**Introduction**

For thousands of years, medicinal plants represented a significant source of remedies, as well as the basis of traditional or indigenous healing systems still highly utilized by the population in the world (1-4). In recent years, medicinal plant therapy has been shown to be useful for treatment of various human and animal diseases (2-15). The unnecessary and frequent use of the same drugs used in modern medicine has resulted in the evolution of antibiotic-resistant microbes (1, 2, 8-10, 16). In traditional medical science, a large number of therapeutic plants have presented antimicrobial effects and many of these have been used for the treatment of different infectious diseases (2, 8, 17-26).

Alzheimer’s disease (AD) is an advancing neurodegenerative disease influencing 25 million people worldwide, and AChE is the most likely mark for the treatment of AD among cholinergic hypothesis (27). Several plants have been established as containing acetylcholinesterase inhibitory (AChEI) activity (28-31).

Tyrosinase (TYR), a multifunctional Cu-containing enzyme, catalyzes melanin synthesis in melanocytes (32). It has also been related to Parkinson’s disease (33). Some TYR inhibitors from natural resources have been documented (34, 35).

Lipoxygenases (LOXs) are enzymes associated with inflammatory and allergic reactions because of the production of eicosanoid leukotrienes and lipoxins, and principal biological mediators of inflammatory processes (36). Alam et al. (37) indicated LOX herb inhibitor that can be a source of valuable alternative therapeutic agent.

Xanthine oxidase (XO) catalyzes the oxidation of hypoxanthine into xanthine and uric acid during the metabolic processes of purines (38) accompanied by the production of reactive oxygen species (ROS) (39). XO is involved in the medical state known as gout, which is marked by hyperuricemia that causes uric acid deposition in the joints resulting in painful inflammation. Some research groups have done screening XO inhibitors from local medicinal plants that can perhaps be developed into clinical products (40-43).

The genus Veronica L. is the largest genus of the Plantaginaceae family, with about 500 species that showed a wide ecological variability. Recently, we reported an antioxidant capacity of V. persica phenolic-rich extracts (1); and according to the literature, there are no other data of its biological activity. In this study, antimicrobial, scolicidal activities and AChE, TYR,
LOX, XO inhibitory activities of the extract prepared from *V. persica* were determined. The goal of this study was to discover a new source of effective medicine for treating infectious, neurodegenerative and inflammatory diseases.

**Materials and Methods**

**Plant material and extraction conditions**

The aerial parts (stems, leaves, and flowers) of *Veronica persica* Poir. were collected at flowering stage, in April 2016, from wild plants in the mountains of Meymand, Firuzabad County, Fars Province, Iran (Coordinates: 28°52'04"N 52°45'12"E). A botanist taxonomically identified the plant. All plant material collected dried in the shade, then pulverized into a fine powder using a grinder sieved through a No. 22 mesh sieve and stored in an air-tight container until required for the experiment. A volume of 200 mL of 70% methanol was added to 20 g of powder and kept on a mechanical shaker for 72 h. The content was filtered and concentrated under reduced pressure under controlled temperature to yield a dark gummy residue. The concentrated extract was stored dry in amber-colored flasks at 4 °C for upcoming experiments.

**Antimicrobial activity assay**

The antimicrobial activity of the extracts was assayed against two Gram-positive bacteria (*Staphylococcus aureus* ATCC 6538 and *Bacillus subtilis* ATCC 6633), two Gram-negative bacteria (*Klebsiella pneumoniae* ATCC 10031 and *Pseudomonas aeruginosa* ATCC 9027) and two fungi (*Candida albicans* ATCC 10231 and *Aspergillus niger* ATCC 9142). All microorganisms were obtained from the Persian Type Culture Collection, Tehran, Iran.

The fungi and bacteria were cultured for 14-24 h at 37 °C and the densities were adjusted to 0.5 McFarland standards at 530 nm. The antibacterial experiments were performed by the disc diffusion method (44). Of the 100 μL microbial suspensions were spread on nutrient agar (Merck, Germany) plates (100 mm × 15 mm). Discs (6 mm diameter) were impregnated with 100 μL of different concentrations of extract (50, 100, 150, and 300 μg/mL) and placed on the inoculated agar. All the inoculated plates were incubated for 24 h at 37 °C. In this assay positive control discs used included ketoconazole, gentamycin and ampicillin (10 mg/disc) for fungi, Gram-negative and Gram-positive bacteria, respectively. Furthermore, we used 5% dimethyl sulfoxide (DMSO) as the negative control. Antimicrobial activity was appraised by measuring the zone of inhibition. Minimum inhibitory concentration (MIC) was determined using serial dilutions of the extracts (0-500 μg/mL) using microdilution experiment approved by Clinical and Laboratory Standards Institute (45). The bacteria and fungi were suspended in Luria-Bertani media and the densities were regulated to 0.5 McFarland standards at 530 nm (10⁸ CFU/mL). The extract (100 μL) and the bacteria and fungi suspensions (100 μL) were added to microtiter plates and incubated at 37 °C for 24 h. The medium with bacteria and fungi but without extract was used as growth control and medium without bacteria and fungi was as sterility control. Growth in each well was compared with the growth in the control well. The MICs values were visually detected in comparison with the growth in the control well and delineated as the lowest concentration of the components with >95% growth inhibition.

**Scolicidal activity**

The scolicidal activity, *Echinococcus granulosus* protoscolices were gained from the infected livers of calves killed in an abattoir. Animals were treated humanely according to the Helsinki Convention. Hydatid fluid was collected together with protoscolices using the Smyth and Barrett (46) assay.

Briefly, the hydatid fluid was transferred to a glass cylinder. Protoscolices, which settled at the bottom of the cylinder after 40 min, were washed three times with normal saline and their viability was verified by motility under a light microscope (Nikon Eclipse E200, Japan). Protoscolices were transferred into a dark receptacle comprising normal saline and stored at 4 °C. Three concentrations of plant extract (5, 10, and 15 mg/mL) were assayed for 10, 20, 30 and 60 min. To prepare these concentrations 50, 150 and 100 μL of extracts, added to test tubes, were dissolved in 9.7 mL of normal saline supplemented with 0.5 mL of Tween-80 (Merck, Darmstadt, Germany) under continuous stirring. For each assay, one drop of protoscolex-rich solution was added to 3 mL of the extract solution, mixed slowly, and incubated at 37 °C. After each incubation period (10, 20, 30 and 60 min), the upper phase was carefully removed so as not to disturb the protoscolices, then 1 mL of 0.1% eosin stain was added to the remaining colonized protoscolices and mixed slowly. After incubating for 20 min at 25 °C, the supernatant was discarded. The remaining pellet of protoscolices (no centrifugation carried out) was then smeared on a manually scaled glass slide, covered with a cover glass, and evaluated under a light microscope. The percentage of dead protoscolices was determined after counting a minimum of 600 protoscolices. In the control, protoscolices were treated only with normal saline + Tween-80.

**Acetylcholinesterase inhibition assay**

In this study, the acetylcholinesterase inhibition activity was determined by the method illustrated by Ingkaninan et al. (47). In brief, 3 mL of 50 mM Tris–HCl buffer (pH 8.0), 100 μL of plant extract at various concentrations (0.5, 1.5, 3 mg/mL) and 20 μL AChE (6 U/mL) solution were mixed and incubated for 15 min at 30 °C; a 50 μL volume of 3 mM 5,5'-dithiobis-(2-nitrobenzoic acid) (DTNB) was added to this mixture. The reaction was then started with the addition of 50 μL of 15 mM acetylthiocholine iodide (AChI). The hydrolysis of this substrate was observed at 405 nm in a Hitachi U-2001 spectrophotometer (Tokyo, Japan). The formation of yellow 5-thio-2-nitrobenzoate anion was noticed as the result of the reaction of DTNB with thiocoline, released by the enzymatic hydrolysis of acetylthiocholine iodide. The enzymatic activity was determined as a percentage of the velocities compared to that of the experiment by buffer instead of inhibitor (plant extract), as follows:

$$EA= \frac{E-S}{E} \times 100$$

In this formula, E is the activity of the enzyme wit-
<table>
<thead>
<tr>
<th>Extract (μg/mL)</th>
<th>Staphylococcus aureus</th>
<th>Bacillus subtilis</th>
<th>Klebsiella pneumoniae</th>
<th>Pseudomonas aeruginosa</th>
</tr>
</thead>
<tbody>
<tr>
<td>50</td>
<td>8.5 ± 0.2 e</td>
<td>9.6 ± 0.3 d</td>
<td>7.3 ± 0.1 e</td>
<td>5.5 ± 0.2 e</td>
</tr>
<tr>
<td>100</td>
<td>11.5 ± 0.1 d</td>
<td>11.2 ± 0.1 c</td>
<td>8.5 ± 0.4 d</td>
<td>7.7 ± 0.1 d</td>
</tr>
<tr>
<td>150</td>
<td>12.3 ± 0.1 c</td>
<td>13.8 ± 0.3 b</td>
<td>9.8 ± 0.1 c</td>
<td>8.3 ± 0.1 c</td>
</tr>
<tr>
<td>300</td>
<td>14.4 ± 0.3 b</td>
<td>17.9 ± 0.5 a</td>
<td>13.6 ± 0.3 b</td>
<td>9.5 ± 0.2 b</td>
</tr>
<tr>
<td>Ampicillin</td>
<td>15.5 ± 0.3 a</td>
<td>17.8 ± 0.5 a</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>-</td>
<td>-</td>
<td>14.5 ± 0.2 a</td>
<td>11.9 ± 0.4 a</td>
</tr>
<tr>
<td>DMSO (negative control)</td>
<td>1± 0.0 f</td>
<td>1± 0.0 e</td>
<td>1±0.0 f</td>
<td>1 ± 0.0 f</td>
</tr>
</tbody>
</table>

Data are expressed as means ± SD of inhibition zone diameter (mm) for different concentrations of the plant extract and controls (μg/mL). The values with different letters within a column are significantly different (P < 0.05; HSD). DMSO: dimethyl sulfoxide; MIC: minimum inhibitory concentration.

hout the experiment sample and S is the activity of the enzyme with the experiment sample.

**Tyrosinase inhibition assay**

To assay, the tyrosinase inhibition used the method described by Liang et al. (48). The tyrosinase (EC 1.14.18.1, Sigma) activity was spectrophotometrically measured on 3-(3,4-dihydroxyphenyl)-L-alanine (L-DOPA) (Sigma-Aldrich) as substrate. Tyrosinase aqueous solution (100 μL, 0.5 mg/mL), plant extract (0.5, 1.5, 3 mg/L) and 1850 μL of 0.2 M phosphate buffer (pH 7.0) were mixed and incubated for 15 min at 30 °C. Following, 10 mM L-DOPA solution (50 μL) was added and the absorbance at 475 nm was measured for 3 min against a blank in a Hitachi U-2001 spectrophotometer (Tokyo, Japan). The same reaction mixture having the plant extract replaced by the equivalent amount of phosphate buffer, as blank. The % inhibition of tyrosinase activity was measured based on the formula:

% Tyrosinase inhibition = \( \frac{A_{\text{control}} - A_{\text{sample}}}{A_{\text{control}}} \times 100 \)

where \( A_{\text{control}} \) is the change of absorbance at 475 nm without a test sample, and \( A_{\text{sample}} \) is the change of absorbance at 475 nm with a test sample.

**Lipoxygenase inhibition assay**

The lipoxygenase (LOX) inhibiting activity was measured using spectrophotometrically as illustrated by Lycklander and Malterud (49) with slight modifications. Briefly, 100 μL of the enzyme solution (at the final concentration of 200 U/mL) was prepared in boric acid buffer (0.2 M; pH= 9) and mixed with 25 μL of extract solution (1 mg/mL in boric acid buffer) and then incubated at room temperature for 3 min. Reaction was initiated by the addition of substrate solution (linoleic acid, 250 μM), and the velocity was recorded for 3 min at 234 nm against a blank in a Hitachi U-2001 spectrophotometer (Tokyo, Japan). Negative control was prepared with contained 1% methanol solution without fraction solution. Quercetin was used as positive control. The percentage of lipoxygenase inhibition was calculated using the following formula:

% Inhibition = \( \frac{(V_{c} - V_{s}) \times 100}{V_{c}} \)

where \( V_{c} \) is the activity of enzyme in absence of extract solution, and \( V_{s} \) is the activity of the enzyme in the presence of extract, quercetin or ibuprofen.

**Xanthine oxidase inhibition assay**

The xanthine oxidase (XO) inhibition activity was measured on a spectrophotometer as illustrated by Owen and Timothy (50) with slight modifications. Briefly, the assay mixture consisted of 150 μL of phosphate buffer (0.066 M; pH 7.5), 50 μL of extract solution (1 mg/mL in phosphate buffer), and 50 μL of enzyme solution (0.28 U/mL). After pre-incubation at room temperature (25 °C) for 3 minutes, the reaction was initiated by addition of 250 μL of substrate solution (Xanthine, 0.15 M in the same buffer). A blank without enzyme solution was also prepared. The reaction was monitored for 3 min at 295 nm and velocity (\( V_{o} \)) was recorded. Phosphate buffer was used as negative control (activity of the enzyme without extract solution). Allopurinol was used as positive control. The percentage of xanthine oxidase inhibition was calculated using the following formula:

% Inhibition = \( \frac{(V_{c} - V_{s}) \times 100}{V_{c}} \)

where \( V_{c} \) control is the activity of enzyme without macerate/fraction and \( V_{s} \) sample is the enzyme activity in presence of macerate/fraction or allopurinol.

**Statistical analysis**

All the experiments were carried out in triplicate. Data were subjected to one-way analysis of variance (ANOVA) followed by Tukey’s HSD (honestly significant difference) post-hoc test at \( P < 0.05 \) using SPSS v. 11.5. Data are expressed as a mean ± standard deviation.

**Results**

The antibacterial activity of the extract is summa-
Biological activities of Veronica persica extract.

The results showed that the extract of *V. persica* demonstrated a dose-dependent antibacterial effect on the growth of all tested bacteria. The *V. persica* extract showed the maximum zones of inhibition at a concentration of 300 μg/mL of extract on the growth of all bacteria. Inhibition zones at a concentration of 300 μg/mL of the extract were 14.4 ± 0.3, 17.9 ± 0.5, 13.6 ± 0.3, and 9.5 ± 0.2 mm for *S. aureus*, *B. subtilis*, *K. pneumoniae*, *P. aeruginosa*, respectively. Among bacteria, *B. subtilis* (MIC = 40.3 μg/mL) revealed a high sensitivity to extract of *V. persica*. The results of antifungal assays are shown in Table 2. The *V. persica* extract inhibited the growth of *C. albicans* and *A. niger* in all the assayed concentrations (*P* < 0.05). The maximum inhibition zone was detected in concentration 300 μg/mL plant extract. Indeed, the *V. persica* extract exhibited a strong activity against *C. albicans* and *A. niger* fungi with an inhibition zone of 9.9 ± 0.1 and 66.7 ± 0.2 μg/mL of *V. persica* extract, respectively. Mortality rates of *E. granulosus* protoscolices after treatment with various concentrations of *V. persica* extract is presented in Table 3. As exposure time and extracts concentration increased, % mortality was also increased. Therefore, exposure to the extracts for 60 min, at 5, 10 and 15 mg/mL led to 58.38%, 72.6%, 91.45% inhibition, respectively. The mortality in the control was 3.84%, after 60 min.

The anti-neurodegenerative activity of *V. persica* extract was also evaluated. Table 4 shows the AChE inhibitory activity of *V. persica* extract.

### Table 2. Antifungal activity of *V. persica* extracts.

<table>
<thead>
<tr>
<th>Extract (μg/mL)</th>
<th><em>Candida albicans</em></th>
<th><em>Aspergillus niger</em></th>
</tr>
</thead>
<tbody>
<tr>
<td>50</td>
<td>3.9 ± 0.5 e</td>
<td>5.7 ± 0.5 d</td>
</tr>
<tr>
<td>100</td>
<td>4.7 ± 0.8 d</td>
<td>6.6 ± 0.3 c</td>
</tr>
<tr>
<td>150</td>
<td>7.9 ± 0.3 c</td>
<td>7.9 ± 0.2 b</td>
</tr>
<tr>
<td>300</td>
<td>9.9 ± 0.1 b</td>
<td>10.9 ± 0.5 a</td>
</tr>
<tr>
<td>Ketoconazole (μg/mL)</td>
<td>10.5 ± 0.1 a</td>
<td>10.5 ± 0.1 a</td>
</tr>
<tr>
<td>DMSO (negative control)</td>
<td>1.3 ± 0.1 f</td>
<td>1.1 ± 0.0 e</td>
</tr>
<tr>
<td>MIC</td>
<td>95.5 ± 0.1</td>
<td>66.7 ± 0.2</td>
</tr>
</tbody>
</table>

Data are expressed as means ± SD of inhibition zone diameter (mm) for different concentrations of the plant extract and controls (μg/mL). The values with different letters within a column are significantly different (*P* < 0.05; HSD). DMSO: dimethyl sulfoxide; MIC: minimum inhibitory concentration.

### Table 3. Scolicidal activity of *V. persica* extract against *E. granulosus*.

<table>
<thead>
<tr>
<th>Concentrations (mg/mL)</th>
<th>Exposure time (min)</th>
<th>Protoscolices</th>
<th>Dead protoscolices</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td>10</td>
<td>1829.00 ± 54.21</td>
<td>395.44 ± 18.22</td>
<td>21.59</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>1453.94 ± 70.22</td>
<td>432.25 ± 51.11</td>
<td>29.73</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>1222.53 ± 42.45</td>
<td>588.33 ± 25.14</td>
<td>48.11</td>
</tr>
<tr>
<td></td>
<td>60</td>
<td>1192.74 ± 32.17</td>
<td>696.55 ± 25.51</td>
<td>58.38</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>1455.00</td>
<td>56.00</td>
<td>3.84</td>
</tr>
<tr>
<td>10</td>
<td>10</td>
<td>945.92 ± 22.62</td>
<td>611.45 ± 77.22</td>
<td>64.65</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>799.91 ± 32.81</td>
<td>527.22 ± 17.15</td>
<td>65.95</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>993.74 ± 65.44</td>
<td>669.22 ± 25.33</td>
<td>67.37</td>
</tr>
<tr>
<td></td>
<td>60</td>
<td>732.23 ± 33.17</td>
<td>532.00 ± 25.11</td>
<td>72.67</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>1455.00</td>
<td>56.00</td>
<td>3.84</td>
</tr>
<tr>
<td>10</td>
<td>10</td>
<td>934.91 ± 81.12</td>
<td>684.54 ± 17.82</td>
<td>73.23</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>788.45 ± 13.17</td>
<td>675.39 ± 32.11</td>
<td>85.65</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>993.74 ± 65.44</td>
<td>669.22 ± 25.33</td>
<td>67.37</td>
</tr>
<tr>
<td></td>
<td>60</td>
<td>732.23 ± 33.17</td>
<td>532.00 ± 25.11</td>
<td>72.67</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>1455.00</td>
<td>56.00</td>
<td>3.84</td>
</tr>
<tr>
<td>15</td>
<td>10</td>
<td>934.91 ± 81.12</td>
<td>684.54 ± 17.82</td>
<td>73.23</td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>788.45 ± 13.17</td>
<td>675.39 ± 32.11</td>
<td>85.65</td>
</tr>
<tr>
<td></td>
<td>30</td>
<td>993.74 ± 65.44</td>
<td>669.22 ± 25.33</td>
<td>67.37</td>
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<tr>
<td></td>
<td>60</td>
<td>732.23 ± 33.17</td>
<td>532.00 ± 25.11</td>
<td>72.67</td>
</tr>
<tr>
<td>Control</td>
<td></td>
<td>1455.00</td>
<td>56.00</td>
<td>3.84</td>
</tr>
</tbody>
</table>

Values are mean ± SD of three replicates. In the control, protoscolices were treated only with normal saline + Tween-80.

### Table 4. Acetylcholinesterase inhibitory activity of *V. persica* extract.

<table>
<thead>
<tr>
<th>Sample</th>
<th>0.5 mg/mL</th>
<th>1.5 mg/mL</th>
<th>3 mg/mL</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Veronica persica</em></td>
<td>35.5 ± 0.48 e</td>
<td>46.45 ± 0.32 d</td>
<td>55.32 ± 0.29 e</td>
</tr>
<tr>
<td>Galanthamine</td>
<td>99.86± 0.58 a</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Rutin</td>
<td>55.35 ± 0.19 b</td>
<td>85.85 ± 0.54 a</td>
<td>90.42 ± 0.39 a</td>
</tr>
<tr>
<td>Caffeic acid</td>
<td>52.52 ± 0.32 c</td>
<td>75.99 ± 0.69 c</td>
<td>85.36 ± 0.19 b</td>
</tr>
<tr>
<td>Rosmarinic acid</td>
<td>50.45 ± 0.11 d</td>
<td>77.69 ± 0.51 b</td>
<td>85.55 ± 0.23 b</td>
</tr>
</tbody>
</table>

Data represent the mean ± SD of three independent replicates. The values with different letters within a column are significantly different (*P* < 0.05; HSD).
extract was examined at concentrations of 0.5, 1.5, and 3 mg/mL using AChE and TYR assays (Tables 4 and 5). Inhibition of both enzymes by the extract varied between 35.5% to 55.32% for AChE, and from 25.45 to 52.7% for TYR. A statistically marked difference was observed among the different concentrations of the extract. The extract exhibited statistically weaker activity than the standards galanthamine, rutin, caffeic acid and rosmarinic acid for AChE assay, and ellagic acid, quercetin, rutin and kojic acid for TYR experiment. The extract of V. persica presented stronger inhibitory effect at the highest concentration (3 mg/mL) in both assays.

V. persica extract demonstrated high anti-inflammatory properties, both in the LOX and XO experiments. In the XO experiment, the results showed a high inhibition of enzyme activity. Finally, when assayed using the LOX assay, the extract presented moderate inhibition (Table 6).

Discussion

It is well known that active phytochemicals are produced for protection of plants against microbial pathogens and those plants can be hopeful sources of new compounds with biological activities such as antioxidants and antimicrobials (7, 51, 52). This study demonstrates that the extract of V. persica possesses extensive antibiotic activity against Gram-positive and Gram-negative bacteria, fungi and E. granulosus. The genus Veronica exhibited strong antibacterial activity probably due to the high content of flavonoids, phenol carboxylic acids and tannin (53). Dunki et al. (54) showed that V. spicata is beneficial as phytotherapeutical, antioxidative and antimicrobial agent. Also, Moca et al. (2015) indicated that the use of three Veronica species V. officinalis L., V. teucrium L. and V. orchidea Crantz as antimicrobial agents (55). Probably, the antimicrobial potential of V. persica we observed can be ascribed to the presence of polyphenolic compounds.

AD influences memory and other features of human mind and is characterized by the loss of activities referring to the acetylcholine in the cerebral cortex. Noteworthy, most of the AChE inhibitors such as galanthamine were originally isolated from plants (56). Furthermore, it has been demonstrated that TYR might be related to the damaged neurons typical for another progressive neurological disorder, Parkinson’s disease (57). A number of polyphenols isolated from plants were established as AChE and TYR inhibitors, such as quercetin, kaempferol and caffeic acid (58). Our study documented the activity of V. persica extract against enzymes involved in neurodegenerative disorders. Although inhibitory activities of the extract were lower than the standard ones for those enzymes, the results of our study propose that V. persica could be of interest for the development of food supplements that could prevent neurodegenerative diseases. In a previous study, a moderate neuroprotective activity of V. jacquini and V. teucrium extracts was reported on human neuroblastoma SH-SY5Y cell line (59). In addition, Živković el al. (2017) showed that V. teucrium and V. jacquini methanol extracts inhibit AChE and TYR enzymes (60).

Although Veronica species have been widely utilized in the oriental medicine for inflammatory disorders, the pharmacological effects of these species have not been fully examined. In the present study, V. persica was assessed for its inhibitory effect on LOX and XO. XO is an enzyme that generates ROS (reactive oxygen species) from the chemical reaction it catalyzes. ROS react with cellular lipids, resulting in the formation of lipid peroxides, which are metabolized to malondialdehyde, a major product of lipid peroxidation (61). In our paper, the high inhibition of XO by V. persica extract may contribute to its antioxidant effect. V. persica extract also demonstrated a moderate inhibition of LOX enzyme. LOX are involved in the metabolism of leukotrienes (62). The moderate LOX inhibition by V. persica extract could partially contribute to the anti-inflammatory activity of the plant extract. The anti-inflammatory effects of V. officinalis extract on human lung epithelial cell line A549 were previously (63). Similarly, the extracts of three Veronica species, V. jacquini Baumg., V. teucrium L. and V. irticifolia Jacq exhibited LOX inhibitory activity (64). In conclusion, our results indicate that V. persica extract could be a promising food supplement for human health, particularly in the preven-

### Table 5. Tyrosinase inhibitory activity of V. persica extract.

<table>
<thead>
<tr>
<th>Sample</th>
<th>0.5 mg/mL (%)</th>
<th>1.5 mg/mL (%)</th>
<th>3 mg/mL (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Veronica persica</td>
<td>25.45 ± 0.31 e</td>
<td>35.33 ± 0.12 e</td>
<td>52.7 ± 0.5 e</td>
</tr>
<tr>
<td>Ellagic acid</td>
<td>46.95 ± 0.47 b</td>
<td>66.5 ± 0.26 b</td>
<td>89.42 ± 0.29 b</td>
</tr>
<tr>
<td>Quercetin</td>
<td>35.65 ± 0.21 d</td>
<td>55.49 ± 0.11 c</td>
<td>79.84 ± 0.51 d</td>
</tr>
<tr>
<td>Rutin</td>
<td>39.59 ± 0.41 c</td>
<td>45.59 ± 0.33 d</td>
<td>80.45 ± 0.33 c</td>
</tr>
<tr>
<td>Kojic acid</td>
<td>75.55 ± 0.33 a</td>
<td>85.45 ± 0.39 a</td>
<td>94.44 ± 0.37 a</td>
</tr>
</tbody>
</table>

Data represent the mean ± SD of three independent replicates. The values with different letters within a column are significantly different (P < 0.05; HSD).

### Table 6. Lipoxygenase and xanthine oxidase inhibitory activities of V. persica extract.

<table>
<thead>
<tr>
<th>Sample</th>
<th>Lipoxygenase inhibition (%)</th>
<th>Xanthine oxidase inhibition (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Veronica persica</td>
<td>55.35 ± 0.45 b</td>
<td>84.99 ± 0.22 b</td>
</tr>
<tr>
<td>Quercetin</td>
<td>55.69 ± 0.22 b</td>
<td>85.44 ± 0.32 b</td>
</tr>
<tr>
<td>Allopurinol</td>
<td>ND</td>
<td>92.54 ± 0.43 a</td>
</tr>
<tr>
<td>Ibuprofen</td>
<td>80.38 ± 0.25 a</td>
<td>ND</td>
</tr>
</tbody>
</table>

Data represent the mean ± SD of three independent replicates. The values with different letters within a column are significantly different (P < 0.05; HSD).
tion of infectious, neurodegenerative and inflammatory disorders.

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Conflict of interest
The authors declare no financial or other conflicts of interest.

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