Is the male reproductive system poorly designed?

Jerry Bergman

One of the latest proofs of human evolution is the poor design claim, namely that an intelligent Creator would not design some human body part in a certain way. An example is the human male reproductive system, which Rowe listed as number four in his list of the top 10 design flaws in the human body.[1] The human male reproductive system poor design claim focuses on the view that "if testicles were designed", then why didn't God "protect them better. Couldn't the Designer have put them inside the body, or encased them in bone" like the brain which is surrounded by a hard skull?[2]

Concluding that a body structure is poorly designed, as Oxford University Ph.D. Professor Hafer claims, instead of asking *why* the existing design exists, is a science stopper. The 'why' question motivates research into the reasons for the design. When this approach was applied to the human appendix, the tonsils, the backward retina, and the many putative other examples of supposed poor design, good reasons for the existing designs were found in all cases. The same is true of the male reproductive system.

Hafer explained that when she was looking for new approaches to refute Intelligent Design, she knew she "had a winner when … in the middle of an Anatomy and Physiology lecture" she concluded that the male reproduction system "is a great first argument against ID".[3] She believed that she also had a good "political-style argument" against ID.[4] Her main argument is that because male testicles are outside of the body, they are prone to injury. She adds that in many animals, including cold-blooded reptiles, they are located inside the body where they are fully protected.

The reasons for the design

Male testicles exist outside of the body in humans and most mammals for several important reasons, including effective regulation of scrotal temperature for optimal spermatogenesis development. Another reason is to keep sperm relatively inactive until they enter the warm confines of the female reproductive system.[5] Even just a few degrees above the optimal temperature is detrimental to both sperm production, specifically in the later stages of spermatogenesis, and sperm maturation.[6]

A low ambient temperature is essential for normal spermatogenesis in humans and *most* mammals because the enzymes required for the process are denatured if their temperature is not finely regulated. One study in mice found temperatures of 37°C or higher caused "a significant reduction in the percentage of motile sperm", producing an increase in the number of spermatozoa with plasma membrane damage.[7] The mammal exceptions include monotremes, mammals that lay eggs instead of giving birth to live young, and have intra-abdominal testes. Also, some placental mammals, such as insectivores (shrews, hedgehogs, and moles), plus elephants and hippopotamuses, all have intra-abdominal testicles.[8] One factor is externalized testes are found only in certain mammals whose lifestyle involves jumping, leaping, or galloping. In large animals with this lifestyle this behaviour would be expected to put great pressure on the testicles and even expel their contents by creating concussive hydrostatic rises in peritoneal pressure.[9]

Compared to core body temperature, the average temperature drop achieved in the current design architecture is ideal. The sperm production system is maintained at a temperature very close to 4˚C cooler than the normal body temperature of 37°C.[10] An increase in temperature by as little as 2°C adversely affects sperm formation. The result of this 2˚C increase in humans includes a lower sperm count, and a significant increase in the number of abnormal sperm.[11]

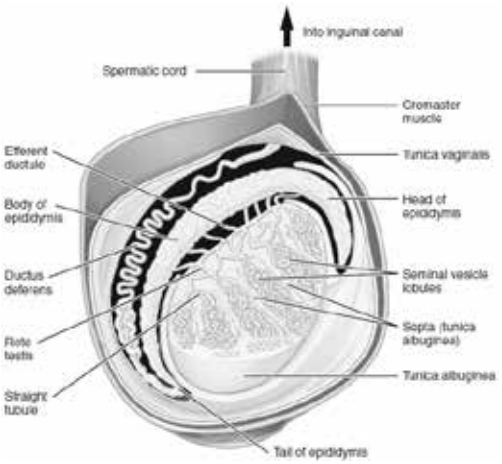

Figure 1. Diagram of a male testicle showing its internal structure (From Wikimedia Commons)

If the testes were inside the body, the enzymes sperm require to be healthy would be denatured in a matter of hours. New sperm would have to constantly be produced to allow humans to be fertile year-round, as is normal for humans. This issue would not be a concern for most animals that are fertile only during very short windows each year.

Several complex mechanisms exist to ensure that the 4˚C difference is maintained at 37˚C down to 33˚C. When testicle temperature drops below 33˚C, a complex feedback system causes the cremaster muscle that surrounds the testicles to contract, which moves the testicles closer to the warm 37˚C body in order to compensate for the heat loss.[12] When their temperature rises above 33˚C, the cremaster muscle relaxes, allowing the testicles to move away from the body. This insures that the ideal male reproductive system temperature is maintained within a very narrow tolerance of the 33˚C ideal.

Their 33˚C temperature is also maintained by increasing or decreasing the surface area of the tissue surrounding the testicles, the scrotum, allowing faster or slower dissipation of their heat. It does this by expanding like a wrinkled balloon does when air is blown into it. Furthermore, scrotal skin is very thin, allowing the testes to easily lose heat into the surrounding environment. The air circulating around the scrotum sack further helps to facilitate the cooling of the scrotal skin, in turn helping to cool the sperm development system.

Furthermore, to maintain the proper temperature, the arteries carrying blood into the scrotum run alongside the veins that carry blood away. This sophisticated heat-exchanger mechanism lowers the temperature of the blood supply travelling to the testicles.[13] The warm arterial blood coming from the abdomen loses heat to the cooler venous blood coming away from the testes. The result is that the blood is cooled slightly before even entering the scrotum. For these reasons, the existing system is an excellent design, well known to engineers as *counter-current exchange*, to maintain optimal spermatogenesis temperature control. This design is widespread in biological systems.

Year-round fertility

One major reason for the rigid temperature regulation is because humans are fertile throughout the entire year, and virtually all animals with internal testicles, including all cold-blooded vertebrates and birds, are not. Most animals need to be fertile only during a very short period of time during their mating season. This is often when outdoor temperature allows maintenance of the proper temperature for spermatogenesis, such as is the case for reptiles. In harmony with this observation, Freeman found that "taxa with internal testes produce large volumes of low-quality sperm while taxa with scrotal testes produce smaller volumes of higher-quality sperm".[14]

Another reason for the rigid temperature regulation is that sperm have a very short lifespan and must be stored at lower than body temperature to keep them dormant longer. Sperm are stored in the epididymis where they mature, and the warmth of the female reproductive system serves to help activate them. If the testicles were located inside of the male body, the sperm would be activated much sooner, and thus, given their short lifespan, measured in hours, large numbers of sperm would die before they could even enter the vagina.

Evidence for the temperature effect includes the fact that semen quality is lower in the warm summer months compared with the cold winter months. The semen volume does not change significantly, but the total sperm count falls in the summer compared to winter, especially in the northern hemisphere during the hottest summer months of July and August.[15]

Several protection designs

Several designs reduce the likelihood of testicular injury, including the left testicle usually hanging lower than the right one and, as a result, pressure causes one to slip past the other without pain or injury. Each testicle is housed in a strong fibrous outer

covering called the tunica albuginea, and an effective lubrication system allows the slippage to occur without pain or problems. Injury is rare, and the main source of injury is in sports, which is why it is recommended that sport participants always use protective equipment, such as a jockstrap or hard cup, while playing.

Cryptorchidism

Another reason for the existing placement of the human male reproductive system outside of the body is that the

> "… postpartum testicle is designed to function at this lower temperature. Failure of the testicles to descend into the scrotum, called cryptorchidism, causes an increased risk of malignancy and other major health problems. The process of testicle descent is also both complex and poorly understood."[16]

Failure of the testicles to descend following birth leads to progressive abnormality in both the biochemistry and physiology of the testis, often causing infertility.[17]

One example is that the abnormal biochemistry caused by descent failure interferes with many of the necessary reproductive system developments. Examples including the transformation of neonatal gonocytes into type A spermatogenesis, a step required to produce viable sperm.[18] This is one reason why failure of the testicles to descend is a major reason for male infertility.

Evolution of the scrotum?

How the many complex male temperature regulative system parts could have evolved can only be speculated, and not be based on observation and science.[19] Consequently, evolutionists must produce many just-so stories in an attempt to explain their existence and function.[20] In short, a literature review found that "all of the current hypotheses regarding the origin and evolution of the scrotum" and external testicles are seriously problematic. Reasons include, assuming external testicle evolution from lower life forms with internal reproductive organs is problematic because it is "why the scrotum has been lost in so many groups, that should be explained".[21] The authors even speculate that the scrotum may have evolved before mammals did

> "… in concert with the evolution of endothermy in the mammalian lineage, and that the scrotum has been lost in many groups because descent in many respects is a costly process that will be lost in mammal lineages as soon as an alternative solution to the problem of the temperature sensitivity of spermatogenesis is available."[22]

Conclusions

In conclusion, clear evidence exists that year-round reproductive cycles, plus the requirement that human sperm must be kept close to a constant temperature of 4˚C below that of the core body temperature, effectively explains the existing design of testicles. Men who have uncorrected non-descended testicles are usually infertile and prone to many other health problems, including cancer. In short, the existing complex design is required for many reasons, including fertility and health reasons. It is, therefore, clear that Hafer's poor design claim, along with those of other evolutionists, is grossly irresponsible.

References

1. Rowe, C., The 10 Design Flaws in the Human Body, *Nautilus*, 24 March 2016, p. 2.
2. Hafer, A., *The Not-So-Intelligent Designer: Why evolution explains the human body and Intelligent Design does not*, Cascade Books, Eugene, OR, p. 5, 2016.
3. Hafer, A., No data required: why intelligent design is not science, *The American Biology Teacher* **77**(7):507–513, 2015; Hafer, ref. 2 p. 2.
4. Hafer, ref. 2, pp. 1–2.
5. Wechalekar, H., Setchell, B.P., Peirce, E.J., Ricci, M., Leigh, C., and Breed, W.G., Whole-body heat exposure induces membrane changes in spermatozoa from the cauda epididymis of laboratory mice, *Asian J. Andrology* **12**(4): 591–598, 2010.
6. Setchell, B.P., The effects of heat on the testes of mammals, *Animal Reproduction* **3**(2):81–91, 2006.
7. Wechalekar *et al.*, ref. 5, pp. 591–592.
8. Sodera, V., *One Small Speck to Man—The evolution myth, 2nd edn*, Vija Sodera Productions, pp. 109–110, 2009.
9. Chance, M.R.A., Reason for externalization of the testis of mammals, *J. Zoology* **239**(4): 691–695, 1996.
10. Hutson, J.M. and Clarke, M., Current Management of the Undescended Testicle, *Seminars in Pediatric Surgery* **16**:64–70, 2007; p. 65.
11. Ahmad, G. Moinard, N., Esquerre-Lamare, C., Mieusset, R., and Bujan, L., Mild induced testicular and epididymal hyperthermia alters sperm chromatin integrity in men, *Fertility and Sterility* **97**(3):546–553, 2012.
12. Van Niekerk, E., Vas deferens—refuting 'bad design' arguments, *J. Creation* **26**(3):60–67, 2012; creation.com/vas-deferens
13. Wechalekar, *et al.*, ref. 5, p. 591.
14. Freeman, S., The evolution of the scrotum: a new hypothesis, *J. Theoretical Biology* **145**:429–445, 1990; p. 429.
15. Chen, Z., Toth, T., Godfrey-Bailey, L., Mercedat, N., Schiff, I., and Hauser, R., Seasonal variation and age-related changes in human semen parameters, *J. Andrology* **24**(2):226–231, 2003.
16. Hutson and Clarke, ref. 10, p. 65.
17. Chung, E. and Brock, G., Cryptorchidism and its impact on male fertility: a state of art review of current literature, *Canadian Urological Association J.* **5**(3):210–214, 2011.
18. Hutson and Clarke, ref. 10, p. 66.
19. Gallup, G.G., Finn, M.M., and Sammis, B., On the origin of descended scrotal testicles: the activation hypothesis, *Evolutionary Psychology* **7**(4):517–526, 2009; Freeman, S., The evolution of the scrotum: a new hypothesis, *J. Theoretical Biology* **145**:429–445, 1990.
20. Heyns, C.F. and Hutson, J.M., Historical review of theories on testicular descent, *J. Urology* **153**:754–767, 1995; Ivell, R., Lifestyle impact and the biology of the human scrotum, *Reproductive Biology and Endocrinology* **5**(15):1–8, 2007.
21. Werdelin, L. and Nilsonne, A., The evolution of the scrotum and testicular descent in mammals, *J. Theoretical Biology* **196**(1):61–72, 1999; pp. 61–62.
22. Werdelin and Nilsonne, ref. 21, p. 61.