Future Natural Products
doi: 10.34172/fnp.2023.04 2023;9(1):25-30 http://futurenatprod.skums.ac.ir

Original Article

The protective effects of lavender herbal tea on spasticity and ataxia in patients with multiple sclerosis

Nahid Jivad[1], Zahra Forouzandeh Shahrakei[2], Armin Khaghani[3], Mahbubeh Setorki[4*]

[1]Medical Plants Research Center, Shahrekord University of Medical Sciences, Shahrekord, Iran
[2]Deputy of Research and Technology, Shahrekord University of Medical Sciences, Shahrekord, Iran
[3]Student Research Committee, Shahrekord University of Medical Sciences, Shahrekord, Iran
[4]Department of Biology, Izeh Branch, Islamic Azad University, Izeh, Iran

*Corresponding Author: Mahbubeh Setorki, Email: Doctor.setorgi@gmail.com

Received: January 3, 2023
Accepted: May 2, 2023
ePublished: June 12, 2023

Abstract

Background and aims: Multiple sclerosis (MS) is a neurological disease with (an) unknown cause(s) that affects the central nervous system, the brain, and the spinal cord and causes loss of control, vision, balance, and sensation. Ataxia and spasm are commonly observed in patients with MS and cause a significant decrease in functioning and quality of life. Pharmacotherapy, physiotherapy, and rehabilitation are usually used to deal with ataxia and spasms. Given the World Health Organization's emphasis on using herbal medicines with fewer side effects and the anti-inflammatory and neuroprotective effects of lavender extract, the present study aims to investigate the effect of hydroalcoholic lavender extract on ataxia and spasm in MS patients.

Methods: The present study is a double-blind, placebo-controlled clinical trial. Eighty-four patients with MS referred to Imam Ali Clinic and Hajar Hospital of Shahrekord were enrolled in the study and randomly divided into two groups of intervention and control. Patients in the intervention group were given lavender tea, and those in the placebo group received the placebo for 60 days. Before and after the intervention, spasticity was assessed using the Ashworth Scale and the spasm repetition scale. International Cooperative Ataxia Rating Scale (ICARS) and Berg Balance Scale (BBS) were used to evaluate ataxia. Data were analyzed using SPSS 16.

Results: The results showed the mean Ashworth and spasm index before and after intervention did not differ significantly between the two groups of control and placebo ($P<0.05$), but lavender tea could increase BBS and decrease ICARS significantly in the group receiving it ($P<0.05$).

Conclusion: Due to phenolic and flavonoid compounds and high antioxidant properties, lavender can decrease spasticity and ataxia and improve the functioning of other chemicals and drugs used to treat MS in patients.

Keywords: Spasm, Ataxia, Lavender, Multiple sclerosis

Introduction

Multiple sclerosis (MS) is the most common disorder caused by inflammation in the central nervous system, which is characterized by the destruction of the myelin covering axons and the creation of numerous small and large plaques in the brain and spinal cord. Myelin facilitates the rapid and constant transmission of electrochemical signals among the brain, spinal cord, and body. When myelin is damaged, the transmission of nerve signals becomes slower, leading to reduced function. The disease starts with repeated attacks on the white matter and can cause a wide range of disabilities (1). On average, 2.5 million people worldwide suffer from this disease. The probability of infection in women is twice that of men. The age of onset of the disease is mainly between 20-40 years old, but rarely people under two or over 74 years old may also develop this disease (2).

The prevalence rate in different regions worldwide substantially varies, which can be due to the difference in genetic and environmental factors involved in the disease (3). With a prevalence of 50 people per 100 000 people, Iran is considered one of the countries with an average prevalence of MS (4). The clinical symptoms of this disease are different depending on which areas of the central nervous system are involved. In general, the most common symptoms of the disease include sensory disorders, vision disorders, balance and speech disorders, muscle stiffness, fatigue, bladder disorders, defecation disorders, sexual disorders, sensitivity to heat, and cognitive disorders. Unpredictable symptoms may vary from mild to severe and may occur for a short or extended period. Besides this, a combination of symptoms may present depending on the location of the central nervous system. Each patient may have his/her symptoms (5).

Medicinal plants have been known worldwide for thousands of years in different nations and over many centuries. Due to their safe use, efficiency, cultural acceptability, and fewer side effects than chemical drugs,

they have been used by the people of most societies and countries, and herbal medicines have been well-known all over the world due to their unique and distinguished benefits. Research has also shown that these plants, in addition to their economic value, have a special status for the health of different societies due to their phenolic compounds and the antioxidant role of these compounds. Therefore, the approach globally has been towards identifying new species and active ingredients in plants. Therefore, plants currently play an essential part in the treatment of diseases even despite the availability of new drugs.

Lavender is a herbaceous plant from the mint family, Lamiaceae. This plant is fragrant and evergreen and grows up to 90 cm. Its fresh flowering branches are removed after collection and used for medicinal purposes. Lavender is a native plant from the Mediterranean and Arabian coasts to Asia Minor, and in Iran, it grows in Azerbaijan (mountains of Mianeh, Marand, and Hashtrud). This plant is used as a fragrance in cosmetics and health products, and the flower is its main medicinal part (6-8). It is traditionally believed that lavender oil has antibacterial, antifungal, anti-flatulent, anti-epileptic, analgesic, and antidepressant properties. It also effectively treats stomach ailments, tension headaches, burns, and insect bites. Ibn Sina (Avicenna) has recommended its long-term use for nervous diseases (9). Scientific research on this plant has revealed that lavender has analgesic, antispasmodic, and sedative effects, increases the levels of antioxidants, reduces anxiety during childbirth, reduces the secretion of cortisol from the adrenal gland, and increases the secretion of serotonin from the digestive system. In addition, recent scientific reports have shown the properties of lavender in improving sleep problems, anxiety, and pain during the active phase of labor (10-12). In Husseini *and colleagues*' study, the anti-inflammatory and analgesic effects of the lavender extract were investigated in rats. The extract exhibited pronounced analgesic effects in the hot plate test. Different doses of the extract also showed analgesic and anti-inflammatory effects similar to indomethacin, morphine, and dexamethasone in the formalin test (13).

In Hancianu *and colleagues*' study with rats stimulated with scopolamine, lavender essential oil showed potent antioxidant and anti-apoptotic activities. Intraperitoneal injection of essential oil for seven days led to an increase in antioxidant enzymes (SOD, GPX, and CAT) and a decrease in lipid peroxidation (MDA) in the anterior lobes of rats, which indicates the antioxidant capacity of the essential oil (14).

Given the neuroprotective, sedative, analgesic, and anti-inflammatory effects of lavender extract as well as its traditional use as an antispasmodic and analgesic agent, the plant extract seems to have therapeutic effects on MS. With regards to the neuroprotective effects reported for lavender, the present study was designed to investigate the effect of lavender tea on the rate of ataxia and spasm in patients with MS.

Materials and Methods

The present study is a single-blind, controlled clinical trial conducted after obtaining approval for its protocol from the Ethics Committee of Shahrekord University of Medical Sciences and issuance of the ethics code (IR.SKUMS.REC.1398.078 and IRCT; 20220818055740N1) by the Committee. In this study, a total of 84 MS patients (the number of samples with a 95% confidence interval and 80% test power was estimated to be 38 people in each group using the sample size formula, and taking into account a 10% chance of dropping out, the number of samples in each group was 42 people) referred to Imam Ali (AS) Clinic and Kashani Hospital of Shahrekord (March 2019 to May 2020) were enrolled. Inclusion criteria were being 20 to 50 years, diagnosis of MS, scores 3-5 on the Expanded Disability Status Scale, stable spasticity scored as ≥ 2 for one of the joints on the Ashworth Scale, presence of ataxia and absence of neurological disorders (other than MS), cardiovascular diseases and infectious diseases, no exacerbation of MS clinical symptoms, no treatment with steroids two months before enrolment into the study, no physiotherapy two months before enrolment into the study, no history of alcohol and narcotic drug use, no depression (based on the Beck Depression Inventory), the lack of history of psychotherapy, the absence of cannabinoid use one week before enrolment into the study, and the absence of significant cognitive impairment (based on the Mini-Mental State Examination).

Exclusion criteria were non-compliance with the study protocol, lack of allergy to drugs, and no use of drugs other than prescribed drugs. First, sufficient information regarding the study objectives was given to the patients, and written consent to participate in the study was obtained from them if they volunteered to do so. Patients could withdraw from the study whenever they wished. All information collected from the patients from the time they were enrolled until they completed or withdrew from the study remained confidential. The teas were the same in terms of appearance and were labeled as A and B. During the study, each patient was contacted every week, and the necessary explanations were given to him/her about the general conditions and how to take the drugs (each bag was left in boiling water for 10 minutes and then consumed) and, if needed for clinical and paraclinical examinations, the patient was referred to the doctor and the laboratory. In addition, demographic data and clinical course of the disease, including age, gender, duration of MS, type of MS, corticosteroid use, and history of drug use by the patients, were recorded. Patients were randomly assigned to control and placebo groups using Random Allocation software. The control group patients received a bag of lavender tea containing 5-10 mcg of lavender essential oil (manufactured by Kooh Gol Company, where the bag was left in boiling water for 10 minutes and then consumed). Besides, the placebo group (receiving granule base without lavender essential oil produced by Kooh Gol Company) received a placebo tea daily for 60 days.

Before and after the intervention, spasticity was evaluated based on the Ashworth scale and the spasm repetition scale (The amount of spasm in the lower and upper limbs was considered).

Patients were randomly assigned to intervention and control groups based on Random Allocation software. The patients in the intervention group received one tea per day (manufactured by Kooh Gol Company), and those in the placebo group received one for 60 days. Before and after the intervention, spasticity was evaluated based on the Ashworth scale and spasm repetition scale. The International Cooperative Ataxia Rating Scale (ICARS) and the Berg Balance Scale (BBS) were used to evaluate ataxia.

The spasm repetition scale and Ashworth scale were used to evaluate spasms. The spasm repetition scale patients completed the spasm repetition scale five times a day, which evaluates the spasm in the previous 4 hours (0: No spasm, 1: 1-3 spasms, 2: 4-6 spasms, and 3: More than six spasms). The most accepted clinical scale for evaluating muscle tone is the Ashworth Scale. In this scale, muscle strength is scored based on a 5-point scale (0: No increase in muscle tone, 1: A slight increase in muscle tone, 2: A more significant increase in muscle tone but the movement is performed, 3: A significant increase in muscle tone and movement is complex, and 4: Constant contracture (stiffness of the limb when bending or pulling). This scale is used bilaterally for elbow flexors and extensors, wrist flexors and flexors, hip flexors, extensors and adductors, knee flexors and extensors, and plantar extensors and flexors. Then, the average scores of the left and right joints for all eleven joints in question will be calculated**.** The total score on this test ranges from 0 to 88 (in the present study, a minimum score of 2 and a maximum score of 88 were considered).

The validity and reliability of the Ashworth Scale have been confirmed in previous studies. To assess ataxia, the ICARS and BBS were used. The BBS is a gold standard for investigating functional balance. This test takes 15 to 20 minutes and consists of 14 balance tests. The minimum attainable score on the test is 0, and the maximum attainable score is 4. If the sum of attained points of the patient is less than or equal to 20, the patient will need a wheelchair. If the sum of the patient's points is greater than 20 and less than or equal to 40, the patient will need assistance in walking. Patients whose score is more than 40 can walk independently. The BBS is a valid and reliable test for MS patients, but using another scale in patients with ataxia symptoms in addition to the BBS is recommended. The ICARS is a valid and reliable scale for patients with cerebellar damage and has robust psychometric properties to assess balance. The validity and reliability of the BBS have been demonstrated in previous studies.

Data analysis

The data was entered into SPSS 16 software and analyzed using descriptive statistics (including mean, frequency percentage, and standard deviation) and analytical statistics (including independent *t* test, paired *t* test, and chi-square). The significance level (*P* value) was considered to be < 0.05.

Results

In the current clinical trial study, 84 patients with MS referred to Imam Ali Clinic (AS) and Kashani Hospital of Shahrekord were randomly assigned to two groups, namely, control and placebo. Regarding gender, both groups mainly included women. The mean standard deviation age of the control group was 38.02 ± 6.88 years, and that of the placebo group was 38.10 ± 6.76. The two groups were matched in terms of gender and age ($P > 0.05$) (Table 1). The mean Ashworth Scale score before and after the intervention and changes before and after the intervention were not significantly different between the control and placebo groups ($P > 0.05$). The comparison of Ashworth Scale scores before and after the intervention in each of the groups showed no significant difference ($P > 0.05$) (Table 2). The mean spasm scale score before and after the intervention and changes before and after the intervention were not significantly different between the control and placebo groups ($P > 0.05$). The comparison of spasm scale scores before and after the intervention in each of the groups showed no significant difference before and after the intervention ($P > 0.05$) (Table 2). The mean BBS score before and after the intervention was not significantly different between the control and placebo groups ($P > 0.05$), but the comparison of the changes in the BBS score before and after intervention in the two groups showed that the increase in the control group was significantly higher than that in the placebo group ($P < 0.05$).

A comparison of the BBS scores before and after intervention in each of the groups showed that both groups exhibited a significant increase after the intervention ($P < 0.01$) (Table 2). The mean ICARS score before the intervention, after the intervention, and the changes before and after the intervention were not significantly different between the two groups ($P > 0.05$). A comparison of the ICARS scores before and after intervention in each group showed that the mean ICARS score in the control group increased significantly after the intervention compared to before the intervention ($P < 0.05$). However, in the placebo group, no significant difference was observed in the score before and after the intervention ($P > 0.05$) (Table 2).

Table 1. Comparison of gender, education level and age between the control and placebo groups

Group	Variable	Drug (%) Frequency	Placebo (%) Frequency	*P* value
Gender	Male	10 (23.8)	11(26.2)	0.801(a)
	Female	32 (76.2)	31 (73.8)	
Age (y), mean±standard deviation		38.02 ± 6.38	38.6 ± 10.76	0.962(b)

Analyzes used (a) chi-square test (b) independent t-test.

Table 2. Comparison of Ashworth Scale, Spasms, Berg Balance Scale, and International Cooperative Ataxia Rating Scale scores in studied groups before and after intervention

Variable	Intervention time	Drug Group	Placebo group	*P* value
Ashworth	Before	17.16 ± 8.24	16.81 ± 7.99	0.830
	After	16.19 ± 7.42	17.05 ±6.79	0.590
P value		0.071	0.768	
Changes before and after		1.00± 3.5	0.16 ± 3.28	0.271
Spasms	Before	1.14 ± 0.57	1.1 ± 0.43	0.666
	After	1.07 ± 0.26	0.34 ± 1.07	1.00
P value		0.323	0.570	
Changes before and after		0.07±0.46	0.02 ± 0.27	0. 566
BBS	Before	41.6 ± 5.33	5.125 ± 42.45	0.455
	After	43.55±5.33	4.77 ± 43.31	0.83
P value		000.0 ***	0. 003 **	
Changes before and after		-1.95± 2.66	76.1 ± 86.0-	0.029*
ICARS	Before	-1.95± 2.66	10.88 ± 5.22	0.951
	After	10.24 ± 4.28	10.45 ± 3.52	0.803
P value		0.011 *	0.282	
Changes before and after		3.71 ± 1.73	0.43± 2.55	0.549

*$P<0.05$, **$P<0.01$, ***$P<0.0001$ (Independent *t*-test and paired *t*-test).

Discussion

MS is a degenerative disease of neurons in the central nervous system, which causes inflammation, myelin degeneration, and sometimes loss of axons. The use of medicinal plants in traditional medicine in Iran has a long history. Lavender is traditionally used as an analgesic, soothing, and anti-migraine treatment. The results of the present study showed that the two-month consumption of lavender tea leads to a significant increase in the average BBS index and a decrease in the average ICARS index in the control group compared to the placebo group. Also, the results of the present study showed that although the consumption of lavender tea did not lead to a significant decrease in spasm and Ashworth scale results, the reduction of these two indices was more pronounced in the control group than in the placebo group, showing that that lavender tea could lead to a decrease in spasm and Ashworth scale score. In the study of Hancianu et al (14) in rats induced with scopolamine, lavender essential oil showed potent antioxidant and anti-apoptotic activities.

Intraperitoneal injection of the essential oil for seven days increased antioxidant enzymes (SOD, GPX, and CAT) and decreased lipid (MDA) peroxidation in the anterior lobes of rats, which indicates the antioxidant potential of this essential oil. There were also no DNA break patterns in lavender-treated groups that showed anti-apoptotic activity of the plant. That study showed that lavender essential oil's antioxidant and anti-apoptotic activities were the primary mechanism of neuroprotective action against scopolamine-induced damage. In addition, it was revealed that there was a direct and significant correlation between SOD versus MDA and GPX and CAT versus MDA, indicating that increasing antioxidant defense and reducing lipid peroxidation can effectively protect neurons against neural oxidative stress following lavender essential oil treatment.

In the study of Wang et al (15), the protective effect of lavender oil on neuronal cells was investigated in a mouse ischemia model. In that study, point cerebral ischemia was induced in mice. Their results showed that lavender oil significantly reduced the degree of ischemia, MDA, carbonyl, and ROS. On the other hand, it reduced neuronal damage and increased the activity of SOD, CAT, and GSH-Px, as well as the GSH/GSSG ratio. Taken together, the results of that study showed that the neuroprotective effect of this extract against ischemic damage could depend on its antioxidant properties. The cited studies also show the protective effect of the lavender extract on neuronal cells and its antioxidant properties.

In a review by Alnamer et al, the methanolic lavender extract showed more significant sedative effects at 200, 400, and 600 mg/kg (by oral administration) than diazepam. The lavender extract also showed hypnotic effects in mice at 800 and 1000 mg/kg (16). In the study of Azizzadeh Delshad et al, the lavender extract's effect in preventing spinal motor neuron damage was investigated. After axotomy, rats received different doses of lavender extract for three days. Lavender treatment for three days after axotomy increased viability and decreased apoptosis of motor neurons, and the best effect was produced after administration of the extract at 500 mg/kg. The study's results showed that removal of the sciatic nerve in all groups caused programmed death and a significant reduction of motor neurons and that lavender extract treatment led to a decrease in apoptotic neuron count, yet the difference was not statistically significant. It could be argued that more time was needed for the difference to turn significant (17).

Various studies have also addressed the protective effects of lavender extract on neuronal cells *in vitro*, including the study conducted by Xu et al (18). Lavender extract at 100 mg/kg concentration improved cognitive function in mice stimulated with scopolamine. However, 12 µg/ml protected PC12 (neuronal-like) cells against H2O2-induced damage by reducing LDH, NO, and intracellular accumulation of ROS and MMP. H2O2, as a source of ROS production, can lead to oxidative stress in various cells and ultimately lead to apoptosis and changes in the cell signaling system at high concentrations. Free oxygen radicals can also react with NO and produce reactive nitrogen species, ultimately leading to the oxidation of lipids, DNA, and proteins. The proximity of PC12 cells to H2O2 significantly decreases cell viability and increases LDH release from cells. The proximity of PC12 cells with different concentrations of lavender extract protects cells against the damage caused by H2O2 and prevents the increase of ROS and NO. This extract could also prevent the reduction of MMP in PC12 cells exposed to H2O2, altogether indicating the protective effect of this extract on neuronal cells. Given the presence of many flavonoids in the lavender plant, its neuroprotective effects can be attributed to these flavonoids' antioxidant and anti-inflammatory role, which leads to the reduction of neuronal cell death and protects neurons against damaging factors. For the neuroprotective effects to be produced, the plant's flavonoids must cross the blood-brain barrier to an acceptable extent, and their amount in the spinal cord must suffice to affect the function of neurons. It has been reported that following a single intraperitoneal injection of flavonoids in rats, significant amounts of these substances are found in the animals' brains, which confirms this argument. So it can be said that flavonoids can easily pass through the blood-brain barrier in living organisms (19). In another study conducted by Neelamma et al, compounds such as flavonoids, tannins, terpenes, rutin, quercetin, and anthocyanins could reduce spasticity by affecting the central nervous system in patients with MS (20). Therefore, the presence of these compounds in lavender can reduce ataxia and spasm in people with MS.

In the study of Rahnama-ye Bashm et al on the effect of the hydroalcoholic lavender extract on the learning and memory of diabetic rats, the extract of this plant, along with reducing blood sugar, resulted in a significant reduction in the number of errors related to diabetes-related damage in learning and memory tests. Further investigations showed that the antidiabetic effects of lavender were due to the presence of phenolic and flavonoid compounds in this plant, and due to the high antioxidant properties of this plant, it can lead to improved memory and learning in diabetic rats, which sufficiently indicates its neuroprotective effects (21). In addition to the effects of lavender, we can also mention its moderating effects. In a study conducted by Standen et al, they showed that the essential oil of this plant at low concentrations enhanced the activity of natural killer cells and peripheral blood mononuclear cells (22).

In another study conducted by Azadmehr et al, the results of lymphocyte proliferation in a cell proliferation test showed poor peripheral blood lymphocyte-proliferative activity following treatment with lavender extract at 50 µg/mL and less. No significant cell-proliferative effect was observed at concentrations over 50 µg/mL. The researchers observed that lavender extract could significantly reduce TNF-α as one of the critical cytokines and mediators in inflammatory responses (23).

Conclusion

Given the cited studies' findings, although lavender could not lead to a significant reduction in spasm and ataxia in patients with MS, the reduction in spasm and ataxia, as well as the higher rate of changes in the group receiving the drug compared to placebo can show the achievement of more pronounced impacts with increasing the duration of intervention. The presence of various compounds in lavender, especially flavonoids, can lead to the reduction of inflammation and protective effects on neurons, which in this way can reduce the amount of spasm and ataxia in patients with MS; however, it is necessary to conduct further studies and also investigate the active ingredients of this plant in other studies.

The present study showed that lavender tea leads to a decrease in ataxia-related indicators in patients with MS, which can be due to the presence of flavonoids and antioxidant, anti-inflammatory, and neuroprotective properties of the plant. However, increasing the duration of the treatment led to a significant increase in antispasmodic and anti-ataxia effects in MS patients, which is recommended to be considered in studies conducted in the future.

Acknowledgments

This article was derived from a student's thesis entitled Evaluation of the effect of lavender tea on the rate of ataxia and spasm in patients with multiple sclerosis. We appreciate the financial support of the Research and Technology Deputy of the University, the Clinical Research Development Unit of Shahrekord Ayatollah Kashani Hospital, and all those who assisted in implementing the study.

Authors' Contribution

Conceptualization: Nahid Jivad, Zahra Forouzandeh Shahrakei.
Data curation: Zahra Forouzandeh Shahrakei, Armin Khaghani.
Formal analysis: Mahbubeh Setorki.
Funding acquisition: Nahid Jivad, Zahra Forouzandeh Shahrakei.
Investigation: Armin Khaghani, Mahbubeh Setorki.
Methodology: Nahid Jivad, Mahbubeh Setorki.
Project administration: Nahid Jivad, Mahbubeh Setorki.
Resources: Armin Khaghani, Nahid Jivad.
Supervision: Nahid Jivad, Zahra Forouzandeh Shahrakei.
Validation: Nahid Jivad, Armin Khaghani.
Visualization: Nahid Jivad.
Writing–review & editing: Nahid Jivad, Zahra Forouzandeh Shahrakei.

Competing Interests

The authors declare no conflict of interest.

Ethical Approval

This study was approved by the Ethical Committee of Shahrekord University of Medical Sciences with Ethics code IR.SKUMS.REC.1398.078.

References

1. Goldenberg MM. Multiple sclerosis review. P T. 2012;37(3):175-84.
2. Bach JF. Infections and autoimmune diseases. J Autoimmun. 2005;25 Suppl:74-80. doi: 10.1016/j.jaut.2005.09.024.
3. Sahraian MA, Khorramnia S, Mohammad Ebrahim M, Moinfar Z, Lotfi J, Pakdaman H. Multiple sclerosis in Iran: a demographic study of 8,000 patients and changes over time. Eur Neurol. 2010;64(6):331-6. doi: 10.1159/000321649.
4. Etemadifar M, Nourian SM, Akbari M, Abtahi SH, Nasri P, Fereidan-Esfahani M. The distinctive contrast of multiple sclerosis epidemiology between Persians and Armenian minority community of Isfahan city, Iran. Neurol Sci. 2015;36(4):657-8. doi: 10.1007/s10072-014-2013-0.
5. Owens T. The complex immunology of multiple sclerosis. Mult Scler. 2014;20(8):1023-4. doi: 10.1177/1352458514521312.
6. Prusinowska R, Śmigielski KB. Composition, biological properties and therapeutic effects of lavender (*Lavandula angustifolia* L). A review. Herba Pol. 2014;60(2):56-66. doi: 10.2478/hepo-2014-0010.
7. Da Porto C, Decorti D, Kikic I. Flavour compounds of *Lavandula angustifolia* L. to use in food manufacturing: comparison of three different extraction methods. Food Chem. 2009;112(4):1072-8. doi: 10.1016/j.foodchem.2008.07.015.
8. Cavanagh HMA, Wilkinson JM. Lavender essential oil: a review. Aust Infect Control. 2005;10(1):35-7. doi: 10.1071/hi05035.
9. Selamoglu Z, Alinia-Ahandani E, Alizadeh-Tarpoei Z, Hajipour S, Rafeie F. A Mini-Review of the Medicinal Properties of the Lavender Plant and Ways to Increase Its Effective Compounds. jhehp 2023; 9 (1) :1-4. doi: 10.52547/jhehp.9.1.1.
10. Basch E, Foppa I, Liebowitz R, Nelson J, Smith M, Sollars D, et al. Lavender (*Lavandula angustifolia* Miller). J Herb Pharmacother. 2004;4(2):63-78.
11. Denner SS. *Lavandula angustifolia* Miller: English lavender. Holist Nurs Pract. 2009;23(1):57-64. doi: 10.1097/01.HNP.0000343210.56710.fc.
12. Raut JS, Karuppayil SM. A status review on the medicinal properties of essential oils. Ind Crops Prod. 2014;62:250-64. doi: 10.1016/j.indcrop.2014.05.055.
13. Husseini Y, Sahraei H, Meftahi GH, Dargahian M, Mohammadi A, Hatef B, et al. Analgesic and anti-inflammatory activities of hydro-alcoholic extract of *Lavandula officinalis* in mice: possible involvement of the cyclooxygenase type 1 and 2 enzymes. Revista Brasileira de Farmacognosia. 2016;26(1):102-8. doi: 10.1016/j.bjp.2015.10.003.
14. Hancianu M, Cioanca O, Mihasan M, Hritcu L. Neuroprotective effects of inhaled lavender oil on scopolamine-induced dementia via anti-oxidative activities in rats. Phytomedicine. 2013;20(5):446-52. doi: 10.1016/j.phymed.2012.12.005.
15. Wang D, Yuan X, Liu T, Liu L, Hu Y, Wang Z, et al. Neuroprotective activity of lavender oil on transient focal cerebral ischemia in mice. Molecules. 2012;17(8):9803-17. doi: 10.3390/molecules17089803.
16. Alnamer R, Alaoui K, Bouidida el H, Benjouad A, Cherrah Y. Sedative and hypnotic activities of the methanolic and aqueous extracts of *Lavandula officinalis* from Morocco. Adv Pharmacol Sci. 2012;2012:270824. doi: 10.1155/2012/270824.
17. Azizzadeh Delshad A, Naseri M, Parvizi M, Fattah N, Sharayeli M. The Iranian traditional herbal medicine ostokhodus can prevent axotomy-induced apoptosis in spinal motoneurons in neonate rats. J Med Plants Res. 2011 Sep 16;5(18):4446-51.
18. Xu P, Wang K, Lu C, Dong L, Gao L, Yan M, et al. Protective effect of lavender oil on scopolamine induced cognitive deficits in mice and H2O2 induced cytotoxicity in PC12 cells. J Ethnopharmacol. 2016;193:408-15. doi: 10.1016/j.jep.2016.08.030.
19. Youdim KA, Qaiser MZ, Begley DJ, Rice-Evans CA, Abbott NJ. Flavonoid permeability across an in situ model of the blood-brain barrier. Free Radic Biol Med. 2004;36(5):592-604. doi: 10.1016/j.freeradbiomed.2003.11.023.
20. Neelamma G, Swamy BD, Dhamodaran P, Vanitha B. Bioactive molecules present in plants play a potential role in the treatment of spasticity in multiple sclerosis: a new perspective in future. Saudi J Med Pharm Sci. 2016;2(6):122-8. doi: 10.36348/sjmps.2016.v02i06.001.
21. Rahnama-ye Bashm M, Rahmati B, Poorgholam M. The effect of *Lavandula dentata* aerial parts hydroalcoholic extract on learning and memory in male streptozotocin-induced diabetic rat. Daneshvar Medicine. 2019;27(141):1-8. doi: 10.22070/27.141.1. [Persian].
22. Standen MD, Connellan PA, Leach DN. Natural killer cell activity and lymphocyte activation: Investigating the effects of a selection of essential oils and components in vitro. Int J Aromather. 2006;16(3):133-9. doi: 10.1016/j.ijat.2006.09.006.
23. Azadmehr A, Hajiaghaee R, Rezazadeh S, Afshari A, Kiani Amin M, Baradaran B, et al. Evaluation of *Lavandula officinalis* extract on lymphocyte proliferation and tumor necrosis factor-alpha production. J Med Plants. 2011;10(38):142-7.

Cite this article as: Jivad N, Forouzandeh Shahrakei Z, Khaghani A, Setorki M. The protective effects of lavender herbal tea on spasticity and ataxia in patients with multiple sclerosis. Future Nat Prod. 2023;9(1):25-30. doi: 10.34172/fnp.2023.04.