ROLE OF SELENIUM AND VITAMIN E IN OCCUPATIONAL EXPOSURE TO HEAVY METALS (MERCURY, LEAD AND CADMIUM) : IMPACT OF WORKING IN LAMP FACTORY

BY

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ABSTRACT

Heavy metals are environmental contaminants that may pose long-term health risks. Unfortunately, the consequent implementation of preventive measures was generally delayed, causing important negative effects to the exposed populations. The objective of this study was to determine whether co-consumption of nutritional supplements as selenium and vitamin E would treat the hazardous effects of exposure to mercury, lead and cadmium. Thirty-six workers (20 males and 16 females) were the subjects of this study, their ages ranged from 19-63 years, (M = 29.5±10.12). They were working in lamp factory for an average of 0.5-40 years (M= 5.3±8.8). Twenty control subjects matched for age and gender were used for comparison. All workers were subjected to neuropsychiatric evaluation. General Health Questionnaire (GHQ-28) revealed that 44.4% were complaining of anxiety, 52.7% of depression, 41.6% of social dysfunction and 22.2% of somatic symptoms. Cognitive tests revealed that long-term memory was not affected significantly when compared with controls, while short term memory and perceptual ability were affected significantly. Blood metal levels were measured by energy dispersive X-ray fluorescence (EDXRF), and revealed that the mean blood mercury, lead and cadmium concentrations before treatment were 1.6 mg/l, 0.39 mg/l and 1.7 μg/l, while they decreased significantly after treatment to 1.2 mg/l, 0.29 mg/l and 1.3 μg/l respectively. Anti-oxidative enzymes (paraoxonase and catalase) and lipid peroxidation product (malondialdehyde) were measured before and after treatment with selenium and vitamin E, and showed significant improvement. It could be concluded that co-consumption of selenium and vitamin E produces significant decrease in mercury, lead and cadmium levels in blood, and significant improvement in the antioxidative activity which may treat the neuropsychiatric disorders occasioned by chronic occupational exposure to heavy metals.

Keywords: mercury, lead, cadmium, neuropsychiatric impairment, selenium, vit E.

INTRODUCTION

The sulfhydryl-reactive metals (mercury, cadmium, lead, and also arsenic) are particularly insidious and can affect a vast array of biochemical and nutritional processes. The symptoms associated with the slow accumulation of toxic metals are multiple and rather non descriptive, and overt expression of toxic effects may not appear until later in life (Quig, 1998).

Mercury (Hg) is used in many industrial implications as several types of electric lamps, including fluorescent lamps and high intensity discharge (HID) lamps; in electric light switches and thermostats, in dental amalgams and many other industries. Chronic exposure to elemental mercury yields a classic triad of gingivitis and salivation, tremor and neuropsychiatric changes (Goldwater, 1972), known as erethism including, irritability, emotional liability, memory loss, combativeness, anxiety, withdrawal and depression (Smith, 1978). The harmful effects of long-term exposure to elemental mercury are generally thought to be caused by inhalation exposure. However, mercury liquid and vapor are absorbed through the skin in small amounts and this route of exposure can contribute to the overall exposure. Effects following absorption through the skin are expected to be similar to those reported for long-term inhalation exposure. Long term mercury exposure causes harmful effects on the nervous, digestive and respiratory systems, and the kidneys (Agency for Toxic Substances and Disease Registry, 1990).

During the past century, lead (Pb) exposures in the workplace are common because of the widespread use of lead compounds in paints, gasoline, and industry. Human lead exposure occurs when dust and fumes are inhaled and when lead is ingested via lead-contaminated hands, food, water, cigarettes, and clothing. Lead entering the respiratory and digestive systems is released to the blood and distributed throughout the body. The most important aspects of lead toxicity are its effects on the central nervous system, which may be irreversible; however, lead affects all organs and functions of the body to varying degrees (Agency for Toxic Substances and Disease Registry, 1990). The frequency and severity of symptoms among exposed workers depend upon the level of exposure. Although much progress has been made in reducing lead exposures, exposures in the workplace continue to be a significant public health problem (Centers for Disease Control and Prevention, 1994).

Cadmium (Cd) is listed by the US Environmental Protection Agency as one of 126 priority pollutants. The most dangerous characteristic of cadmium is that it accumulates throughout a lifetime. Cadmium accumulates mostly in the liver and

kidneys and has a long biological half-life of 17 to 30 years in humans (Hideaki et al., 2008). Cadmium's uses vary from an electrode component in alkaline batteries to a stabilizer in plastics. Although animal studies have shown that cadmium is a potent neurotoxicant, little is known about its toxicity for the human central nervous system (Viaene et al., 2000). Occupational exposure to cadmium is associated with cognitive impairment in adults. Cadmium interferes with several important nervous system functions, but the mechanisms of neurotoxicity remain uncertain (Hart et al., 1989).

Lead and cadmium induce clastogenic as well as aneugenic effects in peripheral lymphocytes, indicating a potential health risk for working populations with significant exposures to these heavy metals (Palus et al., 2003).

One of the major mechanisms behind heavy metal toxicity has been attributed to oxidative stress. Antioxidants such as selenium and vitamin E act to protect cells against the effects of free radicals, which are potentially damaging by-products of energy metabolism. Free radicals can damage cells and may contribute to the development of many human diseases (Van Gaal et al., 2006).

Selenium is an "essential" trace element, which has a variety of functions. It is a key component of several functional selenoproteins required for normal health. Whereas selenium is poisonous to man and animal in large amounts (De Lorgeril and Salen, 2006). Role of selenium as a protective against exposure to metals is a conflict issue. Most studies conclude that selenium has a beneficial role while few show no role (Hansen, 1988).

Vitamin E has also been shown to play a role in immune function, in DNA repair, and other metabolic processes (Farrell and Roberts, 1994). The antioxidant properties of vitamin E seemed to be responsible for protection from cadmium toxicity (Tandon et al., 1992) as well as iron-induced lipid perioxidation specially in combination with glutathione "GSH", and N-acetyl-cysteine "NAC" (Milchak and Douglas, 2002).

Co-exposure to cadmium, mercury, lead and other heavy metals occurs in many occupational settings, such as pigment and batteries production, galvanization and recycling of electric tools lamp factories. However, little is known about co-exposure to heavy metals.

This study aims to investigate the benefit of co-consumption of selenium and vitamin E either alone or in combination in case of co-exposure to mercury, lead and cadmium during working in a lamp factory.

SUBJECTS, MATERIAL AND METHODS

Subjects:

The study was conducted on 56 subjects, 36 workers in a lamp factory at Monoufiya Governorate in Egypt (20 males and 16 females), ages ranged from 19-63 years, (M = 29.5±10.12). Subjects were working for 0.5 to 40 years (M= 5.3±8.8) and 20 control subjects matched for age, gender, dietary habits, smoking, dental amalgam and life style with the workers.

Subjects were classified as follows:

Group I : Control group (10 males and 10 females).

Group II: 36 factory workers divided into three subgroups a, b and c.

Group II a: 12 subjects (3 females and 9 males) received only selenium 100 µg orally once daily before breakfast for 2 months.

Group II b: 12 subjects (6 females and 6 males) received only 100mg vitamin E orally once daily before breakfast for 2 months.

Group II c: 12 subjects (7 females and 5 males) received combination of selenium and vitamin E with the same doses as mentioned in the previous groups, once daily before breakfast for 2 months.

Material

Selenium : 100 µg tablet, 250 tablet yeast free, dietary supplement, vegetarian formula. Manufactured by NOW FOOD, BLOOMINGDALE, IL 60108, USA.

Vitamin E : 100mg capsule, 24 capsules, manufactured by Pharco.

Methods :

Blood sample collection: After taking written consent, venous radial blood samples were collected from each subject the day before starting medications, and at the end of research, as 2.5 ml on sterile heparinized vacutainer tube and 5ml on sterile vacutainer plain tube. Separation of serum was done at the same day of sampling by centrifugation at 1500 rpm for 15 minutes at 4ºC. Samples were preserved at - 20ºC till conduction of analysis. The examination included blood samples analysis for determination of metals including, selenium, cadmium, mercury and lead. Serum levels of malondialdehyde (MDA), serum activity of catalase, and paraoxanse enzymes were also detected. Remeasuring metals and oxidative enzymes levels after 2 months treatment were done for the workers.

Heavy metal analysis: Selenium, lead, cadmium and mercury quantitative analysis was performed by means of micro analysis e.g. energy dispersive X-ray fluorescence (EDXRF), a highly sensitive multi elemental method of micro-analysis. The EDXRF technique was chosen for its simplicity, accuracy, and sensitivity, as well

as the samples do not need complex preparation. Plasma samples were collected and dried as powder being placed on KIMPOL polycarbonate film as a backing material previously mounted on a polyethylene target frame and fixed by 1% polystyrene in benzene. The samples collected by deposition on a filter paper and were put in an electric oven at 80°C for 4 days until dryness. The samples were grinded for full homogenization and very fine powder formation until analysis with Tube Excited X- ray Fluorescence Analysis apparatus (El Nimr et al., 1992).

Biochemical analysis:

1- Measurement of Serum PON1 (paraoxonase) enzyme activity : PON1 activity was measured by adding 15μl serum to 285μl Tris buffer (100mmol/L, pH 8.0) containing 2 mmol/L CaCL$_2$ and 1mmol/L paraoxon (O,O-diethyl-O-nitrophenylphosphate (Sigma Chemical Co.). The rate of generation of P-nitrophenol was determined at 405nm, 25°C with the use of continuously recording spectrophotometer as described previously(Mackness et al.,1991)

2- Measurement of Catalase (CAT) activity: CAT was measured by determining the constant rate of hydrogen peroxide decomposition. The assay was performed according to the method described by Pifferi et al. (1981) with modifications as set forth by Nowak et al. (1997). The reaction mixture prepared in an Eppendorf tube

consisted of 500 μL 0.05 M phosphate buffer (pH 7.0), 300 μL distilled water, 50 μL 1.1 mM H$_2$O$_2$ in distilled water, and 50 μL serum sample (or 50 μL distilled water as blank). After 5 minutes of incubation at 25°C, 100μL 50% trichloroacetic acid (Sigma Chemical) was added to each tube and the tubes were centrifuged (1,000 g for 5 minutes). To each tube 10 μL titanium (IV) reagent was added and 200 μL supernatant was transferred into wells on a 96-well micro plate. The absorbance was read at 405 nm in the micro plate reader. The results were expressed as CAT activity in units per milliliter of serum, after comparison with the standard curve. The standard curve was prepared by plotting the absorbance at 405 nm (ordinate) as a function of the standard CAT (Sigma Chemical) concentration between 0 to 33 U/ml. One unit CAT decomposed 1 μmole H$_2$O$_2$ per minute at a pH 7.0 and 25°C.

3- Measurement of malondialdehyde (MDA) serum level: MDA was based on the coupling of MDA with thiobarbituric acid briefly, serum samples were diluted in phosphate buffer (20mM, pH7.4) and heated together with a thiobarbituric acid TBA solution (375 mg/ml) in a boiling water bath for 15 min. The tubes were cooled, and the absorbance was measured by spectrophotometry at 535nm. 1,1,3,3-Tetraethoxypropane. Sigma, St. Louis, MO was used as a stander (Bird and Draper, 1984).

Medical and neurological examination:

The medical examination included a medical history, an evaluation of some behavioral and biological risk factors for cardiovascular disease (Marmot, 1988) and an evaluation of clinical neurological status: postural tremors of the upper extremities and a hand writing test, coordination, reflexes, the presence or absence of pathological reflexes, including snout reflex and pin pain sensation, touch pressure, two- point discrimination and vibratory sensation. Neurological examination was performed to detect complaints consistent with peripheral neuropathy and dysfunction of the autonomic nervous system.

Neuropsychological examination : All the workers were screened for the presence of psychiatric symptoms and / or disorders by General Health Questionnaire (GHQ-28), Arabic version (Godlberg and Hiller, 1979). GHQ-28 is widely used to screen populations for the presence of psychiatric illness. A number of validity studies indicate that GHQ is a reasonably sensitive and specific indicator of psychopathology (Marder, 1995).

The general health Questionnaire (GHQ-28) is a self-administered instrument, designed to detect four elements of distress : depression, anxiety, social impairment, and hypochondriasis.

(GHQ-28) consists of four subscale items:
  A- Somatic symptoms
  B- Anxiety and insomnia.
  C- Social dysfunction.
  D- Depression.

The scoring method was 0-0-1-1 for all 28 items, for each symptom in each group a total score of 5 or more indicates the presence of the symptom, while below 5 score means no symptoms (Goldberg et al., 1997).

The (GHQ-28) was distributed to the studied subjects at their work settings. The educated subjects take usually 15 -25 minutes to complete the GHQ. The examiner helped the non- educated subjects. After that, the data were collected for analysis.

RESULTS

I- Results of metal analysis by energy dispersive X-ray fluorescence (EDXRF):

It was found that there is significant elevation in blood concentrations of mercury, cadmium and lead in factory workers (1.576 ± 0.26, 1.77 ± 0.344 and 0.3831 ± 0.0669 respectively), when compared with control subjects (0.234 ± 0.026, 1.223 ± 0.0709, and 0.245 ± 0.035 respectively) (Table 1).

No significant changes were observed in different age groups or between both

sexes in the factory workers group in blood metal levels (Tables 2-3).

Regarding the duration of exposure of the workers, which ranges from 6 months to 40 years, it was noticed that there was so significant changes in different durations of exposure (Table 4). Following treatment by selenium and vitamin E, there was significant decrease in blood levels of Hg, Cd and Pb when compared with the same workers before treatment (Table 5). As regards type of treatment they received, no significant differences were noticed between different groups (Table 6).

II- Results of biochemical analysis:

As regards the biochemical results, there is significant decrease in serum CAT and PON1 activity and increase in serum MDA level in the exposed workers in comparison with the control group (Table 7). Following treatment with selenium and vitamin E, there was significant increase in serum CAT and PON1 and decrease in serum MDA level in workers after 2 months treatment in comparison with themselves before treatment (Table 8).

III- Results of the neuropsychological evaluation:
A- The General Health Questionnaire (GHQ):
Using the general health questionnaire,

it was found that anxiety, insomnia and depression were significantly noticed among workers, and no significant differences as regards social dysfunction and somatic symptoms in comparison with control group (Table 9).

B- Cognitive tests:
Cognitive tests revealed significant deterioration in short term memory, perceptual ability, short term visual memory as well as in trail making test in comparison with the control group, while no significant differences were detected as regards long term memory (Table 10).

DISCUSSION

Metal induced toxicity is very well reported in the literature. A growing amount of data provide evidence that metals are capable of interacting with nuclear proteins and DNA causing oxidative deterioration of biological macromolecules (Leonard et al., 2004).

Normal levels of mercury, cadmium and lead in blood are < 0.04mg/l, 0.1-0.4 ug/l and 0.1-0.4 mg/l respectively (Gothe, 1985; Roper, 1991 and Young-Jin, 2006). This means that mercury, cadmium and lead blood levels in the current study both in the control group and the factory workers group were above the normal levels, whereas in workers were significantly higher than the control. This was associat-

ed with significant decrease in serum CAT and PON1 activity and increase in serum MDA levels as well as higher incidence of neuropsychiatric disorders in the factory workers in comparison with the control. The elevated levels of the mercury, cadmium and lead may posed these neuropsychiatric disorders.

A number of researchers have also shown enhanced rate of lipid peroxidation in brain of lead exposed rats (Adegbesan and Adenuga, 2007 and Bokara et al., 2008). One of the suggested mechanisms is inhibition of d-aminolevulinic acid dehydratase (ALAD). There was an excessive production of n-NOS (neural nitric oxide synthase) and MAO (mono-amino oxidase), depletion of GABA, 5HT and AchE, which are important neurotransmitters that control neurobehavioral changes (Flora et al., 2007).

Several studies have shown that lead alters the activity of antioxidant enzymes like superoxide dismutase (SOD), catalase, glutathione peroxidase (GPx) and glucose 6-phosphate dehydrogenase (G6PD) and antioxidant molecules like GSH in animals, and human beings (Gurer and Ercal, 2000).

Whanger (1992) reported that selenium is essential for GPx activity, and lead forms a complex with selenium, thereby decreases its activity.

Galan et al. (2001) showed that cadmium, unlike other heavy metals is unable to generate free radicals by itself, however, reports have indicated superoxide radical, hydroxyl radical and nitric oxide radicals could be generated indirectly

Watanabe et al. (2003) showed generation of non-radical hydrogen peroxide which by itself became a significant source of free radicals via the Fenton chemistry.

Numerous reports on animal models have depicted that cadmium intoxication significantly increased the malondialdehyde (MDA) and glutathione peroxidase (GSH-Px) (Yang et al., 2003; Cosic et al., 2007).

Neuronal inhibition by CdCl2-induced oxidative stress is a new mechanism of cadmium action which may directly produce neurotoxic symptoms as well as implicate cadmium and related metals as environmental factors in the etiology of neurodegenerative diseases (Monroe and Halvorsen, 2006).

Mahboob et al. (2001) showed that when mice were exposed to mercuric chloride, there were alterations in the lipid peroxidation (LPO), glutathione reductase (GR), glutathione peroxidase (GPx), superoxide dismutase (SOD) and GSH levels in different organs. Toxic effects of mercury have also been observed

in oligodendrocytes, astrocytes, cerebral cortical and cerebellar granular neurons obtained from embryonic and neonatal rat brains (Yee and Choi, 1996).

However, Peter et al. (2002) concluded that oxidative stress and changes in GSH and activities of antioxidant enzymes do not play a major role in Hg vapor toxicity in brain and kidney. Exposure to low levels of mercury vapor has been associated with impairment of neurobehavioral function.

Some studies concluded that there was no strong evidence that blood mercury levels are associated with worse neurobehavioral performance in older urban adult (Mutter and Naumann, 2005 and Weil et al., 2005).

The biochemical results in our study revealed significant decrease in serum CAT and PON1 activity and increase in serum MDA level in the exposed workers in comparison with the control group.

The production of malondialdehyde is used as a biomarker to measure the level of oxidative stress in an organism (Del Rio et al., 2005). Elevation of its level is an indication of oxidative stress exposure.

The protective role of paraoxonase against lipoprotein lipid peroxidation was suggested to be related to the ability of PON1 to hydrolyze oxidized lipids including oxidized phospholipids and cholesteryl ester hydroperoxides (Aviram et al., 1999). Compounds (from diet or drugs) with antioxidative activity against lipoprotein oxidation preserved PON1 activity, in association with their ability to protect lipoproteins against oxidation (Aviram et al., 2000).

Catalase is a common enzyme found in nearly all living organisms which are exposed to oxygen, where it functions to catalyze the decomposition of hydrogen peroxide to water and oxygen (Chelikani et al., 2004).

The protective effect of selenium against lead toxicity may be attributed to a competition between selenium and lead for binding with the functional bioligands or to the in vivo formation of lead selenide (Flora et al., 1983). It is known that the oxidation- reduction processes of mercury and selenium play a mediated role in the accumulation of inorganic mercury in tissue and its loss from tissue cells (Clarkson and Kilpper, 1978; Magos, 1991). This can explain the decrease in blood lead and mercury level following treatment by selenium in the current study.

Experimental studies conducted on the protective effect of selenium against the

hepatotoxicity and renal toxicity of cadmium concluded that selenium could have a protective effect by its antioxidant effect which counter the oxidative stress induced by cadmium that may cause its hepatotoxic and nephrotoxic effects (El-Sharaky et al., 2007; Newairy et al., 2007).

Vitamin-E could be useful in order to protect membrane-lipids and, notably, to prevent protein oxidation produced by lead intoxication. It has been claimed that α-tocopherol is the most important lipid-soluble antioxidant, and that it protects cell membranes from oxidation by reacting with lipid radicals produced in the lipid peroxidation chain reaction (Rendon-Ramirez et al., 2007; Traber and Atkinson, 2007).

This would remove the free radical intermediates and prevent the oxidation reaction from continuing. Vitamin E has a greater effect than selenium in reducing lipid peroxidation in various brain regions (Whanger, 2001).

Reports have shown that antioxidants like vitamin C and Vitamin E have shown protection against cadmium induced toxicity in different animal models (Ognja-novic et al., 2003 and Beytut et al., 2003). Cosic et al. (2007), showed that presence of antioxidants like cysteine, glutathione and ascorbate induced more DNA damage in in-vitro experiments. This DNA damage was considered to be due to the generation of reactive species. Experimental studies explore the protecting effect of vitamin E and selenium either alone or in combination against the toxic metal effects especially cadmium, mercury and lead (Beyrouty and Chan, 2006) which agrees with the current study.

The higher incidence of neuropsychiatric disorders observed in the workers in comparison with control may be mediated by the oxidative stress caused by mercury, cadmium, and lead. Decrement in this oxidative stress occurs after treatment by selenium and vitamin E and was measured by significant increase in serum CAT and PON1 activity and decrease in serum MDA level following treatment. As the controls also have high levels of metals, so it may be related to the increased incidence of neuropsychiatric disorders among non-exposed population, so selenium with vitamin E could play a role in decreasing incidence of these disorders.

It could be concluded that supplemental consumption of selenium or vitamin E by workers occupationally exposed to lead, cadmium and mercury could protect against neurobehavioral disorders that could appear along chronic exposure.

Table (1): Comparison between blood levels of mercury, cadmium and lead of the control group and the factory workers group.

	Factory workers group (n =36)	Control group (n =20)	t	P
	Mean ± SE	Mean ± SE		
Hg (mg/l)	1.576± 0.26	0.234± 0.026	-30.66	0.000*
Cd (ug/l)	1.77± 0.344	1,223 ± 0.0709	-8.82	0.000*
Pb (mg/l)	0.3831 ± 0.0669	0.245 ±0.035	-10.06	0.000*

*Significant difference (p<0.05)

Table (2): Comparison between blood metal levels of different age groups.

Age group	Mean Hg± SE	Mean Pb± SE	Mean Cd± SE
< 30 Y (n=26)	1.602± 0.054	0.386± 0.014	1.743± 0.066
> 30 Y (n= 10)	1.509± 0.067	0.374± 0.014	1.755± 0.12
T	1.08	0.61	0.08
P	0.293	0.546	0.936

*Significant difference (p<0.05)

Table (3): Comparison between blood metal levels of both sexes.

Sex	Mean Hg± SE	Mean Pb± SE	Mean Cd± SE
♂ **(n= 20)**	1.611± 0.06	0.373± 0.011	1.831±0.096
♀ **(n=16)**	1.532± 0.063	0.395± 0.021	1.641±0.035
T	0.91	0.91	1.85
P	0.371	0.373	0.078

*Significant difference (p<0.05)

Table (4): Comparison between blood metal levels in different durations of exposure.

Duration of Exposure	Mean Hg± SE	Mean Pb± SE	Mean Cd± SE
0.5 - 2 ys (n=20)	1.578± 0.054	0.397± 0.016	1.697± 0.067
>2-5 ys (n=9)	1.68± 0.1	0.382± 0.022	1.918± 0.17
>5-10 ys (n=5)	1.388± 0.11	0.346±0.019	1.652± 0.032
>10 ys (n=2)	1.565± 0.11	0.34± 0.02	1.705±0.015
P value	0.262	0.369	0.393

*Significant difference (p<0.05)

Table (5): Comparison between blood levels of mercury, cadmium and lead in the factory workers before and after treatment with selenium and vitamin E.

	Factory workers group before treatment	Factory workers group after treatment	t	P
	Mean ± SE	Mean ± SE		
Hg	1.576± 0.26	1.231±0.194	6.38	0.000*
Cd	1.77± 0.344	1.366± 0.209	5.68	0.000*
Pb	0.3831 ± 0.0669	0.2967 ± 0.0464	6.37	0.000*

*Significant difference (p<0.05)

Table (6): Comparison between blood metal levels in the factory workers as regards type of treatment.

Type of treatment	No.	Mean Hg± SE	Mean Pb± SE	Mean Cd± SE
Vit. E (n=12)	12	1.612± 0.097	0.367± 0.014	1.769±0.1
Selenium (n=12)	12	1.520±0.075	0.398±0.024	1.729± 0.11
Both Vit. E and Selenium (n=12)	12	1.595± 0.05	0.383±0.019	1.742± 0.1
P value		0.668	0.542	0.961

*Significant difference (p<0.05)

Table (7) : Comparison between serum levels of CAT, PON1 activity and MDA level between factory workers and the control group.

	Factory workers group (n=36)	Control group (n=20)	t	P
Serum Catalase (U/ml)	10.3± 2.45	16.45 ± 5.07	5.11	0.000*
MDA (nmol/ml)	5.6± 0.6	2.2± 0.4	24.8	0.000*
Paraoxonase (U/ml)	23.3± 7.7	39.7± 14.4	-4.7	0.000*

*Significant difference (p<0.05)

Table (8): Comparison of serum levels of CAT, PON1 activity and MDA level in the factory workers group before and after treatment with selenium and vitamin E.

	Factory workers group before treatment (n=36)	Factory workers group after treatment (n=36)	t	p
Serum Catalase	10.3± 2.45	15.21± 4.1	6.17	0.000*
MDA	5.6± 0.6	3.2± 0.3	-19.4	0.000*
Paraoxonase	23.3± 7.7	44.6± 70.4	-1.8	0.080

*Significant difference (p<0.05)

Table (9): Results of the General Health Questionnaire (GHQ).

GHQ results	Factory workers group before treatment (n =36)		Control group (n=20)		X^2	P
	n.	%	n.	%		
Anxiety-insomnia	16	44.44%	4	11.11%	9.97	0.002*
Depression	19	52.78%	7	19.44	8.67	0.003*
Social dysfunction	15	41.67%	8	22.22%	3.13	0.08
Somatic symptoms	8	22.22%	5	13.89%	0.08	0.8

*Significant difference (p<0.05) X^2 : chi square test

Table (10): Comparison between control group and factory workers before treatment as regards cognitive tests.

	Factory workers before treatment (n. =36)	**Control group (n. =36)**	**t**	**P**
Information *(Long-term memory)*	12±3.95	12.94±4.57	1.27	0.2
Digit span (Short-term memory)	7.33±3.09	9.14±3.56	3.75	0.001*
Digit symbol *(Perceptual ability)*	12.19±5.50	14.19±6.83	2.37	0.02*
Bender Gestalt (Short-term visual memory)	17.44±2.34	16±1.66	3.64	0.001*
Trail Making (A)	50.22±12.65	54.86±15.79	2.78	0.009*

*Significant difference (p<0.05)

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دور السيلينيام وفيتامين هـ فى حالة التعرض المهنى للمعادن الثقيلة (الزئبق، الرصاص والكادميوم) : أثر العمل فى مصنع لمبات

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المعادن الثقيلة هى ملوثات بيئية تسبب تأثيرات صحية طويلة المدى، ولسوء الحظ أن هناك قصور فى تنفيذ الإجراءات الواقية مما يؤدى إلى تأثيرات سلبية خطيرة على القطاع المتعرض. الهدف من هذا البحث هو تحديد ما إذا كان تناول المكملات الغذائية مثل السيلينيام وفيتامين هـ قد يعالج التأثيرات الضارة نتيجة للتعرض المتزامن لكل من الرصاص والزئبق والكادميوم، وقد تم عمل الدراسة على ٣٦ عامل بمصنع لمبات، تتراوح أعمارهم مابين ١٩ إلى ٦٣ عام (متوسط ٢٩٫٥ ± ١٢٫١٠) : ٢٠ ذكور و١٦ إناث، تراوحت مدة عملهم بمصنع لمبات مابين ستة أشهر إلى أربعين عام (متوسط = ٥٫٥ ± ٨٫٨). المجموعة الضابطة كانت عشرون شخصاً متطابقين معهم فى العمر والنوع والعادات والظروف المحيطة، جميع العمال خضعوا للتقييم النفسى العصبى، وقد أسفرت الأسئلة الصحية العامة ٢٨ عن وجود ٤٤٫٤٪ يعانون من قلق و٥٢٫٧٪ يعانون من إكتئاب و٤١٫٦ من خلل فى التكيف الاجتماعى و٢٢٫٢٪ من أعراض جسدية، وقد بينت الاختبارات المعرفية على عـدم تأثر الذاكرة البعيدة بالمقارنة بالمجموعة الضابطة ولكن تأثرت الذاكرة القريبة والقدرة على الإدراك الحسى، كذلك تم قياس مستويات المعادن بالدم بجهاز (EDXRF) وقد بينت نتائج التحليل أن متوسط تركيز كل من الزئبق والرصاص والكادميوم قبل العلاج كانت مرتفعة إرتفاعاً ذو دلالة إحصائية، وقد انخفضت هذه المستويات إنخفاضاً ذو دلالة إحصائية بعد تناول كل من السيلينيام وفيتامين هـ. أما بالنسبة لإنزيمات مضادات الأكسدة وتتضمن باروكسينيز وكتاليز ومالوندالدهايد فقد تم قياسها قبل وبعد العلاج وذلك بعد مقارنتها قبل العلاج بالمجموعة الضابطة وقد

وجد أن هناك تحسن فى النشاط المضاد للأكسدة، وعلى ضوء هذه النتائج نتبين أن تناول كل من السيلينيام وفيتامين هـ منفردين أو مجتمعين

يساعد على خفض مستويات كل من الزئبق والرصاص والكادميام فى الدم وتحسين النشاط المضاد للأكسدة الذى من الممكن أن يعالج

الاضطرابات النفسية العصبية الحادثة نتيجة التعرض المزمن للمعادن الثقيلة المذكورة.