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

Alireza Chamkori, Mehrdad Shariati, Darab Moshtaghi, Parviz Farzadinia

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 adrenocorticotropic 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 testes, 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
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 refrigerate 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 ± 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 ($P > 0.05$). 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 average serum level in the 119-decibel group increased significantly compared to the control group ($P \leq 0.05$). Moreover, the average serum level of this hormone in the 107-decibel group increased 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 concentration 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).

**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, 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

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

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.
Chamkori et al
Crescent Journal of Medical and Biological Sciences, Vol. 3, No. 2, April 2016

26.23
(II) 107db
Groups
10.41 ± 2.44%
31.14
(II) 107db
5.13 ± 3.10
34.45 ± 6.43%
-
-
35.01 ± 10.11
Normal
-
Creamy
5.21 ± 1.40%
Creamy
Normal
29.17 ± 2.36
None
18.15 ± 6.63%
Creamy
7.31 ± 1.43%
7.11 ± 3.18
38.15 ± 6.63%
26.81 ± 2.43%
6.10 ± 2.61%
3.86 ± 1.54
None
-
86.25 ± 1.09
105.19 ± 18.1
-
(II) 119db
Groups
33.12 ± 4.24
36.15 ± 4.25
-
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8.15 ± 3.16%
None
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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 hormonal 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: 34/727).

Conflict of interests

None.

Financial support

This project was done in the Animal House of Bushehr University of Medical Sciences, Bushehr, Iran and fully financially supported by Azad University, Kazeroon branch.

Acknowledgments

This research was conducted at the Infertility Center of Omid Khalaji Fars, Bushehr, Iran and was supported by Islamic Azad University, Kazeroon Branch.

References
8. Gonzalez-Quijano MI, Ariznavarreta C, Martin AI, Treguerres JA, Lopez-Calderon A. Naltrexone does not reverse the inhibitory effect of chronic
doi:10.1159/000125933.

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