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Insights into the daily functions of radix *Panax notoginseng*

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A total of 48 compounds, including 6 undescribed triterpenoid saponins, notoginsenosides V1–V6 (**1–6**), were isolated and identified from the radix of *Panax notoginseng*. Their structures were elucidated by combining various spectroscopic techniques, such as NMR and MS analyses. Among them, (2*S*)-1-*O*-(9*Z*,12*Z*-octadecadienoyl)-3-*O*-β-galactopyranosylglycerol (**43**) exhibited the strongest anti-inflammatory activity against cyclooxygenase-2. The compounds **7**, **14**, **15**, **16**, **28**, **29**, **30**, **39**, **43**, and **45** promoted the proliferation of human oral mucosa fibroblasts (hOMFs). Additionally, the compounds **1**, **8**, **9**, **14**, **35**, **43**, and **45** promoted the proliferation of human dermal papilla cells (hDPCs). These results revealed the potential daily functional benefits of *P. notoginseng*, including ameliorating oral ulcer conditions and preventing hair loss.

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Introduction

Panax notoginseng (Burk.) F. H. Chen, commonly known as Sanqi or Tianqi, is an agricultural product of high economic value.[1,2] Nowadays, all Sanqi is artificially cultivated, particularly in Yunnan, China. It has been used to treat cardiovascular diseases, various aches, inflammations, and trauma.[2–4] Extensively cultivated in the Yunnan and Guangxi provinces in China, *P. notoginseng* serves as the main raw material for the production of traditional Chinese medicines, such as the Yunnan Baiyao, Compound Danshen dripping pills, Xuesaitong injection, and Pientzehuang.[5] To date, more than 200 secondary metabolites have been isolated from *P. notoginseng*, including saponins, polysaccharides, flavonoids, sterols, carbenes, and amino acids.[4,6–8] Generally, *Panax notoginseng* saponins (PNS) are the major and characteristic components of *P. notoginseng*[4] and are considered the foundation of its pharmacological efficacy. Beyond the pharmaceutical field, the application of *P. notoginseng* is expected to see a linear growth in the cosmetic and health-supplement industries according to the "*Global Notoginseng Root Extract Market report*".[9] The use of its extracts in oral care has been recognized by the market,[10] and several recent studies have suggested its potential benefits for hair growth.[11–13] However, the reasons behind the benefits of the *Panax notoginseng* extract on oral health and hair remain unclear. Specifically, the components responsible for these daily functions and their mechanisms of action still need to be investigated. To address these scientific questions, we combined phytochemistry and bioactivity screening in an attempt to elucidate the underlying substances.

Results and discussion

Isolation and structure elucidation of compounds

The phytochemical investigation of the roots of *P. notoginseng* led to the isolation of 48 compounds. Herein, we describe their structure elucidation. Compound **1** (Fig. 1) was obtained as a white powder; its molecular formula was determined as $C_{42}H_{70}O_{14}$ based on its HRESIMS m/z = 821.4657 $[M + Na]^+$ (calcd. for 821.4658, $C_{42}H_{70}O_{14}Na$), indicating 8 degrees of unsaturation. The ^{1}H NMR spectrum revealed seven methyl singlets at δ_H 0.93 (3H, s), 0.95 (3H, s), 1.02 (3H, s), 1.09 (3H, s), 1.37 (3H, s), 1.62 (3H, s) and 1.68 (3H, s) and 2 sugar anomeric protons at δ_H 4.59 (1H, d, J = 7.7 Hz) and δ_H 4.35 (1H, d, J = 7.8 Hz) (Table 1). The ^{13}C NMR and DEPT data of **1** (Table 2), in conjunction with the MS data, suggested that **1** possessed 42 carbons, including 11 methylenes, 18 methines, and six quaternary carbons. The presence of two glucose units was supported by two anomeric carbons [δ_C 105.0 (d) and 98.1 (d)] and two methylenes [δ_C 70.2 and 62.8]. The HMBC correlations of δ_H 4.59 (1H, d, J = 7.7 Hz, H-1′) with δ_C 85.0 (C-20) and of δ_H 4.35 (1H, d, J = 7.8 Hz, H-1″) with δ_C 70.2 (C-6′) indicated a 1→6 linkage at C-20. The comparison of the mass spectrum and NMR spectroscopic data of the compound **1** with those of notoginsenoside U revealed high structural similarities. The major difference was the presence of a methylene group (δ_C 84.1) in Compound **1**, replacing a methyl group at δ_C 32.1 (C-28)

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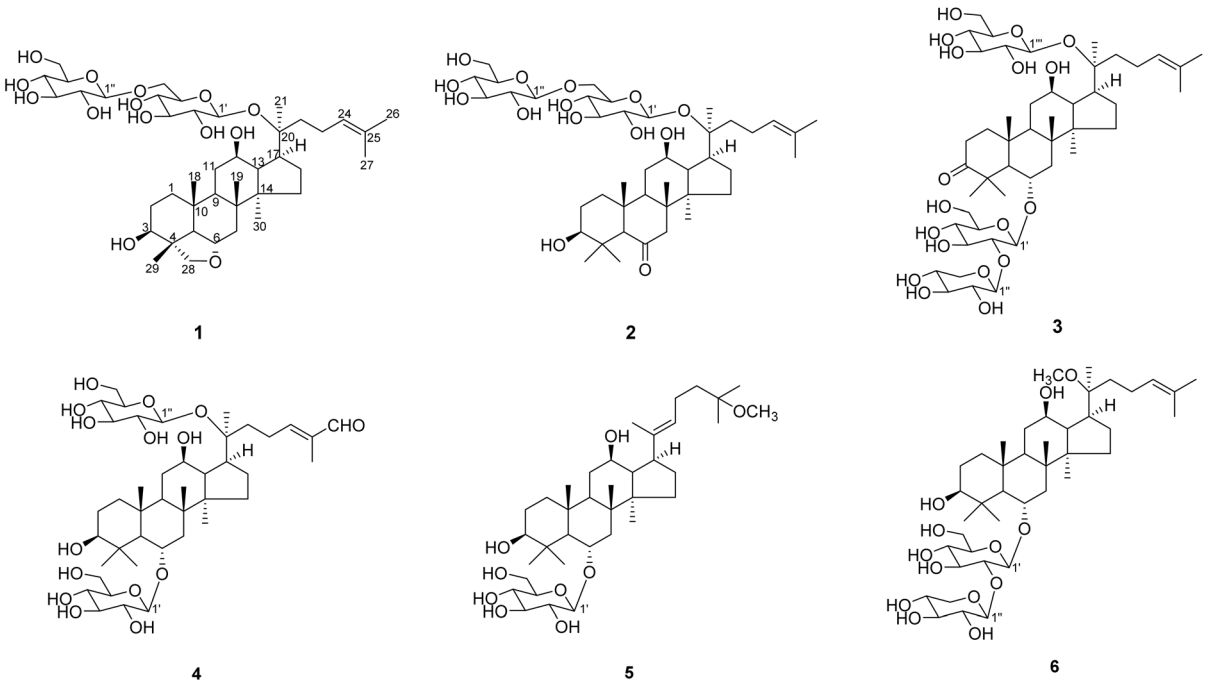

Fig. 1 Structures of the new compounds **1**–**6**.

in notoginsenoside U.[14] The ^{1}H–^{1}H correlation between δ_H 4.00 (H-6) and δ_H 0.89 (H-5), as well as with δ_H 1.42 and 1.78 (H-7), established the H-5/H-6/H-7 fragment. In the HMBC spectrum (Fig. 2), correlations from δ_H 3.54 and 3.72 (H-28) to δ_C 46.2 (C-4), δ_C 61.2 (C-5), and δ_C 74.1 (C-6) indicated long-range coupling, suggesting spatial proximity. These data imply that the methyl group at C-28 and the hydroxyl group at the C-6 position may have cyclized to form a new furan ring. This was further supported by the additional degree of unsaturation of compound **1** and confirmed by HMBC correlations.

The relative configuration of **1** was established by the ROESY spectrum (Fig. 2). The NOE correlations of H-5 with H-3/H-9, H-9 with H_3-30, and H-17 with H-12/H_3-30/H-1′ indicated that H-3, H-5, H-9, H-12, H-17, H-1′, and Me-30 were co-oriented (α-configuration). The β-orientation of H-6 was assigned based on the NOE correlations of H-6 with H_3-19/H_3-18/H_3-29. The relative configurations of sugar units were determined as β according to the coupling constant ($J = 7.6$, 7.8 Hz). The sugar unit was assigned as β-D-glucopyranose based on its ^{1}H and ^{13}C NMR data (Tables 1 and 2) and comparison with other dammarane triterpenoid saponins from this plant. The absolute configuration at C-20 was assigned by the comparison of chemical shift differences with those of known (20*S*)- and (20*R*)-ginsenosides.[15] For C-17, C-21, and C-22, the differences ($\delta_S - \delta_R$) were approximately +4.1 ± 0.1, +4.3 ± 0.1, and −7.4 ± 0.1 ppm, respectively. The chemical shifts of compound **1** at C-17 (δ_C 52.8), C-21 (δ_C 22.4), and C-22 (δ_C 36.8) were consistent with those of notoginsenoside U,[14] supporting the 20S configuration. The consistency between the calculated and experimental ECD spectra of **1** (Fig. 3) further confirmed the absolute configuration of compound **1**, as shown in Fig. 1. Through the detailed analysis of the 2D NMR data, the structure of compound **1** was finally determined and named notoginsenoside V1 (Fig. 1).

The molecular formula ($C_{42}H_{70}O_{14}$) of compound **2** was determined from the molecular ion peak at *m*/*z* 797.4695 $[M - H]^-$ (calcd. For 797.4693, $C_{42}H_{69}O_{14}$) and supported by the ^{13}C NMR spectroscopic data (Table 2). Two terminal carbon signals at δ_C 98.1 and δ_C 105.0, along with the two methylene signals at δ_C 62.8 and δ_C 70.2, indicated the presence of two glucose units. The HMBC cross-peaks from δ_H 4.59 (1H, d, $J = 7.9$ Hz, H-1′) to δ_C 84.8 (C-20) and from δ_H 4.35 (1H, d, $J = 7.8$ Hz, H-1″) to δ_H 70.2 (C-6′) confirmed a (1→6) linkage between the two glucose units, with the inner glucose attached to the aglycone at C-20. The ^{1}H and ^{13}C NMR data (Tables 1 and 2) were similar to those of notoginsenoside U,[14] except that a carbonyl signal (δ_C 215.0) in **2** replaced the methylene signal (δ_C 67.4) in notoginsenoside U. HMBC cross-peaks of δ_H 2.28 (H-5) and δ_H 1.76, 2.70 (H-7) with δ_C 215.0 confirmed a ketone carbonyl group at C-6. The structure of compound **2** was established by the detailed analysis of the NMR spectra data and named notoginsenoside V2 (Fig. 1).

Compound **3** was obtained as a white powder, with the molecular formula of $C_{47}H_{78}O_{18}$, requiring 9 degrees of unsaturation, as established by HRESIMS at *m*/*z* 953.5086 $[M + Na]^+$ (calcd. for 953.5080, $C_{47}H_{78}O_{18}Na$). The ^{1}H NMR spectrum of **3** showed three anomeric protons at δ_H 4.47 (1H, d, $J = 7.3$ Hz), 4.60 (1H, d, $J = 7.8$ Hz), and 4.88 (1H, d, $J = 7.4$ Hz), indicating that all the sugar units were β-glucopyranosyl. Three terminal carbon signals at δ_C 98.3, 103.6, and 103.9, along with the three methylene signals at δ_C 63.0, 63.5, and 66.8, in the ^{13}C-NMR spectrum indicated the presence of two glucose units and one xylose unit. The sugar chain, xylosyl (1-O-2) glucosyl O-, and glycosidic site at C-6 were confirmed by the HMBC correlations

Table 1 ^{1}H (500 MHz) NMR spectroscopic data for the compounds **1–6** in methanol-d_4 (δ in ppm and J in Hz)

No.	1	2	3	4	5	6
1	1.09, m	1.33, m	1.77, m	1.05, m	1.05, m	1.03, m
	1.73, m	1.81, m	1.84, m	1.73, m	1.72, m	1.73, m
2	1.66, m	1.60, m	2.17, m	1.59, m	1.58, m	1.57, m
				1.64, m	1.63, m	1.63, m
3	3.46, dd (10.5, 5.0)	3.10, overlap		3.10, overlap	3.09, dd (11.8, 4.6)	3.08, overlap
5	0.89, d (11.7)	2.28, s	1.93, d (10.7)	1.11, d (10.5)	1.10, overlap	1.09, d (10.5)
6	4.00, m		3.97, td (10.7, 3.8)	4.09, td (10.7, 2.6)	4.09, td (10.5, 3.2)	4.06, m
	1.42, m	1.76, overlap	1.66, m	1.64, m	1.63, m	1.65, m
7	1.78, m	2.70, d (11.6)	2.14, m	2.05, m	2.04, m	1.99, m
9	1.46, m	2.08, m	1.64, m	1.48, m	1.47, m	1.45, m
	1.32, m	1.00, m		1.18, m	1.15, m	
11	1.74, m	1.54, m	1.80, m	1.84, m	1.77, m	1.84, m
12	3.74, m	3.82, m	3.27, m	3.73, m	3.60, m	3.28, m
13	1.75, m	1.75, m	1.75, m	1.73, m	1.77, m	1.67, m
15	1.10, m	1.31, m	1.19, m	1.13, m	1.15, m	1.14, m
	1.58, m	1.89, m	1.58, m	1.59, m	1.66, m	1.57, m
16	1.33, m	1.35, m	1.38, m	1.37, m	1.44, m	1.30, m
	1.89, m	1.89, m	1.93, m	1.94, m	1.85, m	1.84, m
17	2.32, m	2.32, m	2.31, m	2.33, m	2.57, m	2.36, m
18	0.93, s	0.91, s	0.79, s	0.99, s	0.98, s	0.97, s
19	1.02, s	0.97, s	1.04, s	1.09, s	1.12, s	1.06, s
21	1.37, s	1.36, s	3.34, s	1.40, s	1.61, s	1.21, s
	1.53, m	1.54, m	1.61, m	1.71, m		1.44, m
22	1.79, m	1.81, m	1.80, m	2.04, m	5.28, td (7.2, 1.5)	1.58, m
23	2.04, m	2.03, m		2.47, m		1.99, m
	2.13, m	2.14, m	2.06, m	2.55, m	1.99, m	2.09, m
24	5.13, t (7.1)	5.12, t (7.1)	5.10, t (6.9)	6.65, t (6.7)	1.47, overlap	5.11, t (7.1)
26	1.68, s	1.67, s	1.67, s	9.36, s	1.14, s	1.67, s
27	1.62, s	1.62, s	1.61, s	1.74, s	1.14, s	1.60, s
28	3.54, d (7.6)	0.99, s	1.37, s	1.32, s	1.32, s	1.31, s
	3.72, d (7.6)					
29	1.09, s	1.16, s	1.33, s	1.00, s	0.99, s	0.98, s
30	0.95, s	1.04, s	0.98, s	0.96, s	0.93, s	0.95, s
1′	4.59, d (7.7)	4.59, d (7.9)	4.47, d (7.3)	4.35, d (7.8)	4.34, d (7.8)	4.43, d (7.1)
2′	3.11, m	3.12, m	3.60, m	3.20, m	3.20, m	3.61, m
3′	3.34, m	3.20, m	3.61, m	3.34, m	3.32, m	3.61, m
4′	3.27, m	3.26, m	3.41, m	3.25, m	3.27, m	3.55, m
5′	3.42, m	3.43, m	3.26, m	3.26, m	3.26, m	3.25, m
	3.79, dd (11.7, 5.6)	3.79, overlap	3.68, overlap	3.79, d (5.6)	3.64, overlap	3.64, overlap
6′	4.05, dd (11.7, 2.1)	4.05, dd (11.6, 2.0)	3.76, overlap	3.82, dd (5.1, 1.9)	3.81, dd (11.7, 1.8)	3.80, overlap
1″	4.35, d (7.8)	4.35, d (7.8)	4.88, d (7.4)			4.87, m
2″	3.20, m	3.20, m	3.07, m			3.24, m
3″	3.25, m	3.34, m	3.34, m			3.25, m
4″	3.33, m	3.34, m	3.32, m			3.45, m
5″	3.25, m	3.26, m	3.09, m			3.10, m
			3.75, m			3.80, m
6″	3.64, dd (11.9, 5.1)	3.64, overlap				
	3.85, dd (11.9, 1.4)	3.85, overlap				
1‴			4.60, d (7.8)			
2‴			3.22, m			
3‴			3.34, m			
4‴			3.67, m			
5‴			3.20, m			
6‴			3.63, overlap			
			3.80, overlap			
OCH_3					3.17, s	3.27, s

observed between glucosyl H-1' (δ_H 4.47) and C-6 (δ_C 80.4) of the aglycone and between xylosyl H-1'' (δ_H 4.88) and glucosyl C-2′ (δ_C 78.8). An additional HMBC correlation from the terminal glucosyl, H-1‴ (δ_H 4.60), to C-20 (δ_C 84.9) of the aglycone confirmed that another sugar moiety was attached at C-20 of the aglycone. The ^{1}H NMR and ^{13}C NMR data (Tables 1 and 2) suggested that **3** was similar to notoginsenosides-R1,[16] except that a carbonyl group (δ_C 233.0) in **3** replaced a methine group in

Table 2 ^{13}C (125 MHz) NMR spectroscopic data for the compounds **1–6** in methanol-d_4 (δ in ppm)

No.	1	2	3	4	5	6
1	42.3 t	40.9 t	40.8 t	40.2 t	40.3 t	40.2 t
2	29.2 t	27.7 t	33.7 t	27.6 t	27.6 t	27.5 t
3	78.5 d	79.0 d	223.0 s	79.9 d	79.8 d	80.1 d
4	46.2 s	38.7 s	49.1 s	40.4 s	40.5 s	40.5 s
5	61.2 d	66.4 d	58.7 d	61.8 d	61.8 d	61.9 d
6	74.1 d	215.0 s	80.4 d	80.9 d	80.9 d	80.8 d
7	41.7 t	53.4 t	43.4 t	45.3 t	45.5 t	45.2 t
8	43.7 s	47.6 s	41.1 s	41.9 s	42.1 s	41.9 s
9	51.1 d	51.0 d	49.1 d	50.6 d	51.4 d	50.6 d
10	36.8 s	44.8 s	39.4 s	40.5 s	40.5 s	40.4 s
11	31.0 t	31.4 t	31.8 t	30.9 t	32.4 t	31.1 t
12	71.3 d	71.1 d	71.7 d	71.7 d	74.0 d	71.7 d
13	49.2 d	49.5 d	49.4 d	49.6 d	51.0 d	49.1 d
14	52.3 s	52.2 s	52.6 s	52.4 s	51.9 s	52.5 s
15	31.6 t	31.1 t	31.6 t	31.5 t	33.3 t	31.7 t
16	26.9 t	26.9 t	27.2 t	27.3 t	29.4 t	27.0 t
17	52.8 d	53.0 d	53.0 d	53.0 d	51.3 d	48.2 d
18	16.3 q	17.6 q	18.4 q	17.8 q	17.9 q	17.8 q
19	16.7 q	16.3 q	16.2 q	17.7 q	17.5 q	17.7 q
20	85.0 s	84.8 s	84.9 s	84.5 s	140.3 s	81.1 s
21	22.4 q	22.4 q	22.8 q	22.4 q	12.8 q	21.5 q
22	36.8 t	36.7 t	36.7 t	34.8 t	125.7 d	36.0 t
23	23.8 t	23.8 t	24.2 t	25.2 t	23.5 t	23.4 t
24	126.0 d	126.0 d	125.8 d	157.4 d	40.1 t	125.7 d
25	132.3 s	132.3 s	132.3 s	140.3 s	76.2 s	132.4 s
26	25.9 q	25.9 q	25.9 q	197.3 q	25.3 q	25.9 q
27	18.0 q	18.0 q	17.9 q	9.2 q	25.3 q	17.8 q
28	84.1 t	27.9 q	32.3 q	31.4 q	31.4 q	31.4 q
29	14.8 q	16.0 q	20.1 q	16.1 q	16.1 q	16.6 q
30	17.6 q	17.5 q	17.0 q	17.2 q	17.0 q	17.1 q
1′	98.1 d	98.1 d	103.6 d	105.6 d	105.5 d	103.9 d
2′	75.3 d	75.3 d	78.8 d	75.5 d	75.5 d	79.2 d
3′	78.5 d	77.9 d	79.8 d	79.1 d	79.1 d	79.7 d
4′	71.7 d	71.7 d	71.2 d	71.7 d	71.7 d	71.8 d
5′	76.8 d	76.8 d	77.5 d	78.0 d	77.7 d	77.6 d
6′	70.2 t	70.2 t	62.5 t	62.9 t	62.9 t	62.9 t
1″	105.0 d	105.0 d	103.9 d			103.8 d
2″	75.1 d	75.1 d	75.4 d			75.6 d
3″	77.9 d	78.5 d	78.2 d			78.3 d
4″	71.5 d	71.5 d	71.3 d			71.3 d
5″	77.9 d	77.9 d	66.8 t			66.9 t
6″	62.8 t	62.8 t				
1‴			98.3 d			
2‴			75.5 d			
3‴			78.3 d			
4‴			71.8 d			
5‴			77.9 d			
6‴			62.9 t			
OCH_3					49.7 q	49.4 q

notoginsenoside R1. The HMBC cross-peaks of δ_H 1.33 (H-29) with δ_C 223.0, 49.1 (C-4), and 58.7 (C-5), as well as the cross-peaks of δ_H 1.37 (H-28) with δ_C 223.0, 49.1 (C-4), and 58.7 (C-5), indicated the carbonyl at C-3. The structure of compound **3** was established by the detailed NMR analysis and named notoginsenoside V3 (Fig. 1).

Compound **4** had the molecular formula $C_{42}H_{70}O_{15}$, determined based on the molecular ion peak at *m/z* 837.4601 $[M + Na]^+$ in the HRESIMS data and the ^{13}C NMR spectroscopic data

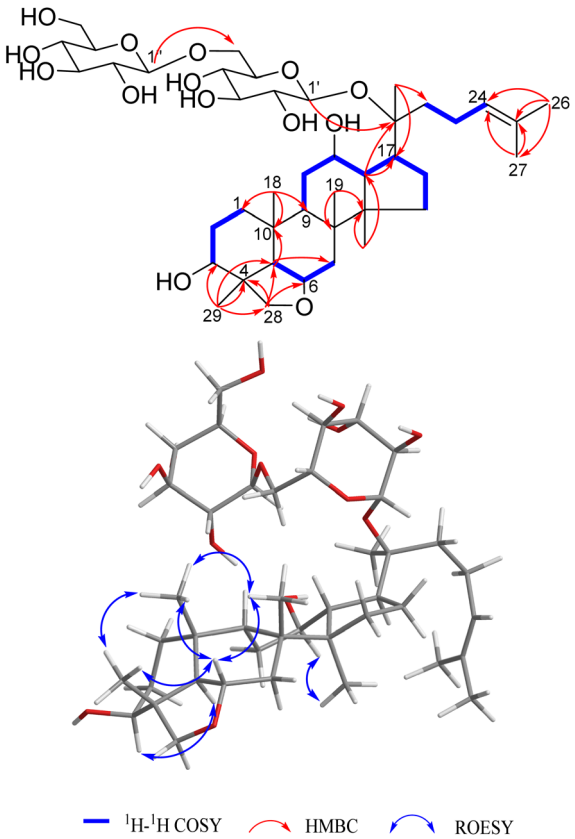

Fig. 2 Key HMBC, ^{1}H–^{1}H COSY, and ROESY correlations of the compound **1**.

(Table 2). Two terminal carbon signals at δ_C 98.2 and δ_C 105.6, along with the two methylene signals at δ_C 62.8 and δ_C 62.9, in the ^{13}C-NMR spectrum indicated the presence of two glucose units. The HMBC correlations of δ_H 4.35 (1H, d, $J = 7.8$ Hz, H-1′) with δ_C 80.9 (C-6) and of 4.63 (1H, d, $J = 7.8$ Hz, H-1″) with δ_C 84.5 (C-20) established that the two glucose units were attached to the aglycone at C-6 and C-20, respectively. The ^{1}H and ^{13}C NMR data of **4** were similar to those of ginsenoside Rg1,[17] except for the absence of one methine signal and the presence of a downfield methine signal (δ_C 197.3) in **4**. The HMBC cross-peaks from δ_H 9.36 (δ_C 197.3) with δ_C 157.4 (C-24) and δ_C 9.2 (C-27) and from δ_H 1.74 (H-27) to δ_C 157.4 (C-24) and δ_C 140.3 (C-25) indicated the oxidation of the methyl group at C-26 to an aldehyde group (δ 9.36, 197.3). The structure of compound **4** was confirmed by the detailed analysis of the NMR data and named notoginsenoside V4 (Fig. 1).

Compound **5** was obtained as a white powder. Its molecular formula was deduced as $C_{37}H_{64}O_9$ based on a HRESIMS ion peak at *m/z* 697.4533 $[M + HCOO]^-$ (calcd. for 697.4532, $C_{38}H_{65}O_{11}$). The ^{1}H NMR spectrum of **5** (Table 1) displayed eight methyl singlets at δ_H 0.93 (3H, s), 0.98 (3H, s), 0.99 (3H, s), 1.12 (3H, s), 1.14 (3H, overlap), 1.14 (3H, overlap), 1.32 (3H, s), 1.61 (3H, s); a methoxy signal at δ_H 3.17 (3H, s); a sugar anomeric proton signal at δ_H 4.34 (1H, d, $J = 7.8$ Hz); and an olefinic proton signal at δ_H 5.28 (1H, td, $J = 7.2$, 1.5 Hz). These results suggested **5** to be a dammarane-type saponin. The attachment

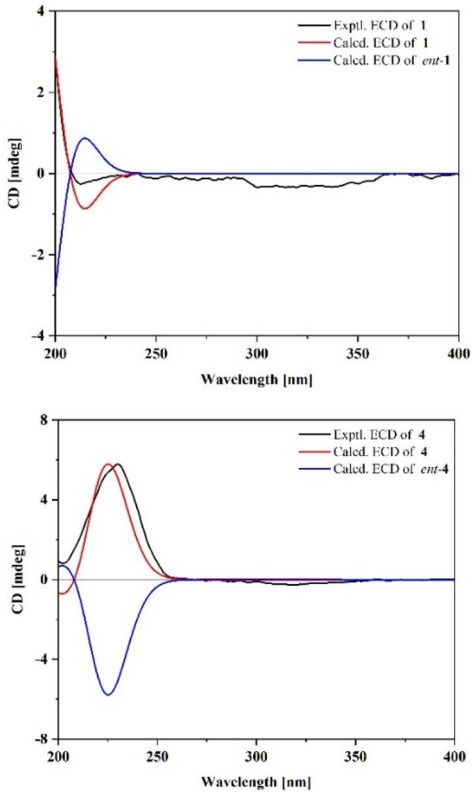

Fig. 3 Experimental and calculated ECD spectra of the compounds **1** and **4**.

of the sugar at C-6 was confirmed by the key HMBC correlation signals from δ_H 4.34 (1H, d, J = 7.8 Hz) to δ_C 80.9 (C-6). The location of the double bond at C-20/C-22 was established through the COSY correlations of H-23/H-22 and H-24, as well as key HMBC correlations from H-21 to C-17, C-20, and C-22. The NMR data (Tables 1 and 2) of **5** were similar to those of notoginsenoside ST13,[18] except for an additional methoxy signal (δ 3.17, 49.7) in compound **5**. The position of the methoxy group in C-25 was determined by the HMBC correlations of δ_H 3.17 (25-OCH_3) with δ_C 76.2 (C-25). The double bond was assigned at *E*-configuration based on the chemical shift of C-21 (δ_C 12.8), which was consistent with the E form (δ_C 12.5 for C-21 in sanchinoside B1 [19]) rather than the Z form (δ_C 19.9 (C-21) in notoginsenoside ST13 [18]). The analysis of the 2D NMR data confirmed the remainder of the structure. The structure of **5** was elucidated as shown in Fig. 1 and named V5 (Fig. 1).

Compound **6** was obtained as a white powder with the molecular formula of $C_{42}H_{72}O_{13}$, requiring 7 degrees of unsaturation, as established by HRESIMS at 783.4903 ($[M - H]^-$, calcd. for $C_{42}H_{71}O_{13}$ 783.4900). Two terminal carbon signals at δ_C 103.8 and δ_C 103.9, along with the three methylene signals at δ_C 62.9 and δ_C 66.9, in the ^{13}C-NMR spectrum indicated the presence of one glucose unit and one xylose unit. The HMBC correlations observed between glucosyl H-1' (δ_H 4.43) and C-6 (δ_C 80.8) of the aglycone and between xylosyl H-1'' (δ_H 4.87) and glucosyl C-2' (δ_C 79.2) confirmed a xylosyl-(1→2)-glucosyl chain attached at C-6. The ^{1}H NMR and ^{13}C NMR data (Tables 1 and 2) suggested that **6** was similar to notoginsenoside R2, except for the presence of an additional methoxy signal (δ 3.27, 49.4) in **6**. The HMBC correlations of H-21 (δ_H 1.21) with C-17 (δ_C 48.2), C-20 (δ_C 81.1), and C-22 (δ_C 26.0), as well as OCH_3 (δ_H 3.27) with C-20 (δ_C 81.1), confirmed the connection of –OCH_3 to the C-20 position. Therefore, the structure of **6** was elucidated as shown in Fig. 1 and named V6 (Fig. 1).

Other compounds were identified as ginsenoside Rb1 (**7**),[20] pseudoginsenoside RC1 (**8**),[21] quinquenoside III (**9**),[22] ginsenoside Rd (**10**),[20] ginsenoside K (**11**),[23] ginsenoside Rh2 (**12**),[24] majoroside F4 (**13**),[25] 3-*O*-[β-D-glucopyranosyl(1→2)-β-D-glucopyranosyl]-3β, 12β, 20(S), 25-tetra-hydroxydammar-23-ene (**14**),[26] notoginsenoside SY3 (**15**),[27] ginsenoside Rh7 (**16**),[28] 20(S)-sanchirhinoside A8 (**17**),[29] ginsenoside Rg2 (**18**),[30] ginsenoside Rg1 (**19**),[31] notoginsenoside R2 (**20**),[16] 20(S)-sanchirhinoside A3 (**21**),[32] 20(S)-sanchirhinoside A4 (**22**),[32] ginsenoside Rh1 (**23**),[33] ginsenoside Rh5 (**24**),[34] ginsenoside Rf (**25**),[30] notoginsenoside R1 (**26**),[16] 20(S)-sanchirhinoside A1 (27),[32] ginsenoside Rh1 6′-acetate (**28**),[35] 3-oxo-ginsenoside Rh1 (**29**),[36] ginsenoside F1 (**30**),[37] notoginsenoside R3 (**31**),[38] notoginsenoside R6 (**32**),[38] 20-glucoginsenoside Rf (**33**),[39] ginsenoside Re_3 (**34**),[40] dihydroginsenoside Rg1 (**35**),[41] 20(R)-ginsenoside Rh1 (**36**),[42,43] 20(*R*)-ginsenoside Rh_5 (**37**)[34] (3β,6α,12β,20S,23E)-3,12,20,25-tetrahydroxydammar-23-en-6-yl-2-*O*-β-D-xylopyranosyl-β-D-glucopyranoside (**38**),[26] ginsenoside Rk3 (**39**),[44] notoginsenoside T5 (**40**),[45] ginsenoside Rh_4 (**41**),[46] notoginsenoside ST8 (**42**),[18], (2*S*)-1-*O*-(9*Z*,12*Z*-octadecadienoyl)-3-*O*-β-galactopyranosylglycerol (**43**),[47] panaxydol (**44**),[48] panaxytriol (**45**),[49] ginsenoyne C (**46**),[50] 6,7-dihydroxytetradeca-1,3-diyne (**47**),[51] and panaxyne (**48**).[52]

Cyclooxygenase inhibitory activities. The preliminary screening of the inhibitory activities of the isolated compounds against COX-2 cyclooxygenase at a concentration of 50 μM revealed that **43** exhibited better inhibitory activity compared to other compounds. The EC_{50} value of 5.94 μM for **43** was calculated from the concentration-inhibition response curve (Fig. 4), as compared with that of a positive control, celecoxib, with an IC_{50} value of 0.54 μM.

The compounds promote the proliferation of hOMFs. To evaluate the effects of the isolated compounds on oral ulcer, human oral mucosa fibroblasts were used. Compounds **7**, **12**, **13**, **14**, **15**, **16**, **18**, **19**, **25**, **28**, **29**, **30**, **35**, **39**, **43**, **45** promoted the proliferation of fibroblasts in human oral mucosa at a concentration of 50 μM over 48 hours, compared to the control. A concentration-dependent relationship was further observed for compounds **7**, **14**, **15**, **16**, **28**, **29**, **30**, **39**, **43**, and **45**, suggesting

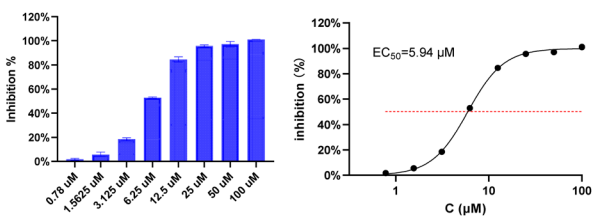

Fig. 4 Dose-effect relationship plots of the compound **43** in inhibiting cyclooxygenase-2.

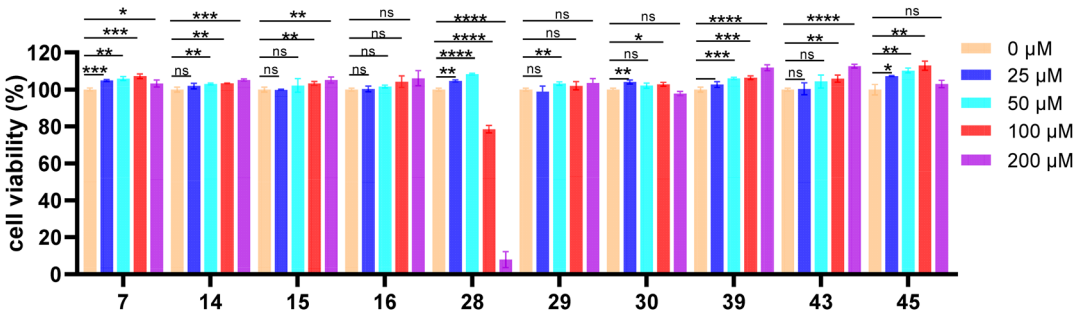

Fig. 5 Proliferation evaluation of the compounds **7**, **14**, **15**, **16**, **28**, **29**, **30**, **39**, **43**, and **45** on hOMF cells at multiple concentrations. The significance was determined by a Student's test (*p < 0.05, **p < 0.01, and ***p < 0.001 *vs.* the control group).

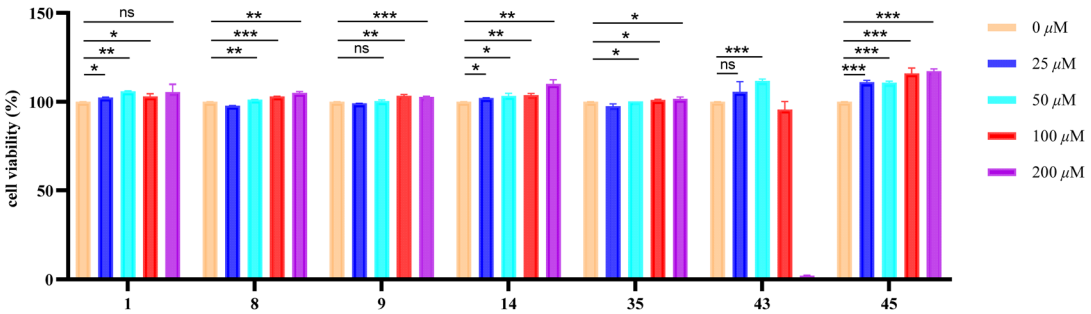

Fig. 6 Proliferation evaluation of the compounds **1**, **8**, **9**, **14**, **35**, **43**, and **45** on hDPCs at multiple concentrations. The significance was determined by a Student's test (*p < 0.05, **p < 0.01, and ***p < 0.001 *vs.* the control group).

a potential beneficial effect on the healing of oral soft tissue defects (Fig. 5).

The compounds promote proliferation of the hDPCs. To investigate the effects of isolated compounds on the viability of human dermal papilla cells (hDPCs), an MTS-reducing assay was performed for all compounds. Compounds **1**, **8**, **9**, **14**, **21**, **35**, **43**, and **45** were able to promote the proliferation of hDPCs at a concentration of 50 μM over 48 hours compared to the control. A concentration-dependent relationship was further observed for these compounds, suggesting a potential hair growth-promoting effect (Fig. 6).

Conclusions

Recent studies have shown that scaffolds containing notoginsenoside can effectively alleviate the local hypoxia state in diabetic mice after implantation, promote angiogenesis, and thereby accelerate wound closure. It can also reduce the production of pro-inflammatory cytokines and inhibit kidney inflammation by increasing the abundance of specific beneficial bacterial populations. This suggests that it may play a broad-spectrum role in the prevention and treatment of chronic inflammatory diseases.[53] Total notoginsenoside can significantly reduce the volume of cerebral infarction and alleviate the necrosis of hippocampal neurons.[54] In the present study, 48 compounds were identified from the roots of *Panax notoginseng*. These compounds were evaluated for their bioactivities, including anti-inflammatory effects and the proliferation of human oral mucosal fibroblasts (hOMFs) and human dental pulp cells (hDPCs). A wealth of bioactive compounds with the potential to promote the proliferation of hOMFs and hDPCs was revealed. This research not only expands the understanding of the chemical constituents of *Panax notoginseng*, but also reveals its diverse biological activities. Compound **43**, (2*S*)-1-*O*-(9*Z*,12*Z*-octadecadienoyl)-3-*O*-β-galactopyranosylglycerol, exhibited significant anti-inflammatory activity and promoted the proliferation of hOMFs, distinguishing it from the saponins. Notably, the saponins promoted the proliferation of human hair follicle cells, as well as oral mucosa fibroblasts. These findings provide a scientific basis for the improved development and utilization of *Panax notoginseng*, particularly in the fields of oral health and hair growth. Of course, the absence of mechanistic studies or more advanced models, such as molecular pathway analyses, differentiation markers, or *in vivo* models, significantly weakens the biological impact of the work. In the future, we will attempt to conduct *in vivo* tests to verify its functionality and explore its mechanism of action.

Experimental section

General experimental procedures

Optical rotations were measured with a Horiba SEPA-300 polarimeter. UV spectra were recorded on a Shimadzu UV-2700 spectrophotometer. 1D and 2D NMR spectra were obtained on AVANCE 800, 600, 500, and 400 spectrometers (Bruker

BioSpin GmbH, Rheinstetten, Germany) with tetramethylsilane (TMS) as an internal standard. HRESIMS data were recorded on a Shimadzu UPLC-IT-TOF. Column chromatography (CC) was performed on either silica gel (200–300 mesh, Qingdao Marine Chemical Co., Ltd., Qingdao, China) or RP-18 silica gel (20–45 μm, Fuji Silysia Chemical Ltd., Japan). Fractions were monitored by TLC on silica gel plates (GF254, Qingdao Haiyang Chemical Co., Ltd., Qingdao, China), and spots were visualized with Dragendorff's reagent. Medium-pressure liquid chromatography (MPLC) was performed using a BUCHI pump system coupled with RP-18 silica gel-packed glass columns. High-performance liquid chromatography (HPLC) was performed using Waters 1525 EF pumps (Waters Corp., Milford, MA, USA) coupled with analytical and preparative Xbridge C_{18} columns (4.6 × 150 and 19 × 250 mm, respectively). The HPLC system employed a Waters 2998 photodiode array detector and a Waters fraction collector III.

Plant material

The roots of *Panax notoginseng* (Burk.) F. H. Chen were supplied by Infinitus (China) Co., Ltd. in October 2022 and was identified by Professor Peng Guangtian of the Guangzhou University of Traditional Chinese Medicine. The voucher specimen (SQ20201012) was deposited in Infinitus (China) Co., Ltd.

Extraction and isolation

The air-dried roots of *P. notoginseng* (6 kg) were powdered and extracted with 70% ethanol water, and the solvent was evaporated under a vacuum to obtain an extract. The extract was applied to a macroporous adsorption resin, eluted with water, and then eluted with 80% ethanol water to obtain 930 g of fraction. This fraction was subjected to column chromatography (CC) over silica gel and eluted with gradient $CHCl_3$–MeOH (4 : 1, 3 : 1, 2 : 1, and 1 : 1, v/v) to afford fifteen fractions (Frs. A–O).

Fraction Fr. B (3 g) was separated by reversed-phase medium-pressure liquid chromatography (MPLC) eluted with a gradient of MeOH–H_2O (30 : 80–100 : 0, v/v) to give the nine sub-fractions (Frs. B-1–B-8). Fr. B-5 was separated using a Sephadex LH-20 column, eluting with methanol, and further fractionated using a preparative Xbridge C_{18} HPLC column with a gradient of MeCN (45 : 55–60 : 40, v/v) in H_2O to get **46** (20.6 mg, Rt = 24.1 min) and **48** (20.6 mg, Rt = 35.4 min). Fr. B-6 was chromatographed on Sephadex LH-20 (MeOH) and further purified on the HPLC preparative column with a gradient of MeCN (50 : 50–65 : 35, v/v) in H_2O to obtain **47** (3.0 mg, Rt = 27.3 min). Fr. B-9 was subjected to a Sephadex LH-20 column using MeOH as the eluent and further fractionated using a preparative Xbridge C_{18} HPLC column with a gradient of MeCN (65 : 35–80 : 20, v/v) in H_2O to get **44** (41.7 mg, Rt = 35.8 min) and **45** (12.7 mg, Rt = 14.8 min).

Fraction D (3 g) was fractionated using a C_{18} MPLC column, eluting with a gradient of MeOH–H_2O (35 : 65–90 : 10, v/v), to obtain Fr. D-1 to Fr. D-13. Fr. D-1 was subjected to a Sephadex LH-20 column using MeOH as the eluent and further fractionated using a preparative Xbridge C_{18} HPLC column with a gradient of MeCN (60 : 40–75 : 25, v/v) in H_2O to yield **43** (3.7 mg, Rt = 42.0 min). Fr. D-7 (0.8 g) was loaded to a Sephadex LH-20 column, eluting with MeOH, and further separated by RP-C18 preparative HPLC, eluting with gradients of MeCN–H_2O (35 : 65–50 : 50, v/v), to afford **16** (3.8 mg, Rt = 23.5 min), **29** (3.8 mg, Rt = 27.1 min) and **30** (54.4 mg, Rt = 28.3 min). Fr. D-8 was further purified on the HPLC preparative column with a gradient of MeCN (30 : 60–45 : 55, v/v) in H_2O to obtain **28** (116.1 mg, Rt = 36.3 min). Fr. D-9 was further purified on a preparative Xbridge C_{18} HPLC column with a gradient of MeCN (45 : 55–60 : 40, v/v) in H_2O to get **17** (60.6 mg, Rt = 23.1 min) and **27** (4.5 mg, Rt = 25.7 min). Fr. D-13 was separated using a Sephadex LH-20 column, eluting with methanol, and further fractionated using a preparative Xbridge C_{18} HPLC column with a gradient of MeCN (55 : 45–75 : 25, v/v) in H_2O to get **11** (60.0 mg, Rt = 29.9 min) and **12** (4.5 mg, Rt = 34.3 min).

Fr. H (20 g) was chromatographed on a C_{18} MPLC column and eluted with a gradient of MeOH–H_2O (10 : 90–70 : 30, v/v) to give ten subfractions (Frs. H-1–H-10). Compound **18** (43.0 g, Rt = 47.3 min) was crystallized from fraction Fr. H-6. Fr. H-8 underwent elution using a Sephadex LH-20 column with methanol and was further purified using a preparative C_{18} HPLC on a Xbridge column with a gradient of H_2O (20 : 80–40 : 60, v/v) in MeCN to afford **35** (16.5 mg, Rt = 36.2 min), **20** (399.6 mg, Rt = 43.9 min), **21** (6.9 mg, Rt = 27.7 min), and **22** (127.4 mg, Rt = 29.9 min). Fr. H-9 was refined by preparative C_{18} HPLC on a Xbridge column with a gradient of H_2O (30 : 70–45 : 55, v/v) in MeOH to furnish **1** (3.0 mg, Rt = 22.1 min), **2** (1.3 mg, Rt = 30.5 min), and **15** (3.7 mg, Rt = 24.7 min). **8** (4.3 mg, Rt = 30.3 min) and **9** (10.1 mg, Rt = 34.3 min) were obtained from Fr. H-10 using preparative HPLC with a gradient of MeCN–H_2O (35 : 65–55 : 40, v/v).

Fraction I (150.0 g) was chromatographed on a C_{18} MPLC column eluted with a gradient of MeOH–H_2O (20 : 80–100 : 0, v/v) to give nine subfractions (Frs. I-1–I-9). Fr. I-2 was subjected to a Sephadex LH-20 column, using MeOH as the eluent, and further fractionated using a preparative Xbridge C_{18} HPLC column with a gradient of MeOH (40 : 60–55 : 45, v/v) in H_2O to get **23** (90.8 mg, Rt = 14.1 min), **24** (43.5 mg, Rt = 29.9 min), **36** (6.1 mg, Rt = 15.4 min), and **39** (83.2 mg, Rt = 33.3 min). The most abundant compound, **19** (133.0 g), was crystallized from fraction Fr. I-4. Compound **41** (2.0 g) was crystallized from fraction Fr. I-5. Fr. I-7 was refined by preparative C_{18} HPLC on a Sunfire column with a gradient of H_2O (30 : 70–45 : 55, v/v) in MeCN to afford **25** (4.0 mg, Rt = 29.0 min), **13** (2.5 mg, Rt = 30.6 min), and **14** (5.9 mg, Rt = 34.1 min).

Fraction Fr. K (40 g) was chromatographed on a C_{18} MPLC column eluted with a gradient of MeOH–H_2O (35 : 65–85 : 15, v/v) to give twelve subfractions (Frs. K-1–K-12). Fr. K-2 was refined by preparative C_{18} HPLC on an Xbridge column with a gradient of H_2O (15 : 85–30 : 70, v/v) in MeCN to get **42** (1.1 mg, Rt = 21.7 min). Fr. K-7 was further purified by preparative C_{18} HPLC on a Xbridge column with a gradient of H_2O (35 : 65–50 : 50, v/v) in MeCN to obtain **5** (11.1 mg, Rt = 30.8 min), **6** (134.9 mg, Rt = 32.7 min), and **40** (117.9 mg, Rt = 39.6 min). Fr. K-12 was subjected to a Sephadex LH-20 column, using MeOH as the eluent, and further fractionated using a preparative Xbridge C_{18}

HPLC column with a gradient of MeCN (40 : 60–55 : 45, v/v) in H_2O to yield **37** (36.0 mg, Rt = 33.2 min).

Fraction Fr. J (40 g) was also chromatographed on a C_{18} MPLC column eluted with a gradient of MeOH–H_2O (30 : 70–80 : 20, v/v) to give four sub-fractions (Frs. J-1–J-4). Fr. J-3 was separated on Sephadex LH-20 (CH_3OH) to produce three sub-fractions (Frs. J-3-1–J-3-3). Compound **4** (15.3 mg, Rt = 25.4 min) was obtained from J-3-1 using preparative HPLC with a gradient of MeCN–H_2O (30 : 70–40 : 60, v/v). Through the same method as Fr. J-4, **3** (7.1 mg, Rt = 35.8 min) and **38** (9.1 mg, Rt = (t_R = 25.2 min) were obtained from Fr. J-4-2.

Fraction Fr. L (180 g) was fractionated by RP-18 CC and eluted using MeOH (30 : 70–85 : 15, v/v) in H_2O, affording six sub-fractions (Frs. L-1–L-6). Compounds **26** (42.0 g) and **10** (28.0 g) were crystallized from fraction Fr. L-3 and Fr. L-5, respectively.

Fraction Fr. M (87 g) was separated by reversed-phase medium-pressure liquid chromatography (MPLC), eluted with a gradient of MeOH–H_2O (25 : 75–80 : 20, v/v), to give four sub-fractions (Frs. M-1–M-4). Fr. M-2 was applied to a Sephadex LH-20 column, eluting with methanol, and was divided into two parts, Fr. M-2-1–M-2-3. Fr. M-2-1 was further purified by preparative C_{18} HPLC on a Sunfire column with a gradient of H_2O (20 : 80–30 : 70, v/v) in MeCN to obtain **31** (49.5 mg, Rt = 26.4 min), **32** (32.5 mg, Rt = 28.2 min), **33** (359.3 mg, Rt = 33.2 min), and **34** (15.3 mg, Rt = 28.2 min).

Compound **7** (114.0 g), one of the main ingredients, was crystallized from fraction Fr. N (130.0 g).

Notoginsenoside V1 (1). White powder; $[\alpha]_{20}^{D}$ = −25.78 (*c* 0.09, MeOH); UV (MeOH) λ_{max} (log ε) 203.0 (4.68), 274.5 (3.62) nm; ^{1}H and ^{13}C NMR data, see Tables 1 and 2; HRESIMS *m*/*z* 821.4657 $[M + Na]^+$ (calcd. for 821.4658, $C_{42}H_{70}O_{14}Na$).

Notoginsenoside V2 (2). White powder; $[\alpha]_{20}^{D}$ = +16.22 (*c* 0.09, MeOH); UV (MeOH) λ_{max} (log ε) 282.0 (3.91), 203.5 (4.76) nm; ^{1}H and ^{13}C NMR data, see Tables 1 and 2; HRESIMS *m*/*z* 797.4695 $[M - H]^-$ (calcd. for 797.4693, $C_{42}H_{69}O_{14}$).

Notoginsenoside V3 (3). White powder; $[\alpha]_{20}^{D}$ = +26.36 (*c* 0.11, MeOH); UV (MeOH) λ_{max} (log ε) 275.5 (3.47), 203.5 (4.66) nm; ^{1}H and ^{13}C NMR data, see Tables 1 and 2; HRESIMS *m*/*z* 953.5086 $[M + Na]^+$ (calcd. for 953.5080, $C_{47}H_{78}O_{18}Na$).

Notoginsenoside V4 (4). White powder; $[\alpha]_{20}^{D}$ = −19.00 (*c* 0.10, MeOH); UV (MeOH) λ_{max} (log ε) 196.5 (4.50), 228.0 (4.84), 277.0 (3.50) nm; ^{1}H and ^{13}C NMR data, see Tables 1 and 2; HRESIMS *m*/*z* 837.4601 $[M + Na]^+$ (calcd. for 837.4607, $C_{42}H_{70}O_{15}Na$).

Notoginsenoside V5 (5). White powder; $[\alpha]_{20}^{D}$ = −15.80 (*c* 0.10, MeOH); UV (MeOH) λ_{max} (log ε) 204.5 (4.74), 269.0 (3.24) nm; ^{1}H and ^{13}C NMR data, see Tables 1 and 2; HRESIMS *m*/*z* 697.4533 $[M + HCOO]^-$ (calcd. for 697.4532, $C_{38}H_{65}O_{11}$).

Notoginsenoside V6 (6). White powder; $[\alpha]_{20}^{D}$ = −15.57 (*c* 0.14, MeOH); UV (MeOH) λ_{max} (log ε) 203.0 (4.61), 278.5 (3.06) nm; ^{1}H and ^{13}C NMR data, see Tables 1 and 2; HRESIMS *m*/*z* 783.4903 $[M - H]^-$ (calcd. for 783.4900, $C_{42}H_{71}O_{13}$).

***In vitro* COX inhibitory assay.** The inhibitory activities of the compounds against COX-2 were determined using a colorimetric COX (ovine) inhibitor screening assay kit (Cayman, no. 760111) according to the manufacturer's instructions.[55] The 50% effective concentration (EC_{50}) values were calculated from the concentration-inhibition response curve. Celecoxib was used as a positive control for comparison.

Human oral mucosa fibroblasts proliferation assay. hOMFs (human oral mucosa fibroblasts), as primary cells, were purchased from Shanghai Jinyuan Biotechnology Co., Ltd. and cultured in Fibroblast Medium-2 (FM-2) in 5% CO_2 at 37 °C. We collected hOMFs in the logarithmic growth phase and digested them with trypsin to create a single-cell suspension, and then, they were seeded into 96-well tissue culture dishes with a density of 4×10^3 cells per well and cultured overnight. The cells were then incubated in a culture medium with each compound for 48 h. The MTS-reducing activity was evaluated by measuring the absorbance at 490 nm using the CellTiter 96 Aqueous One Solution Cell Proliferation Assay kit (Promega, USA) and an Infinite M200 Pro (Tecan, Austria) microplate reader.[56]

Human dermal papilla cells assay. Human dermal papilla cells (hDPCs), as primary cells, were purchased from Shanghai Jinyuan Biotechnology Co., Ltd. and cultured in a basal medium supplemented with 10% fetal bovine serum, 1% growth factor, and 1% penicillin/streptomycin (P/S) in 5% CO_2 at 37 °C.[57] Cells were maintained in a humidified incubator at 37 °C and in 5% CO_2. We collected hDPCs in the logarithmic growth phase and digested them with trypsin to create a single-cell suspension, and then, they were seeded into 96-well culture plates with a density of 5×10^3 cells/well and cultured overnight. Cells were then incubated in a culture medium with each compound for 48 h. The MTS-reducing activity was evaluated by measuring the absorbance at 490 nm using the CellTiter 96 Aqueous One Solution Cell Proliferation Assay kit (Promega, USA) and an Infinite M200 Pro (Tecan, Austria) microplate reader.[56]

Author contributions

L. X and Z. J. Q contributed equally to this work. L. X and Z. J. Q were responsible for the academic manuscript composition and data and phytochemical experiments. J. W, L. L. Y, L. L, and B. L were responsible for the activity experiments. M. F. B was responsible for the manuscript framework guidance and revisions, and X. H. C provided academic mentoring and experimental support. All authors accepted the final version of the manuscript.

Conflicts of interest

There are no conflicts to declare.

Data availability

The data used to support the findings of this study are available from the corresponding author upon request.

The data of ^{1}H and ^{13}C-NMR, HMBC, HSQC, COSY and ROESY spectra of the compounds **1–6** (PDF) in this article have been included as part of the supplementary information (SI). Supplementary information is available. See DOI: https://doi.org/10.1039/d6ra01935h.

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