The 6th international- 20th Arabic conference for Home Economics
Home Economics and Educational quality assurance December 23rd -24th, 2018
http://homeEcon.menofia.edu.eg

Journal of Home Economics

ISSN 1110-2578

Therapeutic Effect of Selected Plants on Autistic Rats

Adel A. Ahmed,Nehad R. Eltahan,Sara A. Elsherif, MohamedA. Elsaadany.
Department of Nutrition &Food Science,Faculty of Home Economics, Menufia University, Egypt

Abstract

Introduction: Autism is a lifelong neurodevelopmental disorder impairs social reciprocity and communication. The incidence of autism has been rising at an alarming rate over the past three decades, although the prevalence seems to differ between countries. **Aim:** The aim of this Study is to investigate the effects of gluten-free casein freediet (GFCF)fortified with/out Sage (*Salvia officinalis*) orGinkgo *(Ginkgo Biloba)*as a possible aid in the improvement of neurological, behavioral, neurochemical and histopathological changes associated with autism.**Methods:** Agroup of pregnant rats received a single intraperitoneal injection of 600 mg/kg Valproic acid (VPA) then their offspring were randomly distributed into 4 groups each 5 rats as following;group 1: fed with standard diet (VPA-SD) and served as control positive,group 2: fed with GFCF diet(VPA-GFCF diet), group 3: fed with GFCF diet plusSage (VPA-GFCF+ Sage), and group 4: fed with GFCF diet (VPA-GFCF + Ginkgo), where five offspring from healthy pregnant rats (not injected with VPA), fed standard diet were served as control negative group, Therapeutic potential ware measured using;the feed efficiency ratio (FER), serum level of (SOD),(T.A.C), (GSH), (MDA) and (5-HT) in addition to histopathological study of the cerebellum. **Results:**results showed that diet with Ginkgo significantly illustrated the highest effect on the FER compared to all other groups ($p<0.05$).There weremild differences in serummarkers,where sage group expressed the highest effect followed by ginkgo and then the GFCF diet.

Histopathological of Cerebellum of rat from control positive showing necrosis, pyknosis and abnormal shape of Purkinje cells where Ginkgo group showingan improvements in necrosis and pyknosis. **Conclusions:** GFCF diets with herbal supplement demonstrated benefits effects in the treatment of disorders associated with autism.

Keywords : Autism, Gluten, Casein, Sage, Ginkgo

Introduction

Autism spectrum disorder (ASD) is a complicated neurological disorder with impairments within the social relationship **Maski*et al.*, 2011.**It considered asa multi-factorial disorder that is influenced by immunological, genetic and environmental factors. The prevalence lately increased with a ratio of 4:1 boysto girls, **Loomes*et al.*, 2017**.ASD children suffer from abnormal gluten and casein-related digestive enzymes and increased gut permeability. Without adequate levels of digestive enzymes, peptides resulting from gluten and casein fail to become amino acids in huge numbers. Increased gut permeability then allows the peptides to leak into the bloodstream, where they circulate and eventually cross the brain blood barrier**Elder *et al.*, 2006;Mulloy *et al.*, 2010.** Adequate nutrition help to the relief, both digestive and metabolic as well as psychological changes **Meguid *et al.*, 2017.** The gluten-free/casein-free (GFCF) diet is a common dietary intervention for autistic children. The Diet Mechanism it is according to the opioid-excess theory**Compart and Laake, 2012.**

The increased of oxidative stress have been reported in autism. This can be a common pathogenic mechanism in many major psychiatric disorders because the brain has a relatively greater vulnerability to oxidative damage. Oxidative stress can participate in the evolution and clinical manifestations of autism**Meguid *et al.*, 2011.**

Valproic acid (VPA) is an anti-epileptic drug and is used to treat manic-type bipolar disorder and migraine. The use of VPA through pregnancy is associated with a raised incidence of autism. Based on this observation, prenatal exposure to VPA has been used as a reliable animal model for autism. This model shows neuroanatomical, and biochemical results that summarize the main characteristics of autistic children **Bambini-Junior*et al.*, 2011.**The brain of autistic

children shows several neuropathological abnormalities like reduced number of Purkinje cells in the cerebellum, while neurochemical, the most consistent finding in ASD is an increase in blood serotonin. Interestingly, serotonin is known to play an active role in brain development **Abdelrahman,2008.**

Ginkgo biloba (*Ginkgo Biloba*)belonging to the Ginkgoaceae family, The active componantsincludes flavones, ginkgolides,andbilobalides**Chen *et al.*, 1998; Hauser*et al.*, 2002.**Flavonoid glycosides are antioxidants that can protect neurodegenerative diseases due to antioxidant stress **Ramassamy, 2006.**The extract has effects on improving blood flow to organs and tissues to protect against free radicals that are related to the nervous system disorders **Smith *et al.*, 1996.** It is effective on the neurotransmitter system and on the antioxidant effect that related to the pathogenesis of ASD. Ginkgo Biloba, effective as an additional treatment of deviation in reciprocal social communication, verbal and non-verbal communication is the key to the characteristics of ASD**Niederhofer, 2009; HasanZadeh *et al*., 2012.**

Sage (*Salvia officinalis*)belongs to the mint family.There are sage types that studies suggest to help in the treatment of various diseases such as dementia, autism, lupus, cancer, heart disease, obesity, includes depression and diabetes **Hamidpour*et al.*, 2014.** In addition to cholinergic activity, a wide range of activities has reported for the Salvia, these include tannic acid, oleic acid, ursolic acid, cornsole, cornsolic acid, fumaric acid, chlorogenic acid, caffeic acid, niacin, nicotinamide, flavones, flavonoid glycosides, and estrogenic substances**.** It is used in heritage medicine for reducing oxidative stress and free radical damage**Anamaria*et al*.,2013.**

Material and Method:

Materials

This experiment was conducted using Albino Wistar rats at national animal welfare,the national research center, dokki, Egypt.Standard or basal diet was formulated to contain 14% casein, 10% sucrose, 5% corn oil, 5% fiber (cellulose), 3.5% mineral mixture, 1% vitamin mixture, 0.25% choline chloride, 0.3 % D-L methionine, and

60.95% corn starch **Reeves *et al.*,1993.**GFCF was prepared by replacing gluten orcaseinwith soy protein. VPA was purchased from (Sigma, St. Louis, MO). Dried *Salvia officinalis* was purchased from Environment Fund and the Community Service, Research and experiences of medicinal plants and aromatic Center, Faculty of Pharmacy, Cairo University, Egypt, *Ginkgo biloba* extract 260 mg/5ml was purchased from EIMC united pharmaceuticals for EMA pharm pharmaceuticals, Egypt.

Experimental design

The experiments were conducted according to national animal welfare standards and the ethics committee for institutional animals. On the 12th day of pregnancy, 20 pregnant Albino Wistar rats weighing 150-200g have been given a single intraperitoneal injection of 600 mg/kg VPA (using VPA dissolved in a saline pH 7.31at concentration of 50mg/ml) **Kim *et al.*, 2013**.

At the 21st of birth, the offspring male, were randomly distributed into 4 groups each 5 rats as following; group1: fed with standard diet (VPA-SD) and served as control positive, group 2: fed with GFCF diet (VPA-GFCF diet), group 3: fed with GFCF diet plus Sage (VPA-GFCF+ Sage), and group 4: fed with GFCF diet (VPA-GFCF + Ginkgo), where five offspring from healthy pregnant rats (injected only saline), fed standard diet were served as control negative group.Groups were given sage or ginkgo as fresh solution daily. Experiment was continued for 40 days. Therapeutic potential ware measured using; the feed efficiency ratio (FER), serum level of (SOD), (T.A.C), (GSH), (MDA) and (5-HT) in addition to histopathological study of the cerebellum.

Plant extraction and phenolic determination:

The sage tea was routinely prepared by pouring 300 mL of boiling waterwith4g of dried plant material and allowing steep for 5 min. Where Ginkgo extract 260 mg/5 ml was adjusted to 300 ml with water. These solutions are given to rats instead of drinking water **Tisserand & Young, 2013.** Phenolic content was determined in water extract using Folin-Ciocalteu reagent and Gallic acid as a standard where Flavonoid content was determined Spectrophotometrically according to a standard method**Quettier-Deleu *et al.*, 2000 .**

Biological Evolution:

During the experimental period (40days), the diet consumed was recorded every day and body weight was recorded every week. The food efficiency ratio (FER), were determined according to**chapman*et al.*, *1959***.The catalase is determined according to **Aebi,1984**superoxide dismutases (SOD) according to**Nishikimi*et al*., 1972,** malondialdehyde (MDA) is determined according to **Ohkawa*et al*., 1979**. Total antioxidant assay is determined according to**koracevic, *et al*, 2001,**glutathione (GSH) determined according to **Beutler*et al*., 1963.**

Histopathology Examinations

Small specimens of the organs brain were taken from each experimental animal, fixed in neutral buffered formalin, dehydrated in ascending concentration of ethanol (70.80 and 90%), cleared in xylene and embedded in paraffin. Sections of 4- 6 mm thicknesses were prepared and stained with hematoxylin and eosin according to**Bancroft*et al*., 1996.**

Statistical analysis:

Data were expressed as Mean ±SD. T-test and analysis of Variance (ANOVA)between groups mean were calculated using the SPSS software for Windows **Base, 2010.**

Results

Flavonoid and phenolic content of the sage and ginkgo illustrated in **Figure (1)**. As shown Ginkgo was significantly higher ($p \leq 0.05$) in both flavonoid and phenolic content compared to sage.

The feed efficiency ratio**Figure (2)**, rats of control negative grouprecorded higher FERin comparison with control positive group ($p \leq 0.05$). On the other hand, the rats of GFCF diet with/out herbal administrationshowed a significant elevation in FER compared to the control negativegroup. FER of Ginkgogroup was the highest compared to all other groups ($p \leq 0.001$).

Serum level of(SOD),(TAC), (GSH), (MDA) and (5-HT**)**values from different treated groups are illustrated in **Figure (3)**. As represented there washarmony between results obtained from different markers.

Control positive groupshowed asignificant reduction of all markers compared to control negative group ($p<0.05$). On the other hand, a significant increase was noticedby the administration of the GFCF diet, Ginkgo and Sage.Diet with sage expressed the highest effect followed by ginkgo and then the GFCF diet.

Histopathological study of the cerebellum ofdifferent treated groups showed the Purkinje cellslayer lying between the superficialmolecular layer and a deep granular layer of Cerebellum. Cerebellum of rat from control negative **(Figure. 4a)** showing no histopathological changes. Where Cerebellum of rat from control positive **(Figure. 4b)** showing necrosis and pyknosis and abnormal shape of Purkinje cells as well as loss of Purkinje cells. Cerebellum of rat from GFCF diet group showing slight modulation in a number of necrosis of sporadic Purkinje cells compared to positivecontrol. Cerebellum of rat from GFCF diet+sage group showing necrosis, pyknosis and atrophy of Purkinje cells and loss of Purkinje cells and chromatolysis. GFCF diet + ginkgo group showing improvements in necrosis and pyknosis of somePurkinje cells **(Figure. 4c-4e)**

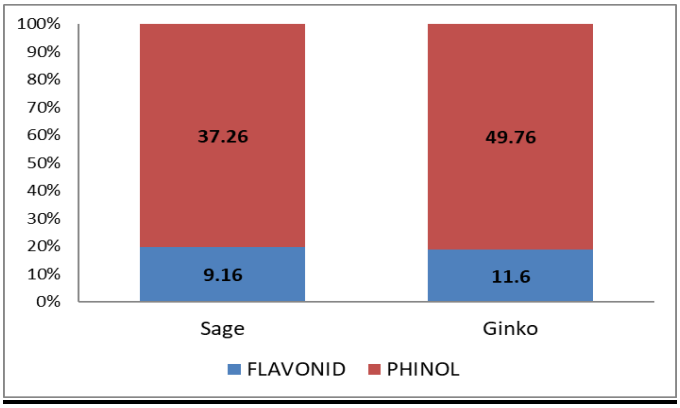


Figure (1):Flavonoid and phenolic contents of the sage and ginkgo. Data represented as mean of three replicates

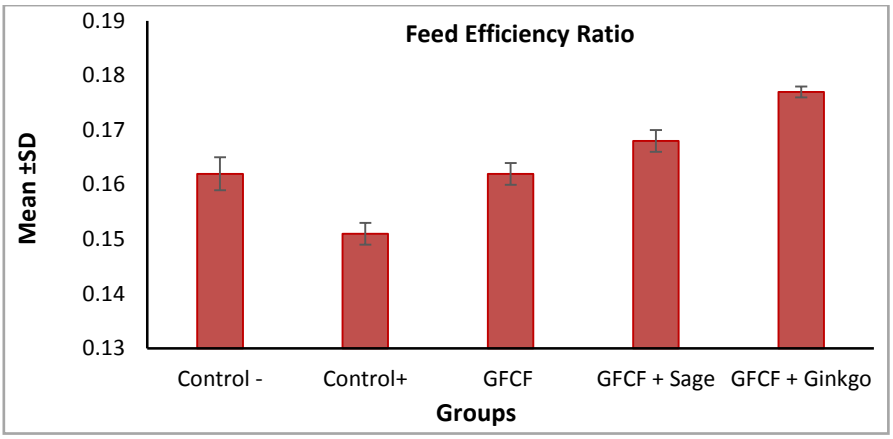
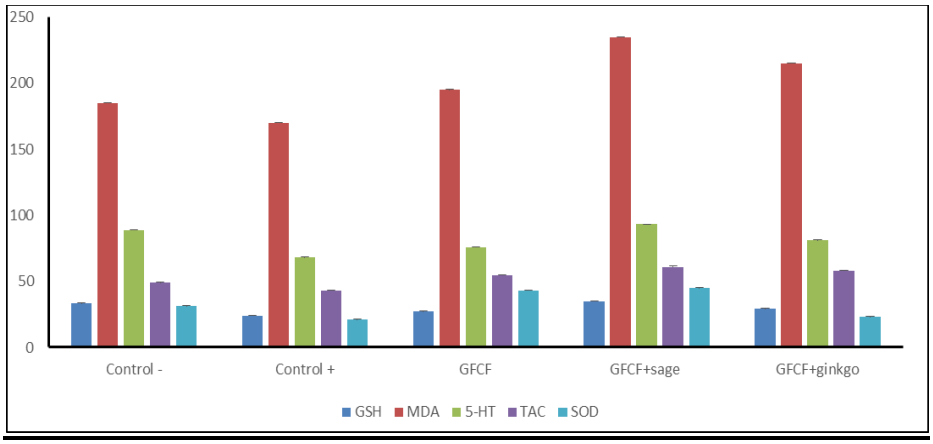


Figure (2): The Feed Efficiency Ratio (FER)of different groups. Data represented as mean ± SD.



Figure(3): Serumlevel of(SOD),(TAC), (GSH), (MDA) and (5-HT)in different treatedgroups. Data represented as mean of three replicates± SD.

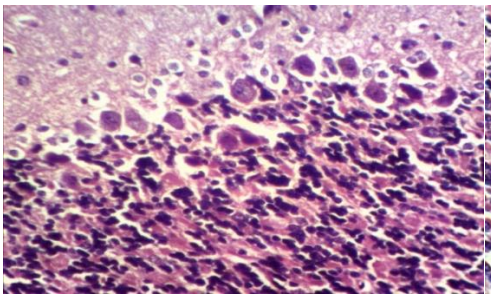


Fig. (1a): Cerebellum of rat from control- group showing no histopathological changes. Note normal Purkinje cells (H & E X 400).

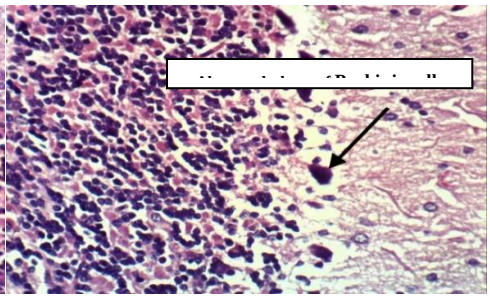


Fig.(1b): Cerebellum of rat from control+group showing necrosis and pyknosis and abnormal shape of Purkinje cells and loss of Purkinje cells

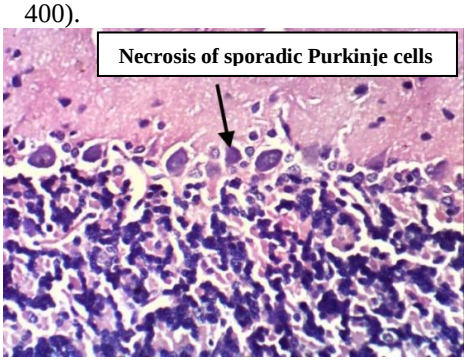


Fig. (1c): Cerebellum of rat from GFCF group showing necrosis of sporadic Purkinje cells

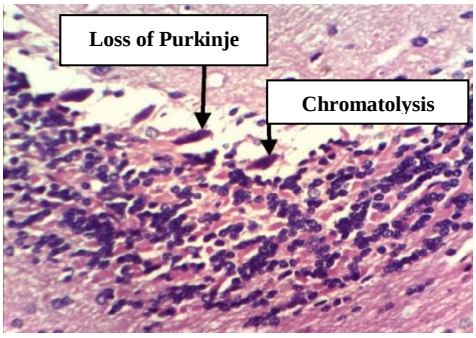


Fig.. (1d): Cerebellum of rat from GFCF+sage group showing necrosis, pyknosis and atrophy of Purkinje cells and loss of Purkinje cells and chromatolysis

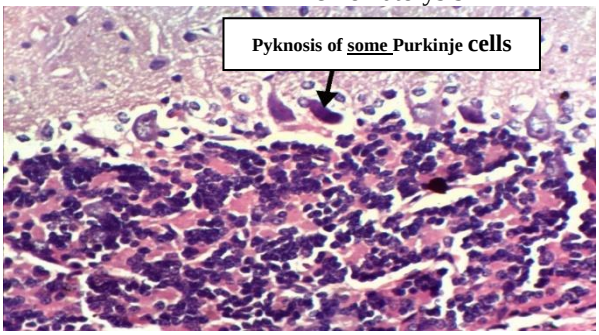


Fig. (4e) : Cerebellum of rat from GFCF+ginko group showing necrosis and pyknosis of some Purkinje cells.

Figure (4): The histopathological changes indifferent treated groups.

Discussion

The present study confirmed the simulation of an experimentally induced animal model of ASD to the human one as regards neurochemical and histopathological parameters.It can be also used to study the effect of oraladministration of plant extract on rats memory, concentration, mental alertness, and a decrease in mental fatigue **Farooqui,2013**.

Rats fed GFCF diet exhibiteda higher FER. Thesedataare agreed with **Whiteley*et al.,* 2013**who suggested that the use of GFCF diet ameliorate symptoms, gastrointestinal disturbances,and improved developmental outcome.Moreover,when sage or ginkgo added to the diet the FERbecomeshigher. Ginkgo showed the highest effect on FER compared to all other groups. This date was agreed with **Hasanzadeh,*et al.*,2012**who reported anincrement in food intake by 26%of autistic children receivingGinkgo biloba added to their medication. Increased appetitemay explain the high FER, especially in rats,received Ginkgoin their diet.

Patients with ASD and co-occurring gastrointestinal disturbances are at higher risk for oxidative stress. Oxidative stress in addition to other factors could contribute to the development and clinical manifestations of ASD**Gorrindo*et al*., 2013**. Elevatedlevel of serum (SOD), (T.A.C), (GSH), (MDA) and (5-HT)reported in autistic patients as indication for oxidative stress. The pathogenic mechanism of psychiatric disorders implicated oxidative stress because of the high risk of brain damage through oxidative stress **Annelies*et al*, 2018.**

Data showed that sage followed by ginkgo werehelpful in reducing the oxidative stress. Sam results werereportedby**Hamidpour*et al.*, 2014 w**ho demonstrated that sage had a therapeutic effect in treating several diseases such as dementia, ASD, lupus, cancer, heart disorder, obesity, incorporates depression and diabetes**Hasanzadeh*et al.*, 2012.**Ginkgo was effective on the neurotransmitter system and the antioxidant effect that may be related to the pathogenesis of ASD. Ginkgo also effective as a complementary treatment to the abnormality in reciprocal social communication, verbal and nonverbal communication**Bahmani *et al.*, 2016.**Ginkgo extract exhibited its effect

through increasing the flow of blood to organ and tissue to protect from free radicals **Marchezan *et al*.,2018**.

Histopathology result of the present study confirmed that the most consistent neurological abnormalities in ASD is marked Purkinje cell loss in the cerebellum,**Valko, 2007.** The loss of Purkinje cells leads to social behavior deficits and increased and increased repetitive behaviors.Traditionally, the neurological basis of ASD has thought to lie mainly in the cerebral cortex**Fatemi*et al*.,2012.**The cerebellum has vast interconnections with the cerebral cortex and other parts of the brain, and evidence suggests that the cerebellum modulates and coordinates different functions throughout the brain**Nadeem *et al*., 2019**.

The brain in the early part of a development has a high risk of oxidative stress that's leading to the pathogenesis of neurodevelopment disorders and neuropsychiatric disorders similar to ASD**Reith *et al*., 2013.**Sage and ginkgo extract improve and protectPurkinje cell loss in the cerebellum by acting as potent antioxidants. Both extracts prevented the loss of Purkinje cells and retained the number and the shape of the cells. **BistandBhatt.,2010;Dhar*et al*., 2018.**

References

Abdelrahman, O.R.(2008):*Neopterin and 8-isoprostane as a marker for ASD in children.* M. Sc. Thesis, Microbiology. Dept., Fac. medicine., Alexandria Univ., Egypt.

Aebi, H. (1984): Catalase in vitro. In *Methods in Enzymology* (Vol. 105, pp. 121-126). Academic Press.

Anamaria, P.; Muste, S.; Mureşan, C.; Pop, C.and Salanţă, L. (2013): Comparative study regarding the importance of sage *(Salvia officinalis L.)* in terms of antioxidant capacity and antimicrobial activities. *Hop Med. Plants*, *1*(2), 41-42.

Annelies, V.; Harry, R.; Ines, W.; Annelies, B.;and Tess, D. B. (2018). Evaluation of Biomarkers of Oxidative Stress in Attention-Deficit/Hyperactivity Disorder (ADHD). *J Mol Biomark Diagn*, *9*(390), 2.

Bahmani, M.; Sarrafchi, A.; Shirzad, H.;and Rafieian-Kopaei, M. (2016): Autism: Pathophysiology and promising herbal remedies. *Current pharmaceutical design*, *22*(3), 277-285.

Bambini-Junior, V.; Rodrigues, L.; Behr, G. A.; Moreira, J. C. F., Riesgo, R., and Gottfried, C. (2011): An animal model of autism induced by prenatal exposure to valproate: behavioral changes and liver parameters. *Brain research*, *1408*, 8-16.

Bancroft, G. D.; Stevens, A.;and Turner, D. R. (1996): Theory and practice of technique. *Churchill: Livingston. New York*.

Base, P. S.(2010). Statistics, Core System User's Guide. *SPSS Inc.*, *Chicago, IL*.

Beutler, E.; Duron, O.;and Kelly, M. B. (1963): Colorimetric method for determination of glutathione reduced. *J. Lab. Clin. Med*, *61*, 882.

Bist, R., and Bhatt, D. K. (2010): Augmentation of cholinesterases and ATPase activities in the cerebellum and pons-medulla oblongata, by a combination of antioxidants (resveratrol, ascorbic acid, alpha-lipoic acid and vitamin E), in acutely lindane intoxicated mice. *Journal of the neurological sciences*, *296*(1-2), 83-87.

Chapman, D. G.; Castillo, R., and Campbell, J. A. (1959): Evaluation of protein in foods: 1. A method for the determination of protein efficiency ratios. *Canadian Journal of Biochemistry and Physiology*, *37*(5), 679-686.

Chen, P.; Su, X. L.; Nie, L. H.; Yao, S. Z.;andZeng, J. G. (1998): Analysis of Ginkgolides and Bilobalides in Ginkgo biloba L. extract for its production process control by high-performance liquid chromatography. *Journal of chromatographic science*, *36*(4), 197-200.

Compart, P. and Laake, D. (2012): The Kid-Friendly ADHD and Autism Cookbook, Updated and Revised: The Ultimate Guide to the Gluten-Free, Casein-Free Diet. Fair Winds Press (MA).

Dhar, P.; Kaushal, P. and Kumar, P. (2018): Antioxidant supplementation upregulates calbindin expression in cerebellar Purkinje cells of rat pups subjected to postnatal exposure to sodium arsenite. *Brain research, 1690*, 23-30.

Elder, J. H.; Shankar, M.; Shuster, J.; Theriaque, D.; Burns, S. and Sherrill, L. (2006): The gluten-free, casein-free diet in autism: results of a preliminary double blind clinical trial. Journal Of Autism And Developmental Disorders, 36(3), 413-420.

Farooqui, A. A. (2013): Beneficial effects of ginkgo biloba in neurological disorders. In *Phytochemicals, Signal Transduction, and Neurological Disorders* (pp. 237-270). Springer New York.

Fatemi, S. H.; Aldinger, K. A.; Ashwood, P.; Bauman, M. L.; Blaha, C. D.; Blatt, G. J. and Estes, A. M. (2012): Consensus paper: pathological role of the cerebellum in autism. *The Cerebellum*, *11*(3), 777-807.

Gorrindo, P.; Lane, C. J.; Lee, E. B.; McLaughlin, B. and Levitt, P. (2013): Enrichment of elevated plasma F2t-isoprostane levels in individuals with autism who are stratified by presence of gastrointestinal dysfunction. *PloS one*, *8*(7), e68444.

Hamidpour, M.; Hamidpour, R.; Hamidpour, S. andShahlari, M. (2014): Chemistry, Pharmacology, and Medicinal Property of Sage (Salvia) to Prevent and Cure Illnesses such as Obesity, Diabetes, Depression, Dementia, Lupus, Autism, Heart Disease, and Cancer. *Journal of Traditional and Complementary Medicine*, *4*(2), 82-88. doi:10.4103/2225-4110.130373

Hasanzadeh, E.; Mohammadi, M. R.; Ghanizadeh, A.; Rezazadeh, S. A.; Tabrizi, M.; Rezaei, F. andAkhondzadeh, S. (2012): A double-blind placebo controlled trial of Ginkgo biloba added to risperidone in patients with autistic disorders. *Child Psychiatry and Human Development*, *43*(5), 674-682.

Hauser, D.; Gayowski, T.and Singh, N. (2002): Bleeding complications precipitated by unrecognized Gingko biloba use after liver transplantation. *Transplant international*, *15*(7), 377-379.

Kim, P.; Park, J. H.; Kwon, K. J.; Kim, K. C.; Kim, H. J.; Lee, J. M. and Shin, C. Y. (2013). Effects of Korean red ginseng extracts on neural tube defects and impairment of social interaction induced by prenatal exposure to valproic acid. *Food and Chemical Toxicology*, *51*, 288-296.

Koracevic, D.; Koracevic, G.; Djordjevic, V.; Andrejevic, S. and Cosic, V. (2001): Method for the measurement of antioxidant activity in human fluids. *Journal of Clinical Pathology*, *54*(5), 356-361.

Loomes, R.; Hull, L. and Mandy, W. P. L. (2017): What is the male-to-female ratio in autism spectrum disorder? A systematic review

and meta-analysis. *Journal of the American Academy of Child and Adolescent Psychiatry*, *56*(6), 466-474.

Marchezan, J.; dos Santos, E. G. A. W.;Deckmann, I. and dos Santos Riesgo, R. (2018): Immunological Dysfunction in Autism Spectrum Disorder: A Potential Target for Therapy. *Neuroimmunomodulation*, 1-20.

Maski, K. P.; Jeste, S. S.;and Spence, S. J. (2011): Common neurological co-morbidities in autism spectrum disorders. *Current Opinion In Pediatrics*, *23*(6), 609.

Meguid, N. A.; Anwar, M.; Bjørklund, G., Hashish, A.; Chirumbolo, S.; Hemimi, M. and Sultan, E. (2017): Dietary adequacy of Egyptian children with autism spectrum disorder compared to healthy developing children. *Metabolic Brain Disease*, *32*(2), 607-615.

Meguid, N. A.; Dardir, A. A.; Abdel-Raouf, E. R. and Hashish, A. (2011): Evaluation of oxidative stress in autism: defective antioxidant enzymes and increased lipid peroxidation. *Biological Trace Element Research*, *143*(1), 58-65.

Mulloy, A.; Lang, R.; O'Reilly, M.; Sigafoos, J.; Lancioni, G. and Rispoli, M. (2010): Gluten-free and casein-free diets in the treatment of autism spectrum disorders: a systematic review. Research in Autism Spectrum Disorders, 4(3), 328-339.

Nadeem, A.; Ahmad, S. F.; Al-Harbi, N. O.; Attia, S. M.; Alshammari, M. A.;Alzahrani, K. S. and Bakheet, S. A. (2019): Increased oxidative stress in the cerebellum and peripheral immune cells leads to exaggerated autism-like repetitive behavior due to deficiency of antioxidant response in BTBR T+ tf/J mice. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, *89*, 245-253.

Niederhofer, H. (2009): First preliminary results of an observation of Ginkgo Biloba treating patients with autistic disorder. *Phytotherapy Research*, *23*(11), 1645-1646.

Nishikimi, M.; Rao, N. A. and Yagi, K. (1972): The occurrence of superoxide anion in the reaction of reduced phenazine methosulfate and molecular oxygen. *Biochemical and Biophysical Research Communications*, *46*(2), 849-854.

Ohkawa, H.; Ohishi, N. and Yagi, K. (1979): Assay for lipid peroxides in animal tissues by thiobarbituric acid reaction. *Analytical Biochemistry*, *95*(2), 351-358.

Quettier-Deleu, C.; Gressier, B.; Vasseur, J.; Dine, T.; Brunet, C.; Luyckx, M. and Trotin, F. (2000): Phenolic compounds and antioxidant activities of buckwheat (Fagopyrum esculentum Moench) hulls and flour. *Journal of Ethnopharmacology*, *72*(1-2), 35-42.

Ramassamy, C. (2006): Emerging role of polyphenolic compounds in the treatment of neurodegenerative diseases: a review of their intracellular targets. *European Journal of Pharmacology*, *545*(1), 51-64.

Reeves, P. G.; Nielsen, F. H. and Fahey Jr, G. C. (1993): AIN-93 purified diets for laboratory rodents: final report of the American Institute of Nutrition ad hoc writing committee on the reformulation of the AIN-76A rodent diet.

Reith, R. M.; McKenna, J.; Wu, H.; Hashmi, S. S.; Cho, S. H.; Dash, P. K. and Gambello, M. J. (2013): Loss of Tsc2 in Purkinje cells is associated with autistic-like behavior in a mouse model of tuberous sclerosis complex. *Neurobiology of disease*, *51*, 93-103.

Smith, P. F.; Maclennan, K. and Darlington, C. L. (1996): The neuroprotective properties of the Ginkgo biloba leaf: a review of the possible relationship to platelet-activating factor (PAF). *Journal of Ethnopharmacology*, *50*(3), 131-139.

Tisserand, R. and Young, R. (2013): *Essential Oil Safety-E-Book: A Guide for Health Care Professionals*. Elsevier Health Sciences.

Valko, M.; Leibfritz, D., Moncol, J.; Cronin, M. T.; Mazur, M.and Telser, J. (2007): Free radicals and antioxidants in normal physiological functions and human disease. *The international journal of biochemistry and cell biology*, *39*(1), 44-84.

Whiteley, P.; Shattock, P.; Knivsberg, A. M., Seim, A.; Reichelt, K. L.; Todd, L. and Hooper, M. (2013): Gluten-and casein-free dietary intervention for ASD spectrum conditions. *Frontiersinhumanneuroscience*, *6*, 344.

Journal of Home Economics

المؤتمر الدولى السادس – العربى العشرون للإقتصاد المنزلى
" الاقتصاد المنزلى وجودة التعليم"
٢٣-٢٤ ديسمبر ٢٠١٨ م

http://homeEcon.menofia.edu.eg **ISSN 1110-2578**

التأثير العلاجي لبعض النباتات على الفئران المصابة بالتوحد

عادل عبد المعطى احمد, نهاد رشاد الطحان, ساره عبدالوهاب الشريف ,
محمد عبدالمجيد السعدني .
قسم التغذية وعلوم الأطعمة ,كليه الاقتصاد المنزلي , جامعه المنوفية, مصر

الملخص العربى:

التوحد هو اضطراب عصبي مزمن يؤثر على السلوك والقدرة على التواصل الاجتماعي. قد زاد انتشار المرض بشكل ملحوظ خلال العقود الثلاث الماضية رغم اختلاف نسب الانتشار اقليميا. كان الهدف من هذا البحث هو دراسة مدى تأثير الوجبات الخالية من الكازين والجلوتين مع أو بدون اعشاب المريمية او الجنكة في تحسين الحالة العصبية والتغيرات المرضية التشريحية المرتبطة بالتوحد. تم حقن مجموعة من إناث الفئران الحوامل بجرعه واحدة من محلول حمض فالبوريك داخل الغشاء البروتوني بتركيز ٦٠٠ مجم / كجم ، بعد الفطام تم توزع النسل عشوائياً الى ٤ مجموعات بكل منها ٥ فئران على النحو التالي :- مجموعة ١: كونترول موجب تتغذى على وجبة قياسية، مجموعة ٢: تم تغذيتها على وجبة خاليه من الكازين والجلوين، مجموعة ٣: تم تغذيتها على وجبة خاليه الكازين والجلوتين + مريمية، مجموعة٤: تم تغذيتها على وجبة خاليه من الكازين والجلوتين + جنكة. أخذ نسل امهات سليمه غير محقونه بحمض الفالبوريك، وغذي على وجبة قياسية لاستخدامه كمجموعه كونترول سالبة. تم تقدير التأثير العلاجى من خلال قياس معدل النمو، مستويات السيرم من دلالات مضادات الاكسدة من سوبر أكسيد ديسميوتاز، جلوتاثيون، المحتوى الكلى لمضادات الاكسدهوالمالونالدهيد بالاضافة الى التشريح النسيجى للمخيخ. أظهرت النتائج أن للجنكة تأثير معنوي واضح على معدل النمو مقارنه بالمجموعات الاخرى، كان هناك توافق بين دلالات مضادات التأكسد مع اختلاف نسب التأثير، فقد اظهرت المريمية أعلى تأثير، تلى ذلك الجنكة ثم الوجبة الخالية من الكازين والجلوتين. وفيما يخص التركيب التشريحي فقد اظهر مخيخ مجموعه الكونترول الموجبة تشوها وضمورا في الخلايا العصبية بينما المجموعة المعالجة بالجنكة أظهرت تحسن واضح في نسبه الضمور. تؤكد النتائج ان التغذية على وجبات خاليه الكازين والجلوتين بالإضافة الى عشبة المريمة والجنكة ان لهم تأثير إيجابي معنوي في علاج الاضطرابات المرتبطة بالتوحد لدى فئران التجارب.

الكلمات المفتاحية: التوحد، الكازين، الجلوتين، المريمية، الجنكة