REVIEW
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Advances in ethnopharmacology, phytochemistry and pharmacology of *Erigerontis Herba* and its Chinese medicine prescriptions

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Author contributions
Zhou Y conceived the study and wrote the original draft. Huang YY wrote the original draft. Shang DY searched the references and drawn pictures. Chen Y searched the references. Tian M organized the tables. Jin CY searched the references. Lu X searched the references. Yan BC supervised the study. Pang HQ conceived and designed the study.

Competing interests
The authors declare no conflicts of interest.

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Abbreviations
IS, ischemic stroke; MAPKs, mitogen-activated protein kinases; AD, Alzheimer's disease; NF-κB, nuclear factor-kappa B; ECM, extracellular matrix; EH, *Erigerontis Herba*; EBI, *Erigeron breviscapus* injection; DZSMC, Dengzhan Shengmai capsule; EMT, epithelial-mesenchymal transition; OA, osteoarthritis; IVDD, intervertebral disc degeneration; NPCs, nucleus pulposus cells; NLRP3, nucleotide-binding oligomerization domain, leucine-rich repeat and pyrin domain-containing 3; PTEN, phosphatase and tensin homolog deleted on chromosome ten; AKT, protein kinase B.

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Abstract
Erigerontis Herba (EH), the dried whole plant of *Erigeron breviscapus*, is well-known for circulating blood, activating meridians to alleviate pain, expelling wind, and clearing away cold. It has been extensively utilized in southern China for the treatment of stroke hemiplegia, chest stuffiness and pains, rheumatic arthralgia, headache, and toothache. This review focuses on the botany, ethnopharmacology, phytochemistry, pharmacology and toxicity of EH and its related prescriptions to offer new insights for prospective research of EH. Relevant information about EH was retrieved from ancient records and books, PubMed, China National Knowledge Infrastructure, *Chinese Pharmacopoeia*, Web of Science, Doctoral and Master's Theses, and various electronic databases. EH is a member of Compositae family and is mainly grown in southern China. Traditional Chinese medicine records that EH has the effects of circulating blood and removing blood stasis, expelling wind, and removing cold, as well as relieving rigidity of muscle and relieving pain. By now, nearly 200 ingredients have been characterized from EH, including flavonoids, caffeoyls, aromatic acids, coumarins, pentacyclic terpenoids, volatile oil and other compounds. EH extracts, EH related prescriptions (Dengzhan Xixin injection, Dengzhan Shengmai capsules, etc.) or compounds (scutellarin, scutellarein, etc.) possessed obvious therapeutic effects of ischemic stroke, cerebral hemorrhage, myocardial infarction, Alzheimer's disease, diabetes and its complications, gastric cancer, bone, and joint degenerative diseases. Scutellarin, the major active compound of EH, has been used as a quality marker. And no obvious toxicity of EH has been reported. According to its traditional applications, ethnopharmacology, phytochemistry, pharmacology, and toxicity, EH was applied as a valuable herb for clinical application in food and medicine fields. While several compounds have been shown to possess diverse biological activities, the underlying mechanisms of their actions remain elusive. To fully exploit the medicinal potential of EH, further studies on understanding the effective material basis and mechanisms are warranted.

Keywords: *Erigeron breviscapus*; traditional applications; ethnopharmacology; phytochemistry; pharmacology

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Highlights

This review summarizes the modern research progress on the botany, ethnopharmacology, phytochemistry, and pharmacological effects of *Erigerontis Herba* (EH). It is the first to elaborate on the pharmacological effects of EH on diabetes and its complications, as well as on degenerative bone and joint diseases. Additionally, it reviews the clinical applications of EH prescriptions, highlighting their advantages and potential side effects. This work lays a foundation for the further development and utilization of EH.

Medical history of objective

Dengzhan Xixin is a medicinal herb commonly used by ethnic minorities, originating from the book "*Dian Nan Ben Cao – Volume 1*" (1436 C.E.) written by the Ming Dynasty scholar Lan Mao. In ancient times, Dengzhan Xixin was often used to treat hemiplegia. In current research, modern pharmacological studies have demonstrated that Dengzhan Xixin has anti-inflammatory, anticoagulant, antioxidant, anti-apoptotic, anti-ischemic cardiovascular disease, anti-rheumatic, and cardioprotective and hepatoprotective effects.

Background

Erigerontis Herba (EH, Dengzhanxixin in Chinese), the dried grass of *Erigeron breviscapus* (Vant.) Hand.-Mazz., has a long medicinal utilization history in Guangxi, Yunnan, Guizhou, Hunan, Tibet, and Sichuan provinces in China [1]. EH was firstly recorded in the *Materia Medica in South Yunnan* (Ming Dynasty), and was good at, eliminating wind, dissipating cold (relieve discomfort caused by cold and wind), removing dampness (reduce issues related to excess moisture in the body), tonifying Qi (enhance physical strength and immunity), and invigorating blood circulation [2–4]. Flavonoids, caffeoyl derivatives, and volatile compounds were the main active components of EH. Among these compounds, scutellarin and apigenin-7-O-glucuronide are the major flavonoids; while caffeic acid, dicaffeoylquinic acid, and tricaffeoylquinic acid are the major caffeoyl derivates [5]. Modern pharmacological research indicated that EH and its active compounds possess various of pharmacological activities, such as anti-coagulation, anti-oxidation, anti-inflammation, and anti-apoptosis, anti-ischemic cardio-cerebrovascular diseases, anti-rheumatism, heart and liver protection [6–11]. The clinical studies also demonstrated EH has definite curative effects on cardio-cerebrovascular diseases with minimal side effects, and the total effective rates were more than 95% [1].

In traditional applications, EH has been used to treat various symptoms, such as hemiplegia after stroke, chest pain, rheumatic arthralgia, headache, and toothache. For the treatment of stroke, EH could prevent the stroke sequela (headache, hemiplegia, and cognitive impairment, etc.) through inhibiting platelet aggregation and improving microcirculatory disorders [10, 12]. In the treatment of chest pain, EH could increase blood supply to the myocardium via vasodilating the coronary arteries, thus relieving symptoms of chest pain [13]. EH also has the effects of circulating blood, dispelling wind, and removing dampness, as well as relieving pain, which could alleviate inflammation, rheumatic arthralgia, toothache, and other symptoms [14].

Owing to its excellent treatment effects, EH has been listed as the four major cardio-cerebral-vascular protection medicines along with *Salviae Miltiorrhizae Radix et Rhizoma*, *Ginkgo biloba* and *Notoginseng Radix et Rhizoma* [1]. EH and its related prescriptions (*Erigeron breviscapus* injection (EBI), Dengzhan Shengmai capsule, Breviscapine tablets, etc.) have been widely applied for the clinical treatment of coronary heart disease, ischemic stroke (IS), and angina pectoris [8, 15, 16]. For example, Dengzhan Shengmai capsule (DZSMC) is the only Chinese patent drug used for the treatment of secondary prevention for stroke, which ranks the first among TCMs for neurological treatment [17]. With in-depth exploration on EH, more and more effects were found (anti-platelet aggregation, anti-atherosclerosis, and inhibiting brain edema, etc. [1, 8, 10, 14, 18]), which attracted increasing attention by researchers. Hence, the social and economic values of EH are great. To our best knowledge, the pharmacological effects of EH on diabetes and its complications, as well as the bone and joint degenerative diseases were reviewed for the first time. It also summarized the clinical applications of EH's prescriptions in recent years.

The present review concluded the modern research progress on the ethnopharmacology, botany, phytochemistry, and pharmacological activities of EH and its related prescriptions. It offers overall knowledge of EH and some deficiencies were also proposed, which could promote the further development of EH related products.

Methods

Information was acquired by searching online databases, including PubMed, Elsevier, Springer, Baidu Scholar, Google Scholar, and CNKI. Remarkably, publications on EH are reported exclusively in English. We also obtained some information about this herb from classical Chinese herbal writings.

Botany

The genus of *Erigeron* included over 200 species, mainly distributed in Asia, North America, and Europe, with a few species in Oceania and Africa. There are 35 species in China, mainly located in Xinjiang and the southwestern mountainous areas. *Erigeron* is divided into two subgenera: *Subgen Erigeron* and *Subgen Trimorpha*. According to the 2020 edition of *Chinese Pharmacopoeia*, the length of EH is about 15–25 cm. The rhizome is 0.2–0.5 cm in diameter and 1–3 cm long; the surface is uneven with many attached cylindrical fine roots, which are about 0.1 cm in diameter. The stem is cylindrical, 0.1–0.2 cm in diameter and 14–22 cm long; it is light brown to yellow-green with fine longitudinal ridges, which were covered with white pubescent hair; it is brittle in texture, and the cross-section is yellow-white with hollow or pith. The basal leaves are wrinkled and broken, and when flattened, they are inverted egg-shaped lanceolate, spoon-shaped, broad lanceolate or broad inverted egg-shaped, 0.5–1.3 cm wide and 1.5–9 cm long. The stem leaves are alternate, lanceolate, and the base leaves wraps around the stem. The capitulum is terminal. The achene is flat and oval. The illustration of EH was shown in Figure 1.

This plant is found throughout Tibet, Yunnan, Hunan, Guizhou, Guangxi, and Sichuan provinces in China. It is a distinctively valuable medicinal herb in Yunnan [1]. It grows exclusively in forest margins and open slope grasslands, specifically at altitudes ranging from 1,200 to 3,500 meters, and has been designated as a nationally protected species of TCMs [19]. The entire plant is harvested during the summer and used for medicinal purposes [20, 21].

Ethnopharmacology

Based on the *Chinese Pharmacopoeia (version 2020)*, the dried grass of EH is slightly bitter and spicy with a warm character. Ancient books recorded that it predominantly enters the heart and liver meridians. EH has diverse pharmacological activities such as antithrombosis, anti-tumor, anti-inflammatory, and anti-osteoporotic effects [6, 7, 10, 11]. The aqueous extract of EH is also utilized as its main disease resistant compound. The multiple pharmacological activities are matched with the capability of dispelling wind and removing dampness, circulating blood, and relieving pain. Clinically, EH is mainly applied to treat stroke, heart disease, Alzheimer's disease (AD), diabetes, bone and joint degenerative diseases [15–18]. The recommended administration dose for patients is 9–15 g. Almost no side effects of EH could be found in the clinic.

The dried grass of EH has a long history of clinical application in the field of health maintain. In China, the application of EH can date from

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over 500 years ago (Ming Dynasty). It was medicinally used in several ethnic groups, including Miao, Zhuang, Yi, Bai, Naxi, and Tibetan [8]. EH was firstly recorded in Materia Medica of South Yunnan. This is an early record of EH clinical compatibility in the treatment of injuries from falls, fractures, contusions, and strains. After the Ming Dynasty, several EH related prescriptions emerged gradually and have a long application history from generation to generation in southern China. Up to the last century, EH was mainly utilized to deal with cold and rheumatic arthralgia. After the founding of the People's Republic of China, scientists first discovered that EH possessed the clinical efficacy of hemiplegia after stroke, cerebral hemorrhage, and coronary heart disease [15]. Subsequently, EH related prescriptions (EBI, Breviscapine tablets, and DZSMC, etc.) are extensively developed and used to treat rheumatism, cerebral thrombosis, hemiplegia, microcirculation disorders, and coronary heart diseases. These prescriptions exhibited significant curative effects with minimal side effects. Especially in the treatment of cardio-cerebral-vascular diseases, the curative effect of EBI is remarkably high, with therapeutic efficacy exceeding 95% [1]. The Chinese medicine prescriptions and minority applications of EH were exhibited in Figure 2.

Figure 1 An illustration of EH.

Figure 2 Chinese medicine prescriptions and minority traditional applications of EH

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Chemical composition

Currently, 25 flavonoids and flavonoid glycosides, 46 caffeoyls, 78 volatile oils, and approximately 40 other compounds have been isolated from EH.

Flavonoids and flavonoid glycosides

Flavonoids and their glycosides, represented by scutellarin, are the main active ingredients of EH, and literature search revealed that the flavonoids in EH mainly included five types: flavanones (1–2), flavones (3–7), flavonols (8–11), flavonol glycosides (12–15), and flavonoid glycosides (16–25). At present, 25 flavonoids and their glycosides have been isolated from EH, as shown in Table 1, Figure 3 [22–30]. Breviscapine, the total flavonoid extract of EH, which contains scutellarin (no less than 90%) and apigenin-7-O-glucuronide (no more than 10%) [5].

Caffeoyl compounds

Caffeoyl compounds are abundant in EH, most of them occurred as the polymers of various caffeic acids. Until now, four types of caffeic acid polymers have been discovered, including quinic acids (compounds 26–52), 2,7-anhydro-3-deoxy-2-octulopyranosonic acids (compounds 53–59), 2,7-anhydro-2-octulopyranosonic acids (compounds 60–65), 1-(2'-γ-pyrones) (66–68) and caffeic acid derivatives (compounds 69–71). In total, 46 caffeoyls have been identified from EH, which are presented in Figure 4, Table 2 [31–39]. The compound CQA consists of caffeic acid, 3-O-caffeoylquinic acid, 3,5-dicaffeoylquinic acid, and 3,4,5-tricaffeoylquinic acid, which accounts for 29.7% of EH compounds [40].

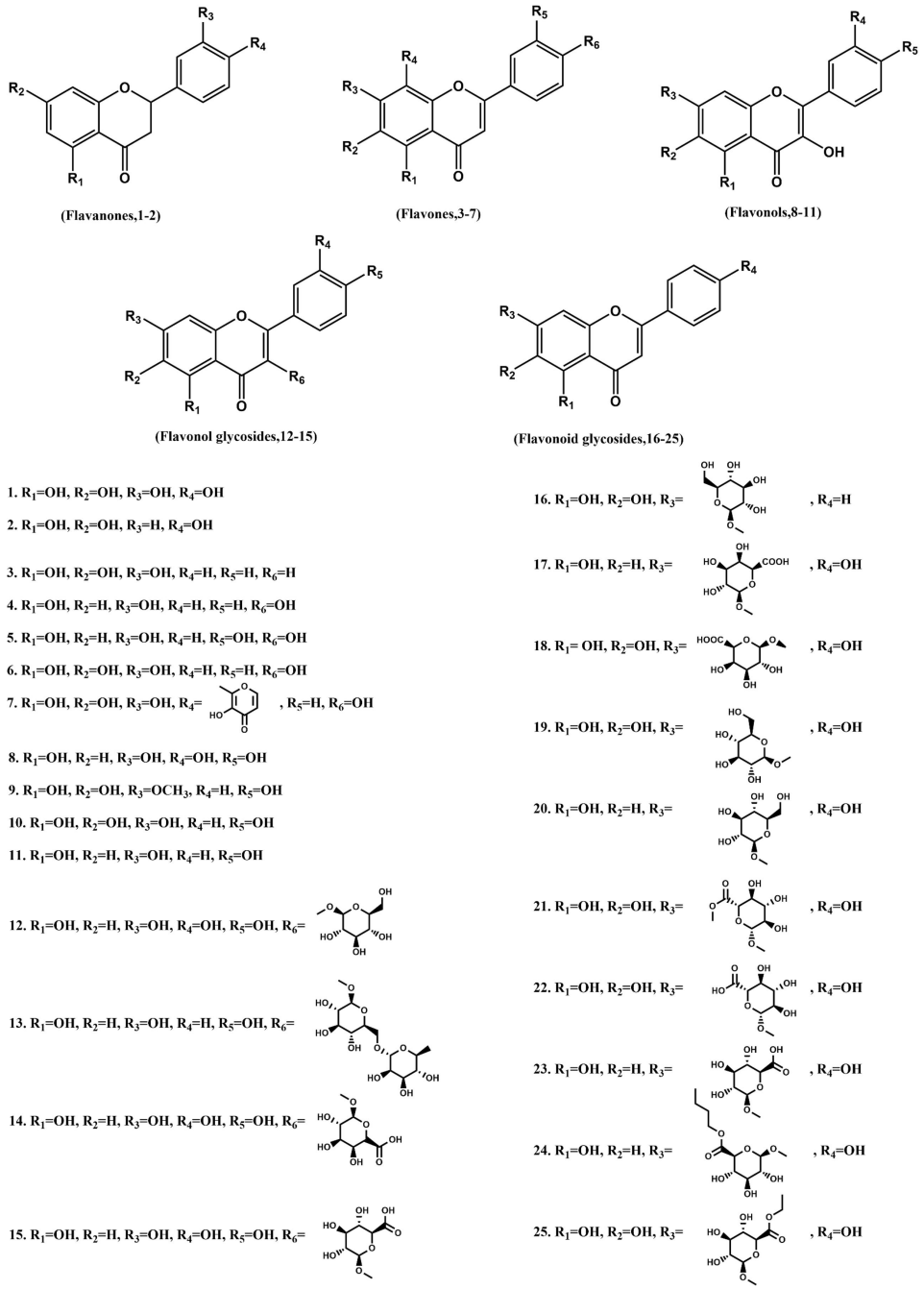
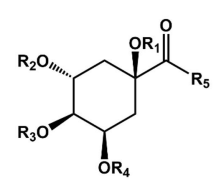
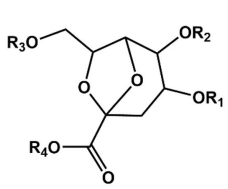


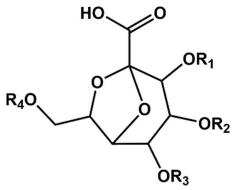
Figure 3 Structures of flavonoids and flavonoid glycosides



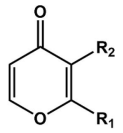
(Caffeoyl compounds,26-52)



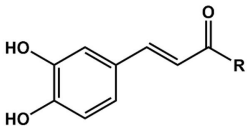
(Caffeoyl compounds,53-59)



(Caffeoyl compounds,60-65)



(Caffeoyl compounds,66-68)



(Caffeoyl compounds,69-71)

26. R_1=R_2=R_3=R_5=H, R_4=caffeoyl

27. R_1=R_2=R_4=R_5=H, R_3=caffeoyl

28. R_1=H, R_2=R_3=R_4=R_5=caffeoyl

29. R_1=R_3=R_4=R_5=H, R_2=caffeoyl

30. R_2=R_4=R_5=H, R_1=R_3=caffeoyl

31. R_1=R_4=caffeoyl, R_2=R_3=R_5=H

32. R_1=R_4=R_5=H, R_2=R_3=caffeoyl

33. R_1=R_3=R_5=H, R_2=R_4=caffeoyl

34. R_1=R_2=R_5=H, R_3=R_4=caffeoyl

35. R_1=malonyl, R_2=R_4=caffeoyl, R_3=R_5=H

36. R_1=R_5=H, R_2=R_4=caffeoyl, R_3=acetyl

37. R_1=R_5=H, R_2=R_4=caffeoyl, R_3=malonyl

38. R_1=R_5=H, R_2=R_3=R_4=caffeoyl

39. R_1=R_2=R_3=caffeoyl, R_4=R_5=H

40. R_1=R_2=R_4=caffeoyl, R_3=R_5=H

41. R_1=R_3=R_4=caffeoyl, R_2=R_5=H

42. R_1=R_2=R_4=H, R_3=caffeoyl, R_5=CH_3

43. R_1=R_2=R_3=H, R_4=caffeoyl, R_5=CH_3

44. R_1=R_2=R_4=caffeoyl, R_3=malonyl, R_5=H

45. R_1=R_5=CH_3, R_2=R_4=caffeoyl, R_3=H

46. R_1=R_2=H, R_3=R_4=caffeoyl, R_5=CH_3

47. R_1=R_3=H, R_2=R_4=caffeoyl, R_5=CH_3

48. R_1=R_4=H, R_2=R_3=caffeoyl, R_5=CH_3

49. R_1=R_3=R_4=H, R_2=caffeoyl, R_5=Butyl

50. R_1=R_3=R_5=H, R_2=caffeoyl, R_4=caffeoyl-4-O-glucoside

51. R_1=R_2=R_3=R_5=H, R_4=caffeoyl-4-O-glucoside

52. R_1=caffeoyl, R_2=R_3=R_5=H, R_4=caffeoyl-4-O-glucoside

53. R_1=R_2=R_4=H, R_3=caffeoyl

54. R_1=R_3=R_4=H, R_2=caffeoyl

55. R_1=R_4=H, R_2=R_3=caffeoyl

56. R_1=caffeoy$_1$, R_2=R_3=R_4=caffeoyl

57. R_1=R_3=caffeoyl, R_2=H, R_4=CH_3

58. R_1=caffeoyl, R_2=R_4=H, R_3=caffeoyl-4-*O*-glucoside

59. R_1=R_2=R_3=caffeoyl, R_4=H

60. R_1=caffeoyl, R_2=R_3=R_4=H

61. R_1=R_2=R_4=H, R_3=caffeoyl

62. R_1=R_2=R_3=H, R_4=caffeoyl

63. R_1=R_3=H, R_2=R_4=caffeoyl

64. R_1=R_2=H, R_3=R_4=caffeoyl

65. R_1=H, R_2=R_3=R_4=caffeoyl

66. R_1=H, R_2=-6-caffeoyl-α-D-glucoside

67. R_1=-6-caffeoyl-β-D-glucoside, R_2=H

68. R_1=H, R_2=-6-caffeoyl-β-D-glucoside

69. R=OH

70. R=OCH_2CH_3

71. R= 

Figure 4 Structures of caffeoyl compounds

Table 1 Flavonoids and flavonoid glycosides of EH

No.	Compound name	References
1	Eriodictyol	[22]
2	Naringenin	[23]
3	Baicalein	[24]
4	Apigenin	[25, 26]
5	Luteolin	[25]
6	Scutellarein	[26]

Table 1 Flavonoids and flavonoid glycosides of EH (*continued*)

No.	Compound name	References
7	Erigerononones A	[27]
8	Quercetin	[24]
9	3,5,6,4'-Tetrahydroxy-7-methoxyflavone	[28]
10	3,5,6,7,4'-Pentahydroxyflavone	[28]
11	Kaempferol	[24]
12	Quercetin 3-O-β-D-glucoside	[23]
13	Kaempferol-3-O-rutinoside	[29]
14	Quercetin-3-O-β-galacturonide	[27]
15	Quercetin-3-O-glucuronide	[26]
16	Baicalein-7-O-β-D-glucopyranoside	[24]
17	Apigenin-7-O-β-galacturonide	[27]
18	5,6,4'-Trihydroxyflavone-7-O-β-D-galacturonide	[28]
19	4'-Hydroxy baicalein-7-O-β-D-glucopyranoside	[30]
20	Apigenin 7-glucoside	[26]
21	Scutellarin	[26, 30]
22	Apigenin-7-O-glucronide	[28]
23	Apigenin-7-O-β-D-glucuronide	[26, 30]
24	5,4'-Dihydroxyflavone-7-O-butyl beta-D-glucuronide	[28]
25	5,6,4'-Trihydroxyflavone-7-O-β-D-glucuronide ethyl ester	[28]

Table 2 Caffeoyl compounds of EH

No.	Compound name	References
26	5-caffeoylquinic acid	[12]
27	4-caffeoylquinic acid	[31]
28	1-caffeoylquinic acid	[31]
29	3-caffeoylquinic acid	[31]
30	1,3-dicaffeoylquinic acid	[12]
31	1,5-dicaffeoylquinic acid	[31]
32	3,4-dicaffeoylquinic acid	[12]
33	3,5-dicaffeoylquinic acid	[12]
34	4,5-dicaffeoylquinic acid	[12]
35	1-malonyl-3,5-dicaffeoylquinic acid	[31]
36	Acetyl-dicaffeoylquinic acid	[31]
37	4-malonyl-3,5-dicaffeoylquinic acid	[31]
38	3,4,5-tricaffeoylquinic acid	[31]
39	1,3,4-tricaffeoylquinic acid	[31]
40	1,3,5-tricaffeoylquinic acid	[31]
41	1,4,5-tricaffeoylquinic acid	[31]
42	4-O-caffeoylquinic acid methyl ester	[32]
43	5-O-caffeoylquinic acid methyl ester	[32]
44	4-malonyl-1,3,5-tricaffeoylquinic acid	[31]
45	1-O-methyl-3,5-O-dicaffeoylquinic acid methy lester	[33]
46	4,5-O-dicaffeoylquinic acid methyl ester	[34]
47	3,5-O-dicaffeoylquinic acid methyl ester	[34]
48	3,4-O-dicaffeoylquinic acid methyl ester	[35]
49	5-O-caffeoylquinic acid butyl ester	[33]
50	3,5-dicaffeoylquinic acid glucoside	[31]
51	5-caffeoylquinic acid glucoside	[31]
52	1,5-dicaffeoylquinic acid glucoside	[31]
53	9-caffeoyl-2,7-anhydro-3-deoxy-2-octulopyranosonic acid	[31]

Table 2 Caffeoyl compounds of EH (*continued*)

No.	Compound name	References
54	4-caffeoyl-2,7-anhydro-3-deoxy-2-octulopyranosonic acid	[31]
55	Erigoster B	[31]
56	3-caffeoyl-2,7-anhydro-3-deoxy-2-octulopyranosonic acid	[31]
57	Erigoster A	[36]
58	3,9-dicaffeoyl-2,7-anhydro-3-deoxy-2-octulopyranosonic acid glucoside	[31]
59	3,4,9-tricaffeoyl-2,7-anhydro-3-deoxy-2-octulopyranosonic acid	[31]
60	2-caffeoyl-2,7-anhydro-2-octulopyranosonic acid	[37]
61	4-caffeoyl-2,7-anhydro-2-octulopyranosonic acid	[37]
62	9-caffeoyl-2,7-anhydro-2-octulopyranosonic acid	[31]
63	3,9-dicaffeoyl-2,7-anhydro-2-octulopyranosonic acid	[31]
64	4,9-dicaffeoyl-2,7-anhydro-2-octulopyranosonic acid	[31]
65	3,4,9-tricaffeoyl-2,7-anhydro-2-octulopyranosonic acid	[31]
66	1-(2'-γ-pyranone) -6- caffeoyl-α-d-pyranglucose	[38]
67	Erigeside I	[31]
68	1-(2'-γ-pyranone) -6- caffeoyl-β-d-pyranglucose	[38]
69	Caffeic acid	[31]
70	Ethyl caffeate	[38]
71	3-O-caffeoyl-γ-quinide	[39]

Volatile oil components

Studies have shown that EH contains a large number of long-chain aliphatic alkanes, long-chain fatty acids, and other compounds. A total of 78 volatile oil components (72–149) have been identified from this plant, as shown in Table 3 [41–43].

Other components

In EH, there are also coumarins, pentacyclic triterpenes, aromatic acids, phytosterols, xanthones, and other constituents (150–188), as shown in Table 4 [22, 23, 25, 27, 28, 35, 36, 44–49].

Pharmacological effects

EH and its Chinese medicine prescriptions possess pharmacological effects of anti-oxidant, vasodilatation and neuroprotection, anti-inflammatory, anti-platelet aggregation, and anti-coagulant [3, 8, 50–53]. Table 5 lists the diseases that EH could be used to treat [2, 3, 5, 8, 11, 15, 16, 20, 40, 54–91].

Cerebrovascular diseases

The active compounds of EH play an anti-inflammatory role to reduce the damage after IS. Scutellarin obviously decreased the volume of cerebral infarction, blood-brain barrier permeability, and neural damage in the cortex and hippocampus by activating the Nrf2 and mitogen-activated protein kinases (MAPK) signaling pathways [15, 54]. Besides, the anti-neuroinflammation effect of scutellarin is comparable to that of p38 MAPK inhibitor [15]. These results indicated that scutellarin might be a promising candidate drug for the treatment of IS. EH's active compounds can exert therapeutic effects not only by directly acting on relevant targets but also through regulating intestinal flora. Breviscapine could protect from cerebral ischemia-reperfusion injury via regulating intestinal flora, suppressing the TLR4/MyD88/NF-κB pathway [5]. The key mechanisms of scutellarin against IS (Figure 5).

EH related prescriptions have been proven to exert therapeutic effects in IS by inhibiting apoptosis. DZSMC could alleviate middle cerebral artery occlusion-induced neuronal apoptosis via enhancing the transport of short-chain fatty acids from the intestine to the brain tissue [92, 93]. EBI could improve brain injury by promoting the expression of phosphatase and tensin homolog deleted on chromosome ten induced putative kinase 1 (PINK1) and Parkin, enhancing mitochondrial autophagy, and inhibiting mitochondrial-mediated apoptosis [8]. It is also noteworthy that EH related prescriptions could exert neuroprotective effects. DZSMC could inhibit platelet activation and inflammation, enhance endothelial cell function, promote neovascularization, and exert neuroprotective effects on transient ischemic attacks [92, 94]. EBI could maintain the structural integrity of blood-brain barrier by inhibiting the expression of matrix metalloproteinase-9 (MMP-9) and claudin-5 [3]. Multiple clinical studies have shown that EBI could improve blood viscosity, lower blood lipids, and improve microcirculation in patients with acute IS [13, 95–99]. In summary, EH's related prescriptions have strong pharmacological effects in anti-apoptosis, neuroprotection, and improvement of microcirculation, which is matched well with the EH's active compounds as promising candidate drugs for IS.

Cardiovascular disease

Scutellarin has the ability to minimize the injury caused by myocardial infarction through anti-inflammatory and anti-apoptotic defenses [57]. Mechanistically, scutellarin effectively suppressed the activation of the nucleotide-binding oligomerization domain, leucine-rich repeat and pyrin domain-containing 3 (NLRP3) inflammasome by activating protein kinase B (AKT) and inhibiting mammalian target of rapamycin complex 1 (mTORC1) [56]. Additionally, scutellarin exhibited the anti-apoptotic effects via modulating ERK1/2-CREB phosphorylation [58]. Overall, the cardioprotective benefits of scutellarin were achieved through its anti-inflammatory and antioxidant mechanisms (Figure 6).

EH's related prescriptions have been proven to treat cardiovascular diseases by improving heart function. EBI has been used to treat unstable angina pectoris and stable angina pectoris [100, 101]. In a randomized controlled trial, the incidences of cardiogenic shock, heart failure, severe arrhythmia, and mortality during hospitalization were lower in the EBI group compared to the standard medication group [102]. DZSMC can reduce cardiac fibrosis, possibly through the suppression of cardiac fibroblasts activation and reduction of excessive extracellular matrix (ECM) deposition via regulating TGF-β1/Smad3 and LTBP2 signaling pathways [103].

Table 3 Volatile oil components of EH

No.	Compound name	References
72	1,3,5-Cycloheptatriene	[41]
73	3-Methylbutyric acid	[41]
74	Benzaldehyde	[41]
75	2-Methylbenzaldehyde	[41]
76	6-Methyl-3,5-heptadien-2-one	[41]
77	2-Heptanal	[41]
78	Heptanoic acid	[41]
79	1,3,5,7-Cyclooctatetraene-1-carbaldehyde	[41]
80	2,3-Dihydro-1-indanone	[41]
81	1-Methyl-4-(1-methylethyl)benzene	[41]
82	β-pinene	[41]
83	Norbornene	[41]
84	DL-Limonene	[41]
85	6,6-Dimethyl-2-methylenebicyclo[3,1,1]hept-3-one	[41]
86	3-Methyl-6-(1-methylethylidene)cyclohexene	[41]
87	(E)-2-Nonenal	[41]
88	Dimethyl-2-butenedioic acid	[41]
89	p-Methoxy-β-cyclopropylthujaplicene	[41]
90	1-Methyl-4-(1-methylethyl)toluene	[41]
91	2,3,5,6-Tetramethylphenol	[41]
92	4,6,6-Trimethylbicyclo[3,1,1]hept-3-en-2-one	[41]
93	Thymol	[41]
94	p-Cymene-8-ol	[41]
95	1,7,7-Trimethyl-[2,2,1]heptan-2-one	[41]
96	1,3,3-Trimethylbicyclo[2,2,1]heptan-2-ol	[41]
97	1,8-oxideo-p-menthane	[41]
98	4-Methyl-1-(1-methylethyl)-3-cyclohexen-1-ol	[41]
99	α-Terpineol	[41]
100	Borneol	[41]
101	Undec-2-ene	[41]
102	Nonanoic acid	[41]
103	Eugenol	[41]
104	Cis-Jasmone	[41]
105	(E,E,E)-1,4,8-Dodecatriene	[41]
106	Phthalic acid	[41]
107	p-Meng-3-en-9-aldehyde	[41]
108	4-(p-Methoxyphenyl)-3-buten-2-one	[41]
109	2-Methyl-5-propylnonane	[41]
110	Thymolhydroquinone	[41]
111	α-Farnesene	[41]
112	4-(1,5-Dimethylhex-4-enyl)cyclohex-2-enone	[41]
113	1,2,3,5,6,7,8,8a-Octahydro-1,4-dimethyl-7-(1-methylethyl)au	[41]
114	2,3,8,8-Tetramethyltricyclo[5,2,2,0(1,6)]	[41]
115	N-Nonylcyclohexane	[41]
116	2,7,10-Trimethyldodecane	[41]
117	N-Pentadecane	[41]
118	2,3,5,9-Tetramethyltricyclo[6,3,0,0(1,5)]undec-2-en-4-one	[41]
119	Caryophylene oxide	[41]
120	3,7,11-Trimethyl-2,6,10-dodecatrien-1-ol	[41]
121	Decylcyclohexane	[41]
122	Tetradecanoic acid	[41]
123	8-Methylhexadecane	[41]
124	Pentadecanoic acid	[41]

Table 3 Volatile oil components of EH (*continued*)

No.	Compound name	References
125	Octadecane	[41]
126	Cetyl carbonate	[41]
127	2,6,10,14-Tetramethylpentadecane	[41]
128	Nineteenthane	[41]
129	Methyl 14-methylpentadecanoate	[41]
130	9-Octadecadienoic acid	[41]
131	N-Octane	[41]
132	Methyl 9,12,15-octadecatrienoate	[41]
133	Eicosane	[41]
134	3,7-Dimethylbenzene-1,6-octadien-3-ol	[41]
135	1,1-Dimethylethyl hexadecanoate	[41]
136	(−)-Myrtenal	[42]
137	Carveol	[42]
138	Nerolidol	[42]
139	Linoleic acid	[42]
140	β-Ocimene	[43]
141	β-Linalool	[43]
142	2,2-Dimethyl-3,5-decadiyne	[43]
143	1-Naphthalenol,4-methoxy-	[43]
144	3-Phenyl-2-propyn-1-ol	[43]
145	3,5,9-Trimethyl-2,4,8-decatrien-1-ol	[43]
146	6,10,14-Trimethyl-2-pentadecanone	[43]
147	10,12-Octadecanediynoic acid	[43]
148	3,7,11,15-Tetramethyl-2-hexadecen-1-ol	[43]
149	10,12-Dipentadecanediynoic acid	[43]

Table 4 Other components of EH

No.	Compound name	References
150	Succinic anhydride	[35]
151	3-hydroxy-4H-pyran-4-one	[35]
152	α-Methoxy-pyrone	[44]
153	4-Hydroxybenzoic acid	[28]
154	Cinnamic acid	[28]
155	3,4-Dihydroxybenzoic acid	[28]
156	4-Hydroxyphenylacrylic acid	[36]
157	5,7-Dihydroxychromanone	[23]
158	p-Methoxycinnamic acid	[28]
159	3,4-Dihydroxycinnamic acid	[28]
160	Scopoletin	[25]
161	6-Hydroxy-7-methoxy-2H-chromen-2-one	[28]
162	Ferulic acid	[36]
163	3,5-Dimethoxy-4-hydroxybenzyl	[28]
164	Sinapic acid	[36]
165	Erigeside C	[45]
166	3-hydroxy-4H-pyran-4-one	[46]
167	4,7-dihydroxy-3-(3-hydroxy-4-oxo-4H-pyran-2-yl)-1H-isochromen-1-one	[45]
168	Erigerononones B	[27]
169	1-hydroxy-2,3,5-trimethoxyxanthone	[28]
170	Betulabuside A	[46]
171	Esculin sesquihydrate	[28]
172	6-(β-D-Glucopyranosyloxy)-7-methoxy-2H-1-benzopyran-2-one	[46]
173	2,6-Dimethoxy-4-allylphenyl-1-β-D-glucopyranoside	[28]

Table 4 Other components of EH (*continued*)

No.	Compound name	References
174	Erigeside II	[47]
175	3,5-Dimethoxybenzoic acid-4-O-β-D-glucopyranoside	[28]
176	3,5-Dimethoxy-4-hydroxybenzoic acid-7-O-β-D-glucopyranoside	[46]
177	Ergost-7,22-diene-3-ol	[48]
178	Stigmasterol	[35]
179	Friedelan	[48]
180	β-sitosterol	[22]
181	Erigeside E	[49]
182	Friedelin	[48]
183	Friedelan-3.α.-ol	[48]
184	Friedelanol	[48]
185	Erigeside D	[49]
186	Glucopyranoside	[35]
187	Daucosterol	[44]
188	Pentadecanoic acid	[48]

Table 5 Potential diseases that EH and its TCMs prescriptions could be used to treat

Disease type	Disease name	References
Cardiovascular and cerebrovascular diseases	IS	[3, 5, 8, 15, 54]
	Ischemic heart diseases	[40, 55–58]
	Cerebral hemorrhage	[40]
	Myocardial infarction	[59]
	Angina pectoris	[16, 40]
	Coronary heart disease	[8, 11]
	Hypertension	[8, 40]
Neurodegenerative diseases	AD	[40, 60–62]
	Stress-induced anxiety disorders	[63]
Bone and joint degenerative diseases	Osteoarthritis	[15, 64–67]
	Intervertebral disc degeneration	[68, 69]
Cancer	Colorectal cancer	[2]
	Non-small cell lung cancer	[70, 71]
	Gastric cancer	[72, 73]
	Lung cancer	[74]
	Osteosarcoma	[53, 75]
	Malignant melanoma	[76]
	Hepatocellular carcinoma	[77]
Diabetes and its complications	Diabetes	[61, 78, 79]
	Type 2 diabetic cardiomyopathy	[80]
	Diabeticnephropathy	[51, 81]
	Diabetic cognitive dysfunction	[20]
Other diseases	Liver injury	[11]
	Idiopathic pulmonary fibrosis	[82]
	Asthma	[83]
	Atherosclerosis	[84, 85]
	Pulmonary embolism	[86]
	Nonalcoholic fatty liver disease	[87]
	Hyperlipidemic	[55]
	Hyperuricemic nephropathy	[88]
	Obesity	[89]
	Gastritis	[90]
	Glaucoma	[40, 91]

IS, ischemic stroke; AD, Alzheimer's disease.

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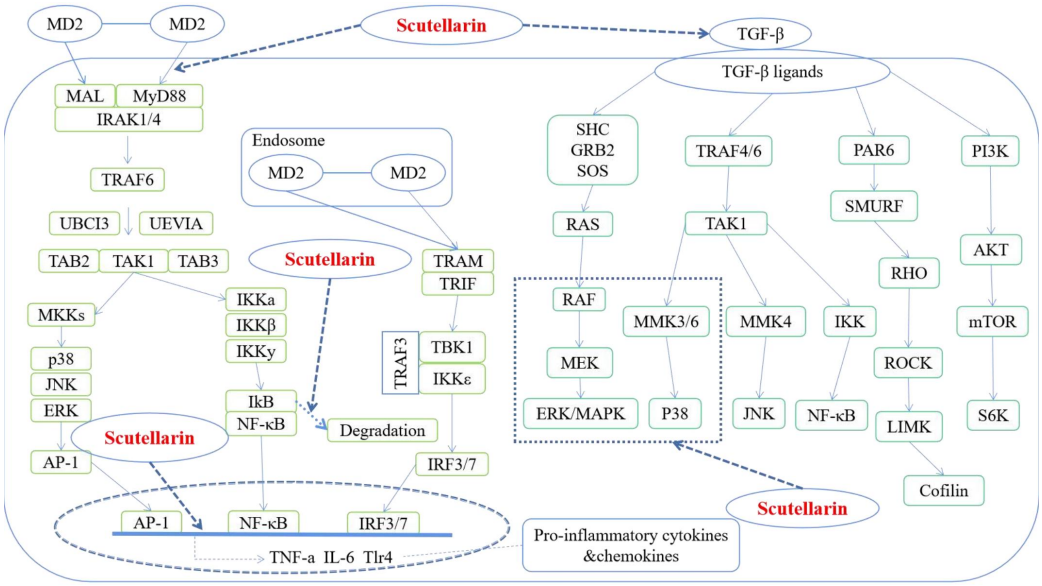

Figure 5 Pattern diagrams for the mechanism of scutellarin in IS. NF-κB, nuclear factor-kappa B; MD2, myeloid differentiation protein 2; JNK, c-Jun N-terminal kinase; ERK, extracellular regulated protein kinases; AP-1, activated protein 1; TRAF6, TNF receptor associated factor 6; UBC13, Ubiquitin-conjugating enzyme E2 13; UEV1A, Ubiquitin-conjugating enzyme E2 variant 1A; TRAM, Translocating chain-associated membrane protein; TRIF, Toll like receptor adaptor molecule 1; TAB, transforming growth factor-beta activated kinase binding protein; IκB, inhibitor of NF-κB; SHC, SHC-transforming protein; GRB2, GRB2-associated-binding protein 2; SOS, Son of sevenless homolog; RAS, Ras-like protein; TAK1, transforming growth factor activated kinase 1; IKK, inhibitor of kappa B kinase.

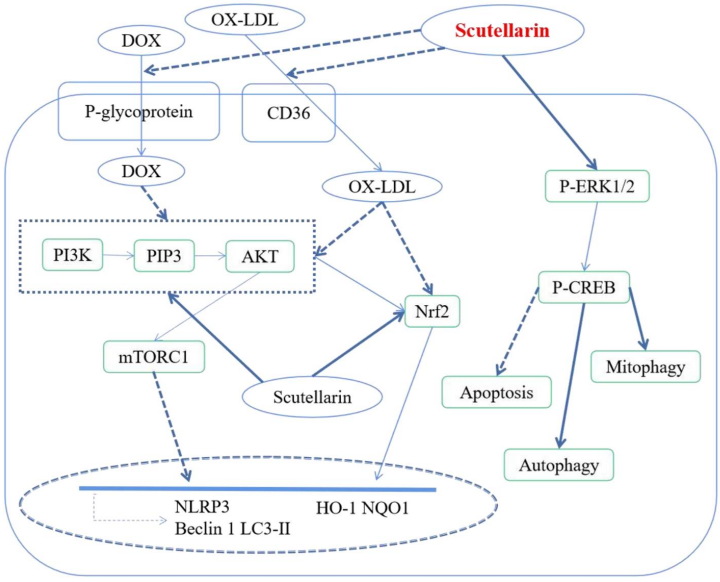

Figure 6 Pattern diagrams for the mechanism of scutellarin in ischemia heart disease. AKT, protein kinase B; PI3K, phosphatidylinositol-3-kinase; mTORC1, mammalian target of rapamycin complex 1; PIP3, 3,4,5-triphosphate phosphatidylinositol; Nrf2, nuclear factor erythroid 2-related factor 2; DOX, dioxygenase.

Neurodegenerative diseases

Current studies have exerted that the active ingredients of EH, such as scutellarin, baicalin, and CQA, possess good pharmacological activities and research prospects in the treatment of cognitive dysfunction. Their mechanisms included reducing A toxicity, lowering tau phosphorylation, inhibiting oxidative stress, regulating the central cholinergic system, anti-inflammatory, anti-apoptotic, promoting the proliferation and differentiation of nerve cells [20, 31]. In addition to active compounds, DZSMC has also been reported to protect against cognitive defects of AD by accelerating A aggregation into fibrils or protofibrils, and reducing soluble A oligomers [104]. The anti-AD mechanism of EH's active components was summarized in Figure 7.

Diabetes and its complications

In diabetes, oxidative stress could accelerate the occurrence and progression of diabetes, and exert adverse effects on microvascular lesions and renal lesions. EH's active compounds exerted essential roles in the treatment of diabetes through antioxidant and anti-inflammatory. Scutellarin could reduce low-density lipoprotein, total cholesterol, serum triglycerides, cholesterol, and insulin levels, upregulate high-density lipoprotein levels, and improve insulin resistance caused by high-fat, high-sugar diet and streptozotocin treatment [1, 79]. Additionally, inflammatory factors can affect the structure and function of islet B cells, leading to insulin resistance and decreased islet function. Scutellarin could inhibit high glucose-mediated vascular inflammation [79]. And breviscapine could inhibit high glucose-induced nuclear factor-kappa B (NF-κB) signaling

activation and subsequently decrease NLRP3 activation [51]. Besides the antioxidant and anti-inflammatory effects, scutellarin can also play a therapeutic role in diabetes by regulating mitochondrial autophagy [74].

In addition to its therapeutic effects on diabetes, EH's active compounds also have an ameliorative effect on diabetic nephropathy. Breviscapine effectively ameliorated various features of diabetic nephropathy in vivo, including renal fibrosis, glomerular expansion, proteinuria, podocyte injury, and mesangial matrix accumulation. Mechanistically, Breviscapine appeared to exert its treatment effects through modulation of the transforming growth factor-β1 (TGF-β1) and Wnt/β-catenin signaling pathways [81].

Cancer

Recent studies have demonstrated that the active components of EBI, including scutellarin, breviscapine, and caffeoylquinic acid esters, possess potential anti-tumor effects. Scutellarin exerted its antitumor actions by arresting the cell cycle, inducing apoptosis and autophagy, inhibiting cell proliferation and epithelial-mesenchymal transition (EMT), and angiogenesis [2]. Scutellarin inhibited the proliferation and EMT of gastric cancer cells, promoting their apoptosis in a dose-dependent manner. Additionally, it enhanced the expression of phosphatase and tensin homolog deleted on chromosome ten (PTEN) while reducing the phosphorylation level of phosphatidylinositol-3-kinase (PI3K), thereby suppressing the progression of gastric cancer [73]. Also, scutellarin could significantly reduce the invasive potential of melanoma cell lines and inhibit EMT and angiogenesis via activating ERK1/2 pathway and inhibiting Akt/mTOR signaling pathway [70, 71, 76]. Furthermore, scutellarin has been found to potentially suppress osteosarcoma cell growth by regulating the EGR1/LINC00857/miR-105-5p/c-Myc axis [75].

Apart from scutellarin, other active components in EBI also exhibited significant antitumor effects. Numerous studies have shown that breviscapine inhibited the growth of liver cancer, prostate cancer, and lung cancer. Breviscapine suppresses the division of osteosarcoma cells by inhibiting the expression of cyclin A and ki67 [53]. Some studies have revealed that EBI significantly suppressed the proliferation of three human colorectal cancer cell lines by activating the RIPK3/MLKL signaling pathway, a crucial pathway in necroptosis [2, 53]. However, the active components responsible for its anti-colorectal cancer effects have yet to be identified.

Bone and joint degenerative diseases

Current research has revealed that scutellarin could reduce the occurrence of inflammation, maintain joint stability, protect cartilage, and delay the progression of osteoarthritis (OA) by inhibiting the formation of osteoclasts, degrading the ECM of cartilage, and suppressing the rupture of collagen fibers and the leakage of proteoglycans on the surface of cartilage. Scutellarin suppressed the degradation of ECM by suppressing the MAPK and NF-κB pathways, thereby reducing abnormal bone remodeling [66]. On the other hand, scutellarin inhibited the expression of Frizzled7, MMP1, ADAMTS-5, Wnt3a, β-catenin, MMP13, and promoted the production of collagen II and aggrecan by regulating the MAPK and Wnt/β-catenin pathways [64]. Additionally, scutellarin decreased the protein expression levels of p-AKT and p-mTOR in IL-1-induced SW1353 cells [65, 67]. All these studies indicated that scutellarin has the potential to be developed as a novel and effective drug for the treatment of OA. The key mechanisms of scutellarin against OA were shown in Figure 8.

Scutellarin exerted therapeutic effects on intervertebral disc degeneration (IVDD) by suppressing inflammation and autophagy in human primary nucleus pulposus cells (NPCs), thereby mitigating the loss of crucial ECM components. Scutellarin reduced the amount of reactive oxygen species generated by mitochondrial damage, which in turn inhibited the activity of NLRP3 inflammasomes by antagonizing the activation of MAPK and NF-κB pathways [68]. Additionally, scutellarin stimulated autophagy activation in NPCs by regulating the PI3K/PTEN/Akt pathway, leading to increased Rab8a expression and exosome release [69]. These studies collectively revealed the protective effects of scutellarin against NPCs degeneration, potentially providing clues for future therapeutic approaches in IVDD. The essential mechanisms are outlined in Figure 9.

Quality control

According to the *Chinese Pharmacopoeia (2020) edition*, scutellarin is the quality control marker of EH, and the content of scutellarin should be no less than 0.30%. Scutellarin and 1,3-dicaffeoylquinic acid are the quality control markers of EBI. The content of flavonoids in this prescription, calculated as scutellarin, should be 0.40–0.60 mg per 1 mL. The total contents of caffeic acid esters in this prescription, calculated as 1,3-dicaffeoylquinic acid, should be 2.0–3.0 mg/mL. Moreover, scutellarin and 4,5-dicaffeoylquinic acid are the quality control markers of DZSMC. The content of EH in each capsule, calculated as scutellarin and 4,5-dicaffeoylquinic acid, should be no less than 15.0 mg and 1.2 mg, respectively.

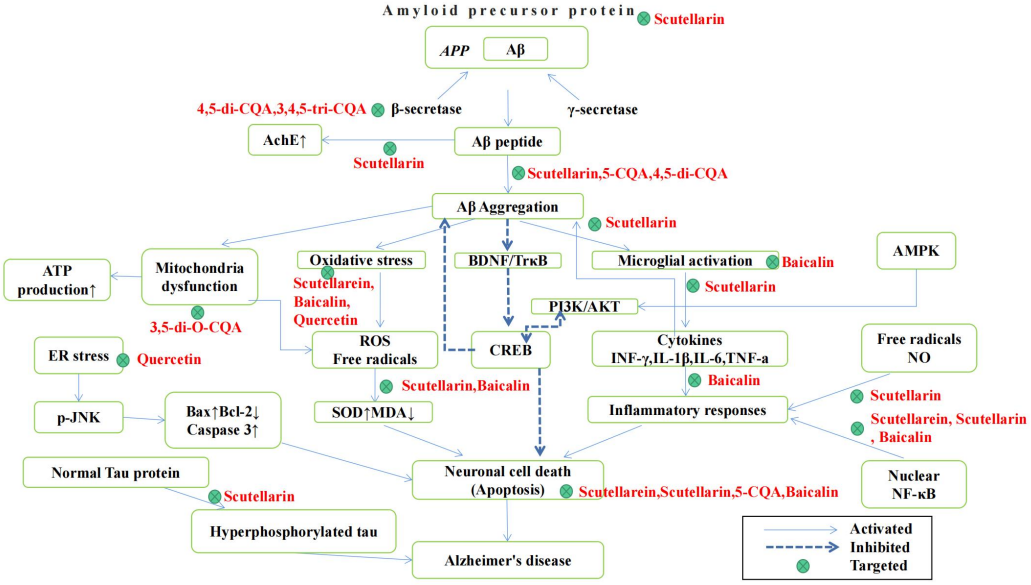

Figure 7 The anti-AD mechanism of EH's active components. ROS, reactive oxygen species; NF-κB, nuclear factor-kappa B; ATP, adenosine triphosphate; ER, endoplasmic reticulum; SOD, superoxide dismutase; MDA, malonaldehyde; NO, nitric oxide; AMPK, Adenosine 5'-monophosphate (AMP)-activated protein kinase.

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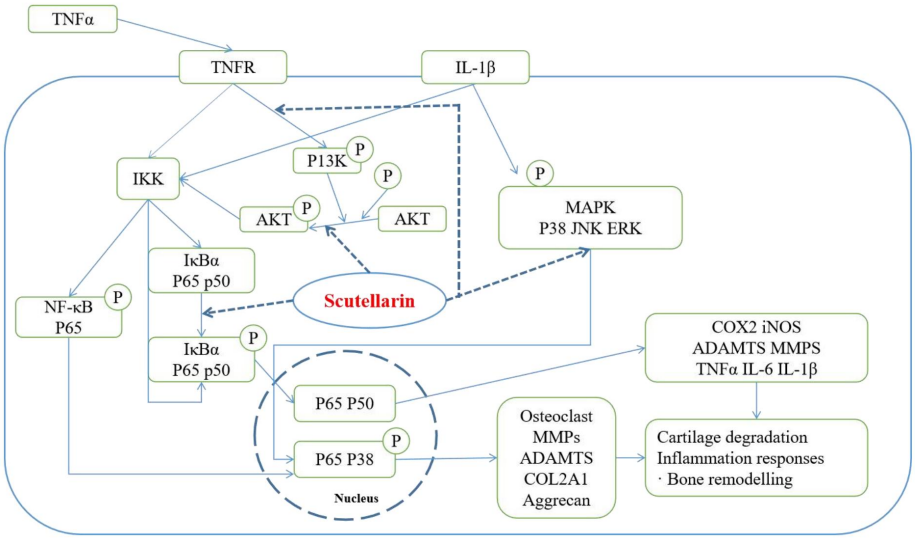

Figure 8 Pattern diagrams for the mechanism of scutellarin in OA. AKT, protein kinase B; PI3K, phosphatidylinositol-3-kinase; NF-κB, nuclear factor-kappa B; MMP, matrix metalloproteinase; IKK, inhibitor of kappa B kinase; COX2, cyclooxygenase-2; iNOS, inducible nitric oxide synthase; TNFR, tumor necrosis factor receptor; MARK, MAP/microtubule affinity-regulating kinase; JNK, c-Jun N-terminal kinase; ERK, extracellular regulated protein kinases; IκB, inhibitor of NF-κB.

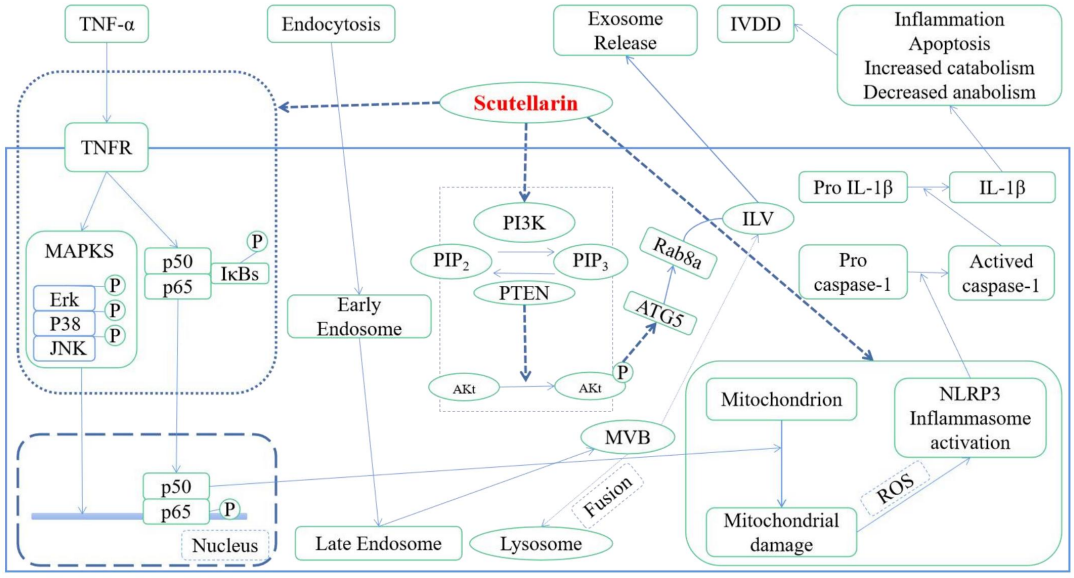

Figure 9 Pattern diagrams for the mechanism of scutellarin in IVDD. IVDD, intervertebral disc degeneration; AKT, protein kinase B; PI3K, phosphatidylinositol-3-kinase; ROS, reactive oxygen species; NLRP3, nucleotide-binding oligomerization domain, leucine-rich repeat and pyrin domain-containing 3; PTEN, phosphatase and tensin homolog deleted on chromosome ten; MVB, multivesicular body; ILV, intraluminal vesicles; MARK, MAP/microtubule affinity-regulating kinase; JNK, c-Jun N-terminal kinase; IκB, inhibitor of NF-κB; PIP3, 3,4,5-triphosphate phosphatidylinositol; TNFR, tumor necrosis factor receptor.

Toxicity

According to the historical records of traditional literature, EH has been found to possess low toxicity, thus it was regarded as a safe medicinal material. The adverse drug reactions associated with EH preparations primarily included digestive system reactions, allergic reactions, cardiovascular system reactions, nervous system reactions, blood system reactions, and respiratory system reactions [105]. The subacute toxicity test further demonstrated that scutellarin, primarily composed of scutellarin B and scutellarin A, does not exert any adverse effects on kidney, liver, and blood functions, with no significant alterations in any of the organs [106].

The acute toxicity test of EBI revealed abnormal mental behavior, behavioral disorders, and convulsions could be observed in mice. In the long-term toxicity test, rats intraperitoneally injected with 480 mg/kg EH daily for two months exhibited slow body weight gain. Pathological examination revealed mild turbid swelling could be found in some renal tubular epithelia [107]. The acute toxicity test of Dengzhanhua dripping pills revealed that no significant pathological changes (organ coefficient, organ tissue structure) were observed [108].

Conclusion

EH, as a traditional Chinese medicine, possessed high medicinal values, low toxicity, and long-term clinical application. This article summarized the botany, ethnopharmacology, phytochemistry, pharmacological effects, quality control, and toxicity of EH. In recent decades, EH has made significant breakthroughs in phytochemistry

and pharmacology. In terms of phytochemistry, this herb has been found to contain compounds mainly categorized as flavonoid, caffeoyl, and volatile. Scutellarin, the main active compound of EH, was extensively studied due to its excellent effects on cardiovascular and cerebrovascular diseases, neurological diseases, and cancer. However, the intrinsic relationships between scutellarin and other active ingredients and the mechanisms of disease treatment are still unclear. Furthermore, the distribution, absorption, metabolism, and excretion pathways of EH's potential pharmaceutically active components still need to be further elucidated through in vivo metabolism studies.

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