Journal of Medicine, Physiology and Biophysics
ISSN 2422-8427 An International Peer-reviewed Journal
Vol.31, 2017

Autism Electromagnetic and Diet Therapy

Hussein Abu Elasal[1] Ahmad A. Gharaibeh[2*]
1- King Abudallah ll. Design and Development Bureau (Retired)
2- Department of Food Science and Nutrition, Faculty of Agriculture, Jerash University, P.O.Box 311, Jerash 26110, Jordan

Abstract
Autism is a neurological disorder of childhood with poorly understood, in terms of its etiology and pathology. Oxidative stress may play a role in the disease pathophysiology. Oxidative stress results in an excess of free radicals which leading to organ dysfunction. Mitochondria are the main intracellular source of free radicals. In this experimental study, an innovative approach of using a DC electrical pulsating device with an antioxidants supplements in early treatments of disease is introduced. The current study included (20) autistic children as the intervention group and (20) non-autistic children as a control. The intervention group received an electromagnetic treatments and the antioxidants supplements for four months. Their autistic states were evaluated according to the Autism Treatment Evaluation Checklist (ATEC). Lactate (The mitochondrial dysfunction biomarker), Ceruloplasmin and Transferrin were tested for the presence of the oxidative stress. Pre-treatment values were compared with post- treatment values and with standard values. Results indicate that there is a significant decrease in lactate values after intervention (mean ± SD 2.6 ± 0.82 and 1.88 ± 0.61) pre- and post intervention. Assessment of the autism state by (ATEC) reported that 90% of the respondents showed a decrease in total ATEC scores after the treatment process by an average of 69.28%. The strong correlation of the oxidative stress biomarkers associated with autism severity. Suggesting a good ground to use electromagnetic and antioxidant supplements treatment for autistic children.
Keywords: electromagnetic therapy, autism, oxidative stress, mitochondrial dysfunction, food supplements.

1. Introduction

It is well known that electrical fields is generated by cells and tissues activity. Each cell in body is positively and negatively charged and needs to be kept balanced .i.e., polarized. The biomagnetic fields throughout the body maintain this balance in the cells. Different studies stated that an imbalance of the electromagnetic frequencies of energy can cause illness.

The depolarization of cells is due to the accumulation of the free radicals, since they are atoms, molecules or ions with positive and negative charges surrounded the cell membrane. Free radicals are produced from either endogenous sources (Halliwell et. al., 1995), or from exogenous sources (Lobo,et.al.,2010). Mitochondria are the primary source of reactive oxygen species (ROS) by electron leak from the electron transport chain.

Different studies suggested that the Electromagnetic therapy is effective on any living cell to restore its polarization, by reducing its free radicals content.

An Asian study reported that electromagnetic pulse prevents free radical generation by activating antioxidant enzyme activity and reducing oxygen consumption. Wang, et.al., (2013).

In addition to that another study reported that the electromagnetic pulses accelerate healing by acting as a catalyst for the antioxidants to connect with the free radicals which are the primary culprit in diseases. Glen Gordan (2007)

Moreover, a study proposed that Pulses Electromagnetic Frequencies (PEMF) devices offer abundant free electrons to the human body, in addition to plentiful negative ions. Such a flood of free electrons, penetrating through permeable membrane throughout the tissues, and affect the ATP production in the Krebs cycle. Thomas F. Valone (2003)

Two earlier research studies showed that there is a statistical correlation between the electro-stimulation of key acupuncture points with a reduction in free radicals and an improved emotional state. Russell (2005) and Shealy (2002)

Autism is a prevalent neuro-developmental disorder disease, the disorder has an association with language severe impairment, in addition to cognition as well as socialization (Lord et.al., 2000 ; Austin, 2008)

Many studies reported the role of the oxidative stress in the pathophysiology of autism disease. (McGinnis,R. 2004; Maria E. et.al., 2013; El-Ansary A. et.al., 2010). Oxidative stress results in a surplus of free radicals, which can lead to a react with cellular lipids, proteins, and nucleic acids ending in injury and final organ dysfunction. (Jacobson MD,1996).

A strong connection between mitochondrial diseases and autism and oxidative stress has been found, possibly due to an intervention between genetics and the environment. Mitochondria are primary source of ROS electron leak from the electron transport chain. A recent study (2010) at University of California Davis showed

that 80% of children with ASD enrolled in their study had blood tests indicating mitochondrial dysfunction (Giulivi, et.al.,2010). Another study reviewed eighteen publications representing a total of 112 children with ASD and MD (ASD/MD) were identified. They found that the prevalence of development regression (52%), seizures (41%), motor delay (51%), gastrointestinal abnormalities (78%), and pyruvate (45%) was significantly higher in ASD/MD compared with the general ASD population (Rossignol, DA, and Frye RE, 2012). A lactate level is a simple, non-evasive , low-cost means for the initial diagnostic approach of mitochondrial dysfunction. (Zeviani 1996).

Both Ceruloplasmin as well transferrin are major antioxidant proteins. In addition Ceruloplasmin works as ferroxidase, it also protects polyunsaturated fatty acids from active oxygen radicals in red blood cell membranes.(Arnaud et al.,1988). Transferrin works as an antioxidant by reducing the concentration of free ferrous ion (Loeffler et al., 1995).

Objectives of this experimental study, were to introduce an innovative approach of using a simple DC electrical pulsating device with an antioxidants supplements in early treatments of autism.

2. Materials and Methods

2.1. Ethical statement

Participation jn the study was voluntary and anonymous. A written consent was obtained from the parents of each individual case. During the experiment time, each participants assigned with a code number in conjunction with initial selection. These numbers were in lieu of their names for all data records. The study followed principles in the Declaration of Helsinki.

2.2. Description of the device:

The device was invented by the first author (H. Abu Elasal). The device consists of three parts; the first is a timing circuit with a 9 volt supply voltage which produce a number of pulses per seconds according to the nature of the disease, the second is a switching circuit and the third is a two copper electrodes (positive and negative) the first was connected with a specific point on the human body and with the other was connected to the right or the left hand.

This research inspects the effectiveness of electro-stimulation on specific skin points, in the attempt to reduce the free radical activity and increase the antioxidants capacity, as well as to determine the outcome of the intervention on the autistic state of the participants.

2.3. Sampling and Setting:

Participants were selected from autism-specific schools in Irbid- Jordan. All children met criteria for diagnosis of autism on (SCARS) scores (Scopler, et.al., 1980), while the diagnosis was established in a previous assessment by either a child psychiatrist or psychologist.

Twenty children diagnosed with autism were selected (14 males and 6 females) the age ranged between 3 to 15 years. All children were in good physical health.

A local clinic was used to control the experiment, so that the area can supervised, it was conducted at the My Health Medical Center (Irbid –Jordan), and the biochemical tests were carried out at Biolab and Al-Amal Specialized Lab. (Irbid –Jordan).

2.4. Experimental Design:

The study was conducted over a period of four months. A pre-post testing was conducted for each participants.

Pre-test: During the first day of the study, all participants children were surveyed and examined for the following :1- using (ATEC) scores to measure their autistic state; 2- the lactate test to assess the mitochondrial dysfunction; and 3- ceruloplasmin and transferrin test to evaluate the antioxidant capacity of the participants.

Over a period of four months the intervention group received the electro- stimulation treatments on specific skin points, each participants received one to two session per week, each session lasted for 15 to 20 minutes. At the same time the intervention group supplied with vitamin C, vitamin E and Selenium (50 mg, 10 IU, and 20 mcg) respectively. Since, The Recommended Dietary Allowances (RDA) for children (1-10 years) were (40-50 mg, 10.5 IU and 30 mcg) respectively. Food and Nutrition Board (FNB) (1989).

Post-test: At the end of the interventions, all of the participants were re-tested using the same measure to determine post- intervention values and the data learnt through the different tests can be analyzed for any statistical significance.

2.5. Biochemical Analysis:

2.5.1. Lactate in blood:

The levels of lactate was measured by enzymatic determination from BioMeriex, France according to manufacturer instruction. The reference limit (venous) is: 0.5 – 2.2 mmol/L.

2.5.2. Ceruloplasmin and Transferrin levels in serum:
The levels of Ceruloplasmin and Transferrin were analyzed for the eighteen (18) autistic children as well as the control group using the NEPHSTAR protein analysis system and the assay kits. (Goldsite Diagnostics In. China). In this test the immunephelo-meter is applied.
The normal range of ceruloplasmin concentration of healthy adults is: 0.2- 0.6 g/L.
The normal range of transferrin concentration of healthy adults is: 2- 3.6 g/L.

2.6. State Evaluation:
The study was based on the ATEC (Autistic Treatment Evaluation Checklist), which developed by ARI (the Autism Research Institute in San Diago, USA). The tool is provided for free and is available at (www.autism.com/atec/index.html). The tool is composed of four subscales:
Scale I: Speech/Language Communication (14 items) –(Range 0 -28)
Scale II: Sociability (20 items) –(Range 0 -40)
Scale III: Sensory/ Cognition/Awareness (18 items) –(Range 0 -36)
Scale IV: Health/ Physical behavior (25 items) –(Range 0 -75)

Items on subscales 1-3 are scored from 0 (not a problem) to 2 (serious problem). The maximum true score is 179, the higher score indicates higher severity of autistic behaviors and poorer social developmental skills, lower scores indicates progress and improvement in autistic problems. The level of ATEC <30 places the child in the top 10 percentile. Such children have high chances of leading normal lives as independent individuals, while any child with a score of more than 104 would already be in the 90^{th} percentile and be considered very severely autistic.

During the study, children were evaluated by the Autism Treatment Evaluation Checklist (ATEC), (Rimland, 1999). The ATEC is an assessment tool designed to be completed by either the parents or the researchers. The tool consists of four subscales: a) Speech/Language Communication; b) Sociability; c) Sensory/Cognition Awareness and d) Health/Physical/Behavior. The ATEC has been successfully used to measure treatment effects and progress over time in several studies in autism. (Geire.2013; Magiati 2011).

The original purpose of ATEC is not to provide diagnostic tool. The tool included several subscale scores, in addition to the total score to be used for comparison.

2.7. Statistical analysis:
Lactate levels, in addition to the levels of ceruloplasmin and transferrin, as well as the (ATEC) scores for autism state were analyzed statically. Comparison was made between group, (a) autism group and their developmentally normal control group (b) autism group pre-intervention and post intervention. The measured values, expressed as the mean ± SD, were compared using unpaired students t-test, $p<0.05$ was considered as significant level.

3. Results:
3.1. lactate level in autism
The reference interval of lactate in control children is 0.13 to 2- 2.2 mmol/L. Table 1. illustrates the levels of lactate in 18 children with autism and their non-autistic control. Statistical analysis (a) showed that lactate levels were significantly increased in whole autism group (mean± SD=2.6 ± 0.82) mmol/L as compared to whole non-autistic control group (mean ± SD=1.3 ± 0.7 mmol/L , and (b) also, statistical analysis showed that lactate levels were significantly decreased in whole autism group post-intervention (mean ± SD=1.88 ± 0.61) mmol/L as compared to the same group pre- electromagnetic intervention.

3.2. Reduced Serum Ceruloplasmin levels in Autism
Table 1. illustrates the levels of ceruloplasmin in 18 children with autism and their non-autistic control. Statistical analysis (a) showed that ceruloplasmin levels were significantly decreased in whole autism group (mean± SD=0.2200 ± 0.0418) mg/ml as compared to whole non-autistic control group(mean± SD=0.3717 ±0.0498) mg/ml , and (b) also, statistical analysis showed that ceruloplasmin levels were significantly increased in whole autism group post-intervention (mean± SD=0.3494± 0.0417) mg/ml as compared to the same group pre- electromagnetic intervention (mean± SD=0.2200 ± 0.0418)mg/ml. The data was however, not significant when the mean levels of ceruloplasmin in autistic group post-intervention (0.349 ± 0.0417) mg/ml were compared with non-autistic control group mean levels (0.3717 ± 0.0498) mg/ml.

Table1. Oxidative stress indicators of autistic (Pre- and Post- intervention) and control children

Oxidative stress indicators	Group	No	Mean ± SD	P value
	Pre-inter.	18	0.220±0.0418	p ≤o.o5
Ceruloplasmin	Post-inter.	18	0.349±0.0417	
	Control	18	0.371±0.0498	
	Pre-inter.	18	1.993±0.13	p ≤o.o5
Transferrin	Post-inter.	18	3.107±0.25	
	Control	18	3.072± 0.23	
	Pre-inter.	18	2.60 ±0.82	p≤ o.o5
Lactate	Post-inter.	18	1.88 ±0.61	
	Control	18	1.30±0.72	

3.3. Reduced Serum Transferrin levels in Autism

The levels of transferrin in serum samples of(18) children with autism, and their non-autistic control group are shown in table 1. Statistical analysis (a) showed that transferrin levels were significantly decreased in whole autism group (mean ± SD=1.9933± 0.1303) mg/ml as compared to whole non-autistic control group(mean ± SD=3.0722± 0.2396) mg/ml , and (b) also, statistical analysis showed that transferrin levels were significantly increased in whole autism group post-intervention (mean ± SD=3.1072± 0.2519) mg/ml as compared to the same group pre- electromagnetic intervention (mean± SD=1.9933± 0.1303)mg/ml. The data was however, not significant when the mean levels of transferrin in autistic group post-intervention (3.1072±0.2519) mg/ml were compared with non-autistic control group mean levels (3.0722± 0.2396) mg/ml.

3.4. Assessment of the autism state by The Autism Treatment Evaluation Chicklist (ATEC)

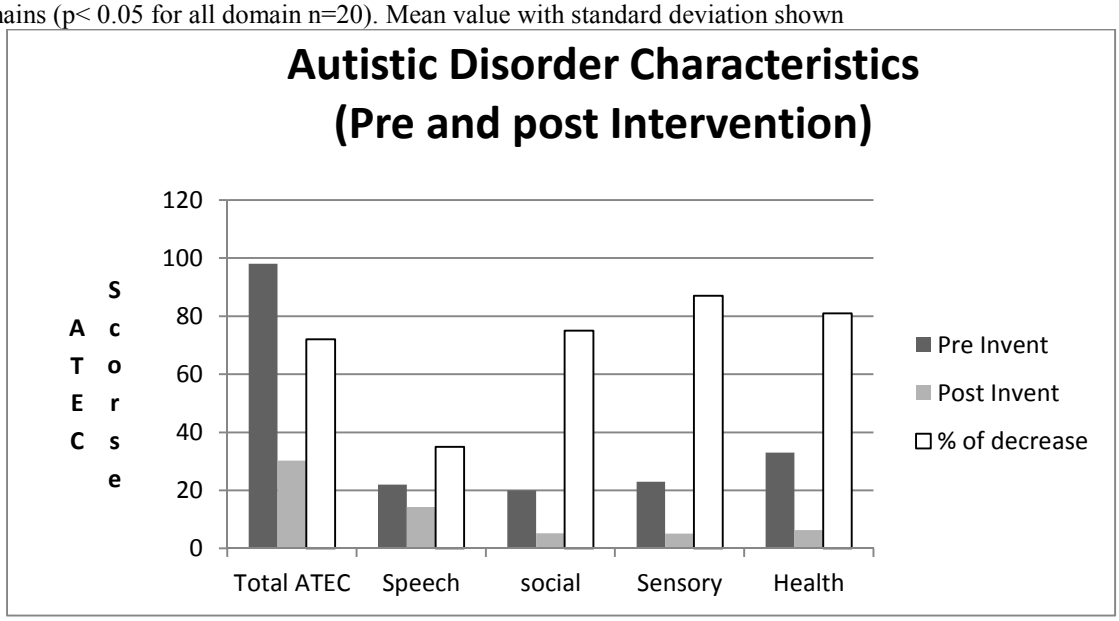
Mean ATEC score significantly decreased following initiation of treatment, indicating a decrease in severity of autism symptoms (table 2). Of the 20 respondents, 90% reported a decrease in total ATEC score. The remaining two respondents did not respond to EM treatment, registering reduction in total ATEC scores of less than 10 points. It is not clear if they would improve if they had continued the treatment. Mean total ATEC values decreased by an average of 69.28% from 98.00 prior to treatment to 30.30 following treatment initiation (Figure 1, P<0.05).

Table 2. Autistic Disorder Characteristics (Pre- and post- Intervention)

ATEC Domain Scores	No	Mean ±SD		Percent of decrease
		Pre-Inter.	Post-Inter.	
Total ATEC	20	98.00±16.46	30.30 ±7.74	69.08
Speech	20	22.00± 3.27	14.30±2.71	36.40
Social	20	20.35± 2.15	5.25±1.37	73.75
Sensory	20	23.o5±3.33	5.15±1.30	77.65
Health	20	31.95 ±3.67	6.30±1.38	80.28

There were significant decreases observed following treatment initiation in all four of the ATEC categories (Figure 1). Mean speech/ language/communication ATEC scores decreased from 22.00 prior to treatment to 14.00 following treatment initiation (average decrease of -36.4%; P<0.05), with 56% of respondents reporting improvements. Sociability ATEC scores decreased by an average of 73.75% following treatment initiation, with improvements reported among 75% of respondents (P<.05). Sensory/Cognitive Awareness ATEC scores were decreased by an average of 77.65%, following treatment initiation with 72% of respondents reporting improvements (P<.05). In the health/physical/behavior domain, ATEC scores decreased by an average of 80.28%, from 31.95 prior to treatment to 6.30 following treatment initiation; 92% percent of respondents reported improvements in this domain (P<.05).

Figure 1: Decrease in Autism Severity Post Intervention. Mean ATEC Scores significantly decreased across all domains ($p < 0.05$ for all domain n=20). Mean value with standard deviation shown



4. Discussion

Several recent studies described the treatment of some neurological disorders with an externally applied of electromagnetic field (Jacobson, et. al.,1994; Sandyk, R., 1992).

For the treatment process, the device offers a flood of electrons, penetrating through permeable membranes throughout the tissues and neutralize these charges and correct the cells polarization. We correlate statically the electrical stimulation effects with the oxidative stress biomarkers as well as the improvement of autistic symptoms.

Autism is a severe neurodevelopment disorder. According to the U.S.CDC (July 2016), the prevalence of the autism spectrum disorder is about 1 in 68 children. Exclusive studies have demonstrated that oxidative stress plays a vital role in the pathology of several neurological diseases. Oxidative stress results in an excess of free radicals which leading to organ dysfunction. (Halliwell B. 2012) suggested that oxidative stress can damage neurons and brain cells via product of lipid peroxidation. Also, (Chauhan et. al., 2004), reported that phospholipid composition of erythrocyte membrane is altered in autism. The mechanism of oxidative stress in autism is due to increased production of pro-oxidants, as well as the deficiencies of antioxidants enzymes and antioxidant proteins or both.

Mitochondria are the main intracellular source of free radicals. The mechanism by which a mitochondrial dysfunction might lead to autistic behavior is not known, but the direct involvement of an oxidative defect in the disturbance of early brain development is a plausible hypothesis (Lombard 1998). Recently, evidence has accrued that autistic children have concomitant diseases such as mitochondrial disease and abnormality of energy generation (Siddiqui F.M. et.al., 2016). Many recent studies finding support the hypothesis that there is an association of ASD with impaired mitochondrial function. There are several main outcome measures that revealed the mitochondrial dysfunction in autism such as oxidative phosphorylation capacity, mtDNA copy number and deletions, mitochondrial rate of hydrogen peroxide production, and plasma lactate and pyruvate. If the mitochondria is not working, glucose will turn into pyruvate, which will turn into lactate, which will turn into alanine. A lactate level is a simple, non-evasive, low- cost means for the initial diagnostic approach of mitochondrial problems (Zeviani 1996). Elevated of lactate level blood samples from autistic children ranged from 17% to 76% (Olivera 2005). Mostafa et. al., (2005) studied the mitochondrial dysfunction in autistic children and they found 77% of the children had high lactate levels than normal group. Also, Weissman et.al.,(2008) found that lactate level were high in 76% of the autistic children.

Our results suggest that the levels of lactate, were significantly increased in children with autism as compared to their non-autistic control, (13) out of (18) autistic children ,about 73,3% had higher lactate levels regarding the laboratory markers. This result is concomitant with that of (Rossignol,D.A. and R.E.Frye 2012) who found that mean lactate content in 114 children with ASD was (1.73mmol/L), while in control group was (0.9mmol/L). On the other hand we found that children with severe autism had higher lactate compared to less severity. In fact, we found one case had high level of lactate (3.0 mmol/L) and this result remain without changing even after the intervention treatment, although his behavioral symptoms were improved more than 80%.

In general, we found that autism symptoms were improved in all cases that had decreasing levels in lactate contents. The different values of plasma lactate may be explained by the differences in chosen cut-off value above which the patient was considered to have lactatemia, number and age of studied autistic children.

Our results suggest that the levels of ceruloplasmin and transferring, which are the major antioxidant proteins in the blood, are significantly reduced in children with autism as compared to their non-autistic control. This result is concomitant with that of (Chauhan, et al., 2004), who reported lower levels of ceruloplasmin and transferrin (68% and 84% respectively) in children with autism as compared to their developmentally normal siblings. Also (Essa, et al., 2012), their study showed marked reduction in the levels of ceruloplasmin and transferring in Omani autistic children.

Ceruloplasmin protects polyunsaturated fatty acids in the red blood cell membranes from active oxygen radicals (Arnaud et al.,1988). It acts as a ferroxidase and superoxide dismutase.

Transferrin acts as an antioxidant by reducing the concentration of free ferrous ion that catalyzes the conversion of hydrogen peroxide to highly toxic hydroxyl radical by Fenton reaction. It has been reported that the lower levels of ceruloplasmin and transferrin are directly manifesting the state of autism in children with loss of acquired language skills(Chauhan,2004).

Despite the extensive research in the use of antioxidants, there is not a clear-cut consensus that these antioxidants are totally successful in reducing free radicals. Other than antioxidants, there are different methods used to reduce free radicals such as the electro stimulation of specific acupuncture points (Shealy, et al., 2002; Russell 2006; Wang,et.al., 2013). By using types of transcutaneous electrical nerve stimulators they found that in individuals with positive urinary evidence of free radicals, there are a significant reduction in total free radical activity.

In the current study, after the EM stimulation we found that lactae levels were decreased while ceruloplasmin and transferrin levels were increased in autistic group as compared to the same group pre-intervention. Furthermore, the results suggest that there was a significant inverse correlation between protein antioxidant and autism severity measured using ATEC scoring. There were significant decreases observed following EM stimulation in all four of the ATEC categories. These finding confirmed that there could be significant health benefits from EM stimulation. However, the lowest improvement level of the ATEC categories was with speech subscale. Previous studies showed a positive correlation between reduced levels of these antioxidant proteins and loss of previously acquired language skills in children with autism. However, the underlying mechanism is still unclear and further extensive studies are warranted.

The results confirmed that the EM stimulation making it easier for the antioxidants to connect with the free radicals, and thus prevent depolarization of cells.

We applied this principle of treatment on several chronic diseases such as mental illness, diabetes, heart diseases, glands and food allergies, and we obtained prospective and encourage results with no known side effects.

In conclusion, the strong correlation of the oxidative stress biomarkers and the ATEC scores, strongly suggested that oxidative stress biomarkers are associated with autism severity.

The present study is a novel prospective study conducted to evaluate the autistic symptoms improvement after using a DC pulsating treatment. The highly decrease in ATEC scores and the parents reports, strongly emphasize the DC pulsating effects in healing and decreasing of autism severity with more than 80% of the participants.

Finally, further practical studies with sophisticated instruments were needed to strictly correlate the treatment processes with a fixed mathematical equation responding to the biomagnetic field for every disease.

In considering the potential limitation of the present study, the number of study participants was of moderate size. Despite this potential limitation, the consistency and specificity of the results observed were strengths of the present study.

5. Acknowledgement

The authors wish to acknowledge the help of parents and children who participated in the study; without their participation this type of investigation would not be possible.

6. References

Arnaud,P., Gianazza ,E ., Miribel., 1988. Ceruloplasmin . Methods in Enzymology 163,144-452.

Austin, D .2008. An epidemiological analysis of the " autism as mercury poisoning " hypothesis . International Journal of Risk and Safety in Medicine , 20:135-142.

Chauhan A, Chauhan V, Brown WT, Cohen I. 2004. Oxidative stress in autism: Increased lipid peroxidation and reduced serum levels of ceruloplasmin and transferrin –the autioxidand proteins. Life Sci. 75:2539-2549.

EL. Ansary A, Al- Daihan S, Al-Dbass A, Al-Ayadhi L .2010. Measurement of selected ions related to oxidative

stress and energy metabolism in Saudi autistic children. Clin Biochem. Jan, 43(1-2): 63-70.

Essa M.M ., Guillemiu , G.J ., Waly ,M.I,AL-Sharbati ,M.M., AL-Farsi, Y.M., Hakkim ,F.L.,Ali,A., AL-Shfaee ,M.A. 2012. Reduced level of antioxidant proteins in children with autism in Oman . International Journal of Nutrition , pharmacology ,Neurological Disease. Vol. (2) , Issue (1) :53-56.

Food and Nutrition Board (FNB) (1989). Recommended Dietary Allowances, 9th.ed. Washington,D.C. National Research Council, National Academy of Sciences.

Gordon, G.A., 2007. Designed electromagnetic pulsed therapy: Clinical application. J. Cell. Physiol. 212:579-582.

Giulivi C, Zhang YF, Omanska-Klusek A, Ross-Inta C, Wong S, Hertz-Picciotto I, Tassone F, Pessah IN.2010. Mitochondrial dysfunction in Autism. J.of the American Medical Association 304:2389-2396.

Halliwell,B., and Gutteridge,J.M.C.1995. The definition and measurement of antioxidants in biological systems. Free Radic Boil Med.18:125-126.

Halliwell, B., 2012. Free radicals and antioxidants: updating a personal view. Nutrition Reviews. 70(5):257-265.

Jacobson, MD., 1996. Reactive oxygen species and programmed cell death. Trends Biochem Sci.243(21): 81-119.

Lobo,V. Patil, A. Patak,A. Chandra, N. 2010. Free radicals, antioxidants and functional foods: Impact on human health. Pharmacogn Rev. 4(8):118-126.

Loeffler, D.A., Connor, J.R, Juneau, P.L., Snyder, B.O.S, Kanaley, L, DeMaggio, A.J., Nguyen,H,Brickman,C.M.,Lewitt,P.A,1995. Trasferrin and iron in normal, Alzheimer's disease, and Parkinson's disease brain regions. Journal of Neurochemistry 65(2): 710-724.

Lombard J. 1998. Autism: A Mitochondrial Disorder Med Hypothesis. 50:497-500.

Lord**,** C, Cook, E.H, Leventhal, B.L., Amaral, D.G., 2000. Autism spectrum disorders. Neron 28(2): 355-363.

Maria Elena Gonzalez Fraguela et.al., 2013. Oxidative stress markers in children with autism spectrum Disorders. British Journal of medicine and Medical Research. 3(2) : 307-317.

Mc Ginnis WR.2004. Oxidative stress in autism. Altern. Ther. Health Med. vol. (10), No. (6) :22-36.

Mostafa.G.A. EL-Gamal.H, El-Wakkad.A, El-Shorbagy.O, Hamza. M. 2005. Saturated Fatty Acids , Carnitine and Lactate as Biological Markers of Brain Energy in Autistic Children. Int. J. Ch. Neuropsychiatry, 2 (2):179-188.

Olivera G. Diogo.L, Grazina.M, Garcia.P, Ataide.A, Marques.C, Miguel.T, Icente. AM, and Oliveira.CR. 2005. Mitochondrial Dysfunction in Autism Spectrum Disorders : A Population – Based Study. Dev. Child.Neurol. 47:185-189.

Rimland B., Eldelson M.1999. Autism Treatment Evaluation Checklist. Autism Research Institute. 4812 Adams Avenue, Sam Digo, CA 92116. http:// www.autismeval.com/ari-atec/report1.html.

Rossignol DA an Frye RE 2012. Mitochondrial Dysfunction in Autism Spectrum Disorders: A Systematic Review and Meta-Analysis. Mol. Psychiatry 17 (3):290-314.

Russell,S. And Shealy,C.N. 2006. Free radicals reduction through the use of eletro stimulation of acupuncture needles on the Shealy Ring of Crystal. Subtle Energies and Energy Medicine. VOL(16), NO(2):85-88.

Sandyk,R. 1992.Magnetic field in the therapy of Parkinsonism. International Journal of Neuroscience. 66:209-235.

Schopler, E.,Reichler,R. J., DeVellis, R. F. and Daly,K. 1980.Towards objective classification of Childhood Autism: Childhood Autism Rating Scale (CARS). Journal of Autism and Developmental Disorders, 10: 91-103.

Shealy,C.N., And Thomlinson, K.P.2002. Reduction of free radical by electrical stimulation of specific acupuncture points. Subtle Energies and Energy Medicine VOL.(13), NO(3);251-250.

Siddiqui MF, Elwell C, Jonhnson MH. 2016. Mitochondrial Dysfunction in Autism Spectrum Disorders. Autism Open Access.27; 6 (5).

U .S. CDC 2016. The Centers for Diseases Control and Prevention.

Valone ,T.F .2003. Bioelectromagretic healing, its history and a rationale for its use. www.Integrity Research Institue.org. Proceeding of the whole Person healing conference Bethesda ,MD .

Wang.c, Zhon. H, Peng. R, Wang.L, Su.Z.2013.Electron pulse reduces free radical generation in rat liver mitochondria in vitro. Free Radical Research, April, vol. (47), No.4:276-282.

Weissman JR, Kelley RI, Bauman ML, Cohen BH, Murray KF, Mitchell RL, et al. (2008) Mitochondrial Disease in Autism Spectrum Disorder Patients: A Cohort Analysis. PLoS ONE 3(11): e3815.

Zeviani,M. Bertagnolio,B. Uziel,G. 1996. Neurological Presentation of Mitochondrial Diseases. J. of Inherited. Metabolic Diseases.19 (4):504-520.