REVIEW ARTICLE
IMPACT OF OCCUPATIONAL HEAT EXPOSURE ON MALE REPRODUCTIVE HEALTH

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Humans are exposed to several exogenous chemicals used in industrial processes, development activities and also through food chain. The male reproductive system is vulnerable to the effects of the physical and chemical factors. This is a review of literature to determine the influence of occupations involving high temperature on the male reproductive health. We concluded that occupational heat exposure will alter spermatogenesis and also affect sperm morphology, therefore it is overall a risk factor for male infertility. A few studies have shown that the occupational heat exposure to male partner can cause low birth weight, preterm delivery, and spontaneous abortion which may be due to genetic mutations in human sperm. However, more prospective studies are needed to ascertain this fact.

Keywords: Occupational heat exposure, Male infertility, Semen analysis

INTRODUCTION

Reproduction disorders and occupational hazards to reproductive health have become prominent issues in recent decades after reports to adverse effects of certain chemicals on reproductive function. Occupational hazards are by far the best documented in reproductive epidemiological research. Commonly occupational exposures have been divided into physical exposures (heat and radiation), chemical exposures (solvents and pesticides), psychological exposures (distress) and exposure to metals and welding. Several occupational hazards to male reproductive function are known but exposure prevalence is hardly sufficient to play a role for reduced sperm count in the general male population. Sedentary work may be an exception. Perhaps prolonged time in the sedentary position exhausts the testicular heat regulation. But so far studies addressing implications of the heat hypothesis in the general population are few. Neither change of sexual behavior nor reduced period of sexual continence seems to be a likely explanation. Temperature plays an important role in the spermatogenesis of human beings. Therefore, nature has kept the scrotum outside the body so that the temperature of the testes may be lower than that of the body temperature. This article reviews the impact of occupation involving high temperature on the reproductive health.

Temperature influences the development of germ cells and the reproductive cycle of human. In human beings, generation of male germ cells depends generally on the comparative low temperature condition in scrotum. The spermatogenesis of scrotum also depends on the relatively low temperature condition in scrotum. Temperature higher than the scrotal temperature (e.g., abdominal temperature) would block the process of spermatogenesis. Therefore, the scrotal mammals were often chosen as the models to study the influences of temperature on the structures and functions of spermatogenic or other cells in testis. In general, spermatogenic and other cells of testis were sensitive to heat, and if testis is submitted even to the so called “normal body temperature” (such as 37°C in humans) for a long time, not only spermatogenesis will be blocked, but even Leydig and Sertoli cells will be damaged to a large extent. Histological studies have showed that heat would differentially hurt male germ cells in different developmental stages during spermatogenesis, especially the pachytene primary spermatocytes.

The first report of occupational heat exposure was in 1775 when an English physician Percival Pott documented a high occurrence of scrotal cancer in chimney sweepers. The observation made in this study lead to the implementation of safety regulations in the form of bathing requirements for these workers.

McLeod and Hotchkiss noted the detrimental effect of elevated temperature on sperm production. They mentioned that induced hyperpyrexia in 6 healthy men, in a high-temperature cabinet caused a dramatic drop in sperm count after 3 weeks, with a mean duration of 50 days. Also, the role of frequent hot baths and hot tubs has consequently been described as possible causes of infertility.

Sas and Szollosi established that 291 of the 2984 patients seeking advice for infertility in Szeged (Hungary) were professional drivers. These professional drivers had a high incidence of the severe sperm anomalies (oligoasthenozoospermia and azoospermia), which was correlated with the number of years of driving and with the use of agricultural and industrial heavy machinery with elevated levels of vibration.

In another case control study of data from medical records and dispatched questionnaires from 1977 to 1980 Rachootin et al found out that there is an association between infertility and occupational
exposure to heat (with heat exposure being deduced from the job description).12

Figa-Talamanca et al have demonstrated the effect of chronic occupational exposure to high temperature on sperm production in the ceramic industry. They have pointed out a higher prevalence of pathologic sperm profiles in the exposed subjects when compared to healthy subjects.13 In another study Bonde et al studied a group of welders exposed to high temperatures but well-protected against inhalation of toxic substances. These authors found reversible but significant modifications in the sperm morphology after 6 weeks of elevated heat exposure. No other semen parameters were altered, and no hormonal imbalances were detected.14

In Rome Figa-Talamanca et al studied 72 taxi drivers and 50 control subjects (of similar age and smoking habits). They compared salivary testosterone concentrations, sperm quality (concentration, morphology and motility), and time taken to obtain conception. The taxi drivers had a significantly lower occurrence of normal sperm forms (45.8% compared with 64.0%) than the control group. This observation was more pronounced in those who had been working as drivers for a long time. A considerable association between smoking and poor sperm morphology was also observed.15

The causal relationship between paternal employment in the ceramic industry and an increased risk of low birth weight and preterm delivery was suggested by a national Scottish study.16 In support of these results, Kline et al elucidated an association between increased heat exposure of the male partner and more frequent spontaneous abortions.17 In contrast, Lindbohm et al did not discover any association in increased fetal loss in wives of men with a mild exposure to elevated heat.18 Thonneau et al found an association between male occupational heat exposure and the time required to achieve a pregnancy in a retrospective survey of 402 fertile couples. This study recommended that occupational exposure to heat is a ‘weak’ risk factor for male subfertility.19

Zorgniotti and McLeod have reported an improvement in sperm changes in men wearing a cooling device inducing chronic hypothermia of the testis.20

Carlsen et al have demonstrated a significant adverse effect of reported fever on sperm concentration, sperm morphology and motility, and indicated that the stages during spermatogenesis most vulnerable to fever with respect to sperm concentration are the meiotic phase and the postmeiotic phase (spermiogenesis) from early primary spermatocytes to early spermatids. Sperm morphology and motility are most susceptible to fever during the postmeiotic phase.21

### Table 1: Review of studies on effects of occupational heat exposure on male reproductive health

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<td>Macleod et al15</td>
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<td>Figa-Talamanca et al20</td>
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<tr>
<td>Thonneau et al21</td>
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Fever can cause transient oligospermia and a reduction of serum Luteinizing hormone (LH) as well as serum testosterone levels, possibly by increasing corticotropin-releasing hormone (CRH), resulting in suppression of GnRH pulsatility and, consequently, a reduced LH release.22-24 There are also reports that sperm densities decrease in the summer months in men who work outdoors, supporting the theory that environmental heat may damage spermatogenesis or possibly the function of epididymis.25

### CONCLUSION

In conclusion, this review suggests that exposure to high temperatures has a harmful effect on male reproductive functions and it must be considered as an important risk factor for male infertility. The altered spermatogenesis is seen in men who are continually exposed to high temperatures and perhaps also in men with impaired arteriovenous testicular systems as there may be chronic thermo-dysregulation. Moreover lower birth weight, preterm delivery, and spontaneous abortion rates can be related directly to elevated heat exposure of the male partner, which could be due to genetic mutations in sperm. A full reproductive toxicity study is warranted to confirm on how the occupational heat exposure to male partner can affect the offspring.
REFERENCES


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