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Health hazards among workers in plastic industry

Sawsan Farouk Helal and Wessam Sabry Elshafy

Abstract

Styrene is a basic building block for manufacturing thousands of products throughout the world.

The present study aimed to (1) detect the presence of styrene and/or its metabolites in the workers in one of the Egyptian plastic factories; (2) demonstrate some common health effects of styrene exposure among the same group by some laboratory investigations and compare them with the unexposed healthy individuals; and (3) correlate the duration of styrene exposure and its level in the blood with the severity of the demonstrated health effects.

This study was conducted in one of Egyptian plastic factories. The exposed group was 40 male workers, ranging in age from 18 to 33 years (23.20 ± 4.09), working 12 h/day with 1 day off, and working without any protective equipment. A control group of 50 unexposed healthy males matched with the exposed group for age (21–35 yrs (23.40 ± 4.05)), sex, socioeconomic status, and smoking habit is selected. Written individual consent is obtained from all participants followed by (a) a full medical and occupational history and full clinical examination; (b) ventilatory function tests: forced vital capacity (FVC), slow vital capacity, forced expiratory volume in the 1st second (FEV_1)%, FEV_1/FVC %, peak expiratory flow, and mid-expiratory flow 25–75%; (c) analyses of β_2 microglobulin; blood styrene level; and urinary mandelic acid; and (d) cytogenetic study.

The study results showed a statistically significant difference between the exposed and the control groups as regard the blood styrene level, urinary mandelic acid level, β_2 microglobulin in urine, and chromosomal study. The study also showed a statistically significant correlation between the duration of styrene exposure and ventilatory function parameters, also between the duration of styrene exposure and some detectable chromosomal aberrations.

Our study recommends the implementation of preemployment and periodic medical examinations and health education programs using personal protective equipments and following the recommended allowable concentrations of styrene exposure.

Keywords

Chromosomal aberration, styrene, health hazards, plastic, kidney affection, ventilatory affection

Introduction

Styrene is a clear, colourless liquid that is derived from petroleum and natural gas by-products but also occurs naturally. Styrene helps to manufacture plastic materials used in thousands of remarkably strong, flexible and lightweight products that represent a vital part of our health and well-being.

Most people are exposed to styrene in tiny amounts that may be present in the air or that occur in food (styrene is a natural component of foods such as cinnamon, beef, coffee beans, peanuts, wheat, oats, strawberries, and peaches) generally as trace amounts, whose detection was difficult until the

recent technological advances. We recognize styrene by its distinctive odour, when using certain products containing styrene such as polyester resin solutions (www.styrene.org, 2011). In humans, absorption of styrene mainly occurs through inhalation, and more

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than 90% of the inhaled styrene is retained in the body (Lauwerys, 1999).

At low doses, styrene may cause irritation in the respiratory tract, and at high concentrations ($>100 \text{ mg/m}^3$) cause chronic bronchitis and obstructive pulmonary changes. Irritation of skin and eyes, elevated urinary excretion of albumin, evaluation of the potential adverse effects of styrene on the liver led to human and animal studies. Electroencephalographic, dopaminergic, functional, and psychiatric impairments have been noted (most effects have been seen at concentrations of about 100 ppm) and decreased nerve conduction velocities have been observed (IARC, 1994).

Based on classic cytogenetic effects in human biomarkers, the possibility that occupational exposure to styrene causes genotoxicity has been the focus of many biomonitoring studies (Stefano et al., 1996).

In humans, styrene metabolism involves oxidation by cytochrome P450 monooxygenases to form styrene-7,8-oxide, an epoxide thought to be responsible for the genotoxic effects of styrene exposure (Teixeira et al., 2007).

Aim of the work

The present study aimed to (1) detect the presence of styrene and/or its metabolites in workers in an Egyptian plastic factory; (2) demonstrate some common health effects of styrene exposure among the same group by some laboratory investigations and compare the results with unexposed healthy individuals; (3) correlate the duration of styrene exposure and its blood level with the severity of the demonstrated health effects.

Subjects and methods

This study was conducted in one of Egyptian plastic factories in El Oboor City. The study group consisted of 40 workers, ranging in age between 18 and 33 years (23.20 ± 4.09), working on the basis of 12 h/day with 1 day off, and working without any protective equipment during their working hours.

The control group consisted of 50 unexposed healthy males matched with the study group for age (which ranged from 21 to 35 yrs (23.40 ± 4.05)), sex, socioeconomic status, and smoking habits; they were selected randomly from the employers in the administration section of the factory, which was away from the exposure zone.

All participants in the study were volunteers; a written individual consent was obtained from every participant in the study either the exposed or the control group. These consents were reviewed by the ethical committee of Industrial Medicine and Occupational Diseases Department, Faculty of Medicine, Cairo University. The present study started after the approval of the ethical committee on the study protocol.

Methodology

(1) Full medical and occupational history, duration of styrene exposure, and history of other exposures outside the working area were recorded; followed by full clinical examination to detect any systemic affection. (2) Ventilatory pulmonary function tests were performed using a portable spirometer to measure forced vital capacity (FVC), slow vital capacity (SVC), forced expiratory volume in the 1st second ($\text{FEV}_1\%$), $\text{FEV}_1/\text{FVC}\%$, peak expiratory flow (PEF), and mid-expiratory flow 25–75% ($\text{MEF}_{25-75}\%$). (3) Laboratory investigations included (a) analysis of urine β_2 microglobulin levels. Sample urine, volume ranged between 1 and 10 ml, was taken in plastic nonacidic urine container. Participants were instructed to empty their bladders, drink a large amount of water, and then collect their urine samples in the containers during the work day. The samples were kept in the refrigerator then transferred to the laboratory. As a significant loss of β_2 microglobulin activity may occur in acidic urine ($\text{pH} \leq 6$), the pH of urine was adjusted to be between 6 and 8 with 1 mol/L sodium hydroxide. Analysis of β_2 microglobulin in urine was carried out using immunochemiluminometric assay. The normal range is 0–160 $\mu\text{g/L}$ (Henne et al., 1997). (b) Urine mandelic acid was analyzed using gas–liquid chromatography. (c) Measuring the styrene level in the blood and (d) cytogenetic analysis of lymphocytes using tissue culture and slide preparation from peripheral blood (karyotyping): Lymphocyte cultures were set up using a modified techniques of Moorhead and his colleagues as mentioned below:

(a) Blood sampling

From each subject, 5 ml of venous blood was obtained under strict aseptic measures in a heparinized syringe and mixed gently to prevent clotting. Culturing was performed in a sterilized

laminar air flow to avoid contamination of the samples and ensure successful cultures.

(b) Culture technique

- (1) Two culture tubes were used for each subject. Each tube contained:
 - 4 ml RPMI 1640 medium.
 - 1 ml foetal bovine serum.
 - L-Glutamine.
 - 0.02 ml penicillin streptomycin.
 - 0.3 ml phytohaemagglutinin.
- (2) 0.5 ml of blood was added to each culture tube then the contents were gently mixed by shaking for a few seconds.
- (3) The tubes were incubated at 37°C for 3 days in a slanting position. This position created more surface area between the liquid and the gaseous phase and allowed the cells to settle over a larger area of the culture tube.

(c) Harvesting

Lymphocytes were harvested on the third day.

(1) Colchicine treatment

- At the end of the incubation, 0.2 ml of colchicine was added to each of the culture tubes and mixed by gentle shaking again incubated at 37°C for 30 min.
- The addition of colchicine arrests cell division at the metaphase stage and the chromosomes at this stage are considered most suitable for the study.
- The culture tubes were centrifuged at 1000 r/min for 10–15 min.
- The supernatant was discarded using Pasteur pipette, leaving as little medium as possible over the cell button (pellet). The cells were then resuspended by gentle shaking.

(2) Hypotonic treatment

- 5 ml of hypotonic KCl solution (0.04) were added drop by drop from the side of the tube and shaken gently. The hypotonic solution was prewarmed to 37°C before use.
- The use of hypotonic solution causes the cells to swell, disperses the chromosomes, and makes them easier to identify and count.
- The cell suspension was incubated at 37°C for 30 min.

- The tubes were rotated in the centrifuge for 10 min.
- The supernatant was discarded.

(3) Fixation

- 5 ml of freshly prepared fixative (1 part glacial acetic acid + 3 parts absolute methyl alcohol) were added drop by drop from the side of the tube while mixing gently for few minutes. The addition of fixative helps in cell fixation.
- The tubes were left in the refrigerator for at least 1 h.
- The tubes were rotated in the centrifuge for 10–15 min.
- The supernatant was discarded.
- The steps were repeated 4 times.
- After the final centrifugation, the cells were suspended in 0.05 ml of fixative.

(d) Slide preparation:

- (1) Using a Pasteur pipette, 2–3 drops of the cell suspension were allowed to fall from a height of 20–30 cm on a 45° tilted cold slide. This procedure enabled the rupturing of the swollen cells and spreading the chromosomes.
- (2) The slides were blown gently to help spreading the drops.
- (3) The slides were left to dry on the hot plate at 40°C for 30–45s.

(e) Staining

(1) Preparation of Giemsa stock solution:

- 1 g of Giemsa powder was added to 66 ml of glycerol and left for 1.5–2 h in a water bath at 60°C with continuous shaking.
- After it cooled down, 66 ml of methyl alcohol were added. Then the mixture was filtered and stored in a dark bottle for at least 1 week before use.

(2) Slide staining (Giemsa staining):

- Preparation of 4% Giemsa solution: To each 1 ml of Giemsa stock solution, 1 ml of phosphate buffer (pH 6.8) and 23 ml distilled water were added to obtain the 4% Giemsa solution.
- Staining: The 4% Giemsa solution was poured in a coplin jar and 2–3 slides from each tube were left in the stain for 5 min. The slides were then washed in tap water and left to dry, in the vertical position, before examination.

Table 1. Comparison between the exposed and control groups as regard some laboratory investigations

| The study group | Exposed group, N: 40 | | Control group, N: 50 | | t test | p value |
|--|----------------------|--------|----------------------|-------|--------|---------|
| | Mean | ±SD | Mean | ±SD | | |
| Investigations | | | | | | |
| Blood styrene level (0–0.22 µg/L) | 1117 | ±64.52 | 0.24 | ±0.15 | 122.5 | <0.001 |
| Mandelic acid in urine (1.25–10 µmol/L) | 246 | ±21.60 | 4.20 | ±1.21 | 79.04 | <0.001 |
| Creatinine (0.7–1.2 mg/dl) | 1.02 | ±0.12 | 0.8 | ±6.1 | 6.6 | <0.05 |
| β ₂ microglobulin in urine (0–160 µg/L) | 145.9 | ±11.7 | 52.9 | ±18.4 | 18.2 | <0.001 |

Table 2. Comparison between the exposed and control groups as regard some ventilator pulmonary function parameters

| The study group | Exposed group, N: 40 | | Control group, N: 50 | | t test | p value |
|-------------------------------------|----------------------|-------|----------------------|-------|--------|---------|
| | Mean | ±SD | Mean | ±SD | | |
| Investigations | | | | | | |
| FVC% of the predicted | 77.20 | ±6.75 | 86.10 | ±4.3 | 5.07 | <0.001 |
| SVC% of the predicted | 80.20 | ±6.74 | 89.75 | ±3.8 | 5.6 | <0.001 |
| FEV ₁ % of the predicted | 76.91 | ±6.5 | 87.40 | ±3.67 | 6.3 | <0.001 |
| FEV ₁ /FVC | 74.16 | ±7.4 | 82.75 | ±2.3 | 4.9 | <0.001 |
| PEF% of the predicted | 69.62 | ±7.9 | 80.20 | ±3.1 | 5.5 | <0.001 |
| MEF 25–75% of the predicted | 61.1 | ±8.3 | 75.5 | ±4.6 | 6.6 | <0.001 |

FEV₁: forced expiratory volume in the 1st second; FVC: forced vital capacity; MEF: mid-expiratory flow; PEF: peak expiratory flow; SVC: slow vital capacity.

- Examination: For various structural and numerical aberrations, using the low-power objective, the slides were screened for well-spread metaphases. If the culture was found to be successful with a satisfactory mitotic index, 30 metaphases were scored for chromosomes.

Strict aseptic measures were followed while withdrawing the blood samples, and setting up the cultures should be performed inside a thoroughly sterilized laminar air flow, and sterile gloves were used in order to avoid infection of the cultures (Moorhead et al., 1991).

Abnormal structural chromosomal study was detected in the form of chromatid gap. It is a chromatid discontinuity, the width of which is less than the chromatid width (Connor and Ferguson, 1984); chromatid break occurs when there is a complete separation of the two segments of a chromatid leaving a distance between the two fragments greater than the width of the same chromatid (Connor and Ferguson, 1984). If the gap or break involves both the chromatids of a chromosome the lesion is named an iso-gap (chromosome gap) or an iso-break (chromosome break), respectively (Borganokar, 1989); deletion is

the loss of chromosome segment. It may be terminal or interstitial (Borganokar, 1989); unbalanced rearrangement involving the phenotype in this type of rearrangement is likely to be abnormal because of deletion, duplication, or both (Thompson et al., 1991); and balanced rearrangements do not usually have a phenotypic effect because all the genetic information are present even if it is packaged differently, but their carriers are likely to produce a high frequency of unbalanced offsprings (Borganokar, 1989).

Statistical analysis. The present study is a case–control study; the data were analyzed using SPSS statistical programmed version 16. Paired *t* test was used to compare the quantitative variables and Pearson correlation coefficient test was used to correlate them. All differences and correlations were considered statistically significant at *p* value ≤ 0.05.

Results

Table 1 shows a statistical significant difference between the exposed and the control groups as regard the styrene level in the blood, urinary mandelic acid level, serum creatinine, and β₂ microglobulin in urine.

Table 2 shows a statistically significant difference between the exposed and the control groups as regard

Table 3. Chromosomal study among the exposed and the control groups

| Examined groups | Exposed group, N: 40 | | Control group, N: 50 | | t test | p value |
|--------------------------|----------------------|--------|----------------------|--------|--------|---------|
| | Mean | ±SD | Mean | ±SD | | |
| Investigations | | | | | | |
| Gap | 8.29 | ± 1.68 | 1.05 | ± 0.82 | 17.5 | <0.001 |
| Iso-gap | 4.20 | ± 0.93 | 0.90 | ± 0.64 | 13.44 | <0.001 |
| Break | 1.91 | ± 0.71 | 0.65 | ± 0.48 | 6.69 | <0.001 |
| Iso-break | 1.95 | ± 0.62 | 0.50 | ± 0.60 | 7.81 | <0.001 |
| Centromere separation | 1.6 | ± 0.88 | 0.80 | ± 0.67 | 3.05 | <0.001 |
| Deletion | 1.75 | ± 1.07 | 0.75 | ± 0.71 | 3.55 | <0.001 |
| Unbalanced rearrangement | 0.20 | ± 0.50 | 0.00 | ± 0.00 | 1.82 | >0.05 |
| Total aberrations | 19.91 | ± 3.3 | 4.7 | ± 2.8 | 17.9 | <0.001 |

Table 4. Correlation coefficient between the duration of styrene exposure among the exposed group and ventilator pulmonary function parameters

| Parameters | R | F | p value |
|-----------------------|--------|-------|---------|
| FVC% | -0.608 | 0.002 | <0.05 |
| SVC% | -0.603 | 0.002 | <0.05 |
| FEV ₁ % | -0.655 | 0.001 | <0.05 |
| FEV ₁ /FVC | -0.671 | 0.000 | <0.001 |
| PEF% | -0.642 | 0.001 | <0.05 |
| MEF 25–75% | -0.764 | 0.000 | <0.001 |

FEV₁: forced expiratory volume in the 1st second; FVC: forced vital capacity; MEF: mid-expiratory flow; PEF: peak expiratory flow; SVC: slow vital capacity.

Table 5. Correlation coefficient between the duration of styrene exposure among the exposed group and the detectable chromosomal aberrations

| Parameters | R | F | p value |
|--------------------------|-------|-------|---------|
| Gap | 0.601 | 0.002 | <0.05 |
| Iso-gap | 0.485 | 0.01 | <0.05 |
| Break | 0.216 | 0.31 | >0.05 |
| Iso-break | 0.23 | 0.26 | >0.05 |
| Central separation | 0.192 | 0.63 | >0.05 |
| Deletion | 0.205 | 0.33 | >0.05 |
| Unbalanced rearrangement | 0.42 | 0.04 | <0.05 |
| Total aberrations | 0.559 | 0.005 | <0.05 |

different ventilatory pulmonary function parameters with obstructive pulmonary impairment.

Table 3 shows a statistically significant difference between the exposed and the control groups as regard some parameters of the chromosomal study such as gap, iso-gap, break, iso-break, centromere separation, deletion, and total aberrations.

Table 4 demonstrates a statistically significant negative correlation coefficient between duration of

Table 6. Correlation coefficient between the blood styrene level in the exposed group and some measurable parameters

| Parameters | R | F | p value |
|---------------------------------------|--------|-------|---------|
| FEV ₁ /FVC% | -0.488 | 0.002 | <0.05 |
| MEF 25–75% | -0.764 | 0.00 | <0.001 |
| Mandelic acid in urine | 0.910 | 0.00 | <0.001 |
| β ₂ Microglobulin in urine | 0.867 | 0.00 | <0.001 |

FEV₁: forced expiratory volume in the 1st second; FVC: forced vital capacity; MEF: mid-expiratory flow.

exposure to styrene among the exposed group and ventilatory pulmonary function parameters.

Table 5 shows a statistically significant positive correlation coefficient between duration of styrene exposure among the exposed group and some detectable chromosomal aberrations such as gap, iso-gap, unbalanced rearrangement, and total aberration. In contrast, the correlations between the duration of styrene exposure with the rest of detectable chromosomal aberrations such as break, iso-break, central separation, and deletion were statistically nonsignificant.

Table 6 shows a statistically significant positive correlation coefficient between the duration of styrene exposure among the exposed group with the level of mandelic acid and β₂ microglobulin in urine. On the other hand, the table shows statistically significant negative correlation coefficient between the duration of styrene exposure and the parameters (FEV₁/FVC%, MEF 25–75%) ventilator functions.

Discussion

Styrene is one of the most important industrial intermediates consumed in the world. Human exposure to styrene occurs mainly in the reinforced plastics



Figure 1. Chromosomal study shows break.



Figure 3. Chromosomal study shows gap.

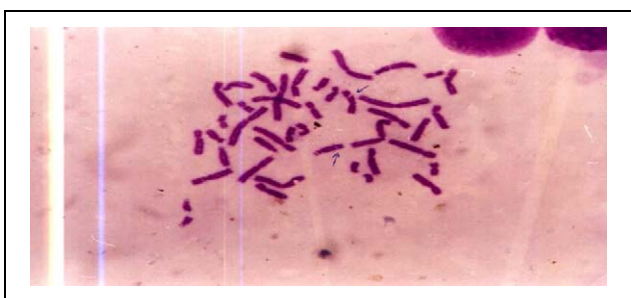


Figure 2. Chromosomal study shows iso-break.

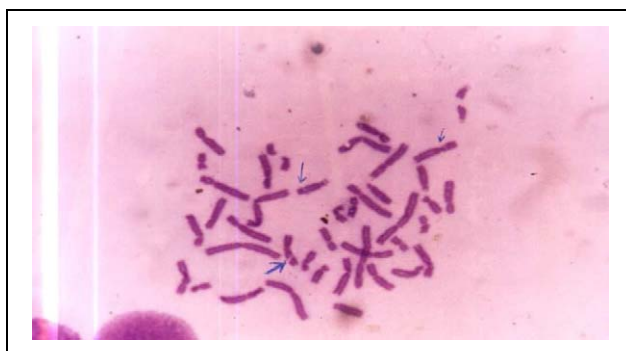


Figure 4. Chromosomal study shows iso-gap.

industry, particularly in developing countries (Yuan et al., 2010).

The present study aimed to detect the presence of styrene and/or its metabolites in the studied groups and correlates the level of detected styrene with some health effects related to styrene exposure. Such correlations are underestimated and underinvestigated among workers in the Egyptian plastic factories.

The study proved that the styrene level in blood and its metabolites (mandelic acid in urine, as biomarkers of exposure to styrene) were detected, with statistically significant difference between exposed workers and control group (Table 1).

Supporting the previous results, some laboratory findings (as measurement of blood creatinine and β_2 microglobulin in urine which are known as biomarkers of styrene effect on the kidney) were detected and showed statistically significant difference between the exposed and control groups (Table 1). Rueff et al. (2009) mentioned the same findings due to styrene exposure in the manufacture of plastics and synthetic rubber. On the other hand, Karami and his colleagues studied another effect of styrene on the kidney; the results indicate a possible association between occupational styrene exposure and renal cell carcinoma (RCC) risk. Additional studies are needed to replicate findings, as this is the first time these associations have been reported, which could be by chance (Karami et al., 2011).

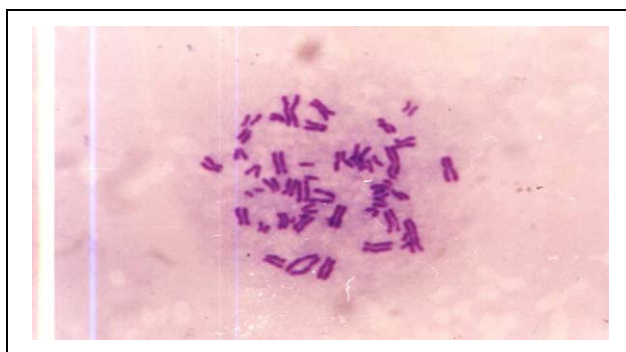


Figure 5. Chromosomal study shows centromere separation.

One of the most common health effects due to styrene exposure among the exposed group is ventilatory effects or pneumotoxic effects (Yuan et al., 2010) which was demonstrated in the present study as obstructive ventilatory pulmonary function impairment with statistical significant difference between the exposed workers and control group (Table 2). These findings agree with Checkoway and Cullen (1998) who mentioned that styrene exposure leads to chronic bronchitis and obstructive pulmonary changes.

On the other hand, Oner et al. (2004) and Robins et al. (1990) demonstrated another type of obstructive

impairment in the form of occupational asthma due to styrene exposure.

Mogel and his colleagues explained the effect of styrene in the lung cells by the expression of cyclooxygenase-2 (COX-2) which is the rate-limiting enzyme in the prostaglandin pathway which increased markedly following styrene exposure. In addition, prostaglandin E_2 and prostaglandin $F_{2\alpha}$, which are the major products of the COX enzyme, were found to be upregulated with induction of inflammatory reactions in lung cells in response to styrene exposure (Mögel et al., 2011). These findings can explain our study results because inflammation especially the chronic inflammation due to prolonged repeated exposure leads to obstructive pulmonary diseases and/or bronchial asthma.

Correlation of previous ventilatory findings with the duration of styrene exposure (Table 4) demonstrated statistically significant negative correlations (i.e. increasing the duration of styrene exposure among the exposed workers associated with decreasing the ventilatory pulmonary function parameters). This correlation is supporting the findings of Mogel and also the results of the study as the prolonged duration of styrene exposure leads to chronic repeated inflammation associated with increased severity of the obstructive impairment and the liability to chronic obstructive pulmonary diseases.

The present study agrees with Nestmann et al. (2005) and Wongvijitsuk et al. (2011) who mentioned the presence of different types of chromosomal aberrations among plastic workers due to styrene exposure with statistically significant difference in comparison with the control group (Table 3).

Huff and Infante in a previous study mentioned that styrene is genotoxic and form DNA adducts in humans, as proved in the present study. In addition, long-term chemical carcinogenesis bioassays showed that styrene caused lung cancers in several strains of mice and mammary cancer in rats. Subsequent epidemiologic studies found styrene workers had increased mortality or incidences of lymphohaematopoietic cancers (leukaemia or lymphoma or all), with suggestive evidence for pancreatic and esophageal tumours (Huff and Infante, 2011).

In addition, the present study confirmed the previous results of Fracasso and his colleagues when they studied the genotoxic effect of styrene exposure in 34 workers employed in the production of fiberglass-reinforced plastic sheets and compared with 29 unexposed healthy controls. They found that the exposed workers showed significantly high levels

of DNA strand breaks ($p < 0.0001$) and a drastic decrease in DNA repair activity as compared with the controls (Fracasso et al., 2009), as shown in Figure 1).

Correlation of the previous chromosomal aberration findings with the duration of styrene exposure revealed statistically significant positive correlations with some types of aberrations (Table 5) (i.e. increasing the duration of styrene exposure associated with the increase in the number of detectable chromosomal aberrations), as shown in figures 2-5. On the other hand, Wongvijitsuk and his colleagues in a previous study among reinforced-fiberglass plastics workers proved that the effects of styrene exposure at levels below the recommended standards of the threshold limit value (TLV-TWA (8)) of 20 ppm with variable exposure times in reinforced-fiberglass plastics workers associated with DNA damage such as DNA strand breaks of exposed groups were significantly higher compared to the control group ($p < 0.05$) (Wongvijitsuk et al., 2011).

The study also revealed statistically significant positive correlations between the styrene level in the blood of the exposed workers (the level of mandelic acid and β_2 microglobulin in urine) (Table 6), and these findings are matched with that of Wongvijitsuk et al. (2011) who demonstrated significant increase in the styrene urinary metabolites, mandelic acid, and phenylglyoxylic acid with an increase in the styrene level in blood and styrene exposure. Also the deteriorations regarding the severity of the obstructive impairment as shown in some parameters of ventilatory pulmonary functions ($FEV_1/FVC\%$ and $MEF_{25-75\%}$) increased significantly with an increase in the styrene level in blood. So keeping the styrene level in blood within the recommended standards of the TLV will be helpful in decreasing the severity of its health hazards on the exposed workers.

Recommendations. Preemployment and periodic medical examinations should be performed to exclude any susceptibility to styrene exposure and permit a strict monitoring of the workers. Workers were supposed to use the proper personal protective equipments during the working hours in the factory. The occupational culture among the workers was improved by conducting health education programs about the health hazards of styrene exposure and by explaining the benefits of the personal protective equipments and how to use them perfectly. Proper and regular measurement of environmental styrene level to keep it within the recommended allowable concentrations. Additional

studies regarding styrene exposure in the Egyptian plastic factories are recommended to demonstrate the associations between the effect of styrene exposure on the chromosomes with some genetic and carcinogenic diseases as RCC.

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