The Egyptian Journal of Hospital Medicine Vol., 17 : 197 – 206 Dec.2004
I.S.S.N: 12084
1687 -2002

Effect Of L-Cysteine On Blood Picture And Some Serum Parameters In Rats Exposed To 2 Gauss Electro-Magnetic Field

Mervat Abdel-Rahman
Clinical pathology laboratory, Student hospital, Cairo University, Giza.

Abstract

Objective: investigation of the bio-effects of exposure to 2 gauss electromagnetic field (EMF) on blood elements, blood glucose, hepatocellular enzymes and bilirubin of mice and their possible modification by L-cysteine. Methods: the following groups were studied; (1) normal rats treated with saline; (2) normal rats treated with L-cysteine (18 mg/kg); (3) rats exposed to EMF for 21 days and treated with the vehicle (saline) during the exposure period; (4) rats exposed to EMF for 21 days and treated with L-cysteine (18 mg/kg orally, 3 times per week) during the exposure period; (5) rats exposed to EMF for 21 days and treated with the vehicle (saline) during the exposure period and for 45 days after exposure; (6) rats exposed to EMF for 21 days and treated with L-cysteine (18 mg/kg orally, 3 times per week) during the exposure period and for 45 days after exposure; Results: in rats exposed to low frequency EMF for 21 days (group2), marked increase in aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities in serum was observed. Plasma bilirubin level was raised. Meanwhile, significant decrease in plasma glucose levels occured after exposure to EMF. No significant changes were observed in haemoglobin level, red blood cells or total leucocytic count noted in rats exposed to EMF. The elevations in serum bilirubin, AST and ALT levels were reduced to near normal values in rats given L-cysteine druing the 21 days of exposure (group3). On the other hand, in rats examined 45 days after the end of the exposure period (group 3), no significant alterations were noted as regards bilirubin, AST, ALT and glucose levels in serum. Conclusions: these results suggest that (1) exposure to low frequency EMF of 50 Hz is associated with some degree of liver injury reflected in increased leakage of hepatoceular enzymes into plasma as well as an increase in serum bilirubin; (2) these alterations can be ameliorated by the administration of L-cysteine, as well as; (3) by limiting exposure to EMF.

Key Words: Electro-magnetic field, liver enzymes, rat, blood picture

Introduction

In the recent years, there has been considerable research effort undertaken on the biological effects and potential hazards of low frequency electric and magnetic fields. Especialy in Egypt, where electric equipments are being used without earthing, there exists EMF of 1-3 gauss, because of the long extension of electric cables in work places, houses, laboratories, etc…

Studies suggested that EMF may influence the physiological processes in biological systems. In particular, the effects of low frequency EMF has received considerable interest (Pasquinelli *et al*., 1993; Petrini *et al*., 1990). It has been suggested that exposure to low frequency EMF of 75 Hz and an intensity of 20 Gauss caused an increase in mouse life span (Pasquinelli *et al*., 1993), while sub-acute exposure to EMF (128 mT 1hour/day for 10 consecutive days) stimulated plasma corticosterone and liver metallothionein activities in rats (Chater *et al*., 2004). In addition, epidemiological studies have implicated EMF exposure with

increased risk of various types of cancer, including leukemia, brain and breast tumours (Wrensch *et al*., 1993; Savitz *et al*., 1988; Loomis *et al*., 1994).

One important mechanism, by which low frequency electro-magnetic field (EMF) may affect biological systems is the increasing generation of free radicals. Lalo *et al* (1994) and Kabuto *et al* (2001) suggested that steady MF could accelerate lipid peroxidation. It was also reported that 60 Hz, MF increased the phorbol 12-myristate 13-acetate (PMA) induced oxidative burst in neutrophils. Fiorani *et al* (1997) reported that MF (50 Hz, 0.5 mT) increased the damage in an oxidative stressed rabbit erythrocyte system. Lee *et al* (2003), investigated whether ELF (60 Hz) MF can modulate antioxidant system in mouse brain by detecting chemilum-inescence and measuring superoxide dismutase (SOD) activity in homogenated organs. Their results suggest that 60 Hz, MF could deteriorate antioxidant defensive system by reactive oxygen species (ROS), other than superoxide radicals.

Thiol compounds, such as glutathione (GSH), cysteine (CSH) and homocysteine (HCSH) are a natural reservoir of the reductive capacity of the cell. The most significant role played by thiols *in vivo* is their function as components of the intracellular and extracellular redox buffer. A diminished cellular GSH level accom-panies such pathological states as AIDS, liver cirrhosis, Crohn's disease and ulcerative colitis and also malnutrition (Reimund *et al*., 1998; Hadi Yasa *et al.*, 1999; Choi *et al.*, 2000). The pivotal role of redox cycle in maintaining the integrity of the biological system in the face of oxidative stress and other challenges is, therefore, of particular clinical relevance. In this respect, *N*-acetyl-L-cysteine (NAC), an antioxidant and a GSH precursor, has been shown to ameliorate cytokine transcription and synthesis, in addition to suppressing ROS-mediated lung injury (Barrett *et al*., 1999; Haddad *et al*., 2001; Behrend *et al*., 2003).

The aim of this work is therefore to assess the potential impact of 2 gauss EMF on blood elements, blood glucose, liver and kidney functions of mice. In addition, the effect of the anti-oxidant L-cysteine was assessed.

Materials and methods

Animals

Male rats in the weight range of 100-120 g maintained on standard laboratory diet were used. Rats were randomized into 5 groups (6 rats/group). The following groups were studied; (1) normal rats treated with saline; (2) normal rats treated with L-cysteine (18 mg/kg); (3) rats exposed to EMF for 21 days and treated with the vehicle (saline) during the exposure period; (4) rats exposed to EMF for 21 days and treated with L-cysteine (18 mg/kg orally, 3 times per week) during the exposure period; (5) rats exposed to EMF for 21 days and treated with the vehicle (saline) during the exposure period and for 45 days after exposure; (6) rats exposed to EMF for 21 days and treated with L-cysteine (18 mg/kg orally, 3 times per week) during the exposure period and for 45 days therafter;

Exposure facility

Animals were exposed to a homogenous magnetic field generator in which animals can be housed and exposed as described earlier (Abdel Rahman, 2004). There was no measurable difference in temperature between the room and the chamber. The animals were kept in special plastic cages that permit normal ventilation and daylight. Food and water were allowed *ad libitum* and kept in special open containers fixed on the walls of the cages. The magnetic field exposure was locally manufactured (Cairo University).

Biochemical assessment

At the end of the experiments (21 days for group 1, 3 and 4 and 66 days for groups 2, 5 and 6), animals from different groups were

anaesthetized with ether and blood samples were obtained from the retro-orbital vein plexsus. Laboratory investigations included complete blood picture, serum glucose, bilirubin, serum Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities and bilirubin. Aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities in serum were measured colorimetrically (Crowley, 1967). Bilirubin in serum was determined as described by Bauer (1982).

Statistical analysis

All results are expressed as means ± SE. A two-tailed Student's t test was used. A probability value less than 0.05 was considered statistically significant (Loether & Mc Tavish 1976).

Results

Biochemical changes after exposure to EMF Early changes

In rats exposed to low frequency EMF for 21 days, marked increase in aspartate aminotransferase (AST) and alanine aminotransferase (ALT) activities in serum was observed, indicating a form of hepatic injury. The serum AST and ALT levels were significantly elevated ($P < 0.01$) from 92.4 ± 2.2 and 42.6 ± 2.6 (saline control) to 148 ± 5.5 and 84.6 ± 3.4 IU/L, respectively. This represented 60.2 and 98.6% increases over control values, respectively. Total bilirubin was also increased in plasma from control values of 0.19 ± 0.29 to 0.5 ± 0.09 mg% ($P<0.001$). On the other hand, blood glucose was reduced by 35.6% (54.7 ± 2.9 mg%). Control value was 84.8 ± 4.6 mg% ($P<0.01$). (Table 1).

In rats treated with L-cysteine during the time of exposure to EMF (21 days) and killed therafter, the elevations in AST, ALT and bilirubin were reduced to near normal values (AST; 88.1 ± 3, ALT; 50.3 ± 4.1; bilirubin; 0.16 ± 0.02). The decrease in plasma glucose, on the other hand, was not improved by L-cysteine treatment (Table 1).

Late changes

Rats exposed to EMF for 21 days, but killed 45 days after the end of the exposure period showed no significant alterations in AST, ALT, bilirubin or glucose levels in serum, indicating that removal from the EMF can ameliorate the abnormalities seen during the exposure period. L-cysteine administration did not significantly change any of the parameters studied. However and unexpectedly, serum GPT was raised in those received L-cysteine for this long period (Table 1).

Blood picture

In rats exposed to EMF, no significant changes were observed as regards total leucocytic count, red blood cell count, haemoglobin level, haematocrite values, mean cell volume, or mean cell haemoglobin concentration.

Table 1. AST, ALT, bilirubin and glucose levels in serum of rats exposed to EMF (2 gauss) with or without L-cysteine treatment

	Normal control	Normal controls + L-Cysteine	Exposed (2 gauss) for 21 days	Exposed 21 days + L-Cysteine during EMF exposure	Exposed (2 gauss) for 21 days and killed 45 days after the end of exposure period	Exposed (2 gauss) for 21 days and killed 45 days after the end of exposure period + L-cysteine treatment since beginning of EMF exposure
AST	92.4 ± 2.2	56.8 ± 3.1	148 ± 5.5	88.1 ± 3	70.6 ± 4.0	92.8 ± 6.4
ALT	42.6 ± 2.6	47.8 ± 4.2	84.6 ± 3.4	50.3 ± 4.1	50.6 ± 3.7	80.8 ± 6.9
Bilirubin	0.19 ± 0.29	0.22 ± 0.01	0.5 ± 0.09	0.16 ± 0.02	0.18 ± 0.02	0.17 ± 0.03
Glucose	84.8 ± 4.6	94 ± 3.9	54.7 ± 2.9	40.8 ± 4.3	96 ± 6.2	91.7± 7.9

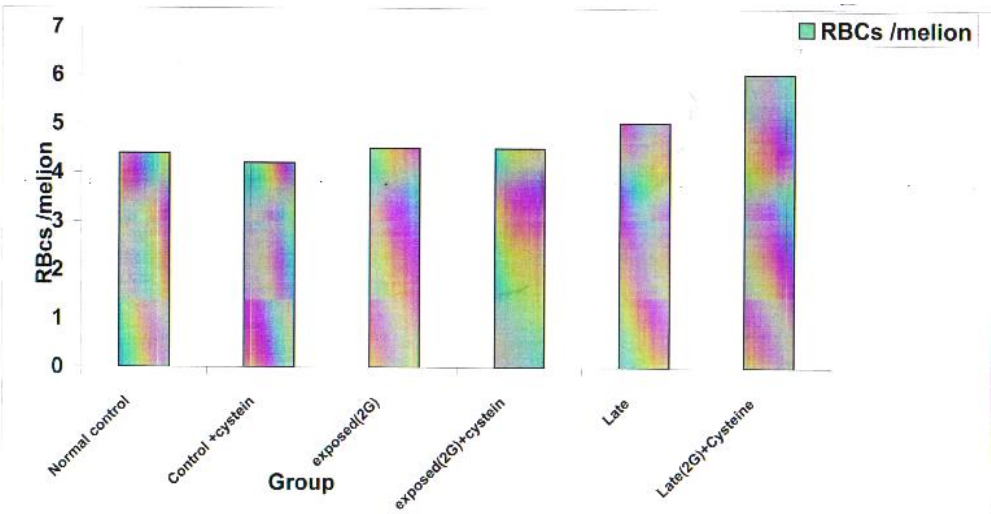

Fig 1:- **Effect of EMF at (2 Gauss) on red blood cell count (RBCs /melion) for control groups and group exposed +Cystein**

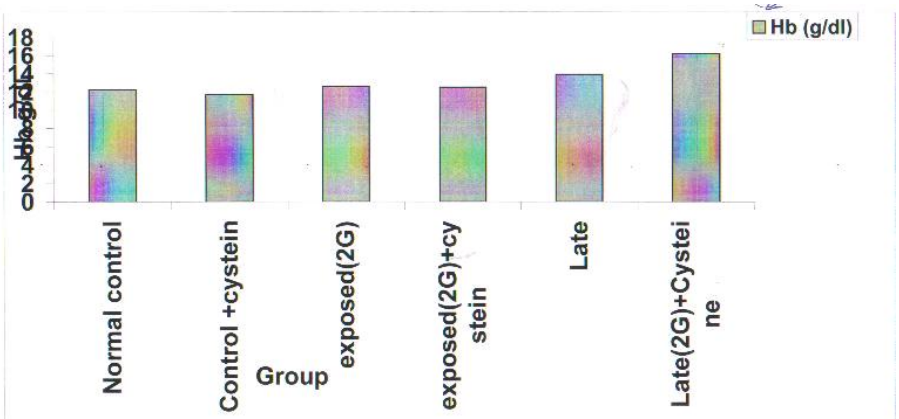

Fig 2:- **Effect of EMF of (2 Gauss) of group exposed + cystein on hemoglobin concentration (Hb g/dl).**

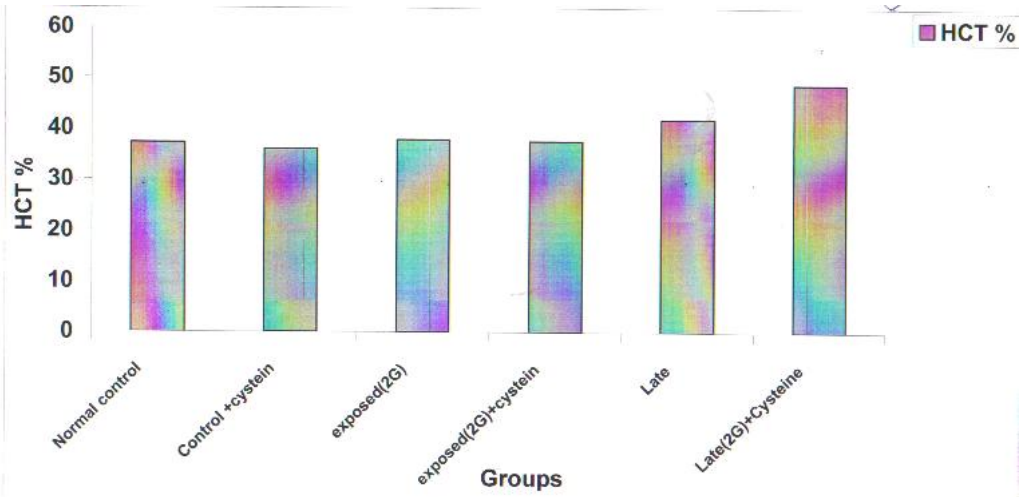

Fig 3:- Effect of EMF of (2 Gauss) on hematocait value (HCT %) for control groups and group exposed + cystein

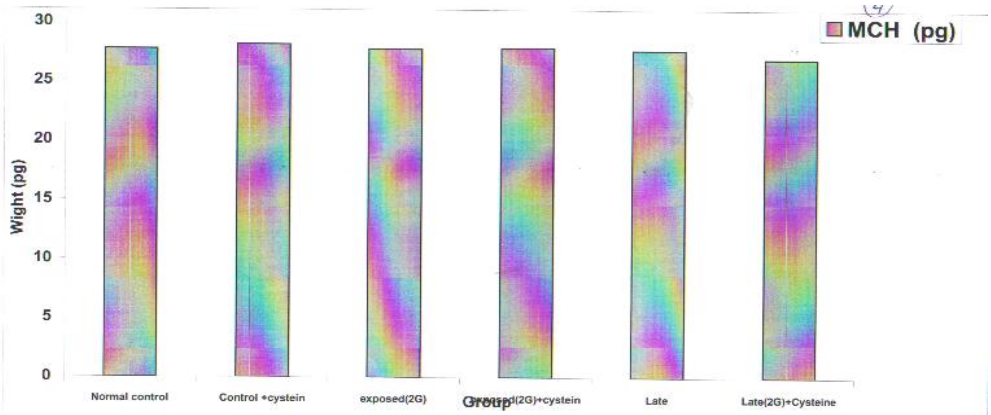

Fig 4:- Effect of EMF of (2 Gauss) on mean corpuscular hemoglobin (MCH pg) fof control group and group exposed +Cystein

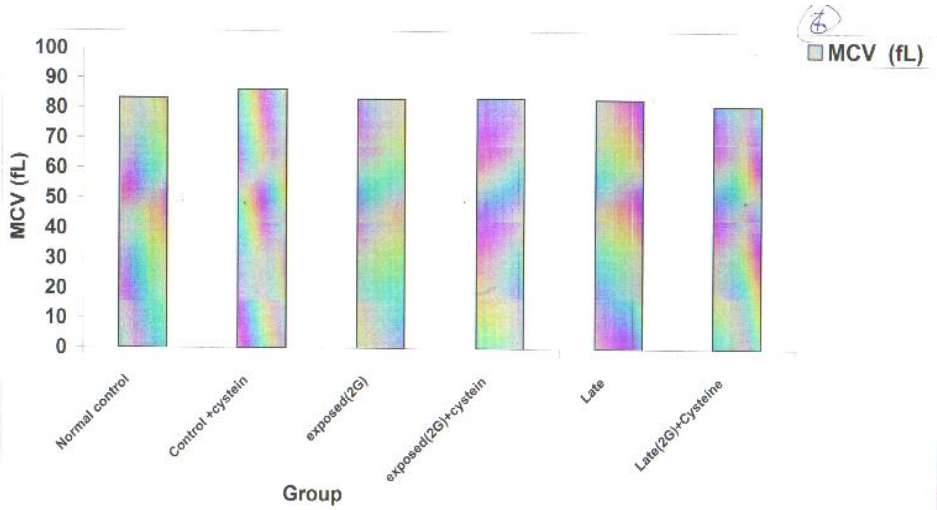

Fig 5:- Effect of EMF of (2Gauss) on mean corpuscular volume (MCV fL) for control group and group exposed +Cystein

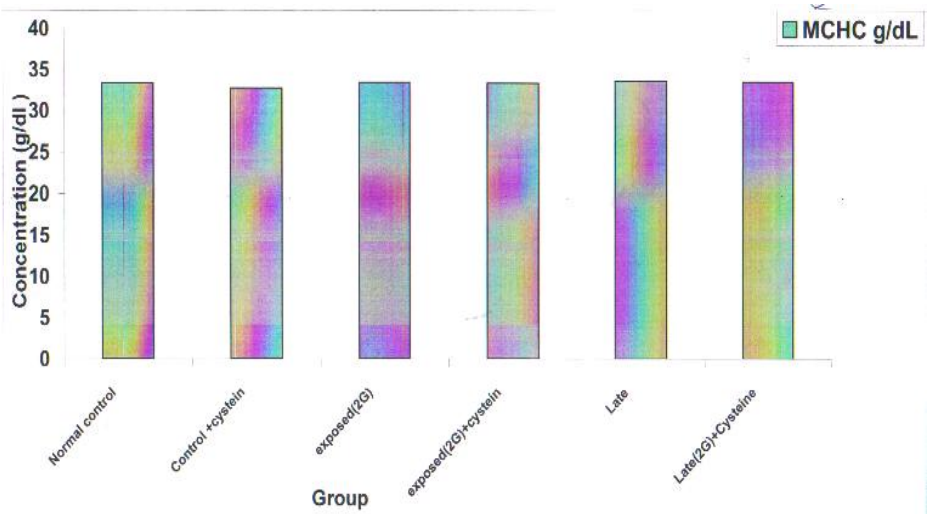

Fig 6:- Effect of EMF of (2 Gauss) on mean corpuscular hemoglobin (MCHC g/dl) for control group and group exposed + Cystein .

Discussion

The present study provide evidence that exposure to 2 gauss EMF can result in some form of liver injury. This is because significant elevations of the hepatocellular enzymes AST and ALT were observed in plasma of rats at 21 days of exposure to EMF. These enzymes contained within the hepatocytes are released into blood after hepatocyte injury and increased cell membrane permeability. Serum bilirubin, a marker of liver damage and/or cholestatsis was also increased. Plasma glucose was significantly decreased by exposure to EMF, suggesting derangement of normal glucose homeostasis, which could be also of liver origin. The changes mentioned above were not observed in plasma of rat after the recovering period. This important observation, suggest that the biochemical changes observed can be reversed after avoiding exposure to the field.

Various reports suggest that the interaction site for ELF-EMF is the plasma membrane, since exposure determines altered Ca^{+2} influx (Flipo *et al*., 1998; Fanelli *et al*., 1999), as well as the clustering of integral plasma membrane proteins (Bersani *et al*., 1997). In addition, ELF-EMF-induced oscillations of intracellular Ca^{+2} concentration (Loschinger *et al*., 1998) have been correlated with changes in DNA, RNA and protein synthesis activities (Liburdy, 1992). More recently, the application of ELF-EMF has been involved in the reduction (Fanelli *et al*., 1999) or stimulation (Flipo *et al*., 1998) of somatic cell apoptosis.

One mechanism by which EMF can affect biological systems, may be through increasing the generation of oxygen derived free radicals and oxidaive stress. Free radicals are generated under normal conditions in mitochondria as a consequence of oxidative phosphorylation in the respiratory chain. Additionally, they may be produced in response to many different endogenous and exogenous stimuli in the endoplasmatic reticulum, the peroxisomes, and the cytosol. Under certain conditions, reactive oxygen species may induce oxidative cell injury. Oxygen radical species such as superoxide radical, hydrogen peroxide and hydroxyl radical are highly reactive resulting in oxidative damage to lipids, proteins, and DNA (Haddad 2001; Papp *et al*., 2003).

The protection is provided by the antioxidant defense system, including enzymatic and non-enzymatic components**.** Thiol compounds, such as glutathione (GSH), cysteine (CSH) and homocysteine (HCSH) are a natural reservoir of the reductive capacity of the cell. The tripeptide L-γ-glutamyl-L-cysteinyl-glycine, or glutathione (GSH), plays a major role in maintaining intracellular reduction-oxidation (redox) equilibrium in the lung. The cysteinyl moiety of GSH provides the reactive thiol as a functional element responsible for the diverse properties of glutathione (Haddad, 2001)**.** *N*-acetyl-L-cysteine (NAC), an antioxidant and a GSH precursor, has been shown to ameliorate cytokine transcription and synthesis, in addition to suppressing ROS-mediated lung injury. In contrast, irreversible inhibition of γ-glutamylcysteine synthetase (γ- GCS), the rate-limiting enzyme in GSH biosynthesis, by the action of L-buthionine-(*S,R*)-sulfoximine (BSO), has been shown to enhance cytokine release by inducing the intracellular accumulation of ROS (Behrend *et al*., 2003). NAC (N-acetyl cysteine) is frequently used as a mucolitic and as an antidote in paracetamol intoxication ((Kucukardali *et al*., 2002; Zhao *et al*., 1998). NAC may maintain cell integrity by increasing the amount of glutathione within the cell or coming into direct reaction with spontaneous conjugation and/or reduction (Flanagan and Meredith , 1991).

In conclusion, the results obtained from the present observations indicated that animals treated with the anti-oxidant L-cysteine during the period of EMF exposure did not suffer from liver cell injury. This is in contrast to rats not treated with L-cysteine.

This indicates that oxidative stress has been involved in the changes observed with liver enzymes and bilirubin. Whereas treatment after removal from the field of exposure did not significantly affect the studied parameters, except serum activity of GPT. This result is of particular interest since L-cysteine does not appear to be beneficial in preventing injury in this case. The results of the present work also indicated that removal from the field of exposure markedly lessened the changes seen in bilirubin, hepatocellular enzymes and blood glucose. This suggest that stopping exposure to EMF, e.g., by changing the job circumstances is necessary for those individuals whose work conditions subject them to long periods of exposure to EMF.

Acknowledgement

The author acknowledge the Biophysics Department at Bini Suif University for the facilities provided.

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تأثيرالسيستين علي صورة الدم وعلي دلالات مختلفة بالدم في الفئران التي تم تعريضها للمجال الكهرومغناطيسى منخفض الترددات

د. ميرفت عبد الرحمن
معمل الباثولوجيا الاكلينيكية، مستشفي الطلبة، جامعة القاهرة. الجيزة

هدف هذا البحث دراسة تأثير التعرض للمجال الكهرومغناطيسى منخفض الترددات على صورة الدم، وانزيمات الكبد والبليروبن ومستوي السكر بالدم في فئران التجارب البيضاء ومدي تأثير مضاد الاكسدة السيستين. اشتملت الدراسة علي 5 مجموعات من الفئران : المجموعة الاولي فئران لم تتعرض للمجال الكهرومغناطيسى، المجموعة الثانية فئران لم تتعرض للمجال الكهرومغناطيسى وتم علاجها بالسيستين، المجموعة الثالثة فئران تم تعرضها للمجال الكهرومغناطيسى ، المجموعة الرابعة فئران تم تعرضها للمجال الكهرومغناطيسى لمدة 21 يوما وتم علاجها بالسيستين اثناء فترة التعرض للمجال ، المجموعة الخامسة فئران تم تعرضها للمجال الكهرومغناطيسى لمدة 21 يوما بالاضافة الي فترة ابتعاد عن المجال لمدة 45 يوما اضافية ، المجموعة السادسة فئران تم تعرضها للمجال الكهرومغناطيسى لمدة 21 يوما بالاضافة الي فترة ابتعاد عن المجال لمدة 45 يوما اضافية وتم علاجها بالسيستين اثناء فترة التعرض للمجال وكذلك اثناء فترة النقاهة من المجال.

أظهرت النتائج ان التعرض للمجال الكهرومغناطيسى منخفض الترددات ادي الي ارتفاع ملحوظ في انزيمات الكبد، البليروبن,بالاضافة الي انخفاض في مستوي الجلوكوز بالدم. هذه التغييرات عادت الي المستوي الطبيعي مع استخدام السيستين. اما في الفئران التي قضت فترة نقاهه مدتها 45 يوما بعد انتهاء فترة التعرض للمجال فقد انخفضت التغييرات السابق ذكرها بصورة كبيرة مما يوضح ان الابتعاد عن المجال الكهرومغناطيسى يؤدي الي تحسن وضع خلايا الكبد. كذلك فأن العلاج بأستخدام مضادات الاكسدة اثناء فترة التعرض للمجال الكهرومغناطيسى له فائدة علاجية.