-H. Gao, Y.-H. Liu and X.-W. Luo (2022). Osteoclastogenesis inhibitory phenolic derivatives produced by the Beibu Gulf coral-associated fungus *Acremonium sclerotigenum* GXIMD 02501, *Fitoterapia* **159**, 105201.

[3] C.-N. Wang, H.-M. Lu, C.-H. Gao, L. Guo, Z.-Y. Zhang, J.-J. Wang, Y.-H. Liu, S.-T. Xiang, J. Wang and X.-W. Luo (2021). Cytotoxic benzopyranone and xanthone derivatives from a coral symbiotic fungus *Cladosporium halotolerans* GXIMD 02502, *Nat. Prod. Res.* **35**, 5596–5603.

[4] X.-W. Luo, G.-D. Cai, Y.-F. Guo, C.-H. Gao, W.-F. Huang, Z.-H. Zhang, H.-M. Lu, K. Liu, J.-H. Chen, X.-F. Xiong, J.-P. Lei, X.-F. Zhou, J.-J. Wang and Y.-H. Liu (2021). Exploring marine-derived ascochlorins as novel human dihydroorotate dehydrogenase inhibitors for treatment of triple-negative breast cancer, *J. Med. Chem.* **64**, 13918–13932.

[5] C.-M. Chen, W.-H. Chen, X.-Y. Pang, S.-R. Liao, J.-F. Wang, X.-P. Lin, B. Yang, X.-F. Zhou, X.-W. Luo and Y.-H. Liu (2021). Pyrrolyl 4-quinolone alkaloids from the mangrove endophytic fungus *Penicillium steckii* SCSIO 41025: Chiral resolution, configurational assignment, and enzyme inhibitory activities, *Phytochemistry* **186**, 112730.