Occupational risks in families at a battery factory in Jordan

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ABSTRACT

The effect of lead exposure on rate of spontaneous abortion of workers at a battery factory was studied. The study comprised 25 male workers in a lead-smelter and controls of 24 male university employees without any known occupational exposure to toxic agent. The most interesting outcome of the present study has been the discovery of high incidence of spontaneous abortion in lead-exposed workers compared to that of controls. Comparative evaluation between smokers and non-smokers with respect to the incidence of spontaneous abortion does not exhibit any statistically significant variations in lead-exposed workers and controls as well. However, such findings are considered to be indicative of the possible existence of a genetic risk and sufficient for justifying further work on a wider scale of risk assessment and occupational hazard in Jordan.

Key Words: Spontaneous abortions, Lead.

المخاطر المهنية عند عائلات عمال مصنع البطاريات في الأردن

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الملخص

يهدف هذا البحث إلى دراسة معدل الإجهاض التلقائي عند العمال الذين يتعرضون للتلوث بالرصاص. وقد تمت دراسة هذا المعدل على 25 فرداً تعرضوا لهذا التلوث. وفي مجموعة الضبط تمت دراسة 24 فرداً سليماً لم يتعرضوا من قبل إلى أي عامل ملوث معروف. وتشير النتائج إلى وجود نسبة عالية من الإجهاض التلقائي عند زوجات العمال الذين تعرضوا للرصاص إذا ما تمت مقارنتها مع تلك التابعة لمجموعة الشاهد. كذلك لم تظهر الدراسة وجود فروقاً ذات أهمية إحصائية في معدل الإجهاض التلقائي بين المدخنين وغير المدخنين في كل من مجموعة العمال ومجموعة الشاهد مما يشير إلى أن عامل التدخين ليس فعالاً في هذه الدراسة وأن الرصاص قد يكون العامل الفعال في إحداث نسبة عالية من الإجهاض التلقائي عند زوجات العمال الذين تعرضوا للرصاص. تشير هذه النتائج إلى احتمال وجود ضرر وراثي من التعرض للرصاص هذا الضرر يعد مبرراً كافياً لإجراء دراسات تفصيلية لمعرفة حجم الضرر الناشيء من التعرض للرصاص في الأردن.

الكلمات المفتاحية: الإجهاد التلقائي، الرصاص.

Introduction

Lead is one of the most widely spread contaminants that accumulates in the environment (Godish, 1986). Occupational and environmental exposure to this element has attracted interest in recent years due to its deleterious health effects on reproduction and development (Apostoli *et al*., 2000; Bakali *et al*., 1995; Beach and Watt; 2003; Chowdhury *et al*., 1999; Czeizel and Mosonyi, 1997; Griebel *et al.*, 2005; Joffe *et al.*, 2003; Lanphear, 1998; McMurry *et al.*, 1995; Mecheva and Milanov, 1993; Montesanti *et al.*, 1995. Ryan *et al*., 1999; Sallmen *et al*., 1992; Vill *et al.*, 1993; WHO, 1989).

The hazard to health stimulated investigations of mutagenic activity of lead in the human organism (Högstedt *et al.*, 1979; Lin *et al.*, 1998; Nordenson *et al.*, 1982; Omari and Al-Mahasneh, 1997). The possible involvement of lead as gametotoxic (Apostoli *et al.*, 1998; Bauchinger *et al.*, 1976; Lancranjan *et al.*, 1975; Shiau *et al.*, 2004; Sokol *et al.*, 1994; Vaglenov *et al.*, 2001) and embryotoxic (Beckman and Nordström, 1982; Nordström *et al.*, 1979a) has been reported. Moreover, a tendency towards an increased rate of spontaneous abortion (mean number of spontaneous abortions per family) (Baghurst *et al.*, 1991; 'Czeizel and Mosonyi, 1997; Geronimus and Hillemeier, 1992; Lin *et al*., 1998; Murphy *et al*., 2001; Nordström *et al.*, 1979b) and congenital malformations (Nordstrom *et al*., 1979b; Sallmen *et al*., 1992) in pregnancies where the mothers had worked at the smelter of lead has been reported. In this work, we undertake exploratory study to present data on the occupational risks in families where their husbands are workers in a lead-smelter of a battery factory in Jordan.

Materials and Methods

Biomonitoring study was conducted in the years 1997 and 1998, investigating the occupational risks in a group of 25 male workers (age range 25-62 years) occupationally exposed to lead at a smelting plant who were occupied for 3-35 years. During their work lead may be inhaled as dust or fumes. A reference group (control) of 24 male university employees without any known occupational exposure to toxic agent was used. Their age varied between 34 and 60 years. The blood lead level of the lead-exposed workers ranged from 37 to 190 microgram Pb/dl blood, while that of the control group was 1 to 4 microgram Pb/dl blood. All of the workers completed an interview-administered questionnaire which included an occupational history, smoking status, age, history of medication use, drug and alcohol use.

Nobody was previously irradiated or treated with clastostatic drugs, and had suffered no viral or bacterial infections for two months before the study. The questionnaire was extended to their wives. All wives were healthy at the time of abortions. The questionnaires were filled out by each family under our supervision. The data obtained were statistically analyzed using t-test and the chi-square test.

Results

Table (1) and (2) show individual data on the parental age, period of exposure and preganancies outcome in the families of the lead-exposed workers and control males, respectively.

Table 1. Data on families whose fathers are workers at a lead battery factory.

			Pregnancies outcomes		
Subject number	Parental Age (years)	Period of exposure (years)	No. of live births	No. of spont. abortions	No. of pregnancies
Smokers					
1	62	35	10	0	10
6	31	7	4	0	4
9	52	10	6	1	7
10	33	14	5	0	5
11	43	6	5	2	7
13	29	5	2	0	2
15	54	18	6	0	6
19	50	18	6	4	10
22	55	28	10	0	10
23	26	7	2	2	4
24	26	7	0	0	0
25	26	5	0	2	2
26	29	6	4	1	5
27	50	7	8	1	9
28	34	3	0	0	0
Total	600	176	68	13	81
Mean	40.00 ± 3.38*	11.73 ± 2.41	4.53 ± 0.86	0.87 ± 0.29	5.40 ± 0.79
Non-Smokers					
5	58	26	8	0	8
7	50	28	6	4	10
8	50	13	0	0	0
12	45	9	3	0	3
16	35	5	8	1	9
21	30	6	5	0	5
29	32	10	4	3	7
30	35	3	2	1	3
31	25	1	5	0	5
32	34	7	3	0	3
Total	419	108	44	9	53
mean	41.90 ± 3.38	10.80 ± 2.91	4.48 ± 0.81	0.90 ± 0.45	5.30 ± 1.01
Combined Mean (S+NS)	40.76	11.76	4.48	0.88 ± 0.22	5.36 ± 0.66

*= standard error; S = smokers; NS = non-smokers

Table 2. Data on families whose fathers are workers at Jordan University (Controls).

Subject Number	Parental Age (years)	Period of exposure (years)	Pregnancies outcomes: No. of live births	No. of spont. abortions	No. of pregnancies
Smokers					
1	42	25	6	0	6
2	35	12	4	2	6
3	49	25	5	0	5
4	41	15	6	2	8
5	36	13	5	0	5
6	35	8	6	0	6
7	45	24	3	0	3
8	40	16	2	0	2
9	37	14	3	1	4
10	39	14	2	0	2
11	38	15	2	0	2
12	52	20	3	0	3
Total	489	201	47	5	52
Mean	40.75 ± 1.59*	16.75 ± 1.59	3.92 ± 0.47	0.42 ± 0.23	4.33 ± 0.60
Non-Smokers					
1	30	12	2	0	2
2	60	21	4	0	4
3	34	10	4	0	4
4	37	15	3	0	3
5	40	16	2	1	3
6	36	11	3	0	3
7	39	15	2	0	2
8	41	18	3	0	3
9	35	12	2	0	2
10	39	14	2	0	2
11	42	18	3	0	3
12	44	14	2	0	2
Total	482	176	32	1	33
mean	40.17 ± 2.01	14.67 ± 0.72	2.67 ± 0.23	0.08 ± 0.24	2.75 ± 0.22
Combined Mean (S+NS)	40.45	15.76	3.29	0.25 ± 0.12	3.54 ± 0.33

- * = standard error; S = smokers; NS = non-smokers.

The two groups, workers and control, of males are shown to be comparable in their mean parental age (Workers: 40.76; Control: 40.45 years). The mean number of spontaneous abortion (Smokers: 0.87±0.29; Non-smokers: 090 ± 0.45; Combined: 0.88 ± 0.22) (Table1) and the percentage of this abortion (Smokers: 16.04, Non-smokers: 16.98; Combined: 16.42) (Table 4) were higher in the occupationally exposed workers to lead as compared to that of control (Mean = Smokers: 0.42±0.23, Non-smokers: 0.08± 0.24, Combined:

0.25 ± 0.12, Table 2; Percentage = Smokers 9.61, Non-smokers 3.03, Combined: 7.06, Table 4).

When the corresponding data were compared between smokers or non-smokers of both groups, the occupationally exposed workers of non-smokers showed significantly higher mean number of pregenancies than the control group ($t = 2.6879$, $P < 0.05$) (Table 3). Similar finding is also observed in the combined mean ($t = 2.4364$, $P<0.05$) (Table3). Cigarette smoking appeared to have no such influence on this mean ($t = 1.0319$, $P > 0.05$) (Table 3).

Table 3. Statistical analysis of the means.

Means	Comparison	t	P
I. Smokers			
Number of pregnancies	W vs. E	1.0310	> 0.05
Number of spont. Abortions	W vs.E	1.1317	> 0.05
II. Non-smokers			
Number of pregnancies	W vs.E	2.6879	> 0.05
Number of spont. abortions	W vs.E	1.6858	> 0.05
III. Combined (S + NS)			
Number of pregnancies	W vs. E	2.4364	< 0.05
Number of spont. abortions	W vs.E	2.4600	< 0.05

W = lead-exposed workers; E = employees (Control); S = smokers; NS = non-smokers; t = t – valve; P = probability.

The difference between occupationally exposed workers and controls may also be described on the basis of the rate of spontaneous abortions (mean number of spontaneous abortion per family). The workers has significantly higher rate of spontaneous abortion (0.88 ± 0.22) (Table 1) as compared to that of controls (0.25 ± 0.12) (Table 2) when the corresponding combined mean was compared between the two groups ($t = 2.4600$, $P < 0.05$) (Table 3).

Table 4. Proportions of spontaneous abortions in lead-exposed workers

and controls.

Comparison		Spontaneous abortion Percentage	No. of pregnancies
I.	Lead-exposed workers		
	Smokers	16.04	81
	Non-smokers	16.98	53
II.	University employees (Controls)		
	Smokers	9.61	52
	Non-smokers	3.03	33
III.	Combined % (S+NS)		
	Battery workers	16.42	134
	University employees	7.06	85

S = smokers; NS = non-smokers.

This difference could also be illustrated when the percentage of spontaneous abortion to the total preganancies (Combined%) was compared between the two groups, workers and controls (Combined%: Workers = 16.42; Controls = 7.06) (Table 4). The Chi-square value between them is 5.9199, 1 df, $0.01 < P < 0.02$ (Table 5). Cigarette smoking appeared to have no such influence on the percentage of spontaneous abortions since comparison between smokers and non-smokers of each group revealed insignificant differences (Workers: x^2- value = 0.0248, 1 df, $0.80 < P < 0.90$; Control: x^2-value = 1.3345, 1 df, $0.20 < P < 0.30$) (Table 5).

Table 5. Statistical analysis of the proportions of spontaneous abortions.

		Comparison	Chi-square	df	P-value
I.	Workers	S vs. NS	0.0248	1	0.80<P<0.90
II.	Employees (Controls)	S vs. NS	1.3345	1	0.20<P<0.30
III.	Combined % (S + NS)	W vs. E	5.9199	1	0.01<P<0.02

S = smokers; NS = non-smokers; W = workers; E = employees; P = probability

Discussion

The genetic risk posed by the action of environmental exposure to heavy metals on the DNA of living cells becomes a question of increasing importance (Lanphear, 1998; Ryan *et al.*, 1999; Sakai, 1995; WHO, 1989; Zarocosta, 2005). The toxicity of lead, as one of these heavy metals, has long been recognized (Huseman *et al.*, 1992; Walden, 1973). The importance of this element as an environmental contaminant has attracted increasing interest in the last two decades as a major concern of public health authorities (Apostoli *et al.*, 200; Czeizel and Mosonyi, 1997; Joffe *et al.*, 2003; Lin *et al.*, 1998; National Research Council, 1980; Schwartz and Otto, 1991; shiau *et al.*, 2004; Sokol *et al.*, 1994; WHO, 1977, 1989) and as modern industry requires this easily workable metal (Bender, 1980; Bender et. al. 1974; Gerber *et al.*, 1980; Hemminki *et al.*, 1979; Murphy *et al.*, 2001; Rosen, 1982; Skreb and Habazin-Novak, 1975; Stover and Leob, 1976).

In the pooled data (Smokers and non-smokers)(Tables 1 and 2), comparative evaluation of the rate of spontaneous abortion between lead-exposed workers and controls exhibits significant differences (Combined rate, Smokers and Non-smokers: Workers vs. Employees, $t = 2.46$, $P < 0.05$) (Table 3). Such differences suggest a possible association between rate of spontaneous abortion and lead-exposed workers which could be explained on the bases that toxic agents emitted from the lead smelter might, somehow, affect the germ cells of these lead-workers, a condition which has been reported in several studies (Beckman *et.al.*, 1982; Lancranjan *et al.*, 1975; Lin *et al.*, 1996; McMurry *et al.*, 1995; Nordstrom *et al.*, 1978, 1979a, 1979b; Wyoobeck and Bruce, 1978).

Although cigarette smoking represents an indirect exposure by possible contamination with lead on cigarette from the workers' hands or work surfaces, no possible association between smoking, lead exposure and rate of spontaneous abortion has been reported (Smokers: Workers vs. Employees, $t = 1.1317$, $P > 0.05$) (Table 3). Similar finding is also observed in non-smokers group (Non-smokers: Workers vs Employees, $t = 1.6858$, $P > 0.05$) (Table 3). These findings revealed that smoking has no impact on the rate of spontaneous abortion. Such finding has been previously confirmed by Beckman and Nordström (1982).

When the percentage of spontaneous abortion to the total pregenanceis (Combined%) was compared between lead-exposed workers (16.42%) and control (7.06%) (Table 4), the Chi-square value between them is 5.9199, 1 df, $0.01 < P < 0.02$ (Table 5). Such results demonstrate marked differences in the incidence of spontaneous abortion between the workers and controls. This finding is in good agreement with the earlier finding of other workers (Anttila and Sallmen, 1995; Beckman and Nordström; 1982; Brundage, 2002). In this connection, cigarette smoking appeared to have no such effect on the incidence of spontaneous abortion since comparison between smokers and non-smokers in workers as well as in controls revealed insignificant differences (Workers: x^2 – value = 0.0248, 1 df, $0.80 < P < 0.90$; Control: x^2 – value = 1.3345, 1 df, $0.20 < P < 0.30$) (Table 5).

Based on the findings of the present study, the most interesting outcome has been the discovery of high incidence of spontaneous abortion in lead-exposed workers as compared to that of controls. However, the material of the present study is too small to allow a meaningful statistical analysis. Therefore, it may be of significance that all these important observations reported in this study are considered to be indicative of the possible existence of a genetic risk and sufficient for justifying further work on a wider scale of risk assessment of occupational hazard in Jordan.

REFERENCES

Anttila, A., and Sallmen, M. 1995. Effects of parental occupational exposure to lead and other metals on spontaneous abortion. Occup Environ Med., 37(8): 915-921.

Apostoli, P., kiss, P., and Porru, S. 1998. Male reproductive toxicity of lead in animals and human. ASCLEPIOS study Group. Occup Environ Med., 55: 364-374.

Apostoli, P., Bellini, A., and Porru, S. 2000. The effect of lead on male fertility: a time to pregnancy (T TP) study. Am J Ind Med., 38: 310-315.

Baghurst, P. A., Robertson, E. F., Oldfield, R. K., King, B. M., McMichael, A. J., Vimpani, G. V., and Wigg, N. R. 1991. Lead in the placenta, membranes and umbilical cord in relation to pregnancy outcome in a lead-smelter community. Environ. Health Perspect., 90: 315-320.

Bakali, R. I., Pesti, G. M., Ragland, G. M., Konjufca, W. L., and Novak, R. 1995. Delta-aminolevulinic acid dehydratase: a sensitive indicator of lead exposure in broiler chick (*Gallus olomisticus*). Bull. Environ. Contam. Toxicol., 55(6): 833-839.

Bauchinger, M., Schmid, E., Einbrodt, H. J., and Dresp, J. 1976. Chromosome aberrations in lymphocytes after occupational exposure to lead and cadmium. Mutation Res., 40: 57-62.

Beach, J., and Watt, D. 2003. General practioners and occupational health professionals. BMJ, 327: 302-303.

Beckman, L., Nordenson, I., and Nordström, S. 1982. Occupational and environmental risks in and around a smelter in northern Sweden. Hereditas, 96: 261-264.

Beckman, L., and Nordström, S. 1982. Occupational and environmental risks in and around a smelter in northern Sweden: IX. Fetal mortality among wives of smelter workers. Hereditas, 97: 1-7.

Bender, M. A. 1980. Relatinship of DNA lesions and their repair to chromosome aberration production. In Generoso W. M., Shelby M. D., de Serres, F. J. (Eds): "DNA Repair and Mutagenesis in Eukaryotes" Plenum, NY. pp. 245-265.

Bender, M. A., Griggs, H. G., and Bedford, J. S. 1974. Mechanisms of chromosome aberration production. III. Chemicals and Ionizing Radiation. Mutation Res., 23: 197-212.

Brundage, S.C. 2002. Preconception health care. Am Fam Physician, 65(12): 2521-2522.

Chowdhury, B. A., and Chandra, R. K. 1999. Biological and health implication of toxic heavy metal and essential trace element interaction. J. progress In Food And Nutrition, 11:57-113.

Czeizel, A. E., and Mosonyi, A. 1997. Monitoring of early human fetal development in women exposed to large doses of chemicals. Environ. Mol. Mutagen, 30(2): 240-244.

Gerber, G. B., Leonard, A., and Jacquet, P. 1980. Toxicity mutagenicity of lead. Mutation Res., 76: 115-141.

Geronimus, A. T., and Hillemeier, M. M. 1992. Patterns of blood lead levels in VS black and white women of childbearing age. Ethn. Dis., 2(3): 222-231.

Godish, T. 1986. Air Quality. 2nd ed. Lewis Publishers, Inc., Michigan.

Griebel, C. P., Halvorsen, J., Golemon, T. B., and Day. A. A. 2005. Management of spontaneous abortion. Am Fam physician, 72: 1243-1250.

Hemminki, K., Sorsa, M., and Vaino, H. 1979. Genetic risks caused by occupational chemicals. Scand. J. Work Environ. Health, 5:307-327.

Hogstedt, B., Koling, A. M., Mitelman, F., and Schutz, A. 1979. Correlation between blood-lead and chromosomal aberration. Lancet, 2: 262.

Huseman, C. A., Varma, M. M., and Angle, C. R. 1992. Neuroendocrine effects of toxic and low blood lead levels in children. Pediatrics, 90(6): 1001.

Joffe, M., Bisanti, L., and Apostoli, P. 2003. Time to pregnancy and occupational lead exposure. Occup environ Med., 60: 752-758.

Lancranjan, T., Popescu, H., Gavanescu, O., Klepsch, J., and Serbanescu, M. 1975. Reproductive ability of workmen occupationally exposed to lead. Arch. Environ. Health, 30:396-401.

Lanphear, B.P. 1998. The paradox of Lead Poisoning Prevention Science, 281:1617-1618.

Lin, S., Hwang, S. A., Marshall, E. G., and Marion, D. 1998. Does paternal occupational lead exposure increase the risks of low birth weight or prematurity? Am. J. Epidemiol., 148 (2): 173-181.

Lin, S., Hwang, S. A., Marsahall, E. G., Stone, R., and Chen, J. 1996. Fertility rates among lead workers and professional bus drivers: a comparative study. Ann. Epidemiol., 6(3): 201-208.

McMurry, S. T., Lochmiller, R. L., Chandra, S. A., and Qualls, C. W. 1995. Sensitivity of selected immunological, hematological and reproductive parameters in the cotton rat (*Sigmodon htsptdus*) to subchronic lead exposure. J. Wildi. Dis., 31(2): 193-204.

Mecheva, R., and Milanov, Z. 1993. Heavy metals and trace elements in agrocoenoses of different levels of pollution. Nauka za Gorata, 30(2): 89-95.

Montesanti, M., Morgantini, F., Landucci, C., Biagi, C., and Castelli, S. 1995. Assessment of lead blood level in children living in Lucca (Italy) in 1993. Minerva-Pediatr, 47(4): 119-125.

Murphy, M. J.; Graziano, J. H., Popovac, D., Kline, J. K., Mehmeti, A., Factor-litvak, P., Ahmedi, G., Shrout, P., Rajovic, B., and Nemezic, D. V. 2001. Post pregnancy outcomes among women living in the vicinity of a lead smelter in Kosovo, Yugoslavia. Am. J. Public Health, 80(1): 33-35.

National Research Council. 1980. Lead in the human environment. Washington, D.C. National Academy of Science.

Nordström, S., Beckman, L., and Nordenson, I. 1978. Occupational and environmental risks in and around a smelter in northern Sweden. III. Frequencies of spontaneous abortion. Hereditas, 88: 51-54.

Nordström, S., Beckman, L., and Nordenson, I. 1978. Occupational and environmental risks in and around a smelter in northern Sweden. VI. Conginital malformations. Hereditas, 90:297-302.

Nordström, S., Beckman, L., and Nordenson, 1. 1979b. Occupational and environmental risks in and around a smelter in northern Sweden. V Spontaneous abortion among female employees and decreased birth weight in their offspring. Hereditas, 90: 291-296.

Nordenson, I., Nordström, S., Sweins, A., and Beckman, L. 1982. Chromosome aberrations in lead-exposed workers. Hereditas, 96: 265-268.

Omari, Y. I., and Al-Mahasneh, Q. M. 1997. Chromosome aberrations in lead-exposed workers. Dirasat, 27 (1): 92-98.

Rosen, J. F. 1982. Testimony presented at the EPA hearing on lead in gasoline. Washington, D. C. April 16th, 1982.

Ryan, D. and others. 1999. "Protecting children from Land poisoning and building healthy communities. Am. J. Public Health., 89(11): 1690-1695.

Sakai, T. 1995. Reviews on biochemical markers of lead exposure with special emphasis on heme and nucleotide metabolism. Sangyo Eiseigaku Zasshi, 37(2): 99-112.

Sallmen, M., Lindbohm, M.L., Anttila, A., Taskinen, H., and Hemminki, K. 1992. Paternal occupational lead exposure and congenital malformations. J. Epidemiol Community Health, 46(5): 519 - 522.

Schwartz, J., and Otto, D. 1991. Lead and minor hearing impairment Arch. Environ. Health, 46(5): 300-305.

Shiau, C. Y., Wang, J. D., and Chen, P. C. 2004. Decreased fecundity among male lead workers. Occupational and Environmental Medicine, 61: 915-923.

Skreb, Y., Habazin-Novak, V. 1975 Reversible inhibition of DNA, RNA, and protein synthesis in human cells by lead chloride. Toxicology, 5: 167-174.

Sokol, R. Z., Okuda, H., and Nagler, H. M. 1994. Lead exposure in vivo alters the fertility potential of sperm in vitro. Toxicol Appl Pharmacol., 124: 310-316.

Stover, H. A., and Loeb, L. A. 1976. Infidelity of DNA synthesis in vitro screening for potential metal mutagens and carcinogens. Science, 194: 1434-1436.

Vaglenov, A., Creus, A., Laltchev, S., Petkova, V., Pavlova, S. and Marcos, R. 2001. Occupational exposure to lead and induction of genetic damage. Environ Health Perspect., 109: 295-298.

Villa, R. B., Paez, O. F., and Perez, M. H. 1993. Heavy metal concentrations in heart, liver, and kidney tissues in the vaquita phocoena sinus (Mammalia: Phoconidae). Ser. ie Zoologia, 64(1): 61-71.

Walden, H. A. 1973. Lead poisoning in the ancient world. Med. Hist., 17: 391- 399.

World Health Organization. 1989. Lead-Environmental Aspects. WHO Publication Center, Albany, New York.

Wyoobeck, A. J., and Bruce, W. R. 1978. The induction of sperm cell abnormalities in mice and animals. In A. Hoilaender (Ed.): Chemical Mutagens, Vol. 5, Plenum, New York, pp. 257-285.

Zarocosta, G. J. 2005. International Labour Organization tackles work related injuries. BMJ, 331: 656-658.