

Original Article

On the Origin of Descended Scrotal Testicles: The Activation Hypothesis

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Abstract: Male gonads contained in an unprotected skin sack located outside the body cavity are a peculiar mammalian anomaly. We advance the hypothesis that descended scrotal testicles in humans and many other mammals evolved to provide a situation specific means of activating sperm. As a result of consistent temperature differences between the male and female reproductive tracts (i.e., scrotal temperatures are typically maintained at 2-3°C below body temperature), we propose that the rise to body temperature that accompanies insemination into the vagina serves as one of several triggers for the activation of sperm. We explore some of the features of mammalian reproduction and behavior that are consistent with this hypothesis, make some testable predictions, and examine the psychological and behavioral adaptations that have evolved to protect otherwise vulnerable scrotal testicles from damage.

Keywords: testicles, scrotum, cremasteric reflex, pain, nocturnal copulation, temperature, sperm motility, capacitation, hyperactivation

Introduction

It is almost unthinkable to ask why ovaries do not descend during embryological development and emerge outside the female's body cavity in a thin, unprotected sack. Evolution is based on reproductive competition among individuals for genetic representation in future generations. The integrity of the gonads is of paramount importance when it comes to reproduction. Because of vulnerability to damage, insult, and temperature variation, unprotected ovaries located outside the body cavity would be an enormous handicap/serious reproductive disadvantage. The same reasoning applies with equal force to the testicles. But unlike ovaries, descended testicles located outside the body

cavity in the scrotum are common among many mammals in spite of all the obvious risks and disadvantages.

We propose a simple mechanism in the common ancestry of mammals (or at least placentals) that may have evolved to promote descended, scrotal testicles. We also examine a number of adaptations that appear to have evolved to maintain scrotal testicles and minimize the costs of this peculiar gonadal arrangement. Finally we offer testable predictions about the minority of mammals without descended testicles (testicondy).

Species Differences in Testicular Descent

Werdelin and Nilsson (1999) provide a valuable account of the evolution of testicular descent. Not all mammals have scrotal testicles, or even descended testicles. Categorizing mammals into those with and without a scrotum obscures the fact that some have descended but ascrotal testicles. Werdelin and Nilsson distinguish, therefore, between species with testicles that are 1) descended and scrotal, 2) testicles that are descended but ascrotal, as in the case of seals where the testicles are located subcutaneously, and 3) testicles that are not descended and remain deeply embedded in the body cavity, as in the case of elephants. While descended scrotal testicles are widespread and represent the prevailing arrangement among mammals, the loss of the scrotum is more common than the loss of testicular descent. Werdelin and Nilsson conclude that the scrotum is an older primitive adaptation, and more recent testicular evolution among mammals “has proceeded from scrotal to ascrotal but never the reverse,” while the “loss of descensus is relatively rare” (p. 69).

Theories of Scrotal Testicular Descent

Why some mammals have testicles located outside the body cavity has puzzled biologists. “The adaptive significance of the scrotum is unresolved after more than 60 years of debate and experimentation” (Freeman, 1990, p. 429). Thoughtful reviews of the different theories that have been advanced to explain scrotal testicles are provided by Birkhead (2000) and Werdelin and Nilsson (1999).

The traditional interpretation is that the scrotum evolved to provide a cool environment that would ensure for optimal spermatogenesis (Moore, 1926). As an interesting extension of this notion, Bedford (1978) has argued that the epididymis functions as a cold-storage site for sperm, and therefore it was the epididymis that was the prime mover for the evolution of the scrotum. We now know that both the storage and formation of viable sperm for most mammals requires that the testicles be maintained below body temperature (Setchell, 1998), but why this is the case has never been adequately explained.

Some theories of testicular descent include the idea that reduced scrotal temperatures function to minimize gamete mutation rates (Short, 1997). Not only are scrotal temperatures typically below body temperature, but because of reduced blood flow to the testicles another hypothesis is that these conditions combine to create a hostile environment that functions to train or test sperm as a means of selecting for a high quality ejaculate (Freeman, 1990).

It has even been suggested that the scrotum evolved as a signaling device or form of sexual ornamentation to promote male social/sexual competition (Portman, 1952). As an extension of this hypothesis, it is interesting to note that scrotal testicles could be related to

what is known as the “handicap hypothesis” (Andersson, 1986; Zahavi and Zahavi, 1997). According to this idea, some exaggerated instances of different sexually dimorphic traits (such as coloration and plumage in birds) have no functional significance other than the fact that they evolved to advertise or signal fitness to members of the opposite sex. So, for example, if a male peacock could survive in spite of all the costs associated with such elaborate, brightly colored, and conspicuous tail feathers, he might be an especially suitable and attractive mate because in order to survive with such a handicap he might have traits that contribute to fitness in other important compensating ways. While this might seem to be a plausible account of scrotal testicles because the handicap they entail carries enormous potential costs, most traits that fit the handicap hypothesis become more exaggerated and more costly over time as a consequence of female choice and competition among males for limited reproductive opportunities. With the possible exception of colored scrota among a few species of primates, there is little evidence that this has been the case.

Effects of Temperature

In species with descended scrotal testicles, a failure to achieve descensus or incomplete/partial descensus during embryological development results in sterility. Infertility as a consequence of undescended testicles (cryptorchid) is often prevented through early surgical descension (Docimo, 1995). It is apparent, however, that the sperm producing capacity of the testicles is not permanently affected by descension failure as the effects on fertility can be reversed even in adulthood (Frankenhuis and Wensing, 1979). Extreme instances of testicular ascension among children can result in descended testicles being drawn back up into the body cavity (Redman, 2005) and if left uncorrected can also result in sterility (Fenton, Woodward, Hudson, and Marschner, 1990).

Not only does the production of viable sperm require that testicular temperatures remain below body temperature (Setchell, 1998), the same is true for the maintenance of viable sperm (Appell, Evans, and Blandy, 1977). Raising ambient temperature from room temperature (20-24°C) to body temperature (37°C) produces a temporary increase in human sperm motility (Birks et al., 1994). But at body temperature sperm only remain mobile and viable for several hours in vitro (Makler, Deutch, Vilensky, and Palti, 1981). When temperature rises much above 37°C sperm motility rapidly diminishes (Makler et al., 1981), and sperm viability is adversely affected.

The Scrotum

There is general consensus that one of the principal functions of the scrotum is to keep testicular temperatures below body temperature as a means of providing an optimal environment for sperm production and storage. Not only is the skin of the scrotal sack thin to promote heat dissipation, the arteries that supply blood to the scrotum are positioned adjacent to the veins taking blood away from the scrotum and function as an additional cooling/heat exchange mechanism. As a consequence of these adaptations average scrotal temperatures in humans are typically 2.5 to 3°C lower than body temperature (Valeri et al., 1993), and spermatogenesis is most efficient at 34°C.

The scrotum, however, is more than a passive cooling device or cold storage mechanism. There is considerable evidence that the scrotum plays an active thermoregulatory role. When ambient temperature rises not only do testicles descend, but conversely when temperatures fall below a certain point the testicles are drawn up toward

the body to conserve heat. The cremasteric muscle, triggered by the cremasteric reflex mediates these moment to moment changes in testicular descent and ascent.

Clothing that restricts testicular descent has been implicated in lowering sperm counts and reduced viability (e.g., Parazzini et al., 1995). Whole body heating or local heating of the testicles through self-induced hyperthermia of the scrotum may also diminish sperm viability (see Setchell, 1998 for a review).

Kumar and Kumar (2008) have extended this analysis of testicular thermoregulation to the question of why human testicles are typically suspended asymmetrically at slightly different levels of descent. In contrast to what would obtain for scrotal symmetry where the testicles were positioned immediately adjacent to each other, with one testicle suspended slightly above (or below) the other there is a corresponding increase in the available surface area subject to heat dissipation and cooling.

Among some marine mammals, such as dolphins and seals with descended but non-scrotal testicles, cooling is achieved as a consequence of the fact that the testicles are located subcutaneously and cooling is also accomplished through venous blood flow from the extremities (see Rommel, Pabst, and McLellan, 1998). A few mammals, however, are testicond; i.e., the testicles are neither scrotal nor descended, and remain deeply embedded inside the body cavity (Werdelin, Nilsson, and Fortelius, 1999). The fact that elephants as a case in point have un-descended testicles with no apparent means of testicular cooling is a point to which we will return.

The Activation Hypothesis

In a review of the literature on the effect of heat on testicular function, Setchell (1998) concludes "...it is not yet possible to explain why spermatogenesis is so sensitive to heat..." (p. 189). Stated another way, why is it that the optimal temperature for sperm production and storage is several degrees below body temperature?

Most sperm are kept inactive by different factors while stored in the epididymis and activation of sperm is initiated as a consequence of exposure to glandular secretions during ejaculation (e.g., Okamura, Tajima, Soejima, Masuda, and Sugita, 1985).

Among mammals with descended scrotal testicles one of the key features that distinguish the male reproductive system from the female is that the female reproductive tract is maintained at body temperature. Although chemical features of the vagina exert an influence, it has been shown that a rise in temperature is sufficient to produce activation of sperm under laboratory conditions. Marin-Briggiler, Tezon, Miranda, and Vazquez-Levin (2002) demonstrated that capacitation of human sperm maintained *in vitro* at 20°C could be produced by raising the temperature to body temperature (37°C). It has also been discovered that capacitated human sperm are chemotactically responsive to follicular fluid. However, both chemotaxis and capacitation are short-lived and only last from 50 min to 4 hrs (Eisenbach, 1999). Raising the temperature of sperm to body temperature also produces a time-bound increase in sperm motility (Makler et al., 1981; Si, 1999).

Why should raising sperm temperature to body temperature be so critical in preparing sperm not only to achieve their fertilization potential, but in enabling sperm to acquire the necessary motility to penetrate the cervix and reach the fallopian tubes? As a primitive adaptation, perhaps descended scrotal testicles evolved as a consequence of an equally primitive algorithm. The chance for sperm to travel up through the female reproductive tract and fertilize an egg is represented by a relatively short, time-bound

opportunity. Once activation and capacitation are achieved, sperm viability is short lived (Eisenbach, 1999).

Recall that sperm motility increases as temperature rises to body temperature. Among mammals with descended scrotal testicles a consistent and universal feature of ejaculation into the female reproductive tract is that it raises the ambient temperature of the ejaculate to body temperature. Thus, the rise to body temperature occasioned by depositing sperm into the vagina functions as a primitive but highly appropriate, situation specific trigger that augments sperm activation and functions to further increase the likelihood that a sufficient number of sperm will be able to pass through the cervix and reach the oviducts to fertilize an egg. In our view, descended scrotal testicles evolved to both capitalize on this copulation/insemination contingent temperature enhancement and function to prevent premature activation of sperm by keeping testicular temperatures below the critical value set by body temperature.

Capacitation and Hyperactivation

After sperm penetrate the cervix and reach the oviduct they appear to be trapped in a reservoir, where capacitation and hyperactivation occur and only a few sperm are released to fertilize an egg when ovulation occurs (Suarez, 2001). Capacitation and hyperactivation are required for sperm to achieve their fertilization potential and penetrate the zona pellucida, and occur as a result of complex biochemical interactions (involving cAMP/PKA and tyrosine kinase/phosphatase signaling pathways and a Ca^{2+} signaling pathway) that come into play during the time sperm are trapped in the oviduct (Marquez and Suarez, 2004).

Capacitation involves a series of structural changes in the membrane of the spermatozoa, culminating in the acrosome reaction, which allows the sperm to bind to the oocyte. As sperm capacitate, there is also a concomitant change in sperm motility resulting in hyperactivation. This is characterized by a dramatic increase in flagellar bend amplitude and beat asymmetry and a curved and highly convoluted pattern of movement. Hyperactivation is an important part of capacitation, as it is thought to be the mechanism by which the sperm are able to detach from the thick mucus of the oviduct and propel themselves vigorously towards the egg (Suarez and Ho, 2003). Thus, there appear to be several phases of activation: glandular activation during ejaculation, temperature induced activation upon the release of semen into the vagina, and subsequent capacitation and hyperactivation that occur after sperm reach the oviduct.

The timing of hyperactivation and the acrosome reaction are critical to successful fertilization. A premature acrosome reaction is believed to be a cause of male infertility (de Lamirande, Leclerc, and Gagnon, 1997). The effect of scrotal temperature on the timing of this reaction may be important. Si (1999) has shown that hyperactivity can be induced in sperm incubated at body temperature, and effectively stopped when the temperature drops. It is suggested that as temperature increases, so does lipid diffusibility in the sperm plasma membrane, which may alter membrane permeability and subsequent membrane-bound enzyme reactions. These reactions include signaling pathways for essential ions such as calcium bicarbonate, which are responsible for hyperactivation and the acrosome reaction.

Due to the effect of temperature, sperm created and maintained at body temperature would be in a constant state of activation. By depleting metabolic stores this would create significant obstacles to successful fertilization. There is evidence, for example, that sperm

that become hyperactivated prematurely cannot penetrate the cervix (Zinaman et al., 1989).

According to Suarez and Pacey (2006) the female reproductive tract plays a role in ensuring that only sperm with normal morphology and the capacity for vigorous movement reach the oviduct. Of the several hundred million sperm contained in a single human ejaculate only a few thousand penetrate the cervix and have a chance to reach the fallopian tubes. Sperm that achieve advanced stages of capacitation before reaching the cervix lack the necessary energy reserves to successfully navigate the remainder of the female reproductive tract, and may even be prevented from advancing towards the oviduct by the female's own reproductive defenses.

Corollary Adaptations

Several features of mammalian reproduction are consistent with our hypothesis. For instance, sexual arousal in males activates the cremasteric reflex and draws the testicles up close to the body (e.g., Johnson, Kolodny, and Masters, 1994). In addition to minimizing testicular vulnerability occasioned by thrusting, the cremasteric reflex may also function to raise testicular temperature and the corresponding temperature in the caudal portion of the epididymis to activate sperm as they move into the vas deferens for pending ejaculation into the vagina.

Humans show a peculiar cross-cultural nocturnal copulation bias (Ford and Beach, 1951). Having sex at night could promote any number of adaptive outcomes. Nocturnal copulation may have functioned to minimize vulnerability to predation and/or detection and interference from rival males, accommodate clandestine copulations, promote incest avoidance, and even enhance the likelihood of sperm retention by postponing the resumption of an upright posture by the female during the post ejaculatory period (Gallup and Burch, 2006). Patterns of nocturnal copulation may also be a circadian adaptation to descended scrotal testicles. When daytime temperatures approach or exceed body temperature which is common in equatorial regions, testicular adjustments would fail to function favorably. However, in the evening and at night when ambient temperatures fall back below body temperature, more effective scrotal temperature adjustments are reinstated and the likelihood of optimal sperm viability may increase. Even though sperm maturation extends over 70 days, it would nonetheless be a relatively simple matter to collect semen samples from males living under such circumstances at different times during the day and night to see if there were corresponding circadian differences in sperm viability that match the differences in ambient temperature and the likelihood of copulation. Recall that the activation of sperm that occurs at body temperature can be reversed by a reduction in temperature (Si, 1999).

Not unrelated to the activation effect is the fact that many mammalian females show a slight increase in basal body temperature at about the time of ovulation. Thus, ovulating females of different species are often described as being "in heat." Indeed, monitoring changes in basal body temperature in human females has been used to identify periods of maximum fertility for purposes of increasing or decreasing the likelihood of impregnation through appropriate timing of insemination (McCarthy and Rockette, 1986). This small but consistent rise in basal body temperature at around the point of ovulation may be what originally set the stage as a temperature trigger for the time-bound activation of sperm, and the subsequent evolution of descended scrotal testicles may simply serve to further accentuate this difference.

Thus, for most mammalian species the conditional rise in temperature that occurs when sperm are deposited in the vagina may represent an ancient algorithm that evolved to provide a contextually appropriate trigger to promote further activation of sperm. Even among testicond mammals it is possible that this mechanism may still be intact. Perhaps the threshold for activation in these species has simply been reset to a higher value. That is, among mammals with undescended testicles maintained at body temperature, activation of sperm may require an increase that exceeds body temperature. Indeed, in such species it may be that the increase in female basal body temperature that occurs during ovulation may be even more accentuated than in other mammals, as a means of triggering activation and compensating for the fact that testicular temperatures are maintained at body temperature. Thus the primitive algorithm based on the activation effect with a rise in temperature may continue to function even in testicond mammals as consequence of a more substantial ovulation-induced rise in female basal body temperature (for an alternative account as it applies to elephants see Werdelin, Nilsson, and Fortelius, 1999).

Adaptations to Testicular Vulnerability

Any account of descended scrotal testicles must also address the enormous potential costs of having the testicles located outside the body cavity where they are left virtually unprotected and especially vulnerable to insult and damage. To be consistent with evolutionary theory the potential costs of scrotal testicles would have to be offset not only by compensating benefits (e.g., sperm activation upon insemination), but one would also expect to find corresponding adaptations that function to minimize or negate these costs.

One especially salient psychological adaptation to descended scrotal testicles is the propensity among males to experience intense, often excruciating pain in response to testicular insults. Testicles have an unusually low pain threshold; i.e., the magnitude of insult needed to trigger the experience of testicular pain is often innocuous when applied to many other parts of the body. Pain is highly adaptive. Pain evolved to 1) promote the avoidance of situations that are not in an organism's best biological interests, and 2) to enable organisms to learn to refrain from engaging in behaviors that may not be adaptive (Thornhill and Thornhill, 1991).

From an evolutionary perspective, one would expect the magnitude of pain experienced by insults of the same intensity to be proportional to the extent to which damage to affected areas might detract from the likelihood of reproduction (see Suarez and Gallup, 1985; Thornhill and Thornhill, 1991). Based on this logic, variation in pain sensitivity at different body sites (e.g., eyes, testicles, legs, back) ought to be related to the vulnerability and importance these areas serve in promoting reproductive success. Given the role the testicles play in gamete production, it should come as no surprise that scrotal testicles are especially susceptible to pain. The experience of pain in response to testicular insult prompts males to avoid situations that led to testicular pain in the past, and provides a powerful source of motivation to learn to refrain from engaging in behaviors that might lead to testicular damage. Other things being equal, males that could learn to protect their gonads probably left more descendants, and this would have been particularly true of those with scrotal testicles.

As noted previously, the cremasteric reflex participates in testicular thermoregulation by raising or lowering the testicles within the scrotum, and thereby adjusts the proximity of the testicles to the body to compensate for changes in ambient

temperature. The cremasteric reflex also pulls the testicles up close to the body under conditions of sexual arousal, which we contend may serve to prime the activation of sperm prior to ejaculation.

The cremasteric reflex also appears to function in several other ways to compensate for the vulnerability of descended scrotal testicles and to minimize testicular damage. Fear and the threat of danger have been shown to promote reflexive contractions of the cremasteric muscle and cause the testicles to be drawn up against the body where they are less vulnerable (Fenton et al., 1990). As further evidence that this response evolved to accommodate problems posed by having the testicles located outside the body cavity, pain is another trigger for the cremasteric reflex and the application of a pin prick to the inner thigh to assess the absence of this response has been used to index spinal anesthesia in patients being prepped for surgery (Okuda, Mishio, Kitajima, and Asai, 2000). Thus, there are at least four conditions that activate the cremasteric reflex: activation in response to changes in ambient temperature and sexual arousal both evolved to regulate scrotal/testicular temperature, whereas activation as a consequence of fear and pain are adaptations that function to minimize testicular injury/insult.

Conclusion

Consistent with evidence that descended scrotal testicles are a primitive adaptation among mammals, we advance the hypothesis that this peculiar gonadal arrangement evolved to insure consistent differences in temperature between the male and female reproductive tracts. Because scrotal testicles are maintained at several degrees below body temperature, sperm are kept in an inactive, dormant state. Whereas, upon ejaculation into the vagina there is a conditional rise in temperature that combines with other mechanisms that trigger the activation of sperm and function to increase the likelihood that sperm will reach and penetrate the cervix. Several features of mammalian reproduction are consistent with this hypothesis, including predicted temperature changes in sperm motility, behaviors that function to raise scrotal temperature as a means of preparing sperm for release into the vagina, and behavioral adaptations that compensate for testicular vulnerability.

Acknowledgements: The authors thank Glenn Sanders, Jennifer Davis, Steven Platek, Todd Shackelford, Daniel White, Lars Werdelin, Josh Plotnik, and Andrew Gallup for helpful comments on earlier drafts of this paper.

Received 06 August 2009; Revision submitted 16 October 2009; Accepted 24 October 2009

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