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Traditional uses, chemical diversity and biological activities of *Panax* L. (Araliaceae): A review

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ABSTRACT:

Ethnopharmacological relevance: *Panax* L. (Araliaceae) is globally-recognized plant resource suitable for the globalization of traditional Chinese medicines. It has traditionally been used as tonic agents in various ethnomedicinal systems of East Asia, especially in China. It is often used to regulate bodily functions and considered as adjuvant therapy for tumor, resuscitation of traumatic hemorrhagic shock, etc.

Aim of this review: This review systematically summarized the information on distributions, botanical characteristics, traditional uses, chemical components and biological activities of the genus *Panax*, in order to explore and exploit the therapeutic potential of this plant.

Materials and methods: The available information about genus *Panax* was collected via the online search on Web of Science, Google Scholar, PubMed, Baidu Scholar, Science Direct, China National Knowledge Infrastructure and Springer search. The keywords used include *Panax*, saponin, secondary metabolites, chemical components, biological activity, pharmacology, traditional medicinal uses, safety and other related words. The Plant List (www.theplantlist.org) and Catalogue of Life: 2019 Annual Checklist (www.catalogueoflife.org/col/) databases were used to provide the scientific names, subspecies classification and distribution information of *Panax*.

Results: *Panax* is widely assessed concerning its phytochemistry and biological activities. To date, at least 748 chemical compounds from genus *Panax* were isolated, including saponins, flavonoids, polysaccharides, steroids and phenols. Among them, triterpenoid saponins and polysaccharides were the representative active ingredients of *Panax* plants, which have been widely investigated. Modern pharmacological studies showed that these compounds exhibited a wide range of biological activities *in vitro* and *in vivo* including antineoplastic, anti-inflammatory, hepatorenal protective, neuroprotective, immunoregulatory, cardioprotective and antidiabetic activities. Many studies also confirmed that the mechanisms of organ-protective were closely related to molecular signaling pathways, the expression of related proteins and antioxidant reactions. To sum up, genus *Panax* has high medicinal and social value, deserving further investigation.

Conclusions: The genus *Panax* is very promising to be fully utilized in the development of nutraceutical and pharmaceutical products. However, there is a lack of in-depth studies on ethnomedicinal uses of *Panax* plants. In addition, further studies of single chemical component should be performed based on the diversity of chemical structure, significant biological activities and clinical application. If the bioactive molecules and multi-component interactions are discovered, it will be of great significance to the clinical application of *Panax* plants. It is an urgent requirement to carry out detailed phytochemical, pharmacology and clinical research on *Panax* classical prescriptions for the establishment of modern medication guidelines. Exploring the molecular basis of herbal synergistic actions may provide a new understanding of the complex disease mechanisms and accelerate the process of pharmaceutical development.

1. Introduction

With the enhancement of public health awareness, great efforts have been constantly made to explore reliable alternative therapies and the medicinal natural products, especially those derived from plants due to the appearance of toxic side of chemical drugs and

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Table 1
Name and distribution of genus *Panax* plants.

Scientific name	Rank	Common name	Distribution Area
Panax assamicus R.N.Banerjee	Species	–	Assam and East Himalaya
Panax bipinnatifidus Seem.	Species	"Yuye San-chi"	Assam, China North-Central, China South-Central, East Himalaya; Myanmar, Nepal, Thailand, Tibet and West Himalaya
Panax bipinnatifidus var. *angustifolius* (Burk.) J. Wen	Infraspecific taxon	–	Assam, China South-Central, East Himalaya, Nepal, Thailand, Tibet and West Himalaya
Panax bipinnatifidus var. *bipinnatifidus*	Infraspecific taxon	–	China North-Central, South-Central, East Himalaya, Myanmar, Nepal and Thailand
Panax ginseng C. A. Mey.	Species	Korean ginseng, Ginseng	China North-Central, Khabarovsk, Korea, Manchuria and Primorye
Panax japonicus (T. Nees) C. A. Mey.	Species	Japanese ginseng or "Zhu-Jie-Shen"	From the southern foot of the Himalayas to the east, through southern China to the Japanese islands, the western end of Nepal, Bhutan and the hinterland of the Himalayas
Panax notoginseng (Burk.) F. H. Chen	Species	Chinese ginseng, "San-chi"	South Yunnan, Alpine Mountains in Western Sichuan, China South-Central and Southeast
Panax pseudoginseng Wall.	Species	Himalayan ginseng	The narrow mountains of southern Tibet, China and Nepal in the middle of the Himalayas
Panax quinquefolium L.	Species	American ginseng	From Quebec in eastern Canada to Mambatoba in the West and then south to Florida, Alabama and Oklahoma in the United States
Panax sokpayensis Shiva K. Sharma & Pandit	Species	–	East Himalaya
Panax stipuleanatus H. T. Tsai & K. M. Feng	Species	"Wild-San-chi", "Xiang-ci" and "slub San-chi"	From southern Yunnan to tropical monsoon rain forest in northern Vietnam
Panax trifolius L.	Species	Dwarf ginseng	Connecticut, Delaware, Georgia, Indiana, Kentucky, Maine, Maryland, Masachusettes, Michigan, Minnesota, New Brunswick, New Hampshire, New Jersey, New York, North Carolina, Nova Scotia, Ohio, Ontario, Pennsylvania, Prince Edward I., Quebec, Rhode I., Tennessee, Vermont, Virginia, West Virginia and Wisconsin
Panax vietnamensis Ha & Grusha.	Species	Vietnamese ginseng	China North-Central, China South-Central, China Southeast and Vietnam
Panax vietnamensis var. *fuscidiscus* K. Komatsu, S. Zhu & S. Q. Cai	Infraspecific taxon	Vietnamese ginseng	China South-Central
Panax vietnamensis var. *langbianensis* N. V. Duy, V. T. Tran & L. N. Trieu	Infraspecific taxon	Vietnamese ginseng	Vietnam
Panax vietnamensis var. *vietnamensis*	Infraspecific taxon	Vietnamese ginseng	China North-Central, China South-Central, China Southeast and Vietnam
Panax wangianus S. C. Sun	Species	–	China South-Central
Panax zingiberensis C. Y. Wu & K. M. Feng	Species	Ginger ginseng	From southern Yunnan to Tropical monsoon rain forest in northern Vietnam

—: not mentioned.

uncontrollable risks of biological agents. Botanical (plant-based) natural product is defined as the substance produced by a variety of natural sources which can be either a complex mixture extracted from raw material or a single-compound (Kellogg et al., 2019). In this regard, some medicinal plants, such as genus *Panax* plants, have been well acknowledged to show great advantages over other chemical drugs. *Panax* was derived from the Greek word meaning "all-healing" and primarily coined by the Russian botanist Carl A. Meyer. Genus *Panax* belongs to the Araliaceae family (Commission, 2015). Currently, a total of 18 plant species including infraspecific taxa, have been proved to be members of the *Panax* globally, which can be found in Table 1 (Yahara et al., 1978). The representative members of the *Panax* include *P. ginseng* C.A. Mey, *P. quinquefolius* L., *P. notoginseng* (Burk.) F.H. Chen, etc (Yang and Fang, 1991). Originated from "East Asia-North America" in paleotropical mountainous area of Paleogene, *Panax* is a floristic composition and southwest China is known as the modern distribution center (Wang, 2001).

Panax is a kind of medicinal and edible tonic with a medicinal history of over four thousand years. It mainly regulates bodily functions, prolongs lifespan and exerts antineoplastic and neuroprotective functions, which is one of the reasons for the gradually increasing demand for genus *Panax* plant. The main medicinal part of *Panax* plant is root. However, other various parts, including the leaves, flowers, rhizomes and fibrils, can also be used as medicines. Ginseng Radix et Rhizoma, Ginseng Folium and Panax Japonicus Rhizome have been officially recorded in Chinese Pharmacopoeia (2015 edition) and Japanese Pharmacopoeia (17 edition) (Commission, 2015; Ri, 2016). Based on recent studies, the aerial parts of this genus had different pharmacological activities and chemical components from those of rhizomes, which was similar to the expression of the ancient classical monograph (Bai et al., 2014). "*Ben Cao Gang Mu Shi Yi*" (On Supplement to Compendium of Material Medica), a classical Chinese medicinal treatise, recorded that *P. ginseng* leaves had the effect of "replenishing qi, invigorating lung, expelling the heat and promoting the production of the body fluid". Flowers of *P. ginseng* played a role of aromatic resuscitation (Zhao, 1998). In other medical monographs, "*Ben Jing Feng Yuan*" (in Chinese) demonstrated that the rhizomes of *P. ginseng* could induce vomiting, however, which was denied by modern studies. In addition, fibrils of *P. ginseng* were also reported to be capable of effectively treating vomiting, cough and blood loss, as well as other syndromes (Zhang, 1996). Although different species and parts of *Panax* plants have diverse usages in the Traditional Chinese medicines (TCMs) system, most of them was considered saponins as the main active ingredients (Bai et al., 2014). So far, the main chemical compounds have been isolated from the genus *Panax* include saponins, flavonoids, polysaccharides, phytosterols, polyacetylenes, amino acids and fatty acids. The main aglycones of genus *Panax* are protopanaxadiol, protopanaxatriol, oleanolic acid and ocotillol. Despite similar components to some extent, their contents are different.

Genus *Panax* plants have been recognized as precious tonic Chinese medicines since ancient times. However, the price of its medicinal materials varies greatly by species and planting pattern, which is one of the most important factors for the extensive studies on the plants. The summary of its abundant traditional uses, chemical constituents and pharmacological activities can provide better guidance on the rational utilization of the genus *Panax*. In this review, a comprehensive compilation is primarily made concerning the botany information, phytochemistry, traditional uses and bioactivities of genus *Panax*. The compounds that were found in *Panax* plants in the past 60 years, including their corresponding chemical structures and biological activities were mainly introduced. We aim to provide potential development value to analyze metabolic mechanism of its important natural products and to discover new drugs. Moreover, a more comprehensive review of structure-activity relationships of chemical components will provide

certain theoretical basis for the quality control and rational use of genus *Panax*.

2. Materials and methods

The available information about genus *Panax* was collected via Web of Science, Google Scholar, PubMed, Baidu Scholar, Science Direct, China National Knowledge Infrastructure (CNKI), and Springer search. The keywords used include *Panax*, saponin, secondary metabolites, chemical components, biological activity, pharmacology, traditional medicinal uses, safety, and other related words. The Plant List (www.theplantlist.org) and Catalogue of Life: 2019 Annual Checklist (www.catalogueoflife.org/col/) databases were used to verify the scientific names and provide subspecies classification and distribution information of *Panax*.

3. Botanical studies

According to *Flora of China*, *Panax* belongs to perennial herbs with one stalk having palmately compound leaves, and erect stems without branches. *Panax* is a self-pollinated plant that blooms at the third year of growth with a solitary inflorescence arranged in terminal umbels. When flowers bloom in May, they grow into red globose berries. Ovoid seeds are collected from the red berries with two pale yellow seeds in each fruit. The roots are fleshy and spindle-shaped with two or five rootlets and root hairs. The rhizome (neck) is considered as the important identifier that determines plant quality. (Flora of China Editorial Committee, 2001; Lan, 1978).

Genus *Panax* can be divided into two groups. The first group is characterized by short rhizomes, fleshy roots and large seeds, with tetracyclic triterpene dammarane-type saponins as its characteristic chemical components. As one of the ancient taxa, it is characterized by narrow or intermittent distribution on geographical distribution. Moreover, *P. ginseng*, *P. quinquefolius* and *P. notoginseng* are regarded as typical plants. The second group is morphologically characterized by long rhizomes, underdeveloped or incomplete fleshy roots and small seeds, rich in pentacyclic triterpene oleanolic saponin, with extensive and continuous geographical distribution. As an evolutionary group, its representative plants contain *P. japonicas*, *P. notoginseng*. and *P. pseudoginseng*, belonging to the first group in morphology. However, its chemical components are consistent with those in the second group, which is recognized as a transitional group between the two groups (Lu et al., 1992).

4. Traditional uses

TCMs advocates individualized therapy, mainly by using Chinese herbal medicine to determine the specific type of syndromes and to modulate human balance. As a significant species of genus *Panax*, *P. ginseng* has been considered as an emblematic plant in folk medicine since ancient times, with recorded nature since two thousand years ago. According to "*Shen Nong Ben Cao Jing*" (Shen Nong's Herbal), *P. ginseng* harbors diverse pharmacological effects, such as nourishing, intelligence improving, mind tranquilizing, eyesight improving and anti-aging activities. *P. ginseng* returns to the spleen meridian, which is the key medicine for invigorating the spleen (Tao, 1994). It is often compatible with TCMs Astragali Radix, Atractylodes Macrocephalae Rhizoma and other qi-invigorating and spleen-invigorating drugs for fever, high humidity, diabetes, etc. (Lan, 1978). In addition, *P. ginseng* is also used to treat hemorrhage and impotence, and to treat critically ill patients with double dose or compatibility with aconitum roots (Park et al., 2012). However, *P. ginseng* cannot be used in combination with Radix et Rhizoma Veratri Nigri or Faeces trogopterori (the dry excrement of *Trogopterus xanthipes*) (Jia et al., 2009). *P. ginseng* and its products, as the potent natural tonics, rank among the top ten in the natural medicine market in Europe and America, which have earned a high reputation among consumers. In terms of processing and administration, *P. ginseng* is usually sliced and dried. It can also be directly chewed after peeling fresh roots or soaked in wine for drinking and chewing. *P. ginseng* is usually boiled with chicken in China and Korea and made into energy drinks, tea varieties, or candies in America as well (Wu and Kang, 2019). However, most of them are mainly used medicinally, and the traditional Chinese medicine preparations represented by oral liquid are especially popular among women in Japan.

P. quinquefolius possesses a medicinal history of over 300 years in China. The earliest record found in the literature was "*Ben Cao Gang Mu*" (General Outline of Materia Medica) by Li Shizhen, demonstrating that *P. quinquefolius* was bitter in taste and cool-natured. It was mainly used to remove pathogenic fire, generate body fluid and eliminate tiredness, especially for patients with dryness-heat constitution. Zhang Xichun, a famous modern doctor, discussed *P. quinquefolius* in his book "*Yi Xue Zhong Zhong Can Xi Lu*" (in Chinese). Specifically, Zhang et al. reported that *P. quinquefolius* was a cool-natured herb with the function of supplementing energy and promoting blood circulation (Zhang, 2001). People who cannot use *P. ginseng* as tonics can use *P. quinquefolius* as a substitute. Another effect of *P. quinquefolius* is to treat hematochezia, which was found in Japanese Medical Book "*Lei Ju Yao Fang*" (in Chinese). In general, *P. quinquefolius* is administered at a dose of 3–5 g per day, which can be added or subtracted appropriately according to different disease conditions. *P. ginseng* in the original prescription of TCMs of Qing Shu Yi Qi Tang was replaced by *P. quinquefolius*, which was used clinically in combination with TCMs to treat children with summer heatstroke and other symptoms (Wang, 1852).

Originated from the remnants of the Tertiary ancient tropical mountains 25 million years ago, *P. notoginseng* was taken as the most precious Chinese medicine in *Ben Cao Gang Mu* (Compendium of Materia Medica). The application of *P. notoginseng* was first recorded in the "*Xian Zhuan Wai Ke Mi Fang*" (in Chinese), which had been nearly 600 years. At the beginning of the 20th century, Qu Huanzhang, a folk doctor, used *P. notoginseng* as one of the main ingredients to invent the famous Yunnan Baiyao. It was used for trauma and various hemorrhagic diseases largely due to remarkable curative effect (Zhou et al., 2017). In TCMs, raw *P. notoginseng* is used for hemostasis and promoting blood circulation while processed *P. notoginseng* is used for improving immunity. That is so-called "fresh hits cooked tonic" (Zhao, 2018). Meanwhile, edible *P. notoginseng* has a long history in China. Among the wide variety of *P. notoginseng*-related food (including *P. notoginseng* stewed chicken, *P. notoginseng* root fried meat and *P. notoginseng* tea), it is worthwhile to mention that the *P.* notoginseng vinegar can be used as not only seasonings but also direct health supplements (Wu and Kang, 2019). The annual sales income of *P. notoginseng* products is nearly $14.6 million, and it is exported to the Asian and European countries as food additives. Table 2 presents the traditional uses (edible and medicinal use) of *P. ginseng*, *P. quinquefolius* and *P. notoginseng*.

5. Chemical components

The chemical components of genus *Panax* (as shown in Table S1.) include saponins (**1–516**), phytosterols (**517–523**), flavonoids (**524–545**), polyacetylenes (**546–568**), polysaccharides (**569–598**), fatty acids (**599–621**), glycosides (**622–627**), coumarins (**628–630**), polyphenols (**631–632**), phenolic acids (**633–643**), sulfonic acids (**644–647**), aldehydes (**648**), ketones (**649–650**), lactams (**651–655**), amino acids (**656–673**), inorganic elements (**674–735**) and cyclic dipeptides (**736–748**).

Studies on the chemical components of *Panax* plants can be tracked back to the mid-19th century. In 1985, the first ginsenoside "panaquilon" was isolated from *P. quinquefolius* by Garrignes S (Taik-Koo Yun et al., 2001). Since 1970s, some new species of genus *Panax* (e.g. *P. vietnamensis* Ha et Grushv. and *P. sokpayensis*) have gradually attracted the attention of researchers (Duc et al., 1994; Sharma and

Table 2
The traditional uses of *P. ginseng*, *P. quinquefolius* and *P. notoginseng*.

Panax plants	Edible methods	Edible products	Classical prescriptions in China	Traditional and clinical uses	Origin
P. ginseng	Chewing; brewing tea; stewing; steaming; making porridge; grinding powder; brewing wine	Active ginseng instant tablets; ginseng nectar; ginseng wine; active ginseng sugar; ginseng coffee; ginseng pork; ginseng protein preserved beans; shenrong tonic wine	Bu zhong yi qi tang (补中益气汤)*	Curing for the weakness of spleen and stomach, kidney-Yang deficiency.	"Pi wei lun" (Treatise on the spleen and stomach)
			Li zhong wan (理中丸)*	Curing for digestive system disorders such as loss of appetite, nausea, burping, vomiting, or diarrhea	"Shanghan lun" (Treatise on treatment of diseases induced by cold)
			Si jun zi tang (四君子汤)*	Curing for chronic gastric ulcer and peptic ulcer disease	"Taiping Huimin Hejiju Fang" (Formulary of the people's benevolent pharmacy of the Taiping era)
			Gui pi tang (归脾汤)*	Curing for mental agitation, anxiety, and insomnia (guipi = restore the spleen)	"Ji sheng fang" (Prescriptions for succouring the sick)
P. quinquefolius		American ginseng buccal tablet; American ginseng qingrun tea; American ginseng powder; American ginseng drink	Qing shu yi qi tang (清暑益气汤)*	Clearing heat and detoxicating, invigorating strength and spleen nutrition	"Pi wei lun" (Treatise on the spleen and stomach)
			Yang shen bao fei wan (洋参保肺丸)*	Curing the swell of throat, dry mouth and cough	Pharmacopoeia of PR China
			Shen mai fu shen tang (参脉茯神汤)*	Curing for the syndrome of dampness-heat due to spleen deficiency	"Wen re jing wei"
			Jia wei jie du sheng mai san (加味解毒生脉散)*	Curing for the toxic shock syndrome caused by escherichia coli septicemia	"Qian jia miao fang"
P. notoginseng		Shenqi buccal tablet; radix notoginseng seasoning; radix notoginseng flower tea; radix notoginseng powder; radix notoginseng wine	An shen zhi tong tang (安神止痛汤)*	Curing for excruciating pain caused by serious injury and restlessness	"Lin ru gao gu shang yan fang ge jue fang jie"
			Hua xue dan (化血丹)*	Curing for hemoptysis, hematuria and hematochezia	"Yi xue zhong zhong can xi lu"
			Fu fang xue shuan tong jiao nang (复方血栓通胶囊)*	Curing for coronary heart disease stable angina pectoris and early diabetic nephropathy	Pharmacopoeia of PR China
			Die da huo xue san (跌打活血散)*	Curing for falls, contusions, or sprains	Pharmacopoeia of PR China

Note. *Cited from the website: https://www.wiki8.com/.

Pandit, 2009). At the same time, two-dimensional nuclear magnetic resonance (2D NMR) and quadrupole time of flight mass spectrometer (Q-TOF-MS) were used as the promising technique for identifying chemical compounds of genus *Panax* and clarifying stereo configurations (Ma et al., 1999; Wang et al., 2016a,b,c,d). From 1970 to 2000, the reported saponins compounds mostly belonged to C17 side chain varied type. However, due to the substitution of –OH and –OOH, the absolute configuration of some chiral carbon atoms has not been solved. Since 2000, various new saponins had been isolated with the development of chromatography, spectroscopy and mass spectrometry, making it possible to rapidly screen natural products of *Panax*. Yao et al. constructed a two-dimensional liquid chromatography (2D-LC) separation system based on high-performance liquid chromatography-high resolution mass spectrometry (HPLC-HRMS) platform to identify 945 ginsenosides in the leaves of *P. notoginseng* and inferred 662 ginsenosides to be the new ones (Yao et al., 2014). Chen et al. established a method for nondestructive differentiation of *Panax* species (including America ginseng and Asian ginseng) by visible and short-wave near-infrared spectroscopy (Vis-SWNIR) (Chen et al., 2011). Spectral analysis is becoming increasingly as a key technology for the quality control of genus *Panax* owing to the advantage of being rapid, non-destructive and cost-effective. Fig. 1 exhibites the trend of annually reported new saponins from the *Panax* genus within the time 1978–2019.

5.1. Saponins (1–516)

Saponins are complex glycosides composed of steroids or triterpenoid glycosides ligands and sugar chains. They are mainly distributed in terrestrial higher plants, such as *P. ginseng*, and exerted extensive pharmacological activities. In ancient folk medicine, they were used as hemolytic agents, antimicrobial agents and anti-inflammatory agents (María et al., 2015). However, the saponins exhibited effective activities are hydrolyzed aglycones or secondary glycosides.

Among the 18 species of genus *Panax*, *P. ginseng*, *P. quinquefolius* and *P. notoginseng* with similar genetic relationships take dammarane-type ginsenosides as main effective components. In addition, the content of saponins is affected by species, parts, growing period and producing area, which means their pharmacological activities are not identical. Bai et al. (2014) reviewed the saponins in the aerial parts (stems, leaves, flowers and fruits) of *P. ginseng*, *P. notoginseng*, *P. quinquefolius* and *P. japonicus*. It was found that most of the saponins in the aerial parts of *Panax* plants were dammarane-type saponins and ocotillol-type saponins. Structurally speaking, triterpenoid saponins can be divided into tetracyclic triterpene saponins (e.g. dammaran-type) and pentacyclic triterpene saponins (e.g. oleanolic-type and ocotillol-type saponins) according to the different aglycones. Protopanaxatriol (PPT) saponins and protopanaxadiol (PPD) saponins belong to dammarane-type saponins which have 1–4 glycosyl groups combined with their aglycones in general. They were considered as one of the main active components of *Panax*. In PPD type saponins, the sugar chains are usually connected with C3 or C4 position of aglycones while PPT aglycones are usually linked to the C6 or C20 position. In this way, a variety of saponins are formed due to the different types of glycosyl groups and the linking orders such as PPT saponins Re, Rf and Rg_1 and PPD saponins Rb_1, Rb_2, Rc and Rd. The number of hydroxyl groups showed a decreasing trend of $Rb_1 > Rb_2 = Rc > Rd = Re > Rg_1 = Rf$ by comparing the molecular structures of ginsenosides, which may be the key to their differences in bioactivities. The saponins isolated from different parts of *Panax* plants are summarized as follows (Tables 3–9).

5.1.1. Protopanaxadiol type saponins (PPD, 1–94)

Among all the discovered dammarane tetracyclic triterpene saponins of genus *Panax*, 94 saponins are classified as PPD type and 93 saponins are classified as PPT type according to whether there is hydroxyl on the C-6 site. In the PPDs, the sugar moieties are attached to the C3 and/or C20 in the ring of the triterpene dammarane (as in Rg_3, Rb_1, Rb_2, Rc and Rd), while acylation of 6-OH of glucose tends to occur at the end of the 3-sugar chain. It can be confirmed that acylation has become an important source of new structures in the PPDs. For example, six new acylated PPD type ginsenosides (Ra_4–Ra_9) (**7–12**) were isolated from the roots of *P. ginseng*, which were very minor acylated ginsenosides of genus *Panax* (Zhu et al., 2011). In these ginsenosides, the 4-OH in the terminal glucose of ginsenoside Ra_8 was acylated. Ginsenoside Rs_2 (**24**) (Tung et al., 2010a,b) and Rs_3 (**25**) (Baek et al., 1997) were acetylated ginsenosides of ginsenoside Rc, and 20(*S*)/(*R*) Rg_3, respectively. In addition, it reported that malonyl-substitution was the unique polar acylation mode, such as m-Ra_3 (**27**) (Ruan et al., 2010), m-Rb_1 (**28**), m-Rb_2 (**29**), m-Rc (**30**), m-Rd (**31**) (KITAGAWA et al., 1989) and m-notoginoside R_4 (**32**) (Sun et al., 2007), which are easily hydrolyzed from dry green parts of ginseng into Rb_1, Rb_2, Rc and

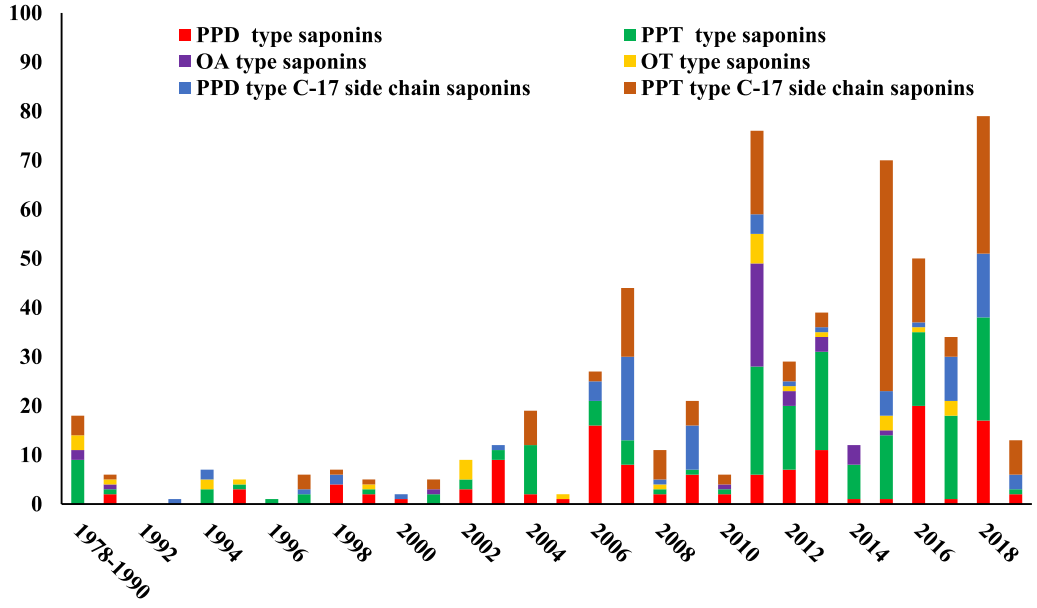
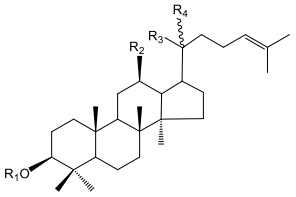
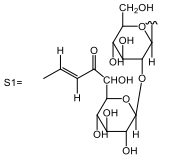


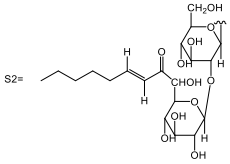
Fig. 1. The cumulative histogram plotting the number of annually reported new saponins from 1978 to 2019.

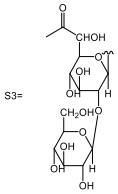
Table 3
The structure of protopanaxadiol type saponins.

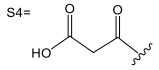


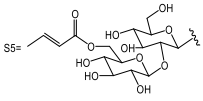
PPD type saponins core structure

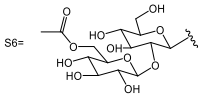


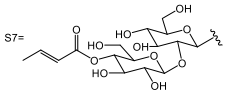












NO.	Compound	R_1	R_2	R_3	R_4	C_{20}
1	20(*S*)-25-OCH_3-PPD	H	OH	OH	OCH_3	*S*
2	20(*R*)-Protopanaxadiol	H	OH	OH	CH_3	*S*
3	20(*S*)- Protopanaxadiol	H	OH	CH_3	OH	*R*
4	Ginsenoside Ra_1	Glc^2-Glc	OH	*O*-Glc^6-Ara(*p*)^4-Xyl	CH_3	*S*
5	Ginsenoside Ra_2	Glc^2-Glc	OH	*O*-Glc^6-Ara(*f*)^2-Xyl	CH_3	*S*
6	Ginsenoside Ra_3	Glc^2-Glc	OH	*O*-Glc^6-Glc^3-Xyl	CH_3	*S*
7	Ginsenosides Ra_4	S5	OH	*O*-Glc^6-Ara(*p*)^4-Xyl	CH_3	*S*
8	Ginsenosides Ra_5	S6	OH	*O*-Glc^6-Ara(*p*)^4-Xyl	CH_3	*S*
9	Ginsenosides Ra_6	S5	OH	*O*-Glc^6-Glc	CH_3	*S*
10	Ginsenosides Ra_7	S5	OH	*O*Glc^6-Ara(*f*)	CH_3	*S*
11	Ginsenosides Ra_8	*S7*	OH	*O*Glc^6-Ara(*f*)	CH_3	*S*
12	Ginsenosides Ra_9	S5	OH	*O*Glc^6-Ara(*f*)	CH_3	*S*
13	Ginsenoside Rb_1	Glc^2-Glc	OH	*O*-Glc^6-Glc	CH_3	*S*
14	Ginsenoside Rb_2	Glc^2-Glc	OH	*O*-Glc^6-Ara(*p*)	CH_3	*S*
15	Ginsenoside Rb_3	Glc^2-Glc	OH	*O*-Glc^6-Xyl	CH_3	*S*
16	Ginsenoside Rc	Glc^2-Glc	OH	*O*-Glc^6-Ara(*f*)	CH_3	*S*
17	Ginsenoside Rd	Glc^2-Glc	OH	*O*-Glc	CH_3	*S*
18	20(*S*)-Ginsenoside Rg_3	Glc^2-Glc	OH	OH	CH_3	*S*
19	20(*R*)-Ginsenoside Rg_3	Glc^2-Glc	OH	CH_3	OH	*R*
20	20(*S*)-ginsenoside Rh_2	Glc	OH	OH	CH_3	*S*
21	20(*R*)-ginsenoside Rh_2	Glc	OH	CH_3	OH	*R*
22	Ginsenoside F_2	Glc	OH	*O*-Glc	CH_3	*S*
23	Ginsenoside Mc	H	OH	*O*-Glc^6-Ara(*f*)	CH_3	*S*
24	Ginsenoside Rs_2	Glc^2-Glc^6-AC	OH	*O*-Glc^2-Ara(*f*)	CH_3	*S*
25	Ginsenoside Rs_3	Glc^2-Glc-(6-*O*-AC)	OH	OH	CH_3	*S*
26	6″-Acetyl-ginsenoside-Rd	Glc^2-Glc^6-$COCH_3$	OH	*O*-Glc	CH_3	*S*
27	Malonyl ginsenoside Ra_3	Glc^2-Glc-COCOCH2O	OH	*O*-Glc^6-Glc^3-Xyl	CH_3	*S*
28	Malonyl ginsenoside Rb_1	Glc^2-Glc-(6-*O*-Mal)	OH	*O*-Glc^6-Glc	CH_3	*S*
29	Malonyl ginsenoside Rb_2	Glc^2-Glc-(6-*O*-Mal)	OH	*O*-Glc^6-Ara(*p*)	CH_3	*S*
30	Malonyl ginsenoside Rc	Glc^2-Glc-(6-*O*-Mal)	OH	*O*-Glc^6-Ara(*f*)	CH_3	*S*
31	Malonyl ginsenoside Rd	Glc^2-Glc-(6-*O*-Mal)	OH	*O*-Glc	CH_3	*S*
32	Malonyl notoginsenoside R_4	Glc^2-(6-Mal)Glc	OH	*O*-Glc^6-Glc^6-Xyl	CH_3	*S*
33	Notoginsenoside Fa	Glc^2-Glc^2-Xyl	OH	*O*-Glc^6-Glc	CH_3	*S*
34	Notoginsenoside Fc	Glc^2-Glc^2-Xyl	OH	*O*-Glc^6-Xyl	CH_3	*S*
35	Notoginsenoside Fe	Glc	OH	*O*-Glc^6-Ara(*p*)	CH_3	*S*
36	Notoginsenoside Ft_1	Glc^2-Glc^2-Xyl	OH	OH	CH_3	*S*
37	Notoginsenoside FP_2	Glc^2-Glc^2-Xyl	OH	*O*-Glc^6-Ara(*p*)	CH_3	*S*
38	Notoginsenoside D	Glc^2-Glc^2-Xyl	OH	*O*-Glc^6-Glc^6-Xyl	CH_3	*S*
39	Notoginsenoside K	Glc^6-Glc	OH	*O*-Glc	CH_3	*S*
40	Notoginsenoside L	Glc^2-Xyl	OH	*O*-Glc^6-Glc	CH_3	*S*
41	Notoginsenoside O	Glc	OH	*O*-Glc^6-Xyl^3-Xyl	CH_3	*S*
42	Notoginsenoside P	Glc	OH	*O*-Glc^6-Xyl^4-Xyl	CH_3	*S*
43	Notoginsenoside Q	Glc^2-Glc^2-Xyl	OH	*O*-Glc^6-Xyl^4-Xyl	CH_3	*S*
44	Notoginsenoside S	Glc^2-Glc^2-Xyl	OH	*O*-Glc^6-Ara(*f*)^5-Xyl	CH_3	*S*
45	Notoginsenoside T	Glc^2-Glc^2-Xyl	OH	*O*-Glc^6-Glc^3-Xyl	CH_3	*S*
46	Notoginsenoside R_4	Glc^2-Glc	OH	*O*-Glc^6-Glc^6-Xyl	CH_3	*S*
47	Notoginsenoside ST_4	Glc^2-Glc^2-Xyl	OH	OH	CH_3	*S*
48	Notoginsenoside FZ	Glc^2-Glc^2-Xyl	OH	*O*-Glc^6-Ara(p)	CH_3	*S*
49	Notoginsenoside Fh_1	Glc^2-Glc^2-Xyl	OH	*O*-Glc^6-Ara(*p*)^4-Xyl	CH_3	*S*
50	Notoginsenoside L_5	S4	OH	*O*-Glc^6-Ara(*f*)	CH_3	*S*
51	Notoginsenoside L_6	S4	OH	*O*-Glc^6-Ara(*p*)	CH_3	*S*
52	Notoginsenoside L_7	S4	OH	*O*-Glc^6-Xyl	CH_3	*S*
53	Notoginsenoside L_8	S4	OH	*O*-Glc^6-Glc	CH_3	*S*
54	Gypenoside IX	Glc	OH	*O*-Glc^6-Xyl	CH_3	*S*
55	Gypenoside V	Glc^2-Glc	OH	*O*-Glc^6-Rha	CH_3	*S*
56	Gypenoside XVII	Glc	OH	*O*-Glc^6-Glc	CH_3	*S*
57	Gypenoside XIII	H	OH	*O*-Glc^6-Xyl	CH_3	*S*
58	Chikusetsusaponin VI	Glc-Xyl^6-Xyl	OH	*O*-Glc-Glc^6	CH_3	*S*
59	Chikusetsusaponin III	Glc-Glc^6-Xyl	OH	OH	CH_3	*S*
60	Chikusetsusaponin VII	Glc^6-Xyl	OH	*O*-Glc^6-Glc	CH_3	*S*

(*continued on next page*)

Table 3 (*continued*)

NO.	Compound	R_1	R_2	R_3	R_4	C_{20}
61	Chikusetsusaponin FK_4	Xyl-^{6}Glc^2-Glc	OH	*O*-Glc^6-Ara(*f*)	CH_3	*S*
62	Chikusetsusaponin FK_5	Xyl-^{6}Glc^2-Glc	OH	*O*-Glc^6-Xyl	CH_3	*S*
63	Chikusetsusaponin FK_6	Xyl-^{6}Glc^2-Glc	OH	*O*-Glc	CH_3	*S*
64	Chikusetsusaponin FK_7	Glc^2-Glc	*O*-Glc	OH	CH_3	*S*
65	Quinquenoside I	S1	OH	*O*-Glc	CH_3	*S*
66	Quinquenoside II	S2	OH	*O*-Glc^6-Glc	CH_3	*S*
67	Quinquenoside III	S3	OH	*O*-Glc	CH_3	*S*
68	Quinquenoside V	Glc^2-Glc	OH	*O*-Glc^6-Glc^4-Glc	CH_3	*S*
69	Quinquenoside L_2	Glc^2-Glc	OH	*O*-Glc	CH_3	*S*
70	Quinquenosides L_{10}	Glc	OH	*O*-Glc^6-Ara(*p*)	CH_3	*S*
71	Quinquenosides L_{14}	Glc^2-Glc	OH	*O*-Ara(*p*)	CH_3	*S*
72	Quinquenosides L_{16}	Glc^2-Glc	OH	*O*-Glc^6-Glc	CH_3	*S*
73	Compound O	Glc	OH	*O*-Glc^2-Ara(*p*)	CH_3	*S*
74	Compound K	H	OH	*O*-Glc	CH_3	*S*
75	Compound Y	H	OH	OH	CH_3	*S*
76	Pseu-ginsenoside Rc_1	Glc^2-Glc^6-AC	OH	*O*-Glc	CH_3	*S*
77	Yesanchinoside J	Glc^2-Glc^6-AC	OH	*O*-Glc^6-Glc^6-Xyl	CH_3	*S*
78	Vinaginsenoside R_{16}	Glc^2-Xyl	*O*-Glc	OH	CH_3	*S*
79	Vinaginsenoside R_3	Glc^2-Glc	H	*O*-Glc	CH_3	*S*
80	Quinquenoside Jb	Glc^2-Glc	OH	*O*-Glc^6-Glc^6-Ara(*f*)	CH_3	*S*
81	20(*R*)methoxyl-ginsenoside Rg_3	Glc^2-Glc	OH	OCH_3	CH_3	*R*
82	20(*S*)methoxyl-ginsenoside Rg_3	Glc^2-Glc	OH	OCH_3	CH_3	*S*
83	Malonyl-floralginsenosides Rb_1	Glc^2-Glc	H	*O*-Glc^6-(4-Mal)Glc	CH_3	*S*
84	Malonyl-floralginsenosides Rb_2	Glc^2-(3-Mal)Glc	H	*O*-Glc^6-Glc	CH_3	*S*
85	Malonyl-floralginsenosides Rd_1	Glc^2-(2-Mal)Glc	H	*O*-Glc	CH_3	*S*
86	Malonyl-floralginsenosides Rd_2	Glc^2-(3-Mal)Glc	H	*O*-Glc	CH_3	*S*
87	Malonyl-floralginsenosides Rd_3	Glc^2-(4-Mal)Glc	H	*O*-Glc	CH_3	*S*
88	Malonyl-floralginsenosides Rd_4	Glc^2-Glc	H	*O*-(3-Mal)Glc	CH_3	*S*
89	Malonyl-floralginsenosides Rd_5	Glc^2-Glc	H	*O*-(6-Mal)Glc	CH_3	*S*
90	Malonyl-floralginsenosides Rd_6	Glc^2-(6-Mal)Glc	H	*O*-(6-Mal)Glc	CH_3	*S*
91	Malonyl-floralginsenosides Rc_1	Glc^2-(6-Mal)Glc	H	*O*-Glc^6-Xyl	CH_3	*S*
92	Malonyl-floralginsenosides Rc_2	Glc^2-(4-Mal)Glc	H	*O*-Glc^6-Ara(*p*)	CH_3	*S*
93	Malonyl-floralginsenosides Rc_3	Glc^2-(3-Mal)Glc	H	*O*-Glc^6-Ara(*p*)	CH_3	*S*
94	Malonyl-floralginsenosides Rc_4	Glc^2-(3-Mal)Glc	H	*O*-Glc^6-Ara(*f*)	CH_3	*S*

Note: Glc: *β*-D-glucopyranosyl; Glc∗: *α*-D-glucopyranosyl; Xyl: *β*-D-xylopyranosyl; Ara(*p*): *α*-D-arabinopyranosyl; Ara(*f*): *α*-L-arabinofuranosyl; Rha: *α*-L-rhamnopyranosyl; AC: acetyl; Mal: malonyl.

Rd (Wang, 2001). Pseu-ginsenoside Rc_1 (**76**) and yesanchinoside J (**77**) were isolated from *P. ginseng* and *P. japonicus* respectively, with the same acylation mode as Rg_3. The structures of parent nucleus and related compounds in PPD type saponins are shown in Table 3.

5.1.2. Protopanaxatriol type saponins (PPT, 95–187)

As a class of saponins with important biological activities, 93 kinds of PPT type saponins have been reported so far. In the PPT type saponins, the sugar moieties are attached to the ring at the C6 (as in Rg_1, Re and Rg_5) and C20 position normally. Ginsenoside Re_1 (**105**) and Re_2 (**106**) are the first example of ginsenosides moiety containing [*α*-D-glucopyranosyl-(1 → 3)-*β*-D-glucopyranosyl] isolated from the genus *Panax* (Wang et al., 2013a,b). In addition, some interesting studies showed that the substitution of malonyl or acetyl at C-60 or C-3 position could increase the antiproliferative activity by comparing the ginsenoside Rh_{24} (**111**) and ginsenoside Rh_{26} (**115**) (Li et al., 2018). 20(*S*)-sanchirhinosides A_1-A_6 (**160–165**) as minor PPT type saponins were isolated from the root extract of *P. notoginseng*. The olefine acid ester group and acetyl were attached to the sugar chain of C-6 position of **160** and **161** respectively, which might be related to inhibit mitochondrial oxidative stress (Zhang et al., 2013). Qiu et al. (2017) obtained 15 new malonyl-substituted triterpenoid saponins from the flower buds of *P. ginseng* including malonyl-floralginsenosides Re_1–Re_3 (**178–180**), through the Liquid chromatography -Mass spectrometry (LC-MS)-guided phytochemical isolation. The structures of parent nucleus and related compounds in PPT type saponins are shown in Table 4.

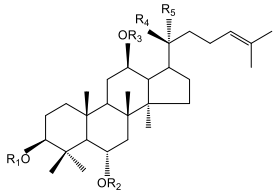
5.1.3. Oleanolic acid type saponins (OA, 188–210)

The OA saponins are minor ginsenosides with a total of thirty-four known compounds. They are characterized by C-3- and/or C-28-glycosyl chains and the presence of an inner glucuronic acid (GlcA) residue attached to C-3. Ginsenoside Ro, belonging to oleanane-type pentacyclic triterpene, is considered to be synthesized from oleanolic acid. It was found only at low levels in *P. ginseng*. Yang et al. isolated two oleanolic acid type saponins (stipuleanoside R_1 **209**, R_2 **210**) from the rhizome of *P. stipuleanatus* for the first time (Shukla et al., 1992). Stipuleanoside R_2 could be converted to R_1 by potassium hydroxide-methanol saponification, or turned into chikusetsusaponin IV by attaching a portion of the *β*-D-glucopyranosyl group at the C3 position of its glucuronic acid. Bifinosides A-C (**198–200**) were isolated from the polar fractions of a methanol extract of *P. bipinnatifidus* roots (Nguyen et al., 2011). Furthermore, a liquid chromatography-tandem mass spectrometry (LC-MS/MS) method was established to determine the natural compounds pseudoginsenoside RT_1 butyl ester, taibaienoside I (**201**), and chikusetsusaponin-IVa butyl ester (**206**), which were different from the artifactual compounds containing a butylester group (Chan et al., 2011). The structures of parent nucleus and related compounds in oleanolic acid type saponins are shown in Table 5.

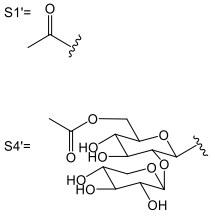
5.1.4. Ocotillol type saponins (OT, 221–243)

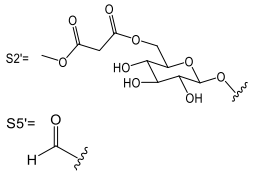
Ocotillol type saponin is a class of tetracyclic triterpene saponins containing furan ring in the side chain, which are only found in a few natural products, such as *P. pseudoginseng*, *P. quinquefolius*, *P. vietnamensis* and *P. japonicus*. Botanical natural ocotillol-type saponins mainly include pseudoginsenoside F_{11} (PF_{11}, **225**), pseudo-ginsenoside RT_5 (**229**), RT_2 (**230**), RT_4 (**236**), vina-ginsenoside R_1 (VR_1, **231**), VR_2 (**232**), VR_5 (**233**), VR_6 (**234**), VR_{13} (**235**), majonoside R_1 (MR_1, **239**) and yesanchinoside A-C (**241–243**). The PF_{11} and RT_5 in the ocotillol-type ginsenosides are the main characteristic compounds of *P. quinquefolius* different from the *P. ginseng*. VR_1 (**231**) and VR_2 (**232**) were ocotillol saponins with an acetyl group on the sugar chain at

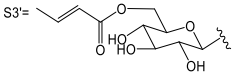
Table 4
The structure of protopanaxatriol type saponins.



PPT type saponins core structure







NO.	Compound	R_1	R_2	R_3	R_4	R_5
95	20(*S*)- protopanaxatriol	H	H	H	OH	CH_3
96	20(*R*)-protopanaxatriol	H	H	H	CH_3	OH
97	Ginsenoside Rg_1	H	Glc	H	*O*-Glc	CH_3
98	20(*S*)-ginsenoside Rg_2	H	Glc^6-Rha	H	OH	CH_3
99	20(*R*)-ginsenoside Rg_2	H	Glc^6-Rha	H	CH_3	OH
100	20(*S*)-ginsenoside Rh_1	H	Glc	H	OH	CH_3
101	20(*R*)-ginsenoside Rh_1	H	Glc	H	CH_3	OH
102	Ginsenoside Rf	H	Glc^2-Glc	H	OH	CH_3
103	Ginsenoside F_1	H	H	H	*O*-Glc	CH_3
104	Ginsenoside Re	H	Glc^2-Rha	H	*O*-Glc	CH_3
105	Ginsenoside Re_1	H	Glc	H	*O*-Glc^3-Glc	CH_3
106	Ginsenoside Re_2	H	Glc^3-Glc	H	*O*-Glc	CH_3
107	Ginsenoside Re_3	H	Glc	H	*O*-Glc^4-Glc	CH_3
108	Ginsenoside Re_4	H	Glc	H	*O*-Glc^6-Ara(*f*)	CH_3
109	Ginsenoside F_3	H	H	H	*O*-Glc^6-Ara(*p*)	CH_3
110	Ginsenoside F_5	H	H	H	*O*-Glc^6-Ara(*f*)	CH_3
111	Ginsenoside Rh_{24}	H	Ara(*p*)^6-Rha	H	*O*-Glc	CH_3
112	Ginsenoside Rg_{18}	H	Glc	H	*O*-Glc^2-Rha	CH_3
113	Ginsenoside Rs_{11}	H	Glc^2-*O*Glc^6-AC	H	*O*-Glc^6-Ara(*f*)	CH_3
114	Ginsenoside Rh_{25}	*O*-Xyl-Glc^2	H	H	*O*-Ara-Glc^6	CH_3
115	Ginsenoside Rh_{26}	*O*-(*E*)-but-2-enoyl-Glc-Glc^2	H	H	*O*-Glc	CH_3
116	Ginsenoside Re_5	H	Glc^2-Glc	H	OH	CH_3
117	Ginsenoside Mb	Glc	H	H	*O*-Glc^6-Ara(*p*)	CH_3
118	20(*R*)-ginsenoside Rh_{19}	Glc	H	H	CH_3	OH
119	Notoginsenoside R_1	H	Glc^2-Xyl	H	*O*-Glc	CH_3
120	20(*R*)-Notoginsenoside R_2	H	Glc^2-Xyl	H	CH_3	OH
121	Notoginsenoside R_3	H	Glc	H	*O*-Glc^6-Glc	CH_3
122	Notoginsenoside R_6	H	Glc	H	*O*-Glc^6-Glc*	CH_3
123	Notoginsenoside N	H	Glc^4-Glc*	H	*O*-Glc	CH_3
124	Notoginsenoside Rt	H	Glc^6-OAC	H	*O*-Glc	CH_3
125	Notoginsenoside FP_1	H	Glc	H	*O*-Glc^6-Ara(*p*)	CH_3
126	Notoginsenoside Rw_1	H	Xyl	H	*O*-Glc^6-Xyl	CH_3
127	Notoginsenoside Fh_7	H	Glc^2-Glc	H	*O*-Glc^6-Glc	CH_3
128	Notoginsenoside M	H	Glc^6-Glc*	H	*O*-Glc	CH_3
129	Notoginsenoside L_4	S1′	H	H	*O*-Glc	OH
130	Notoginsenoside L_9	S4	H	H	*O*-Glc^6-Ara(*f*)	CH_3
131	Notoginsenoside L_{10}	S4	H	H	*O*-Glc^6-Ara(*p*)	CH_3
132	Notoginsenoside L_{11}	S4	H	H	*O*-Glc^6-Xyl	CH_3
133	Notoginsenoside L_{12}	S4	H	H	*O*-Glc^6-Glc	CH_3
134	Chikusetsusaponin LM_1	H	H	H	*O*-Glc^6-Xyl	CH_3
135	Chikusetsusaponin LM_2	H	H	H	*O*-Glc^6-Xyl^3-Xyl	CH_3
136	Chikusetsusaponin LM_3	H	H	H	*O*-Glc^6-Ara(*p*)^4-Xyl	CH_3

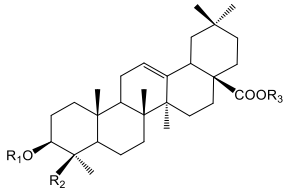
(*continued on next page*)

Table 4 (*continued*)

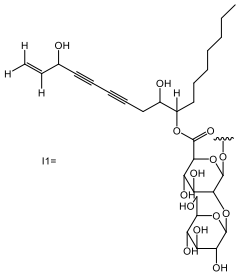
NO.	Compound	R_1	R_2	R_3	R_4	R_5
137	Chikusetsusaponin LM_4	Glc^2-Glc	H	Glc	OH	CH_3
138	Chikusetsusaponin LM_5	Glc^2-Glc	H	H	*O*-Glc^6-Ara(*f*)	CH_3
139	Chikusetsusaponin LM_6	Glc^2-Glc	H	H	*O*-Glc^6-Ara(*p*)^4-Ara(*f*)	CH_3
140	Chikusetsusaponin FK_1	Glc^2-Rha	Glc	H	CH_3	CH_3
141	Chikusetsusaponin L_{10}	H	H	Glc	OH	CH_3
142	Chikusetsusaponin L_5	H	H	H	*O*-Glc^6-Ara(*p*)^4-Xyl	CH_3
143	3-acetyl ginsenoside F_1	AC	H	H	*O*-Glc	CH_3
144	6′-acetyl-ginsenoside F_1	H	H	H	*O*-Glc^6-AC	CH_3
145	3*β*-acetoxyl ginsenoside F_1	COO	H	H	*O*-Glc	CH_3
146	6′-acetyl ginsenoside Rg_3	H	Glc^2-Glc^2	H	O-Glc^6-AC	CH_3
147	20-*O*-Gluco-ginsenoside R_f	H	Glc^2-Glc	H	*O*-Glc	CH_3
148	6′-malonyl formyl-ginsenoside F_1	H	H	H	*O*-Glc	CH_3
149	6′-emalonyl formyl ginsenoside F_1	H	H	H	S2′	CH_3
150	Pseudoginsenoside RT_3	H	Xyl	H	*O*-Glc	CH_3
151	Pseudo-ginsenosides F_8	AC-$^6Glc^2$-Glc	H	H	*O*-Glc^6-Xyl	CH_3
152	Pseudoginsenoside Rs_1	AC-$^6Glc^2$-Rha	H	H	*O*–CH_3	CH_3
153	Floralquinquenosides E	H	Glc^2-Rha	H	*O*-Glc^6-Xyl	CH_3
154	Floralginsenoside M	H	Glc^2-Rha	H	*O*-Glc^6-Ara(*f*)	CH_3
155	Floralginsenoside N	H	Glc^2-Rha	H	*O*-Glc^6-Ara(*p*)	CH_3
156	Floralginsenoside P	Glc^2-Glc	H	H	*O*-Glc^6-Ara(*p*)	CH_3
157	Quinquenoside L_{17}	H	Glc	H	*O*-Glc^6-Xyl	CH_3
158	Quinquenoside R_1	Glc^2-Glc^6-AC	H	H	*O*-Glc^6-Glc	CH_3
159	Koryoginsenoside R_1	H	Glc^6-Bu	H	*O*-Glc	CH_3
160	20(*S*)-sanchirhinosides A_1	H	S3′	H	OH	CH_3
161	20(*S*)-sanchirhinosides A_2	H	S4′	H	OH	CH_3
162	20(*S*)-sanchirhinosides A_3	H	Glc	H	*O*-Ara(*p*)	CH_3
163	20(*S*)-sanchirhinosides A_4	H	Ara(*p*)	H	*O*-Glc	CH_3
164	20(*S*)-sanchirhinosides A_5	H	Glc^2-Ara(*f*)	H	*O*-Glc	CH_3
165	20(*S*)-sanchirhinosides A_6	H	Glc^2-Xyl	H	*O*-Glc^6-Glc	CH_3
166	6*α*-acetoxy-3*β*,12*β*,20R-trihydroxydammar-24-ene	H	OAC	H	CH_3	OH
167	3-*O*-*β*-D-glucopyranosyl-20(*S*)-protopanaxatriol	Glc	H	H	OH	CH_3
168	3-formyloxy-20-*O*-*β*-D-glucopyranosyl-20(*S*)-protopanaxatriol	S5′	H	H	*O*-Glc	CH_3
169	Vina-ginsenosides R_4	Glc^2-Glc	H	H	*O*-Glc	CH_3
179	Vina-ginsenosides R_7	Glc^2-Glc-Xyl	H	H	*O*-Glc	CH_3
171	Yesanchinoside D	H	Glc^6-AC	H	*O*-Glc	CH_3
172	Yesanchinoside E	Glc^2-Rha	H	H	CH_3	*O*-Glc^6-Xyl
173	20(*R*)-ginsenoside Rh_5	OH	*O*-Glc	H	CH_3	*O*–CH_3
174	6-*O*-[*β*-D-glucopyranosyl-(1 → 2)-*β*-D-glucopyranosyl]-20-*O*-[*β*-D-glucopyranosyl-(1 → 4)-*β*-D-glucopyranosyl]-20(*S*)-protopanaxatriol	H	Glc^2-Glc	H	*O*-Glc^4-Glc	CH_3
175	20(*S*)-6-*O*-[*β*-D-xylopyranosyl-(1 → 2)-*β*-D-xylopyranosyl]dammar-24-ene-3*β*,6*α*,12*β*,20-tetrol	Xyl-Xyl^2	H	H	OH	CH_3
176	Malonyl-ginsenoside Rg_1	H	Glc^6-Mal	H	*O*-Glc	CH_3
177	Malonyl-ginsenoside Re	H	Glc^2-Rha-Mal	H	*O*-Glc	CH_3
178	Malonyl-floralginsenosides Re_1	H	(6-Mal)Glc^2-Rha	H	*O*-Glc	CH_3
179	Malonyl-floralginsenosides Re_2	H	Glc^2-Rha	H	*O*-(2-Mal)Glc	CH_3
180	Malonyl-floralginsenosides Re_3	H	Glc^2-Rha	H	*O*-(4-Mal)Glc	CH_3
181	20(*R*)-ginsenoside Rh_1 6′-acetate	H	Glc^6-Ac	H	CH_3	OH
182	20(*S*)-ginsenoside Rh_1 6′-acetate	H	Glc^6-Ac	H	OH	CH_3
183	(20S)-20-*O*-[*β*-D-xylopyranosyl-(1 → 6)-*β*-D-glucopyranosyl-(1 → 6)-*β*-D-glucopyranosyl]dammar-24-ene-3*β*,6*α*,12*β*,20-tetrol	H	H	H	*O*-Xyl-Glc^6-Glc^6	CH_3
184	(20S)-6-*O*-[(*E*)-but-2-enoyl-(1 → 6)-*β*-D-glucopyranosyl] dammar-24-ene-3*β*,6*α*,12*β*,20-tetrol	H	S3′	H	OH	CH_3
185	Ginsenoside la	Glc	H	H	*O*-Glc	CH_3
186	Pseudo-ginsenoside RT_8	Glc^2-Glc	H	H	CH_3	CH_3
187	Quinquenoside Ja	H	Glc^2-Glc	H	*O*-Glc^4-Glc	CH_3

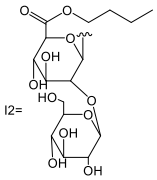
Note: Glc: *β*-D-glucopyranosyl; Glc∗: *α*-D-glucopyranosyl; Xyl: *β*-D-xylopyranosyl; Ara(*p*): *α*-D-arabinopyranosyl; Ara(*f*): *α*-L-arabinofuranosyl; Rha: *α*-L-rhamnopyranosyl; AC: acetyl; Mal: malonyl.

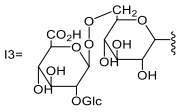
Table 5
The structure of Oleanolic acid type saponins.

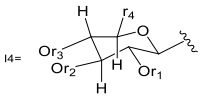


OA type saponins core structure









NO.	Compound	R_1	R_2	R_3
188	Taibaienoside IV	Glc-UA[2]-Glc	CH_3	H
189	Calenduloside E	Glc-UA	CH_3	H
190	Oleanolic acid 3-*O*-[*β*-D-glucopyranosyl-(1 → 2)-*β*-D-glucuronopyranosyl-6′-O-n-butyl ester]	I2	CH_3	H
191	Calenduloside B	Glc[4]-Gal	CH_3	Glc
192	Pjs-1 (oleanolic acid 28-*O*-*β*-D-glucopyranoside)	H	CH_3	Glc
193	Pseudo-ginsenoside-RI_3	I3	Xyl	H
194	Pseudoginsenoside RP_1	GlcUA[2]-Xyl	CH_3	H
195	Polyacetyleneginsenoside Ro	I1	CH_3	Glc
196	28-desglucosyl chikusetsusaponin IV	GlcUA[4]-Ara(*p*)	CH_3	H
197	Oleanolic acid	H	CH_3	H

NO.	Compound	R_1 = I4				R_2	R_3
		r_1	r_2	r_3	r_4		
198	Bifinoside A	Ara(*p*)	H	H	$COOCH_3$	CH_3	H
199	Bifinoside B	H	Xyl[6]-Glc	H	$COOCH_3$	CH_3	H
200	Bifinoside C	Xyl	Ara(*p*)	H	$COOCH_3$	CH_3	Glc
201	Taibaienoside I	H	H	Ara(*f*)	n-Bu	CH_3	Glc
202	Chikusetsusaponin IV	H	H	Ara(*f*)	COOH	CH_3	Glc
203	Chikusetsusaponin IV methyl ester	H	H	Ara(*f*)	$COOCH_3$	CH_3	Glc
204	Chikusetsusaponin IV*α*	H	H	H	COOH	CH_3	Glc
205	Chikusetsusaponin IVa methyl ester	H	H	H	$COOCH_3$	CH_3	Glc
206	Chikusetsusaponin IVa butyl ester	H	H	H	n-Bu	CH_3	Glc
207	Chikusetsusaponin V	H	H	Glc	COOH	CH_3	Glc
208	Chikusetsusaponin Ib	Ara(*f*)	H	H	COOH	CH_3	Glc
209	Stipuleanoside R_1	H	Glc	Ara(*f*)	COOH	CH_3	H
210	Stipuleanoside R_2	H	Glc	Ara(*f*)	COOH	CH_3	Glc
211	Stipuleanoside R_2 methyl ester	H	Glc	Ara(*f*)	$COOCH_3$	CH_3	Glc
212	Pseudoginsenoside RT_1	Xyl	H	H	COOH	CH_3	Glc
213	Pseudoginsenoside RT_1 methyl ester	Xyl	H	H	$COOCH_3$	CH_3	Glc
214	Pseudoginsenoside Rp_1 methyl ester	Xyl	H	H	$COOCH_3$	CH_3	H
215	Ginsenoside Ro	Glc	H	H	COOH	CH_3	H
216	Ginsenoside Ro methyl ester	Glc	H	H	$COOCH_3$	CH_3	Glc
217	Spinasaponin A 28-*O*-glucoside	H	Glc	H	COOH	CH_3	H
218	Araloside A methyl ester	H	H	Ara(*f*)	$COOCH_3$	CH_3	Glc
219	3-*O*-*β*-D-glucopyranosyl (1 → 3)-*β*-D-glucuronopyranoside-28-*O*-*β*-D-glucopyranosyl oleanolic acid methyl ester	H	Glc	H	$COOCH_3$	CH_3	Glc
220	3-*O*-*β*-D-xylopyranosyl (1 → 2)-*β*-D-glucopyranosyl-28-*O*-*β*-D-glucopyranosyl oleanolic acid	Xyl	H	H	CH_2OH	CH_3	Glc

Notes: Glc: *β*-D-glucopyranosyl; Xyl: *β*-D-xylopyranosyl; Ara(*p*): *α*-D-arabinopyranosyl; Rha: *α*-L-rhamnopyranosyl; GlcUA: *β*-D-glucuronopyranosyl; Gal: D-glactopyranoside; Ara(*f*): *α*-L-arabinofuranosyl; n-Bu: n-butyl.

C-6, and were formulated as monoacetylated 24(*S*)-PF_{11} and monoacetylated MR_2 (Yamasaki, 2011). VR_5 (**233**) and VR_6 (**234**) were isolated from the rhizomes and roots of *P. vietnamensis* with an α-glucosyl moiety at the first time (Duc et al., 1994). The structures of parent nucleus and related compounds in ocotillol type saponins are shown in Table 6.

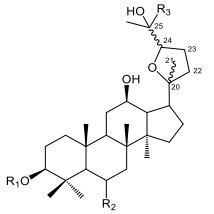
5.1.5. C-17 side chain varied saponins (244–464)

This type of saponins is mainly C-17 side chain changes, including C-24 (25) double bond displacement, hydroxylation, peroxidation, dehydrogenation, cyclization and so on. The main structures of its parent nucleus and C-17 side chain isomeric saponins reported so far are shown in Tables 7–8 In PPD type C-17 side chain varied saponins, floranotoginsenoside A (**246**) was isolated from the flowers of *P. notoginseng*, *P. ginseng* and *P. quinquefolius* with a C-23 (24) double bond and the unconfirmed absolute configuration of C-25 (Seikou Nakamura, 2007; Yoshikawa et al., 2007; Wang et al., 2009). In PPT type C-17 side chain varied saponins, compounds (**350–360**) were identified to possess a C-24-OOH group. Studies showed that Rk_1-Rk_3 (**426–428**) were isolated, which underwent a dehydration reaction of the 20-OH to form a C-20 (21) double bond (Lee et al., 2017a,b). For these C17 side chain varied saponins, it remained to be solved that the absolute configuration of chiral carbons with a hydroxylation or hydrogen peroxy substitution at C-24 or C-23. Taking PPD or PPT saponin with a side chain of 24-hydroxy-25-ene as an example, the absolute configuration of C-24 could be determined by comparing ^{13}C NMR data of C-24 and C-26 (Yang et al., 2014).

5.1.6. Other structural saponins (465–516)

In addition to the five types of saponins mentioned above, a total of 25 kinds of saponins containing isomeric sapogenins in genus *Panax* were summarized, and their specific structures are shown in Table 9. These new structural changes in saponins occur mainly in carbonylation of C-3 or C-18, dehydration between C-1 and C-2, C-5 and C-6, C-12 and

Table 6
The structure of Ocotillol type saponins.



OC type saponins core structure

NO.	Compound	R_1	R_2	R_3	C_{20}	C_{24}
221	Gypenoside F_{11}	H	*O*-Glc-Rha	CH_3	*R*	*S*
222	(20*R*,24*R*)-dammarane-20,24-epoxy-3*β*,6*α*,12*β*,25-tetraol	H	H	CH_3	*R*	*R*
223	Vina-ginsenosides R_{14}	H	*O*-Glc^2-Xyl	CH_3	*S*	*R*
224	24(*R*)-Ocotillol	H	OH	CH_3	*S*	*R*
225	Pseudoginsenoside F_{11}	H	*O*-Glc^2-Rha	CH_3	*S*	*R*
226	24(*R*)-majoroside R_1	H	OH	CH_3	*S*	*R*
227	(20*S*,24*R*,25*R*)-6-*O*-[*β*-D-glucopyranosyl-(1 → 2)-*β*-D-glucopyranosyl]-dammar-20,24-epoxy-3*β*,6*α*,12*β*,25,26-pentaol	H	*O*-Glc^2-Glc	CH_2OH	*S*	*R*
228	(20*S*,24*R*)-dammarane-20,24-epoxy-3*β*,6*α*,12*β*,25-tetraol	H	OH	CH_3	*S*	*R*
229	Pseudoginsenoside RT_5	H	*O*-Glc	CH_3	*S*	*R*
230	Pseudoginsenoside RT_2	H	*O*-Glc^2-Xyl	CH_3	*S*	*R*
231	(20*S*,24*R*)-Pseudoginsenoside F_{11}	H	*O*-Glc^2-Rha	CH_3	*S*	*R*
232	Vina-ginsenosides R_1	H	AC-Glc^2-Rha	CH_3	*S*	*S*
233	Vina-ginsenosides R_2	H	AC-Glc^2-Xyl	CH_3	*S*	*S*
234	Vina-ginsenosides R_5	H	Glc^2-Xyl^4-Glc	CH_3	*S*	*S*
235	Vina-ginsenosides R_6	H	Glc^6-Glc^2-Xyl	CH_3	*S*	*S*
236	Vina-ginsenosides R_{13}	H	Glc^2-Xyl	CH_3	*S*	*S*
237	(20*S*,24*S*,25*R*∗)-6-*O*-[*β*-D-glucopyranosyl-(1 → 2)-*β*-D-glucopyranosyl]-dammar-20,24-epoxy-3*β*,6*α*,12*β*,25,26-pentaol	H	Glc^2-Glc	CH_2OH	*S*	*S*
238	Pseudo-ginsenoside RT_4	H	Glc	CH_3	*S*	*S*
239	24(*S*)-majoroside R_1	H	Glc^2-Glc	CH_3	*S*	*S*
240	24(*S*)-6-*O*-[*β*-D-glucopyranosyl-(1 → 2)-*β*-D-glucopyranosyl]-dammar-20,25-epoxy-3*β*,6*α*,12*β*,24*α*-tetraol	H	Glc^2-Glc	CH_3	*S*	*S*
241	24(*S*)-Majoroside R_2	H	Glc^2-Xyl	CH_3	*S*	*S*
242	(20*S*,24*S*)-dammarane-20,24-epoxy-3*β*,6*α*,12*β*,25-tetraol	H	H	CH_3	*S*	*S*
243	Yesanchinoside A (24*S*)	H	AC-$^6Glc^2$-Glc	CH_3	*S*	*S*
244	Yesanchinoside B (24*S*)	H	Glc-$^6Glc^2$-Glc	CH_3	*S*	*S*
245	Yesanchinoside C (24*S*)	H	Glc^2-Glc^2-Xyl	CH_3	*S*	*S*

Note: Glc: *β*-D-glucopyranosyl; Xyl: *β*-D-xylopyranosyl; Rha: *α*-L-rhamnopyranosyl; AC: acetyl.

C-13, or further hydroxylation of C-7, C-15, C-16 and C-19-dehydroxylation. PPT saponins tend to lose H_2O at C-6, forming a double bond between C-5 and C-6. As a case in point, 5,6-didehydroginsenoside Rg_3 (**489**), 5,6-didehydroginsenoside Rd (**490**) and 5,6-didehydroginsenoside Rb_1 (**491**) derived from two species with a 5 (6) double bond and then further hydroxylated to notoginsenoside G (**476**), yesanchinoside G (**477**) and quinquenoside IV (**478**) (Yoshikawa et al., 1997, 1998; Zou et al., 2002; Wan et al., 2010; Li et al., 2018). The only difference between three new polyacetylenic oleanane-type triterpenoids (baisanqisaponin A-C (**465–467**) and chikusetsusaponin Ivα (**204**) is the rare panaxytriol moiety existed (Liu et al., 2016). Four new 19-dehydroxy-PPD type saponins, including notoginsenoside I (**479**), yesanchinoside I (**480**) and vina-ginsenoside R_3 (**481**) were isolated from the roots of *P. notoginseng*, *P. japonicus*, *P. ginseng* and *P. quinquefolius* respectively (Yoshikawa et al., 1997; Wang et al., 1998; Zou et al., 2002; Ali and Sultana, 2016). Ginsenosides Rh_{18} (**502**) was isolated from the stems and leaves of *P. ginseng* (Li et al., 2012), which could be considered as a compound derived from a precursor with a 22 (23) double bond. It was protonated to an allylic cation and then trapped by the 12-hydroxyl group. The new lupane-triterpene compounds 3*β*-*cis*-feruloyloxy-16*β*-hydroxylup-20 (29)-ene (**474**) and 3*β*-*trans*-feruloyloxy-16*β*-hydroxylup-20 (29)-ene (**475**) were isolated from the ethyl acetate extract of *P. ginseng* seeds. They showed effective inhibitory activity on neutrophil kernel factor kappa B (NF-κB) in HepG2 cells by decreasing the cellular concentrations of NO synthase (iNOS) and Cyclooxygenase-2 (COX-2) induced by inflammatory factors (Kim et al., 2012).

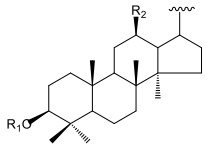
5.2. Phytosterols (517–523)

Phytosterol is a kind of active ingredients, which widely exists in roots, stems, leaves, fruits and seeds of plants. It is called “the key to life” by scientists and has been recognized and applied in the field of food by forty-seven countries. Phytosterols have good antioxidant properties and can be used as antioxidants and nutritional additives to inhibit the absorption of cholesterol and promote the degradation and metabolism of cholesterol. At present, no more than ten plant sterols have been found in *Panax*. Wei et al. obtained *β*-sitosterol (**518**) from petroleum ether of the ethanol extract of *P. notoginseng* villus root and *β*-sitosterol-D-glucoside from ether extract of *P. notoginseng* villus root (Wei et al., 1980). A new sterol glucoside 3-*O*-*β*-D-glucopyranosyl-5,22,24-stigmastatrienol (**519**), and a known sterol 5,22-stigmastadienol (**520**) were isolated from seeds of *P. ginseng* and evaluated for their inhibitory activities on tumor necrosis factor (TNF) by Kim et al. (2013).

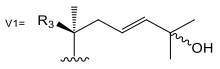
5.3. Flavonoids (524–545)

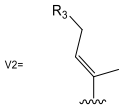
The flavonoid is a kind of low molecular natural plant ingredient, and this kind of compound has a common parent nucleus C6–C3–C6. A total of 22 flavonoids were collected in this study due to the limited reports on flavonoids of *Panax*. The study of flavonoids in *P. notoginseng* has started earlier. Flavonoids A and B were isolated from the villus roots of *P. notoginseng* for the first time by Wei et al., and identified as quercetin (**525**) and quercetin glycoside respectively (Kim et al., 1989).

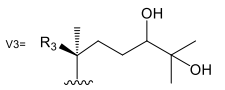
Table 7
The structure of PPD C17-side chain varied saponins.

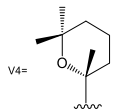


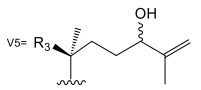
PPD C_{17}-side chain varied

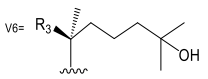


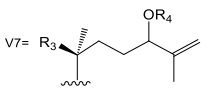


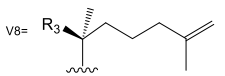


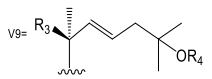


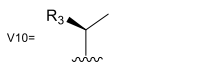


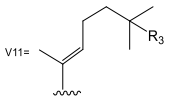


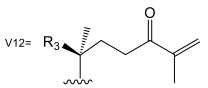


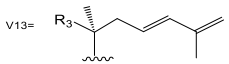


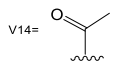


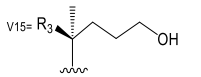


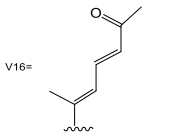


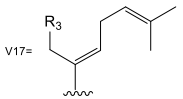


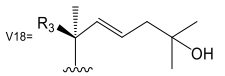


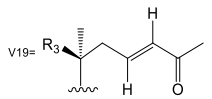


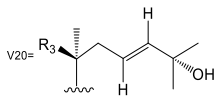


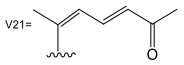


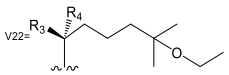


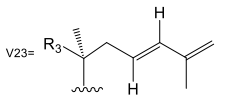


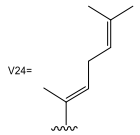


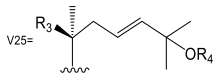


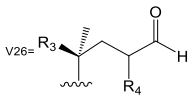


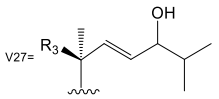


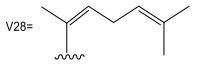


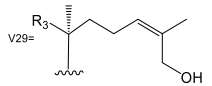


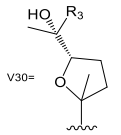












NO.	Type	Compound	R_1	R_2	R_3	R_4	Chiral Carbon
246	V1	25-hydroxy-23-ene-20(*S*)-protopanaxadiol	H	OH	OH	–	C24:*S*
247	V1	Ginsenoside-M6a	Glc^2-Glc	OH	*O*-Glc	–	C25:*S*
248	V1	Floranotoginsenoside A	Glc^2-Glc	OH	*O*-Glc^6-Ara(*f*)	–	C25:*S*
249	V1	Gypenoside XLIX	Glc^2-Glc	OH	*O*-Glc^6-Xyl	–	C25:*S*
250	V1	Notoginsenoside L_{16}	Glc	OH	*O*-Glc^6-Ara(*f*)	–	C25:*S*
251	V1	Vina-ginsenosides R_8	Glc^2-Glc	OH	*O*-Glc	–	C25:*R*
252	V1	Dammar-23 (24)-ene-3*β*,12*β*, 20(*S*), 25-tetraol-20-*O*-*β*-D-glucopyranosyl-3-*O*-*β*-D-glucopyranoside	Glc	OH	Glc	–	C25:*R*
253	V1	Majoroside F_4	Glc	OH	*O*-Glc	–	C25:*R*
254	V1	dammar-23 (24)-ene-3*β*, 12*β*, 20(*S*), 25-tetraol–20-*O*-*β*-D-glucopyranoside	H	OH	*O*-Glc	–	C25:*R*
255	V2	Notoginsenoside Fh_6	Glc^2-Gen	OH	*O*-Glc	–	–
256	V3	Notoginsenoside SFt_2	Glc	OH	OH	–	–
257	V3	dammar-3*β*,12*β*,20(*S*),24(*ζ*),25-pentaol-20-*O*-*β*-D-glucopyranoside	H	OH	*O*-Glc	–	–
258	V3	Chikusetsusaponin FM_1	Glc^2-Glc	OH	*O*-Glc^2-Xyl	–	–
259	V4	Notoginsenoside R_7	Glc	OH	–	–	–
260	V4	3*β*-acetoxy-12*β*-hydroxy-20 (*R*), 25-epoxy dammarane	AC	OH	–	–	–
261	V5	Notoginsenoside SFt_1	Glc	OH	OH	–	C24:*R*
262	V5	Notoginsenoside Fh_3	Glc^2-Glc^2-Xyl	OH	*O*-Glc^2-Xyl	–	C24:*R*
263	V5	Notoginsenoside Fh_4	Glc^2-Glc^2-Xyl	OH	*O*-Glc^6-Glc	–	C24:*R*
264	V5	Floranotoginsenoside D	Glc^2-Glc	OH	*O*-Glc^6-Ara(*f*)	–	C24:*R*
265	V5	Notoginsenosides LK_6	Glc^2-Glc	OH	*O*-Xyl	–	C24:*R*
266	V5	Notoginsenosides LK_7	Glc^2-Glc	OH	*O*-Glc^6-Ara(*f*)	–	C24:*R*
267	V5	Notoginsenosides LK_8	Glc^2-Glc^2-Xyl	OH	OH	–	C24:*R*

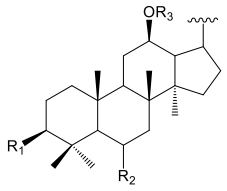
(*continued on next page*)

Table 7 (*continued*)

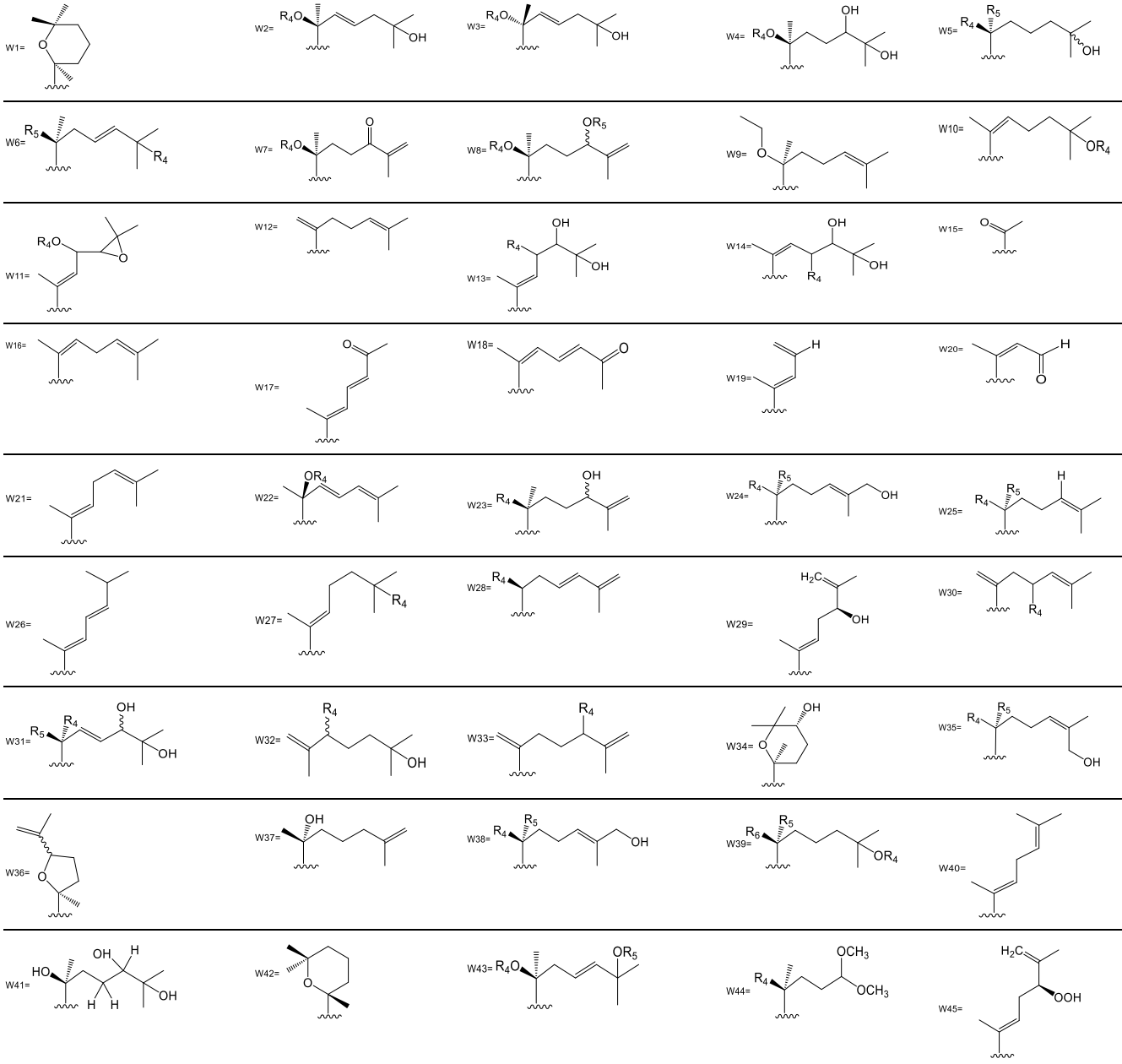
NO.	Type	Compound	R_1	R_2	R_3	R_4	Chiral Carbon
268	V5	Vinaginsenosides R_9	Glc^2-Glc	OH	*O*-Glc	–	C24:*S*
269	V5	Bipinnatifidusoside F_1	Glc^2-Glc	OH	*O*-Glc	–	C24:*R*
270	V5	Majoroside F_1	Glc^2-Glc	OH	*O*-Glc	–	C24:*R*
271	V6	Notoginsenoside Ft_2	Glc^2-Glc^2-Xyl	OH	OH	–	–
272	V7	Ginsenoside II	Glc^2-Glc	OH	*O*-Glc	OH	–
273	V7	Floranotoginsenoside B	Glc^2-Glc	OH	*O*-Glc^2-Xyl	OH	–
274	V7	Floranotoginsenoside C	Glc^2-Glc	OH	*O*-Glc^6-Ara(*f*)	OH	–
275	V7	Floralquinquenoside D	Glc	OH	*O*-Glc	OH	–
276	V8	Ginsenoside III	Glc^2-Glc	OH	*O*-Glc	–	–
277	V8	Notoginsenoside L_{17}	Glc	OH	*O*-Glc^6-Xyl	–	–
278	V8	Notoginsenoside L_{18}	Glc	OH	*O*-Glc^6-Ara(*f*)	–	–
279	V8	Notoginsenoside L_{19}	Glc	OH	*O*-Glc^6-Ara(*f*)	–	–
280	V9	Ginsenoside Rg_{12}	Glc-Glc^2	OH	OH	OH	–
281	V10	Ginsenoside L_1	Glc	OH	OH	–	–
282	V11	Ginsenoside L_2	Glc^2-Glc	OH	OH	–	–
283	V12	Notoginsenosides Ng_2	Glc^2-Glc	OH	*O*-Glc^6-Ara(*f*)	–	–
284	V12	Notoginsenosides LK_1	Glc^2-Glc	OH	*O*-Glc^6-Xyl	–	–
285	V12	Notoginsenosides LK_4	Glc^2-Glc^2-Xyl	OH	*O*-Glc^6-Xyl	–	–
286	V12	Notoginsenosides LK_5	Glc^2-Glc^2-Xyl	OH	*O*-Glc^6-Ara(*p*)	–	–
287	V12	Vinaginsenoside R_{20}	Glc^2-Glc	OH	*O*-Glc	–	–
288	V13	Notoginsenosides LK_2	Glc^2-Glc^2-Xyl	OH	*O*-Glc-Ara(*f*)	–	–
289	V13	Notoginsenosides LK_3	Glc^2-Glc^2-Xyl	OH	*O*-Glc	–	–
290	V13	Notoginsenosides LK_{15}	Glc	OH	*O*-Glc^6-Xyl	–	–
291	V14	Notoginsenoside L_1	H	OH	–	–	–
292	V15	Notoginsenoside L_2	Glc^2-Glc	OH	OH	–	–
293	V16	Notoginsenoside L_3	Glc	OH	–	–	–
294	V17	Sanchirhinoside D	Glc^2-Glc	OH	OH	–	–
295	V18	Koryoginsenoside R_2	Glc^2-Glc	OH	*O*-Glc^6-Glc	–	–
296	V19	3β,6β,12β,20(S)-trihydroxy dammar 24-methyl 1-23-ene-24-carbonyl	H	OH	OH	–	–
297	V20	3β,12β,20(*S*),25-tetrahydroxy dammar 23-ene	H	OH	OH	–	–
298	V20	Quinquenoside L_3	Glc	OH	*O*-Glc^6-Xyl	–	–
299	V21	27-demethyl-(*E*,*E*)-20 (22),23-dien-3β,12β-dihydroxydammar-25-one	H	OH	–	–	–
300	V22	20(*S*)-25-ethoxyl-dammarane-3β,12β,20-triol	H	OH	OH	CH_3	–
301	V22	20(*R*)-25-ethoxyl-dammarane-3β,12β,20-triol	H	OH	CH_3	OH	–
302	V23	Quinquenoside L_1	Glc^2-Glc	OH	*O*-Glc	–	–
303	V24	Isoginsenoside Rh_3	Glc	OH	–	–	–
304	V25	Floralginsenoside E	Glc^2-Glc	OH	OH	OH	–
305	V25	Floralginsenoside F	Glc	OH	*O*-Glc	OH	–
306	V26	Floralginsenoside Kb	Glc^2-Glc	OH	*O*-Glc	H	–
307	V26	Floralginsenoside K_C	Glc^2-Glc	OH	*O*-Glc	OH	–
308	V27	Bipinnatifidusoside F_2	Glc^2-Glc	OH	*O*-Glc	–	–
309	V28	Ginsenoside Rz_1	Glc^2-Glc	OH	–	–	–
310	V29	Vinaginsenoside R_{24}	Glc^2-Glc	OH	Glc	CH_3	–
311	V30	Ginsenoside RT_5	Glc	OH	CH_3	–	–

Glc: β-D-glucopyranosyl; Gen: gentiobiose; Xyl: β-D-xylopyranosyl; Ara(*p*): α-D-arabinopyranosyl; Ara(*f*): α-L-arabinofuranosyl; Rha: α-L-rhamnopyranosyl; —: no substituents.

Table 8
The structure of PPT C17-side chain varied saponins.



PPT C17-side chain varied



(*continued on next page*)

Table 8 (*continued*)

NO.	Type	R_1	R_2	R_3	R_4	R_5	R_6	Chiral Carbon
312	W1	OH	OH	H	–	–	–	–
313	W1	OH	H	H	–	–	–	–
314	W1	OH	OH	H	–	–	–	–
315	W1	*O*-Glc	H	H	–	–	–	–
316	W1	OH	*O*-Glc	H	–	–	–	–
317	W2	OH	*O*-Glc	H	H	–	–	–
318	W2	OH	*O*-Glc^2-Xyl	H	H	–	–	–
319	W3	OH	*O*-Glc	H	H	–	–	–
320	W3	OH	*O*-Glc^2-Rha	H	Glc	–	–	–
321	W3	OH	*O*-Glc^2-Rha	H	H	–	–	–
322	W4	OH	*O*-Glc	H	Glc	–	–	–
323	W4	OH	*O*-Glc	H	H	–	–	–
324	W4	OH	*O*-Glc	H	H	–	–	–
325	W4	OH	*O*-Glc^2-Glc	H	H	–	–	–
326	W4	*O*-Glc^2-Glc	H	H	Glc	–	–	–
327	W4	OH	*O*-Glc	H	Glc	–	–	–
328	W5	OH	OH	H	OH	CH_3	–	C25:*S*
329	W5	OH	*O*-Glc^2-Rha	H	OH	CH_3	–	C25:*S*
330	W5	OH	OH	H	CH_3	OH	–	C25:*S*
331	W5	OH	*O*-Glc^2-Rha	H	CH_3	OH	–	C25:*S*
332	W5	OH	H	H	OH	CH_3	–	C25:*R*
333	W5	OH	H	H	CH_3	OH	–	C25:*R*
334	W5	OH	OH	H	OH	CH_3	–	C25:*R*
335	W5	OH	OH	H	CH_3	OH	–	C25:*R*
336	W5	OH	*O*-Glc^2-Rha	H	CH_3	OH	–	C25:*R*
337	W5	OH	*O*-Glc^2-Rha	H	OH	CH_3	–	C25:*R*
338	W6	OH	*O*-Glc^2-Xyl	H	OH	*O*-Glc	–	–
339	W6	*O*-Glc^2-Glc	OH	H	OH	*O*-Glc	–	–
340	W6	OH	OH	H	OH	*O*-Glc^2-Xyl	–	–
341	W6	*O*-Glc^2-Glc^2-Xyl	H	H	OH	*O*-Glc^6-Glc	–	–
342	W6	OH	H	H	OH	OH	–	–
343	W6	OH	Glc	H	OH	*O*-Glc	–	–
344	W6	OH	*O*-Glc^2-Rha	H	OH	*O*-Glc	–	–
345	W6	OH	*O*-Glc	H	OH	OH	–	–
346	W6	OH	OH	H	CH_3	*O*-Glc	–	–
347	W6	OH	OH	H	OH	*O*-Glc	–	–
348	W6	OH	OH	H	OH	*O*-Glc	–	–
349	W6	*O*-Glc^2-Glc	OH	H	OCH_3	*O*-Glc^6-Xyl	–	–
350	W6	OH	*O*-Glc	H	OOH	OH	–	–
351	W6	*O*-Glc^2-Glc^2-Xyl	OH	H	OOH	*O*-Glc^6-Glc	–	–
352	W6	= O	OH	H	OOH	OH	–	–
353	W6	H	OH	= O	OOH	OH	–	–
354	W6	= O	H	H	OOH	OH	–	–
355	W6	OH	*O*-Glc	H	OOH	*O*-Glc	–	–
356	W6	OH	OH	H	OOH	*O*-Glc^6-Ara(f)	–	–
357	W6	OH	*O*-Glc	H	OOH	OH	–	–
358	W6	OH	*O*-Glc^2-Rha	H	OOH	OH	–	–
359	W6	OH	OH	H	OOH	*O*-Glc	–	–
360	W6	OH	*O*-Glc	H	OOH	OH	–	–
361	W7	*O*-Glc^2-Glc^2-Xyl	H	H	Glc^6-Glc	–	–	–
362	W7	OH	*O*-Glc^2-Rha	H	Glc	–	–	–
363	W7	OH	Glc	H	Glc	–	–	–
364	W7	OH	OH	H	Glc	–	–	–

(*continued on next page*)

Table 8 (*continued*)

365	W8	*O*-Glc^{2}-Glc	OH	H	Glc^{6}-Glc	OH	–	C24:*R*
366	W8	OH	*O*-Glc	H	H	OH	–	C24:*R*
367	W12	OH	*O*-Glc^{2}-Glc	H	H	–	–	C24:*S*
368	W8	OH	OH	H	Glc^{6}-Ara(*p*)	OH	–	C24:*R*
369	W8	OH	*O*-Glc	H	Glc	OH	–	C24:*R*
370	W8	*O*-AC-^{6}Glc^{2}-Glc	H	H	Glc	OH	–	C24:*R*
371	W8	*O*-Glc^{2}-Glc	H	H	Glc^{6}-Ara(*p*)	OH	–	C24:*R*
372	W8	*O*-Glc^{2}-Glc	H	H	Glc^{6}-Ara(*p*)	OH	–	C24:*R*
373	W8	OH	*O*-Glc^{2}-Rha	H	Glc	OH	–	C24:*R*
374	W8	OH	OH	H	Glc	OH	–	C24:*R*
375	W8	OH	*O*-Glc^{2}-Rha	H	H	OH	–	C24:*R*
376	W8	OH	OH	H	Glc	OH	–	C24:*S*
377	W9	OH	*O*-Glc	H	–	–	–	–
378	W10	OH	*O*-Glc	H	H	–	–	–
379	W10	OH	OH	H	H	–	–	–
380	W11	OH	*O*-Glc	H	H	–	–	–
381	W11	OH	*O*-Glc	H	CH_3	–	–	–
382	W11	*O*-Glc^{2}-Rha	OH	H	H	–	–	–
383	W12	OH	*O*-Glc^{3}-Xyl	H	–	–	–	–
384	W12	OH	*O*-Glc^{2}-Rha	H	–	–	–	–
385	W12	OH	OH	H	–	–	–	–
386	W12	*O*-Glc^{2}-Glc^{2}-Xyl	H	H	–	–	–	–
387	W13	OH	*O*-Glc	H	H	–	–	–
388	W13	*O*-Glc^{2}-Glc	H	H	OCH_3	–	–	–
389	W14	*O*-Glc^{2}-Glc	H	H	OCH_3	–	–	–
390	W15	OH	*O*-Glc	H	–	–	–	–
391	W15	OH	OH	H	–	–	–	–
392	W16	OH	Glc^{2}-Rha	H	–	–	–	–
393	W16	*O*-Glc^{2}-Glc^{2}-Xyl	H	H	–	–	–	–
394	W16	OH	OH	H	–	–	–	–
395	W16	*O*-Glc^{2}-Glc^{6}-AC	H	H	–	–	–	–
396	W16	OH	*O*-Glc^{2}-Rha	H	–	–	–	–
397	W17	OH	*O*-Glc	H	–	–	–	–
398	W18	OH	*O*-Glc	H	–	–	–	–
399	W19	OH	*O*-Glc	H	–	–	–	–
400	W19	*O*-Glc	OH	H	–	–	–	–
401	W20	OH	*O*-Glc^{2}-Glc	H	–	–	–	–
402	W21	*O*-Glc^{2}-Xyl	OH	H	–	–	–	–
403	W21	*O*-Glc	OH	H	–	–	–	–
404	W21	OH	OH	H	–	–	–	–
405	W21	OH	*O*-Glc^{2}-Rha	H	–	–	–	–
406	W21	OH	*O*-Glc	Glc	–	–	–	–
407	W22	OH	*O*-Glc	H	–	–	–	–
408	W23	OH	*O*-Glc	H	OH	–	–	C24:*R*
409	W23	OH	*O*-Glc^{2}-Glc	H	OH	–	–	C24:*R*
410	W23	OH	*O*-Glc^{2}-Rha	H	*O*-Glc	–	–	C24:*R*
411	W23	OH	*O*-Glc^{2}-Rha	H	*O*-Glc	–	–	C24:*R*
412	W23	OH	OH	H	*O*-Glc	–	–	C24:*S*
413	W23	OH	OH	H	*O*-Glc	–	–	C24:*S*
414	W23	OH	Glc	H	OH	–	–	C24:*R*
415	W24	OH	OH	H	*O*-Glc	CH_3	–	–
416	W25	OH	*O*-Glc	H	*O*-Glc^{6}-Ara(*f*)	CH_3	–	–
417	W26	*O*-Glc^{2}-Glc	OH	H	–	–	–	–
418	W27	OH	OH	H	O–CH_2CH_3	–	–	–
419	W27	OH	OH	H	OH	–	–	–

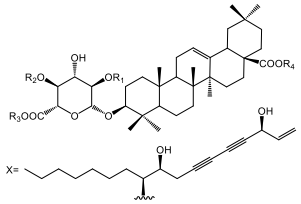
(*continued on next page*)

Table 8 (*continued*)

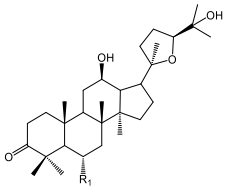
420	W27	*O*-Glc	H	H	OH	–	–	–
	W27	OH	H	H	OH	–	–	–
421								
422	W27	*O*-AC	OH	H	OH	–	–	–
423	W27	OH	OH	AC	OH	–	–	–
424	W28	OH	OH	H	*O*-Glc	–	–	–
425	W29	OH	OH	H	–	–	–	–
426	W30	OH	*O*-Glc	H	CH_3	–	–	–
427	W30	*O*-Glc	H	H	CH_3	–	–	–
428	W30	*O*-Glc^{2}-Glc	OH	H	CH_3	–	–	–
	W30	OH	*O*-Glc	H	OH	–	–	–
429								
430	W30	OH	*O*-Glc	H	OOH	–	–	–
431	W30	OH	*O*-Glc^{2}-Rha	H	OOH	–	–	–
432	W31	OH	*O*-Glc	H	CH_3	CH_3	–	C24:*S*
433	W31	OH	*O*-Glc^{2}-Rha	H	–	*O*-Glc	–	C24:*R*
434	W32	OH	*O*-Glc	H	OH	–	–	C22:*S*
435	W33	OH	*O*-Glc	H	OH	–	–	–
436	W34	OH	*O*-Glc	H	–	–	–	–
437	W34	OH	*O*-Glc^{2}-Xyl	H	–	–	–	–
438	W35	OH	OH	H	CH_3	OH	–	–
439	W35	OH	*O*-Glc	H	OH	CH_3	–	–
440	W35	OH	*O*-Glc^{2}-Xyl	H	OH	CH_3	–	–
441	W35	OH	*O*-Glc^{2}-Glc	H	OH	CH_3	–	–
442	W35	OH	OH	H	*O*-Glc	CH_3	–	–
443	W36	OH	OH	H	–	–	–	C24:*S*
444	W36	OH	OH	H	–	–	–	C24:*R*
445	W37	OH	$OCOCH_3$	H	–	–	–	–
446	W38	OH	OH	H	OH	CH_3	–	–
447	W38	OH	*O*-Glc	H	CH_3	OH	–	–
448	W38	*O*-Glc	H	H	OH	CH_3	–	–
449	W39	OH	H	H	CH_3	OH	CH_3	–
450	W39	OH	H	H	H	OCH_3	H	–
451	W39	OH	OH	H	H	OH	CH_3	–
452	W39	OH	*O*-Glc^{2}-Rha	H	H	OH	CH_3	–
453	W40	OH	OH	H	–	–	–	–
454	W40	OH	*O*-Glc	H	–	–	–	–
455	W41	OH	OH	H	–	–	–	–
456	W42	OH	OAC	H	–	–	–	–
457	W42	AC	OH	H	–	–	–	–
458	W43	*O*-AC-^{6}Glc^{2}-Glc	H	H	Glc	OH	–	–
459	W43	OH	*O*-Glc^{2}-Rha	H	Glc	OH	–	–
460	W43	*O*-Glc^{2}-Glc	H	H	Glc	OH	–	–
461	W43	*O*-Glc^{2}-Glc	H	H	Glc^{6}-Ara(*f*)	OH	–	–
462	W44	OH	OH	H	*O*-Glc	–	–	–
463	W45	OH	*O*-Glc	H	–	–	–	–
464	W45	OH	*O*-Glc^{2}-Rha	H	–	–	–	–

Glc: *β*-D-glucopyranosyl; Xyl: *β*-D-xylopyranosyl; Ara(*p*): *α*-D-arabinopyranosyl; Ara(*f*): *α*-L-arabinofuranosyl; Rha: *α*-L-rhamnopyranosyl; AC: acetyl; —: no substituents.

Table 9
The structure of other saponins.



Type A



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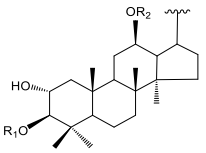

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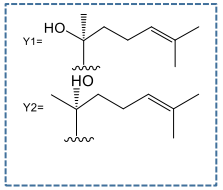

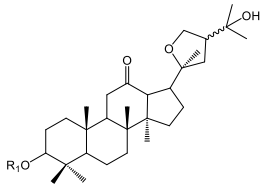

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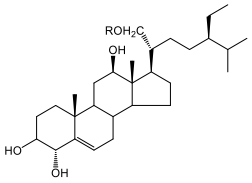
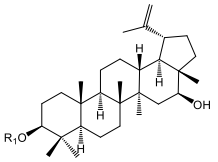


Type E



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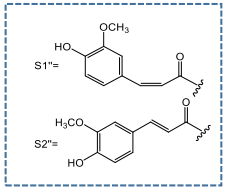

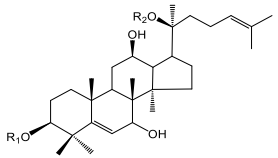

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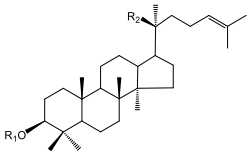

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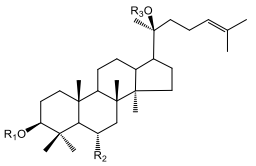
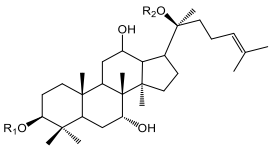
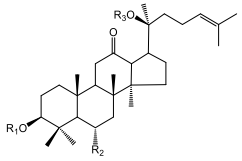


Type I



Type J



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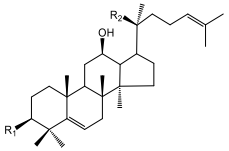
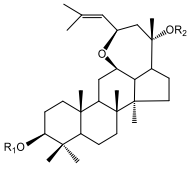


Type L



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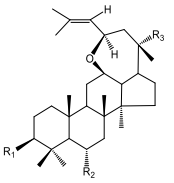

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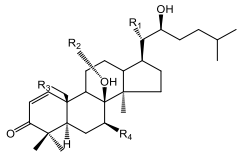
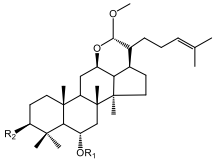


Type O



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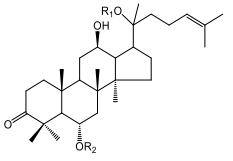

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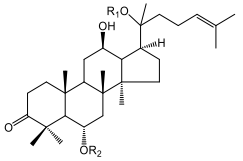

Type R

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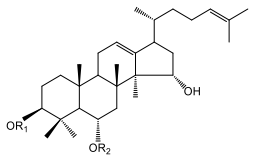
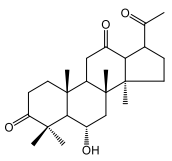
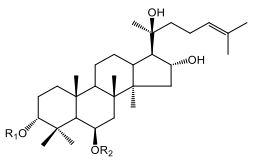
Table 9 (*continued*)



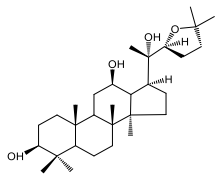
Type S



Type T



Type U



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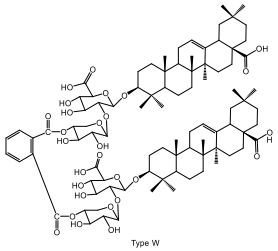
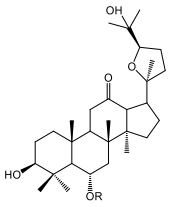
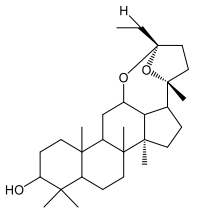


Type W



Type X



Type Y

NO.	Type	Compound	R_1	R_2	R_3	R_4
465	A	Baisanqisaponin A	H	H	X	Glc
466	A	Baisanqisaponin B	H	Ara(*f*)	X	Glc
467	A	Baisanqisaponin C	Glc	H	X	H
468	B	Pseudo-ginsenoside RT_6	*O*-Glc	–	–	–
469	B	Pseudoginsengenin R_1	OH	–	–	–
470	C	Gypenoside L	Glc^2-Glc	H	–	–
471	C	Gypenoside L_1	Glc^2-Glc	H	–	–
472	D	24(*R*)-Pseudoginsenoside G_1	Glc^2-Glc	–	–	–
473	D	24(*S*)-Pseudoginsenoside G_2	Glc^2-Glc	–	–	–
474	E	3*β*,4*α*,12*β*-trihydroxystigmast-5-en-21-yl octadecan-9′,12′-dienoate	R = $CO(CH_2)_7CH{=}CHCH_2CH{=}CH(CH_2)_4CH_3$			
475	E	stigmast-5-en-3*β*,4*α*,12*β*, 21-tetraol-21-octadec-9′,12′-dienoate	R = $CO(CH_2)_7CH{=}CHCH_2(CH_2)_3CH_3$			
476	F	3*β*-*cis*-feruloyloxy-16*β*-hydroxylup-20 (29)-ene	S1"	–	–	–
477	F	3*β*-*trans*-feruloyloxy-16*β*-hydroxylup-20 (29)-ene	S2"	–	–	–
478	G	Notoginsenoside G	Glc^2-Glc	Glc	–	–
479	G	Yesanchinoside G	Glc^2-Glc	Glc^6-Xyl	–	–
480	G	Quinquenoside IV	Glc^2-Glc	Glc^6-Glc	–	–
481	H	Notoginsenoside I	Glc^2-Glc	*O*-Glc^6-Glc	–	–
482	H	Yesanchinoside I	Glc^2-Glc	*O*-Glc^6-Glc-Xyl	–	–
483	H	Lanost-24-en-3*β*-ol-3-*O*-*β*-D-arabinopyranosyl-(2′→1″)-*O*-*β*-D-arabinoside	Glc^2-Ara(*p*)	CH_3	–	–
484	I	Vina-ginsenoside R_3	Glc^2-Glc	H	Glc	–
485	J	Chikusetsusaponins LT_5	Glc	H	Glc^6-Glc	–
486	J	Chikusetsusaponins LT_8	Glc	H	Glc	–
487	J	Chikusetsusaponin LN_4	Glc^6-Xyl	H	Glc^6-Ara(*p*)	–
488	J	3*β*,6*α*-20(*S*)-6,20-bis(*β*-D-glucopyranosyloxy)-3-hydroxy dammar-24-en-12-one	H	Glc	Glc	–
489	J	Chikusetsusaponin FK_2	Glc^2-Glc	Glc	–	–
490	J	Chikusetsusaponin FK_3	Xyl-$^6Glc^2$-Glc	Glc	–	–
491	K	7*β*-hydroxyl ginsenoside Rd	*O*-Glc-Glc^2	Glc	–	–
492	L	5,6-didehydroginsenoside Rg_3	*O*-Glc-Glc^2	OH	–	–
493	L	5,6-didehydroginsenoside Rd	Glc^2-Glc	Glc	–	–
494	L	5,6-didehydroginsenoside Rb_1	Glc^2-Glc	Glc^6-Glc	–	–
495	M	Notoginsenoside LX	Glc	Glc^6-Ara(*f*)	–	–
496	M	Notoginsenoside LY	H	Glc^6-Ara(*f*)	–	–

(*continued on next page*)

Table 9 (*continued*)

497	M	Notoginsenosides Ng_1	Glc	Glc^6-Xyl	–	–	
498	M	Notoginsenoside Fh_8	Glc-Glc	Glc-Ara(*f*)	–	–	
499	M	Notoginsenoside Fh_9	Glc-Glc	Glc-Ara(*p*)	–	–	
500	M	Notoginsenoside L14	Glc	Glc^6-Xyl	C23:*S*	–	
501	M	Notoginsenoside L13	Glc	Glc^6-Xyl	C23:*R*	–	
502	M	3*β*,20*S*-dihydroxy-12*β*, 23*R*-epoxydammar-24-ene 3-*O*-[*β*-D-glucopyranosyl (1 → 2)-*β*-D-glucopyranosyl]-20-*O*-*β*-D-glucopyranoside	Glc^2-Glc	*O*-Glc	–	–	
503	M	Ginsenoside La	Glc	Glc	–	–	
504	N	Ginsenosides Rh_{18}	OH	$O\text{-}Glc^2$-Rha	*O*-Glc	–	
505	N	12*β*,23(*R*)-epoxydammara-24-ene-3*β*,6*α*,20(*S*)-triol	OH	OH	OH	–	
506	N	3*β*,6*α*, 20*S*-trihydroxy-12*β*, 23*R*-epoxydammar-24-ene 6-*O*-[*α*-L-rhamnosyl (1 → 2)-*β*-D-glucopyranosyl]-20-*O*-*β*-D-glucopyranoside	H	$O\text{-}Glc^2$-Rha	*O*-Glc	–	
507	P	Notoginsenoside P_1	Glc	OH	–	–	
508	Q	6-*O*-*β*-D-glucopyranosyl-20*R*-protopanaxadiol-3-one	Glc	H	–	–	
509	R	6-*O*-*β*-D-glucopyranosyl-20-*O*-*β*-D-glucopyranosyl-20(*S*)-protopanaxadiol-3-one	Glc	Glc	–	–	
510	T	6*α*-hydroxy-22,23,24,25,26,27-hexanordammar-3,12,20-trione	Ara(*p*)	Ara(*p*)	–	–	
511	S	Dammar-12,24-dien-3*α*,6*β*,15*α*-triol-3*α*-D-arabinopyranosyl-6*β*-L-arabinopyranoside	–	–	–	–	
512	U	Dammar-24-en-3*α*,6*β*,16*α*,20*β*-tetraol-3*α*-D-arabinopyranosyl-6*β*-D-arabinopyranoside	Xyl	Xyl	–	–	
513	V	(20*S*,22*S*)-dammar-22,25-epoxy-3*β*,12*β*,20-triol	–	–	–	–	
514	W	Pseudoginsenoside-RI_2	–	–	–	–	
515	X	12-one-pseudoginsenoside F_{11}	Glc^2-Rha	–	–	–	
516	Y	(12*R*,20*S*,24*S*)-20,24-;12,24-diepoxy-dammarane-3*β*-ol	–	–	–	–	

Glc: *β*-D-glucopyranosyl; Xyl: *β*-D-xylopyranosyl; Ara(*p*): *α*-D-arabinopyranosyl; Ara(*f*): *α*-L-arabinofuranosyl; Rha: *α*-L-rhamnopyranosyl; —: no substituents.

Then, a flavonoid glycoside, quercetin-3-*O*-sophora glycoside was obtained from *P. notoginseng* leaves (Wei et al., 1980). Subsequently, six flavonoids from the stems and leaves of *P. notoginseng* were separated by Zheng et al. (Zheng, 2004). They were identified as kaempferol (**526**), quercetin (**525**), kaempferol-7-*O*-*α*-L-rhaamnosioside (**529**), kaempferol-3-*O*-*β*-D-galactoside, kaempferol 3-*O*-(2″-*β*-D-glucopyranosyl)-*β*-D-galactopyranoside (**533**) and quercetin3-*O*-(2″-*β*-D-glucopyranosyl)-*β*-D-galactopyranoside (**534**). Four other compounds besides kaempferol (**526**) and quercetin (**525**) were isolated from *P. notoginseng* for the first time. However, at the moment, there are few flavonoids from *P. notoginseng*, most of which exist in the form of flavonols. In addition, some flavonoids were isolated from the flower buds and leaves of *P. ginseng*. Flavonoids are also important chemical components in the aerial part of *P. ginseng*. Three flavonoids, ginsenoside, trifolium glycoside and kaempferol were isolated from stem and leaf of *P. ginseng* (Wang et al., 1985). Xu et al. (Xu and Yang, 2016) also isolated two new compounds from *P. ginseng* buds and identified them as kaempferol-3-*O*-*α*-L-rhamnoside and kaempferol-3-*O*-(2,3-di-*trans*-p-hydroxy cinnamoyl) -*α*-L-rhamnoside (**536**). In recent years, some studies have also found that there are flavonoids in the roots of *P. quinquefolius* (Zhang et al., 2002a,b).

5.4. Polyacetylenes (546–568)

Alkyne hydrocarbons are less distributed in *Panax*, mainly in the underground parts of *P. notoginseng*, *P. ginseng*, *P. quinquefolius* and *P. vietnamensis*. Under guidance of the growth inhibition experiment of staphylococcus aureus, Lin et al. analyzed the chemical components of the fat-soluble parts of *P. notoginseng* (Lin et al., 2002). Panaxynol (**546**) and panaxydol (**548**) were separated from the petroleum ether fraction of *P. notoginseng* for the first time, the yields accounting for 0.01% and 0.033% respectively, which had strong inhibitory effects on staphylococcus aureus. Duan et al. used supercritical CO_2 extraction method to obtain *P. notoginseng* samples with high purity (> 98%) and high yield (0.077%) (Duan et al., 2008). Zhou et al. analyzed the polyacetylene components in different parts of *P. notoginseng* by GC-MS (Zhou et al., 2013). The results showed that the total amount of polyacetylene components was higher in the main roots, flowers and rhizomes of *P. notoginseng* and lower in stems and leaves. The amount of panaxynol (**546**) in *P. notoginseng* flower was higher, but panaxydol (**548**) could hardly be detected. In addition, wei et al. isolated seven polyacetylenes from the underground part of *P. vietnamensis*, two of which were newly discovered compounds, namely 10-acetoxy-heptadeca-8(*E*)-ene-4,6-diyne-3-ol (**561**) and heptadeca-l,8(*E*),10(*E*)-triene-4,6 diyne-3,12-diol (**573**) (Wei, 2001). Two new ones trahydrofuranic polyacetylene glycosides, belonging to a novel biogenetically type in nature, panaxfuraynes A (**563**) and B (**564**) were separated from the root of *P. ginseng* (Lee et al., 2009). Five new polyacetylenes (including ginsenoynes A (**547**), ginsenoynes B (**549**), ginsenoynes C (**552**), ginsenoynes D (**553**) and ginsenoynes E (**551**) were isolated from the hexane extract of the root of *P. ginseng* (Hirakura et al., 1991), whose structures were determined by spectral and chemical methods. New C17-and C14-polyacetylenes were isolated from the dried roots of *P. quinquefolius* by Fujimoto (Fujimoto et al., 1994).

5.5. Polysaccharides (569–598)

Biologically important polysaccharides have been less widespread in genus *Panax*. To date, 30 polysaccharides compounds were isolated from the rhizomes of *P. japonicus* plants, the roots of *P. quinquefolius*, *P. notoginseng* plants and the flower buds of *P. ginseng*.

5.6. Fatty acids (599–621)

Lipid is an important component of *P. ginseng*. Fatty acid compounds not only have a wide range of biological activities but also are synthetic

precursors of many important biological substances. The fatty acid component of *P. ginseng* roots is mainly unsaturated fatty acid and linoleic acid has the highest content. Lu et al. determined the fatty acid content of different processed ginseng products by gas chromatography (GC) method (Lu et al., 1993). It was found that the linoleic acid (**613**) content of *P. ginseng* increased up to 62%–65% after heating treatment, while the relative content of stearic acid was the lowest. The relative content of stearic acid was less than 1% in *P. ginseng* and *P. quinquefolius* root, while *P. notoginseng* did not contain stearic acid (**607**) and its oleic acid (**614**) content was close to the sum of the former two. At present, 24 main fatty acids (including pentadecylic acid (**604**), palmitic acid (**605**), stearic acid (**607**), margaric acid (**606**), oleic acid (**621**) and linolenic acid (**613**), etc.) were found in *P. ginseng* and *P. notoginseng* roots.

5.7. Other compounds (622–748)

In addition to the above-mentioned compounds, Coumarins, such as skimmin (**628**), apiosylskimmin (**629**) and daphnin methyl ether (**630**) were isolated from *P. notoginseng* plants. On the one hand, phenols, esters, aldehydes and ketones compounds were also separated and identified. On the other hand, 14 cyclic dipeptides and five lactamides have been isolated and identified from *P. notoginseng* plants and *P. ginseng* respectively. Further studies on this research field should be conducted. Eighteen amino acids and 72 trace elements were also isolated from *P. notoginseng* and *P. trifolius*.

6. Biological activities

Modern pharmacological studies showed that genus *Panax* had an important role in the antineoplastic, anti-inflammatory, hepatorenal protective, neuroprotective, immunoregulatory, cardioprotective, antidiabetic and anti-hypotensive activities, hemostasis, the activation of blood stasis and etc. The bioactivities of compounds in *Panax* can be found in Table 10.

6.1. Antineoplastic activity

According to the statistics of literature, genus *Panax* exhibited good antineoplastic activity (as shown in Fig. 2). In 1978, Yun et al. reported that extract of Radix et Rhizoma Ginseng Rubra inhibited the proliferation of dimethylolbutanoic acid (DMBA)-induced neoplasms and prolongated the survival time of mice (Yun and Lee, 2001). Upon 30-year development, the antineoplastic research of ginsenosides has become a hot spot in *P. notoginseng* research, with multiple in-depth studies from the metabolism of ginsenosides, antineoplastic mechanisms and other aspects.

A large number of *in vivo* and *in vitro* experiments have proved that ginsenosides compounds exhibit significant antineoplastic activities through some similar pathways. Moreover, the secondary metabolic saponins and their saponins produced by ginsenosides under the action of intestinal bacteria are natural precursors of the antineoplastic effect of *P. notoginseng* (Jin et al., 2006). The main antineoplastic mechanisms can be summarized as follows: (1) cell cycle arrest, induction of apoptosis and inhibition of neoplasm proliferation; (2) inhibitory effects on metastasis of cancer cells; (3) activation of the immune system. A study revealed that the growth of human cervical cancer cells (HeLa) was inhibited by ginsenoside Rd (**17**) in a time- and dose-dependent manner, with an IC_{50} value of 150.5 ± 0.8 μg/mL after 48h incubation (Yang et al., 2006a). Further studies demonstrated that ginsenoside Rb_1 (**13**) exerted calcium antagonism to reverse the production of cancer cells (Lin et al., 2012). Notoginsenoside Ft_1 (**36**) restrained the cell proliferation and induced apoptosis in SH-SY5Y cells through p38 mitogen-activated protein kinases (MAPK) and extracellular regulated protein kinases (ERK)1/2 pathways. It had been shown to have potential therapeutic effects on human neuroblastoma (Gao et al., 2014a,b). Additionally, in a study by Li et al., *P. notoginseng* polysaccharide was added into the culture medium of H22 hepatoma cells *in vitro* and further administered to neoplasm-bearing mice *in vivo*. The results showed that *P. notoginseng* polysaccharide significantly inhibited the growth of H22 cells and prolonged the survival time of neoplasm-bearing mice. The discovery of antineoplastic polysaccharides would broaden the selection of immunotherapeutic drugs for hepatoma (Li et al., 2016). Lee et al. showed that ginseng polysaccharide was a potential non-toxic antineoplastic immune activator, which could activate macrophages to produce active nitrogen intermediates, thus subsequently mediating the tumor killing effects (Lee et al., 1997). Compounds with antineoplastic activity derived from *Panax* are shown in Fig. 3.

6.2. Anti-inflammatory activity

P. ginseng is one of the most widely used alternative drugs to treat inflammation. In recent years, a growing number of studies found that ginsenosides had a variety of pharmacological effects against inflammatory diseases. Owing to the different chemical structures of ginsenosides, they may have different pharmacological activities and mechanisms. Ginsenoside Rh_2 (**20**) significantly alleviates inflammatory bowel disease in mice induced by dextran sodium sulfate (DSS) (Ye et al., 2014). Ginsenoside F_2 is expected to be the alternative natural herbal ingredient with low-side-effect to treat the skin inflammation induced by tetradecanoyl phorbol alcohol acetate (TPA) in mice (Park et al., 2016). Ginsenoside Rg_5 (**417**) has pharmacological effects on anti-neuroinflammation (Xu and Gao, 2017). Ginsenoside Rh_1 (**100**) can effectively stimulate the central nervous system to improve mental acuity and intellectual performance (Tam et al., 2018). In addition, the effects of ginsenoside-compound K (G-CK) on T cells in mice with collagen-induced arthritis showed that G-CK might be a promising drug for the treatment of rheumatoid arthritis (Chen et al., 2018). Dong et al. found that the metabolic pathway of ginsenosides (ginsenoside Ra_1 **4**, Rb_1 **13**, Rb_2 **14**, Rc **16**) *in vivo* was mainly turned into deglycosylated ginsenoside (ginsenoside Rd **17**) through intestinal microflora (Dong et al., 2017). Then it was absorbed into the blood circulation to exert its effect (Kim et al., 2014). Based on literature review, the anti-inflammatory mechanism of ginsenosides can be roughly attributed to: (1) exerting antioxidant effect; (2) inhibiting the expression of inflammatory factors; (3) reducing the phosphorylation and activation of MAPK and activation of Protein Kinase B (PKB/Akt); (4) altering in the intestinal microenvironment. However, a recent study (Han et al., 2018a) showed that the ethanol extract of ginseng berry calyx (Pg-C-EE) from ginseng plants might have anti-inflammatory properties targeting nuclear factor-kB by inhibiting AKT, which suggested that the development of ginseng berry calyx extract might be beneficial in the treatment of inflammatory diseases.

6.3. Hepatorenal protection activity

Chinese herbal medicines have the ability to protect the liver, which have been proved to be the effective anti-inflammatory and antioxidant. *Panax* has the hepatoprotective effects and its mechanism includes blocking fibrosis, inhibiting tumorigenesis, eliminating viruses and inhibiting oxidative damage (Del Prete et al., 2012; Dhiman et al., 2012). Qi et al. first used *P. ginseng* fruit anthocyanin (GFA), an extract of ginseng fruit, to suppress renal injury induced by cisplatin, demonstrating that GFA has a renoprotection effect induced by cisplatin (Qi et al., 2018). The possible mechanisms include inhibition of cisplatin-induced oxidative stress, reduction of inflammatory response and apoptosis. Ginseng oligopeptides can significantly reduce the levels of tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β) and interleukin-6 (IL-6) in serum ($P < 0.05$). Studies have shown that ginseng oligopeptides can protect liver cells by improving alcohol-induced serum inflammatory response.

Table 10
Biological activities of chemical components isolated from the *Panax*.

Biological Activities	Name	Description	*In vivo*/*In vitro*	Reference
Antineoplastic activity	Ginsenoside Rg_1	Rg_1 (1 mg/L) and Rh_1 (100 mg/L) could significantly increase the transcription of dendritic cell IL-12p40 mRNA, which was consistent with its protein expression	*In vitro*	Wang (2004)
	Ginsenoside Rg_3	activated caspase-3, caspase-8, and caspase-9 and regulated the expression of B-cell lymphoma-2 (Bcl-2) and Bax to induce apoptosis of cancer cells; enhanced the anticancer activity of geftinib, making non-small cell lung cancer (NSCLC) cells more sensitive to geftinib; inhibited autophagic flux and enhancing the sensitivity of NSCLC cells to icotinib	*In vitro*	(Park et al., 2014), (Dai et al., 2019), (Wang et al., 2019a,b)
	Ginsenoside Rg_2	suppressed the migration and invasion of liver cancer cells by upregulating the protein expression of Abstract Rho Gtpase activating protein 9 (ARHGAP9)	*In vivo*	Sun et al. (2019)
	Ginsenoside Rg_5	stimulated the apoptosis of human breast cancer cells; regulated proteins to block the G0/G1 phase of cell division; inhibited the increase of breast cancer cells	*In vitro*	Kim and Kim (2015)
	Ginsenoside Rh_1	Rg_1 (1 mg/L) and Rh_1 (100 mg/L) could significantly increase the transcription of DCIL-12p40 mRNA, which was consistent with its protein expression	*In vitro*	Wang (2004)
	Ginsenoside Rh_2	inhibitd the proliferation and induces apoptosis of A375-S2 cell lines cultured *in vitro* by activating the caspase-8 and caspase-3 signaling pathways	*In vitro*	Hyun-Eui and Lee (1999)
	Ginsenoside Rd	inhibited the proliferation and promote cell apoptosis of human glioma U251 cells; depressed miR-18a-mediated Smad2 expression regulation; inhibitd HeLa cell proliferation, and induced cell apoptosis through down-regulating Bcl-2 expression; up-regulated Bax expression; lowered the mitochondrial transmembrane potential, and activated the caspase-3 pathway	*In vitro*	(Gu et al., 2019), (Wang et al., 2016), (Yang et al., 2006b)
	Ginsenoside Re	raised p21 level, reduced phosphorylation of cyclinA-cyclin-dependent kinase2 (CDK2), raised S phase arrest	*In vitro*	Jang et al. (2014)
	Ginsenoside RK_1	inhibited the growth activity of liver cancer cells	*In vitro*	Toh et al. (2011)
	Ginsenoside RK_3	inhibited the growth activity of liver cancer cells	*In vitro*	Toh et al. (2011)
	Ginsenoside Rb_2	inhibited the body pigmentation in the zebrafish i*n vivo* system and reduced melanin contents and tyrosinase activity	*In vivo*	Lee et al. (2015)
	Ginsenoside Rh_6	the melanogenic inhibitory activity of ginsenoside Rh_6 was 23.9% at a concentration of 80 μM	*In vivo* and *In vitro*	Lee et al. (2015)
	Ginsenoside F_4	inhibited Bcl-2 and increased the expression of Bax protein to further promote the apoptosis of JK cells of human lymphocytoma	*In vitro*	Chen et al. (2013)
	Vina-ginsenoside R_4	the melanogenic inhibitory activity of vina-ginsenoside R_4 was 27.8% at a concentration of 81 μM	*In vivo* and *In vitro*	Lee et al. (2015)
	Vina-ginsenoside R_{13}	the melanogenic inhibitory activity of vina-ginsenoside R_{13} was 35.2% at a concentration of 82 μM	*In vivo* and *In vitro*	Lee et al. (2015)
	20(*S*)-dammar-12*β*,20-dihydroxyl-24-ene-3*β*-succinate	inhibited the proliferation of hct-8 human colon cancer cell lines with IC_{50} of 33.5 g/mL	*In vitro*	María et al. (2015)
	20(*S*)-dammar-20-hydroxyl-24-ene-3*β*,6*α*,12*β*-trisuccinate	inhibited the proliferation of hct-8 human colon cancer cell lines with IC_{50} of 38.6 g/mL	*In vitro*	María et al. (2015)
	Notoginsenoside R_1	reduced lung cancer stem cells, reduced epithelial-tomesenchymal transition, inhibited the proliferation of HeLa cells, up-regulated the gap junction function of cells and enhanced the cytotoxicity of cisplatin	*In vitro*	(Lee et al., 2017a,b), (Qi et al., 2012)
	Notoginsenoside R_1	reduced integrin-1 protein, reduced E-selectin, Intercellular adhesion molecule-1 (ICAM-1), enhanced cisplatin cytotoxicity, enhancement of gap junction's activity, enhancement of gap junction's activity	*In vivo*	(Wang et al., 2010a,b,c)
	25-OCH_3-PPD	(10, 20, 40 mg/kg) dosedependently increased the latency time and antinociceptive percentage in hot-plate test	*In vitro*	Zhang et al. (2018)
	25-OH-PPD	(10, 20, 40 mg/kg) dosedependently increased the latency time and antinociceptive percentage in hot-plate test	*In vitro*	Zhang et al. (2018)
	Compound K	inhibited the enzyme activities of CYP2C9 and CYP3A4 in the HLMs, The IC_{50} values were 16.00 μM and 9.83 μM, and Ki values were 14.92 μM and 11.42 μM for CYP2C9 and CYP3A4, respectively	*In vitro*	Xiao et al. (2016)
	Ginsenoside F_1	chromatin condensated and increased in the populationof sub-G1 hypodiploid cellsm, IC_{50} = 23.2 μM	*In vivo*	Tung et al. (2010)
	Ginsenoside F_2	the cytotoxic effect with IC_{50} of 50 μg/mL through apoptosis	*In vivo*	Shin et al. (2012)
	Ginsenoside F_5	chromatin condensated and increased in the populationof sub-G1 hypodiploid cellsm, IC_{50} = 62.4 μM	*In vitro*	(Lee et al., 2017a,b)
	Ginsenoside Rp_1	decreased the stability of the insulin-like growth-factor-1 receptor (IGF-1R) protein in breast cancer cells	*In vitro*	Tung et al. (2010)

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Table 10 (*continued*)

Biological Activities	Name	Description	*In vivo*/*In vitro*	Reference
	Floralginsenosides Ta	chromatin condensated and increased in the populationof sub-G1 hypodiploid cellsm, IC_{50} = 36.3 μM	*In vitro*	Kang et al. (2011)
	β-elemene	inhibited nucleic acid synthesis of tumor cells, induced apoptosis and differentiation of tumor cells, enhanced the immunogenicity of tumor cells, improved the immune function of tumor cells	*In vitro*	Tung et al. (2010)
	*Panax*ynol	inhibited the proliferation of human gastric adenocarcinoma cells *in vitro* and inhibited the synthesis of DNA, RNA and protein in cell L1210	*In vitro*	Lu et al. (2016)
	Quercetin	down-regulated mutated P53 protein, blocked cell cycle, inhibited tyrosine kinase, promoted apoptosis, inhibited heat shock protein and inhibited the expression of Ras and other anticancer	*In vitro*	Lu et al. (2016)
	Polysaccharide	activated CD4 (+) T-cells, raised serum IL-2	*In vitro*	Zhu and He (2004)
	Trilinolein	modulated phosphoinositide 3-kinase/protein kinase B (PI3K/Akt) pathway	*In vitro*	Yoshizaki et al. (2013)
	Ginseng Pectin SA	the maximum inhibition rate of L-929 cell migration was 60%	*In vitro*	Yoshizaki et al. (2013)
	Ginseng Pectin PGP2a	regulated cell cycle and apoptosis to inhibit the proliferation of gastric cancer cells hgc-27	*In vitro*	Fan et al. (2010)
	Ginseng Pectin RGAP	inhibited the proliferation and metastasis of solid tumors in mice	*In vitro*	Li et al. (2014)
	Saponin–phospholipid complex	decreased the tumor progression on Dimethylolbutanoic acid (DMBA)-induced breast cancer rats and increases the levels of antioxidant enzymes including superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPX)	*In vivo* and *In vitro*	Cai et al. (2013)
Anti-inflammatory activity	Ginsenoside Rb_1	enhanced the phagocytic capacity of macrophages for bacteria via activation of the p38/Akt pathway	*In vivo* and *In vitro*	Cai et al. (2013)
	Ginsenoside Rd	dose-dependent inhibited neutrophil kernel factor kappa B (NF-κb) expression with an IC_{50} value of 3.47, down-regulated the expression of major mitogen-activated protein kinases (MAPK) pathway, inhibited the mRNA expressions of inflammatory factors interleukin-1β (il-1β), il-6, tumor necrosis factor α (tnf-*α*) and inducible nitric oxide synthase (iNOS)	*In vitro*	Xin et al. (2018)
	Ginsenoside Re	down-regulated the expression of MAPK pathway and kappaa B pathway, and had an obvious inhibitory effect on the mRNA expressions of inflammatory factors il-1*β*, il-6, tnf-*α* and iNOS	*In vitro*	(Lee, 2014), (Wang, 2011)
	Ginsenoside Rg_1	reduced neuronal death in the ischemic region	*In vivo*	Wang (2011)
	Ginsenoside Rg_5	dose-dependent inhibited the expression of NF-κB, with an IC_{50} value of 0.61, and inhibited the expression levels of cyclooxygenase-2(COX-2) and iNOS genes	*In vivo*	Zheng et al. (2019)
	Ginsenoside Rh_1	inhibitd the expression of ifn-gamma-activated Janus kinase/signal transducer and activator of transcription (JAK/STAT) and extracellular regulated protein kinases (ERK) signaling pathways and their downstream transcription factors NF-κB, interferon regulatory factor-1 (irf-1) and STAT-1 through iNOS promoters	*In vivo* and *In vitro*	(Lee, 2014)
	Ginsenoside Rz_1	inhibited the expression of NF-κB, with an IC_{50} value of 0.63, and inhibited the expression levels of cox-2 and iNOS genes to exert anti-inflammatory effects	*In vitro*	Park et al. (2004)
	Ginsenoside Rf	mediated by the brain-derived neurotrophic factor (BDNF)/tropomyosin receptor kinase B (TrkB)/cAMP response element-binding cAMP response element binding protein (CREB) pathway	*In vivo*	(Lee, 2014)
	Ginsenoside Rk_1	inhibited the expression of NF-κB, with an IC_{50} value of 0.75, and inhibited the expression levels of cox-2 and iNOS genes	*In vitro*	Qin et al. (2019)
	Ginsenoside Rp_1	inhibited the expression of il-1*β* in mouse monocytes induced by lipopolysaccharide (LPS) by blocking the activation of NF-κB signaling pathway	*In vitro*	(Lee, 2014)
	Notoginsenoside R_1	increased Bcl-2 expression and reduced Bax expression in the stomach tissues of rats caused by N-methyl-N′-nitro-N-nitrosoguanidine (MNNG) were eliminated	*In vivo*	Xiang et al. (2013)
	Vina-ginsenoside R_2	be metabolized to ocotillol via presence of recombinant plasmid (PRT4), and the metabolites, particularly ocotillol, may inhibited inflammation by inhibiting the binding of LPS to toll-like receptors 4 (TLR4) on macrophages	*In vivo* and *In vitro*	Luo et al. (2019)
	Majonoside R_2	be metabolized to ocotillol via PRT4, and the metabolites, particularly ocotillol, may inhibited inflammation by inhibiting the binding of LPS to TLR4 on macrophages	*In vivo* and *In vitro*	Jeong et al. (2015)
	Compound K	downregulated the activities of MAPKs, and NF-κB in LPS-treated murine peritoneal macrophages	*In vivo* and *In vitro*	Jeong et al. (2015)
	Ginsenoside Rc	suppressed TANK-binding kinase (TBK1)/IkB kinase ε/interferon regulatory factor-3 and p38/ATF-2 signaling	*In vitro*	Joh et al. (2011)
	Pg-C-EE	nuclear-factor-κB-targeted anti-inflammatory properties through suppression of AKT	*In vivo* and *In vitro*	Yu et al. (2017)

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Table 10 (*continued*)

Biological Activities	Name	Description	*In vivo*/*In vitro*	Reference
	PQS	inhibited apoptosis and inflammation via regulation of endoplasmic, reticulum (ER) stress and the associated inflammatory signaling pathway	*In vivo* and *In vitro*	Han et al. (2018b)
	Ginseng Pectin Y-5	enhanced the activity of natural killer cells (NK) and phagocytes	*In vitro*	Xie et al. (2018)
	G-Rh_2-B2	reduced expression of TNF-α, IL-6, and IL-1β, and activities of p38 MAPK, and NF-κB	*In vitro*	Cho et al. (2014)
	PNFS	down-regulated iNOS gene overexpression and thereby decreased NO overproduction via the inhibition of TLR_4-mediated MAPK/NF-κB signaling pathways, but not the PI3K/Akt signaling pathway	*In vitro*	Bi et al. (2012)
	PPQN	inhibited TNF-α, IL-1, and IL-6 secretions, followed by NO production with respective values of 40.5%, 41.1%, 34.4%, and 11.1% suppression	*In vitro*	Peng et al. (2015)
Anti-oxidative ability	Ginsenoside Rb_1	increased the activity of SOD and CAT by more than 10%	*In vivo*	Wang et al. (2015)
	Ginsenoside Rg_1	increased the activity of SOD and CAT by more than 10%	*In vivo*	Wang et al. (2015)
	Ginsenoside Rf	inhibited hypoxia induced-COX-2 expression and cellular migration	*In vitro*	Wang et al. (2015)
	Ginsenoside C-Mx	increased expression of cytoprotective antioxidants such as heme oxygenase-1 (HO-1) and NADPH quinineoxidoreductase-1 (NQO-1) expression by enhancing the nuclear accumulation of Nuclear factor erythroid 2-related factor 2 (Nrf2)	*In vitro*	Song et al. (2019)
	Majonoside R_2	enhanced gamma-aminobutyric acid (GABAA-ergic) systems in the brain	*In vitro*	Liu et al. (2018)
	Ginsenoside Rb_1	inhibited the production and accumulation of reactive oxygen species (ROS) in the mitochondria under stress, enhanced antioxidant enzyme activity, inhibited oxidase activity, maintained mitochondrial membrane potential stability	*In vitro*	Yobimoto et al. (2000)
	CP-1a	inhibited on superoxide, hydroxyl and 1,1-diphenyl-2-picrylhydrazyl (DPPH) radical	*In vitro*	Zhou et al. (2019)
	CP-2a	inhibited on superoxide, hydroxyl and DPPH radical	*In vitro*	(Wang et al., 2012a,b)
	SLPF	EC50 values of reducing activity, DPPH free radical scavenging activities, the superoxide anion removal ability, and the 2, 2′-azinobis-(3-ethylbenzothiazoline-6-sulfonic acid) (ABTS) free radical removal ability are 7.212, 2.893, 2.949, and 0.855 mg/mL, respectively	*In vitro*	(Wang et al., 2012a,b)
Hepatorenal Protection Activity	Ginsenoside Rg_1	regulated p-erk1/2 and p-jnk pathways, Caspase-3 expression was down-regulated, reduced the expression of oxidation factors such as GSH and SOD, promoted bcl-2 expression and inhibited Bax expression in brain tissue, regulated the expression of NO, activated Nrf2/ho-1 signaling pathway, inhibited tau phosphorylation	*In vitro*	Dai et al. (2018)
	Ginsenoside Rg_1	activated Nrf2 signaling pathway, repressed the expression levels of inflammation-related genes including TNF-α, IL-1β, IL-6, COX-2, and iNOS	*In vivo*	(Wang et al., 2013a,b)
	Ginsenoside Rg_2	regulated oxidative stress, inflammation, and apoptosis to inhibit cisplatin, induced injury to renal cells and LLC-PK1 cells in rats	*In vivo*	Ning et al. (2018)
	Ginsenoside Rg_5	regulated oxidative stress, inflammation, and apoptosis to inhibit cisplatin, induced injury to renal cells and LLC-PK1 cells in rats	*In vivo*	Park et al. (2015)
	Ginsenoside Rg_6	regulated oxidative stress, inflammation, and apoptosis to inhibit cisplatin, induced injury to renal cells and LLC-PK1 cells in rats	*In vivo*	Park et al. (2015)
	Ginsenoside Rb_1	down-regulated expression of il-1β, upregulated brain-derived neurotrophic factor (BDNF) and Caspase3, reduced accumulation of tau and beta powder, regulated FAS/FASL/P53 protein, regulated Ca^{2+} to alleviate glucose (GLU) damage	*In vivo*	Park et al. (2015)
	Ginsenoside Rd	down-regulated reduced GLU concentration, Hif-1 expression, suppressed inflammatory factors NF-κB and P38, activated extracellular regulated protein kinases (ERK) and ark-dependent signal pathways	*In vitro*	Gao et al. (2010)
	Ginsenoside Re	reduced melanoma differentiation-associated gene (MDA) and increased SOD expression, decreased acetylcholinesterase expression, protected the substantia nigra Dopamine (DA), increased cell DA expression	*In vitro*	Waxman and Lynch (2005)
	Notoginsenoside R_1	reduced the accumulation of amyloid beta, increased the expression of choline acetyltransferase, activated Nrf2/ho-1 pathway promotes Nrf2 synthesis, decreased the expression of TNF-α and il-1β, increased the expression of PPAR gamma protein	*In vivo* and *In vitro*	(Jin et al., 2006)
	Ginsenoside F_4	regulated oxidative stress, inflammation, and apoptosis to inhibit cisplatin-induced injury to renal cells and LLC-PK1 cells in rats	*In vivo*	Yang et al. (2016)
	Ginsenoside RK_3	dose-dependent reduction of cisplatin-induced renal injury in LLC-PK1 cells	*In vitro*	Park et al. (2015)
	Ginsenoside Rh_4	dose-dependent reduction of cisplatin-induced renal injury in LLC-PK2 cells	*In vitro*	(Baek et al., 2006)

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Table 10 (*continued*)

Biological Activities	Name	Description	*In vivo*/*In vitro*	Reference
	Ginsenoside Rh_2	increased expression of Bcl-2 and decreased expression of p53, Bax, cytochrome *c*, caspase-8, caspase-9, and caspase-3 in kidney tissues	*In vivo*	(Baek et al., 2006)
	Ginsenoside Rg_3	reduced NF-κB and iNOS protein expressions	*In vivo*	(Qi et al., 2019a,b)
	GOP	0.25 g/kg BW dose of ginseng oligopeptide may be more conducive to improve the liver ethanol metabolism enzyme activity, The serum levels of TNF-*α*, IL-1*β* and IL-6 were reduced, and the serum inflammatory response induced by alcohol was improved	*In vivo*	Kang et al. (2009)
	PQS	possessed protective effects in cisplatin-induced Acute Kidney Injury (AKI) through suppression of oxidative stress, inflammation and apoptosis	*In vivo*	(Liu et al., 2018a,b,c,d)
	PNS	promoted bcl-2 upregulation, activated the PI3K/Akt pathway, cutted Ang II expression, regulated antisense oligonucleotide (ASO) and icam-1, decreased the expression of H_2O_2	*In vitro*	Ma et al. (2017)
	PNS	reduced acute hepatic failure (AHF) acute liver injury, reduced mortality and promoted the proliferation and repair of liver cells	*In vivo*	Si et al. (2016)
Neuroprotective activity	Ginsenoside Rb_1	inhibitd neuronal apoptosis and increased anti-apoptotic genes and modified the neuroprotective effects of glia-derived neurotrophic factors in transient cerebral ischemia	*In vivo*	Ren et al. (2007)
	Notoginsenoside Rb_1	improveed cognitive and sensorimotor deficits by PNS-Rb_1, at least partially, by the modulation of the Akt/mTOR/PTEN signaling pathway	*In vivo*	Yuan et al. (2007)
	Ginsenoside Rb_2	exerted neuroprotective effects in LPS-stimulated N9 microglial cells by blocking TNF-*α* production	*In vitro*	Yan et al. (2018)
	Ginsenoside Rb_3	inhibited the increase of intracellular expansion, the apoptosis of ischemic damaged cells and the activity of aspartic enzyme in the skin to protect the ischemic brain injury	*In vitro*	Wu et al. (2007)
	Ginsenoside Rg_1	modulated microglia-mediated cytokines and the related upstream mediators, protected neuronal activity and promoted neuroplasticity in particular brain regions associated with cognition processing	*In vivo* and *In vitro*	(Zhu et al., 2010)
	Ginsenoside Rg_5	inhibited memory impairment and neuroprotection caused by alcohol or scopolamine	*In vitro*	(Shi et al., 2019a,b)
	Ginsenoside Rd	reduced NO formation and prostaglandin E2 (PGE2) synthesis and inhibited dendrite loss, cell atrophy, cell body changes and nerve cell loss in TH (+) cells	*In vitro*	Bao et al. (2005)
	Ginsenoside Re	increased the expression of bcl-2 protein and bcl-2 mRNA, and reduced the expression of bax, baxmRNA, inducible nitric oxide synthase (INOS) and caspase-3	*In vivo*	Lorenz et al. (2002)
	Ginsenoside Rk_1	inhibited memory impairment and neuroprotection caused by alcohol or scopolamine	*In vivo*	Gando (2010)
	Pseudoginsenoside-F_{11}	antagonized the memory dysfunction induced by scopolamine	*In vivo*	Bao et al. (2005)
	Compound K	regulated $GABA_A$ and gamma-aminobutyric acidB ($GABA_B$) receptors	*In vitro*	Konno (1987)
	PNS	SH-SY5Y cells exposed to oxygen/glucose deprivation injury by inhibiting the overexpression of NgR_1, RhoA, and Rho-associated coiled-coil protein kinase 2 (ROCK2)	*In vivo* and *In vitro*	(Lee et al., 2013a,b)
	WGP	altered the composition and diversity of the gut microbiota in mice with antibiotic-associated diarrhea and promoted the re-establishment of the microbial environment, thus alleviating the symptoms of diarrhea	*In vivo*	Shi et al. (2016)
	TSPJ	upregulated gap-43, a growth-related protein, to improve learning and memory	*In vivo*	Li et al. (2019a,b,c)
Anxiolytic activity	Ginsenoside Rg_1	be related to the GABA-benzodiazepine-chloride channel receptor complex	*In vivo*	(Wang et al., 2013a,b)
	Ginsenoside Rg_5	be related to the GABA-benzodiazepine-chloride channel receptor complex	*In vivo*	Cha et al. (2005)
	Ginsenoside Rk_1	be related to the GABA-benzodiazepine-chloride channel receptor complex	*In vivo*	Cha et al. (2005)
	Ginsenoside Rd	inhibited the anti-inflammatory effect of LPS by stimulating ht-29 cells to secrete inflammatory factor il-8	*In vitro*	Cha et al. (2005)
	Ginsenoside Rb_1	reduced Anxiety index, raised Risk assessment, reduced grooming behaviors in electric powered mobile things (EPMT), raised total number of line crossings of an open field after SPS, reduced SPS-induced decreasement in hypothalamic neuropeptide Y expression, raised in locus cerulean tyrosine hydroxylase expression, reduced expression of BDNF	*In vivo*	Lv (2017)
	Ginsenoside Rg_3	reduced Anxiolytic effect via γ-aminobutyrate A (GABA A) receptor(s)	*In vitro*	Lee et al. (2016)
	Ginsenoside Rh_2	antagonized GABA/benzodiazepines	*In vivo*	(Lee et al., 2013a,b)
	Ginsenoside Rg_1	raised both the frequency and duration of open arm entries	*In vivo*	Kim et al. (2009)
	Ginsenoside Ro	raised Both the frequency and duration of open arm entries	*In vivo*	Carr et al. (2006)
	Pseudoginsenoside-F_{11}	antagonized decreases of DA	*In vivo*	Kim et al. (2009)

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Table 10 (*continued*)

Biological Activities	Name	Description	*In vivo*/*In vitro*	Reference
	PNS	raised levels of 5-hydroxytryptamine (5-HT), DA and Noradrenaline (NE)	*In vitro*	Wu (2003)
	PQS	might be related to its effects on certain neuronal systems	*In vivo*	(Wang et al., 2011a,b)
Immunoregulatory activity	Ginsenoside Rg_1	promoted immune factor IL-2, INF-ɤ, IL-12p40 secretion, and enhanced the antitumor immune after chemotherapy, and reduced cisplatin for the body's immune injury	*In vivo*	Wei et al. (2007)
	Ginsenoside Rb_1	inhibited TNF-α production in LPS-stimulated RAW264.7 macrophages	*In vitro*	Wu (2017)
	Ginsenoside Rb_2	inhibited the production of TNF-α in LPS-stimulated RAW264.7 cells and differentiated U937 cells with IC_{50} values of 27.5 mM and 26.8 mM, respectively	*In vitro*	Cho et al. (2001)
	Ginsenoside Rd	induced immune responses to T helper 1 (TH1) and T helper 2 (TH2) cytokines	*In vivo*	Cho et al. (2001)
	Notoginsenoside L	increased the level of immune gloulin in blood plasma	*In vivo*	Yang et al. (2007)
	Notoginsenoside N	increased the level of immune gloulin in blood plasma	*In vivo*	
	Notoginsenoside R_1	combined with aluminum adjuvant, enhanced the immune effect of aluminum adjuvant, reduced its usage, avoided the adverse reactions caused by high-dose aluminum adjuvant and enhanced the immunogenicity of HAV antigen	*In vivo*	Chen (2002)
	Ginsenoside RT_5	increased the production of interlukin-2 (IL-2) cytokine from PMA/Io-activated EL-4 T cells in a dose-dependent manner	*In vitro*	Tao et al. (2008)
	20(*S*)-Ginsenoside Rh_2	increased the production of IL-2 cytokine from PMA/Io-activated EL-4 T cells in a dose-dependent manner	*In vitro*	Vinh et al. (2019)
	Oleanolic acid β-D-glucopyranosyl ester	increased the production of IL-2 cytokine from PMA/Io-activated EL-4 T cells in a dose-dependent manner	*In vitro*	Vinh et al. (2019)
	RG-CW-EZ-CP	upregulated the phosphorylation of three major MAPKs, including extracellular signal-regulated kinase, and p38	*In vitro*	Vinh et al. (2019)
	Ginseng Pectin S-IIA	induced human monocytes and thp-1 cells to produce interleukin il-8	*In vitro*	Kim et al. (2019)
	Ginseng Pectin SB	promoted the secretion of cytokines il-8 and il-2 in human monocytes thp-1 and mouse spleen cells at low concentrations	*In vitro*	Sonoda et al. (1998)
	Ginseng Pectin SB	inhibited the secretion of cytokines il-8 and il-3 by human monocyte thp-1 and mouse spleen cells in high concentration	*In vitro*	(Tian et al., 2011a,b)
	PNS	enhanced the humoral and cellular immune responses to ovalbumin (OVA) in mice when given together with OVA	*In vitro*	(Tian et al., 2011a,b)
Improve microcirculation activity	Ginsenoside Rg_1	inhibited the adhesion of white blood cells to endothelial cells and the degranulation of mast cells, and Rg_1 and R_1 inhibited the production of Lps-induced granulocytes H_2O_2 at 1.0 mg/mL	*In vivo*	Qin et al. (2006)
	Ginsenoside Rb_1	inhibited the adhesion between white blood cells and endothelial cells and the degranulation of mast cells Rb_1 (1.0 mg/mL) and R_1 (0.2 mg/mL) significantly inhibited the expression of Lps-induced granulocytes CD11b and CD18	*In vivo*	Sun et al. (2007)
	Notoginsenoside R_1	inhibited the adhesion between white blood cells and endothelial cells and the degranulation of mast cells Rb_1 (1.0 mg/mL) and R_1 (0.2 mg/mL) significantly inhibited the expression of Lps-induced granulocytes CD11b and CD18	*In vivo*	Sun et al. (2007)
Cardioprotective activity	Ginsenoside Rb_1	reduced apoptosis of cardiomyocytes caused by ischemia reperfusion injury	*In vivo*	Sun et al. (2007)
	Ginsenoside Rb_3	protected myocardial function during ischemia and inhibited proliferation of vascular smooth muscle cells, Calcium channels have a blocking effect	*In vivo*	Liu and Liu (2002)
	Ginsenoside Rc	calcium channels have a blocking effect	*In vitro*	(Wang et al., 2010a,b,c), (Wang et al., 2010a,b,c)
	Ginsenoside Rd	inhibited the receptor regulation of vascular smooth muscle cells, ameliorated isoproterenol (ISO)-induced cardiotoxicity in rats via upregulation of the activities of antioxidants, and suppression of inflammatory and apoptotic biomarkers	*In vitro*	Sun et al. (1994)
	Ginsenoside Rd	improved cardiac dysfunction and remodeling induced by pressure overload	*In vivo* and *In vitro*	(Guan et al., 2006), (Sun et al., 2019a,b)
	Ginsenoside Rd	ameliorates ISO-induced cardiotoxicity in rats via upregulation of the activities of antioxidants, and suppression of inflammatory and apoptotic biomarkers	*In vivo*	(Zhang et al., 2019a,b)
	Ginsenoside Re	reduced apoptosis of cardiomyocytes caused by ischemia reperfusion injury, attenuated isoproterenol-induced myocardial ischemic injury by regulating the antioxidation function in cardiomyocytes, improved isoproterenol-induced myocardial fbrosis and heart failure by regulation of the transforming growth factor beta 1 (TGF-1)/Smad3 pathway	*In vivo*	(Sun et al., 2019a,b)
	Ginsenoside CK	reduced myocardial ischemia reperfusion in mice with myocardial infarction area and Ca^{2+} induced mitochondrial distention	*In vivo*	(Liu and Liu, 2002), (Wang et al., 2018), (Wang et al., 2019)

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Table 10 (*continued*)

Biological Activities	Name	Description	*In vivo*/*In vitro*	Reference
	Ginsenoside RK_3	inhibited protein kinase B/Nuclear respiratory factor-2/heme oxygenase-1 (AKT/nrf-2/ho-1) and MAPK pathways, and prevented the injury and apoptosis of H9C2 cells	*In vivo*	Tsutsumi et al. (2011)
	Ginsenoside Rh_1	inhibited the activities of L and T Ca^{2+} channels, reducing their opening probability and opening time	*In vitro*	Sun et al. (2013)
	Notoginsenoside R_1	inhibited TNF-α-induced Plasminogen activator inhibitor-1(PAI-1) overexpression in HASMCs by suppressing ERK and PKB signaling pathways	*In vitro*	Zhao et al. (2011)
	Notoinsenoside Rg_1	attenuated pulmonary vasoconstriction which may lead to HHPV through reducing the expression of ERK1/2.	*In vitro*	Zhang and Wang (2006)
	PQS	inhibited excessive endoplasmic reticulum stress (ERS)	*In vivo* and *In vitro*	Zhang et al. (2016)
	PNS	activated PI3K/Akt signaling pathway	*In vivo* and *In vitro*	(Wang et al., 2012a,b)
	PNS	inhibited the changes of NF-κB, reduced the expression of intercellular adhesion factor and neutrophil infiltration	*In vivo*	Chen et al. (2011)
	PNS	increased miR-29c expression and decreased the expression of miR-29c target genes in ISO-challenged mouse hearts	*In vivo*	Tang et al. (2002)
	PNS	reduced the duration of arrhythmias induced by aconitine, $BaCl_2$, and $Cacl_2$-Ach	*In vivo*	Liu et al. (2017)
Antiobesit ability	Chikusetsusaponins III	inhibited pancreatic lipase activity and delaied intestinal dietary absorption	*In vivo*	Leng et al. (2001)
	Chikusetsusaponins IV	inhibited pancreatic lipase activity and delaied intestinal dietary absorption	*In vivo*	Han et al. (2005)
	28-deglucosyl-chikusetsusaponins IV	inhibited pancreatic lipase activity and delaied intestinal dietary absorption	*In vivo*	Han et al. (2005)
	28-deglucosyl-chikusetsusaponins V	inhibited pancreatic lipase activity and delaied intestinal dietary absorption	*In vivo*	Han et al. (2005)
	Ginsenoside Rh_2	induced carnitine palmitoyltransferase-1 (CPT-1) and uncoupling protein-2 (UCP-2)	*In vivo* and *In vitro*	Han et al. (2005)
	Ginsenoside Rc	inhibited pancreatic lipase activity by 100% at 0.5 mg/mL	*In vitro*	Hwang et al. (2007)
	Ginsenoside Rb_1	inhibited pancreatic lipase activity by 96% at 0.5 mg/mL	*In vitro*	(Zhang et al., 2002a,b)
	Ginsenoside Rb_2	attenuated insulin resistance in 3T3-L1 adipocytes, reduces fat mass, and improves insulin sensitivity in high fat diet-obesity mice	*In vivo*	(Zhang et al., 2002a,b)
	Notoginsenoside Fe	through the activation of energysensing neurons in the hypothalamus	*In vivo* and *In vitro*	Dai et al. (2018)
	Ginsenoside Rh_1	inhibited adipocyte differentiation and inflammation	*In vitro*	Li et al. (2019a,b,c)
	Ginsenoside Rg_1	induced AMPK activation, inhibiting lipogenesis, and decreasing intracellular lipid content, adipocyte size, and adipose weight	*In vivo*	Gu et al. (2013)
	PNS	inhibited pancreatic lipase activity by 35.2% at 0.5 mg/mL	*In vitro*	(Liu et al., 2018a,b,c,d)
	PNS	raised transcriptional activation of the liver X receptors (LXR) α gene promoter, reduced NF-κB DNA binding activity	*In vivo* and *In vitro*	(Zhang et al., 2002a,b)
	PQS	inhibited pancreatic lipase activity by 90% at 0.5 mg/mL	*In vitro*	Fan et al. (2012)
	Ginsenoside Rb_1	promoted adipocyte differentiation, inhibited basal lipolysis, decreased fasting blood glucose level (FBGL), improved GT	*In vitro*	(Zhang et al., 2002a,b)
	Ginsenoside Rb_1	improved glucose tolerance (GT), decreased body weight incremental percentage and FBGL	*In vivo*	(Shang et al., 2007), (Park, 2008)
	Ginsenoside Re	improved GT, decreased body weight incremental percentage and FBGL, decreased gluconeogenesis, activated AMPK, inhibitd glycogenolysis, decreased lipogenesis, decreased insulin resistance	*In vivo*	Yang et al. (2010)
	Ginsenoside Rc	induced Reactive oxygen species (ROS) generation, activate AMPK and p38 MAPK	*In vitro*	Yang et al. (2010)
	Ginsenoside Rg_1	protected retinal pigment epithelium (ARPE)-19 cells against HG-induced injury through up-regulating miR-26a, along with inhibition of the ERK and Wnt/β-catenin pathways, relieved the insulin-induced insulin resistance in HepG2 cells, decreased gluconeogenesis, activated AMPK, enhanced of insulin binding in liver membranes	*In vitro*	(Lee et al., 2010a,b)
	Ginsenoside M	showed dose-dependent hypoglycemic actions 7 h after injection and still exhibited significant actions even after 24 h. Z.p. dosing of the main glycan, panaxan N	*In vivo*	(Shi et al., 2019a,b), (Park, 2008), (Tchilian, 1991)
	Ginsenoside N	showed dose-dependent hypoglycemic actions 7 h after injection and still exhibited significant actions even after 24 h. Z.p. dosing of the main glycan, panaxan N	*In vivo*	Konno (1987)
	Ginsenoside O	showed dose-dependent hypoglycemic actions 7 h after injection and still exhibited significant actions even after 24 h. Z.p. dosing of the main glycan, panaxan N	*In vivo*	Konno (1987)

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Table 10 (*continued*)

Biological Activities	Name	Description	*In vivo*/*In vitro*	Reference
	Ginsenoside P	showed dose-dependent hypoglycemic actions 7 h after injection and still exhibited significant actions even after 24 h. Z.p. dosing of the main glycan, panaxan N	*In vivo*	Konno (1987)
	Compound K	protected islet B cells	*In vivo*	Konno (1987)
	PNS	improved glucose homeostasis, increased insulin sensitivity, improved leptin sensitivity, improved glucose uptake, improved insulin-and leptin sensitivity	*In vivo*	Han et al. (2007)
	AF	*in vitro*, AF and AFG inhibited the IC_{50} of intestinal *α*-glycosidase and *α*-amylase in rats by 6.40 and 6.20 mM, and *in vivo*, the IC_{50} inhibited pancreatic *α*-amylase by 36.30 and 37.60 mM	*In vitro and In vivo*	Yang et al. (2010)
	AFG	*in vitro*, AF and AFG inhibited the IC_{50} of intestinal *α*-glycosidase and *α*-amylase in rats by 6.40 and 6.20 mM, and *in vivo*, the IC_{50} inhibited pancreatic *α*-amylase by 36.30 and 37.60 mM	*In vitro and In vivo*	Ha et al. (2011)
	PA	reduced blood glucose and improve glucose tolerance in patients with 2 diabetes mellitus	*In vivo*	Ha et al. (2011)
	TGCG	lowered the fasting blood glucose levels in ob/ob mice (153 ± 16 mg/dL vs 203 ± 9.8 mg/dL, $P < 0.01$, compared to vehicle-treated group)	*In vivo*	Yoshinari and Igarashi (2011)
Hemostatic activity	Dencichine (*β*-N-oxalyl-L-*α*,*β*-diaminopropionic acid)	promoted platelet aggregation induced by low dose trap and ADP	*In vivo*	(Xie et al., 2005)
	Notoginsenoside Ft_1	activated the P2Y12 receptor signaling pathway to promote adp-induced platelet aggregation	*In vitro*	(Li et al., 2018b)
	PNS	lower bleeding times (9.60 ± 1.50 min) than the control group (19.23 ± 4.09 min, $P < 0.001$) or the placebo group (15.18 ± 2.24 min, $P < 0.001$)	*In vivo*	(Gao et al., 2014a,b)
Antithrombotic activity	Ginsenoside Rb_1	promoted the proliferation of erythropoietic progenitor cells	*In vitro*	(White et al., 2000)
	Ginsenoside Rb_1	extended the time from the onset of irradiation to the onset of the clot and reduced the size of the clot	*In vivo*	Zheng et al. (2003a)
	Ginsenoside Rb_3	inhibitd platelet activation and aggregation	*In vitro*	Fang et al. (2008)
	Ginsenoside Rg_1	extended the time from the onset of irradiation to the onset of the clot and reduced the size of the clot	*In vivo*	Cui et al. (2006)
	Ginsenoside Rg_1	promoted the proliferation of human bone marrow granulocytes, inhibitd platelet receptor-activated calcium channels and lowers platelets	*In vitro*	Fang et al. (2008)
	Ginsenoside Rg_2	increased the content of cAMP in platelets	*In vivo*	(Zheng et al., 2003a), (Liu et al., 2007)
	20(*S*)-ginsenoside Rg_3	the affinity of fibrinogen and fibronectin with *α*IIb/*β*3 was inhibited by G-Rg_3 via cyclic AMP-dependent vasodilator-stimulated phosphoprotein (VASP) Ser157 phosphorylation	*In vitro*	Zhang and Chen (1984)
	Ginsenoside Rk_1	inhibited cyclooxygenase activity to reduce thromboxane B 2 (TXB2) levels and reduce 12-HETE levels	*In vitro*	Kwon (2018)
	Chikusetsusaponins Iva	With Gp II b/III a receptor inhibition activity	*In vitro*	(Lee et al., 2010a,b)
	Araloside A	With Gp II b/III a receptor inhibition activity	*In vitro*	Nguyen et al. (2011)
	Chikusetsusaponins Ib	With Gp II b/III a receptor inhibition activity	*In vitro*	Nguyen et al. (2011)
	Ginsenoside Re	proliferated hematopoietic progenitor cells	*In vitro*	Nguyen et al. (2011)
	Ginsenoside Ro	inhibited the formation of 1,2-hydroxy-5,8,10-heptadecatrienoic acid and thromboxane B_2	*In vitro*	Zheng et al. (2003b)
	Ginsenoside R_1	proliferated hematopoietic progenitor cells	*In vitro*	Kuo et al. (1990)
	Notoginsenoside R_1	improved microcirculation and moderately prolonged coagulation time	*In vivo*	Zheng et al. (2003b)
	Notoginsenoside Rd	improved microcirculation and moderately prolonged coagulation time	*In vivo*	(Fang et al., 2008), (Liu et al., 2007)
	Notoginsenoside Rh_1	binded to human platelets	*In vitro*	Liu et al. (2007)
	Notoginsenoside Rf_1	binded to human platelets	*In vitro*	Liu et al. (2012)
	PNS	increased coronary heart disease patients serum NO, endothelin levels drop, reduced vascular endothelin II (Ang II) induced endothelial cell apoptosis rate and Fas and the expression of Bcl-2	*In vitro*	Liu et al. (2012)
	PJSM	accelerated the recovery of red blood cells (RBC), white blood cells (WBC) and haemoglobin (HGB) levels in blood deficiency model mice	*In vitro*	Zheng et al. (2003b)
	PJPS	accelerated the recovery of RBC, WBC and HGB levels in blood deficiency model mice	*In vitro*	Zhang (2015)
	Ocotillol	inhibited the formation of arteriovenous bypass thrombosis in rats	*In vitro*	Zhang (2015)
	Vidarabine	binded to human platelets	*In vitro*	María et al. (2015)
	Guanosine	binded to human platelets	*In vitro*	Liu et al. (2012)
Anti-atherosclerosis activity	Ginsenoside Rd	inhibited the formation of foam cells *in vitro* and reduced atherosclerotic plaques	*In vivo*	Liu et al. (2012)
	Ginsenoside Rh_2	reduced serum il-6 level and inhibited the expression of timp-1 and Vascular endothelial growth factor (VEGF) in aorta of rats	*In vivo*	Li et al. (2011)
	PNS	inhibited the effect of high-fat animal serum on smooth muscle cell (SMCs) and significantly inhibited the occurrence of atherosclerosis and the formation of aortic intima plaque in experimental animals	*In vivo*	Zhang (2015)

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Table 10 (*continued*)

Biological Activities	Name	Description	*In vivo*/*In vitro*	Reference
	PNS	maintained vascular smooth muscle cell (VSMCs) contractile phenotype	*In vitro*	Zhang (2015)
	PNS	inhibited NF-κB DNA binding activity and reduced secretion of IL-6 and MCP-1 in LPS-stimulated THP-1 macrophages	*In vivo* and *In vitro*	Liu et al. (2015)
Haemolytic Activity	Notoginsenoside K	increased the concanavalin A (Con A)-, lipopolysaccharide (LPS)-, and OVA-induced splenocyte proliferation in OVA-immunized mice ($P < 0.05$, $P < 0.01$, or $P < 0.001$)	*In vivo*	Fan et al. (2012)
	Pseudoginsenoside-F_{11}	activated cholinergic transmission to antagonize morphine-induced memory deficits, activated dopaminergic transmission to inhibit morphine-induced conditioned place preference, regulated adenylyl cyclase activity toeliminate morphine-induced tolerance development.	*In vitro*	Qin et al. (2006)
	PNS	haemolytic percents of PNS-treated red blood cell were 11.6% and 3.6% at concentrations of 500 and 250 mg/L, respectively	*In vitro*	(Han et al., 2018a,b)
Autotoxicity	Ginsenoside Rg_1	excessive accumulation of reactive oxygen species (ROS) can be induced to cause oxidative damage to cells, lead to root tip cell necrosis, and finally inhibit root growth	*In vitro*	Qin et al. (2006)
	Ginsenoside Rh_2	selectively inhibited the activity of osteoclast growth factor secreted by RAW264.7 macrophages *in vitro*	*In vitro*	(Luo et al., 2019a,b)
Cytotoxicity activity	Panaxytriol	inhibitd cellular respiration and disrupts cellular energy balance in Breast M25-SE	*In vitro*	Liu et al. (2009)
Allelopathic inhibitory activity	Ginsenoside Rg_1	MSI3 = −0.374, which inhibited seedling height, principal root length, soluble protein content, soluble sugar content and CAT activity	*In vitro*	Matsunaga et al. (1995)
	Ginsenoside R_1	MSI3-0.221, which inhibited seedling height, principal root length, soluble protein content, soluble sugar content and CAT activity	*In vitro*	Ma et al. (2016)
	PNS	MSI3 = −0.426, which inhibited seedling height, principal root length, soluble protein content, soluble sugar content and CAT activity	*In vitro*	Ma et al. (2016)
Anti-muscular atrophy activity	Ginsenoside Rg_1	inhibited the decrease of C2C12 cell activity and apoptosis induced by serum-free culture, inhibited the expression of two muscle-specific ubiquitin ligase E3	*In vitro*	Ma et al. (2016)
	Ginsenoside Rb_1	increased the expression of bcl-2 protein, decreased the expression of Bax protein, and increased the ratio of bcl-2/Bax reduces the apoptosis of hypoxia-induced nerve cells	*In vitro*	Li (2016)
	Ginsenoside Rb_2	upregulated myotube growth and myogenic differentiation through activating Akt/mammalian target of rapamycin signaling and inducing myogenic conversion of fibroblasts	*In vivo* and *In vitro*	Nie et al. (2004)
	20(*S*)-ginsenoside Rg_3	inhibited growth and survival of GBC cells via activation of the p53 pathway	*In vivo* and *In vitro*	Go et al. (2019)
	Ginsenoside Rg_3	attenuated TNF-*α*-induced NPCs impairment via blocking the NF-κB signaling pathway	*In vitro*	Dong et al. (2015)
	PNS	attenuated oxidative damage through oxidative stress- and mitochondrial function-related signaling pathways	*In vivo*	Chen et al. (2019)
	PQS	increased cardiomyocyte viability and decreased cardiomyocyte apoptosis induced by TG	*In vivo* and *In vitro*	Zhou et al. (2018)
Anti-bacterial activity	Oleanolic acid	displayed 98.75% and 97.26% feeding-deterrence at 3000 ppm concentration	*In vivo*	(Ma et al., 2015)
Antiviral activity	Ginsenoside Rb_2	potentiated nonspecific resistance against severe infection of reovirus (RV) in newborn mice	*In vivo*	Shukla (1997)
	20(*S*)-Ginsenoside Rg_3	potentiated nonspecific resistance against severe infection of RV in newborn mice	*In vivo*	Yang et al. (2018)
	Ginsenoside Rg_3	promoted CO cell proliferation, promotes CO cell immune activities, and thereby enhances the resistance of CO to grass carp tissues after reovirus (GCRV) infection	*In vitro*	Yang et al. (2018)
Anti-osteoporosis activity	LPNS	promoted the differentiation of bone marrow mesenchymal stem cells and mononuclear cells into osteoblasts and osteoclasts, respectively, but had no effect on osteoclast activation	*In vivo* and *In vitro*	Dai (2018)
Anti - proliferation activity	Ginsenoside Re	IC_{50} = 0.489 mg/mL, The highest cell proliferation inhibition rates were 71% at 1 mg/mL	*In vitro*	Du et al. (2015)
	Ginsenoside Rg_1	IC_{50} = 0.653 mg/mL, The highest cell proliferation inhibition rates were 59.4% at 1 mg/mL	*In vitro*	Yao et al. (2014)
	Ginsenoside Rb_1	IC_{50} = 0.553 mg/mL, The highest cell proliferation inhibition rates were 68% at 1 mg/mL	*In vitro*	Yao et al. (2014)
Anti-allergic activity	Ginsenoside Rf	inhibited the release of *β*-aminohexidase, IC_{50} = 0.08 mmo/L	*In vivo*	Yao et al. (2014)
	Ginsenoside Rh_2	inhibited the release of *β*-aminohexidase, IC_{50} = 0.03 mmo/L	*In vivo*	Bae et al. (2006)
	Ginsenoside Rg_3	inhibited the release of *β*-aminohexidase	*In vivo*	Bae et al. (2006)
	Ginsenoside Re	reduced blood glucose, total cholesterol and triglyceride levels	*In vivo*	Bae et al. (2006)
	PNS	caused a decrease in platelet activator, Calcium channel blockers, Blocked the norepinephrine induced internal flow	*In vitro*	Cho et al. (2006)

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Table 10 (*continued*)

Biological Activities	Name	Description	*In vivo*/*In vitro*	Reference
Endothelial cell protective activity	Ginsenoside Rb_1	inhibited TGF-β related signal transduction and protected human umbilical vein endothelial cells (HUVECs), with Rb_1 = 80 mg/L	*In vitro*	Chen et al. (2004)
Anti-cell migration activity	Ginseng Pectin WGPA	inhibition was correlated with GalA content (HG domain) and Rha content (rg-i domain)	*In vitro*	Xie et al. (2008)
Anti-apoptosis activity	Ginsenoside Rg_2	down-regulated the expression of pro-apoptotic factors BAX and P53	*In vivo*	(Fan et al., 2010)
Anti-radiation activity	Notoginsenoside R_1	R_1 with a mass concentration of more than 50 μm g/mL decreased cell proliferation activity, hydroxyproline and total collagen secretion, and increased mmp-1 protein secretion in fibroblasts	*In vitro*	Zhang et al. (2008)
	Ginseng Pectin APG	blocked the p53-dependent pathway and the mitochondrial/caspase pathway, and finally protected the small intestinal crypt cells and prevented villi injury	*In vivo* and *In vitro*	Xie et al. (2011)
Myelosuppressive activity	Compound K	controlled apoptosis and promote cells enter the normal cell cycle by bcl-2/bax signaling pathway and/MAP kinase–ERK kinase/extracellular-signal-regulated kinase (MEK/ERK) signaling pathway	*In vitro*	Park et al. (2011)
Anti-acne activity	RGEF	oxidized sebum contents and redness of the skin were reduced, and symptoms of the early to middle stage of acne were effectively improved	*In vivo*	Han et al. (2019)
Gastrointestinal protective activity	PNS	increased vascular endothelial growth factor A (VEGFA) expression	*In vitro*	Hou et al. (2019)
Anti-complement activity	Ginsenoside Re	ginseng saponins imposed their effects on complements C1q, C_2, C_3, C_4, and C_5, however, maybe not C_9	*In vitro*	Zhu et al. (2018)
	Ginsenoside Rf	ginseng saponins imposed their effects on complements C1q, C_2, C_3, C_4, and C_5, however, maybe not C_9	*In vitro*	Gao et al. (2013)
	Ginsenoside Rg_1	ginseng saponins imposed their effects on complements C1q, C_2, C_3, C_4, and C_5, however, maybe not C_9	*In vitro*	Gao et al. (2013)
Anti-complement activity	Ginsenoside Rb_3	ginseng saponins imposed their effects on complements C1q, C_2, C_3, C_4, and C_5, however, maybe not C_9	*In vitro*	Gao et al. (2013)
	Notoginseng R_4	ginseng saponins imposed their effects on complements C1q, C_2, C_3, C_4, and C_5, however, maybe not C_9	*In vitro*	Gao et al. (2013)
	Acidic polysaccharides GL-NIa	through the alternative complement pathway	*In vitro*	Gao et al. (2013)
	Acidic polysaccharides GL-NIb	through the alternative complement pathway	*In vitro*	Gao et al. (2013)
	Acidic polysaccharides GL-AIa	through the alternative complement pathway	*In vitro*	Gao et al. (1991)
	Acidic polysaccharides GL-AIb	through the alternative complement pathway	*In vitro*	Gao et al. (1991)
Anti-hypertensive activity	Ginsenoside Rb_1	increased endothelial-dependent vessel dilatation through the activation of NO by modulating the PI3K/Akt/eNOS pathway and L-arginine transport in endothelial cells	*In vivo* and *In vitro*	Gao et al. (1991)
	Ginsenoside Rg_1	increased endothelial-dependent vessel dilatation through the activation of NO by modulating the PI3K/Akt/eNOS pathway and L-arginine transport in endothelial cells	*In vivo* and *In vitro*	Pan et al. (2012)
Anti-hepatitic Activity	Ginsenoside Ro	inhibited GalN- and CC14-induced cytotoxicity in primary cultured rat hepatocytes	*In vitro*	Pan et al. (2012)
Myelosuppressive activity	Ginsenoside Re	regulated the levels of cytokines, promoting cells enter the normal cell cycle, regulated the balance of bcl-2/bax, and inhibited the expression of caspase-3	*In vivo* and *In vitro*	Matsuda et al. (1991)
	Ginsenoside RK_3	regulated the levels of cytokines, promoted cells enter the normal cell cycle, regulated the balance of bcl-2/bax, and inhibited the expression of caspase-3	*In vivo* and *In vitro*	(Han et al., 2018a,b)
	PNS	promoted DO and mediated by TGF-β1 signaling pathway	*In vivo* and *In vitro*	(Han et al., 2018a,b)
	PNS	inhibited the accumulation of collagen and then inhibit hypertrophic scarring through reducing CTGF expression and increasing MMP1 expression	*In vivo*	Guo et al. (2017)
Analgesic activity	Ginsenoside Rc	suppressed pain induced by chemical stimulation through the non-opioid system	*In vivo*	Zhi (2017)
	Ginsenoside Rd	suppressed pain induced by chemical stimulation through the non-opioid system	*In vivo*	(Lee et al., 2015a,b,c)
	Ginsenoside Re	suppressed pain induced by chemical stimulation through the non-opioid system	*In vivo*	(Lee et al., 2015a,b,c)
Sedation activity	Ginsenoside Rb_1	reduced the amount of synaptic glutamate inhibits the central nervous system	*In vitro*	(Lee et al., 2015a,b,c)
	Notoginsenoside R_1	R_1 of 100 mg kg^{-1} reduced the voluntary activity induced by caffeine in mice	*In vivo*	Cicero et al. (2003)
	PNS	reduced the amount of synaptic glutamate inhibits the central nervous system	*In vitro*	Cui et al. (2009)
Anti-depressant activity	Ginsenoside Rb_1	mediated by central neurotransmitters of serotonergic, noradrenergic and dopaminergic systems, antagonized by 5-HT2AR antagonists (Ritanerin)	*In vivo*	(Ma et al., 1999)
	Ginsenoside Rg_1	reduced CMS-induced increasement of corticosterone levels in serum, increased Chronic unpredictable mild stress (CUMS) -induced CREB phosphorylation in the amygdala of the brain	*In vivo*	Wang et al. (2017)

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Table 10 (*continued*)

Biological Activities	Name	Description	*In vivo*/*In vitro*	Reference
	Ginsenoside Rg_3	reduced IL-6 and TNF-α in plasma and the expression of indoleamine 2,3-dioxygenase (IDO) in brain	*In vivo*	Liu et al. (2016)
	Ginsenoside Rg_5	increased expression of brain neurotrophic derived factor (BNDF)	*In vivo*	Kang et al. (2017)
	Ginsenoside Rh_2	reduced turnover of tryptophan and 5-HT in hippocampal tissue	*In vivo*	Xu and Gao (2017)
	Ginsenoside K	the antidepressant effects of Rb_1 and the metabolite ginsenoside K may be antagonized by 5-HT2AR antagonists (Ritanerin), indicating that Rb_1 has a similar 5-HT transmitter activation effect	*In vivo*	You et al. (2017)
	Ginsenoside Re	regulated the secretion of corticosterone from the Hypothalamic-pituitary-adrenal (HPA) axis	*In vivo*	Carr et al. (2006)
	Ginseng Pectin WGPA	WGPA of 100 mg/kg can significantly increase the social interaction of mice and reduce the aggressive behavior of mice	*In vivo*	Lee (2018)
	PNS	reduced Immobility time in forced swim test (FST) and tail suspension test (TST), increased sucrose intake in sucrose preference test	*In vivo* and *In vitro*	Wang et al. (2014)
	PNS	raised level of animal activity, modulated of brain monoamine neurotransmitters and intracellular Ca^{2+} concentration	*In vivo*	(Wang et al., 2016a,b,c,d)
	GTS	reduced mRNA of IL-1β, IL-6, TNF-α and IDO	*In vivo*	Carr et al. (2006)
Anti-aging activity	Ginsenoside Rb_1	increased the activity of catalase and glutathione peroxidase	*In vivo*	Kang et al. (2017)
	Compound K	CK did not regulate tyrosinase activity and melanin secretion, but increased melanin content in B16F10 cells was observed	*In vitro*	Dai et al. (2018)
	Pg-C-EE	suppressed ROS generation induced by H_2O_2 and undergoing photo therapy (UVB)	*In vivo*	Kim et al. (2018)
Anti-fatigue activity	(24*R*)-Pseudo Ginsenoside HQ	upregulated the innate and adaptive immune response in cyclophosphamide (CTX), induced-immunocompromised mice	*In vitro*	Lee (2018)
	(24*S*)-Pseudo Ginsenoside HQ	upregulated the innate and adaptive immune response in cyclophosphamide (CTX), induced-immunocompromised mice	*In vitro*	(Qi et al., 2019a,b)
	Ginsenoside Rg_1	increased SOD activity, mitochondrial membrane potential and free calcium content in rat skeletal muscle, but decreased MDA content	*In vivo*	(Qi et al., 2019a,b)
	Ginsenoside Rb_1	the intracellular calcium overload was reduced by inhibiting the intracellular calcium flow to protect ischemic nerve cells, and the protective effect was concentration-dependent, reaching the maximum at 60 mol/L	*In vivo*	Yichong et al. (2010)
	Ginsenoside Rb_3	the intracellular calcium overload was reduced by inhibiting the intracellular calcium flow to protect ischemic nerve cells, and the protective effect was concentration-dependent, reaching the maximum at 60 mol/L	*In vivo*	Zhang et al. (2005)
	WGP	the FST-induced reduction in glucose and glutathione peroxidase and increase in creatine phosphokinase, lactic dehydrogenase and malondialdehyde levels	*In vivo*	Zhang et al. (2004)
	Ginseng Pectin WGPA	have therapeutic effects on chronic fatigue syndrome	*In vivo*	(Wang et al., 2010a,b,c)
Antifibrotic activity	Ginsenoside Rd	inhibited CD36 protein expression and reduced lipid intake to inhibit activated HSCs proliferation and COL1A1 protein expression	*In vitro*	Wang et al. (2014)
	Ginsenoside Rg_1	down-regulated the expression of Platelet-derived growth facto (PDGF) receptor-β by reducing the NF-κB activity	*In vivo* and *In vitro*	(Li et al., 2016)
	Ginsenoside Rg_1	restrained the process of EMT maybe via suppressing the expression of P-ERK1/2	*In vitro*	Geng et al. (2010)
	Ginsenoside Rg_1	reduced the deposition of collagen in liver tissue and improved the degree of liver fibrosis	*In vivo*	Xie et al. (2009)
	PNS	reduced the deposition of collagen in liver tissue and improved the degree of liver fibrosis, inhibited on the NF-κB signaling pathway	*In vivo*	Dong et al. (2012)
	PNS	alleviated liver damage and reduced the formation of fibrous septa	*In vitro*	(Zhang et al., 2018a,b)
Anti-vascular aging activity	Ginsenoside Rg_1	reduced p16INK4a/Rb and p53-p21Cip1/Waf1 signaling pathways	*In vitro*	Hui et al. (2016)
Anti-vascular aging activity	Notoginsenoside R_1	via the activation of the Vascular endothelial growth factor (VEGF)-KDR/Flk-1 and phosphatidylinositol-3 kinase (PI3K)-Akt-eNOS signaling pathways	*In vivo* and *In vitro*	(Gao et al., 2014a,b)
	PNS	reduced expression of Proliferating cell nuclear antigen (PCNA), reduced cyclin E, cyclin D1, fibronect, and MMP-9	*In vivo*	(Gao et al., 2014a,b)
	PNS	reduced cell cycle-related factors and ERK signal transduction, raised p53, Bax, and caspase-3 expressions, reduced Bcl-2 expression, protected ECs and in inhibiting platelet adhesion to injured ECs, and the regulation of COX pathway in both ECs and platelets	*In vitro*	Wu et al. (2010)
	PNS	VEGF-KDR/Flk-1and PI3K-Akt-eNOS signaling pathways	*In vivo* and *In vitro*	(Hang, 2012), (Xu et al., 2011), (Wang et al., 2016a,b,c,d)

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Table 10 (*continued*)

Biological Activities	Name	Description	*In vivo*/*In vitro*	Reference
				(Hong and Wang, 2009)

PPD: Protopanaxadiol; PPT: protopanaxatriol; PNS: *Panax notoginseng* saponin; PQS: *Panax quinquefolius* saponin; PJSM: The total saponins of *Panax japonicus*; PJPS: The crude polysaccharides of *Panax japonicus*; G-Rh2-B2: derivative B2 of ginsenoside Rh2; PPQN: neutral polysaccharide from *Panax quinquefolius*; PNFS: *Panax notoginseng* flower saponins; SLPF: stem and leaf of *Panax notoginseng* flavonoid; GOP: Ginseng oligopeptides; WGP: water-soluble ginseng polysaccharide; TSPJ: Total Saponins of *Panax japonicus*; RG-CW-EZ-CP; TGCG: total ginsenosides in Chinese ginseng; RGEF: hydrophobic fraction of red ginseng ethanol extract; WGPA: acidic polysaccharide of ginseng; Pg-C-EE: ethanolic extract of *P. ginseng* berry calyx; AF: Arginyl-fructose; AFG: arginyl-fructosyl-glucose; PA: Pyroglutamic acid; GTS: ginseng total saponin); LPNS: Leaves of *Panax notoginseng* saponin.

6.4. Neuroprotective activity

With the increase of population aging and social life pressure, people are more and more exposed to the risk of nervous system diseases. Common neurological disorders include Alzheimer's disease (AD), Parkinson's disease, epilepsy and depression. Ginsenosides play an increasingly important role in the treatment of nervous system diseases, especially in the central nervous system. Several mechanisms were identified to exhibit significantly neuroprotective activity including the elimination of free radicals to activate brain function, inhibition of oxidative stress and neuroinflammation, the lower levels of toxins-induced apoptosis and regulation of N-methyl-Daspartate receptor channel activity (González-Burgos et al., 2015). Fig. 4 showed the multiple possible neuroprotective mechanisms for extracts and ginsenosides from *Panax*. The abnormal increase of Ca^{2+} level was an important indicator of neurological disorders, which could increase the risk of epilepsy. Take ginsenosides as an example, total ginsenosides and ginsenoside Rg_3 (**18**) could restrain the increase of Ca^{2+} induced by Mg^{2+} (Kim and Rhim, 2004). Besides, studies showed that ginsenoside Rb_2 (**14**) had the potential to become an anticonvulsant drug (Lian et al., 2006). Pseudoginsenoside F_{11} (**225**) could also be a valuable option to slow down the process of neurodegenerative disease. Zhang et al. discovered that pseudoginsenoside F_{11} (**225**) had beneficial effects on the pathological changes of AD in senescence accelerated mouse P8 (SAMP8). The possible mechanisms for improving cognitive impairment act were inhibition of beta-site amyloid precursor protein cleaving enzyme 1 (BACE1) activity and enhancement of protein phosphatase 2A (PP2A) activity (Zhang et al., 2019a,b).

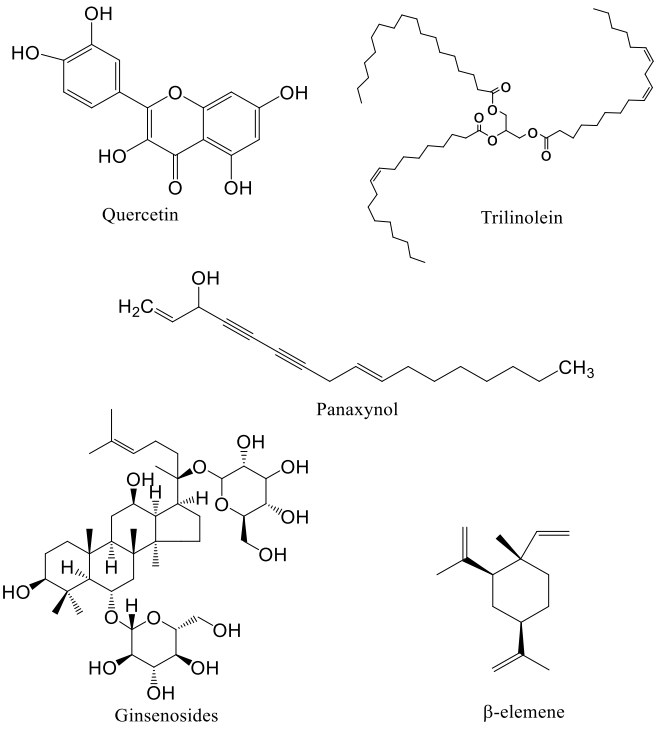

Fig. 3. The structure of some compounds with antineoplastic activity.

6.5. Immunoregulatory activity

Modern pharmacological studies showed that ginseng was the adaptogenic drug, which had bidirectional regulation to be conducive to the recovery and enhancement of body functions (Dou et al., 1999). Ginseng could also be considered as an immunomodulator, which played an immune regulatory role through many ways, such as immune organs (thymus, spleen), immune cells (macrophages, dendritic cells, natural killer cells, etc.) and cytokines (TNF-α, IFN-γ, IL-2, IL-6, IL-12, etc.) (Bai et al., 2019). Ginsenosides and ginseng polysaccharide were the main active components of *P. ginseng* with a wide range of applications in immune regulation (Wang and Wang, 2005). *P. ginseng* pectin

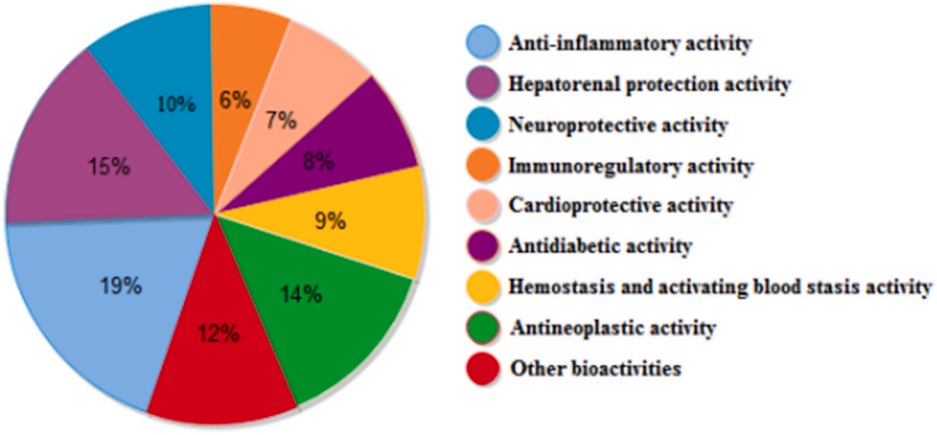

Fig. 2. The comparison of different biological activity related to chemical components reported from the genus *Panax*.

SB was isolated from *P. ginseng* root to have a bidirectional regulation effect on human monocyte THP-1, secreting cytokine interleukin-8 (IL-8) and IL-2 by mouse spleen cell (Tian et al., 2011a,b). Low concentrations of ginseng pectin promoted the secretion of immune cytokines, while high concentrations of ginseng pectin inhibited the process.

Similar immunologically active chemical components have also been found in other species of the *Panax*. In addition, Zhu et al. isolated and purified a galactoside (PPQ) from the roots of *P. quinquefolius* (Zhu et al., 2012). The results showed that PPQ might be expected as a potential antitumor drug with immunoregulatory activity. At a high dose of 400 mg/kg, the production of IL-2 and IFN-γ was significantly increased and the expression of IL-10 was decreased. Then, it regulated the secretion of Th1/Th2 cytokines to enhance immunity. *P. notoginseng* saponins (PNS) possessed immnologic adjuvant activities and enhanced the humoral and cellular immune responses to ovalbumin (OVA) in mice when administered with OVA (Sun et al., 2003).

6.6. Cardioprotective activity

As cardiovascular disease is becoming the major cause of mortality and the limitations of conventional drugs used in therapy, the development of new active substances from medicinal plants is needed in the current clinical and experimental research (Adegbola et al., 2017). Phenolic acids, saponins, flavonoids, alkaloids and other compounds in *Panax* have better pharmacological activities against myocardial ischemia. The research on cardiovascular diseases mainly focuses on the purified ginsenoside monomers from *P. ginseng* rather than the whole extracts. The most frequently studied ginsenosides are Rg_1 (97), Rb_1 (13), Rh_1 (100), Re (104) and Rd (17) (Kim, 2012). The extracts of *P. ginseng* can promote collateral circulation to alleviate myocardial ischemia symptoms. Ginseng stem leaf glucoside can resist chloroform-induced arrhythmias in mice and prevent aconitine-induced arrhythmias in mice (Tang et al., 2009). Ginsenoside Re (104) has a therapeutic effect on triggering ventricular arrhythmia. However, it can cause poisoning or side effects due to the strong tonic effect, if *P. ginseng* is misused or overused (Yang, 2009).

P. notoginseng is also an effective anti-angina medicine. It was quantitatively confirmed in Lei's study that water extracts of *P. notoginseng* improved the cardiovascular function in a dose-related manner (Lei et al., 2012). The research showed that *P. notoginseng* increased coronary blood flow and cardiac contractility without changing heart rate. In addition, Xu et al. showed that *P. quinquefolius* 20(*S*)-protopanaxadiol saponins (PQDS) had cardioprotective effects *in vivo* and *in vitro* (Xu et al., 2013). The mechanism might eliminate the lipid peroxidation products and enhance the function of the endogenous antioxidant enzymes.

6.7. Antidiabetic activity

Since the existing synthetic drugs are often accompanied by considerable side effects, natural hypoglycemic compounds may be effective and safe alternatives to the treatment of diabetes or currently used therapeutic enhancers (Coman et al., 2012). According to the research of Chen et al., *P. notoginseng* was one of the promising medicinal plants had great ability of antidiabetes and antiobesity. PNS and dammarane saponins were the main bioactive components in *P. notoginseng* (Chen et al., 2008). At present, the hypoglycemic and anti-obesity characteristics of PNS are widely reported. PNS had the antidiabetic and anti-obesity effects on KK-Ay mice with type 2 diabetes and its preventive effects on renal lesions (Uzayisenga et al., 2014; Tang et al., 2016). According to literature review, the main mechanisms of PNS exerting anti-diabetic activity were: (1) reducing glucose uptake, lipogenesis; (2) increasing glucose absorption; (3) reducing gluconeogenesis and inhibiting glycogenolysis; (4) increasing insulin sensitivity and reducing insulin resistance.

6.8. Hemostasis and activating blood stasis activity of P. notoginseng

The root of *P. notoginseng*, characterized by the presence of Rb_1 (**13**), Rd (**17**) and Rg_1 (**97**) levels, was described as a unique herb for invigorating the circulation of blood and hemostasis. Dencichine was a special amino acid isolated from the roots of *P. notoginseng*. It could shorten the bleeding time of mice and reduce activated partial thromboplastin time (APTT) and thrombin time (TT), while the concentration of fibrinogen (FIB) in plasma would increase in a dose-dependent manner. Meanwhile, studies showed that dencichine exerted hemostasis activity by regulating intracellular cAMP levels (Huang et al., 2014). Owing to the stability of dencichine was easily destroyed at high temperature, *P. notoginseng* should be used for hemostasis without heating. In addition, the active substances for hemostasis contained calcium ions and quercetin (Dong et al., 2003). Other studies found that PNS had great impact on blood-activating through anticoagulation and antiplatelet aggregation. It suggested that *P. notoginseng* had a dual-directional regulation of hemostasis and blood-activating (Liu et al., 2018a,b,c,d). Some studies also confirmed the dose-effect relationship of *P. notoginseng*. It was found that the small-dose application mainly showed the effect of hematuria, while with the increase of the dose of *P. notoginseng*, its blood-activating effect was enhanced (Yu et al., 2008). Otherwise, the study also confirmed the hematopoietic function of *P. notoginseng*. The main mechanism was that PNS could induce the synthesis of GATA 1 and GATA 2 transcriptional regulatory proteins in hematopoietic cells, thereby regulating the expression of genes related to hematopoietic cell proliferation and differentiation (Gao et al., 2004).

6.9. Other biological activities

In modern research, it was reported that the *Panax* had various other biological activities in addition to those listed above. A series of evidence also indicated that ginsenosides could alleviate the pain caused by toxic chemicals in experimental animals. For example, ginsenoside Rf (**102**) inhibited voltage-dependent Ca^{2+} channels and alleviated the pain reactions induced by a chemical stimulus (Mogil et al., 1998). Ginsenoside Rd (**17**) inhibited the transmission of pain by regulating the central signaling molecule PKCγ (Gao et al., 2017). The analgesic mechanisms of ginseng glycopeptides (GGT) might be related to the regulation of pro-inflammatory cytokines (IL-1, TNF-*α*) and the dynamic balance of anti-inflammatory cytokines (IL-2, IL-4) (Tian et al., 2018). The central analgesic activity of ginsenosides metabolite compound K (CK, **74**) was evaluated by the hot plate method. The results showed that CK could reduce the number of writhing caused by acetic acid and increase the pain threshold of carrageenan-induced inflammatory pain, suggesting that **74** has peripheral analgesic effects (Si, 2018).

In addition, the aboveground parts of *P. notoginseng* had the effect of restraining the central nervous system, which were characterized by sedation, stability and improvement of sleep. PNS and ginsenoside Rb_1 (**13**) had the coordination effects of with central depressant drugs. Although there was relatively limited researches about the toxicity of *P. notoginseng*, several phenomena had been proved that R_1 (**119**), Rg_1, Re, Rb_1, Rg_2 and Rd could inhibit the germination of *P. notoginseng* seeds and had obvious autotoxicity to root cells (Yang et al., 2015). The possible mechanism was that saponins inhibited the synthesis of intracellular antioxidants in the roots of *P. notoginseng*, leading to the excessive accumulation of oxygen free radicals. (Xu et al., 2015).

7. The application and development of *Panax* classical prescriptions in modern pharmacology

With validated safety and reliability, many classical prescriptions in TCMs have been used for thousands of years. This record of long-term clinical experience can provide a more reliable therapeutic basis than

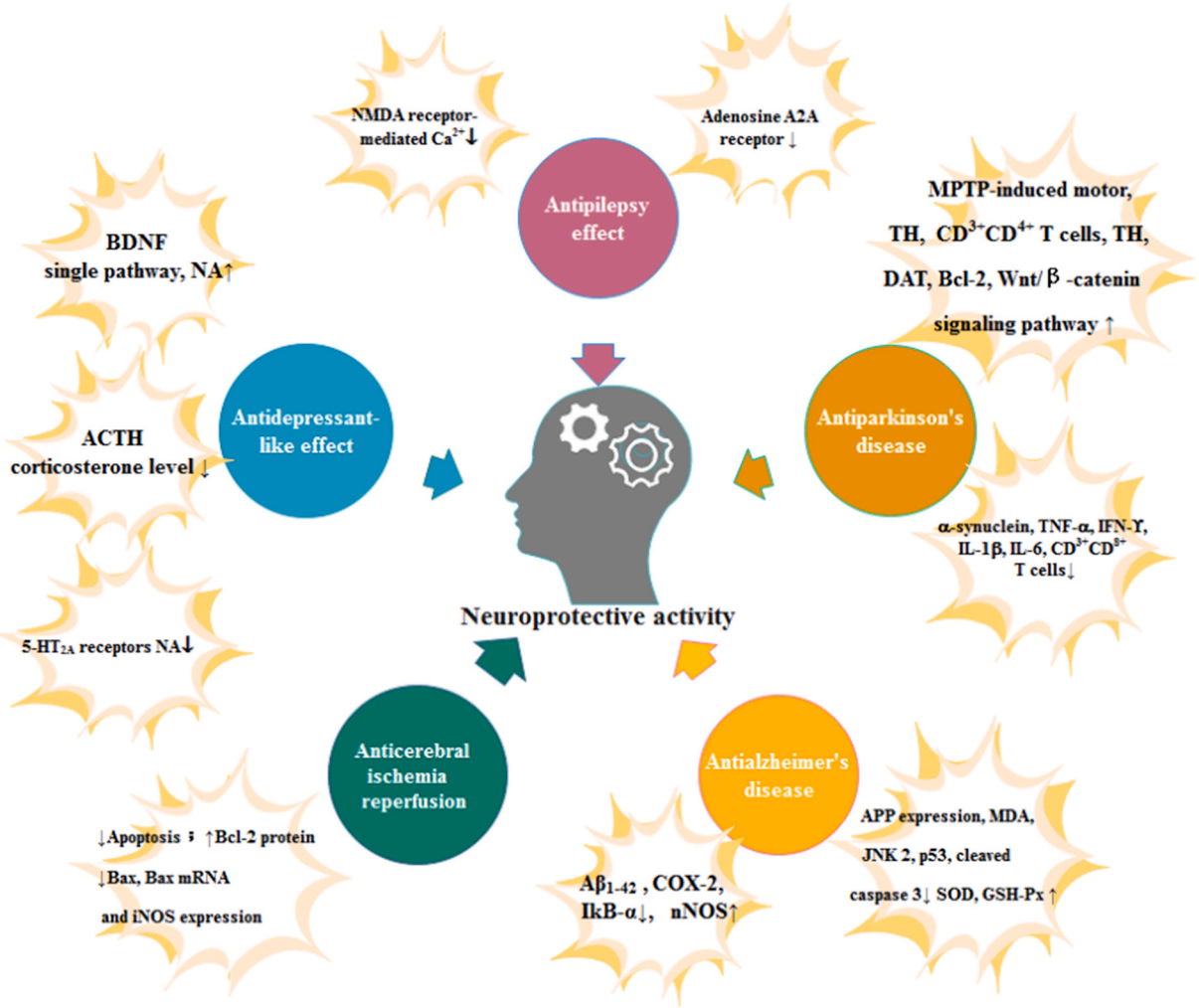

Fig. 4. Several neuroprotection mechanisms of ginsenosides.

the laboratory research with relatively limited time based on modern pharmaceutical standards. Moreover, studies suggested that many classical prescriptions were of good research value. For instance, bojungikki-tang (bu zhong yi qi tang in Chinese) was a prescription composed of eight TCMs including Ginseng Radix and Astragali Radix, which had been extensively used in China, Korea and Japan owing to the therapeutic efficacy on the weakness of spleen and stomach. Modern pharmacological studies showed that bojungikki-tang had antibacterial activity and exhibited positive outcomes in murine models of chronic fatigue syndrome (CFS) (Yan et al., 2002). Moreover, clinical studies confirmed the beneficial effects of bojungikki-tang on cancer-related fatigue (Jeong et al., 2010). Saengmaee-san (Sheng mai san in Chinese) was composed of three herbs (Ginseng Radix, Schisandrae Fructus and Ophiopogon Rhizome). It had been proved to exhibit antioxidant effect *in vitro* and *in vivo* and could treat heart failure and other cardiovascular diseases (Ichikawa and Konishi, 2002). In view of the synergetic and regulatory effects among various components, compound preparations may become a novel therapeutic choice to maintain the balance of Yin and Yang in human body in the future. Therefore, the modern investigation of the TCMs preparations in the ancient classical prescription may also be another potential method for drug development.

8. Conclusion and future perspectives

Genus *Panax*, globally-recognized tonic Chinese herbal medicines, have shown remarkable medicinal value such as in adjuvant therapy for tumor and resuscitation of hemorrhagic shock. In addition, their related compounds have been developed into new drugs. Hence, in the present study, we have comprehensively reviewed the components and bioactivities of the known metabolites from *Panax* and critically discussed the applications and issues of limited availability. To date, at least 748 chemical compounds from genus *Panax* have been isolated, such as saponins, flavonoids, polysaccharide, steroid and phenols. Saponins are considered as the major bioactive components, among which PPD (Rg_3, Rb_1, Rb_2, Rc and Rd) and PPT type ginsenosides (Rg_1, Re and Rg_5) are the most widely distributed in *Panax* plants. These ginsenosides can be recommended as the characteristic indicators for quality evaluation and identification. However, studies on flavonoids, polysaccharides and acetylenic alcohols are inadequate compared to those on saponin components. Moreover, limited attention has been paid to some species such as *P. sokpayensis*, *P. stipuleanatus*, etc. It is feasible to use bioactivity-oriented separation strategies to identify more bioactive components. Further phytochemical studies are suggested to focus on the species with less research or better efficacy chemical components. Additionally, further study concerning single chemical component of *Panax* is inseparable from the diverse chemical structure, significant biological activity and clinical application. The discovery of the bioactive molecules and multicomponent interactions could provide great significance to the clinical application of *Panax* plants. For instance, the binding of ginsenoside Rg_1 to ginsenoside Rb_1 triggers the loss suppression of oxidative stress and inflammatory factors via

ginsenoside Rg_1. Integrated medical research on ancient classical prescriptions is urgently required to perform detailed phytochemical, pharmacology and clinical studies on *Panax* classical prescriptions, aiming to establish modern medication guidelines.

To sum up, this review is designed to provide potential development value to analyze the metabolic mechanism of its important natural products and to investigate new drugs. It is necessary to perform further researches by involving the molecular and cellular mechanisms, toxic animal models and clinical applications. In addition, more comprehensive reviews concerning the structure-activity relationships of chemical components will shed new light on the development of an alternative strategy for quality control of *Panax* based on more rapid and accurate analysis techniques. Hopefully, these studies can maximize and optimize the potential of genus *Panax* as a promising Chinese herbal medicine, thereby further promoting global health.

Author contributions

LL, F-RX, and Y-ZW conceived the review. LL and F-RX collected literature and drafted manuscript. Y-ZW helped in manuscript revision. All authors read and approved the final manuscript for publication.

Declaration of competing interest

The authors declare that there is no conflict of interest regarding publication of this Paper.

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Appendix A. Supplementary data

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