Sperm Production and Variance in Sperm Quality

by

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Abstract

An unusually high level of inter- and intraspecific variability in spermatozoa has been well documented. However, recent evidence indicates that the level of variation within spermatozoa differs markedly across taxa. In particular, it appears that the variability in spermatozoa tends to decrease across species as the risk of sperm competition increases.

In this thesis, I present a model that explains how variability in spermatozoa may arise due to errors made during the sperm production process. In doing so, I also provide an explanation for why variability in sperm traits tends to decrease as the level of sperm competition experienced by males of a given species increases.

The model presented in this study provides a novel perspective on spermatozoa and their production. While many sperm traits are thought to be selected upon, I suggest that variability in spermatozoa may also be the result of evolutionary forces such as sperm competition. Variability in spermatozoa, then, can be adaptive and can represent an optimal reproductive strategy.
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Chapter 1

Introduction

One of the most surprising features of reproductive biology is how variable the fertility of a male’s spermatozoa can be. Spermatozoa are among the most varied cells found in animals ([5, 9, 10, 15, 19, 21, 23, 25, 29, 31, 35]) and spermatozoa from a single male can range from completely nonfunctional to very fertile. For example, more than half of the sperm in a normal human male’s ejaculate may be abnormally shaped or lack motility entirely([19, 31]). Among captive cheetahs (Acinonyx jubatus), males often produce ejaculates that contain more than 70% abnormally shaped sperm ([25]). In fact, some researchers ([15, 29]) have even suggested that the female reproductive tract exerts selection upon sperm such that only the best quality sperm manage to fertilize ova.

Different males within a single species often vary in their sperm fertility as well([8, 36]). The World Health Organization, for example, classifies human males into three categories based upon various sperm quality traits including the motility and acrosome activity of sperm and the proportion of sperm in an ejaculate that are abnormally formed ([31]). Among green sea urchins (Stronglyocentrotus droebachiensis), significant differences in sperm length and morphology were found both between individuals of the same population and between different populations ([28]). Surprisingly, even the size of the genome
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contained in sperm can vary between individuals of the same species; considerable variation in the size of the genome within sperm was found in several species of flour-beetles (Tribolium), for example ([1]).

Unfortunately, we have yet to determine the cause of the variation in spermatozoa produced within and among males adopting the same reproductive tactic. Several studies ([6, 21, 33]) implicate a conflict between haploid and diploid optima in sperm traits as giving rise to the variation within males. Here, I adopt a framework similar to that of Cohen and other authors ([9, 10, 13, 14, 15, 20, 23, 29]) in which the variability in sperm traits arises because of errors in their manufacturing process.

1.1 Source of Variation in Sperm Fertility

When a male produces spermatozoa, he must transcribe several genes in his genome that control, or are involved in, sperm production. The transcription of these genes determines all aspects of the sperm cells produced, and so determines the ability of a given sperm cell to fertilize an ovum. For example, some of these genes impact sperm morphology, which effects the ability of a sperm cell to swim towards an ovum. These genes can also impact other sperm traits such as mitochondria content (or energetic content), acrosome biochemistry, or the ability to detect chemotactic signals from ova.

Since there are usually numerous spermatogenic cells in a male’s testes, and large numbers of sperm are usually produced at a single time, there are many opportunities for transcription mistakes to be made during sperm production. Different transcription mistakes made during spermatogenesis will affect sperm function to different degrees, but, in general, mistakes will only decrease the function of sperm. So, mistakes can be thought of as phenotypic defects in sperm that have varying effects on sperm fertility. Since mistakes, or defects, occur randomly, different sperm produced by a single male will have different combinations of these defects, even though all of the sperm produced by that male are based
upon the same genome. Hence, there will almost always be some variability in the fertility of the sperm produced in a male’s ejaculate.

1.2 Reducing Variation

Recent evidence has shown that the level of variation in sperm fertility can differ widely between species ([8, 9, 10, 23, 36]). In particular, variation in sperm quality tends to decrease as the level of sperm competition increases. For example, several studies have noted a marked decrease in variation of sperm morphology across species in passerine birds as both extrapair paternity and testes size increased ([10, 23]). Thus, species that do not experience significant sperm competition appear to produce the most variable sperm. Interestingly, ejaculates produced by captive cheetahs often contain a very high proportion of malformed sperm. As these cheetahs live in low population densities naturally, it is unlikely that they experience sperm competition ([25]). Among Atlantic Salmon, males that employ a “sneaker” strategy to mating (and so, will almost always experience sperm competition when spawning) produce very high quality sperm, while dominant males (who experience less sperm competition) produce highly variable, poorer quality sperm ([35]). It appears, then, that selection can act to reduce the variation in sperm traits, while the lack of selection allows sperm traits to increase in variability.

If transcription mistakes cause defects in sperm that reduce their fertility, then any male that could reduce or correct some of these mistakes would benefit by an increase in the fertility of his sperm. Under low levels of sperm competition, this benefit may not be very important as males will probably fertilize the ova of any female that they mate with (as long as enough of their sperm function properly). However, as the risk of sperm competition increases, a male would benefit more from increasing the fertility of his sperm, because, under sperm competition, a male’s fertilization success depends upon the ability of his sperm to out-compete the sperm of other males. Thus, we would expect that the number
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of defects in sperm would decrease as the risk of sperm competition increases. Effectively, selection should favour a decrease in the variability in sperm fertility, especially when there is a high risk of sperm competition.

It is important to consider how defects in sperm can be prevented. As mentioned above, mistakes occurring during transcription can cause these defects. Several error checking mechanisms can correct transcriptional errors ([4, 22, 24]). For example, RNA polymerase II is thought to pause during transcription to mediate the repair of transcriptional mistakes ([4]). Many transcription mistakes probably occur during spermatogenesis because huge numbers of sperm are produced very quickly. If a male produced sperm more slowly, he may be able to reduce the number of mistakes that occur during spermatogenesis. Thus, by checking for errors, or by producing sperm more slowly, it is plausible that a male could reduce the number of defects, and the variability, in his spermatozoa. Note that both of these strategies for preventing transcription mistakes involve an increase in the time spent producing sperm. So, males that spend longer producing sperm might be able to decrease the variation in those spermatozoa, whether they do so by error checking, or by producing spermatozoa more carefully.

The purpose of this study is to develop a theoretical model that explains why there is more variation in sperm traits in some species than in others. In particular, I attempt to provide an explanation for the general trend that increasing sperm competition results in decreased variability in sperm traits. To do so, I adopt a framework where variation in spermatozoa arises due to errors in the sperm production process, and these errors may be prevented by error-checking or by taking longer to produce each spermatozoon.
Chapter 2

The Model

I consider defective sperm to be the products of an imperfect transcriptional process in which mistakes lead to a decrease in fertility. Since these mistakes occur stochastically, some sperm in a male’s ejaculate may be free of defects and of very high fertility, but other sperm within the same ejaculate may contain so many defects that they do not function at all.

2.1 The Effect of Defects in Sperm Phenotypes

Different transcriptional mistakes will have different effects. For example, a transcriptional mistake may have no effect at all on fertility if it has only a small effect on the sperm phenotype. Other mistakes may render a sperm cell completely nonfunctional. Because it is impossible to consider the effect of every transcriptional mistake, I consider the average effect of mistakes in transcription. In other words, I consider the effective number of phenotypic defects in a sperm cell as the expected effect of a given number of phenotypic defects.

By considering only the effective number of phenotypic defects in a particular sperm cell, we can define $m$ as the maximum effective number of such defects that a sperm cell can contain when it just begins to cease functioning. In other words, sperm that contain $m$
effective phenotypic defects are those that contain so many defects, or drastic enough defects, that the spermatozoa do not function at all with respect to fertilization. So, the maximum number of effective phenotypic defects that a sperm may contain and still function is \( m - 1 \). Conversely, sperm that contain 0 effective phenotypic defects are those that are defect-free or contain only phenotypic defects that have no effect on fertility. So, maximum fertility is obtained by sperm with 0 effective phenotypic defects, and zero fertility is obtained by sperm that contain \( m \) or more effective phenotypic defects.

### 2.2 Sperm Fertility

I define the fertility, \( f \), of an individual sperm cell as the relative probability that it will reach an ovum and be fully equipped to fertilize it. Thus, the fertility of a sperm cell represents its ability to reach and fertilize an ovum relative to that of a defect-free sperm cell. This fertility depends upon the effective number of phenotypic defects that are contained in a sperm cell. In other words, the fertility of a sperm cell is a decreasing function of the number of phenotypic defects accrued during the production of that sperm.

To contrast sperm production strategies, it is easier to consider the (effective) number of phenotypic defects that are corrected, or prevented, in a sperm cell than the (effective) number of phenotypic defects actually contained within that sperm cell. To do so, I consider the number of phenotypic defects avoided, \( k \), where \( k \in [0, m] \). Then, the effective number of phenotypic defects that a spermatozoa contains is \( m - k \), and \( k \) can be considered an index of sperm quality. The fertility of a sperm cell is a decreasing function of \( m - k \), so must be an increasing function of \( k \), \( f(k) \). I consider several \( f(k) \) functions, as described below.
2.2.1 Linear Fertility Function

A simple form of $f(k)$ is

$$f(k) = \frac{k}{m} \quad (2.1)$$

This function is analytically convenient, but it also offers a simple interpretation of the effect of phenotypic defects on sperm fertility. If we consider an internally fertilizing species, $f(k)$ can be thought of as the relative distance per unit of time that a sperm cell can travel in the female reproductive tract compared to the maximum possible distance that it could travel if it contained no phenotypic defects ($f(m) = \frac{m}{m} = 1$). So, the sperm that travel further (or faster) will be more likely to reach an ova than those that can only travel a short distance in a single time step.

2.2.2 Nonlinear Fertility Function

Externally-fertilizing (spawning) species are defined here as those that simply release their gametes into an aqueous environment instead of releasing sperm into the female’s reproductive tract. If we consider the relative distance that a sperm cell can travel, as in the previous fertility function, (2.1), we can determine the relative size of the sphere that contains all of the locations that a sperm cell can reach by traveling in any direction. Fertility, then, can be represented by

$$f(k) = \left(\frac{k}{m}\right)^3 \quad (2.2)$$

which is shown in figure 2.1

2.3 Male Fertility

I define a male’s fertility as the probability that at least one spermatozoon from an ejaculate produced by that male fertilizes an egg. This probability depends upon the number of effective phenotypic defects in every sperm cell that a male produces in a single ejaculate.
CHAPTER 2. THE MODEL

Figure 2.1: The probability of a spermatozoon fertilizing an egg depends upon the number of phenotypic defects avoided in the production of that spermatozoon. (Evaluated at \( m = 100 \))

In other words, this probability depends upon the distribution of the effective phenotypic defects in the spermatozoa from a male's ejaculate. Male fertility, then, is given as

\[
\text{Male Fertility} = E[P(\text{at least one sperm fertilizes an egg})]

= 1 - E[P(\text{no sperm fertilize an egg})]

= 1 - E[(1 - f(0))^{n_0} \times (1 - f(1))^{n_1} \times ... \times (1 - f(m))^{n_m}]
\]

where \( n(k) \) is the number of sperm that a male produces of type \( k \) (or with \( m - k \) mistakes).

Now, each of these \( n(k) \) terms are random variables that represent the number of sperm of type \( k \) that are produced where \( n(0) + n(1) + ... + n(m) = n \). In other words, the \( n(k) \) terms have a multinomial distribution with parameters \( n, P(0), ..., P(m) \) where \( P(k) \) is the probability that an individual spermatozoon is of type \( k \). Using the Multinomial Theorem,
we get

\[
\text{Male Fertility} = 1 - \sum_{n_0,\ldots,n_m=n}^n \frac{n!}{n_0!\cdots n_m!} (1 - f(0))^{n_0} \cdots (1 - f(m))^{n_m} \]

\[
= 1 - (1 - \overline{f(k)})^n
\]

(2.3)

where \(\overline{f(k)}\) is the average male fertility, and \(n\) is the total number of sperm produced in a single ejaculate.

### 2.4 Increasing Fertility

As mentioned in section 1.2, a male can correct or prevent transcriptional mistakes during sperm production by spending time checking for errors or by spending longer producing sperm. I denote the amount of time that a male spends increasing the fertility of his spermatozoa by \(\tau \in [0, \infty)\).

If a male spends no time producing sperm (\(\tau = 0\)) then his spermatozoa will contain the maximum number of phenotypic defects needed to make his sperm infertile, \(m\). However, by increasing \(\tau\), a male may correct potential phenotypic defects independently during sperm production at a rate \(\lambda\). Thus, if a male devotes \(\Delta t\) extra time to error-checking during sperm production, he will correct (on average)

\[
\lambda \ast (m - k) \ast \Delta t
\]

more phenotypic defects in his sperm that already contain \(m - k\) such potential defects.

Alternatively, increasing \(\tau\) can be thought of as increasing the amount of time that a male spends producing his sperm. Then, we can think of \(\lambda\) as being the independent rate at which a male avoids phenotypic defects in his sperm. So, by devoting \(\Delta t\) more time to sperm production, he will avoid (on average) \(\lambda \ast (m - k) \ast \Delta t\) more phenotypic defects.

Note that, by this characterization, there are diminishing returns to devoting time towards increasing the fertility of sperm. When little time has been devoted to error-checking
(or sperm production), many errors are present in sperm, so error-checking (or producing sperm more slowly) is beneficial. When a male already spends a large amount of time checking for errors during sperm production (or just producing sperm more slowly and carefully), his sperm will contain very few phenotypic defects, so he will benefit much less from increasing the time he spends on error-checking (or on sperm production).

2.5 The Distribution of Sperm Fertility

By increasing the amount of time that he spends on sperm production ($\tau$), a male alters the probability that any given sperm cell will contain any (effective) number, $m - k$, of phenotypic defects. In other words, by spending more time on sperm production, a male will change the distribution of the effective number of phenotypic defects in his sperm. To see this, I derive an expression for the probability that an individual sperm cell will contain $m - k$ phenotypic defects after some time, $t$, of error-checking (or sperm production).

Let $P(k, t)$ represent the probability that a randomly chosen sperm from a male’s ejaculate contains $m - k$ phenotypic defects at time $t$ in the sperm production process. As mentioned in the previous section (2.4), if a male devotes no time to sperm production, his sperm will contain $m$ phenotypic defects. So

$$P(0, 0) = 1 \quad (2.5)$$

If, instead, a male devotes $t + \Delta t$ time to sperm production, the probability that a randomly drawn sperm from his ejaculate will contain $m$ phenotypic defects is

$$P(0, t + \Delta t) = P(0, t) \ast P(\text{no potential defects are corrected (or avoided) in } \Delta t)$$

$$= P(0, t) \ast (1 - \lambda * m * \Delta t) \quad (2.6)$$

Using this expression we can determine the probability that an individual sperm cell has $m$ phenotypic defects at any time $t$ in the sperm production process:

$$P(0, t) = e^{-\lambda m \ast t} \quad (2.7)$$
Likewise, we can derive the probability that a sperm cell contains any number of phenotypic defects $m - k$ at time $t$ in the sperm production process:

\[
P(k, t) = \binom{m}{k} \times (e^{-\lambda t})^{m-k} \times (1 - e^{-\lambda t})^k
\]  
(2.8)

(see appendix A for complete derivation).

In particular, we are interested in the probability that a sperm cell contains some number of phenotypic defects $(m - k)$ at time $\tau$, when sperm production is complete. This is given by

\[
P(k) = \binom{m}{k} \times (e^{-\lambda \tau})^{m-k} \times (1 - e^{-\lambda \tau})^k
\]  
(2.9)

This distribution can be easily intuited if we make a few key observations. First, notice that in order for an individual spermatozoon to be of type $k$ (to have $m - k$ phenotypic defects), $k$ potential phenotypic defects must have been corrected (or avoided). Since each phenotypic defect is corrected with probability $1 - e^{-\lambda \tau}$ in $\tau$ units of time, and since this correction is independent for each phenotypic defect, we can think of each phenotypic defect corrected as a success in $m$ Bernoulli trials. Thus, the number of phenotypic defects in an individual spermatozoa will be random variable from the Binomial distribution with parameters $m$ and $1 - e^{-\lambda \tau}$. Using the Binomial Theorem, then, the probability that a spermatozoa is of type $k$ is just (2.9).

Usually, when measures of sperm fertility are plotted, they appear to be Normally distributed (e.g., in deer [26], salmon [34], fowl [17], and humans [7, 37]). However, the Binomial distribution can be approximated using the Normal distribution for large enough values of $m$. As $m$ here represents the number of (effective) phenotypic defects that a sperm could possibly hold, it will always be relatively large. Thus, the Binomial distribution is also a good representation of most measures of sperm fertility, and so, is a reasonable choice for the distribution of sperm types.

The shape of this distribution depends upon the parameters involved: $\tau$, $\lambda$, and $m$. I discuss the dependence of the distribution on these parameters below.
2.5.1 Differences Due to Changes in $\lambda \ast \tau$

Since $\lambda$ and $\tau$ only appear as a product in the probability distribution (2.9), I only consider the effect of the product $\lambda \ast \tau$ instead of considering $\lambda$ and $\tau$ separately. $\lambda$ represents the rate at which a male can correct (or avoid) potential phenotypic defects in his sperm, and $\tau$ represents how long he spends doing this correction; so the product $\lambda \ast \tau$ is the total amount of correction that is done by that male.

As the total amount of correction that is done increases, the distribution of sperm types should shift towards sperm with fewer phenotypic defects (so towards sperm with more phenotypic defects corrected). In other words, increases in $\lambda \ast \tau$ should shift the distribution of the fertility of sperm in an ejaculate towards higher fertility (fewer phenotypic defects).

As shown in figure 2.2, this is the case under this model. Figure 2.2 also demonstrates the diminishing returns that investments into $\tau$ confer. When $\lambda \ast \tau$ increases at low values (blue and red curves), the distribution is shifted much farther than when $\lambda \ast \tau$ increases by the same amount at larger $\lambda \ast \tau$ values (red and yellow curves).

Changes in the total amount of correction done will also affect the variance in sperm fertility. When $\lambda \ast \tau$ is large enough, increases in $\lambda \ast \tau$ serve to lower the variance in sperm fertility. This is as we would expect because increases in $\lambda \ast \tau$ mean that more phenotypic defects are corrected in sperm, and, so, sperm are more uniform. However, when $\lambda \ast \tau$ is quite low, this pattern is reversed. This happens because we are only considering the effective number of phenotypic defects in sperm, and the fertility that this confers. In reality, many different combinations of sperm defects will correspond with the same effective number of defects. So, while there is a narrow distribution in the effective number of defects when only a few phenotypic defects are corrected, these sperm will be highly variable in traits such as morphology. It is reasonable that we would see a narrow distribution of the effective number of phenotypic defects for very low $\lambda \ast \tau$ because many different combinations of phenotypic defects will result in the same effective number of phenotypic defects. This trend is shown
Figure 2.2: The probability distribution of the effective number of corrected phenotypic defects as depends upon \( \tau \). (Evaluated at \( m = 100, \lambda \ast \tau = 0.5, 2.5, 4.5 \) blue, red, yellow curves)

in figure 2.3, as predicted under this model.

2.5.2 Differences Due to Changes in \( m \)

Perhaps the most intuitive effect on the distribution of sperm types (number of corrected phenotypic defects in sperm) is shown in the maximum effective number of phenotypic defects, \( m \). As \( m \) increases, the distribution of the number of corrected phenotypic defects shifts to the right (towards more corrected phenotypic defects). Since increases in \( m \) mean that there are more phenotypic defects to correct (ie. \( (m - k) \) increases in 2.4), the number of phenotypic defects corrected should increase, and this is the case, as shown in figure 2.4.

When \( m \) increases, and there are more phenotypic defects to correct, the variance in the number of corrected phenotypic defects should also increase. This pattern is also as
CHAPTER 2. THE MODEL

Figure 2.3: The variance in the effective number of phenotypic defects depends upon the number of phenotypic defects corrected, or $\lambda \tau$. (Evaluated at $m = 100$)

The Variability of Sperm

As mentioned above, sperm will be most variable when only a few phenotypic defects are avoided during sperm production, even though the distribution of effective phenotypic defects appears narrow. This happens because any significant defects will cause sperm to be completely nonfunctional. However, these defects may lie in many different regions and combinations throughout sperm phenotype, and need not represent a very large proportion of the entire genome involved in spermatogenesis. So, while many sperm will have the same number of effective phenotypic defects, they will appear very different from one another. Thus, the variability among sperm with a large effective number of phenotypic defects will
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Figure 2.4: The probability distribution of the effective number of corrected phenotypic defects as depends upon $m$. (Evaluated at $\lambda = 0.2$, $\tau = 5$, $m = 100, 500, 1000$ blue, red, yellow curves)

be much higher than that among sperm with few such defects.

In figure 2.6, I approximate the number of different combinations of phenotypic defects that sperm would contain for each number of effective defects. To do so, I calculate the number of combinations that an effective number of defects, $m - k$, could appear in by assuming that sperm will be completely nonfunctional if 10% or more of the genome is transcribed incorrectly (equation (2.10)). In other words, I assume that $m$ is approximately 10% of the genome, in order to illustrate the general trend.

\[
\text{Number of Sperm Morphs} = \binom{10 \times m}{m - k} \quad (2.10)
\]
\[
= \sum_{k=0}^{m} \binom{10 \times m}{m - k} P(k, \tau) \quad (2.11)
\]
As 2.6 demonstrates, when very few phenotypic defects have been avoided during sperm production, sperm will appear highly variable, and a large number of different morphs may be produced. Conversely, when most phenotypic defects have been corrected, only a few potential sperm morphs are possible.

In figure 2.7, I approximate the number of potential sperm morphs based on the amount of time spent producing sperm ($\tau$) by using equation (2.11). Thus, when a male spends longer producing his sperm, he will produce fewer sperm morphs as his sperm will contain only a few phenotypic defects. Since there will only be a few such defects, only a few combinations of these defects are possible, and, so, sperm should be much less variable. When a male produces his sperm “carelessly”, those sperm will contain many defects, in many combinations, and so those sperm will be highly variable.

Figure 2.5: The variance of probability distribution of the effective number of corrected phenotypic defects becomes wider with $m$. (Evaluated at $\lambda = 0.2$, $\tau = 5$)
The number of potential combinations of phenotypic defects depends upon how many such defects have been produced \((m - k)\).

### 2.7 Number of Mating Events

I will assume that a male must spend some time, \(\alpha \in [0, \infty)\), performing tasks between reproductive bouts, such as finding a mate, searching for food, and sleeping. So, each reproductive event that a male participates in takes \(\alpha + \tau\) units of time. Note that \(\tau\) only represents the additional amount of time spent improving sperm beyond a basic amount of time necessary to produce sperm. This time may be spent between some time spent on other tasks. In other words, \(\alpha\) and \(\tau\) need not occur in discrete time periods. \(\tau\) simply provides a measure of how much time is spent improving sperm quality relative to how much time is spent on other tasks \((\alpha)\).

Since males have a finite lifespan, the length of time that a male spends during and between any reproductive events will determine how many such events he can participate...
CHAPTER 2. THE MODEL

Figure 2.7: The number of potential combinations of phenotypic defects depends upon how many such defects are avoided, and so, upon how long a male spends in spermatogenesis ($\tau$).

in. This means that by spending longer on sperm production in each reproductive event (by increasing $\tau$) a male can participate in fewer reproductive events during his lifetime.

Of course, not all males of a given species live for the same lifetime; some will die earlier than others. I assume that every male of a given species spends, on average, the same amount of time mating and suffers the same probability of mortality during a reproductive bout. So, I denote the probability that a male survives a given reproductive bout by the constant $s \in (0, 1]$.

Males may also die between reproductive events. I will assume that this occurs at a constant rate, $\mu$, so if a male spends $\alpha + \tau$ time between reproductive events, then the probability that he survives between reproductive events is $e^{-\mu(\alpha+\tau)}$. Then, the expected
number of reproductive events that a male participates in before he dies is

\[ E[\text{number of matings}] = \sum_{i=0}^{\infty} s^i (e^{-\mu(\alpha + \tau)})^{i+1} \]

\[ = e^{-\mu(\alpha + \tau)} \sum_{i=0}^{\infty} (se^{-\mu(\alpha + \tau)})^i \]

\[ = \frac{1}{e^{\mu(\alpha + \tau)} - s} \]

(2.12)
Chapter 3

Male Fitness: No Sperm Competition

I define male fitness as the number of ova that a male fertilizes in his lifetime. In the absence of sperm competition, this is just the probability that a male fertilizes an ovum in any reproductive event multiplied by the number of such events in which he participates. In other words, a male’s fitness is his fertility multiplied by the number of matings he achieves. This is given as:

\[ w(\tau) = \left( \frac{1}{e^{\mu(\alpha+\tau)} - s} \right) \ast \left( 1 - (1 - f(\tau))^n \right) \]  

(3.1)

3.1 Linear Sperm Fertility Function

Using equation (2.1), the average fertility of the sperm in an ejaculate is

\[ f(\tau) = 1 - e^{-\lambda \tau} \]  

(3.2)

so male fitness is

\[ w(\tau) = \left( \frac{1}{e^{\mu(\alpha+\tau)} - s} \right) \ast \left( 1 - e^{-\lambda \tau \ast n} \right) \]  

(3.3)

Thus, there is an optimal amount of time (\( \tau \)) for a male to spend on sperm production
that maximizes his fitness (figure 3.1). This optimum results from a tradeoff between the number of reproductive events that a male can have in his lifetime and his fertility during those events. Low male fitness can result from producing very low quality sperm so that reproductive events do not usually involve fertilizations. However, low male fitness can also result from producing sperm so slowly that very few reproductive events can occur within a male’s lifespan.

Figure 3.1: The fitness of a male as depends upon $\tau$, the amount of time spent on sperm production. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$)

I now examine the effect that the various parameters have upon the optimal $\tau$ (the length of time spent on sperm production that maximizes male fitness).

3.1.1 The Death Rate Between Matings

The optimal investment into sperm production ($\tau$) decreases as the death rate during sperm production ($\mu$) increases (figure 3.2). When the rate at which males die between mating
events ($\mu$) increases, males are more likely to die during the time that they spend producing their sperm. In other words, as $\mu$ increases, investment into $\tau$ becomes increasingly costly. Since the benefit that a male receives by investing time into sperm production (or error-checking) diminishes with increased $\tau$ values, the optimal choice of $\tau$ will occur when increasing $\tau$ becomes more costly (in terms of survival) than beneficial (in terms of fertility gains). Thus, the optimal amount of time for a male to spend producing his sperm is the result of a trade-off between survival and fertility. So, when investment into $\tau$ becomes more costly (i.e., when $\mu$ increases), this optimal $\tau$ will occur at progressively lower values. Thus, as $\mu$ increases, the optimal $\tau$ decreases to compensate for the decreased probability of survival that increases in $\mu$ confer.

![Figure 3.2: The optimal $\tau$ value as depends upon $\mu$, the death rate between reproductive bouts. (Evaluated at $s = 0.9$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$)](image)

Figure 3.2 also demonstrates that the effect that $\mu$ has on the optimal $\tau$ value diminishes as $\mu$ increases. This occurs because when $\mu$ is large, the optimal $\tau$ is relatively low. As
mentioned above, there are diminishing returns for investments into $\tau$, so changes in $\tau$ have a more marked effect on male fertility when $\tau$ is small. Thus, the larger $\mu$ becomes, the smaller $\tau$ will become and changes in $\tau$ will affect male fertility to a greater degree. In other words, when $\mu$ is large, changes in $\tau$ affect male fertility more than they affect survival. So, changes in $\mu$ have less effect on male fitness when $\mu$ is large because male fitness depends more upon fertility than it does upon survival. This means that changes in $\mu$ will have a diminishing effect as $\mu$ increases.

As figures 3.3(a) and 3.3(b) show, the distribution of sperm quality will shift towards poorer quality sperm as $\mu$ (the death rate during sperm production) increases. Note that although the distribution in this figure narrows when $\mu$ increases, this distribution only represents the effective number of phenotypic defects corrected (or avoided) during the production of sperm cells. Since the same decrease in fertility can arise from many different combinations of phenotypic defects during sperm production, measures of any sperm quality trait will be more variable when $\tau$ is small and few phenotypic defects are corrected (or avoided) in sperm production. Thus, while the distribution of the effective number of phenotypic defects narrows when $\mu$ increases, traits affecting sperm quality will actually become more variable, and so the distribution of any such trait will become wider as $\mu$ increases.

3.1.2 The Probability That a Male Survives Mating

Figures 3.4, 3.5(a) and 3.5(b) demonstrate that, under this model, the optimal $\tau$ value decreases as $s$ increases. Once again, note that although the distribution of sperm types does appear to narrow as the distribution shifts towards lower quality sperm, sperm cells themselves will become much more variable with respect to any given trait.

The probability that a male survives a mating event is not affected by how long he spends producing his sperm. Thus, increases in the probability that a male survives a mating event have a more straight-forward affect on male fitness. When a male is unlikely
Figure 3.3: The optimal distribution of sperm types as depends upon $\mu$, the death rate between reproductive bouts. (Evaluated at $s = 0.9$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$) The effect that $\mu$ has on the full distribution of sperm types is shown in part (a), while only the bottom portion of the distribution is given in part (b) in order to make the trend more evident.

to survive a mating event and to mate again, his current mating event is very important to him. Conversely, when a male can expect to survive his current mating and to mate many times in the future, his current mating event becomes less important relative to future potential matings. So, males should invest more time into sperm production when they are unlikely to survive a mating event (when $s$ is small) than when their survival during mating is relatively certain (when $s$ is large). In other words, the optimal choice of $\tau$ should decrease as $s$ increases.

It is also worth noting that the effect that $s$ has on the optimal $\tau$, and the optimal distribution of sperm types, increases as $s$ becomes larger. This occurs because $s$ only appears within the denominator of the term representing the number of matings that a male can have in his lifetime. So, the effect that changes in $s$ have correspond with changes in $\frac{1}{s}$, which are larger when $s$ is close to 1 than when $s$ is very small. Intuitively this makes sense as well; if a male is likely to survive a mating event, then future events should be
important to him. So, the number of events that the male can take part in have a big impact upon his fitness. Conversely, when it is unlikely that a male will survive reproduction, the number of future reproductive events that he can participate in is relatively unimportant, and so fertility has a much larger impact on male fitness and $s$ has relatively little effect.

Figure 3.4: The optimal $\tau$ value as depends upon $s$, the probability that a male survives a mating event. (Evaluated at $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$)

3.1.3 The Amount of Time Spent Between Mating

As figures 3.6, 3.7(a) and 3.7(b) show, the optimal $\tau$ value tends to increase as the amount of time spent between mating events ($\alpha$) increases.

Males will only live for some finite lifetime, so the longer a male spends between mating events, the fewer such events in which he will be able to participate. Moreover, the longer a male spends between reproductive events, the higher the probability that he will die before he can ever reproduce again (because death between mating events occurs at a fixed rate,
Figure 3.5: The distribution of sperm types as depends upon \( s \), the probability that a male survives a mating event. (Evaluated at \( \mu = 0.05, \alpha = 5, \lambda = 0.2, n = 1000 \)) The effect that \( s \) has on the full distribution of sperm types is shown in part (a), while only the bottom portion of the distribution is given in part (b) in order to make the trend more evident.

Thus, as \( \alpha \) increases (as males spend longer between reproductive events), each current event becomes more important than future potential events, and males should invest more time into sperm production \( (\tau) \).

It is, once again, worth noting that the effect that \( \alpha \) has upon the optimal \( \tau \) value diminishes as \( \alpha \) becomes larger. This occurs because the longer a male spends between matings, the higher the chances that he will die before mating again. When \( \alpha \) gets larger, this probability (that a male will not survive to mate again) becomes very high. At the same time, because the optimal \( \tau \) increases as \( \alpha \) becomes larger, and because fertility benefits to increasing \( \tau \) diminish as \( \tau \) increases, male survival becomes increasingly important relative to male fertility. Thus, we see a diminishing effect of \( \alpha \) on the optimal choice of \( \tau \) when \( \alpha \) becomes large enough.
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3.1.4 The Error Repair Rate

As the error repair rate ($\lambda$) increases, the optimal $\tau$ value tends to decrease (figure 3.8). As errors become easier to correct (or avoid) during sperm production, males should invest less of their time to error correction because of the diminishing returns involved in such investment. Thus, we would expect that as the repair (or avoidance) rate of errors, $\lambda$, increases, the optimal $\tau$ should decrease (figure 3.8). Note that this effect diminishes as $\lambda$ increases. This occurs because the number of errors repaired comes from the product of $\lambda$ and $\tau$, so $\tau$ changes in way that is proportional to $\frac{1}{\lambda}$. Thus, the decrease in the optimal $\tau$ with increases in $\lambda$ diminishes as $\lambda$ gets larger (and $\frac{1}{\lambda}$ becomes smaller).

Although it is difficult to see in both 3.9(a) and 3.9(b), an interesting reversal of the trends discussed between the optimal $\tau$ value and the error repair rate ($\lambda$) occurs when we consider the distribution of sperm types that results from changes in $\lambda$. As $\lambda$ represents

Figure 3.6: The optimal $\tau$ value as depends upon $\alpha$, the amount of time spent between reproductive events. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\lambda = 0.2$, $n = 1000$)
the rate at which errors are repaired, it is an important part of the probability that a given sperm cell is of any particular type. When \( \lambda \) is small, there is a low probability that errors will be repaired, so a low probability of producing any sperm with few phenotypic defects in them. So, although males will spend more time producing their sperm, they still have relatively low quality and highly variable sperm. Conversely, when \( \lambda \) is large, it is quite likely that a given sperm cell is of high quality even though males invest considerable less time into sperm production. In other words, the decrease in \( \tau \) that occurs in response to \( \lambda \) is more than compensated by the fact that errors are much more easily repaired when \( \lambda \) increases.

### 3.1.5 The Total Number of Sperm Produced

As demonstrated in figures 3.10 and 3.11, the optimal \( \tau \) value tends to decrease as the total number of sperm \( (n) \) increases. When a male produces a very large number of sperm, the
fertility of each individual sperm becomes less important. This is due to the stochastic nature by which errors are repaired during sperm production. Since there is always some positive probability that a spermatozoa is defect-free (or relatively defect-free), the chances that some spermatozoa are of very high fertility increases as the number of sperm \((n)\) increases. Because the spermatozoa in an ejaculate typically outnumber fertilizable ova by several orders of magnitude ([13, 14]), only a small number of the of the most fertile sperm are likely to be involved in fertilizations. So, the most fertile sperm are more important in male fertility than other sperm. As males that produce very large numbers of sperm will produce some highly fertile sperm by chance, they should invest less time towards sperm production than males that produce only a small number of sperm.

As before, this effect tends to diminish as \(n\) gets larger. This happens because, again, male fertility depends on the product of \(n\) and \(\tau\), so as \(n\) increases, \(\tau\) should decrease in a
Figure 3.9: The distribution of sperm types as depends upon $\lambda$, the error repair rate. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $n = 1000$) The effect that $\lambda$ has on the full distribution of sperm types is shown in part (a), while only the bottom portion of the distribution is given in part (b) in order to make the trend more evident.

way that is proportional to $\frac{1}{n}$.

### 3.2 Nonlinear Sperm Fertility Function

Using equation (2.2), the average fertility of the sperm in an ejaculate is

$$f(\tau) = \frac{1 - e^{-\lambda \tau}}{m^2}[(m - 1)(1 - e^{-\lambda \tau})(m - 2)(1 - e^{-\lambda \tau}) + 3] + 1$$

(3.4)

and so male fitness is

$$w(\tau) = \left(\frac{1}{e^{\mu(\alpha + \tau)} - s}\right) \left(1 - \left(1 - \frac{1 - e^{-\lambda \tau}}{m^2}[(m - 1)(1 - e^{-\lambda \tau})(m - 2)(1 - e^{-\lambda \tau}) + 3] + 1\right)^n\right)$$

(3.5)

This fitness function (3.12) looks qualitatively quite similar to 3.1, and, once again, there is an optimal value of $\tau$ for which this fitness is maximized (figure 3.12). This optimal $\tau$ value is the result of a tradeoff between the number of matings that a male can participate in and his fertility during those matings.

Before discussing the effect of particular parameter choices on male fitness, and on the
optimal choice of $\tau$, it is worth noting a key difference between (2.2) and (2.1). In (2.1), the average fertility of a sperm cell is $1 - e^{-\lambda \tau}$. In (2.2), however, the average fertility of a sperm cell is on the order of $(1 - e^{-\lambda \tau})^3$. So, changes in $\tau$ will have a more extreme effect on male fertility and fitness with (2.2) than with (2.1).

3.2.1 The Death Rate Between Matings

The effect that $\mu$ (the death rate between mating events) has on male fitness is much the same with (2.2) as it was with (2.1). In general, the optimal choice of $\tau$ decreases as the death rate between mating events increases as this causes the probability of dying between reproductive events to increase. As discussed above, and shown in figures 3.13 and 3.14, the effect that $\mu$ has is more exaggerated with (2.2) because changes in $\tau$ have a more extreme effect on male fitness.
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Also, as discussed in section 3.1, \( \mu \) has a diminishing effect on the optimal \( \tau \) value because of the diminishing returns involved in fertility investments (investments into \( \tau \)).

3.2.2 The Probability That a Male Survives Mating

The effect that the probability of surviving a mating event \( s \) has on the optimal choice of \( \tau \) is qualitatively very similar with (2.2) to its effect in section 3.1. However, once again, this effect is more extreme, and increases in \( s \) cause larger decreases in \( \tau \).

The other major difference between (2.2) and (2.1) is that male fertility depends upon \( \tau \) much more strongly with (2.2). Because of this, there is not the same increase in the effect of \( s \) on the optimal \( \tau \) (or sperm production strategy) with increases in \( s \), and male fertility dominates male fitness to a greater degree (figures 3.15 and 3.16).
Figure 3.12: The fitness of a male as depends upon $\tau$, the amount of time spent on sperm production. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$, $m = 100$)

3.2.3 The Amount of Time Spent Between Mating

The effect that $\alpha$ has on the optimal $\tau$ value is nearly identical with (2.2) as it was with (2.1). Once again, this effect is more extreme, but qualitatively the same. Also, as shown in figure 3.17, fertility is more important to male fitness with (2.2) than when the linear fertility function is used, so, in general, $\tau$ is larger.

3.2.4 The Error Repair Rate

As we would expect, the effect that $\lambda$ (the rate of repair or avoidance of errors) has on the optimal choice of $\tau$ is qualitatively the same with (2.2) as it was in section 3.1, but more extreme.
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3.2.5 The Total Number of Sperm Produced

The effect that the total number of sperm produced \( (n) \) has on the optimal choice of \( \tau \) is also quite similar when we use (2.2) for sperm fertility as it was with (2.1). Once again, increases in \( n \) lead to larger decreases in \( \tau \) under (2.2) than under (2.1). This effect is more clearly visible (as it is more extreme) with (2.2), so the relationship between \( n \) and the optimal distribution of sperm types is shown in figure 3.18.

3.2.6 The Maximum Number of Effective Phenotypic Defects

The biggest difference between (2.2) and (2.1) comes from the occurrence of \( m \) in the average sperm fertility, and so, in male fitness. Recall that \( m \) represents the maximum number of effective phenotypic defects that sperm may contain, and the number of effective phenotypic defects that confers zero fertility. As figure 3.19 displays, the optimal \( \tau \) value

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Figure 3.13: The optimal \( \tau \) value as depends upon \( \mu \), the death rate between reproductive events. (Evaluated at \( s = 0.9, \alpha = 5, \lambda = 0.2, n = 1000, m = 100 \))
Figure 3.14: The distribution of sperm types as depends upon \( \mu \), the death rate between reproductive events. (Evaluated at \( s = 0.9, \alpha = 5, \lambda = 0.2, n = 1000, m = 100 \))

...tends to increase with increases in \( m \).

As \( m \) increases, there are more possible phenotypic defects to correct (or avoid) in each spermatozoa. Since the number of phenotypic defects repaired depends upon how many phenotypic defects are present, increases in \( m \) will cause increases in the number of phenotypic defects repaired. Thus, spending \( \tau \) time on sperm production will be more effective when \( m \) is large than when it is small. However, male fertility also decreases with increases in \( m \). This decrease in fertility occurs because the larger \( m \) is, the more phenotypic defects will exist in male sperm for a given \( \tau \) investment. The decrease in fertility caused by the increase in \( m \) more than compensates for any increase in the efficiency by which defects are repaired. So, males must spend longer producing their sperm (increase \( \tau \)) in order to maintain sperm fertility.

Once again, this effect diminishes as \( m \) increases (figures 3.19 and 3.20). This occurs because the difference in the number of phenotypic defects repaired (or avoided) due to a change in \( m \) is much larger when \( m \) is small than when \( m \) is relatively large. Moreover,
because investments into $\tau$ confer diminishing returns, and because the optimal $\tau$ increases with $m$, fertility changes less when $\tau$ is already relatively large (because $m$ is large) than when it is smaller. Also, increases in $m$ confer fertility costs that change much less when $m$ is large than when $m$ is small. This means that we will see a diminishing effect of $m$ on the optimal $\tau$.

Another way of thinking about this comes from the fact that the number of phenotypic defects repaired depends upon the product of the $m$ and $\tau$. When both $m$ and $\tau$ are large, phenotypic defects will be repaired so quickly that investing more time into sperm production means that only a few more phenotypic defects can be corrected (as most of them are already corrected). Also, when $m$ is very large, changes in $m$ make much less difference to the number of defects to repair than when $m$ is small. So, there is less benefit, in terms of number of defects repaired, to increasing $\tau$ further in response to increases in $m$. 

Figure 3.15: The optimal $\tau$ value as depends upon $s$, the probability of surviving a reproductive event. (Evaluated at $\mu = 0.05, \alpha = 5, \lambda = 0.2, n = 1000, m = 100$)
Figure 3.16: The distribution of sperm types as depends upon $s$, the probability of surviving a reproductive event. (Evaluated at $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$, $m = 100$)

when $m$ is relatively large, and relatively little fertility cost associated with such increases in $m$. Thus, we see a diminishing effect of changes in $m$ on the optimal $\tau$ value when $m$ increases.
Figure 3.17: The distribution of sperm types as depends upon $\alpha$, the probability of surviving a reproductive event. (Evaluated at $\mu = 0.05$, $s = 0.9$, $\lambda = 0.2$, $n = 1000$, $m = 100$)

Figure 3.18: The distribution of sperm types as depends upon $n$, the total number of sperm produced. (Evaluated at $\mu = 0.05$, $s = 0.9$, $\lambda = 0.2$, $\lambda = 0.2$, $m = 100$)
CHAPTER 3. MALE FITNESS: NO SPERM COMPETITION

Figure 3.19: The optimal $\tau$ value as depends upon $m$, the number of phenotypic defects that confers zero fertility. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$)

Figure 3.20: The distribution of sperm types as depends upon $m$, the number of phenotypic defects that confers zero fertility. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$)
Chapter 4

Male Fitness: Sperm Competition

Under some risk of sperm competition ($p_{comp}$), the number of ova that a male can fertilize will depend both on the fertility of his own ejaculate and the fertility of the ejaculates of competing males. Male fitness, then, will be the sum of a male’s fitness in competitive interactions and his fitness in the absence of sperm competition. In other words, male fitness is defined as

$$w(\tau, \hat{\tau}) = (1 - p_{comp})w(\tau|\text{no competition}) + p_{comp}w(\tau, \hat{\tau}|\text{competition}) \quad (4.1)$$

$w(\tau|\text{no competition})$ was found in the previous section (equation(3.1)), so we only need to develop an expression for $w(\tau|\text{competition})$.

When the sperm from two different males compete for the fertilization of a single egg, there are several possible outcomes. To examine these, I designate one male as the focal male, and the other as the competing male. The first possibility is that sperm from neither male reaches an egg. In this case, both males have fertility 0. It is also possible that sperm from only one of the males reaches the egg. Then, the male whose sperm does reach the egg will have fertility 1, while the other male has fertility 0. The most interesting possibility occurs when the sperm from both males reach the egg. When this happens, I will define a male’s fertility as the fertility of his ejaculate relative to the fertility of the combined
ejaculates. In other words, when two males compete, a male will receive fertilizations in proportion to the fertility of his ejaculate relative to that of both competing ejaculates. Thus, I define male fitness with sperm competition as

\[
w(\tau, \hat{\tau}|\text{competition}) = \left(\frac{1}{e^{\mu(\alpha+\tau)} - s}\right) \ast \left[\frac{1 - (1 - f(\tau))^n}{1 - (1 - f(\tau))} \ast \frac{1 - (1 - f(\hat{\tau}))^n}{1 - (1 - f(\hat{\tau}))} \ast \frac{f(\tau)}{f(\tau) + f(\hat{\tau})}\right]
\]  

(4.2)

where \( \hat{\tau} \) is the average \( \tau \) of males in the population (and so, the average \( \tau \) of a competing male).

To assess the sperm production strategies of competing males, I seek an evolutionarily stable strategy (ESS) as defined in [30]. In other words, I seek a choice of \( \tau \) that, if adopted by a population, cannot be invaded by any mutant \( \tau \) strategy. Such a strategy, \( \hat{\tau} \), occurs when either of the following conditions are met:

(i) \( w(\hat{\tau}, \hat{\tau}) > w(\tau, \hat{\tau}) \)

(ii) \( w(\hat{\tau}, \hat{\tau}) = w(\tau, \hat{\tau}) \) and \( w(\hat{\tau}, \tau) > w(\tau, \tau) \)

The first of the conditions is met by \( \tau^* \) whenever both of the following are satisfied:

(ia) \( \frac{\partial w(\tau, \tau^*)}{\partial \tau}\big|_{\tau=\tau^*} = 0 \)

(ib) \( \frac{\partial^2 w(\tau, \tau^*)}{\partial \tau^2}\big|_{\tau=\tau^*} < 0 \)

In other words, \( \hat{\tau} \) is an ESS whenever \( w(\hat{\tau}, \hat{\tau}) \) is maximized. I now use this approach to assess the ESS \( \tau \) strategies using the different sperm fertility functions introduced in section 2.2.

4.1 Linear Sperm Fertility Function

Using equation (2.1) in the fitness function, (4.1), we can produce a pairwise invasibility plot (see [32]) in order to determine whether an ESS choice of \( \tau \) exists. There is such an
ESS $\tau$ value when we use (2.1) to represent sperm fertility (figure 4.1). Moreover, as can be seen in figure 4.1, the ESS point is also a convergent stable point. This means that once a population reaches its ESS point, no mutant sperm production strategy can invade that population. If the resident $\hat{\tau}$ value is lower than the ESS $\tau$ value, mutants with larger $\tau$ values can invade that population. Likewise, if the resident $\hat{\tau}$ value is higher than the ESS $\tau$ value, mutants with lower $\tau$ values will be able to invade the population. Thus, a population that has not adopted the ESS $\tau$ will evolve towards the ESS $\tau$ until it reaches it, and then the population will remain at that ESS.

![Figure 4.1](image)

Figure 4.1: The pairwise invasibility plot using (2.1). The blue shading indicates regions where the mutant strategy ($\tau$) can invade the resident strategy ($\hat{\tau}$), the dashed lines represent the boundaries of these regions (where the fitnesses of the the mutant and the resident strategies are the same), and the red line indicates where the resident and the mutant strategies are the same. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$)
4.1.1 The Probability of Sperm Competition

As figure 4.2 demonstrates, this model predicts an increase in the amount of time that a male spends on sperm production ($\tau$) with an increased risk of sperm competition. When there is some risk of sperm competition, sperm fertility is more important to a male than it is when there is no risk of sperm competition. This happens because a male’s fitness depends not only upon his own fertility, but also upon that of his competitors. As the risk of sperm competition, $p_{\text{comp}}$, increases, so does the importance of the fertility of a male’s sperm increase.

![Figure 4.2: The ESS $\tau$ value as depends upon $p_{\text{comp}}$, the probability of sperm competition.
(Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$, $m = 100$)](image)

This trend is also shown in figure 4.3. It is important to note that although the distribution of sperm types appears to widen as the probability of sperm competition increases, this occurs only because sperm types, here, are differentiated only by the number of effective phenotypic defects that they contain. In reality, the distribution of types will narrow as
CHAPTER 4. MALE FITNESS: SPERM COMPETITION

Figure 4.3: The ESS distribution of sperm types as depends upon $p_{\text{comp}}$, the probability of sperm competition. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$, $m = 100$)

the risk of sperm competition increases and the optimal investment into sperm production ($\tau$) increases. So, the decrease in the variance of sperm fertility traits that occurs with increased risk of sperm competition as observed in other studies is predicted by this model. To demonstrate this, I have included a figure displaying how the approximation of the number of sperm morphs made in (2.11) changes with $p_{\text{comp}}$ (figure 4.4).

In the rest of the plots displayed in this section, it is worth noting that the ESS value of $\tau$ is always much higher than it was in section 3.1. Since fertility should be more important to a male under sperm competition, and since these plots are all made by assuming a positive risk of sperm competition ($p_{\text{comp}}$), this is a reasonable prediction.

4.1.2 The Death Rate Between Matings

As in section 3.1, the ESS value of $\tau$ tends to decrease as the death rate between reproductive events increases (figure 4.5). This is unsurprising because as $\mu$ increases, the probability of dying during any sperm production period increases. So, when $\mu$ increases, the survival
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Figure 4.4: An approximation of the number of sperm morphs that would be seen as depends upon $p_{comp}$, the probability of sperm competition. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$, $m = 100$)

cost of devoting time towards sperm production increases. Moreover, as investing time towards sperm production confers diminishing benefits to males, this increase in the cost of sperm production makes it better for a male to reduce the amount of time that he spends producing sperm so that he is more likely to survive to mate again.

When sperm competition occurs, the model predicts that this effect will be more extreme (figure 4.5). This occurs because there is some positive probability that any current reproductive event will be a competitive one. When sperm competition does occur, the fertility of the sperm produced by the males involved becomes much more important in achieving fertilizations than in the absence of sperm competition. So, the ESS $\tau$ value is much higher than in the absence of sperm competition. However, because of the diminishing returns associated with fertility investments, increases in the cost of sperm production have
a more marked effect than in the absence of sperm competition. Thus, we would expect to see a more extreme effect of $\mu$ on the ESS value of $\tau$ under sperm competition than in section 3.1.

Figure 4.5: The ESS $\tau$ value as depends upon $\mu$, the probability of dying between reproductive events. (Evaluated at $s = 0.9$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$, $m = 100$, $p_{\text{comp}} = 0.2$)

As figures 4.6(a) and 4.6(b) display, the variation in sperm types (and fertility) appears to decrease with increases in $\mu$, but in reality will increase if the distinction between different types of phenotypic defects is made. So, distributions of sperm traits will tend to widen as $\mu$ increases. As investments into sperm fertility become more costly, and so males are more likely to die during any sperm production period, they should spend less time correcting (or preventing) errors in sperm production. So, more phenotypic defects will exist in the sperm that are produced. The distribution of sperm fertility, then, should become wider as sperm are produced more “carelessly”, and there are more phenotypic defects in sperm to
make those sperm variable.

Figure 4.6: The ESS distribution of sperm types as depends upon \( \mu \), the death rate between reproductive events. (Evaluated at \( s = 0.9, \alpha = 5, \lambda = 0.2, n = 1000, m = 100, p_{comp} = 0.2 \)) The effect that \( \mu \) has on the full distribution of sperm types is shown in part (a), while only the bottom portion of the distribution is given in part (b) in order to make the trend more evident.

### 4.1.3 The Probability That a Male Survives Mating

As in section 3.1, the ESS time investment towards sperm production (\( \tau \)) tends to decrease as the probability that a male survives a reproductive event (\( s \)) increases. This, once again, reflects the change in relative importance of current and future mating events that occurs with changes in \( s \). However, this decrease in the ESS \( \tau \) value is even more extreme with increases in \( s \) when there is a positive risk of sperm competition than when sperm competition is absent. This is because sperm fertility is more important to males under sperm competition as it impacts male fitness to a greater degree. So, the ESS \( \tau \) value is generally higher under sperm competition than the optimal \( \tau \) value is in absence of sperm competition. Because of the nature by which benefits from investing time (\( \tau \)) towards sperm production diminish, males can “afford” to decrease the higher ESS \( \tau \) value more than they
could the lower optimal $\tau$ value that existed in absence of sperm competition. So, we see a more marked decrease in the ESS $\tau$ value, or a more extreme effect of $s$ on the ESS $\tau$ value than we saw in the absence of sperm competition.

![Figure 4.7: The ESS $\tau$ value as depends upon $s$, the probability of a male surviving a reproductive event. (Evaluated at $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$, $p_{\text{comp}} = 0.2$)](image)

Another major difference between the effect of $s$ with and without a positive risk of sperm competition is that a more linear decrease in the ESS $\tau$ occurs under sperm competition as can seen in figures 4.7, 4.8(a) and 4.8(b). This also arises because of the elevated importance of fertility under sperm competition. Recall that in the absence of sperm competition the effect that $s$ had on the optimal $\tau$ increased as $s$ increased. The accelerated effect found in section 3.1 was due to the fact that the relative importance of the number of future reproductive events and of the current reproductive events changed with changes in $s$. Under sperm competition, fertility in both current and in future reproductive events is
more important to males because every event will not necessarily yield a fertilization. So, we do not see as much of a trade-off between future and current matings as fertility is always quite important.

Figure 4.8: The ESS distribution of sperm types as depends upon \( s \), the probability of a male surviving a reproductive event. (Evaluated at \( s = 0.9, \alpha = 5, \lambda = 0.2, n = 1000, m = 100, p_{\text{comp}} = 0.2 \).) The effect that \( \mu \) has on the full distribution of sperm types is shown in part (a), while only the bottom portion of the distribution is given in part (b) in order to make the trend more evident.

4.1.4 The Amount of Time Spent Between Mating

The effect that the amount of time spent between mating events (\( \alpha \)) has upon the ESS \( \tau \) is qualitatively nearly identical to the effect that it had in the absence of sperm competition (3.1). The only difference is that the effect is more extreme as we would expect because male fertility is more important under sperm competition. Also, it is worth noting that, of course, \( \tau \) is always much higher under a positive risk of sperm competition than in the absence of sperm competition.

Only the plot of the distribution of sperm types is given here as the trend in this distribution due to changes in \( \alpha \) is more clearly seen under sperm competition because \( \tau \) is
larger and because the effect of $\alpha$ is more marked. Once again, be aware that although the distribution appears to become wider with $\alpha$, this only because we are considering only the number of effective phenotypic defects, not the phenotypic defects themselves and because the ESS distribution of sperm types is at the low end of the sperm fertility range.

Figure 4.9: The ESS distribution of sperm types as depends upon $\alpha$, the amount of time a male spends between mating events. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\lambda = 0.2$, $n = 1000$, $p_{\text{comp}} = 0.2$)

4.1.5 The Error Repair Rate

As previously discussed, sperm fertility is much more important under a positive risk of sperm competition than in the absence of any sperm competition. As $\lambda$, the error repair (or avoidance) rate is extremely important in determining sperm fertility, its effect is much more evident under sperm competition. As figure 4.10 demonstrates, $\tau$ decreases much more quickly in response to increased $\lambda$ values when sperm competition is possible.

When $\lambda$ is very small, the ESS $\tau$ value changes with $\frac{1}{\lambda}$ as we saw in the absence of sperm competition. However, when $\lambda$ becomes larger, $\tau$ begins to decrease in a linear fashion with increases in $\lambda$. This happens because, once again, the number of errors repaired results
from the product of $\lambda$ and $\tau$. So, when $\lambda$ increases, the ESS $\tau$ changes with $\frac{1}{\lambda}$ in order to maintain a constant number of repaired errors. This trend does not hold for larger $\lambda$ values because of the elevated importance of sperm fertility under a positive risk of sperm competition. Recall that as $\tau$ becomes small, every decrease in $\tau$ has a large effect on male fertility. So, under sperm competition, the ESS $\tau$ value cannot decrease as quickly as $\frac{1}{\lambda}$ does, as this would mean that male fertility would become extremely low.

![Figure 4.10: The ESS $\tau$ value as depends upon $\lambda$, the error repair rate. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $n = 1000$, $p_{\text{comp}} = 0.2$)](image)

As mentioned in section 3.1, the effect that $\lambda$ has on $\tau$ seems to reverse when we consider the distribution of sperm types that results from changes in $\lambda$. This, once again, occurs because an increase in $\lambda$, or the probability that phenotypic defects are repaired, means that there is an increase in the probability of an individual sperm cell being of high quality. Thus, as in the absence of sperm competition, the distribution of sperm types shifts towards higher quality sperm as $\lambda$ increases. This is more clearly shown here in figure 4.11.
4.1.6 The Total Number of Sperm Produced

As shown in figures 4.12 and 4.13 there is a very similar effect of \( n \) on the ESS \( \tau \) under a risk sperm competition as there was of \( n \) on the optimal \( \tau \) in the absence of sperm competition. It is interesting to note that although the effect of \( n \) on the ESS \( \tau \) is quite similar as it was in the absence of sperm competition, it appears to occur much more quickly. This happens because the ESS \( \tau \) is much higher under sperm competition than in its absence as fertility is more important under sperm competition. As the number of sperm produced increases, however, this elevated importance is more quickly overwhelmed by the fact that large numbers of sperm mean that some sperm will most likely be of very high fertility (will contain few phenotypic defects) just due to chance alone. Since sperm fertility is so important when \( n \) is small and when sperm competition may occur, there is a more extreme change in the ESS \( \tau \) under some competition than there is in the optimal \( \tau \) in the absence of competition. As noted previously, it is important to realize that although the ESS \( \tau \) drops off very quickly, it still approaches a value that is much higher than the optimal \( \tau \).
when no sperm competition occurs.

Figure 4.12: The ESS $\tau$ value as depends upon $n$, the total number of sperm produced for a single ejaculate. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $p_{\text{comp}} = 0.2$)
Figure 4.13: The ESS distribution of sperm types as depends upon \( n \), the total number of sperm produced for a single ejaculate. (Evaluated at \( s = 0.9, \mu = 0.05, \alpha = 5, \lambda = 0.2, p_{\text{comp}} = 0.2 \))

4.2 Nonlinear Sperm Fertility Function

Using the fertility function, (2.2), we also obtain a convergent ESS \( \tau \) point as can be seen in figure 4.14. Thus, when sperm fertility is represented by (2.2), a population with some resident value of \( \tau \) that is not the ESS will evolve towards the ESS, and, once there, it will remain at that ESS.
Figure 4.14: The pairwise invasibility plot using (2.2). The blue shading indicates regions where the mutant strategy ($\tau$) can invade the resident strategy ($\tilde{\tau}$), the dashed lines represent the boundaries of these regions (where the fitnesses of the the mutant and the resident strategies are the same), and the red line indicates where the resident and the mutant strategies are the same. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$, $p_{comp} = 0.2, m = 100$)

It is worthwhile to note that the effect of the various parameters on the ESS value of $\tau$ is generally much the same with the use of (2.1) to represent sperm fertility as it is with the use of (2.2) for fertility except that the effect of each parameter is usually much stronger when (2.2) is used. This is also quite similar to the difference between (2.1) and (2.2) in the absence of sperm competition. Because such similarity exists, I will only provide a detailed discussion of the effects those parameters that are sufficiently different with (2.2) from their effects with (2.1) in the next few sections.
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4.2.1 The Probability of Sperm Competition

As when the linear sperm fertility function was used (section 4.1), the ESS $\tau$ value increases with the probability of sperm competition. This is as expected because male fitness depends upon sperm fertility more strongly under sperm competition. Unlike when the linear sperm fertility function was used, however, the ESS $\tau$ does not increase in a linear fashion with (2.2). Instead, it appears to stay approximately the same for low $p_{\text{comp}}$ values, but then increases linearly with $p_{\text{comp}}$ for higher $p_{\text{comp}}$ values (figure 4.15). This happens because male fertility (and so fitness) depends more strongly upon the number of phenotypic defects that are corrected (or avoided) when (2.2) is used than when (2.1) is used. In other words, the number of phenotypic defects avoided in sperm impact male fertility to a greater degree when (2.2) is used for sperm fertility. Because investments of time to sperm production ($\tau$ investments) confer diminishing returns, small decreases in $\tau$ have very large impacts at low $\tau$ values. So, when $p_{\text{comp}}$ is very low and male fertility is less important, males cannot decrease the amount of time that they spend on sperm production ($\tau$) without a drastic impact on their fertility. Thus, when the risk of sperm competition is relatively low, the ESS $\tau$ does not change very much. Conversely, as changes in $\tau$ matter less in terms of male fertility when $\tau$ is relatively large (which happens when the risk of sperm competition is high), so the ESS $\tau$ changes in accordance with the risk of sperm competition in order to maximize male survival.

The same trend is demonstrated (4.16); the distribution of sperm types shifts towards higher quality sperm as the risk of sperm competition increases, but changes very little for low risks of sperm competition.

Once again, in the next few sections, note that the ESS $\tau$ values are always higher than the optimal $\tau$ values were in the absence of sperm competition because male fertility is more important under sperm competition.
4.2.2 The Death Rate Between Matings

The relationship between the rate of death between matings ($\mu$) and the amount of time that a male spends on sperm production (the ESS $\tau$ value) is nearly identical with the use of (2.2) for sperm fertility as it was with (2.1) for sperm fertility; the ESS $\tau$ value decreases as $\mu$ increases, but this effect diminishes as $\mu$ becomes larger. This is shown in figure 4.17.

Although the effect that $\mu$ has on the distribution of sperm types is much the same with (2.2) as it was with (2.1), it is more clearly seen when (2.2) is used because this distribution is further towards higher fertility sperm types. This pattern is displayed in figure 4.18.

4.2.3 The Probability That a Male Survives Mating

As the probability that a male survives a mating event ($s$) increases, the ESS $\tau$ value decreases in a nearly linear fashion, as we saw when equation (2.1) was used for sperm
fertility under a risk of sperm competition. However, when \( s \to 1 \), or when males always survive a mating event, this effect begins to diminish slightly (figures 4.19 and 4.20). This occurs, again, because of the relationship between male fertility and the ESS \( \tau \) value. When the ESS \( \tau \) value becomes relatively low, small decreases in the amount of time spent on sperm production confer very large fertility costs. Thus, when the ESS \( \tau \) becomes small, it cannot decrease very much without very large decreases in fertility. So, when \( s \to 1 \), the ESS \( \tau \) does not decrease as quickly as the trend for the rest of the range of \( s \) suggests.

4.2.4 The Amount of Time Spent Between Mating

As the amount of time that a male spends between each reproductive event increases, so does the importance of each reproductive event because he can participate in fewer such events. In general, this relationship between the \( \alpha \) value (the amount of time a male spends between reproductive events) and the ESS \( \tau \) value (the amount of time a male devotes to sperm production) is much the same with the use of (2.2) to represent sperm fertility and
under competition as it was in the absence of competition or with (2.1). So, although the ESS $\tau$ value increases with $\alpha$, this increase diminishes as $\tau$ becomes large because of the diminishing returns associated with investment into $\tau$. The sole unique feature that arises from (2.2) under competition is that the ESS $\tau$ value does not become as small at very low $\alpha$ values as we would expect. This happens because sperm fertility is extra important to male fitness when the nonlinear sperm fertility function is used under sperm competition. So, while the ESS $\tau$ changes with $\alpha$, it changes less at the low end of its spectrum. In other words, because sperm fertility is so important under competition, the ESS $\tau$ value cannot become too low as this would confer very low fitness. Thus, when $\alpha$ is very small, the ESS $\tau$ does not follow the same trend as it does for larger $\alpha$ values, and instead, remains slightly higher than the trend would suggest.
Figure 4.18: The ESS distribution of sperm types as depends upon $\mu$, the death rate when between reproductive events. (Evaluated at $s = 0.9$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$, $m = 100$, $p_{\text{comp}} = 0.2$, $m = 100$) The effect that $\mu$ has on the full distribution of sperm types is shown in part (a), while only the bottom portion of the distribution is given in part (b) in order to make the trend more evident.

The relationship between $\alpha$ and the distribution of sperm types is exactly what we would expect given the changes in the ESS $\tau$ value with changes in $\alpha$; the distribution of sperm types tends to shift towards higher fertility as $\alpha$ increases (figure 4.22). When $\alpha$ increases, so does the ESS $\tau$ value, and hence, so does the probability of any spermatozoa having high fertility. The distribution, then, will shift towards higher fertility as $\alpha$ increases. As with the ESS $\tau$ value, the effect that $\alpha$ has upon the distribution diminishes as $\alpha$ becomes relatively large, and is also less potent when $\alpha$ is very small.

4.2.5 The Error Repair Rate

As was the case when sperm competition was absent, the ESS $\tau$ value tends to decrease as the error repair rate ($\lambda$) increases. For low $\lambda$ values, we see much the same pattern between the ESS $\tau$ value and $\lambda$; $\tau$ tends to decrease in proportion to $\frac{1}{\lambda}$. As before, this occurs because the number of errors repaired results from the product of the error repair rate ($\lambda$)
and the amount of time spent repairing (τ), so as λ increases, τ will decrease in such a way that the number of errors repaired is relatively constant. However, unlike in the absence of sperm competition, this trend does not hold when λ becomes larger (figure 4.23). As with the use of (2.1), once λ becomes large enough, and the ESS τ becomes small enough, the increased need for sperm fertility under sperm competition begins to dominate and so τ decreases much more slowly than $\frac{1}{\lambda}$ does. This occurs because, once again, investments into τ confer diminishing returns, and so, decreases in small τ values have drastic effects on sperm fertility. When sperm competition occurs, sperm fertility is very important, so males cannot “afford” such a drastic decrease in sperm fertility.

There is a major difference between (2.1) and (2.2) here. Recall that the ESS τ value started decreasing more slowly than $\frac{1}{\lambda}$ very early (figure 4.10). As figure 4.23 demonstrates,
this change in the rate at which the ESS $\tau$ value decreases happens much later when (2.2) is used. This is because when (2.2) is used, the fertility gain that a male receives for an investment of time into sperm production ($\tau$) diminishes more quickly than when (2.1) is used to represent sperm fertility. So, the ESS $\tau$ value, in general, is much larger when (2.2) is used than when (2.1) is used. Thus, it takes longer for the ESS $\tau$ value to decrease to the point at which fertility becomes so constraining that the ESS $\tau$ value can no longer decrease with $\frac{1}{\lambda}$. 

Figure 4.20: The ESS distribution of sperm types as depends upon $s$, the probability of a male surviving a reproductive event. (Evaluated at $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $n = 1000$, $p_{comp} = 0.2$, $m = 100$)
Figure 4.21: The ESS $\tau$ value as depends upon $\alpha$, the amount of time a male spends between mating events. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\lambda = 0.2$, $n = 1000$, $p_{\text{comp}} = 0.2$, $m = 100$)

Figure 4.23: The ESS $\tau$ value as depends upon $\lambda$, the error repair rate. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $n = 1000$, $p_{\text{comp}} = 0.2$, $m = 100$)
As with in the previous sections, the distribution of sperm types actually shifts towards *increased* fertility as $\lambda$ increases because when the error repair rate increases, so does the probability that any given sperm cell is of higher fertility (figure 4.24). Although the ESS $\tau$ does decrease with increases in $\lambda$, this is more than compensated by the increased probability of sperm being of high fertility that higher $\lambda$ values confer.
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Figure 4.24: The ESS distribution of sperm types as depends upon $\lambda$, the error repair rate.

(Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $n = 1000$, $p_{\text{comp}} = 0.2$, $m = 100$)

4.2.6 The Total Number of Sperm Produced

The effect that the total number of sperm produced ($n$) has on the ESS $\tau$ value is much the same as it had in the absence of sperm competition and when (2.1) was used to represent sperm fertility (figure 4.25). However, it is interesting to note that the effect of changing $n$ is much less extreme with (2.2) than with (2.1), and when sperm competition is present than when it is absent. Interestingly, this trend is the opposite to that of the other parameters. This occurs because equation (2.2) places more importance on the number of phenotypic defects corrected (or avoided) than (2.1) does. So, although a male can expect that at least some sperm may turn out to have high quality by chance, he cannot rely as heavily on chance to ensure that some of his sperm are of high quality when (2.2) is used. Moreover, a positive risk of sperm competition serves to increase the importance of sperm fertility, accentuating this trend. Thus, although the ESS $\tau$ value decreases with $n$ it does so much less than when sperm competition was absent or when (2.1) was used to represent sperm fertility.
Figure 4.25: The ESS $\tau$ value as depends upon $n$, the total number of sperm produced for a single ejaculate. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $p_{comp} = 0.2$, $m = 100$)

Also note that, as with other parameters, the distribution of sperm types is shifted towards higher fertility sperm types (figure 4.26) under the risk of sperm competition and with the use of (2.2) instead of (2.1).
Figure 4.26: The ESS distribution of sperm types as depends upon $n$, the total number of sperm produced for a single ejaculate. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $p_{\text{comp}} = 0.2$, $m = 100$)

4.2.7 The Maximum Number of Effective Phenotypic Defects

The effect that $m$, or the maximum number of effective phenotypic defects possible, has on the ESS $\tau$ value is nearly identical to its effect in the absence of sperm competition. The only difference exists in that the ESS $\tau$ values are slightly higher than the optimal $\tau$ values were in the absence of sperm competition (figures 4.27 and 4.28). This is due to the fact that fertility is more important under sperm competition, so greater weight is placed on investments in $\tau$ than in the absence of sperm competition.
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Figure 4.27: The ESS $\tau$ value as depends upon $m$, the total number of sperm produced for a single ejaculate. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $p_{comp} = 0.2$, $n = 1000$)

Figure 4.28: The ESS distribution of sperm types as depends upon $m$, the number of phenotypic defects that confers zero fertility. (Evaluated at $s = 0.9$, $\mu = 0.05$, $\alpha = 5$, $\lambda = 0.2$, $p_{comp} = 0.2$, $n = 1000$)
Chapter 5

Discussion

The model presented in this thesis is designed to provide a theoretical framework for sperm production that explains why variation in sperm quality differs among species. In particular, recent studies have noted that the variance in sperm quality traits appears to decrease under increased sperm competition ([10, 23]). Thus sperm competition may provide sexual selection for sperm quality traits. Moreover, this selection in some way decreases the variability in sperm, suggesting that variability in spermatozoa is detrimental to males, serving only to reduce their fertility. Thus, evidence seems to indicate that males gain a fertility advantage by reducing the variability of their sperm. The most obvious explanation for this is that variability in sperm comes from harmful defects that may be avoided somehow (as seems to occur under increased selection via sperm competition). The fact that males of some species do produce highly uniform sperm, while others under less sperm competition produce more variable sperm also hints that avoiding harmful defects in sperm comes at some cost to males.

The Atlantic Salmon provides one of the best examples that differing sperm production strategies can be selected for under different circumstances ([35]). In these salmon, the subordinate males only mate by sneaking a mating while a female is mating with a dominant male. As a result, those subordinate males always experience sperm competition, and they
produce sperm that are highly fertile. Conversely, dominant males, who control females and often mate with little sperm competition, produce sperm that are not nearly as fertile, on average ([35]). It appears, then, that selection is acting on the subordinate males to increase the fertility of their ejaculates. The increase in fertility that subordinate males enjoy does seem to come at a cost, as subordinate males expend around twice as much energy during sperm production than dominant males. Unfortunately no direct measure of variance in sperm fertility or quality has been provided for Atlantic Salmon, but subordinate males do appear to produce a much higher proportion of sperm that are motile and metabolically well equipped ([35]) suggesting that these sperm are less variable.

5.1 Assumptions

One major assumption upon which this model relies is that transcriptional errors occur during sperm production and these errors can lead to defects that harm sperm fertility. This certainly seems a reasonable assumption as transcriptional errors do occur and cause defects in other products of transcription. Moreover, sperm function is highly dependent upon sperm morphology ([12, 16, 18, 27]), so phenotypic defects in sperm cells have more impact on the function of those cells than defects in other cells have. In other words, phenotypic defects in spermatozoa are probably more apparent than defects in other cell types because they impact fertility directly. That such elevated variability among spermatozoa does exist ([5, 9, 10, 15, 19, 21, 23, 25, 27, 29, 31, 35]) hints that this variability is, in fact, due to defects in sperm phenotype.

This model also assumes that there is some way for males to either screen for errors during sperm production or to slow down production and avoid errors. Some mechanisms have been suggested for screening for errors during transcription, and for repair once those errors are detected ([4, 22, 24]). Thus, it does seem reasonable to assume that transcriptional errors can be avoided, especially because most transcriptional products are far less variable.
than spermatozoa are ([23]).

We must also assume that defects in the transcription of fewer than half the loci involved in sperm production will usually cause sperm to be nonfunctional. In other words, we must assume that nonfunctional sperm contain (on average) transcriptional errors representing less than half the genome that is involved in sperm production. As the specific loci that are involved in spermatogenesis have not been determined, this assumption is difficult to check. However, it is reasonable that defects representing even a small proportion of the genes involved in spermatogenesis will produce nonfunctional spermatozoa. If this is the case, then this model does predict that with increased sperm quality we will see decreased variability, which would explain the trends seen in other studies ([10, 23]). However, if more than half the genome involved in sperm production must usually be corrupted to produce non-functional sperm, then this model actually predicts the opposite: increasing the quality of sperm will increase the variability of those sperm.

5.2 Predictions

This model allows one to make several testable predictions about sperm production. In particular, it provides predictions as to how much investment into sperm production a male of a given species will make, how varied those resulting sperm will be, and whether those sperm will be of high or relatively low quality. The predictions that can be derived from this model are summarized below in table 5.1. These predictions are best tested in a comparative study; they allow one to predict when the sperm from one species will be more varied than the sperm from another species and so on.
### Table 5.1: Predictions derived from this model

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<th>Trait</th>
<th>Predicted Time Spent Producing Sperm</th>
<th>Predicted Variability in Sperm</th>
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<td>high mean sperm quality</td>
<td>long</td>
<td>low</td>
</tr>
<tr>
<td>low mean sperm quality</td>
<td>short</td>
<td>high</td>
</tr>
<tr>
<td>high sperm competition risk</td>
<td>long</td>
<td>low</td>
</tr>
<tr>
<td>low sperm competition risk</td>
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</tr>
<tr>
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</tr>
<tr>
<td>internal fertilization</td>
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<td>high</td>
</tr>
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<td>high</td>
</tr>
<tr>
<td>low rate of death between matings</td>
<td>long</td>
<td>low</td>
</tr>
<tr>
<td>high rate of death during mating</td>
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<td>low</td>
</tr>
<tr>
<td>low risk of death during mating</td>
<td>short</td>
<td>high</td>
</tr>
<tr>
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<tr>
<td>short time spent between matings</td>
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</tr>
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<tr>
<td>large numbers of sperm produced</td>
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</tr>
<tr>
<td>small numbers of sperm produced</td>
<td>long</td>
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</tr>
</tbody>
</table>

### Mean and Variance in Sperm Quality

The first prediction that comes from this model arises directly from the framework introduced in chapter 2. As discussed above, variability in sperm traits (or quality) comes from phenotypic defects in spermatozoa. So, sperm that are highly variable are the result of many different such defects. Those sperm, then, are of low mean quality. Conversely, sperm that are less variable contain fewer defects, and so, are of higher quality. Thus, species that produce highly variable sperm should have relatively poor quality sperm, and species that produce mostly uniform sperm should produce higher quality sperm. This prediction is best tested by looking at very closely related species or different populations of the same species.
Sperm Competition and Variance in Sperm Quality

According to this model, species under high levels of sperm competition (or sexual selection) should produce sperm that are much more uniform than those under low or no sperm competition. Thus, one can predict that monogamous species will produce sperm with more variability than polyandrous species. The best evidence that this is the case comes from the passerine birds. As both [10] and [23] have noted, there appears to be a negative relationship between the risk of sperm competition and the variability of sperm among these birds. Despite these examples, however, this prediction needs further testing, and is easily testable across large groups of species that need not be closely related.

Internal or External Fertilizers

Recall that the linear sperm fertility function (2.1) was designed to represent the fertility of sperm in an internally fertilizing species while the nonlinear sperm fertility function (2.2) represented the fertility of sperm in externally fertilizing species. The key difference between these functions was that avoiding phenotypic defects in sperm was much more important under the nonlinear fertility function than it was under the linear function. In other words, sperm quality is much more important to males that release their sperm into an external environment than those that release sperm into a female’s reproductive tract. This is somewhat intuitive as only a small proportion of sperm released into an external environment may ever come close to ova while internally fertilizing sperm are usually released in close proximity of ova.

According to this model, then, species that fertilize externally should produce more uniform sperm, or a higher proportion of sperm that are of good quality. Of course, this can only be tested using species for which all of the other parameters of this model are relatively similar. In particular, externally fertilizing species often produce vast numbers of sperm. As discussed below, species that produce large numbers of sperm need to be less
“concerned” about the quality of sperm than those that produce only a few sperm. So, the fact that externally fertilizing species produce often produce large numbers of sperm may partially compensate for the additional need to produce high quality sperm that external fertilizers should experience. In order to test this prediction, then, we must be sure to control for sperm production traits such as the number of sperm produced in an ejaculate.

Efficiency of Defect Repair/Avoidance

When defect repair (or avoidance) is very efficient, males should invest less time into their sperm production than when it is less efficient. However, the decreased in investment of time into sperm production that comes with increased repair efficiency should not completely compensate for this increased efficiency. So, those species for whom transcript repair occurs quickly will produce the least variable sperm while those that must spend a long time repairing transcriptional errors will produce more variable sperm. Ideally, this prediction is best tested by comparing closely related species, one of which contains some transcript repair mechanism that the other does not. As the mechanisms involved in transcript repair have not been determined or described, this prediction cannot be tested as of yet.

The Total Number of Sperm Produced

Those species that produce extremely large numbers of sperm can “afford” to have low average quality of their sperm. This model predicts that these species will produce the most variable sperm while those that produce fewer sperm will also produce more uniform sperm.

It may be more relevant to discuss, here, the number of sperm produced *per ovum available* (on average) than the total number of sperm produced. Then those species with the most sperm redundancy (the most sperm per ova) should produce the most variable sperm while those with little sperm redundancy should produce sperm with less variability.
5.3 Future Directions/Implications

This study was conducted to provide a theoretical framework that could explain why species experiencing high levels of sperm competition seem to produce sperm with lower variability than those with less sperm competition. This pattern has been observed in only a few species and should be confirmed in a wider variety of species. Besides testing the other predictions listed above, determining which genes are involved in sperm production would be very valuable to this model. Also, the mechanisms involved in transcript repair are not completely understood and should be studied in further detail.

A major implication of this study is that producing highly variable sperm may simply represent an alternative strategy to sperm production. Recent concern ([2, 3, 11]) over abnormally formed sperm within human ejaculates may not be warranted; the optimal strategy for human males may simply be to produce relatively variable sperm at low relative cost, as would be expected when the intensity of sperm competition is low. Abnormally formed sperm, then, may just be the result of the human sperm production strategy, not of pollutants in the environment or of poor diet.

This study also has implications for artificial insemination. Among species that produce highly variable sperm it may be important to screen out the best equipped sperm for insemination. Among species that produce relatively uniform sperm, such screening may be avoided without much harm.

This model provides an unique perspective on spermatozoa and on spermatogenesis. In particular, I suggest that variability in sperm traits can be adaptive and selected upon. Production of highly variable sperm may simply be the optimal reproductive strategy for a male, rather than the result of adverse conditions.
Bibliography


Appendix A

Derivation of the Distribution of Sperm Types

Let $P(k, t)$ represent the probability that a randomly chosen sperm from a male’s ejaculate contains $m - k$ phenotypic defects at time $t$ in the sperm production process. If a male devotes minimal ($\tau = 0$) time to sperm production, his sperm will contain $m$ phenotypic defects. So

$$P(0, 0) = 1$$

If a male devotes $t + \Delta t$ time to sperm production, the probability that a randomly drawn sperm from his ejaculate will contain $m$ phenotypic defects is

$$P(0, t + \Delta t) = P(0, t) \times P(\text{no potential defects are corrected (or avoided) in } \Delta t)$$

$$= P(0, t) \times (1 - \lambda \times m \times \Delta t)$$

This expression can be re-written as:

$$\frac{P(0, t + \Delta t) - P(0, t)}{\Delta t} = -\lambda \times m \times P(0, t)$$

Taking the limit as $\Delta t \to 0$ and solving for $P(0, t)$ in this expression, we find the probability that an individual sperm cell has $m$ phenotypic defects at any time $t$ in the sperm production process.
APPENDIX A. DERIVATION OF THE DISTRIBUTION OF SPERM TYPES

process.

\[ P(0, t) = e^{-\lambda m st} \]

Now, consider the probability that a sperm cell has \( m - 1 \) phenotypic defects at time \( t + \Delta t \):

\[
P(1, t + \Delta t) = P(1, t) \times P(\text{no potential defects are corrected (or avoided in } \Delta t)) + P(0, t) \times P(1 \text{ potential defect is corrected (or avoided in } \Delta t))
\]

\[ = P(1, t) \times (1 - \lambda \times (m - 1) \times \Delta t) + P(0, t) \times (\lambda \times m \times \Delta t) \]

Re-writing this expression we get:

\[
\frac{P(1, t + \Delta t) - P(1, t)}{\Delta t} = -\lambda \times (m - 1) \times P(1, t) + \lambda \times m \times P(0, t)
\]

Once again, taking the limit as \( \Delta t \to 0 \) and solving for \( P(1, t) \) gives

\[ P(1, t) = m \times e^{-(m-1)\lambda st} \times (1 - e^{-\lambda st}) \]

In a similar fashion, the probability that a sperm cell contains \( m - 2 \) phenotypic defects at time \( t + \Delta t \) is

\[
P(2, t + \Delta t) = P(2, t) \times P(\text{no potential defects are corrected (or avoided in } \Delta t)) + P(1, t) \times P(1 \text{ potential defect is corrected (or avoided in } \Delta t))
\]

\[ = P(2, t) \times (1 - \lambda \times (m - 2) \times \Delta t) + P(1, t) \times (\lambda \times (m - 1) \times \Delta t) \]

which can be solved to give

\[ P(2, t) = \frac{m \times (m-1)}{2} e^{-\lambda s(m-2)st} \times (1 - e^{-\lambda st})^2 \]

By repeating this process, one can find a general form of \( P(k, t) \), the probability that a spermatozoon is produced with \( k \) potential phenotypic defects repaired. This expression is:

\[ P(k, t) = \frac{m!}{(m-k)!k!} e^{-\lambda s(m-k)st} \times (1 - e^{-\lambda st})^k \]