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Pecanthines A–C, unprecedented dimeric β-carboline alkaloids from the seeds of *Peganum harmala*

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ABSTRACT

Three novel β-carboline alkaloid dimers, pecanthines A–C (**1**–**3**), featuring unprecedented 6/5/6/6–5/6 and 6/5/6/6/5/6 polycyclic frameworks, were isolated from the seeds of *Peganum harmala*. Their structures were elucidated by spectroscopic analyses and X-ray diffraction. Notably, compounds **1** and **2** represent the first heterodimeric alkaloids merging canthin-6-one and melatonin-type scaffolds, with **1** additionally displaying neuroprotective activity. In addition, a gram-scale synthesis of **3** was achieved in six steps, leveraging a key acyl-ketene imine condensation and Suzuki coupling. Biological evaluation revealed that **3** acts as a new topoisomerase I (Topo I) inhibitor, exhibiting remarkable antiproliferative activity. Mechanistic studies demonstrated that **3** induces DNA damage, triggering apoptosis and cell cycle arrest in cancer cells. The discovery and gram-scale synthesis of **3** provide a promising lead compound and a novel molecular scaffold for anticancer drug development.

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DNA topoisomerase I (Topo I), a pivotal enzyme orchestrating DNA replication and transcription, is a validated drug target for cancer therapy [1,2]. Topo I inhibitors exert their therapeutic effects by stabilizing the DNA-Topo I cleavable complex, thereby preventing DNA religation and subsequently inducing DNA damage and tumor cell apoptosis [3–5]. Currently, the clinical use of Topo I inhibitors is largely restricted to camptothecin derivatives such as irinotecan and topotecan [6,7]. Although these agents demonstrate significant efficacy against various malignancies, their clinical utility is substantially limited by dose-limiting toxicities and acquired drug resistance, which severely compromise long-term therapeutic outcomes [8–11]. Therefore, there is an urgent need for Topo I inhibitors with novel chemical scaffolds.

β-Carboline alkaloids are nitrogen-containing heterocycles biosynthesized from tryptophan with a unique tricyclic pyrido[3,4-b]indole skeleton [12]. This structure is associated with a range of pharmacological activities, with its notable antitumor potential attracting significant attention [12–15]. Among natural sources, plants of the *Peganum genus* have been identified as exceptionally rich repositories of structurally diverse β-carboline derivatives [16]. Critically, many of these compounds, such as harmine and its analogs, have been identified as Topo I inhibitors, making them a key focus for phytochemists and medicinal chemists [17–20].

The seeds of *Peganum harmala* have been traditionally employed in Chinese folk medicine for the treatment of various ailments, including cancer, rheumatism, cough, and asthma [21–24]. In a previous study, we reported the isolation of new β-carboline alkaloids [24,25]. As a part of our ongoing research, three novel long conjugated β-carboline alkaloids, pecanthines A–C (**1**–**3**), were isolated from the seeds of *P. harmala* (Fig. 1). Their structures were elucidated by spectroscopic analyses and X-ray diffraction. Compounds **1** and **2** represent the first heterodimeric alkaloids merging canthin-6-one and melatonin-type scaffolds. More strikingly, the structure of peganumium B reported from the seeds of *P. harmala* was revised to be compound **3** (Fig. 1) [26]. Herein, we report the isolation, structural elucidation, hypothetical biogenetic pathway, and biological evaluation of **1**–**3**. Meanwhile, the

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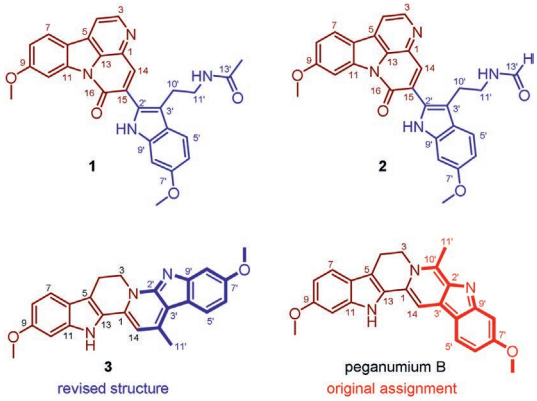


Fig. 1. The structures of compounds **1**–**3** and peganumium B.

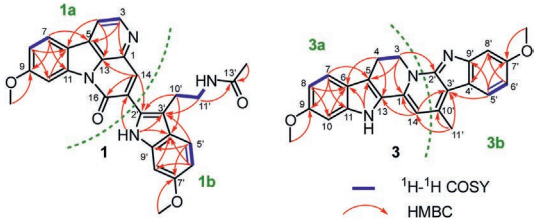


Fig. 2. Key ^{1}H–^{1}H COSY and HMBC correlations of **1** and **3**.

first gram-scale synthesis of **3** was achieved, further confirming the correctness of the structure.

Pecanthine A (**1**) was isolated as pink needle-shaped crystals. The molecular formula of **1** was established to be $C_{28}H_{24}N_4O_4$ from its high-resolution electrospray ionization mass spectrometry (HR-ESI-MS) data (*m/z* 481.1872 $[M+H]^+$, calcd. for $C_{28}H_{25}N_4O_4$ 481.1870). The ^{1}H and ^{13}C nuclear magnetic resonance (NMR) spectra data showed the presence of two AMX spin systems [δ_H 8.32 (1H, d, J = 8.7 Hz), δ_H 8.16 (1H, d, J = 2.4 Hz), δ_H 7.56 (1H, d, J = 8.7 Hz), δ_H 7.24 (1H, dd, J = 8.7, 2.4 Hz), δ_H 6.97 (1H, d, J = 2.3 Hz), δ_H 6.73 (1H, dd, J = 8.7, 2.3 Hz)], three aromatic protons [δ_H 8.83 (1H, d, J = 5.0 Hz), δ_H 8.20 (1H, d, J = 5.0 Hz), δ_H 8.15 (1H, s)], two methoxys [δ_H 3.94 and 3.80 (each 3H, s); δ_C 55.9 and 55.2], a methyl [δ_H 1.71 (3H, s); δ_C 22.6], and an indole NH signal [δ_H 11.14 (1H, s)]. The above data indicated that **1** was a dimeric β-carboline alkaloid. The ^{1}H and ^{13}C NMR data of **1** were definitively assigned by integrating 2D NMR experiments, as documented in Table S1 (Supporting information).

In the ^{1}H–^{1}H COSY spectrum, the correlations between H-3 and H-4, between H-7 and H-8, between H-5′ and H-6′, and between H_2–11′ (δ_H 3.34) and H_2–10′ (δ_H 2.98)/NH-12′ (δ_H 8.00) indicated the presence of four spin systems (C-3 to C-4, C-7 to C-8, C-5′ to C-6′, and C-10′ to N-12′) in **1** (Fig. 2). In the heteronuclear multiple bond correlation (HMBC) spectrum, correlations between H-3 and C-1/C-5, between H-4 and C-6/C-13, between H-7 and C-5/C-9/C-11, between H-8 and C-6/C-10, between H-10 and C-6, between H-14 and C-13/C-16, as well as between 9–OCH_3 and C-9 suggested the existence of a 9-methoxycanthin-6-one alkaloid unit **1a** (Fig. 2). The HMBC correlations between H-5′ and C-3′/C-7′/C-9′, between H-6′ and C-4′/C-8′, between H-8′ and C-4′, between H_2–10′ and C-2′/C-4′, between H_2–11′ and C-3′/C-13′, H_3–14′ and C-13′, between 7′–OCH_3 and C-7′, as well as between NH-1′ and C-3′/C-4′ suggested the presence of a melatonin-type unit **1b** (Fig. 2). Furthermore, the HMBC correlations between H-14 and C-2′, and between NH-1′ and C-15 implied that the substructures unit **1a** and unit **1b** were connected through C-15–C-2′ bonds. Subsequently, crystals of **1** for single-crystal X-ray diffraction were acquired from CH_3OH–CH_2Cl_2 (1:1). Thus, compound **1** was defined

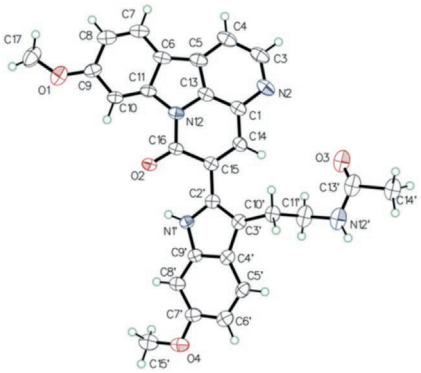


Fig. 3. X-ray ORTEP diagram of **1**.

as an unprecedented heterodimer alkaloid and further determined by single-crystal X-ray diffraction (Cu Kα) analysis (Fig. 3).

The molecular formula of **2** was determined to be $C_{27}H_{22}N_4O_4$ based on its HR-ESI-MS data (*m/z* 467.1711 $[M+H]^+$, calcd. for $C_{27}H_{23}N_4O_4$, 467.1714). Analysis of the ^{1}H and ^{13}C NMR spectra of **2** was similar to those of **1**, except for the absence of a methyl signal (δ_C 22.6, C-14′). The structure was supported by the ^{1}H–^{1}H correlation spectroscopy (COSY) correlation between NH-12′ and H-13′, as well as the HMBC correlations between H-13′ and C-11′, between H_2–11′ and C-13′ (Fig. S1 in Supporting information). Thus, compound **2** was elucidated and named pecanthine B.

Pecanthine C (**3**) was yielded as yellow powder. The molecular formula of **3** was deduced to be $C_{24}H_{21}N_3O_2$ on the basis of its HR-ESI-MS at *m/z* 384.1709 $[M+H]^+$ (calcd. for $C_{24}H_{22}N_3O_2$: 384.1707). The UV spectrum of **3** exhibited absorption maxima at 205, 223, 280, 310 and 432 nm. Analysis of the ^{1}H and ^{13}C NMR spectra of **3** revealed the presence of two AMX spin systems [δ_H 7.91 (1H, d, J = 8.6 Hz), δ_H 7.53 (1H, d, J = 8.7 Hz), δ_H 7.10 (1H, d, J = 2.3 Hz), δ_H 6.92 (1H, d, J = 2.2 Hz), δ_H 6.77 (1H, dd, J = 8.7, 2.2 Hz), δ_H 6.74 (1H, dd, J = 8.6, 2.3 Hz)], two methoxyls [δ_H 3.84 and 3.83 (each 3H, s); δ_H 55.1 and 55.3], a methyl [δ_H 2.82 (3H, s); δ_H 19.6], and an indole NH signal [δ_H 11.73 (1H, s)]. Comprehensive analysis of ^{1}H–^{1}H COSY, heteronuclear single quantum coherence (HSQC), and HMBC spectra of **3** resulted in the unambiguous assignment of its ^{1}H and ^{13}C NMR signals as shown in Table S3.

In the ^{1}H–^{1}H COSY spectrum, the correlations between H_2–3 (δ_H 4.92) and H_2–4 (δ_H 3.26), between H-7 and H-8, and between H-5′ and H-6′ indicated the presence of three spin systems (C-3 to C-4, C-7 to C-8, and C-5′ to C-6′) in **3** (Fig. 2). In the HMBC spectrum, correlations between H_2–3 and C-1/C-5, between H_2–4 and C-6/C-13, between H-7 and C-5/C-9/C-11, between H-8 and C-6/C-10, between H-10 and C-6, between H-14 and C-1/C-13, between 9–OCH_3 and C-9, as well as between 12-NH and C-5/C-6 suggested the existence of a harmaline unit **3a** (Fig. 2). The HMBC correlations between H-5′ and C-3′/C-7′/C-9′, between H-6′ and C-8′, between H-8′ and C-4′, between H_3–11′ and C-3′/C-10′, as well as between 7′–OCH_3 and C-7′ suggested the presence of a 3-ethylidene-indole unit **3b** (Fig. 2). Furthermore, the HMBC correlations between H_2–3 and C-2′, between H-14 and C-3′, and between H_3–11′ and C-14 indicated that the substructures **3a** and **3b** were connected through N-2–C-2′ and C-14–C-10′ bonds. Therefore, the planar structure of **3** was established as a unique long conjugated 6/5/6/6/5/6 fused six-ring system.

Peganumium B, recently reported from the seeds of *P. harmala* as a β-carboline dimer with one novel long conjugated structure, has attracted our attention [26]. Interestingly, compound **3** and peganumium B share identical molecular weights and NMR data, prompting us to re-evaluate peganumium B's structure. In the original literature, the authors deduced peganumium B as a

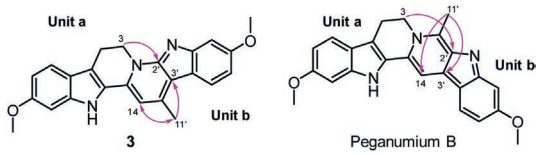


Fig. 4. This unobserved HMBC correlations in Liu *et al.*'s study represents a three-bond coupling in **3**, but a four- to five-bond coupling in peganumium B.

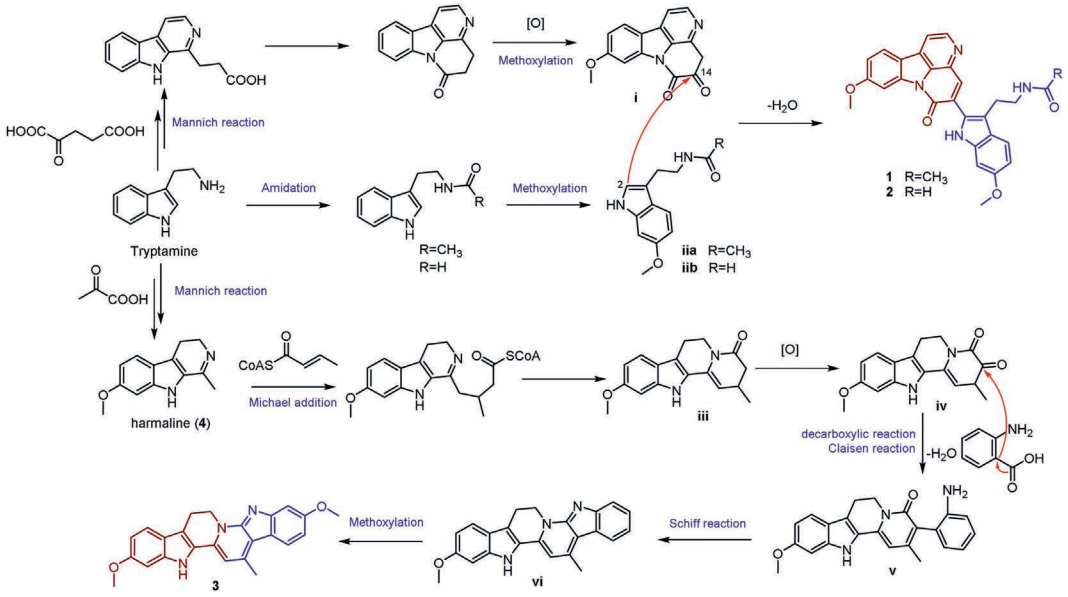
β-carboline dimer based on NMR analysis. They further identified unit **a** as harmaline through HMBC spectroscopy, which is consistent with our analysis of **3** in this study. Subsequently, the linkage between units **a** and **b** was inferred from chemical shifts and HMBC correlations. However, we have found that the HMBC spectrum of peganumium B exhibits strong cross-peaks for H_2–3/C-2′, H-14/C-11′, and H_3–11′/C-14 and C-3′ (Fig. S2 in Supporting information), which were not reported in the original study [26]. These correlations perfectly match those of **3**, where each pair of atoms is separated by three bonds, whereas in peganumium B, they would be separated by four or five bonds (Fig. 4). Thus, a comprehensive re-evaluation of the spectroscopic data led to the revision of peganumium B as structure **3**.

Compounds **1**–**3** represent two novel classes of dimeric β-carboline alkaloid analogues. The plausible biogenetic pathway of **1**–**3** could be proposed based on the findings of key biogenetic precursors in the plant (Scheme 1). The biosynthesis initiates with a Mannich reaction between tryptamine and 2-ketoglutaric acid, followed by successive oxidation, methoxylation, and ring condensation to afford the pivotal precursor **i** [27,28]. Dimerization of **i** with intermediate **iia**/**iib** through nucleophilic addition-elimination accounts for the formation of **1** and **2**. As for compound **3**, its source can be traced back to its precursor, harmaline. First, harmaline (**4**) undergoes a Michael addition with crotonyl-CoA, followed by intramolecular condensation to afford the key intermediate **iii** [29,30]. Subsequent oxidation of **iii** yields intermediate **iv**, which then engages in a Claisen reaction with anthranilic acid to generate the dehydrated intermediate **v** [31]. Finally, **3** is formed through Schiff reaction and methoxylation.

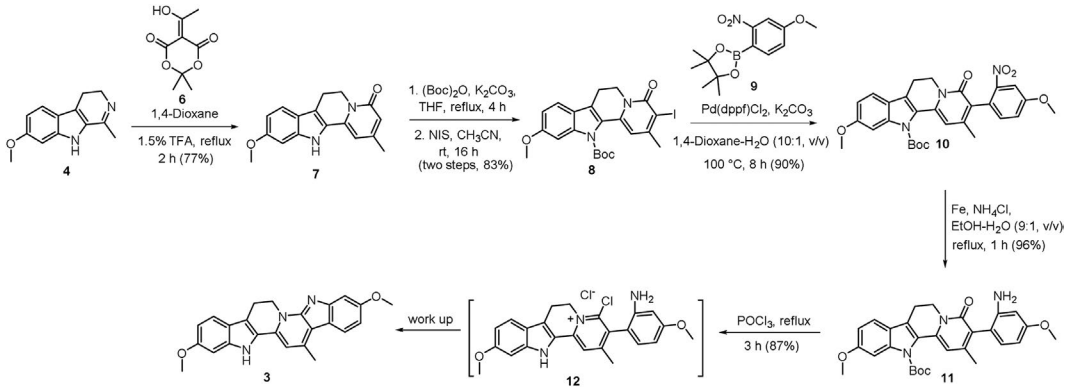
Following the structural revision of peganumium B to **3** and unsuccessful attempts to obtain X-ray-quality crystals, we designed a biomimetic synthesis of **3** according to the biogenic pathway (Scheme 2). The synthesis commenced with harmaline (**4**) from *P. harmala* seeds. Using commercially available Meldrum's acid derivative **6** as an acylketene source, the key intermediate **7** was synthesized in 77 % yield [32]. Subsequent Boc protection of the indole NH followed by iodination at the α-position of the carbonyl group afforded intermediate **8** in 83 % yield over two steps. Next, a Suzuki-Miyaura coupling of **8** with boronic ester **9** was performed using $Pd(dppf)Cl_2$ and K_2CO_3 in 1,4-dioxane at 100 °C, affording the desired **10** in 90 % yield. Subsequent reduction with iron powder and NH_4Cl cleanly afforded intermediate **11** in 96 % yield. The final transformation involved $POCl_3$-mediated intramolecular cyclization under reflux conditions, simultaneously effecting N-Boc deprotection. Ammonia workup delivered **3** in 87 % yield on gram scale. The ^1H and ^{13}C NMR spectra of synthetic **3** matched those of the natural isolate.

β-Carboline alkaloids have attracted considerable attention due to their notable neuroprotective and antitumor activities. Our study demonstrated that compound **1** exhibited dose-dependent neuroprotective effects in an L-glutamate-induced HT-22 cell model (Fig. S3 in Supporting information). Meanwhile, compound **3** displayed significant cytotoxicity against PC-9 and H1299 cells, with the half maximal inhibitory concentration (IC_{50}) values of 0.856 ± 0.13 and 1.312 ± 0.08 μmol/L, respectively (Fig. 5a). Colony formation assays (Figs. 5b and c) and EdU proliferation assays (Figs. S4a and b in Supporting information) revealed that compound **3** effectively suppressed tumor cell clonogenicity and DNA synthesis. Furthermore, wound healing and transwell invasion assays confirmed that **3** significantly inhibited the migratory and invasive capabilities of PC-9 cells (Figs. S4c–g in Supporting information).

To elucidate the anti-tumor molecular mechanism of compound 3, we focused on Topo I, a classic anti-tumor target. The results of relaxation experiments showed that 3 had significantly better inhibitory activity against Topo I than the positive control drug camptothecin (Fig. 5d). Molecular docking analysis revealed that 3 formed hydrogen bond interactions with Topo I and produced π-π stacking effects with DNA base pairs (Fig. 5e). These molecular interactions were the structural basis for its inhibitory activity against Topo I. Given that compound 3 has potent Topo I inhibition and antiproliferative activity, we further examined its effect on



Scheme 1. Hypothetical biosynthetic pathways for **1–3**.



Scheme 2. Synthesis of **3** from **4**, **6**, and **9**.

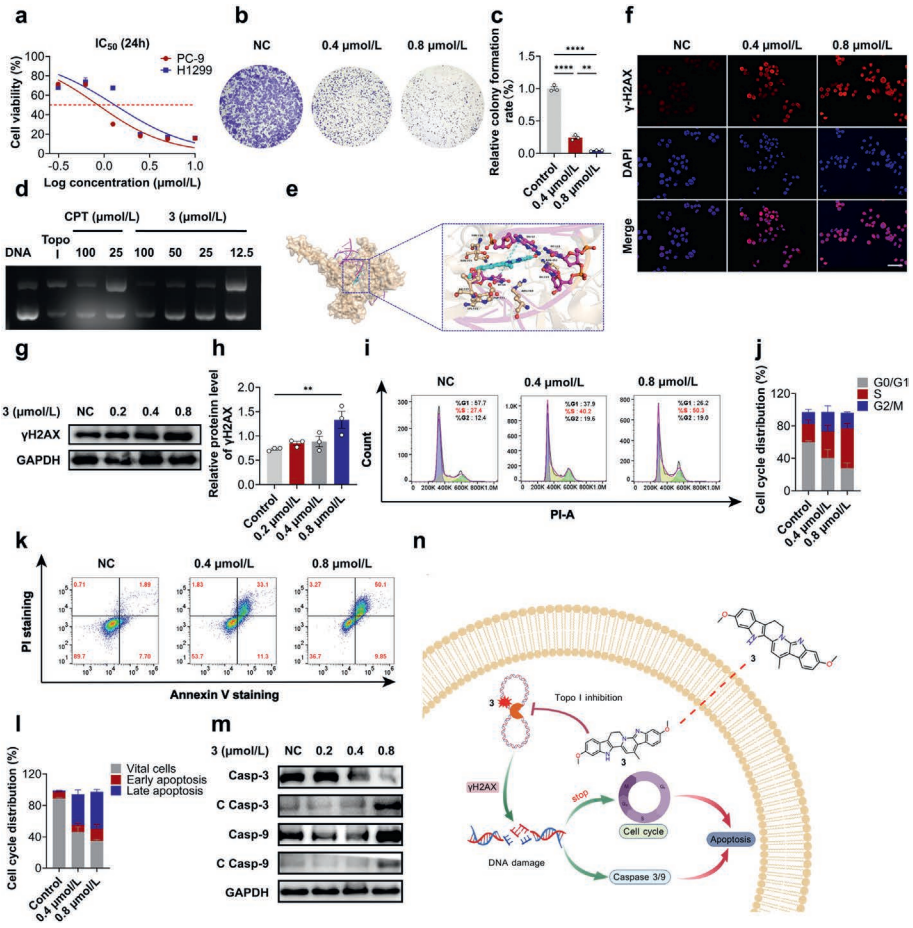


Fig. 5. Biological activity test results of **3**. (a) The cytotoxicity of **3** against PC-9 and H1299 cell lines was evaluated by MTT assay for 24 h. (b) The inhibitory effect of **3** on colony formation in PC-9 cells was assessed by clonogenic assay. (c) Colony formation rates. (d) Compound **3** inhibited Topo I at concentration gradient. (e) Docking of **3** with the Topo I/DNA cleavage complex (PDB: 1T8I). Hydrogen bonds and $\pi-\pi$ stacking interactions are represented by yellow and blue dashed lines, respectively. (f) γ-H2AX fluorescence was measured using fluorescence microscopy. Scale bar: 100 μm. (g) The protein expression levels of γ-H2AX. (h) Statistical analysis of the protein expression levels of γ-H2AX. (i) Cell cycle arrest induced by **3** in PC-9 cells. (j) Statistical analysis of cell cycle distribution. (k) Apoptosis induced by compound **3** in PC-9 cells. (l) Statistical analysis of apoptosis distribution. (m) The expression of apoptosis-related proteins in PC-9 cells treated with **3** was detected by Western blot. (n) Working model: compound **3** inhibits Topo I activity, which results in DNA damage, S-phase cell cycle arrest, and ultimately, apoptosis in tumor cells. The results were the mean ± SD of three independent experiments. $^{**}P < 0.01$, $^{****}P < 0.0001$ *vs.* the control group.

DNA damage. Immunofluorescence detection and Western blot revealed that the expression level of γ-H2AX, a DNA damage marker, was significantly increased in PC-9 cells treated with 3 (Figs. 5f–h). Flow cytometry analysis showed that 3 could arrest the PC-9 cell cycle at the S phase (Figs. 5i and j) and significantly induce cell apoptosis (Figs. 5k–m).

In summary, three novel long-conjugated β-carboline alkaloids, pecanthines A–C (**1**–**3**), were isolated from the seeds of *P. harmala*. Compounds **1** and **2** represent the first heterodimeric alkaloids merging canthin-6-one and melatonin-type scaffolds. The structure of peganumium B reported from the seeds of *P. harmala* was revised to be compound **3**. Meanwhile, a gram-scale synthesis

of **3** was achieved in six steps, leveraging a key acyl-ketene imine condensation and Suzuki coupling, further corroborating its revised structure based on NMR analysis. The bioassays revealed that compound **1** exhibited dose-dependent neuroprotective effects in an L-glutamate-induced HT-22 cell model. Compound **3** displayed significant cytotoxicity against PC-9 and H1299 cells, with IC_{50} values of 0.856 ± 0.13 and 1.312 ± 0.08 μmol/L, respectively. Significantly, compound **3** inhibits tumor cell proliferation by suppressing Topo I activity, inducing DNA damage, arresting the cell cycle, and promoting apoptosis (Fig. 5n). This finding provides a new structural template for the development of Topo I inhibitors.

CRediT authorship contribution statement

Qing Tang: Writing – original draft, Software, Investigation, Data curation, Conceptualization. **Kai Zhao:** Validation, Resources, Investigation, Data curation. **Hui-Hui Zhu:** Software, Investigation, Formal analysis. **Qiang Lin:** Resources. **Hai-Yue Zhao:** Validation. **Wei-Xuan Zheng:** Software. **Zhong-Nan Wu:** Software, Resources. **Hao Wang:** Methodology, Investigation. **Guo-Cai Wang:** Writing – review & editing, Supervision, Project administration, Investigation, Funding acquisition, Data curation. **Yu-Bo Zhang:** Writing – review & editing, Writing – original draft, Visualization, Project administration.

Declaration of competing interest

The authors declare that they have no known competing financial interest or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgments

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.cclet.2025.112172.

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