



Effect of Noise Pollution on the Hormonal and Semen Analysis Parameters in Industrial Workers of Bushehr, Iran

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Abstract

Objective: One of the concerns of health officials is noise pollution and in the realm of health, the problems of sterility and infertility resulting from noise pollution greatly attract the interest of experts nowadays. Noise is one of the harmful environmental factors and one of the most cacophonous of the unavoidable phenomena at home and workplace. Considering Bushehr is one of the cities with high infertility rates, we decided to study labor and industrial environments.

Materials and Methods: Two groups of men volunteer workers, 27 members in each, who were constantly exposed to noisy 107- or 119-decibel environments, were studied together with one group of 27 workers living in quiet environments serving as the control. These people were referred to the Omid Khalij Fars Infertility Center in Bushehr where blood samples were taken and tested for adrenocorticotrophic hormone (ACTH), cortisol, testosterone, prolactin, follicle-stimulating hormone (FSH), luteinizing hormone (LH), and thyroid hormones T3, T4, and Thyroid-stimulating hormone (TSH) and semen samples were taken and sent to the specialized laboratory of the Center.

Results: Statistical studies showed that noise stress in the 119-decibel group significantly reduced the concentrations of the testosterone, prolactin, LH, and FSH hormones and of the thyroid hormones T3, T4, and TSH, and significantly increased the concentrations of the ACTH and cortisol hormones, compared to the control group. Moreover, semen analysis indicated major changes in semen parameters, especially under 119-decibel.

Conclusion: Noise causes changes in hormones involved in the physiological process of fertility and in semen analysis parameters and, hence, has harmful effects on fertility.

Keywords: Hormones, Noise pollution, Semen analysis

Introduction

Noise and vibrations are now one of the major problems of world industry and large numbers of people at workplace or at home are at risk of being harmed by it. Living a machinery life has caused humans to tolerate an uncomfortable coexistence with noise and vibration sources in stressful environments (1). On one hand, large numbers of employees have to confront these two physical factors at workplace and on the other hand, presence of noise and vibrations signifies low levels of technology and undesirable performance or depreciated machinery and equipment (2). Defective or malfunctioning machinery and equipment waste an important part of the input energy through generating noise and vibrations, and it is necessary, both economically and healthwise, to control these two factors (3). Various types of pollution including noise, sound waves produced by cell phones, air pollution and any pollution that causes cells to vibrate, will definitely have negative effect on the formation of the early embryo. Statistics show that in 2011 and 2012 a high percentage of

abortions occurred in women who were exposed more to air and noise pollution. Therefore, it is certain that pollution affects both genders equally and simultaneously (4). Noise stress can also influence male sexual hormones and cause changes in reproductive glands and organs (5). Prolonged exposure to 100-decibel noise has permanent effects on testicular histology and morphology, and changes serum levels of testosterone. Long-term changes in testosterone levels cause structural changes in testicles, stop maturation of germ cells, increase the number of dead and agglutinated sperms and can lead to infertility (6). The negative effect of stress occurs at different levels of the hypothalamic-pituitary-gonadal (HPG) axis. Moreover, stress activates the hypothalamic-pituitary-adrenal (HPA) axis that, in turn, suppresses the HPG axis through inhibiting the secretion of the gonadotropin-releasing hormone (GnRH) (7). With the suppression of GnRH, stress influences fertility through inhibiting secretion of the luteinizing hormone (LH) from the pituitary gland and by suppressing sexual behavior. It was observed in

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researches that there was a relationship between noise pollution at workplace and the thyroid gland diseases of hypothyroidism and hyperthyroidism (8,9). The neural activity required to process environmental noise increases the number of free radicals that are known to be agents of carcinogenic mutations (10). Workers regularly exposed to high levels of noise exhibit symptoms such as nausea, headache, aggression, temperamental changes and anxiety (11).

Therefore, noise pollution can be accompanied by important consequences. That is why this research studied the side effects of noise pollution on fertility power (spermatogenesis) and sperm quality and of hormonal changes on hormones that influence fertility such as thyroid hormones, stress hormones (cortisol and adrenocorticotropic hormone [ACTH]), prolactin and testosterone in industrial workers in Bushehr.

Materials and Methods

Groups

The workers were men in the age range of 25-30 years and were carefully examined by a urologist to make sure they were normal with respect to fertility parameters. Those who had problems related to these parameters were excluded from the research. They were all recommended to refrain from sexual activities for four days before taking samples and were divided into three groups, each with 27 members:

Group I: (the control group) worked away from noise in relatively calm areas; group II: (the experimental group) were exposed to the low noise level or did not directly work with iron cutting machines but carried out regular daily work at the workshop so that they were exposed to the noise level of 107 decibels and group III: Worked directly with iron cutting machines and were exposed to the noise level of 119 decibels. Noise levels were measured using a Phillips sonometer (Model ABC 234).

Features of the Workshop Generating the Noise

In this workshop, sheet iron in various thicknesses were cut for making doors and windows and other domestic and industrial uses; as a result high levels of noise pollution were generated and workers were continuously exposed to this pollution 10 hours a day on average.

Seminal Fluid Analysis

All volunteers were referred to the Omid Khalij Fars Infertility Center and semen samples were collected via masturbation (without using any chemicals or gels) in special containers. All other stages of semen preparation and analysis were carried out at the Andrology laboratory of the Center.

In studying the seminal fluid, macroscopic parameters such as liquefaction time, semen volume, viscosity, color, smell, and sperm pH and microscopic parameters such as sperm agglutination, number of sperms per milliliter of the seminal fluid and total sperm count in the sample, sperm viability, sperm motility, morphology, sperms live

counting, etc were studied.

Sperm Morphology

After sperm motility and sperm count, sperm morphology can be considered an important factor in male fertility. Sperm morphology is evaluated qualitatively and quantitatively and includes distinguishing normal sperms from abnormal ones, etc. Abnormal morphology is often accompanied by poor sperm motility and low sperm count (12).

Sperm-Counting Method

The number of sperms per milliliter is counted with a hemocytometer neubauer. Review of studies by various researchers led to the announcement that the minimum normal number of sperms is 20 million per milliliter of the seminal fluid. This number is sufficient for fertility provided acrosome reaction and sperm morphology and motility are normal. If there are more than 250 million sperms per milliliter of the seminal fluid, the condition is called polyspermy; that is, the number of sperms is higher than normal (13,14).

Sperm Viability Test

Supravital stain can be used to distinguish live and motile sperms from dead ones. In a normal sample of seminal fluid, 75% or a higher percentage of the sperms, are alive.

Hormonal Studies

Blood samples were taken from the left arm of all the workers in the study and were poured into test tubes without adding any anticoagulant agents. After blood coagulation, the test tubes were centrifuged at 3500 rpm for 15 minutes at 25°C. The blood serum on the coagulated part was then carefully removed with a Pasteur pipette. The blood serum samples were immediately placed in a flask containing ice packs and sent to the Razi Laboratory in Bushehr for hormonal measurements.

Since plasma is needed for measuring the ACTH hormone, part of each blood sample taken was poured in a plastic tube impregnated with EDTA and these tubes were placed in a flask containing ice packs and transferred to the Biotechnology Laboratory of Bushehr University of Medical Sciences within 30 minutes. ACTH is stable for 18 hours, if it is in blood plasma inside test tubes impregnated with EDTA and kept at 4°C. A refrigerator centrifuge was used for 15 minutes at 3500 rpm to separate the plasma, and each plasma sample was poured into two plastic tubes with a Pasteur pipette and the tubes were immediately frozen at -20°C (15).

Hormonal Assessment

Hormonal measurements were carried out at the Razi Laboratory in Bushehr using a model Cobas e 411 Alexis machine and employing the electrochemiluminescence method.

Data Analysis

One-way analysis of variance (ANOVA) and Tukey/LSD

tests were used for data analysis and the data was reported as mean \pm standard deviation. After statistical analysis, the data was analyzed using SPSS 20.

Results

Hormonal Evaluation

Average Serum Levels of FSH and LH

Results of LH and FSH measurements in Table 1 show that average serum concentrations of LH in various groups were not significantly different from that of the control ($P > 0.05$). However, results concerning FSH indicate the average serum level of this hormone in the 119-decibel group decreased significantly compared to the control and the 107-decibel groups ($P \leq 0.05$). Moreover, the average serum concentration of FSH in the 107-decibel group declined compared to the control group, but the difference was not statistically significant ($P > 0.05$; Table 1).

Average Serum Levels of T4, T3, and TSH

Results of measuring serum levels of these hormones indicate that their average serum levels in the 119-decibel group increased significantly compared to the control group ($P \leq 0.05$), but they were not significantly different from those of the 107-decibel group ($P \leq 0.05$). Moreover, the average serum concentrations of these hormones in the 107-decibel group increased compared to the control group, but the differences were not statistically significant ($P > 0.05$; Table 1).

Average Serum Levels of Prolactin

Results of the prolactin measurements reveal that its average serum level in the 119-decibel group increased significantly compared to the 107-decibel and the control groups ($P \leq 0.05$). Moreover, its serum level in the 107-decibel group was higher compared to the control group, but the difference was not statistically significant ($P > 0.05$; Table 1).

Average Serum Levels of Testosterone

Results of measuring testosterone show its average serum level in the 119-decibel group decreased significantly compared to the 107-decibel and the control groups ($P \leq 0.05$). Moreover, its average serum level in the 107-decibel group declined compared to the control group, but the difference was not statistically significant ($P > 0.05$; Table 1).

Average Serum Levels of Cortisol and ACTH

Results of measuring these hormones indicate their average serum concentrations in the 119-decibel group increased significantly compared to the 107-decibel and the control groups ($P \leq 0.05$). Moreover, their average serum levels in the 107-decibel were higher compared to the control group, but the differences were not statistically significant ($P > 0.05$; Table 1).

Semen Evaluation

As previously mentioned, semen was evaluated both macroscopically and microscopically.

Macroscopic Results

As shown in Table 2 almost all macroscopic parameters such as liquefaction time, and semen volume, viscosity, color and smell were identical in all groups except for pH and no considerable differences were observed between the groups. The pH level of the seminal fluid in the 119-decibel group was a little more acidic.

Sperm Motility

As shown in Table 3, results of sperm count show the experimental groups were not significantly different from the control group with respect to sperm motility ($P > 0.05$).

- Rapid sperm motility in the 119- and 107-decibel groups decreased significantly compared to the control group ($P \leq 0.05$), but the 119- and 107-decibel groups were not significantly different from each other in this respect ($P > 0.05$).
- Low sperm motility in the 119- and 107-decibel groups increased significantly compared to the control group ($P \leq 0.05$), but these two groups did not differ significantly from each other in this respect ($P > 0.05$).
- Non-progressive sperm motility also increased in the 119- and 107-decibel groups compared to the control group ($P \leq 0.05$), but these two groups were not significantly different from each other in this respect ($P > 0.05$).

Sperm Morphology Quality

As shown in Table 3, the experimental groups were not significantly different from the control group with respect to sperm morphology quality ($P > 0.05$).

Table 1. Comparative of Mean Hormones Levels in Different Groups

Hormones	Groups		
	(I) Control	(II) 107db	(III) 119db
LH	13.55 \pm 6.01	12.23 \pm 5.84	12.11 \pm 4.12
FSH	3.45 \pm 3.19	2.12 \pm 1.08	1.31 \pm 0.13
T3	1.00 \pm 0.47	6.47 \pm 0.38	1.11 \pm 0.35
T4	9.04 \pm 1.06	6.13 \pm 29.28	5.71 \pm 8.19
TSH	4.11 \pm 2.38	2.64 \pm 4.088	2.09 \pm 3.34
Prolactin	3.56 \pm 1.60	2.89 \pm 1.1	2.01 \pm 1.01
Testosterone	30.86 \pm 5.12	22.29 \pm 01.24	19.29 \pm 03.11
Cortisol	189.94 \pm 45.42	164.20 \pm 35.10	281.18 \pm 56.18
ACTH	28.55.12 \pm 26.16	24.94 \pm 45.42	60.27 \pm 23.41

Data were analyzed with *t* test and F-test method. Values were expressed as the mean \pm standard deviation (Mean \pm SD). Significant difference with control group (*); $P < 0.05$; $n = 10$.

Table 2. Comparative of Semen Analysis Macroscopically in Different Groups

Hormones	Groups		
	(I) Control	(II) 107db	(II) 119db
Liquefaction time (min)	35.01 ± 10.11	33.12 ± 4.24	36.15 ± 4.25
Viscosity	Normal	Normal	Normal
Agglutination	None	None	None
Color	Creamy	Creamy	Creamy
pH	7.11 ± 3.18	7.94 ± 1.18	8.89 ± 4.14

Data were analyzed with *t* test and F-test method. Values were expressed as the mean ± standard deviation (Mean ± SD). Significant difference with control group (*); $P < 0.05$; $n = 10$.

Table 3. Comparative of Semen Analysis Microscopically in Different Groups

Microscopic Parameters	Groups		
	(I) Control	(II) 107db	(II) 119db
Immotile %	32.13 ± 4.11%	27.26 ± 6.46%	29.17 ± 2.36%
Rapid progressive %	36.45 ± 6.43%	31.14% ± 5.17%	26.23% ± 9.13%
Slow progressive %	26.81 ± 2.43%	34.45 ± 6.43%	38.15 ± 6.63%
None progressive %	5.21 ± 1.40%	10.41 ± 2.44%	18.15 ± 6.63%
Morphology %	8.15 ± 3.16%	7.31 ± 1.43%	6.10 ± 2.61%
Sperm counts (million/mL)	120.18 ± 4.63	105.19 ± 18.1	86.25 ± 1.09
Semen volume (mL)	4.16 ± 2.12	3.86 ± 1.54	5.13 ± 3.10

Data were analyzed with *t* test and F-test method. Values were expressed as the mean ± standard deviation (Mean ± SD). Significant difference with control group (*); $P < 0.05$; $n = 10$.

Sperm Count

Results show sperm counts were significantly lower in the 119- and 107- decibel groups compared to the control group ($P \leq 0.05$), but these two groups did not significantly differ from each other in this respect ($P > 0.05$).

Semen Volume

Table 3 shows semen volume increased a little in the 119-decibel group, but there were no statistically significant differences between the two experimental groups in this respect ($P > 0.05$) and the increase in semen volume in the 119-decibel group could be accidental.

Discussion

The hormonal cascade of the HPG axis controls the basic performance of the reproductive system and eventually, seminal fluid and sperm production. Moreover, the performance of some hormones such as thyroid hormones (16,17) and stress hormones such as cortisol and ACTH directly influence male and female fertility systems (18). Therefore, we decided to study possible changes in these hormones in workers exposed to acute acoustic stress. In addition, semen study and analysis and the probable changes in semen and in male sexual cells (sperms) are the best criterion of fertility health in men (19). Therefore, in the next step, we studied more carefully the changes resulting from workers' environment through accurate semen analysis. Consequently, results of this research are investigated and discussed from the two aspects of hormonal and semen evaluation.

Nowadays, noise has turned into one of the important threats to human health due to the industrialization of societies, increase in the number of vehicles and due to the ever-increasing expansion of industries, has adverse and destructive effects on most body organs (20). Noise pol-

lution may be one of the reasons why infertility rates have increased to 12%-15% (21).

In this research, results of ACTH, cortisol, and testosterone measurements in the various groups of industrial workers in Bushehr show that 119- and 107-decibel noise levels significantly increased concentrations of ACTH and cortisol and reduced that of testosterone compared to the control group (Table 1). Exposure of workers to the noise generated by electric saws, especially exposure to the 119-decibel noise, increased the activity of the HPA axis due to the rise in ACTH secretion during stress. Moreover, because of higher cortisol levels during stress (that results from increased secretion of ACTH), testosterone levels decline due to the activity of glucocorticoid receptors in Leydig cells and due to decreased response of these cells to LH. Reduced testosterone levels stop and disrupt spermatozoa maturation and result in germ cell atrophy. In general, we can say that chronic stress resulting from noise pollution may change the constant values of hormones and their metabolic purification.

Our study indicated serum levels of FSH and LH decreased with increases in the level of noise pollution and the conducted experiments confirm that the lower level of noise pollution caused insignificant reductions in FSH and LH levels, while the higher level of noise pollution (the 119-decibel level) significantly reduced the concentrations of these hormones.

Moreover, our experiments confirm results of previous studies that indicated thyroid hormones T4, T3, and TSH decreased at noise pollution level of 107 decibels and that further increases in noise pollution levels significantly reduced the concentrations of these hormones (22).

Low levels of testosterone production (23) have harmful effects on ejaculate quality and fertility and reduced levels

of testosterone are associated with lower sperm volumes in the epididymis (23,24). Moreover, the sperms in the epididymis of the group with chronic stress are agglutinated and the number of dead sperms increases. Therefore, changes in the natural morphology of testicles are related to germ cell maturation arrest (24,25).

As shown in Table 3, noise level of 119 decibels in our study caused considerable changes in the volume, number and type of motility of sperms in sperm analysis, increased the number of non-motile sperms, and changed sperm viscosity and sperm head shape and tail length. These results and conclusions completely agree with those of previous studies (23).

A similar study was conducted on two groups of male rats one of which was kept in an environment without noise pollution and the other exposed to 120- decibel noise with the frequency range of 300-350 Hz for 50 days. It was found that the number of embryos declined, and their growth and development faced numerous problems and semen parameters such as sperm motility, number, morphology, and viscosity exhibited very significant changes in the experimental group (26).

In our research, increasing the noise level to 119 decibels caused considerable changes in the volume, number and type of motility of sperms in semen analysis, increased the viscosity of the sperms and number of non-motile sperms and changed sperm head shape and tail length. These results are in complete agreement with those of previous studies.

Noise, as a foreign factor stimulating oxidative stress, disrupts calcium homeostasis that leads to calcium ion imbalance in the mitochondria. This causes release of the active oxygen species in the form of free radicals. The increase in the level of free radicals in the body results in utilization of antioxidants, which reduces the antioxidant capacity of blood. Researchers have proved that oxidative stress influences the male reproductive system. Excessive production of the active oxygen species, or elimination of antioxidant scavengers, that happens at high oxidant levels, changes the performance of sperm and of its supportive tissues including the epididymis. This can, in the end, lead to male infertility because DNA is particularly sensitive to hydroxyl radicals (25,27).

Conclusion

Therefore, considering the effects of noise on the active oxygen species, we can conclude from our research that high noise levels (119 decibels in our study) caused the production of the active oxygen species and free radicals that led to oxidative stress in germ cells and to disruption of the spermatogenesis process. This caused atrophy of germinal epithelial cells followed by reduction in the diameter of seminiferous tubules and reduced thickness of germinal epithelium in the tissues of the testicles, which caused widespread changes in the parameters of semen analysis. Our research showed that acoustic stress caused these changes. In all, we can say that chronic stress resulting from noise pollution may change the constant hor-

monal values and their metabolic purification and that part of this adaptive process can cause some systems, such as the reproductive system, to be sacrificed so that vital body functions can be preserved (23,28).

Ethical issues

All experimental protocols were performed under the approval from the Ethic Committee (clinical trial number is: 34/727).

Conflict of interests

None.

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References

1. Franssen EA, van Wiechen CM, Nagelkerke NJ, Lebre E. Aircraft noise around a large international airport and its impact on general health and medication use. *Occup Environ Med.* 2004;61(5):405-413. doi:10.1136/oem.2002.005488.
2. Mostaghaci M, Mirmohammadi SJ, Mehrparvar AH, Bahaloo M, Mollasadeghi A, Davari MH. Effect of workplace noise on hearing ability in tile and ceramic industry workers in Iran: a 2-year follow-up study. *Scientific World J* 2013;2013:923731. doi:10.1155/2013/923731.
3. Cavacas MA, Tavares V, Oliveira MJ, et al. Effects of industrial noise on circumpulpar dentin-a field emission scanning electron microscopy and energy dispersive spectroscopy analysis. *Int J Clin Exp Pathol.* 2013;6(12):2697-2702.
4. Nedelec SL, Radford AN, Simpson SD, Nedelec B, Lecchini D, Mills SC. Anthropogenic noise playback impairs embryonic development and increases mortality in a marine invertebrate. *Sci Rep.* 2014;4:5891.
5. Swami CG, Ramanathan R, Charan Jeganath A. noise exposure affect testicular histology and on male steroidogenic hormone. *Malays J Med Sci.* 2007; 14(2):28-35.
6. Ogale SB. Noise overload. *Health Nutrients.* 1999;85-89.
7. Rivier C, Rivest S. Effect of stress on the activity of the hypothalamic-pituitary-gonadal axis: Peripheral and central mechanisms. *Biol Reprod.* 1991;45(4):523-532. doi:10.1095/biolreprod45.4.523.
8. Gonzalez-Quijano MI, Ariznavarreta C, Martin AI, Treguerres JA, Lopez-Calderon A. Naltrexone does not reverse the inhibitory effect of chronic

- restraint on gonadotropin secretion in the intact male rat. *Neuroendocrinology*. 1991;54(5):447-453. doi:10.1159/000125933.
9. Veljović V, Jovanović J, Radević L, Radovanović Z, Gligorijević S, Blagojević L. Early detection of thyroid disease in workers professionally exposed to noise through preventive medical check-ups. *Acta Medica Medianae*. 2010;49(3):45-48.
 10. Samson J, Devi RS, Ravindran R, Senthilvelan M. Effect of noise stress. *Environ Toxicol Pharmacol*. 2005;20(1):142-8. doi:10.1016/j.etap.2004.12.059.
 11. Schmidt, Charles W. Noise that annoys: Regulating unwanted sound. *Environ Health Perspect*. 2005; 113(1):A42-A44.
 12. Rothmann SA, Bort AM, Quigley J, Pillow R. Sperm morphology classification: a rational method for schemes adopted by the world health organization. *Spermatogenesis*. 2013; 927:27-37.
 13. Oliveira JB, Massaro FC, Mauri AL, et al. Motile sperm organelle morphology examination is stricter than Tygerberg criteria. *Reprod Biomed Online*. 2009;18(3):320-326. doi:10.1016/S1472-6483(10)60088-0.
 14. Perdrix A, Rives N. Motile sperm organelle morphology examination (MSOME) and sperm head vacuoles: State of the art in 2013. *Hum Reprod Update*. 2013;19(5):527. doi:10.1093/humupd/dmt021.
 15. Evans GW, Lercher P, Meis M, Ising H, Kofler WW. Community noise exposure and stress in children. *J Acoust Soc Am*. 2001;109:1023-1027. doi:10.1121/1.1340642.
 16. Mallidis C, Sanchez V, Wistuba J, et al. Raman microspectroscopy: shining a new light on reproductive medicine. *Hum Reprod Update*. 2013;20(3):403-414. doi:10.1093/humupd/dmt055.
 17. Skorupskaitė K, George JT, Anderson RA. The kisspeptin-GnRH pathway in human reproductive health and disease. *Hum Reprod Update*. 2014;20(4):485-500. doi:10.1093/humupd/dmu009.
 18. Rodrigues MA, Wittwer D, Kitchen DM. Measuring stress responses in female Geoffroy's spider monkeys: Validation and the influence of reproductive state. *Am J Primatol*. 2015;77(9):925-935. doi:10.1002/ajp.22421.
 19. Cooper TG, Noonan E, von Eckardstein S, et al. World Health Organization reference values for human semen characteristics. *Hum Reprod Update*. 2010;16(3):231-245. doi:10.1093/humupd/dmp048.
 20. Passchier-Vermeer W, Passchier WF. Noise exposure and public health. *Environ Health Perspect*. 2000;108(Suppl 1):123-31.
 21. Nurminen T. Female noise exposure, shift work, and reproduction. *J Occup Environ Med*. 1995;37(8):945-950.
 22. Johnson HD, Vanjonack WJ. Effects of environmental and other stressors on blood hormone patterns in lactating animals. *J Dairy Sci*. 1976; 59(9):1603-1617. doi:10.3168/jds.S0022-0302(76)84413-X.
 23. Swami C, Ramanathan J, Jeganath C. Noise exposure effect on testicular histology, morphology and male steroidogenic hormone. *Malaysian J Med Sci*. 2007;14(2):28-35.
 24. Mylchreest E, Sar M, Wallace DG, Foster PM. Fetal testosterone insufficiency and abnormal proliferation of Leydig cells and gonocytes in rats exposed to di (n-butyl) phthalate. *Reprod Toxicol*. 2002;16(1):19-28. doi:10.1016/S0890-6238(01)00201-5.
 25. Beardsel A, O'Donnell L. Characterization of normal spermiation and spermiation failure induced by hormone suppression in adult rats. *Biol Reprod*. 2003;68(4):1299-1307. doi:10.1095/biolreprod.102.009811.
 26. Chia SE, Ong CN, Lee ST, Tsakok FH. Study of the effects of occupation and industry on sperm quality. *Ann Acad Med Singapore*. 1994;23(5):645-649.
 27. Aitken RJ, Baker MA, Sawyer D. Oxidative stress in the male germ line and its role in the aetiology of male infertility and genetic disease. *Reprod Biomed Online*. 2003;7(1):65-70. doi:10.1016/S1472-6483(10)61730-0.
 28. Negro-Vilar AN. Stress and other environmental factors affecting fertility in men and women: overview. *Environ Health Perspect*. 1993;101(Suppl 2):59-64.

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