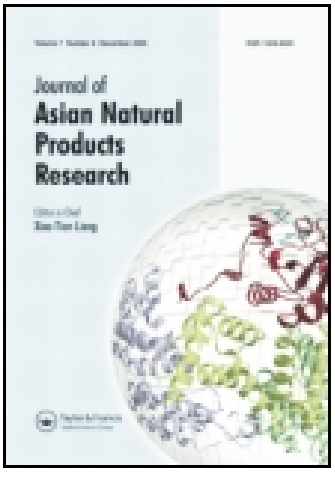

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Natural products as antidepressants documented in Chinese patents from 1992 to 2013

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Depressive disorder is a severe psychiatric problem all over the world. Clinical therapeutic agents for the treatment of depression in the market targeting on monoamine neurotransmitters are far from satisfaction due to their adverse effects. Novel classes of antidepressant agents with different mechanisms and low toxicity are needed. Natural products from traditional Chinese medicines have been revealed as new sources to cure the depressive symptoms with various chemical structures and promising activities. This paper reviews natural products as antidepressants documented in Chinese patents so far.

Keywords: antidepressants; natural products; Chinese patents

1. Introduction

Major depressive disorder is a severe psychiatric syndrome with a lifetime prevalence of 10% and 20% for men and women respectively, and results in heavy social and economic burden [1]. The classical theories on the etiology of depression include monoamine hypothesis and monoamine receptor down-regulation [2]. In addition, hypothalamic–pituitary–adrenal axis, the neurokinin system, brain-derived neurotrophic factor (BDNF), and neuroplastic changes are also related to depression [3,4]. Now most antidepressants in the market target on monoamine neurotransmitters such as 5-hydroxytryptamine (5-HT), noradrenaline (NA) and dopamine (DA) and are classified into four types: monoamine reuptake inhibitors, monoamine oxidase inhibitors, monoamine receptor antagonists, and phytomedicine. However, the delay in the onset of the therapeutic action is ubiquitous among different chemical classes. Moreover, the potent adverse effects to the patients, such as urinary, blurred vision, and sexual dysfunction, still occur severely.

Herbal medicines, especially traditional Chinese medicines (TCMs), are alternative and supplemental sources of new medicine to avoid the adverse effects of synthesized drugs. Previous reviews summarized the progress of natural antidepressants in published literatures [5–7], and this paper concluded the natural antidepressants from Chinese patents up to 2013. According to their structural architectures, they are divided into five types: terpenoids, flavonoids and xanthones, phenylpropanoids, phenols, and other types. The involved main models for predicting the antidepressive efficacy in the patents include forced swim test (FST) and tail suspension test (TST).

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2. Natural products

2.1 Terpenoids

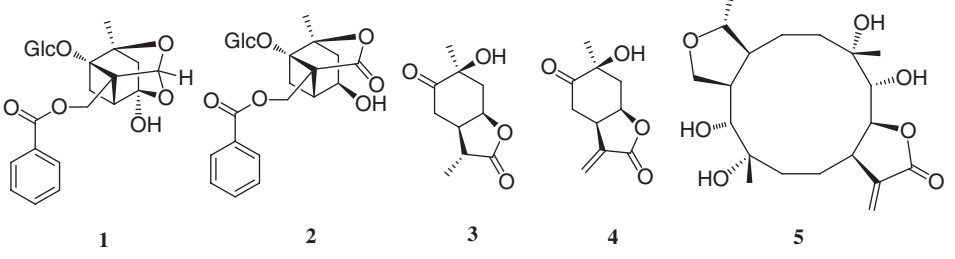
2.1.1 Monoterpenoids and diterpenoids

Paeoniflorin (**1**) and albiflorin (**2**), two bicyclic monoterpenoids isolated from *Paeonia lactiflora* and *Paeonia veitchii*, could decrease immobility time in TST and restrain temperature dropping induced by reserpine at the doses of 50 and 100 mg/kg, and 7 and 14 mg/kg, respectively. The body weight, sucrose water consumption, and the score in open field test of chronic stress depression rats were enhanced at 35 and 70 mg/kg and 7 and 14 mg/kg after successive administration for 21 days. The mechanism showed that the contents of NA and 5-HT in the brain of rats were increased after taking albiflorin (**2**). Besides, paeonilactone A (**3**) and paeonilactone B (**4**) derived from albiflorin (**2**) under anaerobic metabolism also had antidepressive activity [8,9].

Eryngiolide A (**5**) from *Pleurotus eryngii* could decrease immobility time effectively in mouse TST and FST at the doses of 0.2, 1, and 5 mg/kg and showed obvious antidepressive activity [10] (Figure 1).

2.1.2 Dammarane triterpenoids

20(*S*)-Protopanaxadiol and 20(*S*)-protopanaxatriol triterpenoid saponins were the main constituents of *Panax ginseng*. 20(*S*)-Protopanaxadiol (**6**) decreased immobility time in TST and FST at 7.5 and 15 mg/kg, and displayed antagonistic activity of blepharoptosis and akinesia of mouse induced by reserpine at 15 mg/kg. The content of NA, 5-HT was increased in the brain of chronic stress depression rats at 3.33 mg/kg after successive administration for 21 days. The mechanism showed that the uptake of 5-HT and NA was inhibited at doses of 0.1, 1, and 10 μg/ml *in vitro* [11]. 20(*S*)-Protopanaxatriol (**7**) decreased immobility time effectively in TST and FST at 5 and 10 mg/kg, respectively, increased the content of NA and 5-HT in chronic stress depression rats at 8.6 mg/kg, and enhanced the expression of cAMP-response element binding protein (CREB) and BDNF of P12 cells [12]. A series of derivates including dammara -3-*O*-*α*-L-rhamnosyl-3β,12β,20-triol (**8**), dammara-12-*O*-*α*-L-rhamnosyl-3β,12β,20-triol (**9**), ginsenoside Rg_3 (**10**), and ginsenoside Rh_2 (**11**) showed antidepressive activity in TST at different doses between 10 and 50 mg/kg [13]. Ginsenoside Rh_2 (**11**) decreased immobility time effectively in TST and FST, restrained temperature dropping and akinesia induced by reserpine at 15 and 30 mg/kg, inhibited the NA and DA reuptake with IC_{50} values of 21.45 and 3287.1 nM but did not inhibit 5-HT reuptake [14]. Ginsenoside Rg_2 (**12**) enhanced the content of NA, DA, and 5-HT in the brain of rat after administration for 5 days at the doses of 7.5 and 15 mg/kg [15]. Ginsenoside Rb_3 (**13**) was revealed to possess significant activities of decreasing immobility time in TST and FST and restraining akinesia, blepharoptosis, and temperature dropping induced by reser-



1 2 3 4 5

Figure 1. Structures of compounds **1–5**.

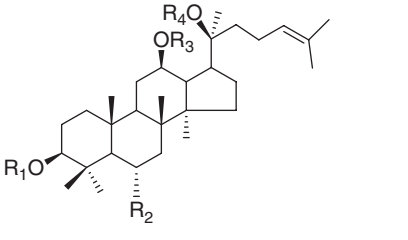
pine in mouse at the doses of 75 and 150 mg/kg. The mechanism might be related to enhancing the content of BDNF and promoting the nerve regeneration to inhibit hippocampal atrophy in the brain [16]. Ginsenosides Rg_1 (**14**) and Rb_1 (**15**), combined with glycyrrhizic acid could decrease immobility time in TST and FST, and the mechanism suggested that it activated cAMP-protein kinase A pathway in hippocampus and enhanced the expression of CREB and BDNF to show antidepressive activity [17]. Chemical modification on 20(*S*)-protopanaxadiol and 20(*S*)-protopanaxatriol led to four active derivatives, dammara-20(*S*)-24(*R*)-epoxy-3β,12β,25-triol (**16**), dammara-20(*S*)-24(*S*)-epoxy-3β,12β,25-triol (**17**), dammara-20(*S*)-24(*R*)-epoxy-3β,6β,12β,25-tetrol (**18**), and dammara-20(*S*)-24(*S*)-epoxy-3β,6β,12β,25β-tetrol (**19**). Among them, compounds **16** and **19** were declared to decrease immobility time in TST at 3.75, 7, and 15 mg/kg and FST at 4, 8, and 16 mg/kg [18].

Pseudojujubogenin (**20**) from *Bacopa monnieri* could decrease immobility time in TST and FST and reverse blepharoptosis and akinesia induced by reserpine significantly, but had no effect on temperature dropping at the doses of 15 and 50 mg/kg, and the mechanism may be correlated to monoamine neurotransmitters in the brain [19] (Figure 2).

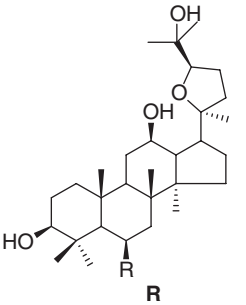
2.1.3 *Oleanane triterpenoids*

Oleanolic acid (**21**) decreased immobility time effectively in TST at 17.5 and 35 mg/kg and FST at 12.25 and 24.5 mg/kg [20]. Total saponins, hederagenin (**22**), akebia saponin D (**23**) from *Akebia quinata* and hederagenin sodium salt could decrease immobility time in TST and FST at 50 and 100 mg/kg. Among them, hederagenin sodium salt exhibited the best activity [21].

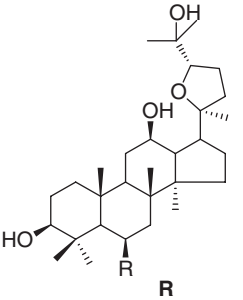
The active part from *Bupleurum chinensis* could decrease immobility time in TST and FST and restrain mouse temperature dropping induced by reserpine at 100 and 200 mg/kg, and involved



	R_1	R_2	R_3	R_4
6	H	H	H	H
7	H	OH	H	H
8	H	H	H	Rha
9	H	H	Rha	H
10	Glc2Glc	H	H	H
11	Glc	H	H	H
12	H	OGlc2Rha	H	H
13	Glc6Xyl	H	H	Glc2Glc
14	H	OGlc	H	Glc
15	Glc2Glc	H	H	Glc6Glc



	R
16	H
18	OH



	R
17	H
19	OH

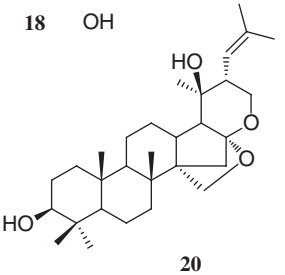


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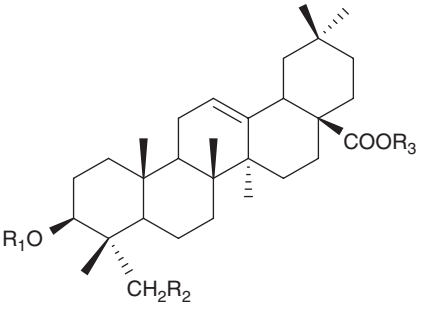
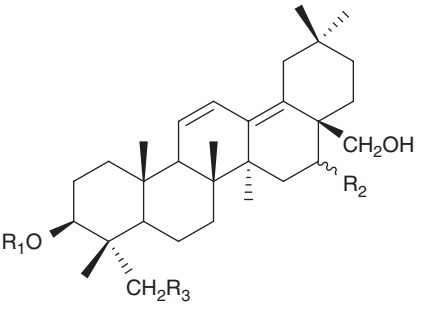
Figure 2. Structures of compounds **6–20**.



	R_1	R_2	R_3
21	H	H	H
22	H	OH	H
23	Ara	OH	Glc6Glc



	R_1	R_2	R_3
24	Glc6Glc |4 Rha	β-OH	H
25	Fuc3Glc	α-OH	OH
26	Fuc3Glc |2 Glc	α-OH	OH
27	Fuc3Glc |6 Ac	α-OH	OH

Figure 3. Structures of compounds **21–27**.

four glycosides, sailkosaponin h (**24**), sailkosaponin b_2 (**25**), 2″-*O*-β-D-glucopyranosylsailkosaponin b_2 (**26**), and 6-*O*-acetyl-saikosaponin b_2 (**27**) [22] (Figure 3).

2.1.4 *Ursane titerpenoids*

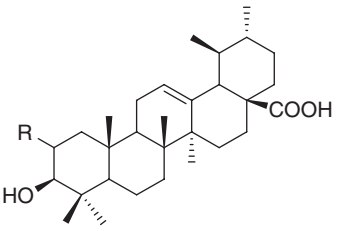
Ursolic acid (**28**), a common triterpenoid, significantly exhibited antidepressive activity in FST at the dose of 10.0 mg/kg [23]. The extract of *Perilla frutescent* containing ursolic acid (**28**) (>20%) decreased immobility time effectively in FST at the doses of 1.0, 5.7, and 18.2 mg/kg and displayed activity against mouse temperature dropping at 5 and 10 mg/kg by enhancing the content of monoamine in synaptic cleft [24].

Total sapogenins, asiatic acid (**29**), and madecassic acid (**30**) from *Centella asiatica*, and asiatic acid sodium salt decreased immobility time in TST and FST at the doses of 30, 60, and 120 mg/kg. All of the four had antagonistic activity on blepharoptosis induced by reserpine and asiatic acid sodium salt exhibited the most potent activity with inhibitory rate of 80% at dose of 60 mg/kg [25]. Besides, the antidepressive activity of asiaticoside (**31**) was also revealed in FST at 3.5 and 7 mg/kg [26].

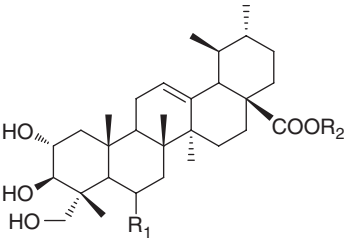
Another ursane pentacyclic triterpenoid, corosolic acid (**32**) isolated from *Lagerstroemia speciosa*, decreased immobility time effectively in TST and FST at 40 and 70 mg/kg and proposed antidepressive activity [27] (Figure 4).

2.1.5 *Other triterpenoids*

Two triterpenoids, gypensapogenin A (**33**) from *Gynostemma pentaphyllum* and aphanamixoid A (**34**) from *Aphanamixis polystachya*, significantly displayed antidepressive activity in TST and FST at doses of 0.2, 1, and 5 mg/kg [28,29] (Figure 5).

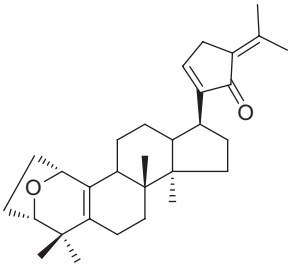


	R
28	H
32	α - OH



	R_1	R_2
29	H	H
30	OH	H
31	H	Glc6Glc4 Rha

Figure 4. Structures of compounds **28–32**.



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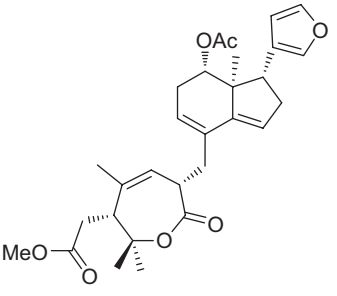

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Figure 5. Structures of compounds **33 and 34**.

2.2 *Flavonoids and xanthones*

Three flavonoid glycosides, naringin (**35**), hesperidin (**36**), and neohesperidin (**37**) from the genus *Citrus*, could resist the reduce of spontaneous activity, treat digestive system dysfunction, and enhance sucrose water consumption of chronic stress depression rats at the doses of 8, 7.2, 8 mg/kg, respectively, and showed inhibitory activity on monoamine oxidase A (MAO-A) with IC_{50} values of 5.82, 12.43, and 13.14 μM [30–32]. A flavonol, houttuynoid D (**38**) isolated from *A. polystachya* also showed antidepressive activity in TST and FST at the doses of 0.2, 1, and 5 mg/kg [33].

Two xanthones, 1-hydroxyl-7-methoxy-xanthone (**39**) and 1,3,5-trihydroxy-xanthone (**40**) isolated from *Hypericum sampsonii*, could decrease immobility time effectively in TST and FST models at 15 and 60 mg/kg [34] (Figure 6).

2.3 *Phenylpropanoids*

The extract of *Acrous atrinowiishott* mainly containing α-asarone (**41**) (4–8%) and β-asarone (**42**) (48–66%) could inhibit MAO-A with IC_{50} values of 20 and 15 μM and monoamine oxidase B (MAO-B) with IC_{50} values of 220 and 600 μM, which showed higher selectivity on MAO-A [35].

Danshensu (**43**) and salvianolic acid B (**44**) isolated from *Salvia miltiorrhiza* demonstrated significantly antidepressive activity. Danshensu sodium salt decreased immobility time in TST and FST mouse models, prolonged sleep duration time, but had no effect on temperature dropping

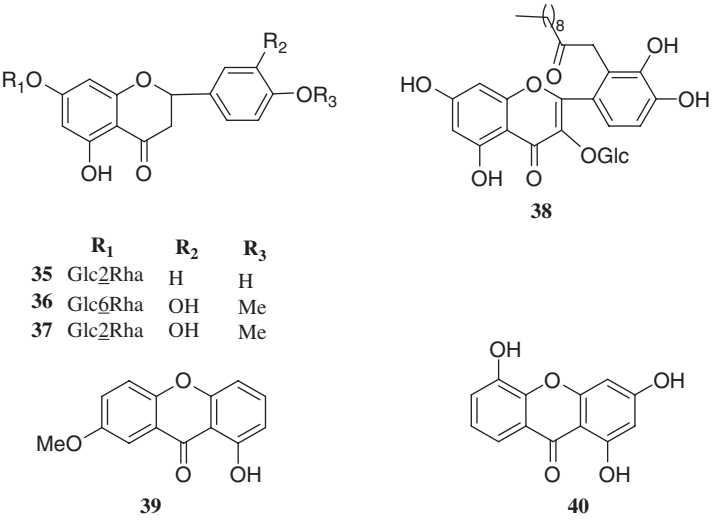

Figure 6. Structures of compounds **35–40**.

induced by reserpine and could not inhibit 5-HT reuptake at the doses of 10, 20, and 40 mg/kg, almost entirely offsetted the cell growth inhibition of P12 induced by dexamethasone at 400 μmol/l *in vitro*, and showed protective effect of cerebral pathological tissue damage of mouse induced by monosodium glutamine at 40 mg/kg *in vivo* [36]. The extract of *S. miltiorrhiza* containing 33% of salvianolic acid B (**44**) decreased immobility time in TST and FST at the doses of 40, 80, and 160 mg/kg, and salvianolic acid B (**44**) could inhibit cyclic adenosine monophosphate-phosphodiesterase to activate cyclic adenosine monophosphate (cAMP) [37].

Rosavin (**45**) from *Rhodiola rosea* exhibited the activity of decreasing immobility time effectively in TST and FST and resisting blepharoptosis, akinesia induced by reserpine at 5, 10, and 20 mg/kg. Mechanism suggested that rosavin (**45**) could inhibit NA and DA reuptake but had no effect on 5-HT reuptake, and thus increases the content of NA and DA in synaptic cleft [38]. Macranthol (**46**) isolated from *Illicium dunnianum* was declared to decrease immobility time obviously in TST and FST at 20 and 40 mg/kg [39] (Figure 7).

2.4 Phenics

Three curcuminoids, curcumin (**47**), demethoxycurcumin (**48**) and bisdemethoxycurcumin (**49**) from *Curcuma longa* were reported to have antidepressive activity. Curcumin (**47**) decreased immobility time effectively in TST and FST at 2.5, 5 and 10 mg/kg, and had antagonistic activity on body dropping induced by reserpine at 5, 10, and 20 mg/kg. Besides, curcumin (**47**) at 5 and 10 mg/kg strengthened action of 5-HT in the brain of mice on the mice head shaking behavior model induced by 5-hydroxytryptophan (5-HTP) [40]. Its metabolite tetrahydrocurcumin (**50**) showed obviously antidepressive activity in TST and FST at 2.5 and 5.0 mg/kg by single or multiple doses and also could strengthen the mice head shaking behavior induced by 5-HTP at 5 mg/kg. Mechanism showed that the contents of NA, DA, and 5-HT in the brain of mouse were increased after administration of tetrahydrocurcumin (**50**) at 5.0 and 10 mg/kg for 14 days [41]. Metabolic half-life *in vivo* was prolonged by chemical modification of curcumin (**47**), demethoxycurcumin (**48**), and bisdemethoxycurmin (**49**). Curcuminoids could still be detected in plasma

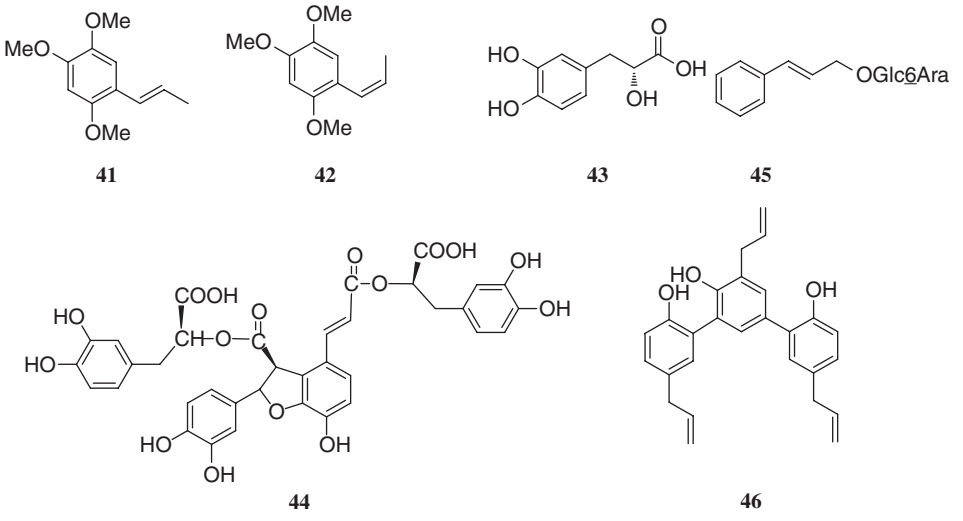

Figure 7. Structures of compounds **41–46**.

after administration of 7 days by a single dose at 18.4 mg/kg in mouse [42].

Resveratrol (**51**) from *Reynoutria japonica* was disclosed with activity of decreasing immobility time in TST and FST at 40 and 80 mg/kg, and could suppress the activity of MAO-A and MAO-B, interestingly inhibit MAO-A selectively, and increase the contents of 5-HT, NA, and DA in the brain of mouse [43]. Stilbeneglycoside (**52**), another polyhydrostilbene widely distributed in bryophyte and higher plants, also showed antidepressive activity in TST at 100 mg/kg and FST at 100 and 200 mg/kg [44].

Curculigoside (**53**), orcinol 1-*O*-β-D-glucopyranoside (**54**), and orcinol (**55**) isolated from *Curculigo orchioides*, together with their derivatives showed antidepressive activity. Curculigoside (**53**) and orcinol-*O*-β-D-glucopyranoside (**54**) decreased immobility time with ED_{50} values of 1.42 and 1.42 mg/kg in FST and 3.27 and 2.06 mg/kg in TST. In addition, the acute toxicity test of orcinol-*O*-β-D-glucopyranoside (**54**) did not demonstrate obvious toxicity [45,46]. Helicid (**56**) and its derivatives were also reported to have antidepressive activity in TST and FST at the doses between 42.0 and 124.6 mg/kg [47] (Figure 8).

2.5 Other types

Timosaponia B-II (**57**), steroid saponin from *Anemarrhena asphodeloides* decreased immobility time in TST and FST and had antagonistic effect on blepharoptosis induced by reserpine with the rate of 40% and 60%, respectively at 60 and 120 mg/kg [48]. Six analogs derived from timosaponia B-III (**58**) could obviously decrease immobility time in TST and FST at 10 mg/kg and showed antidepressive activity [49].

Laetispicine (**59**) isolated from *Piper laetispicum* showed more effective activity at 20 and 50 mg/kg than the positive group (fluoxetine) at 50 mg/kg in FST and obviously inhibited the reuptake of 5-HT, NA at 6.7×10^{-5} and 6.7×10^{-4} mol/l, DA at 1×10^{-5} and 1×10^{-4} mol/l *in vitro* [50,51]. Another amide, 5′-methoxy-3′,4′-methylenedioxy-cinnamic acid isobutylamide (**60**) from the same plant decreased immobility time in TST and FST and restrained temperature dropping induced by reserpine at 20 mg/

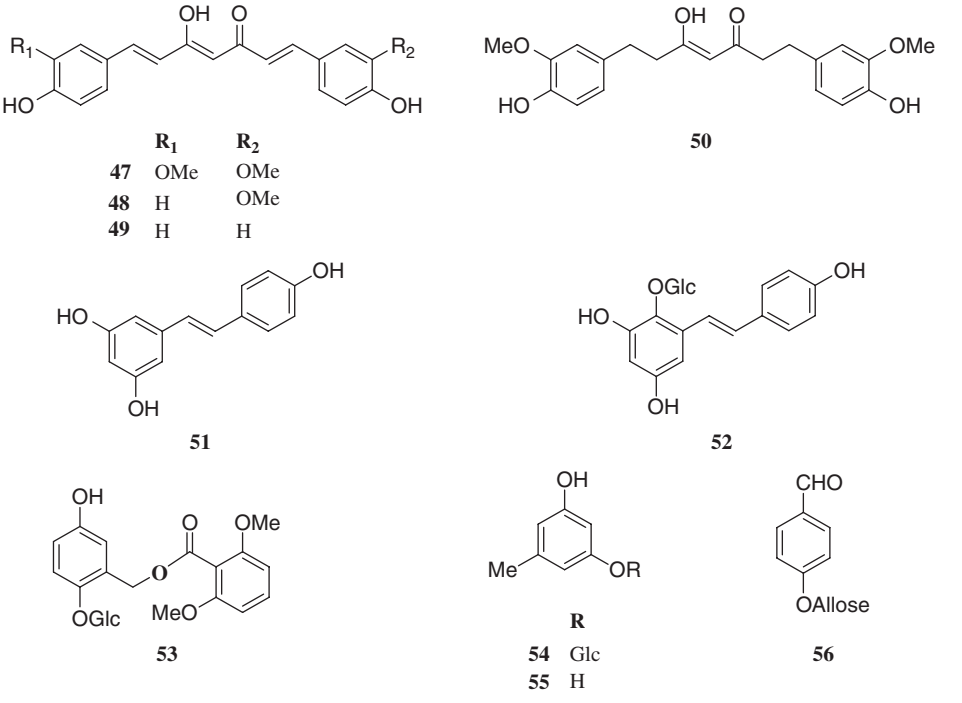

Figure 8. Structures of compounds **47–56**.

kg, increased the content of 5-HT in chronic stress depression rats by continuously administration of 15 mg/kg for 7 days, and showed higher activity than laetispicine (**59**) on 5-HT reuptake with the inhibitory rate of 94% at the concentration of 10 μg/ml [52].

Senkyunolide H (**61**) and senkyunolide I (**62**), phthalides from *Ligusticun chuanxiong*, could increase the content of

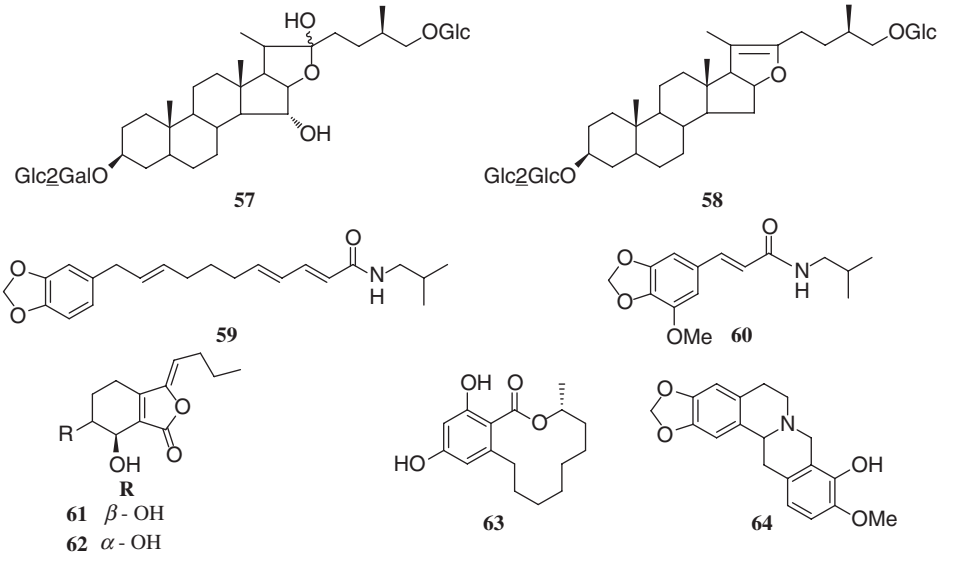

Figure 9. Structures of compounds **57–64**.

monoamine neurotransmitters, especially 5-HT in plasma at 36, 72, and 18, 72 mg/kg, respectively, which could be used to treat depression, cephalagra, and other diseases related to 5-HT nerve system [53,54]. (3*R*)-Des-*O*-methyllasiodiplodin (**63**) isolated from *Cerbera manghas* could significantly decrease immobility time at 10 and 20 mg/kg with no obvious toxicity at the dose of 0.5 g/kg according to acute toxicity tests [55]. Tetrahydroberberrubine (**64**), alkaloid derived from berberine, decreased immobility time in TST and FST at 20 and 30 mg/kg to show antidepressive activity [56] (Figure 9).

3. Conclusions

Many reviews have summarized the progress in the search of natural small-molecule agents of antidepressant, which are mainly concentrated on the result from journal articles instead of patents' advancement [5–7]. Thus, this paper provides a different outlook to summarize 64 compounds from natural products as antidepressants claimed in Chinese patents from 1992 to 2013. The antidepressive properties were concluded based on their structural characteristics and the mechanism research in the patents mostly focused on the inhibition of monoamine reuptake, metabolic inhibition, and BDNF, among which BDNF is an interesting target since most drugs in the market target on monoamine neurotransmitters. Among these compounds, some phenol and amide derivates with simple structures such as tetrahydrocurcumin (**50**), curculigoside (**53**), orcinol 1-*O*-β-D-glucopyranoside (**54**), laetispicine (**59**), and 5′-methoxy-3′,4′-methylenedioxycinnamic acid isobutylamide (**60**) showed high antidepressive activity. Natural active compounds offer major opportunities for finding novel antidepressive targets and mechanisms, which are potential candidates in the development of new antidepressants. Furthermore, some TCMs used to cure depression, such as *B. chinensis*, *P. ginseng*, *A. atrinowiishott*, and *C. longa*, provided a series of active compounds, which showed a short cut of finding novel antidepressant agents from TCMs. However, the active compounds documented in the patents are limited in preliminary animal models, and there is still a long way to be developed as new antidepressive drugs. In conclusion, natural products are one of the important sources of novel antidepressants with novel mechanisms.

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