December 2006
JBMS

The Effect of magnetic field strength as a free radical on diabetic subjects

*Ghalib A. Al-Sharefi and Nameer A. Ali

*Al-Mustansiriyah University
College of Medicine
Department of Physiology

Ghalib A. Al-Sharefi
Ass. Professor

Nameer A. Ali
Lecturer

Abstract:

The study was designed to found out the effect of magnetic field strength on the level of free radicals in the blood of diabetic subjects. It was done in the department of physiology, the medical college of Al-Mustansiriyah University and the research lab of the medical physics section. The study was Design as a prospective, randomized, case-control study. Out came measures: magnetic field (MF) was designed to build field strength of 10, 20 & 30 millteslar. The estimation of oxidative stress and antioxidant was done by measurement of malondialdenyde MDA and Glutathione (GSH) level. This study showed that the level of enythrocyte glutatione was significantly lower in diabetic subjects (P < 0.0.) while plasma MDA was significantly higher in diabetes subject than in the control group. All the measured parameters showed no significant changes when exposed to MF up to 30mTS. We concluded that a magnetic field of up to 30MTS has no harmful effect on the biological system activity in normal and D.M.

Key words: Magnetic field, Glutathione, Malondialdehyde, D.M.

Introduction:

The magnetic field is regarded as one of the occupational hazards, It is one of the elementary field of nature which are caused by the movement of electric charges [1] In the last few years, there had been considerable interest in the biology and health science in relation to possible health risks associated with environmental exposure to magnetic field related to the production, distribution and utilization of electrical energy [2].

It seems responsible to assume that any health related consequences would result if a magnetic field can exert influences at molecular level [3]. Table 1 shows the biological effects of variable magnetic fields at different levels of integration while effects of south and north poles of magnetic field are summarized in table [2].

Correspondence to:
Ghalib A. Al-Sharefi
ghalibalsharefi@yahoo.com

JBMS 2006; Volume 6:1-9

Table (1): Biological effects of variable magnetic fields at different levels of integration

1	Systemic effect (hormonal secretion).
2	Effects on organs (heart contractility).
3	Effects on cells and tissues (membrane transport process, regeneration processes)
4	Effects on molecular and sub molecular processes (e.g. activation of respiratory chain enzymes or change in hemoglobin during oxygen transport [4].
5	Effect on free radicals by disrupting Ca^{++} balance, change biological circadian rhythm or prolong the half life of free radicals [5].

Table (2): biological effects of south & north poles of the magnetic fields

Effect of north pole	**Effects of south pole**
Assist in acid-base balance.	Creates acidity.
Enhances tissue oxygenation.	Creates O_2 deficiency.
Removes Edema from tissue.	Produces Edema.
Inhibit microorganism growth.	Promotes microorganism growth.
Reduces inflammation.	Increase inflammation.
Promotes bright and happy effects.	Promotes dull & depressed effects.
Reduces & dissolves fatty deposits	Encourages fatty deposition.
Promotes trophic hormone release.	Promote catabolic hormones.
Creates order in biological tissue.	Creates disorder in biological tissue.
Cancel free radicals	Produces free radicals.

The free radicals are very reactive species and have very short life span, their activity is due to the unpaired "free" electrons and in certain species e.g. hydrogen and hydroxyl, they are able to give hydrogen free radical and hydroxyl free radical H & OH.[6] The free radicals are molecules which posses an odd number of electron can be viewed as a molecular magnet [2] as a consequence the application of a MF can yield a higher concentration of free radicals and can influence their dynamics.[7]

Free radicals are involved in a multitude of biological processes as enzyme chain reaction, lipid peroxidation and DNA damage,[8] and they can produce a large amount of damage to biological components as DNA or reacts with proteins and lipids.[9] Oxidative reactions are an indispensable component of metabolic processes and are believed to play an essential role in the mechanism of a number of diseases. [10]

The attack of cell membrane by free radicals leads to lipid peroxidation which is assessed by plasma malondialdehyde (MDA) level. [11] The overall of lipid per oxidation are: decrease membrane fluidity, increase the leakiness through the membrane bi-layer and

inactivate membrane bound enzymes. [12] An antioxidant has been defined as a substance that when present in low concentrations can delay or inhibit the oxidation of that substrate. [13] Antioxidants are classified into three types according to their function, primary, secondary & tertiary. [14]

All in vivo antioxidants perform the prevention of oxidative stress which is caused by free radicals attack to cell structure, and they need adequate amount of the so called anti-oxidant minerals which are essential nutrients for the enzymatic reactions of antioxidants against free radicals. [15] In diabetes mellitus hyperglycemia can generate free radicals mainly peroxide and reactive ketoaldehydes by the auto-oxidation of glucose and glycated proteins. [16]

Hyperglycemia and hyper-insulinemia are blamed for the increased production of free radicals. [17] In diabetic subject, extra cellular long-lived proteins such as collagen, elastin and laminin is the target of free radicals attack. These proteins are modified to form glycopootein by glycosylation pathway enhanced by hyperglycemia. The glycoproteins are further broken into fragments by free radicals. [18]

The oxidative stress in biological system results from an imbalance between the production and the removal of reactive oxygen species, such imbalance leads to an increase in reactive intermediate formation in the body, which in turn leads to cellular damage. [19] Enhanced oxidative stress in type II diabetes mellitus has a further variety of important effect in atherogenesis including liporotein oxidation, LDL oxidation, lipid per oxidation of PUFA, one of the radical reaction in vivo, can adequately reflect increased oxidative stress in diabetes.[20] The oxidative stress is significantly increased in diabetes because prolonged exposure to hyperglycemia increases the generation of free radicals and reduces the capacity of antioxidant defense system. [21] It has been demonstrated that scavenging of free radicals can be reduced by the lack of GSH which are associated with diabetes mellitus. [22] The mechanism by which the antioxidant reservoir is reduced is not clear. Protein damage due to protein/glycoprotein disruption may be a mechanism that lowers the activity of primary antioxidant enzymes. [23] This study aimed to study the effect of magnetic field in the strength of 10, 20 and 30 milliteslar on the level of free radicals in the blood of normal and diabetic subjects for exposure time of 1-2 hours.

Materials & Methods:

A total number of 30 healthy subjects were taken as a control group their mean age was 46.4±6.1 years and all were free from glucose intolerance proved by OG TT. The mean age of the thirty three subjects with type II diabetes mellitus was a 50.3±6.3 year & the duration of diabetes was 2-10 years. The devices for generating an in homogenous magnetic field consist of a transformer, variable key, coils, meter, voltmeter and metal core. The plasma malondehyde was estimated according to the modified method of shah of and walker 1988,[24] While the glutathione was estimated with the modified method of Beutler 1963[25] by Tiets 1996. [26]

Results:

The level of MDA in our control subjects was 3.53±0.8 Mml/l which was significantly lower than its level in diabetic subjects (7.8±1.01 Mml/l of P <1.01). Table (3) shows the plasma MDA values of control & diabetic subjects relative to magnetic flux intensity (m T) of 10, 20 & 30mT for 1-2 hours exposure time. The MDA value did not show any statistical difference when compared to initial level what ever the exposure time and irrespective to intensity of magnetic field for both groups. While still the significant difference was noticed for MDA value between the control group & diabetic subjects before and after exposure to magnetic field for different intensities & different exposure time (Figures 1 and 2). The level of erythrocyte glutathione GSH was 59.5±4.53 mg/dl in the control group. While it was significantly lower in the diabetic subject 40.48±5.75 mg/dl and P value is <0.01 with exposure to in homogenous magnetic field for 1hour and 2 hours, the level of GSH showed no significant difference relative to time zero for different levels of intensity of the magnetic field. This was applied for both groups, while still the GSH level showed significantly lower values in comparison between the two groups as shown in table (4), and Figure (2).

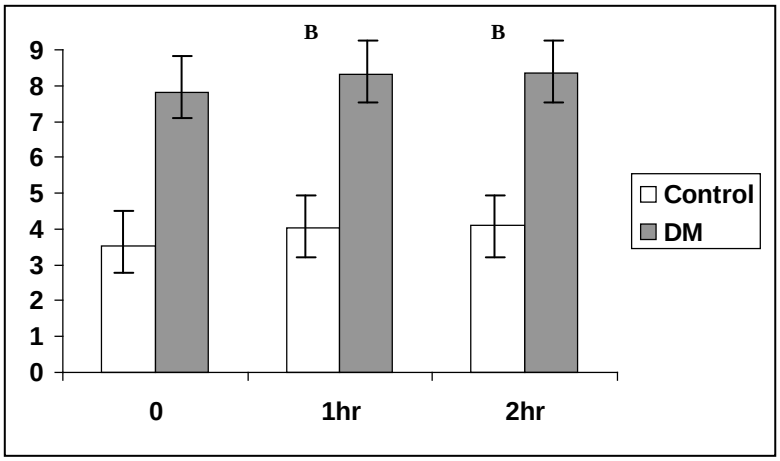

Fig (1): Effect of 10 mT on the plasma MDA level (represented as mean ∓ SD).

B: No significant relative to 0 time diabetic samples
+P < 0.05 relative to control
++P < 0.01 relative to control

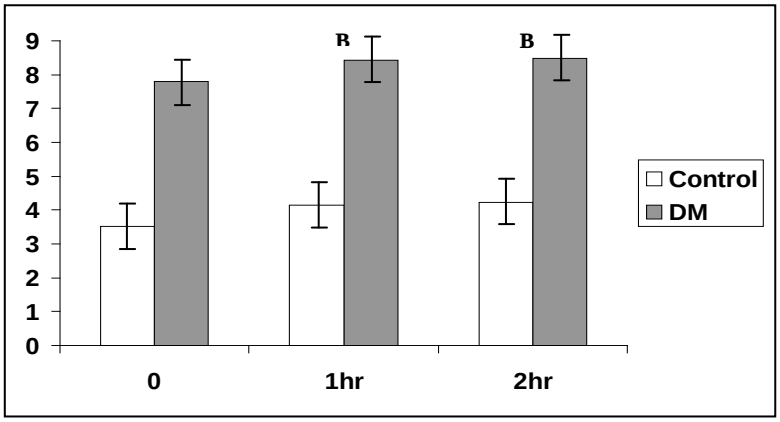

Fig (2): Effect of 20 mT on the plasma MDA level (represented as mean ±SD).

B: No significant relative to 0 time diabetic samples
+P < 0.05 relative to control
++P < 0.01 relative to control

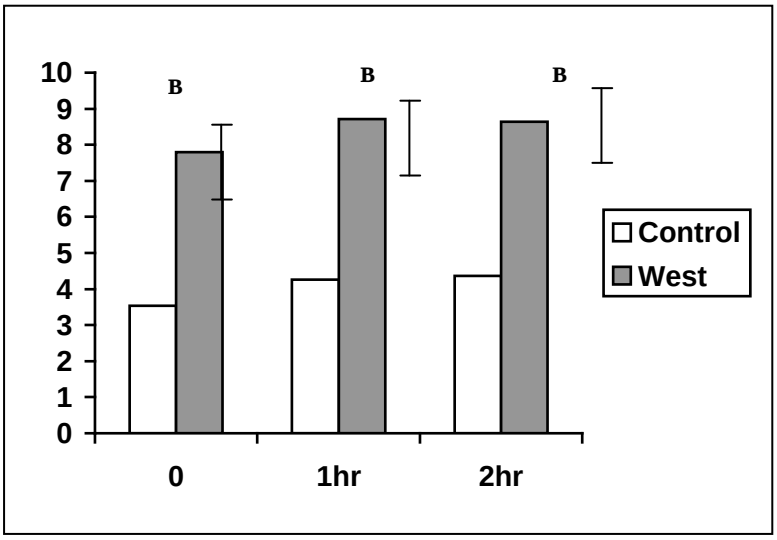

Fig (3): Effect of 30 mT on the plasma MDA level (represented as mean ±SD).

B: No significant relative to 0 time diabetic samples
+P < 0.05 relative to control
++P < 0.01 relative to control

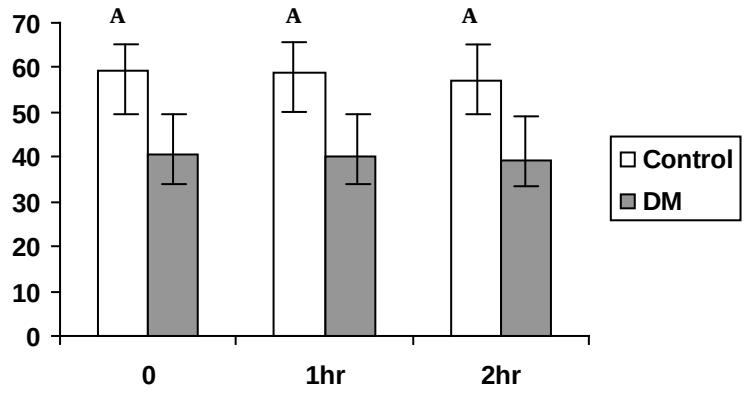

Fig (2-A): Effect of 10 mT on the erythrocytes GSH level (represented as mean ±SD).

A: No significant relative to 0 times in the control sample

+P < 0.05 relative to control

++P < 0.01 relative to control

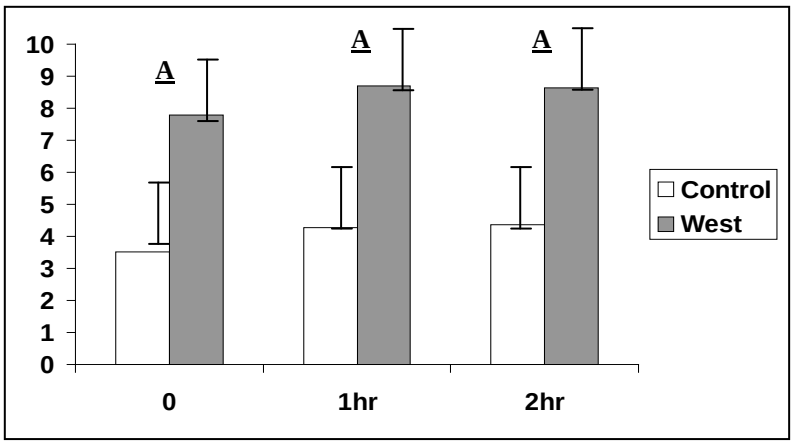

Fig (2-B): Effect of 20 mT on the erythrocytes GSH level (represented as mean ±SD).

A: No significant relative to 0 times in the control sample

+P < 0.05 relative to control

++P < 0.01 relative to control

Table (3): plasma MDA values relative to the magnetic to the magnetic flux intensity (mT) of inhomogenous magnetic field in control and diabetic samples

		Plasma MDA (µmol/L)					
		Control			Diabetes		
Magnetic flux intensity (mT)		Exposure					
		0	1	2	0	1	2
10	Mean	3.53	4.04	4.12	7.80	8.32	8.36
	∓ SD	0.80	0.90	0.82	1.01	0.88	0.91
	P_1-value		NS	NS		NS	NS
	P_2-value				P<0.01	P<0.01	P<0.01
20	Mean	3.53	4.14	4.22	7.80	8.44	8.49
	∓ SD	0.80	0.82	0.79	1.01	0.86	0.90
	P_1-value		NS	NS		NS	NS
	P_2-value				P<0.01	P<0.01	P<0.01
30	Mean	3.53	4.26	4.36	7.80	8.71	8.64
	∓ SD	0.80	0.79	0.81	1.01	0.91	0.89
	P_1-value		NS	NS		NS	NS
	P_2-value				P<0.01	P<0.01	P<0.01

P1 - value: statistical difference before and after expose

P2 – value diabetes mellitus as compared to control

Table (4): *Erythrocyte glutathion values relative to the magnetic to the magnetic flux intensity (mT) of inhomogenous magnetic field in control and diabetic samples*

		Erythrocytes GSH (ml/dL)					
		Control			Diabetes mellitus		
Magnetic flux intensity (mT)		Exposure					
		0	1	2	0	1	2
10	Mean	59.50	58.88	57.13	40.48	40.11	39.21
	∓ SD	4.53	3.90	4.52	4.75	3.52	3.84
	P_1-value		NS	NS		NS	NS
	P_2-value				P<0.01	P<0.05	P<0.05
20	Mean	59.50	58.88	57.18	40.48	38.71	38.09
	∓ SD	4.53	4.15	4.50	5.75	3.33	3.84
	P_1-value		NS	NS		NS	NS
	P_2-value				P<0.01	P<0.01	P<0.01
30	Mean	59.50	57.70	54.20	40.48	37.30	36.20
	∓ SD	4.53	3.73	3.65	5.75	3.35	3.96
	P_1-value		NS	NS		NS	NS
	P_2-value				P<0.01	P<0.01	P<0.01

P1 - value: statistical difference before and after expose

P2 – value diabetes mellitus as compared to control

Discussion:

In our study we found that the level of lipid peroxidation bi-product MDA was significantly higher in diabetic subjects than the control group (P <0.01). This increment in lipid per oxidation maybe attributed to many factors, we think that hyperglycemia induces alterations in intracellular mechanism which yield higher amounts of free radicals leading to imbalance between the free radicals and antioxidant equilibrium and this in turn can cause augmentation in lipid per oxidation detected by increment in MDA level. Same finding was reported by Altomare 1992[27], and Haffner 1995 [28] who started that in type II D.M the lipid peroxidation is increased by the activation of free radicals. Other factors can be blamed for the increment of lipid peroxidation such as increased reactive oxygen species (ROS) production and protein glycatien which both are increased with hyperglycemia [29]. While others found that high MDA levels are associated with poor glycemic control [20]. The erythrocyte glutathione GSH was significantly lower in the diabetic subjects than in normal control group (P <0.01). This result is attributed to the consumption of GSH by the high production of free radicals on which GSH

acts as a scavenger. Identical finding was reported by Marcin 1997[30] and similar conclusion mentioned by Bick 1993[31].

The magnetic field exerts no significant effect on antioxidant level despite the change in strength of M.F and exposure time used in our procedure in normal subjects. The insignificant effect of MF on the level of lipid peroxidation in normal subjects can be regarded as a novel finding as no other study cleared this relationship. While Jin *et al* 2000[31] found that antioxidant level is increased after exposure to MF of 150±25MT strength and with longer duration of exposure, with the notice that study is in vitro while he studied the effect of MF in vivo. Also in diabetic subjects we found no significant changes for antioxidation level or lipid per oxidation in response to MF, a finding which could not be compared to others as there were no previous studies in the same field.

From these results we can calculate that the use of house equipment (radio, CD player.... etc) having MF in their build up system had no harmful effect on humans if the MF strength is within the studied limit.

References:

1. Scaiono JC, Cozens J & Mclean J: Model for the magnetic field effects in vivo. Application of the radical-pair mechanism to biological systems. Photochem. Photobiol. (1994), 59: pp. 585-589 (Abstract).
2. Hileman B: Health effects of electromagnetic fields remain unresolved. C&E. news November (1993), 8: pp 15-29, this is Google's cache of http://www.radicals and magnetism,(2002).
3. Korolenko EC, Cozenes FL & Scanio JC: Magnetic field effect in the dynamics of Nitroxide. Base singlet radical pairs in micelles. J Phys. Chem. (1995), 99: pp 14123-14128.
4. Markov MS: Modulation of cell-free myosin light chain phosphorylation with weak frequency field. Interactions with biological systems. Freg A(ed) landes co. (1994). Internet: this is Google's cache of http://www. Diabetes from net/eng. DM free radical ros. Htm. 2002. (Scientific articles).
5. Reiter RJ, Tan B, Poeggeier A, Menendzpeiaez L, Chen D & Saareia: Melatonin as free radicals scavenger: Implications for aging and age related disease. Ann. N.Y. Acad. Sci (1994), 719: pp. 1-12.
6. Al-Wajidi, Taki A. Musa: Cross-section for inactivation of rabonuclease in solution by low energy X-rays (10 Kev. To 70Kev.), (PhD. Thesis) University of Dundee, UK, (1984): pp54-56.
7. Broklehurst of B: magnetic isotope effect in biology: a marker for radical's pair reaction and electromagnetic effect int. J. Radical. Boil. (1997), 72:pp587-596.
8. Mark H, Kleinman, Tatiana S & Cornelia B: Magnetic field effects on the dynamics of radical pair: The partition effect in vesicles, photochemistry and photobiology, (1998), 68(5): 710-718.
9. Wolff SP & Deen R: Glucose auto and protein modification: the potential role of "auto-oxidative glucosylation" in diabetes. Biochem. J. (1987), 245:pp 243-250.
10. Pawel G: Antioxidant systems-physiology and pharmaco-therapy trends. Material Medica polona. Fasc. (1992), 4(84): pp 217-222.
11. Ben & Baouli A, Aube H & Maupoil V: Plasma lipid peroxidation in critically ill patients, importance
12. Richter C: biophysical consequences of lipid peroxidation in membranes, Chem. Phys. Lipid (1987), 44: pp 175.
13. Halliwell B & Gutteridge JMC: Free radical in biology and medicine. 2nd ed. Oxford Clarendon press. (1989): pp146.

14. Monteiro HP, Bechara EJ & Abd-Alla DS: free radicals involvement in neurological prophyrian and lead poisoning. Mol. Cell biochem. (1991), 103: pp73-83.
15. Sies H & Stahl W: Vitamins E and C, B carotene and other carotenoids as antioxidants. Am J Clin Nutr (1995), 62 (suppl): pp.1315-1321.
16. Baynes JW: Role of oxidative stress in development of complications in diabetes. (1991), 40:pp405-412.
17. Paoissio G & Giugliano D: Oxidative stress and insulin action: is there a relationship. Diabetolegia, (1996), 39: pp. 357-363.
18. Wolff SP, Jiang ZY & Hunt JV: Protein glycation and oxidative stress in diabetes mellitus and and ageing. Free radic. Bio Med. (1991), 10: pp339-352.
19. Thomas E. & Andreoli MD: free radical and oxidative stress. Am. J. Med. (2000), 108: pp.650-651.
20. Jakus V: The role of free radical, stress and antioxidant system in diabetic vascular disease. Bralisl Lek Listy. (2000), 10: pp541-551(Abstract).
21. Colman PG, Wang LI & Lafferty KJ: Molecular biology and autoimmunity of type 1 diabetes mellitus in: Drazini B, Melmed S. Lepoith D. molecular and cellular biology of diabetes mellitus. Insulin secretion. New York: Alon R. Liss inc. (1989), pp125-137.
22. Chari SW, Nath N& Rathi AB: Glutathion and its redox system in diabetic polymorphonuclear Leukocytes. Am J med Sci (1984), 287:pp14-15.
23. Arai K, Lizuka S, Tada Y, Oikawa K & Taniguchi N: Increase in the glycosylated from of erythrocyte Cuzn Sod in diabetes and close association of non-enzymatic glcosylation with enzyme activity. Biochim. Biophys. Acta. (1987), 924: pp292-296.
24. Shah SV & Walker PD: Evidence suggesting a role for hydroxyl radical in glycerol induced acute renal failure. Am. J. physiol. (renal fluid electrolyte physiol. (1988), 24: pp.438-443.
25. Beutler E, Duron O & Kelly BM: improved method for the determination of blood glutathion J. Lab. Clin. Med. (1963), 61:pp882-888.
26. Tietz NW: Fundamentals of clinical chemistry 4th ed. W.b. Saunders com. (1996).
27. Altomare E, Vendemiale G, Chico D, Pocacci V & Cireli F: increased lipid peroxidation in type II poorly controlled diabetic patients. Diabetic metab. Jul-Aug. (1992), 18(4): pp 264-271 (Abstract).
28. Haffner SM, Agil A, Mykkanen L, Stern MP & Jialal: Plasma oxidizability in subjects with normal glucose tolerance impaired glucose tolerance and NIDDM. Diabetes care, (1995), 18: pp646-653 (Abstract).
29. Devaraj S & Jialal I: low-density lipoprotein postsecretory modification, monocyte function and circulating adhesion molecules in type II diabetic patients with and without macrovascular complications. The effect of α. Tocopherol supplementaion. Circulation, (2000), 102: pp191-196.
30. Marcin J, Maciej J, Michal J, Joanna G& Teresa Laskowsko-Klita: Antioxidant defense of red blood cells and plasma in stored human blood. Clinical, Chimica, Acta. (1997), 267: pp 129-142.
31. Bick LR, John MB, Russell KB, Martin JC, Lawrence K, Genesio M, Stephen BS & Partrick CJ: Morphology and biochemistry of the myeloid series in: haematology. Clinical and labrotary practice, (1993), 2:pp1053-1075.