

A review of scrotal temperature regulation and its importance for male fertility

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Abstract

Male fertility is a very important but often neglected area in medical practice. Compared to what is available on female subfertility, there is a dearth in research on male fertility. Fertility in male depends on the normally functioning hypothalamo-pituitary gonadal axis, normal testis and the environment necessary for viable, motile sperm production. The production of healthy germ cells require a temperature which is slightly below the core body temperature and there are several temperature regulatory mechanisms in humans that ensure the maintenance of the scrotal temperature several degrees lower. The aim of the present review is to synthesise and summarise the literature available on scrotal temperature regulation in the healthy male and to recapitulate the disease conditions, environmental, occupational and lifestyle factors that affects scrotal temperature and thus male fertility.

Introduction

Subfertility affects approximately 1 in 7 couples in most countries in the world¹ and in 30% of cases the problem is with the male partner². For many men the only treatment option available is assisted reproductive technology with the use of techniques such as *in vitro* fertilization and intra-cytoplasmic sperm injection³. Compared to female subfertility, relatively little research

has been presented on the factors that contribute to male fertility. What we do know, is that male fertility depends on a normally functioning hypothalamo-pituitary-gonadal hormonal axis and normal sperm production. Spermatogenesis (sperm production) in turn requires structurally and functionally normal testes and the maintenance of the testicles cooler than the body core.

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For effective sperm production, the male gonads in most mammals require an environment that is 2 to 6°C cooler than the body core^{4,5}. The exact cytological changes occurring after heat exposure remain controversial. The most vulnerable cell type to heat seems species specific; Young⁶ reported that in the guinea pig, spermatocytes degenerated first, but in the mouse the earliest heat induced changes occurred in the spermatozoa⁷. In humans, spermatids and spermatozoa are reported to be fragile at high temperatures compared to spermatogonia and spermatocytes⁸. Findings of more recent cellular studies suggest that heat-induced damage to cells occur as a part of cellular stress response by inducing heat shock protein expression. The temperature at which heat shock protein expression is induced varies with the tissue concerned even in the same species⁹ with germ cells having a lower threshold of expression compared to liver cells in the mouse model.

Anatomical and physiological aspects of scrotal temperature regulation

Humans like many mammals have a skin-covered pouch that holds the testes outside the body cavity and helps to maintain the testes at a temperature lower than the body core. The testes are small ovoid organs that are encased in a tough fibrous capsule, known as the *tunica albuginea*. This capsule comprises three layers. The innermost *tunica vasculosa*, the middle *tunica albuginea* proper and the outermost *tunica vaginalis*¹⁰. The wall of the scrotal sac consists of the skin, the tunica dartos muscle, cremasteric fascia, internal spermatic fascia and parietal *tunica vaginalis*¹¹ in order from outside to inside. The subcutaneous tissue does not contain any fat¹². In the embryo, the testes develop from the gonadal ridges located in the posterior abdominal wall and then they descend from this point to the scrotum during development and is induced by fetal testosterone¹⁰.

Although from an evolutionary perspective there appears to be a benefit to having the gonads housed this way for local thermoregulation, there are certain inherent disadvantages also, like being vulnerable for external insults like torsion, rupture and being subjected to trauma. The scrotum plays an active role in providing the necessary thermal environment for the testis. There are five main anatomical features that contribute to the maintenance of the testicular temperature lower than that of the body core. They are, the presence of

the tunica dartos smooth muscle in the scrotal skin, the striated cremaster muscle, a counter-current heat exchange system in the testicular vascular supply, the absence of a subcutaneous fat layer in the scrotal skin, and the abundance of sweat glands in the skin.

The dartos muscle is the smooth muscle that lines the scrotum and contributes to the wrinkled appearance of the skin overlying it^{12,13}. The dartos muscle relaxes when the ambient or core temperature increases⁴. The relaxation of the dartos contributes to cooling of the testes partly by holding them away from the body¹⁰ which is the local heat source; and partly by causing the blood vessels that run in between the muscle to dilate, improving blood flow and facilitating heat loss from the skin¹². Cooler ambient temperatures, on the other hand, stimulate contraction of the muscle, reducing the skin surface area and the blood flow to the skin, while holding the testes closer to the core body heat source.

The cremaster muscle fibres, which are skeletal muscle fibres found in the spermatic cord, are more directly involved in controlling the distance at which the testes are held from the body¹⁴. Contraction of the cremaster causes elevation of the testes and relaxation of cremaster causes descent of the testes. Thus the dartos controls the scrotal skin while the cremaster raises and lowers the testes¹⁵.

The testicular artery that takes blood to the testis from the abdominal aorta descends through the pampiniform plexus of veins that return blood from the testis. Heat exchange between the artery and the surrounding veins via this arrangement also helps in maintaining scrotal temperature^{16,17,18}. Because of this counter-current system, the testes receive blood that is cooler compared to other organs in the body. Blood returning from the testis has lost heat to the environment and is thus cooler than arterial blood. When arterial blood in the testicular artery enters the pampiniform plexus it loses heat to the cooler blood in the pampiniform plexus, and is gradually cooled as it travels towards the testis¹⁹. This mechanism is effective however, only if the scrotum loses heat by radiation²⁰, convection or evaporation¹⁸ to the environment.

A subcutaneous fat layer generally has an insulating effect, reducing conductive heat loss to the environment from the body core. The absence of the subcutaneous fat in the scrotal skin¹³, therefore, facilitates the loss of heat from the testes.

Finally, mechanism of sweating on the scrotal skin due to the presence of a large number of sweat glands on it stimulated by adrenergic sympathetic nerves²¹ helps to maintain the testicular temperature below that of the body core²².

Clinical implications of abnormalities in scrotal temperature

There is no doubt that exposure to excessive heat adversely affects male fertility²³. Excessive heat exposure of the testis due to intrinsic or extrinsic factors decreases sperm concentration, motility and morphologically normal sperms²⁴. There are pathological conditions such as varicocele and cryptorchidism which can adversely affect male fertility through testicular heating²⁵. Similarly, there are lifestyle factors and occupational factors that can disrupt thermoregulatory mechanisms of the testis which in turn adversely affect spermatogenesis process.

Varicocele is the abnormal dilatation and tortuosity of veins in the pampiniform plexus leading to venous stasis within the scrotum. This adversely affects the counter-current heat exchange process in the scrotum leading to a rise in the temperature within the testis²⁶. Exact mechanism of causing male subfertility by a varicocele is not well understood but, higher scrotal temperature and oxidative stress are the hypothesized causes²⁷. Varicocele is a relatively common finding among men with primary and secondary subfertility and it has become the most common treatable cause of male infertility²⁸.

Cryptorchidism is the failure of testis to descend to the scrotal sac and it is a congenital defect seen in 2-4% full-term male infants²⁹. Fifty percent of the cases are reported to get corrected spontaneously within first year of life. However, supra-scrotal position of the testis is at higher risk of male infertility and testicular malignancy. Again, heat induced apoptosis of germ cells and oxidative stress are the main culprits of male infertility associated with cryptorchidism³⁰. Heat induces sperm DNA fragmentation in morphologically normal sperms and it is applicable to both varicocele and cryptorchidism³¹.

Moreover, there are some factors related to the environment, posture, life style and the occupation associated with subfertility. Ambient temperature and seasonal temperature changes may affect male fertility through testicular heat stress.

Postural changes have an effect on scrotal temperature and the lowest testicular temperatures are observed in unclothed standing position. Scrotal temperature is relatively higher in supine and sitting position³². In the supine position scrotum is resting on thighs and in direct contact with relatively higher body temperature. Scrotal temperature is highest during sleep when the body is not moving. Similarly, prolonged sitting position has been shown to increase scrotal temperature in car drivers³³. During sitting, scrotum is trapped in between the thighs with minimal ventilation. Irrespective of whether legs apart, together or crossed, sitting affects scrotal temperature³². This is a contributing factor for paraplegic men to become subfertile³³. Adverse effects of sitting on heated surfaces on scrotal temperature and fertility needs no mention. Apart from posture, the insulating effect of clothes increases the scrotal temperature. In both standing and supine position wearing underwear, increases scrotal temperature on average by 1.5°C-2°C compared to the naked state³⁴. In a clothed state, if air flow is lower the scrotal temperature tends to be higher and therefore, tighter the underwear higher would be the scrotal temperature and adverse effects on male fertility. However, up to date there is no conclusive evidence about the type of underwear which has the highest impact on male fertility by increasing scrotal temperature³⁵.

Obesity is a global pandemic and the prevalence of obesity is higher among subfertile male than in males with normal seminal fluid parameters³⁶. Obesity can directly affects spermatogenesis through scrotal lipomatosis which is deposition of fat around the spermatic cord³⁷. Moreover, due to being sedentary and reduced physical activity, obesity indirectly causes male infertility through scrotal hyperthermia.

Hot baths and sauna also adversely affects spermatogenesis. Hot baths may be used as a way of relaxation or as a daily habit by using hot tubs, heated Jacuzzi or warm baths. Discontinuation of such wet hyperthermia is reported to improve seminal fluid parameters³⁸.

Use of laptop computers on the lap increases scrotal temperature when compared to individuals in same sitting position without using laptops³⁹. From above mentioned mechanisms high temperatures can adversely affect spermatogenesis and it is advisable to avoid the use of laptops placed on the lap for males with abnormal seminal fluid parameters.

Cycling is an activity which gives an exercise to the whole body. Especially among professional cyclists this leads to prolonged seated posture on a saddle seat while wearing tight clothes⁴⁰. At the same time due to the physical activity increased periscrotal circulation will help in dissipation of testicular heat.

Exposure to radiant heat is unavoidable in some occupations like welders, oven operators and bakers. In radiant heat the energy is transmitted by electromagnetic waves in contrast to conduction or convection. Radiant heat is known to cause reversible changes in spermatogenesis⁴¹. Bakers and ceramic oven operators have been reported to have difficulty in conception compared to controls^{23,42}. Long hours of driving in seated position by professional drivers have been reported to be detrimental for male fertility through scrotal hyperthermia. It is reported to cause a reduction in the sperm concentration, abnormal sperm motility and morphology.

Although it is not yet possible to explain fully why spermatogenesis is heat sensitive, there is evidence to support the case that heat causes subfertility in humans^{43,44,45,46}. The production of healthy germ cells require a temperature which is slightly below the core body temperature and there are several temperature regulatory mechanisms in humans that ensure the maintenance of the scrotal temperature several degrees lower. The present review synthesises and summarises the literature available on scrotal temperature regulation in the healthy male and recapitulates the disease conditions, environmental, occupational and lifestyle factors that affects scrotal temperature and thus male fertility.

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