

Guide to
Men's
Reproductive Health
in the Mining
Workplace

A Laurentian University & Workplace Safety North Initiative Sandra C. Dorman, PhD & Céline Larivière PhD

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Introduction

There is accumulating evidence that some workplace environments expose workers to hazards that potentially cause reproductive health problems, many of which are preventable. In the mining industry workers are exposed to a host of potential hazards and some of those hazards may impact upon their reproductive or sexual health. Specific concerns tend to vary by age, with younger workers generally focused on fertility issues that may impact upon their ability to start a family and older workers being more concerned about sexual health. Regardless of age or presenting concern, miners experiencing fertility problems or other sexual health issues may benefit from a formal hazard assessment of their job to determine if occupational exposures are contributing to or causing their reproductive health problem.

What is a reproductive hazard?

A hazard is an agent that has the potential to cause harm to a person. A 'reproductive' hazard is an agent that can adversely affect the reproductive health of women and men and/or negatively impact the growth and development of a fetus. Examples of reproductive problems linked to hazardous agents include: reduced fertility, by harming sperm or ova; miscarriage, if an embryo is damaged (toxic agents can be transferred by sperm); or disruption of hormonal pathways involved in reproduction and sexual function (e.g. testosterone decline). Hazardous agents are present in both non-occupational and occupational settings.

Objectives of this Guide

This Guide has two primary objectives. The first objective is to provide information about the health effects of workplace hazards in the mining industry on male reproductive health. The second objective is to provide information about safety measures and controls that may help inform and therefore prevent or minimize exposure to reproductive hazards. However, it should be noted that it is the employer's responsibility to identify and implement controls for exposures. In many cases, simple measures including minor restructuring of the work environment or work schedule and use of personal protective equipment can ensure the safety of the worker and his future offspring. Effective prevention measures for a given worker vary depending on individual circumstances; therefore, all decisions should be made on a case-by-case basis. The information contained in this Guide can be used to help inform the decision making process.

This Guide serves as an information resource for human resource managers, occupational therapists, industrial hygienists, employers, supervisors and workers. The Guide is intended to be used in conjunction with other resources such as confidential interviews with trained health and safety professionals including nurses and physicians. The material contained in this Guide is for information and reference purposes only and not intended as legal or professional advice. The adoption of practices described in this Guide may not meet the needs, requirements or obligations of individual workplaces.

This Guide has not been designed to address occupational safety matters related to conception or female reproductive biology. See the companion piece: Guide to Healthy Pregnancies in the Mining Workplace (http://www.crosh.ca/publications.html).

How to use this Guide

In this Guide, hazards in the mining workplace are listed under three major subheadings: physical agents (noise, vibration, heat, radiation); chemical agents (gases, dusts, mists, vapors, fumes) and other factors (ergonomic factors, scheduling and workplace stress). Hazardous agents are identified in the first column. Examples of where the agent is encountered are listed in the second column. In the third column, the risk to the worker and, when indicated, the developing fetus, is described. The fourth column provides some examples of safety measures and controls that may help to prevent or minimize the worker's exposure to the agent. When available, recommended exposure limits as defined by the American Conference of Governmental Hygienists (ACGIH) are listed, including the agents "STEL" (short-term exposure limit) and "TWA" (time-weighted average limit), as per Ontario Regulation 490/09 and 833 and have not been proven to specifically protect against reproductive health hazards.

The hazardous agents listed in this Guide have been selected because they are the most commonly encountered reproductive hazards in the mining workplace; however, it is important to note that the list of agents in the Guide is not all-inclusive. For example, biological agents that affect reproductive health such as viruses, fungi and mould are not included because, while these hazards may exist in mining, exposure is not thought to be increased in this workplace compared to other industries or non-occupational settings.

There remains scientific uncertainty about what regulatory agencies should do to protect the reproductive health of workers. For instance, dose-response information is lacking for the majority of reproductive hazards. This is because most of our current knowledge comes from animal studies; there are only a limited number of human studies examining occupational reproductive hazards. Given the current scientific uncertainty, most regulatory agencies recommend exposure levels be kept as low as reasonably possible. Every effort should be made by both the employer and the worker to minimize risk of exposure.

A Man's Role in Reproduction

Because the emphasis of a healthy pregnancy traditionally focuses on the woman's role, the potential for problems resulting from the father's exposure to a reproductive hazard is often overlooked or unknown. On the basis of current understanding, reproductive health problems in men are thought to occur via five main mechanisms:

- 1. Gene/chromosomal mutation of the sperm;
- 2. Hormonal changes (e.g. reduced testosterone);
- 3. Reduction in sperm numbers;
- 4. Disturbance of sperm shape or motility; and
- Erectile dysfunction.

Exposure to reproductive hazards in the workplace generally impact male reproductive function through one or more of these mechanisms ultimately resulting in infertility or unsuccessful pregnancy. Examples of such hazards include vibration, heat, chemicals (arsenic, lead), shift scheduling and occupational stress. It is important for men to have access to information about potential workplace hazards and prevention to prevent exposure and preserve their reproductive health

How are Sperm Made?

Sperm are the male reproductive cells that are transported in semen. Sperm production, known as 'spermatogenesis' occurs in the testicles during an approximate 70-day cycle. Spermatogenesis is constantly occurring over the three-month cycle. For hazardous exposures that directly affect spermatogenesis (for example, heat stress), the effect is usually temporary and reversible, with normal sperm production resuming over the 3 months following removal from exposure. Other hazardous agents cause longer-term effects on reproductive function, either because they remain in the body after removal from exposure (for example, lead) or because the agent directly damages the testicles (for example, hexavalent chromium which may cause testicular atrophy).

In addition to sperm production, the testicles also produce the hormone testosterone. Testosterone is an essential hormone that influences sperm production, reproductive tissue development, sexual drive and sexual performance. Some hazardous agents, for example lead, manganese and mercury, have been demonstrated to alter hormone levels thereby impacting reproductive function and fertility.

Disclaimer

The information contained in this material is provided as a Guide only. Workplace Safety North (WSN) and Laurentian University (LU) recognize that individual companies must develop health and safety policies and programs that apply to their workplaces and comply with appropriate legislation. This material does not constitute legal advice. While the information provided, including references to legislation and established practice, is current at the time of printing, it may become out-of-date or incomplete with the passage of time.

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About the authors

Dr. Sandra Dorman is an Associate Professor in the School of Human Kinetics at Laurentian University. Her research interests are in the area of health and wellness. She teaches courses in physiology, pathophysiology as well as community health and nutrition.

Dr. Céline Larivière is an Associate Professor in the School of Human Kinetics at Laurentian University. Her research interests are in the area of muscle cell physiology. She teaches courses in exercise injury management, physiology as well as human growth and development.

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PHYSICAL HAZARD	EXAMPLES IN THE MINING INDUSTRY	RISK: MALE WORKER AND FETUS (when applicable)	ACTION TO AVOID RISK
Extremes of Heat	Summer mine work, depth, working in any hot and/or humid environment.	 Testicular function is temperature dependent and requires a temperature 2°C to 4°C below core body temperature [1], [3] Elevated scrotal temperature is referred to as scrotal hyperthermia Scrotal hyperthermia causes abnormal spermatogenesis [1], [2], [3], [4], [5] resulting in decreased sperm count and impaired motility and/or morphology [6], [7], [8], [9], [10], [11], [12], [13], [14] which can result in adverse pregnancy outcomes [12] Spermatogenesis requires about 70 days, so the effects of heat may be noted 2-3 months after exposure [29] Sexual function: Insufficient data 	Consult NIOSH Guidelines on monitoring and controlling heat exposures in the workplace. [1], [3] Avoid prolonged sitting / encourage tasks that allow altering seated position frequently (sit in same position for < 28 minutes at a time). [15] Note: despite normal and constant core body temperatures (around 37° C), normal scrotal temperatures (34-36°C) can increase by 2.2 °C with prolonged sitting. [17] Avoid prolonged driving for more than two hours. [17] Avoid laptop usage on lap. [15],[18] Encourage task rotation / switching machines over the course of the shift to facilitate different body postures to avoid scrotal heat stress. Remove worker from the hot environment to facilitate recovery of testicular function. [3] Recovery of testicular function is noted after 4-11 weeks but can take 6 months after exposure to extreme heat ceases. [1] In general, the more prolonged the elevation in testicular temperature the greater the detrimental effect on spermatogenesis. [19], [9], [20]

PHYSICAL HAZARD	EXAMPLES IN THE MINING INDUSTRY	RISK: MALE WORKER AND FETUS (when applicable)	ACTION TO AVOID RISK
Ionizing Radiation	Radiation in mines, especially in mines where thorium or uranium is found. Radon and Radon daughters: No known effects of Radon exposure on male fertility	 Reproduction: Decreased sperm count and/or temporary inability to produce sperm [1], [3], [21], [14] In some cases, permanent sterility [22], [1], [23], [24], [8], [10], [25], [13], [11], [26], [27], [12], [3] Damage to testicular germ cells [1] Sexual function: Insufficient data Fetus: Paternal doses of ≥ 10 mSv (1 rem) in 6 months before conception or total preconception doses ≥ 100 mSv (10 rem) associated with 6-8 fold increased risk of leukemia in offspring [1], [3] 	The annual worker exposure limit for nuclear energy workers is 50 mSv (5 rem). For other workers (ex. workers performing non-destructive testing, servicing nuclear gauges or operating X-ray equipment), the annual exposure limit is 1 mSv for nuclear sources or 5 mSv from all sources. Therefore, keep exposure limits 'as low as reasonably achievable' (28), (29) by monitoring exposure time and distance from hazard. Sperm banking prior to exposure is suggested. Timeto-recovery of testicular function is dose-dependent and may vary between 6-24 months. Delay conception for 3-6 months to ensure that the fertilizing sperm was not produced from a germ cell exposed to radiation.
Noise	Vehicular machinery, power tools, compressed air	Reproduction: No direct effects of noise on male fertility are reported to date, therefore no impact on	Avoid >8 hours exposure or equivalent exposure, to noise greater than 8 dBA. [36], [37] Although direct effects of noise on fertility have not

compressed air discharge, ventilation fans.

- reported to date, therefore no impact on length of time to conception [30]
- In humans, prolonged exposure to noise >80 dBA increases levels of stress hormones^[31] which can in turn be a risk factor for infertility by negatively impacting sperm parameters^[32]

Sexual function:

- Increased stress and suppression of testosterone production supported by animal studies indicating that chronic noise exposure in mice and rats linked to decreased testosterone levels^{[34],[35]}
- Disruption in hormonal pathway may impact sexual function and sex drive

been proven, it is reasonable to suggest that hormone levels may be altered in some men by stress. Therefore, educating the workers regarding the risks of occupational exposure to excessive noise [37] and wearing proper protective equipment during shift are practical, preventative strategies. [3], [37]

PHYSICAL HAZARD	EXAMPLES IN THE MINING INDUSTRY	RISK: MALE WORKER AND FETUS (when applicable)	ACTION TO AVOID RISK
Vibration	Vehicle operation, equipment, vibrating floors near heavy equipment operations, vibrating platforms.	 Significant reduction in spermatogenesis reported in men occupationally exposed to vibration ^[38]; supported by animal studies where male rodents exposed to 4 hours of vibration for 125 consecutive days at 5.07 m/sec/sec showed reduced spermatogenesis ^[3] Sexual Function: Erectile dysfunction due to vascular effects from seat compression and vibration reported in a non-occupational setting ^[39] Single case study of 'Penile Raynauds' in a worker with white finger and white toe ^[40] Androgen hormone levels may increase from whole-body vibration but beneficial effects from this increase are not reported ^[41] 	Keep vibration at acceptable workplace levels. Lower and upper limits of the 8 hr health guidance caution zone are 0.45 m/s² and 0.90 m/s² respectively for frequency-weighted r.m.s. acceleration values. Lower and upper limits of the 8 hr health guidance caution zone are 8.5 m/s¹.75 and 17 m/s¹.75 respectively for the vibration dose values. Avoid whole body vibration above the limit value of frequency-weighted r.m.s. acceleration of 1.15 m/s² or vibration dose value of 21 m/s. It is unclear whether the negative effects of vibration are due to the direct effects of vibration or due to the indirect effects of the vibration-induced increase in temperature. Therefore, see also "Actions to Avoid" for "Extremes of Heat" above.



CHEMICAL HAZARD	EXAMPLES IN THE MINING INDUSTRY	RISK: MALE WORKER AND FETUS (when applicable)	ACTION TO AVOID RISK
Aluminum	May be encountered during bauxite mining or aluminum refining or processing, welding. Also maintenance work such as grinding and machining (fabrication).	 Reproduction: Deterioration in sperm motility Animal (rodent) studies decreased testicular and epididymal weight, reduced sperm counts and fecundity at doses of 100-200 mg/kg/day^{[3], [45]} Sexual function: Insufficient data Fetus: Neurobehavioural abnormalities found in offspring^[3] 	TWA ^{[46], [47]} : 1 mg/m ³ (respirable fraction)
Arsenic	Product of mechanical extraction of ores or minerals, byproduct of smelting lead, copper, and zinc ores.	 Reproduction: High doses associated with testicular damage ^{[3], [48]} No available studies of fertility in humans ^{[49], [50], [51]} but animal studies report impairment of spermatogenesis ^[51] Sexual Function: Insufficient data 	TWA ^{[46], [47]} : 0.01mg/m ³ STEL ^{[46], [47]} : 0.05 mg/m ³
Beryllium	Control rods in nuclear reactors, sheet metal or wire welding. Mining or processing of rare earth element ores; recycling of metals.	 Reproduction: Toxic to testicular structures and associated with abnormal sperm morphology according to animal studies^[3] Sexual function: Insufficient data 	TWA ^{[46], [47]} : 0.05 μg/m ³ STEL ^{[46], [47]} : 0.01 mg/m ³

CHEMICAL HAZARD	EXAMPLES IN THE MINING INDUSTRY	RISK: MALE WORKER AND FETUS (when applicable)	ACTION TO AVOID RISK
Cadmium	Product of mechanical extraction of ores or minerals, byproduct of smelting lead, copper, and zinc ores. Cigarette smoking is a source of cadmium exposure.	 Reproduction: Decreased levels of fertility at blood levels <10 μg/L and reduced sperm motility and/or morphology ^{[52], [53], [54], [11], [51], [55], [56]} Known to affect male reproduction via various mechanisms (affects prostate) ^{[3], [57], [5], [58], [59], [14]} Animal Studies: Selective testicular damage at doses of 1.1-2.2 mg/kg^[I] Sexual function: Insufficient data 	TWA ^{[46],[47]} : 0.002 mg/m ³ (Respirable fraction) 0.01 mg/m ³
Carbon Monoxide	Diesel exhaust, iron and steel foundries, welding, forklift	Reproduction: No known direct effect on male reproductive health	TWA ^{[46], [47]} : 25 ppm

	operations, blasting gases, improper ventilation. Cigarette smoking is a source of carbon monoxide exposure.	Sexual function: No known direct effect on male s function	exual	
Chromium (metal/	Inhalation exposure hazard during	Reproduction: • Testicular atrophy [32]	TWA ^{[46], [47]}	

Chromium	innalation exposure	Reproduction:	TWA ^{[40], [47]}
(metal/	hazard during	• Testicular atrophy [32]	
inorganic)	stainless steel welding, corrosion	 Degree of damage proportional to dose ^[51] Decreased levels of fertility, reduced sperm 	Metal and Cr III compounds: 0.5 mg/m ³
Hexavalent form	inhibitor.	motility/morphology, decreased testosterone levels and increased levels of follicle-	Water- soluble Cr VI compounds: 0.05 mg/m ³
The trivalent form, in trace		stimulating hormone (animal studies) ^{[3], [54],} [11], [51], [73]	Insoluble Cr VI compounds: 0.01 mg/m ³
amounts, is an		Sexual Function:	
essential		Insufficient data	
nutrient.			

CHEMICAL HAZARD	EXAMPLES IN THE MINING INDUSTRY	RISK: MALE WORKER AND FETUS (when applicable)	ACTION TO AVOID RISK
Copper is an essential nutrient, but is toxic at high doses.	Used in alloys and in electroplating.	Reproduction: • Toxic to sperm when in direct contact, decreased sperm count, abnormal sperm Sexual Function: • Insufficient data	TWA ^{[46], [47]} Fume: 0.2 mg/m ³ Dust & mists: 1 mg/m ³
Fire by-	Mine fire.	Reproduction:	Avoid smoke inhalation.

Fire by- products (i.e. hydrogen cyanide, hydrocarbons)	Mine fire.	 Reproduction: Negative effects on spermatogenesis and male fertility have been observed in firefighters. This may be due to a host of toxicants in residential or industrial fires. It is possible some of the same toxicants may be present in mine fires (e.g. PAHs) 	Avoid smoke inhalation. Use of self-contained breathing apparatus (SCBA) to prevent fire smoke exposure in responders to fire events.
		Sexual function: Insufficient data	

Hydrogen Sulfide	Iron smelters, blast emissions. Also a decomposition gas (sewage handling).

Reproduction:No known direct effect on male reproductive health

Sexual function:

• No known direct effect on male sexual function

TWA^[61]: 10 ppm

STEL^[61]: 15 ppm

CHEMICAL HAZARD	EXAMPLES IN THE MINING INDUSTRY	RISK: MALE WORKER AND FETUS (when applicable)	ACTION TO AVOID RISK
Lead (elemental/ inorganic)	Product of mechanical extraction of ores or minerals, by-product of smelting lead, copper and zinc ores.	 Reproduction: Testicular toxicity leading to decreased levels of fertility and reduced sperm count/motility. [62], [63], [23], [12], [24], [3], [56], [16], [53], [32], [13], [10], [51], [11], [26], [14], [64], [65], [5], [59] Blood levels ≥40-50 µg/dl linked to changes in germ cell structure and function leading to abnormal sperm. [1], [12], [66], [56], [54], [68], [69] Sexual function: Reductions in testosterone levels. [8] Fetus: Male preconception blood levels greater than 30 µg/dl associated with increased risk of spontaneous abortion in female partners. [1], [12], [3], [56] Birth defects noted in children where paternal exposure was 2 to 4 times higher than normal. Perinatal death twice as common, increased incidence of cleft lip from paternal exposure. [70] 	TWA ^{[46], [47]} : Elemental Lead: 0.05 mg/m³ Tetraethyl lead: 0.10 mg/m³ STEL ^{[46], [47]} : Tetraethyl lead: 0.30 mg/m³ If not already regularly screening for blood lead levels, as per safety standards (regulation 490), screen during the preconception period. ^[1]

Manganese (elemental/ organic)

Manganese is an essential trace nutrient, but is toxic at high doses.

Used in steel production, as chemical compounds, and in welding rods.

Reproduction:
• Decreased fertility^{[52], [6], [5], [11]}

Sexual function:

• Suspected to affect libido^{[6], [12]}

TWA^{[46], [47]}:

Inorganic forms: 0.2 mg/m³

CHEMICAL HAZARD	EXAMPLES IN THE MINING INDUSTRY	RISK: MALE WORKER AND FETUS (when applicable)	ACTION TO AVOID RISK
Mercury	Used in instruments and older milling equipment. Electricians handling energy-saving light bulbs containing mercury.	 Reproduction: Interferes with male reproductive function/spermatogenesis^[52], ^[24], ^[5], ^[54], ^[59], ^[14] Organic mercury exposure for over 10 years correlated with oligospermia, teratospermia, asthenospermia, and reduced libido^[12] Affects fertility and is toxic in rodents (animal studies) Sexual function: Insufficient data Fetus: Urinary levels greater than 250nmol/L male workers associated with a 2-fold risk of spontaneous abortion in female partners^[71], ^[1], ^[3] 	If exposure is known to occur, discuss with a health care professional and delay attempts at conception for longer than 3 months (i.e. Related to time required for sperm turn-over). [71] TWA ^{[46],[47]} : Alkyl compounds (as Hg): 0.01mg/m³ All forms except alkyl (as Hg): 0.025 mg/m³ STEL ^{[46],[47]} : Alkyl compounds (as Hg): 0.03 mg/m³
Mono- Ammonium Phosphate and Sodium Alkyl Sulfate	ABC, Dry Chemical, fire extinguishers.	 Reproduction: No known direct effect on male reproductive health Sexual function: No known direct effect on male sexual function 	Due to the general safety and health risks of using a fire extinguisher of this type (i.e. hypoxia), use with caution. [72]
Nickel	Used in mining and processing of nickel ores and alloys such as stainless steel and nickel carbonyl produced during nickel refining.	 Reproduction: Decreased fertility^[16] and decline in semen parameters from nickel produced by welding fumes^{[3], [73]} Sexual function: Insufficient data 	TWA ^{[46], [47]} : Elemental/metal: 1 mg/m³ Insoluble compounds: 0.2 mg/m³ Soluble compounds: 0.1 mg/m³ Nickel subsulfide: 0.1 mg/m³ Nickel carbonyl: 0.05 ppm

CHEMICAL HAZARD
Nitrates, Nitrites, and Organic Nitro Compounds

EXAMPLES IN THE MINING INDUSTRY **RISK: MALE WORKER AND FETUS** (when applicable)

ACTION TO AVOID RISK

Explosives, blasting rock and waste water.

Reproduction:

• Toluenediamine and dinitrotoluene are on NIOSH's list of male reproductive hazards [3]

Dinitrobenzene and dinitrotoluene are associated with testicular toxicity^[21]

Animal studies (male rats): decreased epididymal sperm reserves (63% reduction in animals treated with 0.2% dinitrotoluenehighest dose group)[3], [74]

TWA^{[46], [47]}. Dinitrotoluene: 0.2 mg/m³ Nitrobenzene: 1 ppm

Nitroglycerin: 0.05 ppm

Trinitrotoluene: 0.1 mg/m³ or 0.01 ppm Nitrotoluene (all isomers): 2 ppm

STEL^{[46], [47]}.

Trinitrotoluene: 0.02 ppm or 0.2 mg/m³

Nitrogen Oxides (NOx):

Diesel exhaust, blasting gases, welding.

Oxide (NO)

Nitrogen Dioxide Cigarette smoking is a (NO₂) and Nitric source of nitric oxide and one mechanism for reduced fertility in male smokers is via increased production of systemic NO concentrations.[75]

Reproduction:

Sexual function: Insufficient data

• Human sperm is rich in poly unsaturated fatty acids which are sensitive to oxygen induced damage. NOx gases produce free radicals and are thought to damage sperm through high levels via this mechanism. Occupational evidence for this is limited and conclusions are conflicting.

Airborne pollutants of nitrogen oxide and nitrogen dioxide linked to reduced sperm count^[76]

NO decreases sperm motility and induces sperm toxicity in vitro.[77]

 A study in hospital workers with mean NO exposure of <50 ppm found no sperm abnormalities.[78]

Two studies looking at NO₂ exposure in men found a small increase in spontaneous abortion and congenital malformations [79], [80] however, these studies are criticized for confounders (reviewed in Paul) or attributable to the duration of exposure (mean greater than 30hr/week) and levels of NO₂ well above the NIOSH TWA of 25 ppm. (continued next page...)

TWA^{[46], [47]}:

Nitric Oxide: 25 ppm or 45 mg/m³ Nitrogen Dioxide: 3 ppm

STEI [46], [47].

Nitrogen Dioxide: 5 ppm

Maintaining levels below these standards should be sufficient protection.

CHEMICAL HAZARD	EXAMPLES IN THE MINING INDUSTRY	RISK: MALE WORKER AND FETUS (when applicable)	ACTION TO AVOID RISK
		 Notably NO levels increase with cigarette smoke and is thought to be a mechanism for increased infertility in men who smoke. Sexual function: No known direct effect on sexual 	
		function	
Particulate Matter (PM)	Diesel exhaust from machinery, rock dust.	 Reproduction: No known direct effect on male reproductive health, however, PM carrying multiple trace elements including heavy metals and polycycli aromatic hydrocarbons (PAHs) can act as endocrine disruptors affecting both the hypothalamic pituitary axis and testicular spermatogenesis. [81] They also have the potential for causing spermalterations (DNA damage). [81] Sexual function: PM in the respirable range (PM 2.5) can enhan atherosclerosis [82], [83], [84], [85] which can impair blood flow and therefore contribute to erectile dysfunction. [86], [87] PM carrying multiple trace elements including heavy metals and polycyclic aromatic hydrocarbons (PAHs) can act as endocrine disruptors affecting both the hypothalamic pituitary axis and testicular spermatogenesis. [8] 	n ce

CHEMICAL HAZARD	EXAMPLES IN THE MINING INDUSTRY	RISK: MALE WORKER AND FETUS (when applicable)	ACTION TO AVOID RISK
Polycyclic Aromatic Hydrocarbons (PAHs)	Generated from incomplete combustion of organic material and fossil fuels and vehicle exhaust. Cigarette smoking is a source of PAHs.	 Reproduction: Associated with sperm DNA damage [89], [90], [91] and decreased sperm motility [92], [93] In rodents, linked to testicular atrophy, hinders fertility and is associated with reduced sperm motility [3], [91], [93], [94] Sexual function: Insufficient data Fetus: Sperm DNA damage in the male germ line; major contributor to infertility and linked to an increased incidence of miscarriage and the appearance of various kinds of birth defects in the offspring [95], [96] 	Educational workshop; review by worker of safety regulations to keep particulate matter levels at a minimum during shift, (because polycyclic aromatic carbons typically bind to fine particulate matter).
Selenium Selenium is an essential nutrient but potentially toxic at high doses.	Product of mechanical extraction of ores or minerals.	Reproduction: Insufficient data. Sexual function: Insufficient data	TWA ^{[46], [47]} : Selenium and compounds: 0.2 mg/m ³ Selenium hexafluoride: 0.025 ppm or 0.1 mg/m ³
Tellurium	Product of mechanical extraction of ores or minerals.	Reproduction: • Insufficient data Sexual function: • Insufficient data	TWA ^{[46], [47]} : Tellurium and compounds, as Te, excluding hydrogen telluride: 0.1 mg/m ³ Tellurium hexafluoride: 0.01 ppm or 0.1 mg/m ³

CHEMICAL HAZARD	EXAMPLES IN THE MINING INDUSTRY	RISK: MALE WORKER AND FETUS (when applicable)	ACTION TO AVOID RISK
Thallium	Product of mechanical extraction of ores or minerals, smelting of lead and zinc ores.	 Reproduction: Chronic exposure linked to testicular dysfunction, reduced sperm motility^[97] and altered sperm morphology in rodents^[98] Sexual function: Insufficient data 	TWA ^{[46], [47]} : 0.02 mg/m ³ (inhalable fraction) NIOSH has recommended that 15 mg/m ³ of thallium be considered immediately dangerous to life and health. ^[99]
Uranium	Can be a naturally occurring chemical in mines.	 Reproduction: Human data insufficient, though has the potential to pose a radiation risk (see 'ionizing radiation' above). Daily exposure of uranyl acetate in drinking water for 64 days hinders male fertility and decreases sperm count in rodents [100], [3], [101] Sexual function: Insufficient data 	TWA ^{[46], [47]} : 0.2 mg/m ³ STEL ^{[46], [47]} : 0.6 mg/m ³
Vanadium Pentoxide	Used as an alloy, present in crude oil, used in some diesel exhaust catalysts, and catalysts for production of sulphuric acid.	 Reproduction: Human data insufficient Linked to decreased sperm count and fertility rates, lower testosterone levels and prostate atrophy in rodents ^{[3], [102]} Sexual function: Insufficient data 	TWA ^{[46], [47]} : 0.05 mg/m ³

Zinc Chloride Zinc Oxide

Zinc is an essential nutrient, but can be toxic at high doses.

Used as an alloy, as a metal coating and soldering flux and released when welding galvanized surfaces and during smelting of metal concentrates.

Reproduction:

No known health effects on human male reproductive health^[48]

Sexual function:

• No known health effects on human male sexual health^[48]

TWA^{[46], [47]}:

Zinc chloride fume: 1 mg/m³
Zinc oxide: 2 mg/m³ (Respirable fraction)

STEL [46], [47]:

Zinc chloride fume: 2 mg/m³
Zinc oxide: 10 mg/m³ (Respirable fraction)



OTHER HAZARDS	EXAMPLES IN THE MINING INDUSTRY	RISK: MALE WORKER AND FETUS (when applicable)	ACTION TO AVOID RISK
Ergonomic Exposure	Lifting, pushing, pulling, bending, heavy work.	 Reproduction: No adverse effects on male reproductive function have been noted^[3] Sexual function: No adverse effects on male sexual function have been noted^[3] 	None
	Prolonged standing.	 Reproduction: No adverse effects on male reproductive function have been noted Sexual function: No adverse effects on male sexual function have been noted 	None
	Prolonged sitting.	 Reproduction: Insufficient evidence to suggest that sitting causes infertility in men, more than standing [10], [26], [103] However, when prolonged sitting causes a higher scrotal temperature, for example using a laptop computer on your lap, [15] it may lead to decreased sperm count [55] Sitting duration is associated with increase scrotal temperature and both are associated with lower sperm counts [20], [9] Truck and taxi drivers that sit for prolonged periods have negative effects on their fertility [20], [104] Studies comparing tight versus loose underwear show similar results [7] Laptop computers have also been shown to overheat the scrotum [18] 	anything that impedes scrotal heat loss will affect testicular temperature and in turn any elevation of testicular temperature will have a harmful effect on spermatogenesis. In general the more prolonged the elevation in testicular temperature, the greater the negative consequence to the sperm. Therefore, recommendations to remove factors that might impede normal cooling of the scrotum/testes are logical; particularly for men who are attempting to father a pregnancy and especially if they know they have low sperm counts or low sperm mobility take steps to minimize scrotal heating. Sit with thighs apart wear clothing that encourages good air exchange. [105] Change positions, from seated

OTHER HAZARDS	EXAMPLES IN THE MINING INDUSTRY	RISK: MALE WORKER AND FETUS (when applicable)	ACTION TO AVOID RISK
Scheduling	Working hours, shift rotation, and shift duration/night shift.	 Reproduction: Shift work has been shown to be related to infertility and to reduced semen motility/morphology^{[106], [64]} Insufficient evidence that long work schedules (e.g. 14-28 days without interruption) negatively impact fertility Sexual function: Insufficient data 	If fertility issues are suspected and no other causative agent is identified, alterations of work hours/schedule may be considered.
Occupational and Non- occupational Stress	Death of a spouse, divorce, job loss, loss of work, high job demands and minimal job control or workplace conflicts.	 Reproduction: Occupational stress and burnout related to male infertility and reduced semen motility/morphology [107], [32], [10], [64] also shown in animals^[3] Sexual function: Reduced testosterone levels, negatively impacts on spermatogenesis, on semen motility/morphology, may affect libido and sexual performance [108], [64], [3] 	Seek psychosocial support to learn coping/stress management skills. Modify job tasks to support a stress-free environment and promote work-life balance. Si

REFERENCES

- Paul, M., Occupational and environmental reproductive hazards: A guide for clinicians 1993, Baltimore: Williams & Wilkins.
- Bonde, J.P., et al., Identifying environmental risk to male reproductive function by occupational sperm studies: logistics and design options. Occupational and Environmental Medicine, 1996. 53(8): p. 511-519.
- Frazier, L.M. and M.L. Hage, Reproductive hazards of the workplace 1998, New York: John Wiley & Sons.
- Thonneau, P., et al., Occupational heat exposure and male fertility: a review. Human Reproduction, 1998. 13: p. 2122-2125.
- 5. Bonde, J.P. and L. Storgaard, *How work-place conditions, environmental toxicants and lifestyle affect male reproductive function.* International Journal of Andrology, 2002. **25**: p. 262-268.
- Baranski, B., Effects of the workplace on fertility and related reproductive outcomes. Environmental Health Perspectives, 1993. 101 (Suppl. 2): p. 81-90.
- 7. Mieusset, R. and L. Bujan, *Testicular heating and its possible contributions to male infertility: a review.* International Journal of Andrology, 1995. **18**: p. 169-184.
- 8. Paul, M., Occupational reproductive hazards. The Lancet, 1997. 349: p. 1385-1388.
- Hjollund, N.H., et al., Diurnal scrotal skin temperature and semen quality. The Danish First Pregnancy Planner Study Team International Journal of Andrology, 2000. 23: p. 309-318.
- Jensen, T.K., J.P. Bonde, and M. Joffe, The influence of occupational exposure on male reproductive function. Occupational Medicine, 2006. 56: p. 544-553.
- Ashiru, O.A. and O.O. Odusanya, Fertility and occupational hazards: Review of the literature. African Journal of Reproductive Health, 2009. 13(1): p. 159-166.
- Tas, S., R. Lauwerys, and D. Lison, Occupational Hazards for the Male Reproductive System. Critical Reviews in Toxicology, 1996. 26(2): p. 261-307.
- Burdorf, A., et al., Effects of occupational exposure on the reproductive system: core evidence and practical implications. Occupational Medicine, 2006. 56: p. 516-520.
- Brinkworth, M.H. and D.J. Handelsman, Environmental influences on male reproductive health. Andrology, 2010: p. 365-389.
- Sheynkin, Y., et al., Protection from scrotal hyperthermia in laptop computer users. Fertility and Sterility, 2010.
- Figa-Talamanca, I., M.E. Traina, and T. Urbani, Occupational exposures to metals, solvents, and pesticides: recent evidence on male reproductive effects and biological markers. Occupational Medicine, 2001. 51(3): p. 174-188.
- Bujan, L.D., M. Charlet, JP. Thonneau, P. Mieusset. and *Increase in scrotal temperature in car drivers*.
 Human Reproduction, 2000. 15(6): p. 1355-1357.
- 18. Sheynkin, Y., Jung, M., Yoo, P., Schulsinger, D., Komaroff, E., *Increase in scrotal termperature in laptop computer users*. Human Reproduction, 2005. **20**(2): p. 452-5.
- 19. Setchell, B.P., Heat and the testis. Journal of Reproduction and Fertility, 1998. 114: p. 179-184.
- 20. Hjollund, N.S., L. Ernst, E. Bonde, JP. Olsen, J., *The relation between daily activities and scrotal temperature*. Reproductive Toxicology, 2002. **16**: p. 209-214.
- Witorsch, R.J., Reproductive Toxicology Second Edition. Target Organ Toxicology Series, ed. A.W. Hayes, J.A. Thomas, and D.E. Gardner1995, New York: Raven Press.
- 22. Johnston, D.J., G.G. Jamieson, and S. Wright, *Reproductive and developmental hazards and employment policies*. British Journal of Indusrial Medicine, 1992. **49**: p. 85-94.
- 23. Chester, T.J., et al., Caution: work can be hazardous to health. Patient Care, 1996. 30(3): p. 70-84.
- 24. O'Neal, M.A., Warning Signs, Occupational Health & Safety, 1997. 66(12): p. 42-25.

- Sinno-Tellier, S., et al., Male gonadal dose of ionizing radiation delivered during X-ray examinations and monthly probability of pregnancy: A population-based retrospective study. BMC Public Health, 2006. 6(55).
- Bonde, J.P., Male reproductive organs are at risk from environmental hazards. Asian Journal of Andrology, 2010. 12: p. 152-156.
- Ogilvy-Stuart, A.L. and S.M. Shalet, Effect of radiation on the human reproductive system.
 Environmental Health Perspectives Supplements, 1993. 101 (Suppl. 2): p. 109-116.
- Affairs, C.o.S., Effects of physical forces on the reproductive cycle. Journal of the American Medical Association, 1984. 251(2): p. 247-250.
- 29. Gonzalez, C., Occupational reproductive health and pregnancy hazards confronting health care workers. American Association of Occupational Health Nurses, 2011. **59**(9): p. 373-376.
- Rachootin, P. and J. Olsen, The risk of infertility and delayed conception associated with exposures in the Danish workplace. Journal of Occupational Medicine, 1983. 25.
- 31. Fouladi, D.B., Nassiri, P., Monazzam, E.M., Farahani, S., Hassanzadeh, G., Hoseini, M., *Industrial noise exposure and salivary cortisol in blue collar industrial workers*. Noise Health, 2012. **41**(2): p. 184-9.
- 32. Sheiner, E.K., et al., *Effect of occupational exposures on male fertility: Literature Review.* Industrial Health, 2003. **41**: p. 55-62.
- Carosi, L. and F. Calabro, Fertility in couples working in noisy factories. Folia Med (Napoli), 1968.
 51(4): p. 264-8.
- 34. Ruffoli, R., et al., Diazepam administration prevents testosterone decrease and lipofuscin accumulation in testis of mouse exposed to chronic noise stress. Andrologia, 2006. 38: p. 159-165.
- 35. Monder, C., et al., 11 beta-Hydroxysteroid dehydrogenase alleviates glucocorticoid-mediated inhibition of steroidogenesis in rat Leydig cells. Endocrinology, 1994. 134(3): p. 1199-204.
- 36. Smith, A., The World Health Organization and the prevention of deafness and hearing impairment caused by noise. Noise Health, 1998. 1: p. 6-12.
- Gidikova, P., et al., Hearing impairment among workers occupationally exposed to excessive levels of noise. Central European Journal of Medicine, 2007. 2(3): p. 313-318.
- 38. Penkov, A., R. Stanislavov, and D. Tzvetkov, *Male reproductive function in workers exposed to vibration*. Central European Journal of Public Health, 1996, 4(3); p. 185-188.
- Ochiai, A., et al., Do motorcyclists have erectile dysfunction? A preliminary study. International Journal of Impotence Research, 2006. 18: p. 396-399.
- Mooradian, A.D., et al., Penile Raynaud's phenomenon: a possible cause of erectile failure. Am J Med, 1988. 85(5): p. 748-50.
- 41. Cardinale, M. and M.H. Pope, *The effects of whole body vibration on humans: dangerous or advantageous?* Acta Physiologica Hungarica, 2003. **90**(3): p. 195- 206.
- 42. 2631-1, I., Mechanical vibration and shock Evaluation of human exposure to whole-body vibration-Part1: General requirements, in International Organization for Standardization1997: Switzerland.

43.

- Directive 2002/44/EC of the European Department and of the council of 25 June 2002 on the minimum health and safety requirements regarding the exposure of workers to the risks arising from physical agents (vibration) (sixteenth individual Directive within the meaning of Article 16(1) of Directive 89/391/EEC). Official Journal of the European Communities, 2002.
- 44. Dawson, E.B., et al., Comparison of sperm viability with seminal plasma metal levels. Biological Trace Element Research, 1998. 64: p. 215-219.

REFERENCES

- Krewski, D., et al., Human health risk assessment for aluminum, aluminum oxide, and aluminum hydroxide Journal of Toxicology and Environmental Health, Part B: Critical Reviews, 2007.
 10(Suppl.1): p. 1-269.
- American Conference of Governmental Industrial Hygienists. 2011 Threshold Limit Values and Biological Exposure Indices., 2011.
- 47. Ontario Ministry of Labour. Occupational health and safety act in R.R.O. 1990, Regulation 833:

 Control of Exposure to Biological or Chemical Agents2010. p. 1-9.

 http://www.labour.gov.on.ca/english/hs/pdf/ontario_oels.pdf
- 48. Meeker, J.D., et al., Environmental exposure to metals and male reproductive hormones: Circulating testosterone is inversely associated with blood molybdenum. Fertility and Sterility, 2010, 93(1); p. 130-149.
- Schrag, S.D. and R.L. Dixon, Occupational exposures associated with male reproductive dysfunction Annual Review of Pharmacology and Toxicology, 1985. 25: p. 567-592.
- Golub, M.S., M.S. Macintosh, and N. Baumrind, Developmental and reproductive toxicity of inorganic arsenic: Animal studies and human concerns. Journal of Toxicology and Environmental Health, Part B: Critical Reviews, 1998. 1(3): p. 199-237.
- Chowdhury, A.R., Recent advances in heavy metals induced effect on male reproductive function— A retrospective. Al Ameen Journal of Medical Sciences, 2009. 2(2): p. 37-42.
- 52. Danniell, W., Male reproductive toxicity. The Western Journal of Medicine, 1990. 152(2): p. 174.
- 53. Ong, C.N., H.M. Shen, and S.E. Chia, *Biomarkers for male reproductive health hazards: are they available?* Toxicology Letters, 2002. **134**: p. 17-30.
- 54. Kumar, S., Occupational exposure associated with reproductive dysfunction. Journal of Occupational Health, 2004. 46: p. 1-19.
- Sharpe, R.M., Environmental/lifestyle effects on spermatogenesis. Philosophical Transactions of the Royal Society, 2010. 365: p. 1697-1712.
- 56. Telisman, S., et al., Semen quality and reproductive endocrine function in relation to biomarkers of lead, cadmium, zinc, and copper in men. Environmental Health Perspectives, 2000. **108**(1): p. 45-53.
- Hoyer, P.B., Reproductive toxicology: current and future directions. Biochemical Pharmacology, 2001. 62: p. 1557-1564.
- Grajewski, B., et al., Occupational Exposures and Reproductive Health: 2003 Teratology Society Meeting Symposium Summary. Birth Defects Research (Part B), 2005. 74: p. 157-163.
- Woodruff, T.J., et al., Proceedings of the Summit on Environmental Challenges to Reproductive Health and Fertility: executive summary. Fertility and Sterility, 2008. 89(2): p. 281-300.
- 60. McDiarmid, M., et al., *Reproductive Hazards of Fire Fighting II. Chemical Hazards*. American Journal of Industrial Medicine, 1991. **19**: p. 447-472.
- Handbook of Training in Mine Rescue and Recovery Operations Ontario Mine Rescue, W.S. North, Editor 2011, Workplace Safety North: North Bay.
- Nisbet, I.C.T. and N.J. Karch, Chemical Hazards to Human Reproduction 1983, New Jersey: Noyes Data Corporation.
- 63. Sexton, S., *The reproductive hazards of industrial chemicals: The politics of protection.* The Ecologist, 1993. **23**(6): p. 212-213.
- 64. El-Helaly, M., et al., *Workplace exposures and male infertility- a case-control study.* International Journal of Occupational Medicine and Environmental Health, 2010. **23**(4): p. 331-338.
- Mamtani, R., et al., Review Article- Metals and Disease: A Global Primary Health Care Perspective. Journal of Toxicology 2011. 2011: p. 11 pages.

- 66. Apostoli, P., et al., *Male reproductive toxicity of lead in animals and humans*. Occupational and Environmental Medicine, 1998. **55**: p. 364-374.
- Lancranjan, I., Popescu, H.I., Gavănescu, O., Klepsch, I., Serbănescu, M., Reproductive ability of workmen occupationally exposed to lead. Archives of Environmental Health, 1975. 30(8): p. 396-401.
- 68. Shiau, C., J. Wang, and P. Chen, *Decreased fecundity among male lead workers*. Occupational and Environmental Medicine, 2004. **61**: p. 915-923.
- 69. Winker, R. and H.W. Rudiger, *Reproductive toxicology in occupational settings: an update.* International Archives of Occupational and Environmental Health, 2006. **79**: p. 1-10.
- 70. Kristensen, P.e.a., *Perinatal outcome among children of men exposed to lead and organic solvents in the printing industry.* American Journal of Epidemiology, 1993. **137**(2): p. 134-44.
- Cordier, S., et al., Paternal exposure to mercury and spontaneous abortions. British Journal of Industrial Medicine, 1991. 48: p. 375-381.
- 72. Beitland, S., et al., , *Inhalation of fire extinguisher powder*. European Journal of Trauma, 2006. **32**(3): p. 286-291.
- 73. Danadevi, K., et al., , Semen quality of indian welders occupationally exposed to nickel and chromium. Reproductive Toxicology, 2003. 17(4): p. 451-456.
- 74. Pant, N. and S.P. Srivastava, *Testicular and spermatotoxic effect of nitrate in mice*. Human and Experimental Toxicology, 2002. **21**: p. 37-41.
- 75. Ghaffari, M.A.a.R., M., Lipid peroxidation and nitric oxide levels in males smokers' spermatozoa and their relation with sperm motility. Journal of Reproduction and Infertility, 2012. 13(2): p. 81-87.
- Jurewicz, J., et al., Environmental factors and semen quality. International Journal of Occupational Medicine and Environmental Health, 2009. 22(4): p. 305-329.
- Rosselli, M., et al., , Effects of nitric oxide on human spermatozoa: evidence that nitric oxide decreases sperm motility and induces sperm toxicity. Human Reproduction, 1995. 10(7): p. 1789-90.
- 78. Wyrobeck, A.J., et al., , Sperm studies in anesthesiologists. Anesthesiology, 1981. 55: p. 527.
- 79. Cohen, E.N., et al., , *Occupational disease in dentistry and chronic exposure to trace anesthetic gases.* Journal of the American Dental Association, 1980. **101**: p. 21-31.
- Guirguis, S.S., Pelmear, P.L., Roy, M.L., Wong, L., Health effects associated with exposure to anaesthetic gases in Ontario hospital personnel. British Journal of Industrial Medicine, 1990.
 45: p. 490-497.
- 81. Hammoud, A., et al., *Decreased sperm motility is associated with air pollution in Salt Lake City.* Fertility and Sterility, 2010. **93**(6): p. 1875-1879.
- 82. Araujo, J.A., et al., Ambient particulate pollutants in the ultrafine range promote early atherosclerosis and systemic oxidative stress. Circulatation Research, 2008. 102: p. 589-96.
- 83. Sun, Q., et al., Long-term air pollution exposure and acceleration of atherosclerosis and vascular inflammation in an animal model. Journal of the American Medical Association, 2005. **294**(23): p. 3003-10.
- 84. Suwa, T., et al., *Particulate air pollution induces progression of atherosclerosis*. Journal of the American College of Cardiology, 2002. **39**(6): p. 935-42.
- 85. Yatera, K., et al., *Particulate matter air pollution exposure promotes recruitment of monocytes into atherosclerotic plaques*. Journal of American Physiology. Heart and Circulatory Physiology., 2008. **292**(2): p. 944-53.

REFERENCES

- Eaton, C.B., et al., , A retrospective study of the relationship between biomarkers of atherosclerosis and erectile dysfunction in 988 men. International Journal of Impotence Research, 2007. 19: p. 218-225.
- 87. Schwartz, B.G., and Kloner, R.A., *Cardiovascular implications of erectile dysfunction*. Circulation, 2011. **123**: p. 609-611.
- 88. 854 Regulations for Mines and Mining Plants, O.H.a.S. Act, Editor 1990.
- 89. Ji, G., et al., *Joint effects of XRCC1 polymorphisms and polycyclic aromatic hydrocarbons exposure on sperm DNA damage and male Infertility.* Toxicological Sciences, 2010. **116**(1): p. 92-98.
- 90. Sadeu, J.C., et al., Alcohol, drugs, caffeine, tobacco, and environmental contaminant exposure: Reproductive health consequences and clinical implications: Review Article. Critical Reviews in Toxicology, 2010. 40(7): p. 633-652.
- 91. Han, X., et al., Association between urinary polycyclic aromatic hydrocarbon metabolites and sperm DNA damage: A population study in Chongqing, China. Environmental Health Perspectives, 2011. 119(5): p. 652-657.
- 92. Hsu, P.C., et al., Sperm DNA damage correlates with polycyclic aromatic hydrocarbons biomarker in coke-oven workers. International Archives of Occupational and Environmental Health, 2006. **79**: p. 349-356.
- 93. Ramesh, A., et al., Alteration of fertility endpoints in adult male F-344 rats by subchronic exposure to inhaled benzo(a)pyrene. Experimental and Toxicologic Pathology, 2008. **60**(4-5): p. 269-280.
- 94. Jeng, H.A. and L. Yu, *Alteration of sperm quality and hormone levels by polycyclic aromatic hydrocarbons on airborne particulate particles*. Journal of Environmental Science and Health Part A, 2008. **43**: p. 675-681.
- Morris, I.D., et al., The spectrum of DNA damage in human sperm assessed by single cell gel electrophoresis (comet assay) and its relationship to fertilization and embryo development. Human Reproduction, 2002. 17: p. 990-998.
- Aitken, R.J., G.N. lulii, and R.I. McLachlan, Biological and clinical significance of DNA damage in the male germ line. International Journal of Andrology, 2009. 32: p. 46-56.
- Formigli, L., et al., Thallium-Induced testicular toxicity in the rat. Environmental Research, 1986. 40:
 p. 531-539.
- Gregotti, C., et al., Effects of thallium on primary cultures of testicular cells. Journal of Toxicology and Environmental Health, 1992. 36(1): p. 59-69.
- Peter, A.L.J. and T. Viraraghavan, *Thallium: a review of public health and environmental concerns*.
 Environment International, 2005. 31(4): p. 493-501.
- 100. Llobet, J.M., et al., Influence of chronic exposure to uranium on male reproduction in mice. Fundamental and Applied Toxicology, 1991. 16: p. 821-829.
- Domingo, J.L., Reproductive and developmental toxicity of natural and depleted uranium: a review.
 Reproductive Toxicology, 2001. 15: p. 603-609.
- 102. Chandra, A.K., et al., Protection against vanadium-induced testicular toxicity by testosterone propionate in rats. Toxicology Mechanisms
- Frey, K.A., Male reproductive health and infertility. Primary Care: Clinics in Office Practice, 2010.
 37(3): p. 643-652.
- Stoy, J., et al., Semen quality and sedentary work position. International Journal of Andrology, 2004.
 p. 5-11.

- 105. Brindley, G.S., Deep scrotal temperature and the effect on it of clothing, air temperature activity, posture and paraplegia. British Journal of Urology, 1982. 54(1): p. 49-55.
- 106. Irgens, A., K. Kruger, and M. Ulstein, The effect of male occupational exposure in infertile couples in Norway. Journal of Occupational and Environmental Medicine, 1999. 41: p. 1116-20.
- 107. Negro-Vilar, A., Stress and other environmental factors affecting fertility in men and women: overview. Environmental Health Perspectives Supplements, 1993. 101 (Suppl. 2): p. 59-64.
- 108. Mahdi, A.A., et al., Withania somnifera Improves Semen Quality in Stress-RelatedMale Fertility. Evidence-Based Complementary and Alternative Medicine, 2009. 2011: p. 1-9.

