

**Modeling the evolution of visual sexual signaling,
receptivity, and sexual signal reliability among female
primates**

A Dissertation Presented for the
Doctor of Philosophy
Degree
The University of Tennessee, Knoxville

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August 2017

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To Terry, my future husband and forever life partner

Acknowledgments

First and foremost, this dissertation would not have been possible without the help and guidance of my doctoral advisor Sergey Gavrillets. Ever the hardened mathematician, I learned more from him than I ever thought possible. My research would not be as impactful in the scientific community were it not for his keen mathematical eye, and I will always be the first to admit that. One enters his lab a student and leaves a researcher; I was no different. Despite always having challenging and demanding expectations for me, I would not be the mathematician I am today without him.

I also wish to thank my other committee members: Yu-Ting Chen, Judy Day, Lowell Gaertner, and Timothy Schulze for helping me make this dissertation into what it is. In addition, I wish to recognize all the faculty members I have known at UTK who each helped me in their own way. In particular, I thank Lou Gross, for always being a cheery face in the hall and always caring about my professional development. I also thank NIMBioS and all its many visitors, for providing me an enriching mathematical biology environment during my entire time at UTK.

I wish to thank my colleagues in the UTK Math Department for helping to keep me sane: namely Joe Daws, Khoa Dinh, Betsy Heines, Josh Lipsmeyer, Nate Pollesch, and Eddie Tu, among many others. My multiple labmates over the years not only kept me sane and were my friends, but they also helped to shape my research. These include Jeremy Beaulieu, Caroline Farrior, Sarah Flanagan, Mauricio Gonzalez-Forero, Liz Hobson, Ivan Juric, Mahendra Shrestha, Sergei Tarasov, and - most notably - Jeremy Auerbach, Vicken Hillis, and Matt Zefferman.

I also wish to thank RAND Corporation and the U.S. Department of Defense for allowing me to intern with each and learn what industry mathematicians do. In particular, I thank my two mentors at each, Isaac Porche and Bill Hensley, for showing me both true leadership and how to be technically gifted while still having great people skills.

Finally, I was supported throughout graduate school by research fellowships, first SCALE-IT (an NSF IGERT program) and then an NSF Graduate Research Fellowship. My Ph.D. would not have been possible without these, and I am forever grateful for having received them. All numerical simulations presented here were run on the Volos computer cluster in the Gavrillets lab (NIH grant GM56693); my work would have taken some years longer without the help of this cluster.

Abstract

Communication between individuals of a species happens in myriad ways, especially among primates. Not only are verbal cues important, visual and behavioral cues can be just as important as well. Two non-verbal characteristics of the estrous period in many primate species are visual signs of ovulation and sexual receptivity. Visual signs of ovulation take the form of bright colorations and/or sexual swellings around the female's genital region on/around her time of ovulation. Different primate species also have varying lengths of receptivity, that is, willingness of a female to accept a male and permit copulation. In some species, this length of receptivity is equal to the length of time the female has visual ovulation signs present, while in other species such lengths can be much shorter or longer. Species will also vary in the amount of reliability in such signals, i.e. how closely do a female's visible ovulation signs and period of receptivity line up with her period of fertility? In this dissertation, I use mathematical modeling techniques to help answer each of these research questions relating to the evolution of primate non-verbal sexual communication. In Chapter 1, I show how certain ecological factors, such as increased group size and/or the presence of infanticide, can increase visual ovulation signaling among female primates. In Chapter 2, I show female continuous receptivity and concealed ovulation to be correlated, and how only in groups with visible ovulation signs present would one expect to find a relatively short length of time when the female will be receptive to mating. In Chapter 3, I investigate the evolution of sexual signal reliability, evaluating under what conditions will a female's visual sexual signaling line up perfectly (or not) with her peak time of fertility. Finally, in Chapter 4, I outline a future, related biological problem (the evolution of long-term pair-bonding) which could be addressed with many of the same mathematical methods/models used in the earlier chapters. Together, the results presented in this dissertation use mathematics to give new insight into primate evolution and help to resolve old mysteries surrounding primate non-verbal sexual communication.

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Introduction

0.1. Background

Communication between individuals of many species happens in myriad ways, and primates are no exception. Not only are verbal cues important, visual and behavioral cues can be just as important as well. In particular, such visual and behavioral cues as they relate to sexual reproduction have been of much interest to scientists for decades (Semple and Higham, 2013).

Females of most mammal species, including primates, clearly advertise their peak time(s) of fertility (i.e. the period of time where females are able to conceive and become pregnant) (Hrdy and Whitten, 1987). Such “advertisement” of fertility can be displayed as swellings of a female’s genital regions, bright colorations on or near her genital regions, and/or very noticeable behavioral changes marking fertility (Dixon, 1983, 2012). Two of the most well-known non-verbal characteristics of the estrous period in many primate species are visual signs of ovulation and sexual receptivity (Hrdy and Whitten, 1987).

Different primate species have varying levels of visual ovulation signs among their females (Sillen-Tullberg and Moller, 1993). Different primate species also have varying lengths of receptivity, that is, the willingness of a female to sexually accept a male and permit copulation. In some species, this length of receptivity is equal to the length of time the female has visual ovulation signs present, while in other species these lengths can differ (Hrdy and Whitten, 1987). Species with females lacking any visible signs of ovulation throughout the estrous cycle are referred to as having concealed ovulation (Sillen-Tullberg and Moller, 1993). Species with no defined period of receptivity, who instead have females receptive to mating throughout their entire estrous cycles, are said to have continuous receptivity (Hrdy and Whitten, 1987).

0.2. The Estrous Cycle

The female reproductive system of most mammals includes a quite complicated estrous cycle. This cycle consists of relatively discrete stages, each with varying levels of hormonal changes, behavioral changes, and/or visible physiological changes. In particular, estrous marks the phase when the female is “in heat”, meaning able to get pregnant, or fertile. Biologically, this is typically

the time when ovulation occurs in mammals, i.e. when a mature egg is released from the female's ovary, ready to be fertilized. There is much variability in the estrous cycle, both within and between different species of mammals, and also sometimes within any one female individual herself.

Fig. 0.1 illustrates some of the complexity within the mammalian estrous cycle. The follicular phase marks the portion of a female's cycle where her egg is still in the ovary and growing. Ovulation then marks the precise point when this egg gets released. This is followed by the luteal phase, the beginning of which the female will still be fertile, and then menses. Both ovarian and pituitary hormones play important roles in the estrous cycles, and some primate females who have had their ovaries removed will still be receptive to mating (Dixon, 2012).

Fig. 0.2 illustrates some possible complexities between the hormonal changes discussed above (panel (a)) and changes in visible ovulation signs (panel (b)), changes in receptivity (panel (c)), and male mating frequency (panel (d)). In particular, in (b) receptivity is held constant in a female while her amount of visible ovulation signs ("attractiveness") varies over the course of her cycle. Note increased visible ovulation signs are expected during the female's time of highest fertility (i.e. around ovulation). In (c), visible female signs are held constant (for example, in the case of concealed ovulation) while receptivity varies. Again note that increased receptivity is expected to occur during the female's time of highest fertility (i.e. around ovulation). Finally, (d) shows that males are more likely to mate with females with more visible ovulation signs present (more "attractive") *or* with females more receptive to mating. In both cases, this results in males mating more frequently with females around their time of ovulation (Dixon, 2012). Recall from Fig. 0.1 that ovulation happens just after that peak in luteinizing hormone (LH) illustrated in panel (a). Proceptivity (not discussed further here) is female behavioral patterns that initiate and maintain male sexual interaction (Dixon, 2012).

0.3. Phylogeny

Despite the underlying hormonal and physiological similarities among many primates regarding the estrous cycle, there are still many differences between different species of primates. Such differences include the amount, if any, of ovulation signs being visible, as well as the length of

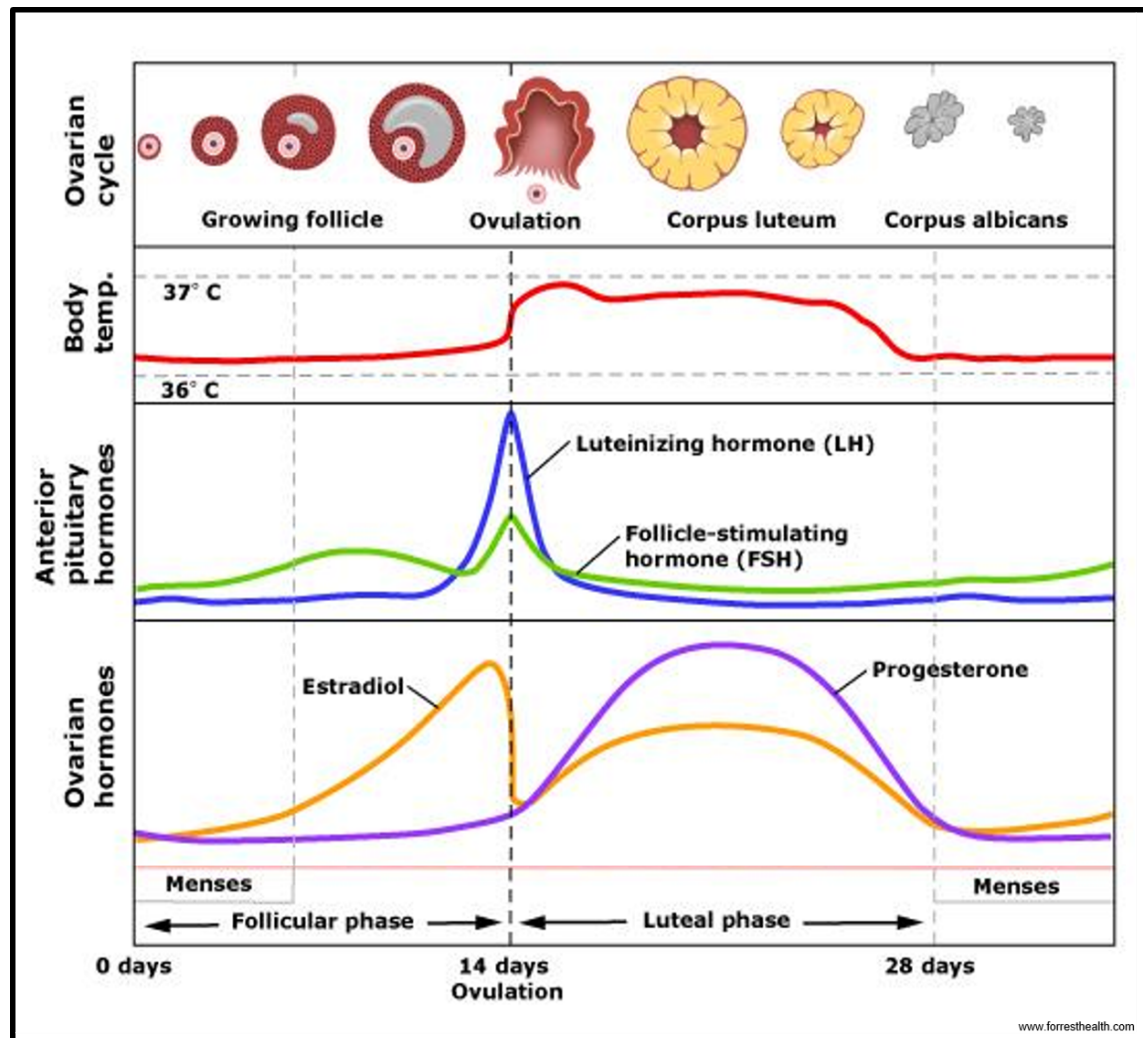


Figure 0.1: Illustration of the complexity of the female estrous cycle, including ovulation and hormonal variation; exact length of cycle here is only an example. Image adapted from: http://sbi4u3.weebly.com/uploads/1/6/4/0/16403200/3972834_orig.jpg

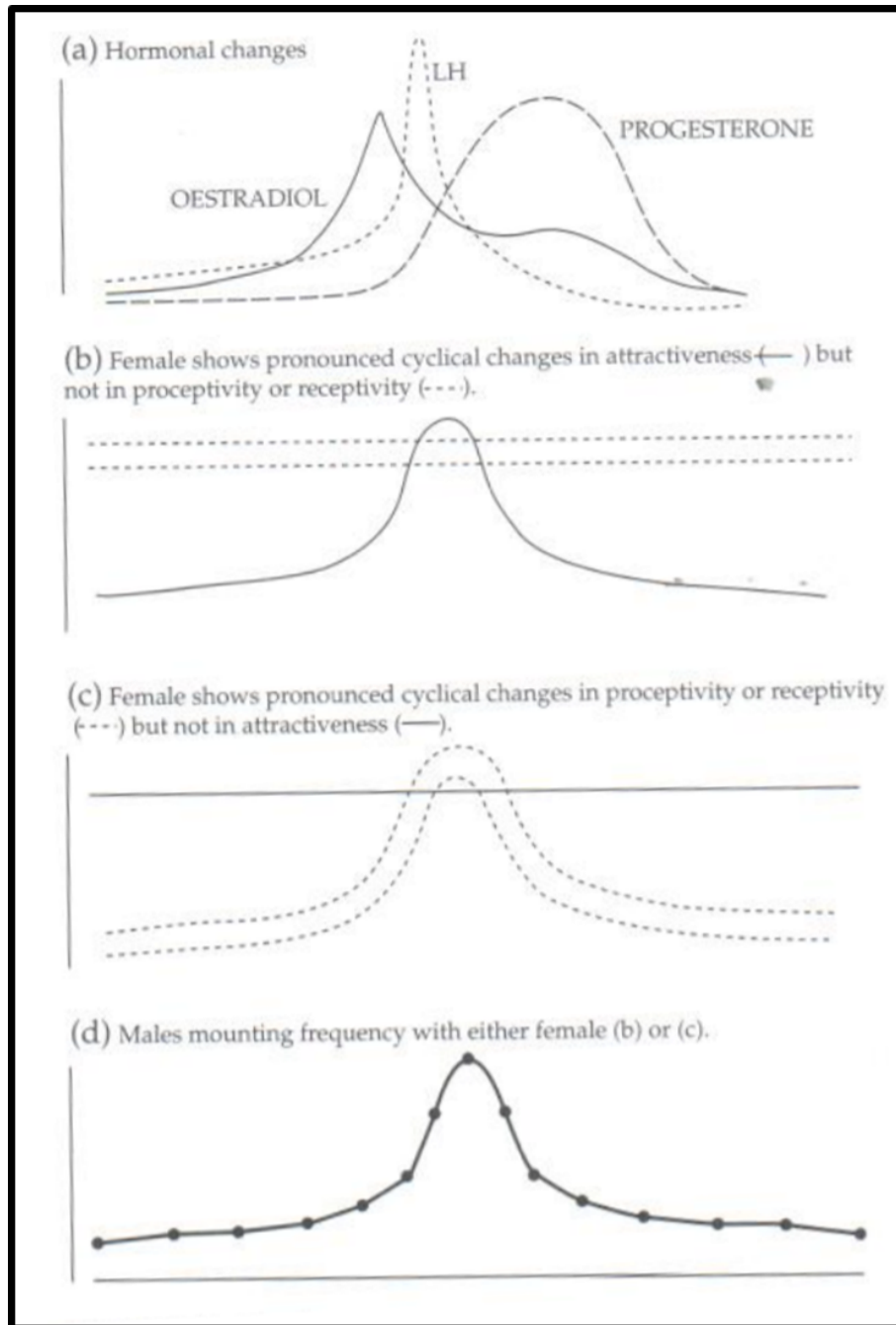


Figure 0.2: Illustration of some possible complexities between the hormonal changes discussed above (panel (a)) and changes in visible ovulation signs (panel (b)), changes in receptivity (panel (c)), and male mating frequency (panel (d)). Recall ovulation happens just after that peak in luteinizing hormone (LH). Image copied from Figure 11.1 in (Dixson, 2012, p.431).

time throughout her cycle that a female is sexually receptive and how reliable these sexual signals are. Some species' females have ovulation signs visible for a majority of their cycle; others have concealed ovulation in which there are *no* visible signs of ovulation at any point in the females' cycles. Some species have continuous receptivity, where they are able to mate throughout the entire estrous cycle; others are only receptive for *hours* (less than one day) of their estrous cycle lasting weeks (Hrdy and Whitten, 1987). Some species have ovulation line up precisely with their day of maximum ovulation signs being visible (e.g. see (Deschner et al., 2004)), while other species show high variability in how fertility and visible ovulation signs match up (e.g. see (Douglas et al., 2016)).

See Fig. 0.3 for a depiction of a possible phylogeny regarding visible ovulation signs. Species are depicted as having absent ("Absent"), slightly visible ("Slight signs"), fully visible ("Sexual swellings"), or unknown ("Equivocal")), as well as the inferred ancestral states. From this, concealed ovulation is estimated to have evolved independently at least 8 times (Sillen-Tullberg and Moller, 1993).

0.4. Hypotheses for the Evolution of Concealed Ovulation

Females of most mammal species very clearly advertise this peak time of fertility. Again, such "advertisement" can be through swellings of her genital region, bright colorations on or near her genital region, and/or very noticeable behavioral changes marking fertility. This clear visual presentation of ovulation is common in not just mammals as a whole, but also more specifically across primates (for example, see Figure 0.3).

With that being said, there are still select species among mammals and primates in particular that have fully concealed ovulation signs. Humans are of course one such species. Despite ovulation being a complicated process physiologically, not only can no one around a human female tell if she is ovulating, not even the female herself ever knows for sure. This uncertainty in the time of ovulation is true on the hormonal level as well. Even with advancements in modern medicine, short of having visual confirmation of the female's egg being released from her ovary, ovulation is very hard to pinpoint in human females (Gangestad et al., 2016a).

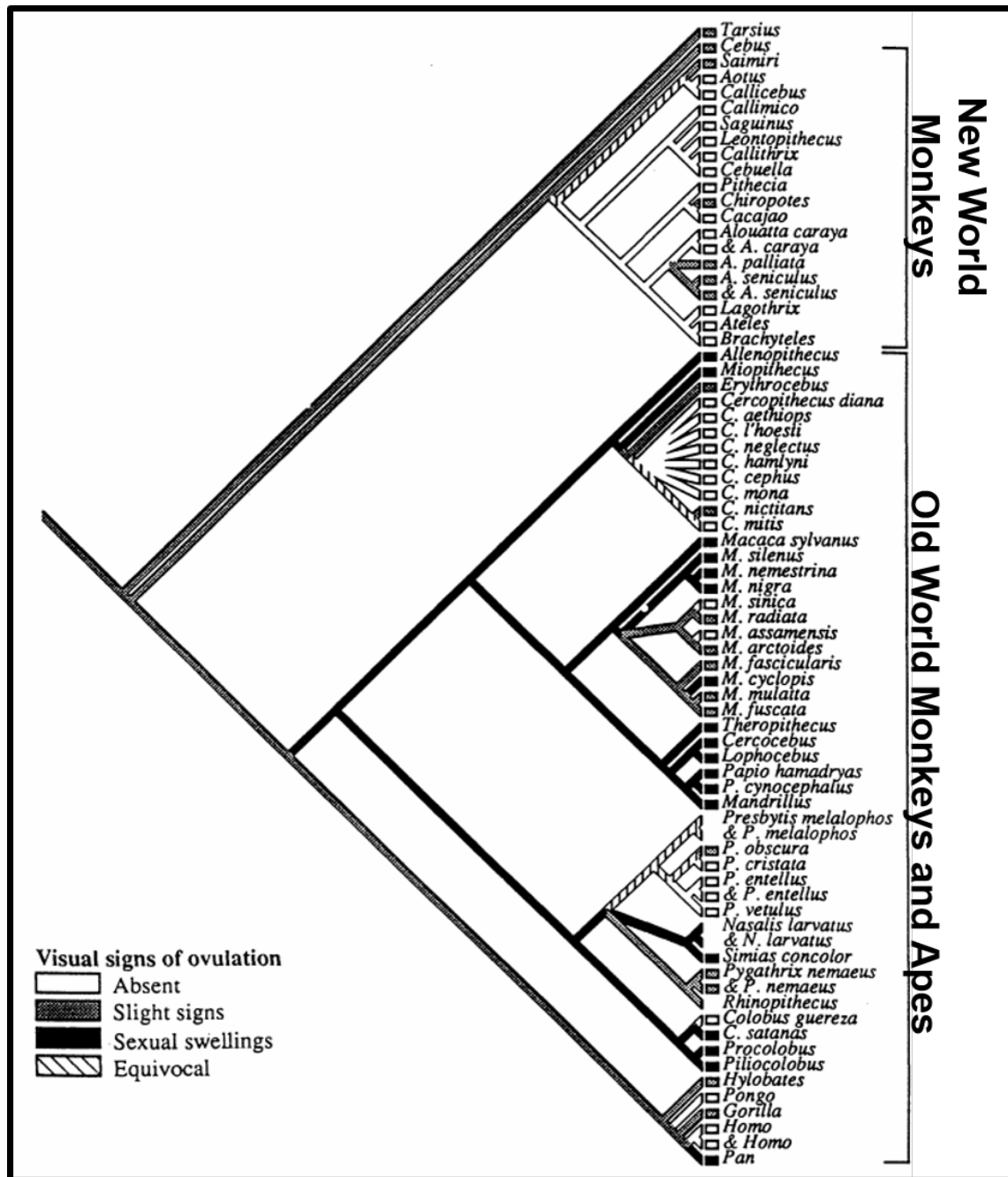


Figure 0.3: Adapted from Figure 2 of (Sillen-Tullberg and Moller, 1993); phylogeny of *Anthropoidea* and *Tarsioidea* depicting the distribution of ovulation signs (classified as one of absent (“Absent”), slightly visible (“Slight signs”), fully visible (“Sexual swellings”), or unknown (“Equivocal”)), as well as the inferred ancestral states

Having ovulation signs fully concealed is quite counter-intuitive from a female fitness perspective, and remains an even bigger mystery. A female who has more visible signs marking her time of fertility would gain more matings during this time, and in particular, more matings from higher-ranking males (since higher-ranking males show more interest in females who are clearly fertile). Such males are presumably of higher genetic quality, and hence would produce offspring also of higher genetic quality. This means that any female with concealed ovulation, in a group of females all with signs very visible, would be at a fitness *disadvantage* compared to all those other females.

This certainly brings up the question, then, of why? If having concealed ovulation signs *decreases* a female's probability of mating and/or *lowers* the quality of the mate she is able to obtain, how did concealed ovulation ever evolve in the first place? This question is nothing new. In fact, it has been argued in the literature since at least the 1940's, in various disciplines such as evolutionary biology, anthropology, and psychology. Many different hypotheses have been put forth in an attempt to explain the evolution of concealed ovulation, with many different researchers over the decades contributing to one or more of these hypotheses.

Below are listed six of these possible hypotheses, in order to get a better idea of the kinds of questions being asked in this field, as many of these ideas will come up again throughout this dissertation. Although these hypotheses are all distinct and come with their own set of assumptions, there is no reason two (or more) of them could not be working in concert. Most of these hypotheses are not mutually exclusive, although some do have contradictory assumptions. Also of note, some of the hypotheses presented below have previously been presented together as a separate hypothesis, have been split up into sub-hypotheses and again presented separately, or have even been presented as hypotheses for why *exaggerated* sexual signals could have evolved (Burt, 1992).

0.4.1. To Support Food-for-Sex

Meat, and hence hunting in order to obtain meat, is a very valuable resource. Since males are typically the predominant hunters, many females rely on males for any substantial meat the females are able to get in their diets. For example, in chimpanzees and baboons, estrous females receive a greater percentage of meat from a male hunt than non-estrous females do. There are also many

anecdotal records of meat being provided by a male in direct exchange for copulation with the female (Hill, 1982).

Since it is in the female’s best interest to have more meat, it would also be in her best interest to copulate with male hunters who bring her meat, and hence for her to have ovulation signs visible for a longer duration within her reproductive cycle than the females around her. As Hill states, “Longer and longer periods of ‘estrous’ were [thus] selected in females, obscuring ovulation and resulting in receptivity to copulation” (Hill, 1982).

0.4.2. Protection against Infanticide

Infanticide is the killing of a female’s offspring prior to weaning. Intuitively, this is extremely detrimental to a female’s fitness (van Schaik et al., 2004; Hrdy, 1979). However, committing an act of infanticide may benefit any male who is not the father of that particular offspring, and hence infanticide is often seen when a new male takes over a group (Pradhan and van Schaik, 2008). Many primate species have prolonged lactational amenorrhoea (van Schaik, 2000), resulting in a male killing a female’s offspring bringing that female back to fertility sooner by prematurely ending her lactation (Lovejoy, 2009; Palombit, 1999). The infanticidal male can then subsequently mate with this female, meaning his offspring will be able to be born sooner than would otherwise be possible had he never committed infanticide (Stallman and Froehlich, 2000). Although infanticidal events are typically rare, they have been documented in many primate species (Hrdy, 1979; van Schaik et al., 1999) and under many different conditions (Clutton-Brock, 2016). In some primate species, male infanticide makes up a substantial proportion of all infant mortality (including over 60%) (Clutton-Brock, 2016; van Schaik et al., 1999).

A species whose females had ovulation concealed would make it easier for a female to attract more than one mating partner throughout her cycle, and hence mate with more than one male during any one of her fertility cycles. Since it has been shown in primates (and even rodents) that males are both: (1) able to accurately assess their approximate probability of paternity for any given offspring, and (2) much less likely to commit infanticide against an offspring of which that male has a positive paternity probability, the higher the number of males a female mates with

during a given fertility cycle, the increased protection her offspring would have against infanticide. It is in this way that infanticide, by greatly increasing the benefit females receive from mating with multiple males during any given cycle, can cause concealed ovulation to increase a female's fitness, and hence serve as selection pressure for concealed ovulation (Hrdy, 1981).

0.4.3. To Reduce Crude Female Contraception

Despite there being many different hypotheses as to why concealed ovulation may have evolved, few take into explicit account the fact that not even the female herself knows when she is ovulating. For most other hypotheses, the fact that the female herself is unaware requires either coincidence or some additional evolutionary explanation (such as an inability to effectively deceive). This hypothesis, however, does explicitly take females' knowledge into account. It states that concealed ovulation evolved as a way to reduce deliberate female contraception. The main proponent of this hypothesis is Nancy Burley (Burley, 1979).

Childbirth has always been a costly process. In early humans, many women died during childbirth, and even among those who survived, many suffered lasting health complications and all suffered great pain. As the early human brain got bigger, not only was childbirth an even riskier and more painful process (since the larger infant head still needed to fit through the small female birth canal), it allowed women to realize that such pain and possible death were in fact quite avoidable.

This hypothesis states that as females began realizing, either in themselves or witnessing it in other women, the connection between sexual intercourse during ovulation and getting pregnant (which then ultimately led to that pain and risk of childbirth), they began actively avoiding having sex during those times of fertility. However, successfully avoiding copulation during ovulation effectively gives a woman zero relative fitness since she would then have no genes being passed on to the next generation. Therefore, females unable to accurately determine their times of fertility would actually be at a fitness *advantage* compared to their counterparts (even though they themselves would be viewing such an outcome as a disadvantage).

It is in this way that concealed ovulation could have evolved over time. Females with diminished

ovulation signs would have a harder time discerning when they were and were not fertile. This in turn would increase their fitness compared to females with fully visible signs since these individuals would be more likely to have sex while fertile than any of the other females. Over evolutionary time, females practicing deliberate contraception would provide sufficient selection pressure for concealed ovulation to evolve.

0.4.4. To Escape Mate-Guarding

One of the most widely cited hypotheses for the evolution of concealed ovulation is concealed ovulation helping females to escape mate-guarding. Although this hypothesis comes in a variety of forms with different researchers supporting several different interpretations of it (for example, see (Schroder, 1993), (Benshoof and Thornhill, 1979)), each of these interpretations essentially boils down to the same underlying hypothesis, presented here.

If a male wants to ensure that he is indeed the father of a female's offspring, he will need to be with ("guard") that female throughout her entire fertile window. If her time of ovulation is clearly apparent, the male would know precisely when he should guard her in order to maximize his chances of paternity. Since this peak time of fertility is only a 3-7 day window, the male would still have the majority of his month available for other life-sustaining activities such as hunting.

If instead a female's ovulation signs are fully concealed, in order for a male to have that same high confidence of paternity, he would instead need to stay around the female for a now more than 3 week window, a much more compromising task for any male, and hence one much less likely to be achieved. It is in this way that concealed ovulation could help females. A female with concealed ovulation is less likely to be guarded (and hence less likely to be guarded during her fertile window), meaning such females get more choice in their ultimate mate, which could then increase her fitness. As Cann and Wilson say, "Concealment [of ovulation] is the ultimate expression of female choice" (Cann and Wilson, 1982).

The two main varieties this hypothesis comes in are (i) deception of the alpha male (e.g. see (Schroder, 1993)), and (ii) deception of the mate (e.g. see (Benshoof and Thornhill, 1979)). Both varieties make assumptions about the social structure at the time concealed ovulation would have

evolved (see Chapter 4 for more details), and both are rooted in the same idea of a female trying to escape guarding by a male; the key difference lies in *who* she is escaping from. In the first, it is assumed the alpha male can successfully dominate mates, meaning concealed ovulation helps a female to mate with males other than the alpha male (clearly to the benefit of lower-ranked males as well). In the second, it is instead assumed there is a “group-living monogamous social setting” present ((Benshoof and Thornhill, 1979)), meaning concealed ovulation helps promote cuckoldry of the male pair-bond by the female.

0.4.5. *To Cement the Pair-Bond*

Another common hypothesis used to explain the evolution of concealed ovulation is the benefit of having hidden ovulation signs while under a monogamous mating system (e.g. (Strassmann, 1981), (Lovejoy, 1981), (Lovejoy, 2009)). Indeed, there is certainly a correlation between species having concealed ovulation signs and monogamous mating systems (Pagel and Meade, 2006). Ovulation signs are estimated to have been lost 0-1 times in a monogamous context and 8-11 times in a non-monogamous context. Conversely, monogamy is believed to have evolved independently seven times in anthropoid primates; 4-6 of these are believed to be under conditions of concealed ovulation and the other 1-3 times with ovulation signs fully visible (Sillen-Tullberg and Moller, 1993). Although far from conclusive, this phylogenetic analysis suggests that concealed ovulation is more likely to help monogamy evolve than monogamy help concealed ovulation evolve.

While this means phylogeny does not strongly support this particular hypothesis, the hypothesis still remains possible. What this hypothesis hinges on is the fact that females benefit from having a pair-bond (for example, due to provisionings, paternal care, etc.). From the male point of view though, why would a male ever invest his time and resources into the costly (and limiting) act of being in a pair-bond? It is because every male faces a trade-off between gaining copulations from *more* mates and increasing their confidence of paternity by staying with *one* mate. While higher-ranking males are more often able to secure enough copulations to have high confidence of paternity from *some* mate, lower-ranking males are typically not so fortunate. Lower-ranked males may then try to increase their confidence of paternity in other ways, one of which is through forming

a pair-bond.

Hence, since females benefit from being in a pair-bond and lower-ranking males are more likely to form such pair-bonds, it can be in females' best interests to prefer lower-ranking males. If a female instead only mated with high-ranking males (presumably of better genetic quality), she would then lose out on the benefit to her fitness coming from being in a pair-bond. Since the incentive to a male here is increasing paternity certainty, pair-bonding males prefer females with concealed ovulation signs since a male pair-bonded to such a female would be more likely to be her only mate (since she would be viewed as less desirable by all the higher-ranking males).

Thus, just as males face a trade-off between having more mates and having higher paternity certainty, there exists a trade-off for any female's fitness. This female trade-off is between mating with multiple higher-ranked males (who are presumably of higher quality/have better genes) vs. mating exclusively with lower-ranked males who pair-bond (since provisioning/paternal care/etc. positively impacts the health and well-being of a female's offspring). These two sides of the trade-off correlate with the female having *more* ovulation signs present and having *less* ovulation signs present, respectively. See Chapter 4 for a more in-depth discussion of these ideas relating to pair-bonding.

0.4.6. To Reduce Male-Male Competition

Even if females having clearly visible ovulation signs is beneficial to those females' individual fitness, it is not necessarily beneficial for the *group* in which those females are living. In particular, males get much more competitive when there is a clearly-ovulating female around. Males' testosterone levels rise, and fighting between males over mates is much more likely to occur. However, for any multi-male group scenario, this kind of in-fighting within the group is quite costly. It is in this way that concealed ovulation could have evolved: groups containing females with concealed ovulation signs had higher fitness than their counterpart groups with females with clear ovulation signs due to less intra-group fighting/competition. Note this hypothesis would be dependent upon group selection (Wilson, 1975).

0.5. Concealed Ovulation in Human Females

Regardless of how exactly it evolved, humans are routinely labeled as being an example of a species with ‘concealed ovulation’ (e.g. see Refs. (Burley, 1979; Daniels, 1983; Gray and Wolfe, 1983; Schroder, 1993)). However, there is still much debate surrounding that claim (e.g. see Refs. (Marlowe, 2004; Gangestad and Thornhill, 2008; Harris, 2011; Haselton and Gildersleeve, 2011; Harris, 2013; Gildersleeve et al., 2013; Gangestad and Haselton, 2015; Haselton and Gildersleeve, 2016; Gangestad et al., 2016b)). While most people would agree there are no sexual swellings or bright colorations marking human females’ times of ovulations, many say there are still other, more subtle cues. As reviewed in (Gildersleeve et al., 2014a), some cues are visual, such as increased facial and breast symmetry around a female’s time of ovulation, others chemical, such as men preferring the smell of ovulating women, others auditory, such as the pitch of women’s voices, and still others behavioral, for instance, how the female dresses or acts towards men around her time of ovulation.

There have been a multitude of studies conducted in the hopes of answering the many questions relating to all of these cues: Do men react to all of them? Are some more important to men in determining interest? How are these cues different within and outside pair-bonds? Are females cognizant of these changes? Do all females exhibit them, or are there other factors at play? Etc. Perhaps most telling, there were recently two meta-analyses conducted using all of the relevant empirical studies from the past several decades. Despite using primarily the same data, each meta-analysis came to a different conclusion as to whether such cues really do exist (Gildersleeve et al., 2014a; Wood et al., 2014). There then ensued more debate following the apparent contradictions, e.g. see Refs. (Harris et al., 2014; Wood and Carden, 2014; Gildersleeve et al., 2014b), and consensus has certainly not been reached yet.

In addition to conflicting empirical results in many cases, there is also the underlying problem of how to know when precisely ovulation even happens. Often crude estimation methods are used, which could clearly affect the final conclusions being reached. This is a known problem in the field, e.g. see Ref. (Gangestad et al., 2016a).

0.6. Methods

The main mathematical/computational methods used in this dissertation are adaptive dynamics (for the analytical work) and agent-based modeling (for the numerical simulations). Because of the complexity of the models being developed, the main results of this dissertation occur primarily via agent-based simulations. The underlying idea behind such methods is to construct models of reality with the numbers of variables/different strategies being as small as possible, while keeping the main ideas as realistic as possible. Mathematics and/or computational methods can then be applied to the model in order to find the predicted patterns or behaviors/traits coming from the system (Bradbury and Vehrencamp, 2011).

0.6.1. Evolutionary Game Theory

Evolutionary game theory studies the behavior of a large number of individuals (a population) who repeatedly interact in strategic decisions (Hofbauer and Sigmund, 1988, 1998; Sandholm, 2009, 2010; Sigmund, 2011). Changes in behavior within populations over time can occur via natural selection (i.e. differences in birth and death rates between individuals playing different strategies) or via pre-determined myopic decision rules utilized by each individual. The former most often occurs via a ‘replicator dynamic’, i.e. when the percentage growth rate of individuals playing a particular strategy is proportional to the difference between that strategy’s payoff and the population’s average payoff (Sandholm, 2009).

Evolutionary game theory models have been used for decades now to predict evolutionary outcomes under frequency dependence, i.e. when the average payoffs for certain strategies are dependent on the frequencies of each strategy in the population (Hofbauer and Sigmund, 1988, 1998). Under this framework, one is looking for evolutionary stable strategies (ESS), meaning strategies that will always do better than any possible mutant when the mutants are rare. ESS’s may thus consist of a single dominant strategy or a stable mixture of different strategies. In addition to one ESS consisting of multiple strategies, a model may also have multiple ESS’s, in which one strategy could become dominant largely by chance (Sigmund, 2011).

0.6.2. Adaptive Dynamics

Overview

Somewhat similar to evolutionary game theory is adaptive dynamics (Hofbauer and Sigmund, 1990; Dieckmann and Law, 1996; Geritz et al., 1998; Waxman and Gavrillets, 2005). The adaptive dynamics approach allows for the investigation of the long-term evolutionary dynamics of continuously-varying traits. This approach is best used when both a large population size and small mutation rate are valid assumptions. Adaptive dynamics explicitly takes into account resident-mutant demographic interactions, or the dynamics of resident and mutant population densities for any given value of the traits. Such interactions are predicting the outcomes should those resident and mutant individual(s) be in the same population together, as in the process of natural selection. In doing so, adaptive dynamics uses an ODE system to define the fitness landscape, provide the evolutionary dynamics, and ultimately allows for evolutionary branching, extinction, and the prediction of any changes in the trait distributions between the parent and offspring generations. Doing so can help predict where an initial population would be after many generations of evolution. Note that adaptive dynamics inherently includes both frequency- and density- dependent effects (Dercole and Rinaldi, 2008).

Not all frequency-dependent models will have stable equilibria, and adaptive dynamics approaches allow for both stable and unstable equilibria to be discovered; such ‘more-inclusive equilibria’ are termed evolutionary singular strategies (ess). Branching points, limit cycles, bifurcations, and chaotic trajectories can all be found using adaptive dynamics. With branching points, one evolutionary trajectory gets divided into two or more different trajectories. Limit cycles result in recurrent cycling between two or more different strategies. Bifurcations result in an evolutionary trajectory having a radical change in state. Finally, chaotic trajectories will never retrace their steps, nor ever converge. While such results may seem quite complex, such dynamics have been shown to closely resemble nature in some systems (Bradbury and Vehrencamp, 2011). In addition, the adaptive dynamics approach has been successfully used to model human and primate systems (for example, see Ref. (Gavrilets, 2012a)).

Mathematical Details

In the simplest scenario of adaptive dynamics, there exists some monomorphic population (termed “residents”), all with the same phenotypic trait value. A mutation (meaning a phenotypic trait value different from this resident population trait value) then gets introduced into the population. Adaptive dynamics is interested in the dynamics of this introduced trait value over evolutionary time, and whether or not the mutant would eventually be able to ‘invade’ the resident population (Waxman and Gavrillets, 2005).

In order to conduct such an analysis, resident type fitnesses are typically assumed to be zero. Intuitively, this assumption makes sense, as populations are bounded, meaning over the long-term, populations will eventually converge to some equilibria, limit cycle, etc. Any mutant introduced, when looking at the short-term, will most likely have some non-zero fitness. It is in this way that mutant fitness will affect the direction and speed of evolution (depending on the exact sign and magnitude of this fitness), as the population gradually moves back towards some, possibly different, equilibria (Dercole and Rinaldi, 2008).

This idea is highly relevant to, and can be thought of as, movement of genotypes along an adaptive landscape. When fitness depends solely on trait values, and not on population density and/or trait frequency, the corresponding fitness landscape will be static. However, in the case of fitness having density- and/or frequency- dependence, the corresponding fitness landscape will be highly dynamic as fitnesses will change as the population composition also changes. In contrast, an *invasion* fitness landscape illustrates these ideas better, by instead plotting a 3D fitness surface with respect to both the resident and mutant trait values (Waxman and Gavrillets, 2005).

Evolutionarily singular strategies are, in a sense, the equilibria of evolution, even if not necessarily stable. They result from any mutants differing from the resident type all having negative fitness, meaning one would expect the entire population to, in time, become the resident type. This idea can be extended to there being more than one resident type present, and is then instead referred to as an evolutionarily steady coalition (esc) (Dercole and Rinaldi, 2008).

In general, adaptive dynamics is a useful technique for predicting the optimal trait strategies corresponding to any given environment. Note this is quite different from being able to predict the

outcome of evolution itself. In order to actually predict the course, one would also have to know how environments vary over time, a much more highly-stochastic process. Another key problem of adaptive dynamics is the number and type of assumptions which must be made. As with approximating any type of complexity, there will always be a trade-off between predictive/descriptive power obtained and mathematical simplicity (Dieckmann and Law, 1996).

To gain better intuition for what an ess is, consider only one mutant and one resident type. The maximum of the invasion fitness landscape is denoted $\rho(y|x)$, where y is the invading mutant type and x is the resident type of the trait-value community in which y is invading. ρ fitness curves can be plotted for varying values of potential mutants and resident types (meaning they can be overlaid on an $x \times y$, or resident vs. mutant trait values, plot). The optimal strategy for the resident environment is then calculated, and can be thought of as being overlaid on top of the $x \times y$ fitness grid (Dercole and Rinaldi, 2008).

Graphically, this optimal strategy will follow the maxima of all the ρ fitness curves plotted earlier. Considering the simple $y = x$ line running through this diagram, an ess will be any intersection of the $y = x$ and optimal strategy lines. Notice that it is at these precise points where $x^* = y^*$, and any other mutants (meaning with $y \neq y^*$) will have negative fitness, as compared to the residents with fitness standardized to zero (Dercole and Rinaldi, 2008). Again, an ess may be an evolutionarily stable strategy (ESS), but is in general a more abstract version of the evolutionary game theory construct (Waxman and Gavrillets, 2005).

A key measurement used in analyzing fitness invasions is the local fitness gradient. This is a measurement of the steepness of the invasion fitness landscape at a point, or the predicted change (if any) of the resident population x given some invading mutant y (Waxman and Gavrillets, 2005). Hence this quantity is given by: $D(x) = \left[\frac{\partial \rho(y|x)}{\partial y} \right]_{y=x}$. A positive D value implies that mutants with trait values larger than those of the residents will invade, while a negative D implies that only mutants with smaller trait values will be able to successfully invade. The curve produced by $D(x) = 0$ is in fact a very useful one, and termed an adaptive isocline. Notice that only at evolutionarily singular strategy points (meaning $x = x^*$) will $D(x) = 0$ (Geritz et al., 1998).

At these precise points, since the first derivative is equal to zero, it makes sense that in order

to analyze any evolutionarily singular strategy further, we will need to take second derivatives into account (Waxman and Gavrillets, 2005). In fact, by calculating just $\left[\frac{\partial^2 \rho(y|x)}{\partial x^2} \right]_{x=x^*, y=x^*}$ and $\left[\frac{\partial^2 \rho(y|x)}{\partial y^2} \right]_{x=x^*, y=x^*}$, we can classify an evolutionarily singular strategy into one of eight categories. These categories then show whether x^* is locally stable (i.e. cannot invade a resident population), can instead invade, has convergence stability, and/or allows for the existence of a protected polymorphism (meaning if two strategies making up a polymorphism are able to invade together). Classification into these eight categories provide much insight into the long-term behavior of the model system of interest (Geritz et al., 1998).

0.6.3. Agent-Based Modeling

Another useful, related tool is genetic algorithm modeling. In particular, such models can be implemented via agent-based modeling. This means these models include agents able to collect information about their environment, make decisions (typically following some predetermined rules) about what actions to take, and then actually act. Collections of agents can then be used, all with the possibility of interacting with one another (Kohler and Gumerman, 2000).

For the work presented here, computer simulations are used to track a population of individuals over time. The population will have an initial condition of strategies (for this work, ‘strategies’ are simply genetic trait values). Heritability, random mutation, and differential survival of strategy types are all explicitly built into the model. The population will then be tracked over a large number of generations, until the asymptotic outcome can be known (Bradbury and Vehrencamp, 2011).

Such computational methods are particularly beneficial as model complexity increases. As is the case for the work presented here, finding ESS’s or other stable/unstable equilibria can be challenging for situations with long sequences of exchanges or multiple interactions being needed (Bradbury and Vehrencamp, 2011).

Indeed, these techniques have been successfully applied to human and primate questions in the past (for example, see Ref. (Kohler and Gumerman, 2000)). As Timothy Kohler states, “[Agent-based modeling is] provoking great interest in the dynamics in human and primate societies in ways

that have not previously been possible” (Kohler and Gumerman, 2000, p.1).

0.6.4. Overview of the Genetic Algorithm Used

The following is a pseudo-code example of how the genetic algorithm works that the modeling work of this dissertation will utilize. This general framework is used in each of Chapters 1,2,3.

```

Define global parameters
Define male/female classes
for (each initial condition) {
    Set initial condition values
    for (each generation) {
        for (each group) {
            for (each individual) {
                Assign each male's quality
                for (each day of the cycle) {
                    Assign each female's fertility, visible ovulation signs, and receptivity
                    Determine male-female mating pairs on each day
                } % End of cycle-day loop
                Calculate and normalize males' perceived probabilities of paternity
                Calculate and normalize males' actual probabilities of paternity
                Determine the father for each offspring
                Calculate and normalize female fitness
            } % End of individual loop
            for (each offspring produced) {
                Determine which parent's trait value the offspring inherits for each evolving trait
                Determine if a mutation occurs, and if so its effect, for each evolving trait
            } % End of offspring loop
            Determine sex (male/female) of the offspring randomly
        } % End of group loop
        Allow females to migrate between groups
        Records the average trait values
    } % End of generation loop
} % End of initial-condition loop

```

0.6.5. Overview of all Parameters Used

Table 0.1 lists all parameters used throughout this dissertation, including both the parameter name and what it represents in the model. Each chapter will list the particular parameters used within it, as well as their respective values. Some parameters remained fixed throughout all simulations, while others were varied, as is outlined in each chapter.

Table 0.1: Names of All Parameters

Parameter Name	Parameter Description
G	Number of groups
T	Time (number of generations)
D	Number of days in each female's cycle
C	Number of days each female has non-zero probability of fertilization
C_v	Probability of fertilization on each of those C days
w_0	Baseline female fitness
N	Number of males and number of females in each group
b	Variation in male quality
c	Cost of having ovulation signs present
c_r	Cost of being receptive to mating
η	Weighting of GC vs. NGC
$\hat{g}$	Mean male genetic quality
ϵ_m	Parameter determining the amount of reproductive stochasticity among males
ϵ_f	Parameter determining the amount of reproductive stochasticity among females
γ	Parameter determining the shape of the function for the amount of visual ovulation signs present
α	Parameter controlling the maximum benefit of protecting against infanticide (note $\alpha = 0$ corresponds to the case with NO infanticide)
β	Parameter controlling the relative maximum cost of infanticide in the paternity probability effect function
τ	Exponent in the paternity probability effect function g
ω	Parameter in the male effectiveness function $f(j)$
κ	Parameter determining the weight males put on the amount of visual ovulation signs in their paternity estimate
ν	Migration rate (between zero and one)
μ	Probability of mutation (per gene per generation)

0.7. Female Sexual Signals, Receptivity, and Signal Reliability

Primate female estrous cycles are one such new possible application for agent-based modeling. There is clearly much variability among primates' estrous cycles, and this variability has been deemed of great interest since the time of Darwin (Darwin, 1876). The evolutionary path for such variation has been investigated by researchers for decades, from many different fields including evolutionary biology, anthropology, psychology, etc. There are many hypotheses as to (1) why visual ovulation signs are present in certain species (e.g. (Nunn, 1999)), (2) the relationship between visible ovulation signs and sexual receptivity (e.g. (Hrdy and Whitten, 1987)), (3) how and why concealed ovulation evolved independently so many times (as discussed earlier), and (4) how sexual signals evolved to be either reliable or unreliable (e.g. (Gouzoules and Gouzoules, 2002)). Despite (1) and (3) above being opposite phenomena, there is ironically still much overlap between these two groups of hypotheses (Burt, 1992).

As discussed previously, having ovulation signs fully concealed is quite counter-intuitive from the perspective of maximizing female fitness (Burley, 1979). A female who has more visible signs marking her time of fertility would gain more matings during this time, and in particular, more matings with higher-ranking males (since higher-ranking males show more interest in females who are clearly fertile) (Strassmann, 1981). Such males are presumably of higher genetic quality, and hence would produce offspring also of higher genetic quality. This means that any female with concealed ovulation, in a group of females all with signs very visible, would be at a large fitness *disadvantage* compared to all those other females (Schroder, 1993).

A female with a receptivity period *longer* than her fertile period is equally counterintuitive. Being receptive to mating, and hence mating, comes with many costs (expending energy resources, higher risk of contracting STD's, etc.). Certainly the fitness benefits of producing offspring outweigh those costs, but if a female is receptive but not fertile, she would only be incurring the costs from being receptive without any benefit of potential offspring. Hence for species to have females with longer receptivity than fertile periods implies there must be some other force affecting this trade-off (such as benefits coming from confusing paternity, for example) (van Schaik et al., 1999).

Similarly, females having reliable signals is intuitive in terms of the evolution of such signals. If

female sexual signals ever became too *unreliable*, males would simply stop paying attention to them. Conversely, in a group of females all with very reliable sexual signals, a female with an unreliable signal could also be at a fitness disadvantage since she would still be primarily mating around the time of her peak signal, whether or not that was also the time of her peak fertility (Gouzoules and Gouzoules, 2002).

0.8. Paternity Concentration vs. Paternity Confusion

The above discussion can really be condensed down into a single question: whether it is more beneficial for a female to concentrate paternity or to confuse paternity. Indeed, both paternity concentration and paternity confusion can be thought of as counterstrategies, or sexual strategies employed by females in order to help increase their fitness (van Schaik et al., 1999).

It is clearly in males' best interests to have paternity concentration. Males do not want competition when it comes to who might be the offspring of a female with whom that male mated. If a male is going to go out of his way to protect an infant (from predators, infanticide, etc.), provision it, or in any other way provide care, the male wants to be certain that the energy and resources being allocated to that child is being allocated towards his actual offspring, passing on his genes (Clutton-Brock, 2016).

Females, however, have a more complex situation. Paternity concentration may be helpful if, for instance, there is one male of a quality far superior to his counterparts (in genetic or non-genetic ways). If that is the case, it would benefit the female to mate exclusively with him, thus guaranteeing her offspring his genetic benefit and/or herself the benefit obtained by mating with him. Conversely, paternity confusion, or a female mating with more than one male during the course of one cycle, could be beneficial (van Schaik et al., 1999). For example, if infanticide is a threat, the female may be better off making multiple males think they may be the father of her offspring, thus affording her offspring protection from infanticide from more than one male. Alternatively, if the female receives provisionings or protection from harassment or predation from each male with whom she mates, she would also have incentive to mate with multiple males, in order to receive multiple provisionings and/or more protection (Clutton-Brock, 2016).

What is seen in nature, then, is a trade-off between paternity concentration and paternity confusion. In most situations, females will receive benefit from both confusing and concentrating paternity, and thus will attempt to confuse paternity (i.e. mate with multiple males) while simultaneously attempting to make a male think his paternity has been concentrated. For example, when just considering infanticide, females will try to concentrate paternity in order to help obtain protection from the likely father, while also trying to confuse paternity (mate with multiple males) in order to keep those males (even if non-likely fathers) from attempting infanticide against her offspring (van Schaik et al., 1999).

In particular, three different counterstrategies have been proposed as ways females could use their sexual behavior in order to manipulate both paternity concentration and paternity confusion among males. First, females could do a better job of attracting *all* males in their group, by having gradually changing and exaggerated visual sexual signals (for example, sexual swellings). For a female benefiting more from paternity concentration, she would want strong ovulation signs visible, but only for a short period of time. In doing so, the female is able to attract high-quality (for example, dominant) male mates, and such males would be able to guard her throughout her short period of attractivity. Conversely, for a female benefiting more from paternity confusion, the female would want weak ovulation signs visible, but for a long period of time. In doing so, the female would be able to attract multiple males with whom to mate over that longer period of time. Note the extreme of this scenario is concealed ovulation, or no visible signs of ovulation present at all.

The second female counterstrategy which has been proposed is altering female sexual receptivity length. Females benefiting from paternity concentration would have short lengths of receptivity. By only being receptive to mating for a short (i.e. less than 1-2 days) period of time, females are allowing males (typically those of high-quality/rank) to be able to guard them throughout their entire period of receptivity. If a male guards a female during her entire period of receptivity, he would know precisely what other males she mated with during that cycle (presumably none), meaning he can be confident in his paternity of any offspring produced. Females instead benefiting more from paternity confusion would have prolonged mating periods, allowing them more time to mate with multiple males throughout the course of one cycle, and make it harder for any one male

to guard her throughout her entire receptivity length.

The third female counterstrategy which has been proposed is altering female sexual signal reliability. Females benefiting more from paternity concentration would want their ovulation signal to honestly signal fertility. For a female investing largely in her ovulation signal (as she would when favoring paternity concentration), she would want to be fertile during this time of signaling, or else all that energy being invested would be for nothing since she would not be getting high-quality mates as the father of her offspring. Females instead benefiting more from paternity confusion would make ovulation more unpredictable, meaning have a more unreliable signal. Rather than having a female's attractivity (via her visual sexual signals) perfectly line up with her fertility, the female could have a blurred correlation between fertility and attractivity, thus allowing males to not be able to accurately estimate when ovulation is happening and hence when the female is at her peak fertility. Doing so would help confuse paternity, as not even herself would know when ovulation was about to happen (van Schaik et al., 1999).

0.9. Dissertation Outline

It is the aim of this dissertation to address the evolution of all three of the above counterstrategies. How could each have evolved? Under what conditions? How, if at all, are each related to the threat of infanticide? And how, if at all, are these counterstrategies related to each other, since more than one could be occurring simultaneously?

If having concealed ovulation signs and unreliable sexual signals *decreases* a female's probability of mating and/or *lowers* the quality of the mate she is able to obtain, how did concealed ovulation and unreliable signals evolve in the first place? Along with the questions of exaggerated female sexual swellings and continuous receptivity, these interesting questions are nothing new. However, with no fossil record or other concrete evidence to utilize for any of these questions, past relevant literature has largely been a verbal debate of ideas. It is my aim here to help answer these questions using mathematical modeling, thus contributing a new and important perspective to this already vast body of literature.

For the first three chapters of this dissertation, I use mathematical modeling techniques to help

answer each of the research questions above relating to the evolution of primate non-verbal sexual communication. In Chapter 1, I show how certain ecological factors, such as increased group size and/or the risk of infanticide, can increase visual ovulation signaling among female primates. In Chapter 2, I show female continuous receptivity and concealed ovulation to be correlated, and how only in groups with strong visible ovulation signs present would one expect to find a relatively short length of time when the female will be receptive to mating. In Chapter 3, I investigate the evolution of female sexual signal reliability, evaluating what conditions are important in the evolution of both reliable and unreliable female sexual signals.

Finally, in Chapter 4, I investigate and describe the case of long-term pair-bonding (i.e. when one breeding male and one breeding female share a common territory, associating with each other for more than just one breeding season regardless of whether or not they currently have any offspring together). While I do not model these ideas here, it is my hope that such work (which could easily be accomplished building off the modeling work presented in this dissertation) will be conducted in the future.

Together, the results presented in the first three chapters of this dissertation use mathematics to give new insight into primate evolution and to help resolve old, important mysteries surrounding primate non-verbal sexual communication. In addition, the final chapter of this dissertation describes in detail one final problem, still open in the fields of evolutionary psychology, anthropology, and biology, which could be solved in the future with similar mathematical models and methods as those described in Chapters 1-3.

Chapter I

On the evolution of visual female sexual signaling

Publication Statement: A version of this Chapter will be submitted soon for publication by its co-authors, Kelly Rooker and Sergey Gavrilets. The modeling and analytical work for this manuscript was primarily done by K. Rooker, and the manuscript also principally composed by K. Rooker.

On the evolution of visual female sexual signaling

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Abstract

A long-standing evolutionary puzzle surrounds female sexual signals visible around the time of ovulation. Even among just primates, why do some species have substantial sexual swellings and/or bright colorations visible around females' genital regions, while other species are like humans, with no signs of ovulation visible? What is the evolutionary purpose behind not just these signs, but also this great variation seen among species? Here we examine the evolutionary trade-offs associated with visual ovulation signaling using mathematical modeling. Our model predicts how various factors, including male genetic heterogeneity and reproductive inequality, female physiological costs, group size, and the weighting of genetic vs. non-genetic benefits coming from males, each influence the strength of ovulation signaling. Our model also predicts that increasing the impacts of infanticide will increase ovulation signaling. We use comparative primate data to show that, as predicted by our model, larger group size and higher risk of infanticide each correlates with having stronger visual ovulation signs. Overall, our work resolves some old controversies and sheds new light on the evolution of visual female sexual signaling.

1. Introduction

1.1. Background

Two general mechanisms for sexual selection are mate competition and mate choice (Darwin, 1871; Andersson, 1994). Traditionally, mate competition has been associated with *male-male* competition for access to females, while mate choice with *female* choice (for example, the bright colors, exaggerated plumage, and mating displays seen among males in many different species of birds to attract female mates) (Hosken and House, 2011; Andersson, 1994; Andersson and Iwasa, 1996; Kokko et al., 2003). However, there are now multiple examples instead of *female-female* competition (e.g. competition among females for rank) and *male* mate choice (e.g. males preferentially mating with females of a specific age, mating call, etc.); see Ref. (Clutton-Brock, 2009) for a review and Ref. (Servedio and Lande, 2006) for modeling work.

A striking example where female-female competition and male mate choice may be occurring is visual female sexual signaling in primates (Darwin, 1876; Zinner et al., 2004). Every primate clade contains species displaying visual signals around the female's time of ovulation (Nunn, 1999), and such signs are thought to have evolved independently at least five times among the catarrhine primates (i.e. Old World monkeys and apes) (Dixson, 2012). Although much varied between species, these signals primarily occur via changes in the size, swelling, shape, and/or color of the female's perineal skin (Dixson, 1983, 2012). See Fig. 1.1 for an example.

1.2. Costs and Benefits of Visual Ovulation Signs

Visual ovulation signals clearly have significant costs (Hrdy and Whitten, 1987). Physiologically, swellings resulting from increased water retention increase body weight, as well as divert fluids away from potentially more important bodily functions (especially in dry/drought conditions) (Krohn and Zuckerman, 1937). Higher body weight (making mobility more challenging), noticeable swellings, and/or genital colorations can increase a female's risk of predation. Sexual swellings expose the female's perineal skin to parasites, as well as to cuts and scratches (Nunn, 1999). Visual ovulation signs can also result in social costs, for example increased competition among males within groups (Etkin, 1954; Clutton-Brock and Harvey, 1976; Pfeiffer, 1978; Daniels, 1983).



Figure 1.1: Olive baboons in Gombe National Park, Tanzania (photograph by Dr. Michael Wilson). Adult female with sexual swelling (left) being groomed by an adult male (right).

Given all these costs, the evolution of visual female sexual signaling was long viewed as a puzzle, including its lack thereof in humans (Lovejoy, 1981). Various potential benefits of having visual ovulation signs have been proposed including signaling higher female genetic quality (Pagel, 1994), fertilization (since such signs simplify the process of obtaining a mate) (van Schaik et al., 2004), and protection by males of ovulating females (against injury, predation, harassment, etc.) (Nunn, 1999). Several other prominent hypotheses instead focus on female mating tactics. For example, having visual ovulation signs could help females attract better mates, giving better genes to offspring (Clutton-Brock and Harvey, 1976), or to attract multiple males to have more than one mating partner, which can be helpful for increasing any of paternity confusion, genetic diversity for offspring, gamete compatibility, post-copulatory sperm competition (“cryptic female choice”), etc. (Hrdy, 1981; Hrdy and Whitten, 1987; Dixson, 2002; Stallman and Froehlich, 2000). Having visual ovulation signals could also help females increase males’ perceived probabilities of paternity (Burt, 1992), increasing paternity certainty (Nunn, 1999) and/or paternal care (W.J. Hamilton, 1984; Alberts and Fitzpatrick, 2012).

The two prominent potential benefits initially emerging from this debate were sexual signals allowing for (1) paternity concentration (i.e. females attracting better mates, giving better genes to their offspring), and (2) paternity confusion (i.e. females attracting multiple mates, allowing

for more than one mating partner). The first became known as the “Best-Male Hypothesis”, and the second the “Many-Male Hypothesis”. The Best-Male Hypothesis says that visual female sexual signals foster male-male competition. Such competition, in turn, allows females the opportunity to be chosen by the “best” male (i.e. the male with the best genes to pass on to her offspring), and mate with him. In doing so, the female is signaling the best time for males to compete, so she can then mate with the “best” male during her peak time of fertility, thus concentrating paternity in a high-quality mate (Clutton-Brock and Harvey, 1976).

The Many-Male Hypothesis, on the other hand, says that females with visual sexual signals are able to attract multiple males with whom to mate. In doing so, a female is able to make more than one male think he may be the father of her offspring (i.e. confuse paternity), which can be beneficial, for example, in reducing the risk of infanticide for the female’s offspring (Hrdy, 1979; Hrdy and Whitten, 1987).

1.3. Infanticide

Infanticide, the killing of a female’s offspring prior to weaning, is extremely detrimental to a female’s fitness (van Schaik et al., 2004; Hrdy, 1979). However, committing an act of infanticide may benefit any male who is not the father of that particular offspring, and hence infanticide is often seen when a new male takes over a group (Pradhan and van Schaik, 2008). Many primate species have prolonged lactational amenorrhoea (van Schaik, 2000), resulting in a male killing a female’s offspring bringing that female back to fertility sooner by prematurely ending her lactation (Lovejoy, 2009; Palombit, 1999). The infanticidal male can then subsequently mate with this female, meaning his offspring will be able to be born sooner than would otherwise be possible had he never committed infanticide (Stallman and Froehlich, 2000). Although infanticidal events are typically rare, they have been documented in many primate species (Hrdy, 1979).

1.4. Reliable Indicator and Graded Signal Hypotheses

Despite much interest in both, neither paternity concentration nor paternity confusion obtained a consensus on being the more likely benefit. Two new hypotheses were then proposed, very different from each other but each attempting to reconcile the fact that *both* benefits (paternity

concentration, i.e. good genes, and paternity confusion, i.e. obtaining multiple mates) could be occurring simultaneously. The first such hypothesis, proposed in 1994, was the Reliable Indicator Hypothesis (Pagel, 1994), and the second, proposed in 1999, was the Graded Signal Hypothesis (Nunn, 1999).

The Reliable Indicator Hypothesis says that it is actually female-female competition which is the primary evolutionary pressure, with visual sexual signals serving as reliable indicators of each female's quality during that competition. Females are actively seeking good genes by competing for mates, and, were it beneficial, 'high-quality' females would also be able to attract more than one quality male (Pagel, 1994). Hence, this hypothesis mainly centers on paternity concentration while indirectly allowing for paternity confusion.

The Graded Signal Hypothesis reconciles these benefits in a very different way by directly accounting for both benefits, paternity concentration and paternity confusion. This hypothesis explicitly recognizes the fact that visual sexual signals serve as 'graded signals' for females, i.e. such signals are probabilistic, increasing and decreasing in strength gradually, with ovulation typically happening around the time of a female's peak signal. In doing so, according to this hypothesis, a female is able to attract male(s) with good genes around the peak of her signal (i.e. when she is likely most fertile), while still attracting other, lesser-quality male(s) during other parts of her cycle, when she still has some sexual signal, just not at its peak, and is likely less fertile (Nunn, 1999). In this way, the Graded Signal Hypothesis is able to directly account for both paternity concentration and paternity confusion as evolutionary benefits for females.

Substantial field and lab work (e.g. Ref. (Nunn et al., 2001; Domb and Pagel, 2001; Zinner et al., 2002; Deschner et al., 2004; Higham et al., 2012; Fitzpatrick et al., 2014, 2015; Douglas et al., 2016)), as well as phylogenetic studies (e.g. Ref. (Nunn, 1999; Sillen-Tullberg and Moller, 1993; Pagel and Meade, 2006)), have all been done in order to help understand visual ovulation signaling in an evolutionary context. As a result of all this work, the Graded Signal Hypothesis is now largely recognized as being the hypothesis to explain the evolution of visual female sexual signaling, while the Reliable Indicator Hypothesis was unable to find support empirically, and has since fallen out of favor (Nunn, 1999; Zinner et al., 2002; Alberts and Fitzpatrick, 2012; Fitzpatrick et al., 2015).

1.5. Research Questions

Many questions, however, still remain. For example, as related to infanticide, is it more likely for infanticide to serve as selection pressure for the evolution of concealed ovulation or of visual sexual signals? It has long been thought that infanticide could be a main selection pressure for *concealed* ovulation to evolve (e.g. see Ref. (Hrdy, 1979, 1981)). A female with concealed ovulation would have the vast majority of her cycle to confuse paternity (lowering her offspring's risk of infanticide) *and* would not have to suffer any of the physiological costs of having visual ovulation signs (Hrdy, 1979).

Completely opposite these beliefs is, as discussed earlier, the idea that females who signal prominently are better able to attract multiple males as mating partners, which (for example) is beneficial in reducing the risk of infanticide occurring to that female's offspring by confusing paternity (Hrdy and Whitten, 1987). Supporting this hypothesis are the facts that females with peak signals often mate with multiple males (Gouzoules and Gouzoules, 2002) and that such signals are not perfectly honest; ovulation rarely occurs exactly at the time of a female's peak signal (Nunn, 1999; Douglas et al., 2016).

Indeed, like the example of infanticide above, many of the proposed benefits of having visual ovulation signs have also instead been proposed as *costs* to females. As Austin Burt says, "Ironically, there is considerable overlap between the hypothesized benefits of sexual signals and those of concealed ovulation." (Burt, 1992, p.4).

Informative experiments relating to this phenomenon are by nature rather challenging to conduct, and many ideas historically have just been presented as verbal arguments which can often be easily challenged. Despite still-unanswered questions on the role and evolution of visual female sexual signaling, even just within the context of the Graded Signal Hypothesis, there has been relatively little theoretical work done in understanding the evolutionary trade-offs behind these signs persisting in so many different species (but see Ref. (Pagel, 1994; Huchard et al., 2009; Nakahashi, 2016)). In fact, two different reviews of visual ovulation signaling appealed specifically for "[More] theoretical approaches [to] be undertaken" (Nunn, 1999, p.241) and "More rigorous modelling, using mathematical tools and simulation approaches, [being] needed" (Zinner et al., 2004, p.83).

Hence, it is our aim here to use a mathematical model to help explore the evolutionary trade-offs associated with visual female sexual signaling. Allowing for both possible benefits to females (i.e. paternity concentration and paternity confusion), we are interested in determining under what conditions will ovulation signaling increase and/or decrease in a multi-male, multi-female population, assuming such signals to be graded.

2. Introduction to the Model

2.1. Model Set-Up

We consider a population of individuals living in a large number of multi-male, multi-female groups. We assume generations are discrete and non-overlapping. Females differ genetically in visual ovulation signs present, while males differ in their ‘quality’ (i.e. benefit to a female). Such quality is explicitly split into a genetic component (GC) and a non-genetic component (NGC). GC is the genetic benefit to a female’s offspring of her mating with a male with good genes, while NGC can be thought of as any non-genetic benefit to a female from mating with that male. For instance, NGC could be protection provided to the female herself (i.e. not her offspring), increased food/provisioning/access to resources, and/or paternal care (see Ref. (Clutton-Brock, 2016) for further description of such benefits). Note that GC and NGC are inversely related such that a male with higher GC will have lower NGC and vice versa.

We account for group size (N), variation in male quality (scaled by parameter b), the weighting of GC vs. NGC (η), costs of having ovulation signs visible (scaled by parameter c), and effects of infanticide. (See Methods for further details, explicit fitness functions, and full definitions of equations and parameters.) Our goal is to understand how visual female ovulation signs evolve over time.

We build an agent-based model such that:

$$\begin{aligned}
\text{female fitness} = & \text{baseline female fitness} \\
& + (1 - \eta) * (\text{benefit of male genetic quality}) \\
& + \eta * (\text{total average benefit of male non-genetic quality}) \\
& \pm \text{benefit/cost due to infanticide from each male} \\
& - \text{cost of having visible ovulation signs.}
\end{aligned}$$

2.2. The Female Cycle

Our model explicitly accounts for the female cycle, which is split into discrete units of time. While such units could be hours, minutes, etc., here we choose to call such units ‘days’ (for example, with 29 days in one female cycle). We treat visual ovulation signs, $x(d)$ for each day d of the cycle, as overlapping curves (Dixon, 2009). Each female is characterized by two genetically controlled traits: magnitude (m) and length (ℓ). m is the maximum amount of ovulation signs a female has visible during her cycle (a non-negative, continuous value), while ℓ is the number of days a female has *some* amount of ovulation signs visible (a non-negative, integer value). For an example of what these ovulation signs $x(d)$ could look like across a cycle for four females with varying values of m and ℓ , see Fig. 1.2.

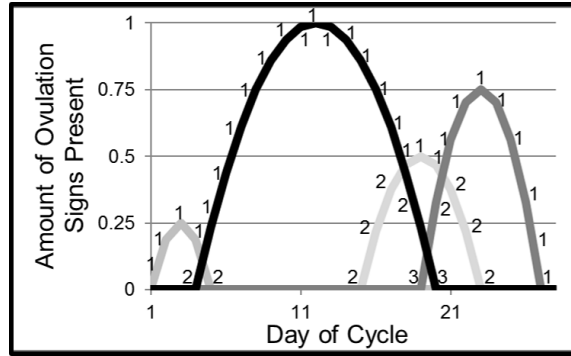


Figure 1.2: Example ovulation signs for females with different trait values over one 29-day cycle. Different colors represent four different female cycles. Numbers beside each curve represent the rank of the male’s GC who is the most likely to mate/guard the female on that particular day of her cycle (assuming no reproductive stochasticity).

For each female, the distribution of probabilities of fertilization across the cycle has the same

shape but is randomly centered (meaning cycle synchrony may only occur probabilistically). Visual ovulation signs are correlated with these days of fertility, but can still evolve freely. This means we assume there exists at least some reliability of the ovulation signal (since a female’s peak day of fertility will also be her day of peak ovulation signs), and that such reliability is the same in all females.

2.3. Male-Female Mating

For every unit of time in the cycle, all males and all females in each group enter the competition for mates. For computational simplicity, we assume each individual to mate exactly once on every unit of time (e.g. day). Males of higher GC are more likely to mate with females with stronger ovulation signs (higher $x(d)$), i.e. when the female is most likely to be most fertile. Males of lower GC mate with females who have fewer ovulation signs visible, i.e. when the female is less likely to be most fertile.

The degree of reproductive stochasticity in males and females is controlled by parameters $\epsilon_m \geq 0$ and $\epsilon_f \geq 0$, respectively. The higher ϵ_m is, the more likely low-GC males are to be able to mate with females with the strongest signal. The higher ϵ_f is, the more unreliable the female signal is in winning high-GC mates. With $\epsilon_m = \epsilon_f = 0$, there is no reproductive stochasticity and mating pairs are formed deterministically so that the highest-GC male mates with a female with the most ovulation signs visible, and so on). (See Methods for a full description of these parameters.)

Mating is followed by offspring production and dispersal. For any female’s potential offspring, every male in the group will have an associated probability of paternity, ranging anywhere from zero to one. This quantity is determined by both the number of times that particular male mates with the female during her cycle, as well as her probability of fertilization on the days of each mating event.

2.4. Infanticide

Modeling infanticide is adapted from Ref. (van Schaik et al., 2004). Any male is able to help or harm (note ‘harm’ here could simply mean not helping) an offspring prior to the infant being weaned, although we typically assume that some males (e.g. those larger and/or stronger) are

able to do so more effectively. Our model does not distinguish between the threat of infanticide via an outsider male or a previously-subordinate male. Infanticide's effects on offspring viability are scaled by parameters α (maximum benefit) and β (relative maximum cost). An additional parameter $0 \leq \kappa \leq 1$ determines the extent to which males take visible female ovulation signs into account when estimating their paternity. $\kappa = 0$ means males estimate their probability of paternity solely by the *number* of matings with an offspring's mother, while $\kappa = 1$ means males instead estimate paternity exclusively on the basis of the female's visible ovulation signs during their time of mating.

3. Methods

3.1. General Model

We consider a population of individuals interacting in G groups comprised of N males and N females. Males are ordered according to their genetic quality to females, denoted y_j such that $y_1 > y_2 > \dots$ for each male j . Intuitively, females will gain more fitness benefit from mating with males of higher GC. Such GC is randomly drawn from the normal distribution $\mathcal{N}(\hat{y}, b)$; parameter $\hat{y}$ characterizes mean male GC and $b > 0$ the extent of variation in male GC. Small b implies small potential benefits to females from mating with even high-GC males. [For simplicity, the distribution of y in the population remains constant, e.g. as a result of mutation-selection balance.] Male NGC is then equal to the reflection of the male's GC over the center of the normal distribution, the vertical line $y = \hat{y}$.

We explicitly incorporate the female estrous cycle using D discrete units of time (e.g. $D = 29$ days). For each of these D days, every female will have an associated probability of fertilization. We assume each fertile period to last $C \leq D$ days (e.g. $C = 7$). For all days lying outside those C fertile days, females are assumed to have zero probability of fertilization but can still mate. We assume a triangular shape for the fertility function to be identical in all females, but with midpoints randomly distributed so as the only cycle synchrony occurring within groups is probabilistic. For all females, we assume ovulation (and hence maximum visual ovulation signs) to happen directly in the middle of the C fertile days.

Considering one focal group, for any female i there are two evolving female traits: $m_i \geq 0$ (ovulation signs magnitude) and $\ell_i \in \mathbb{Z}, \ell_i \in [0, D]$ (ovulation signs length). Together, these determine the amount $x_i(d)$ of ovulation signs visible on any day d of the cycle:

$$x_i(d) = \begin{cases} m_i \cdot \left[1 - \left(\frac{2|d - \lceil \ell_i/2 \rceil|}{\ell_i + 1} \right)^\gamma \right] & \text{for } d = 1, 2, \dots, \ell_i \\ 0 & \text{otherwise} \end{cases} \quad (1)$$

for $\gamma > 0$ (note Fig. 1.2 depicts $x_i(d)$ with $\gamma = 2$) and d measured from the start of ovulation signs being visible. Note m_i represents the peak of $x_i(d)$ and ℓ_i the width of the non-zero portion of $x_i(d)$. Mutations in m_i are continuous, whereas mutations in ℓ_i are discrete, representing adding or subtracting exactly one day to or from a female's time of having ovulation signs visible.

On each day, mating pairs are formed by first randomly perturbing both the male trait y and female trait x by adding to each an independent, normally distributed random variable with standard deviation $\epsilon_m \geq 0$ and $\epsilon_f \geq 0$, respectively. Males and females are then sorted according to these perturbed values and mating occurs between individuals of the same order.

This stochasticity in male ordering reflects stochasticity in the outcome of their competition for mating success; a large ϵ_m means lower reproductive inequality. Stochasticity in female ordering reflects a lack of opportunity to mate with a higher quality male/female, disinterest in mating with a specific male (female) regardless of quality (signs), variation in reliability of the ovulation signal, etc. In addition, males will not always choose to mate with the female with the largest visual sexual signal (e.g. Ref. (Fitzpatrick et al., 2015)), which can be accounted for in our model with $\epsilon_f > 0$.

In the case of no infanticide, we have female fitness:

$$w_i = w_0 + (1 - \eta)y_{m,i} + \frac{\eta}{D} \sum_{\text{all mates } j} y_{p,j} - c \cdot \bar{x}_i. \quad (2)$$

Here, w_0 is baseline fitness, $y_{m,i}$ the benefit from female-female competition (i.e. the GC of the specific male who won the contest of paternity for that particular female's offspring), $y_{p,j}$ the NGC coming from each male j a female mated with throughout her cycle, and $c \cdot \bar{x}_i$ the costs to a female of supporting her visual ovulation signs with $\bar{x}_i = \frac{1}{D} \sum_{d=1}^D x_i(d)$, a female's average visual ovulation

signs. Our choice of the cost term implies there is a fitness trade-off between the m and ℓ traits so that having large values of both traits would imply very large fitness costs.

3.2. Modeling Infanticide

Each male in a female’s group can affect her fitness directly, due to infanticide, although males will differ in their actions’ effectiveness (e.g. due to strength, rank, size, alliances, etc.). We assume each male’s effectiveness to be proportional to $f(j)$ for each male j (sorted by male GC) via an exponential function: $f(j) \sim e^{-\omega j}$ with parameter $\omega > 0$ controlling the amount of disparity among males in their corresponding effectiveness within the group. Note a larger value of ω indicates more disparity. See SI for the case when $\omega = 0$, meaning we are assuming all males to be “equal” within the group, as it relates to their effectiveness.

A male’s contribution $g(p)$ to female fitness (positive via helping protect the offspring, or negative via not helping and/or harming the offspring) depends on his perceived probability of paternity p . Adapted from Ref. (van Schaik et al., 2004), we define $g(p) = \alpha[1 - \beta(1 - p)^\tau]$, with parameters $\alpha, \beta > 0$ and $\tau \geq 1$. Note α determines the maximum benefit a female can obtain from a male protecting her offspring from infanticide, while $\alpha(1 - \beta)$ determines the maximum cost a female can incur from a male *not* protecting her offspring from infanticide. The overall effect of infanticide on female fitness (that gets added to the right-hand side of Eq. 2) is $\sum_j f(j)g(p_j)$, summing over all males j . Note whenever $\alpha = 0$, there are no effects of infanticide.

A male in general would not know his actual paternity probability and would instead have to estimate it. We define this male-estimated (“perceived”) probability by male j of being the father of an offspring born to female i by $p_{i,j} = \sum_{\# \text{ of matings}} (x_i(d))^\kappa$, where $\kappa \in [0, 1]$ represents the weight males put on visual ovulation signs when estimating paternity. Note $\kappa = 0$ represents males estimating their paternity solely on the number of matings with that mother, while $\kappa = 1$ represents males putting full weight instead on her visual ovulation signs. For each male j , these perceived paternity probabilities get normalized such that $\sum_{i=1}^N p_{i,j} = 1$.

3.3. Simulations

This model was implemented independently in both C++ and Matlab with similar results. Migration occurs with only females migrating between groups, although simulations were also run

with only males migrating between groups and the results were unaffected. Mutation occurs with a mutation rate per gene per generation of 10^{-3} . Mutations for m are selected from $\mathbb{N}(0, 0.1)$, while mutations for ℓ from the set $\{-1, 1\}$ with equal probability. $2N$ offspring are created in each group every generation, with N female and N male, determined randomly. The expected number of offspring per female is 2; the actual number is random, proportional to each female's fitness.

The model uses 16 different initial conditions in all simulations: every combination of $m = 0, 0.3, 0.6, 0.9$ and $\ell = 1, 2, 3, 4$. Parameters used include: $G = 400$; $T = 100,000$; $D = 29$; $C = 7$ (with probabilities of fertilization on each of these days being 0.125, 0.25, 0.375, 0.5, 0.375, 0.25, 0.125, respectively); $w_0 = 1$; $N = 4, 8, 16$; $b = 0.1, 0.2, 0.4$; $c = 0.1, 0.2, 0.4$; $\eta = 0.1, 0.2, 0.4$; $\gamma = 0.5, 1, 2$; $\hat{y} = 1$; $\epsilon_m = 0, 0.25, 0.5$; $\epsilon_f = 0, 0.01, 0.05$; $\alpha = 0, 0.2, 0.6, 1$; $\beta = 1.25, 1.5, 1.75$; $\tau = 1, 2, 4$; $\omega = 0, 1, 2$; and $\kappa = 0, 0.5, 1$. Further information on parameter values tested and each of their effects is available in the SI.

4. Results

Numerical simulations of our model show that the average values of the ovulation signs magnitude trait (m) and the ovulation signs length trait (ℓ) converge to the unique equilibria m^*, ℓ^* . Fig. 1.3 illustrates the evolution of m (x -axis) and ℓ (y -axis) over time, both with (bottom row) and without (top row) the effects of infanticide included. In particular, we see that introducing infanticide results in increased visual ovulation signs' magnitude. Due to the costs trade-off present, note that with longer length ℓ^* we typically see smaller magnitude m^* . A more comprehensive discussion of the influences of all parameters can be found in the Supplementary Information (SI).

4.1. Without Infanticide

Fig. 1.4(a) summarizes the effects without infanticide of the benefit b , cost c , and group size N on the equilibria values magnitude (m^*) and length (ℓ^*). As expected, increasing benefits b and decreasing costs c each results in increased m^*, ℓ^* . Our simulations also show that increasing group size N results in increased m^* . This is because group size increasing results in female-female competition for "good genes" increasing, which incentivizes having more visual ovulation signs present.

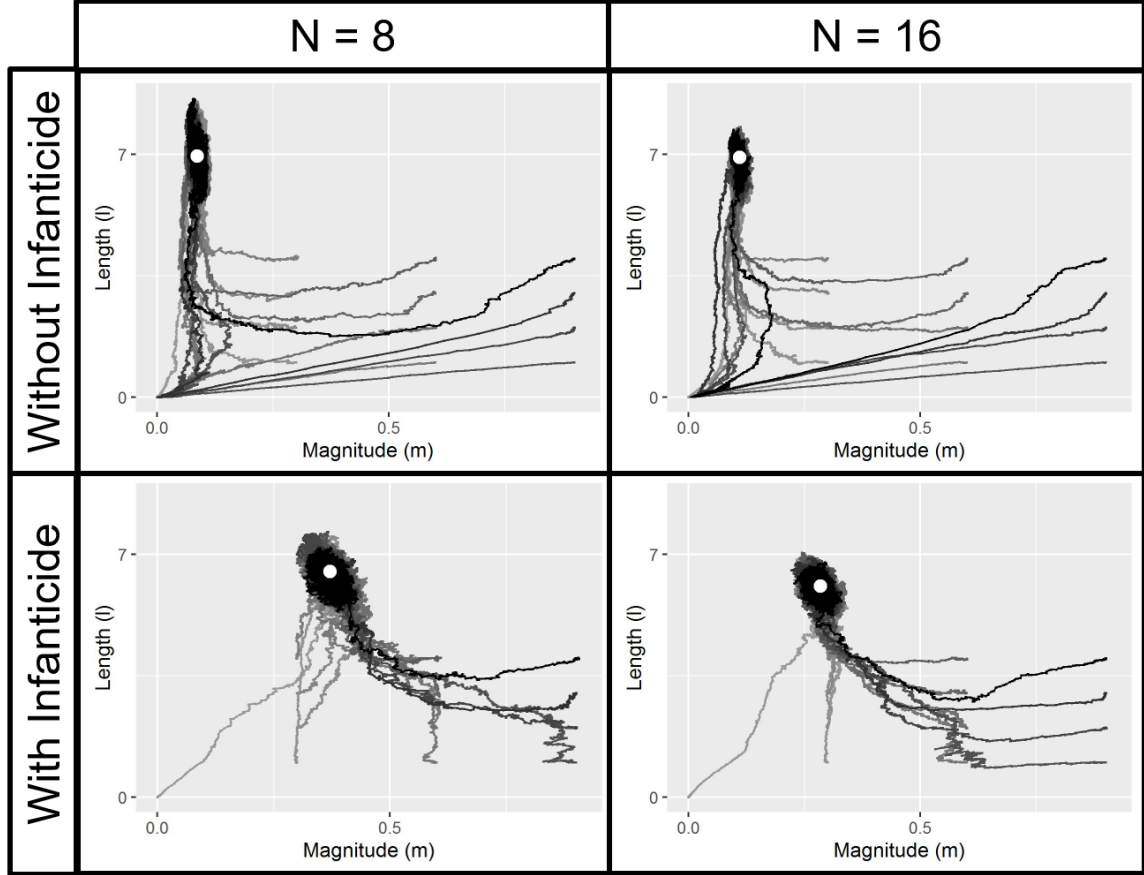


Figure 1.3: Example evolutionary dynamics on the phase plane (m, ℓ) , both with (bottom row) and without (top row) infanticide using two different group sizes (columns). Each graph depicts simulations of the model for 16 different initial conditions (illustrated by the different-colored lines). Notice introducing infanticide increases magnitude m^* , and we see convergence in all cases, as illustrated by the white points depicting the averages over all runs at the end of the simulations. ($\alpha = 0$ (top) and $\alpha = 0.6$ (bottom) with all other parameters held constant: $b = 0.2, c = 0.2, \epsilon_m = 0.25, \epsilon_f = 0.01, \omega = 1, \tau = 2, \beta = 1.5, \kappa = 1, \gamma = 1, \eta = 0$.)

4.2. With Infanticide

Fig. 1.4(b) instead summarizes the effects of parameters relating to infanticide (α, β, κ) on the equilibria values magnitude (m^*) and length (ℓ^*). Increasing α , β , and κ each results in increased values of m^* . Intuitively, as these effects relating to infanticide increase, it becomes in females' best interests to mate with high-GC males (i.e. those more likely to have their actions be effective within their group and hence be better able to help protect an infant). Having strong visual ovulation signs facilitates matings with such high-GC males. The effects on ℓ are somewhat smaller; note in most simulations $\ell^* \leq 7$, since we set the time of fertility to seven days.

4.3. Effects of Reproductive Stochasticity

Fig. 1.4(c) summarizes the effects without infanticide of η and the two reproductive stochasticity parameters, ϵ_m and ϵ_f . Increasing η results in increasing the weighting of NGC compared to GC. Hence, *decreasing* η increases the weight of GC, i.e. the genetic benefit to a female's offspring from mating with a particular male. Intuitively then, decreasing η results in increased m^* , because decreasing η increases the value of the 'prize' to a female for winning the female-female competition for good genes.

Increasing male reproductive stochasticity (meaning decreasing the amount of reproductive inequality among males) decreases the female ovulation traits m^*, ℓ^* . This is because with more male reproductive stochasticity, strongly-signaling females are less likely to get mates with high-GC males, simply by chance, leading to females investing less energy in such signaling. With low male reproductive stochasticity, increasing female reproductive stochasticity increases the female's magnitude trait m^* . This trend reverses as male reproductive stochasticity increases. This is intuitive since with more male reproductive stochasticity, females with the strongest ovulation signals will not necessarily get to mate with the highest-GC males. Hence with low reproductive stochasticity among males, females will invest more in stronger signals to help account for their own reproductive stochasticity. On the other hand, with high reproductive stochasticity among males, ovulation signals for females (both m^*, ℓ^*) are smaller since even females who win the female-female competition will not necessarily be getting the benefit of better genes for their offspring. This effectively takes away the benefit to females of having ovulation signs, and thus such signs remain only a cost.

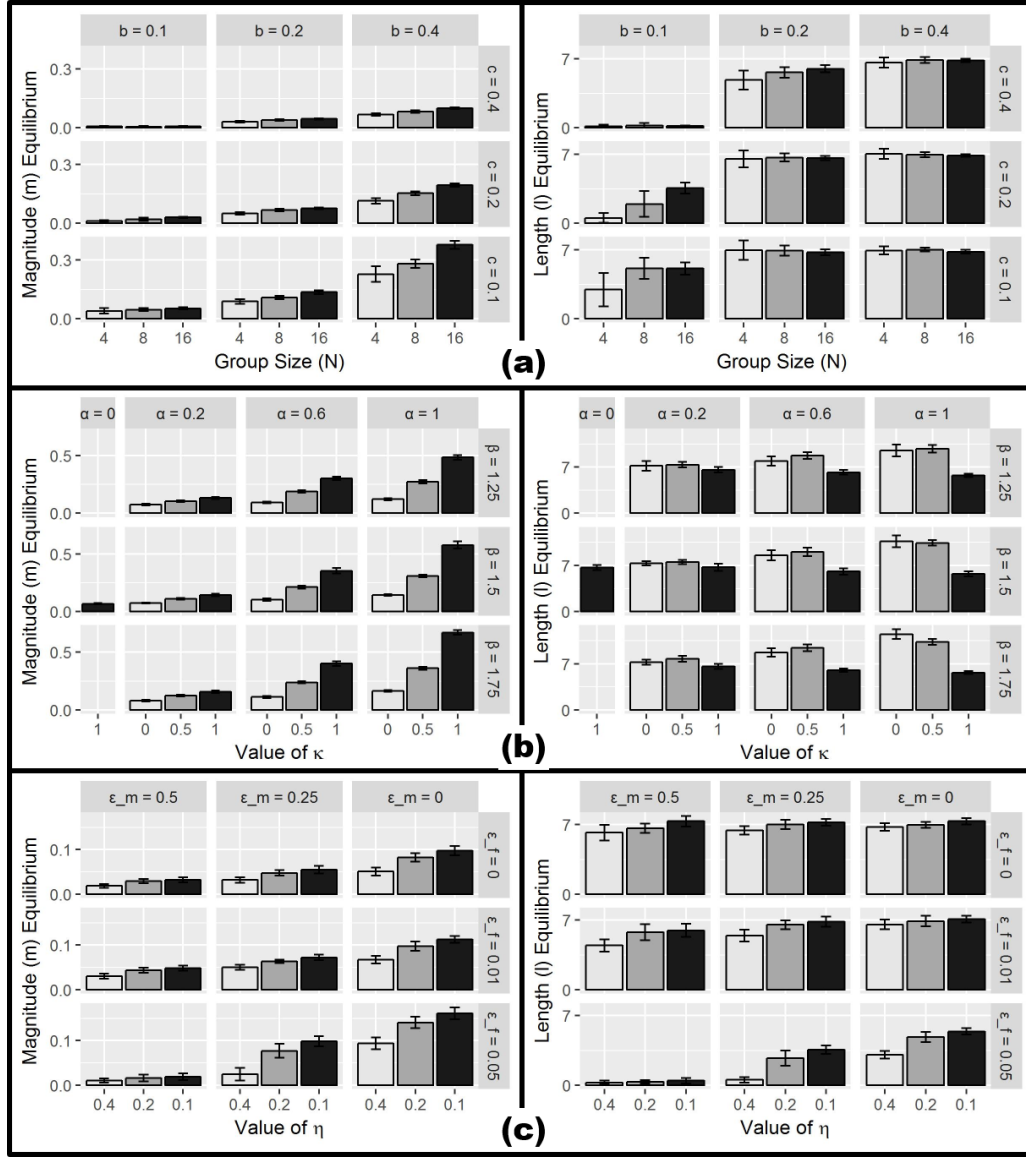


Figure 1.4: Effects of parameters on the average equilibria values of ovulation signs magnitude (m^*) and ovulation signs length (l^*). (a) The effects of variation in male quality (b), costs of having visual ovulation signs (c), and group size (N) on m^* (left graph) and l^* (right graph). (b) Effects relating to infanticide (α, β, κ) on m^* (left graph) and l^* (right graph). $\alpha = 0$ represents the case with no infanticide present (and hence when κ, β are irrelevant). (c) The effects of reproductive stochasticity in males (ϵ_m) and females (ϵ_f) and the weighting of NGC vs. GC (η) on m^* (left graph) and l^* (right graph). Equilibria are obtained by averaging 16 initial condition runs (with standard deviation indicated by error bars). Notice m^* becomes stronger with increasing N , increasing b , decreasing c , increasing α, β, κ , decreasing ϵ_m , and decreasing η . Other parameters: (a) $\epsilon_m = 0.25, \epsilon_f = 0.01, \alpha = 0, \gamma = 1, \eta = 0.2$; (b) $N = 8, b = 0.2, c = 0.2, \epsilon_m = 0.25, \epsilon_f = 0.01, \gamma = 1, \tau = 2, \omega = 1, \eta = 0.2$; (c) $N = 8, b = 0.2, c = 0.2, \alpha = 0, \gamma = 1$.

5. Discussion

5.1. Overview

Here we have developed a realistic framework for modeling the evolution of visual female sexual signaling, allowing for both possible benefits paternity concentration and paternity confusion. We have shown that increased visual ovulation signaling could have evolved for a variety of reasons. For example, we predict increased visual ovulation signals with increased male reproductive inequality (i.e. decreased ϵ_m), increased group size (N), increased variation in male quality (b), and decreased costs of having ovulation signs visible (c).

The effects of b and c are intuitive. The effects of N are due to female-female competition; as group size increases, female-female competition for the best males becomes more intense, thus increasing the ovulation signal. The effects of η are also intuitive: increasing the benefit coming to a female from mating with males with good genes will result in stronger-signaling females. The effects of reproductive stochasticity (ϵ_m, ϵ_f) are a little more varied. In general though, the more stochasticity present, the smaller the benefit to females of having signs, meaning the smaller their ovulation signal.

We also predict increased visual ovulation signals with increased effects of infanticide (α, β, κ). Such signs help females win copulations with high-GC males, who are also more likely to be more effective within their groups (due to strength, rank, etc.). Increased matings give these males increased likelihoods of being the father of any subsequent offspring that female produces (paternity concentration), which increases the likelihood of that male attempting to protect the offspring against any possible infanticide being committed. In our model, we did not impose female cycle synchrony. Assuming so would increase female-female competition and hence visual ovulation signs.

5.2. Comparison to Empirical Data

We were able to test two of these predictions empirically, using data from extant primate species living in multi-male, multi-female groups. All data is available in the Supplementary Information. First, our model predicted that species with larger group sizes would have more visual ovulation signs present. To test this prediction, we used ovulation signs data (Hrdy and Whitten, 1987; Sillen-Tullberg and Moller, 1993; van Schaik et al., 1999; Pagel and Meade, 2006) and added average group

size to each species for which data was available (Jones et al., 2009; Kamilar and Cooper, 2013; Atmoko and van Schaik, 2010; Binford, 2001). Doing so gave 65 species with both a classification of having “Present” or “Absent” visual ovulation signs and the species’ average group size.

Note we restricted this analysis to species with a polygynandrous mating system since our model explicitly considers individuals living in multi-male, multi-female groups and mating system is already a known correlate of visual ovulation signs in primates (Sillen-Tullberg and Moller, 1993; Pagel and Meade, 2006). Also note that we follow (Pagel and Meade, 2006) in grouping “Absent” and “Slight” ovulation signs visible (for example, from the ternary classification as found in Ref. (Sillen-Tullberg and Moller, 1993)) together into the one category of “Absent”. This is due to both the fact that the category “Slight” is vague (e.g. does a species whose females have no sexual swellings, but just a slight reddening of the perineum only visible in immediate proximity count as “Absent” or “Slight”?) and also that it has yet to be established that males use “Slight” swellings as any kind of signal (Pagel and Meade, 2006).

The left graph of Fig. 1.5 shows these breakdowns: species with “Present” and “Absent” visual ovulation signs have different distributions of group size. As predicted by our model, species with smaller group sizes are much more likely to have “Absent” visual ovulation signs, while species with larger group sizes are more likely to have “Present” visual ovulation signs. To test for significance, we controlled for phylogeny using a simulation-based phylogenetic ANOVA test (T. Garland et al., 1993; Revell, 2012) with a recent primate phylogeny (Fabre et al., 2009), and found these differences to be significant ($F = 12.868, p = 0.002$).

Second, we predicted that species with a higher risk of infanticide would have more visual ovulation signs present. To test this prediction, we merged Opie et.al.’s data on lactation and gestation length (Opie et al., 2013) with the same ovulation signs data as above by species name. Note we still restricted our data set to only including those species with a polygynandrous mating system. Doing so gave 48 species with both a classification of “Present” or “Absent” visual ovulation signs, and a ratio of $L/G := \frac{Lactation\ Length}{Gestation\ Length}$. Note this ratio is a known correlate of infanticide risk, with species with higher L/G having higher risks of infanticide (van Schaik, 2000).

To test our prediction, we fit a Bayesian model of continuous trait evolution (Beaulieu et al.,

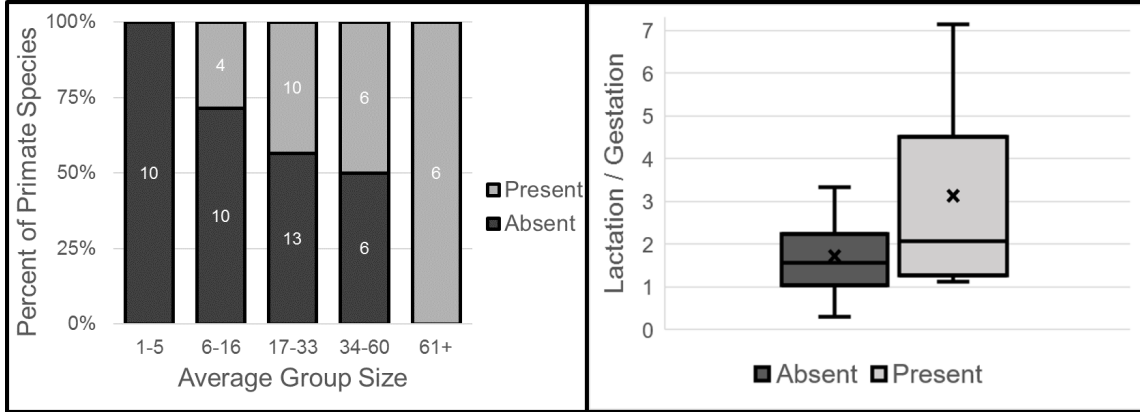


Figure 1.5: Left Graph: Comparing primate species' average group size by females having "Present" or "Absent" visual ovulation signs. After controlling for phylogeny, species with higher group sizes are more likely to have visual ovulation signs, and species with concealed ovulation smaller group sizes (using phyloANOVA: $F = 12.868, p = 0.002$). Numbers inside the bars indicate the numbers of species contained in each category. Right Graph: Box plots depicting the distributions of L/G for species with each "Absent" (33 species) and "Present" (15 species) ovulation signs. Including phylogeny, the model with "Absent" and "Present" having distinct means has a 62% relative probability. Each box plot depicts the minimum, maximum, median, and quartile values, while the black x's mark the means.

2012), again using the primate phylogeny from Ref. (Fabre et al., 2009). The right graph of Fig. 1.5 depicts the box plots of L/G for species with "Absent" and "Present" ovulation signs visible. We see that, as predicted, the mean L/G value for species with "Absent" ovulation signs is less than the mean for species with "Present" ovulation signs. In addition, we see that nearly 75% of species with "Absent" ovulation signs have $L/G < 2$, with about a third of those satisfying $L/G < 1$, meaning in these species average gestation is actually *longer* than average lactation. On the other hand, over 50% of species with "Present" ovulation signs satisfy $L/G \geq 2$, with over 25% satisfying $L/G \geq 4$ and zero species with $L/G < 1$. After considering phylogeny using Ref. (Beaulieu et al., 2012), there is still support for this correlation (62% relative probability from model selection; see Supplementary Information for more details). Again as predicted, in species with less risk of infanticide, we are more likely to expect concealed ovulation to evolve, while in species with a higher risk of infanticide, we are more likely to see visible ovulation signs evolve.

5.3. Conclusion

Hence both our modeling and empirical work support greater infanticide risk leading to increased ovulation signs rather than concealed ovulation. This theoretical work is also helpful in clearing up

past confusion. For example, as Charles Nunn says regarding paternity confusion (i.e. the Many-Male Hypothesis), “[It] is not able to explain the conspicuousness of [the visual ovulation] signal. This hypothesis also cannot explain why features of exaggerated swellings bias certainty towards certain males in the social group.” (Nunn, 1999, p.238).

Our modeling results show that the “conspicuousness of this signal” need not be a result of the selection pressures due to infanticide, but rather to one or more of the potential ecological costs identified here. Moreover, our models show that the risk of infanticide can indeed explain why exaggerated swellings would bias certainty towards “certain males” (i.e. more powerful/effective ones). More powerful/effective males are both more likely to take over a group from a current alpha-male (and hence the risk of infanticide from them be imminent), and more likely to be stronger/better able to protect a female’s infant from any possible infanticide attempt by a new alpha-male.

Our work complements the sparse earlier modeling work on visual ovulation signaling. Pagel (1994) mathematically showed that male-male competition for females will always occur (Pagel, 1994). Huchard et.al. (2009) included an individual-based model, attempting to reconcile the Reliable Indicator and Graded Signal Hypotheses (Huchard et al., 2009). Nakahashi (2016) discusses infanticide while not explicitly modeling its effects (Nakahashi, 2016). In addition, our model considers the female cycle as a factor, assuming males can estimate the fertility of females with whom they mate, rather than know it exactly (as assumed in Ref. (Nakahashi, 2016)).

Although our work considers primates as a whole, in particular there has been much interest on the question of why do *human* females have concealed ovulation? Is this related to some larger “adaptive suite” in human evolution (Lovejoy, 2009)? While our results do not support the infanticide hypothesis for the evolution of concealed ovulation in humans (Hrdy, 1981), they *do* provide new ecological predictions as to why concealed ovulation may have evolved in our ancestors.

In particular, such ecological causes for the disappearance of visual ovulation signals include: (i) group size decreasing (potentially as a result of changes in ecological resource distributions, predators, etc.), (ii) within-group variation in male quality decreasing or males becoming more egalitarian (Boehm, 2001; Gavrilets, 2012b), (iii) non-genetic components of male quality becoming highly ben-

eficial compared to genetic components, (iv) physiological costs of having ovulation signs increasing (due to increased water retention, body weight, infection/damage, predation, etc.; potentially as a result of the adaptation of bipedalism), and/or (v) the effects of infanticide decreasing.

Note with regards to (i) above, visual ovulation signs are expected to disappear with the transition to monogamy since the genetic benefits of visual ovulation signaling (i.e. attracting good and/or more mates) would disappear while the costs remain present (Lovejoy, 2009; Gavrillets, 2012a). This is in line with both previous phylogenetic studies on primate mating system and visual ovulation signs (Sillen-Tullberg and Moller, 1993; Pagel and Meade, 2006), and also our model should we set $N = 1$.

Our approach also comes with several limitations. Like many models, we assume discrete generations for mathematical simplicity. Explicitly accounting for the female cycle has made obtaining informative analytical results not feasible, leading to us relying on agent-based simulations. We also do not consider the effects of any extended mate-guarding (i.e. mate-guarding lasting longer than our one discrete unit of time). We do not explicitly allow for variation in signal reliability, instead assuming a female’s peak day of fertility and day of peak visible ovulation signs to line up. We also only consider multi-male, multi-female mating systems, excluding other mating systems from direct consideration in our model. In addition, our description of ‘male quality’ is purposefully general; we do not explicitly take into account male age, rank, or any other similar known factor in primate mating.

While much work still needs to go into understanding the evolution of visual female sexual signaling, our model is able to resolve some old controversies and shed new light on this important issue. In particular, our work suggests several ecological pathways by which visual ovulation signals could have evolved or diminished, while also showing that infanticide can provide important selection pressure on the evolution of visual ovulation signs. Aside from the two predictions explored in Fig. 1.5, applying more of our theoretical insights to other specific systems/species would require further detailed information on fitness components and ecological factors, which we hope to have inspired for future empirical work.

6. Acknowledgments

We thank Jeremy Auerbach for comments and suggestions, and Jeremy Beaulieu for help with the phylogenetic analysis. K.R. was supported by a National Science Foundation Graduate Research Fellowship. S.G. was supported by the National Institute for Mathematical and Biological Synthesis through National Science Foundation awards EF-0832858 and DBI-1300426, with additional support from The University of Tennessee. All numerical simulations were run on the Volos cluster, which is supported by National Institutes of Health award GM56693 (S.G.).

7. Supplementary Information: Analytical Results

While the exact model used for the numerical work is too complicated from which to get useful analytical results, interesting analytical results can still be obtained after making some simplifications.

7.1. The Two-Male Infanticide Model

7.1.1. Analytical Model Set-Up

We let $N = 2$ in order to consider the case when there are only two males present and/or of importance in each group, namely the males with the highest and second-highest quality. For simplicity, we will refer to these as the dominant male and subordinate male, respectively. We also here are only considering the case where the only benefits/costs to females come from the threat of infanticide (i.e. $\eta = 0, c = 0$), and there is no reproductive stochasticity among either males nor females ($\epsilon_m = \epsilon_f = 0$).

We also consider perceived paternity probability ($p_{i,j}$ for male j of female i 's offspring) instead as a function. Letting x_i be the ovulation signs trait for female i , we write perceived paternity probability as the function $p(j, x_i)$ for male j of female i 's offspring.

Note in this two-male simplification, we need only consider $f(1), f(2)$ (from the probability of takeover function $f(j)$ and $p(1, x_i), p(2, x_i)$ here. For this probability of takeover function, any function $f(j) \in [0, 1]$ satisfying $f(1) + f(2) = 1$ will work. For the perceived paternity probability function, we show that the exact equations for $p(j, x_i)$ will not matter for the analyses of this model below, only their respective slopes.

To consider these slopes, note we expect $p(1, x_i)$ to be an *increasing* function of x_i since in the case of fewer ovulation signs, a dominant male would find it increasingly challenging to keep watch over any given female throughout her entire fertile period. In contrast, in the case of increasingly visible ovulation signs, a dominant male would not only know that he mated with a female during her fertile period, he would also be much more likely to successfully guard her during her entire fertile window. Similarly, we expect $p(2, x_i)$ to be a *decreasing* function of x_i , since (analogous to the above) the fewer visible ovulation signs a female has, the less likely she is to be desirable by the dominant male and hence the more opportunities there would be for a subordinate male to mate with (and/or guard) said female throughout her cycle. Conversely, if the female had strong ovulation signs visible, the subordinate male would likely not be able to keep the dominant male from mating with her.

Since there are only two males to consider in this model and we are not considering any benefit/cost to females other than from infanticide, the fitness function is simply:

$$w_i = w_0 + f(1)g(p(1, x_i)) + f(2)g(p(2, x_i)). \quad (3)$$

7.1.2. Invasion Female Fitness

In the most general scenario, the invasion fitness for a mutant female with trait value $\tilde{x}$ (in a population of females all with trait value x) is:

$$w(\tilde{x}|x) = \sum_{r=1}^N [f(r)g(p(r, \tilde{x}))] - \sum_{r=1}^N [f(r)g(p(r, x))]. \quad (4)$$

7.1.3. Invasion Fitness Gradient

We can then find the invasion fitness gradient in this general case:

$$\left. \frac{dw}{d\tilde{x}} \right|_{\tilde{x}=x} = \sum_{r=1}^N \left[f(r) \frac{dg}{dx}(p(r, x)) \right]. \quad (5)$$

Keeping $f(r)$ and $p(r, x)$ in their general forms, we plug in the derivative of the given function for g . The above invasion fitness gradient thus becomes:

$$\left. \frac{dw}{d\tilde{x}} \right|_{\tilde{x}=x} = \alpha\beta\tau \sum_{r=1}^N \left[f(r)(1 - p(r, x))^{\tau-1} \frac{dp}{dx}(r, x) \right]. \quad (6)$$

The invasion fitness for the mutant female (with ovulatory trait $\tilde{x}$) in the two-male simplification becomes much simpler:

$$w(\tilde{x}|x) = f(1)g(p(1, \tilde{x})) + f(2)g(p(2, \tilde{x})) - f(1)g(p(1, x)) - f(2)g(p(2, v)), \quad (7)$$

as well as the invasion fitness gradient:

$$\left. \frac{dw}{d\tilde{x}} \right|_{\tilde{x}=x} = \alpha\beta\tau \left[f(1) (1 - p(1, x))^{\tau-1} \frac{d}{dx} (p(1, x)) + f(2) (1 - p(2, x))^{\tau-1} \frac{d}{dx} (p(2, x)) \right]. \quad (8)$$

We then use the substitutions $p(2, x) = 1 - p(1, x)$ and $\frac{d}{dx} (p(2, x)) = -\frac{d}{dx} (p(1, x))$, to instead rewrite the above as:

$$\left. \frac{dw}{d\tilde{x}} \right|_{\tilde{x}=x} = \alpha\beta\tau \left(\frac{d}{dx} (p(1, x)) \right) [f(1)(1 - p(1, x))^{\tau-1} - f(2)(p(1, x))^{\tau-1}]. \quad (9)$$

Notice each of the terms in the above is non-negative, meaning in order for $\left. \frac{dw}{d\tilde{x}} \right|_{\tilde{x}=x} < 0$ to be satisfied (and x predicted to decrease in value), we would need:

$$f(1)(1 - p(1, x))^{\tau-1} < f(2)(p(1, x))^{\tau-1}. \quad (10)$$

7.1.4. Considering the Case $\tau = 1$

Hence for $\tau = 1$, we would need:

$$f(1) < f(2) \quad (11)$$

to be satisfied in order for x to decrease. Thus we would need the probability of takeover to be greater than the probability that the dominant male will remain dominant. Note this makes sense since the *less* likely usurpation is to occur (i.e. the smaller the value of $f(2)$), the more desirable it would be for a female to have *more* matings with the dominant male. Since the $p(1, x)$ curve largely reflects a dominant male's preference for mates, the more ovulation signs any given female has, the more attention/matings she will get from that dominant male. Hence, in the case $\tau = 1$, the condition $u(1) < u(2)$ makes sense since as soon as usurpation is *more* likely to occur than not, we expect ovulation signs (x) to decrease (to allow the female increased matings with the subordinate male), while when usurpation is instead *less* likely to occur, we expect ovulation signs (x) to increase (to instead allow the female increased matings with the dominant male).

7.1.5. Considering the Case $\tau = 2$

Then for $\tau \neq 1$, we obtain the condition for x to decrease:

$$p(1, x) > \frac{f(1)^{\frac{1}{\tau-1}}}{f(2)^{\frac{1}{\tau-1}} + f(1)^{\frac{1}{\tau-1}}}. \quad (12)$$

To take a closer look at this general $\tau \neq 1$ case, we consider $\tau = 2$. Then the above condition becomes:

$$p(1, x) > \frac{f(1)}{f(2) + f(1)} \quad (13)$$

$$= f(1), \text{ or:} \quad (14)$$

$$p(2, x) < f(2) \quad (15)$$

needs to be satisfied in order for x to decrease.

In this case, the condition $p(2, x) < f(2)$ also makes intuitive sense. The closer $f(2)$ is to 1 the more likely this condition is to be satisfied, meaning the higher the probability of usurpation, the more likely it is for ovulation signs (x) to decrease. Again, the higher the chance of usurpation, the higher the benefit to the female of mating with the subordinate male (since if he took over, that female's infant would then gain his associated protection), and mating with the subordinate male is more likely to occur when ovulation signs are less visible (i.e. x closer to zero).

Hence for $\tau = 2$:

- If $f(2) < \inf\{p(2, x) : x \in [0, 1]\}$, x will increase;
- If $f(2) > \sup\{p(2, x) : x \in [0, 1]\}$, x will decrease; and
- If $\inf\{p(2, x) : x \in [0, 1]\} < f(2) < \sup\{p(2, x) : x \in [0, 1]\}$, we expect x to evolve towards the equilibrium value given by $p^{-1}(2, f(2))$ (see Figure 1.6).

In the event of an equilibrium occurring (as demonstrated in Figure 1.6), it is intuitive that this equilibrium would occur precisely where subordinate paternity probability equals subordinate takeover probability. If $p(2, x) > f(2)$, it is more likely for the subordinate male to be the father of the offspring than for him to take over as the dominant male, meaning the female would have been better off (i.e. have a smaller chance of infanticide being committed against her offspring)

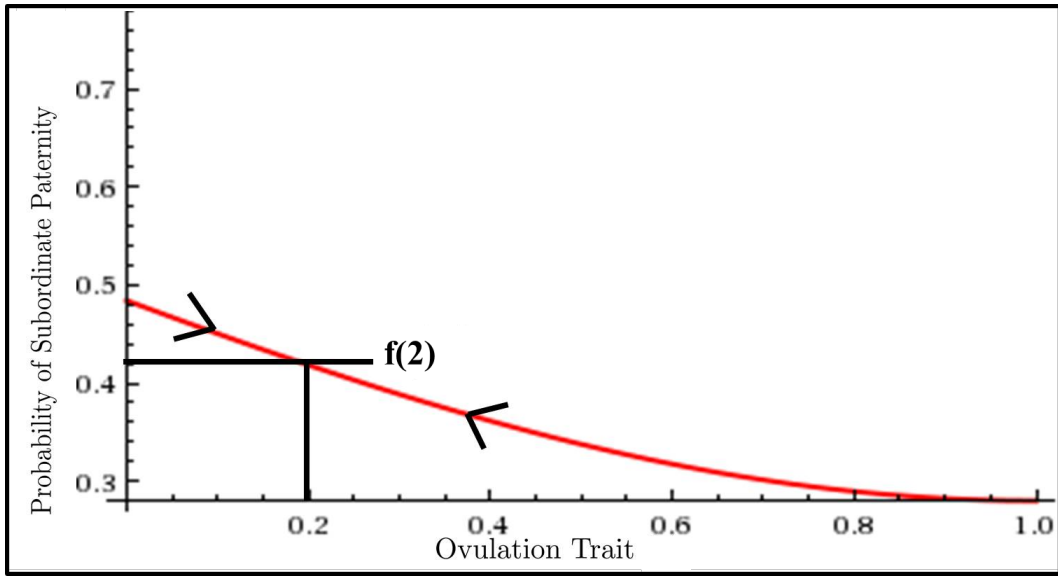


Figure 1.6: Depiction of the evolution of x to the equilibrium value $p^{-1}(2, f(2))$ when $\inf\{p(2, x) : x \in [0, 1]\} < f(2) < \sup\{p(2, x) : x \in [0, 1]\}$ is satisfied.

with the dominant male having a higher paternity probability. Hence we would expect x to increase since larger x gives higher perceived paternity probability to the dominant male (since $p(1, x)$ is an increasing function of x).

Similarly, when $p(2, x) < f(2)$, it is now more likely for the subordinate male to take over as the dominant male than be the father of an offspring, meaning it is in the female's best interest (i.e. minimizes her chances of infanticide being committed against her offspring) for the subordinate male to have increased paternity probability. Then in this case, we would expect x to decrease since smaller x values give higher perceived paternity probability to the subordinate male (since $p(2, x)$ is a decreasing function of x). Thus we obtain the logical equilibrium value of $p(2, x) = f(2)$.

8. Supplementary Information: Numerical Results

8.1. Further Details of the Model

8.1.1. The Female Cycle

Female estrous cycles are divided into D discrete units of time (e.g. $D = 29$ days) with $C \leq D$ days of fertility (e.g. $C = 7$). Although the form of such fertile periods will be identical in all females, the timing of each fertile period will be randomly distributed among females. For instance, when $C = 7$ (calling each of these fertile days $C1, C2, \dots, C7$) and $D = 29$ (calling each of these days $D1, D2, \dots, D29$), $C1$ in every female has an equal probability of landing on any of $D1, D2, \dots, D29$. Since C is a cycle, we similarly assume $C7$ can land on any such day. For example, if $C1$ landed on $D24$, then $C7$ would 'wrap around' the cycle to land on $D1$. Moreover, for all females we assume ovulation happens directly in the middle of this C cycle. Hence, for $C = 7$ we assume ovulation happens on $C4$ and the probabilities of fertilization increase from $C1$ to $C4$ and then decrease from $C4$ to $C7$.

Since having visible ovulation signs at least loosely correlates with a female's fertility, we assume the day(s) of having maximum visual ovulation signs align with the day(s) of that female's maximum fertilization probability, and align these cycles accordingly (note in many cases, $C \neq \ell_i$). Recall $x_i(d)$ denotes the amount of visual ovulation signs present on each day d of the female cycle, with $x_i(d)$ defined by the function in the Methods section of the main text. This function is determined by m_i (ovulation magnitude), ℓ_i (ovulation length), and parameter γ (controlling the shape of the

resulting curve). Note the exact function $x_i(d)$ is chosen such that (i) m_i will denote the peak of the curve, (ii) ℓ_i will denote the width of the non-zero portion of the curve, (iii) m_i, ℓ_i remain independent of each other, and (iv) m_i, ℓ_i can both decrease all the way to zero. Example values of $x_i(d)$ are displayed in Fig. 1.7 for varying values of m_i, ℓ_i , and γ , with $C = 7$ (as indicated by the red shaded bars).

8.1.2. Determining Male-Female Mate Pairs

We assume that, for every day of the cycle, males of higher GC will preferentially mate with females with more ovulation signs visible. We also assume each individual to mate precisely once on each day of the cycle. Parameters ϵ_m and ϵ_f control the amount of stochasticity in the process of mating pair formation. With $\epsilon_m, \epsilon_f \rightarrow \infty$, matings are determined completely randomly.

8.1.3. Calculating Probabilities of Paternity

To calculate the probability of paternity for each male, first the fertility of each female with which that male mated is summed up for every day a particular male mates with her. These values are normalized across males for each female in the group, meaning a male who mates with the female on a day where she has a higher fertility probability will have a higher paternity probability. The actual father for that female's offspring is then determined randomly, proportional to each male's probability of paternity for her offspring.

Note these *actual* probabilities of paternity are different from the *perceived* probabilities of paternity, as detailed in the Methods section of the main text. We make this distinction because males will not know any female's probability of fertility at the time he mates with her; males will only know how many visible ovulation signs she has present. Separating these quantities allows actual probabilities of paternity (which males do not know) to be calculated using females' probabilities of fertility, and perceived probabilities of paternity (which males *do* know) to be calculated using females' visible ovulation signs.

8.1.4. Infanticide

We define the exponential function of male effectiveness to be:

$$f(j) = \frac{e^{-\omega j}}{\sum_{k=1}^N e^{-\omega k}}, \quad (16)$$

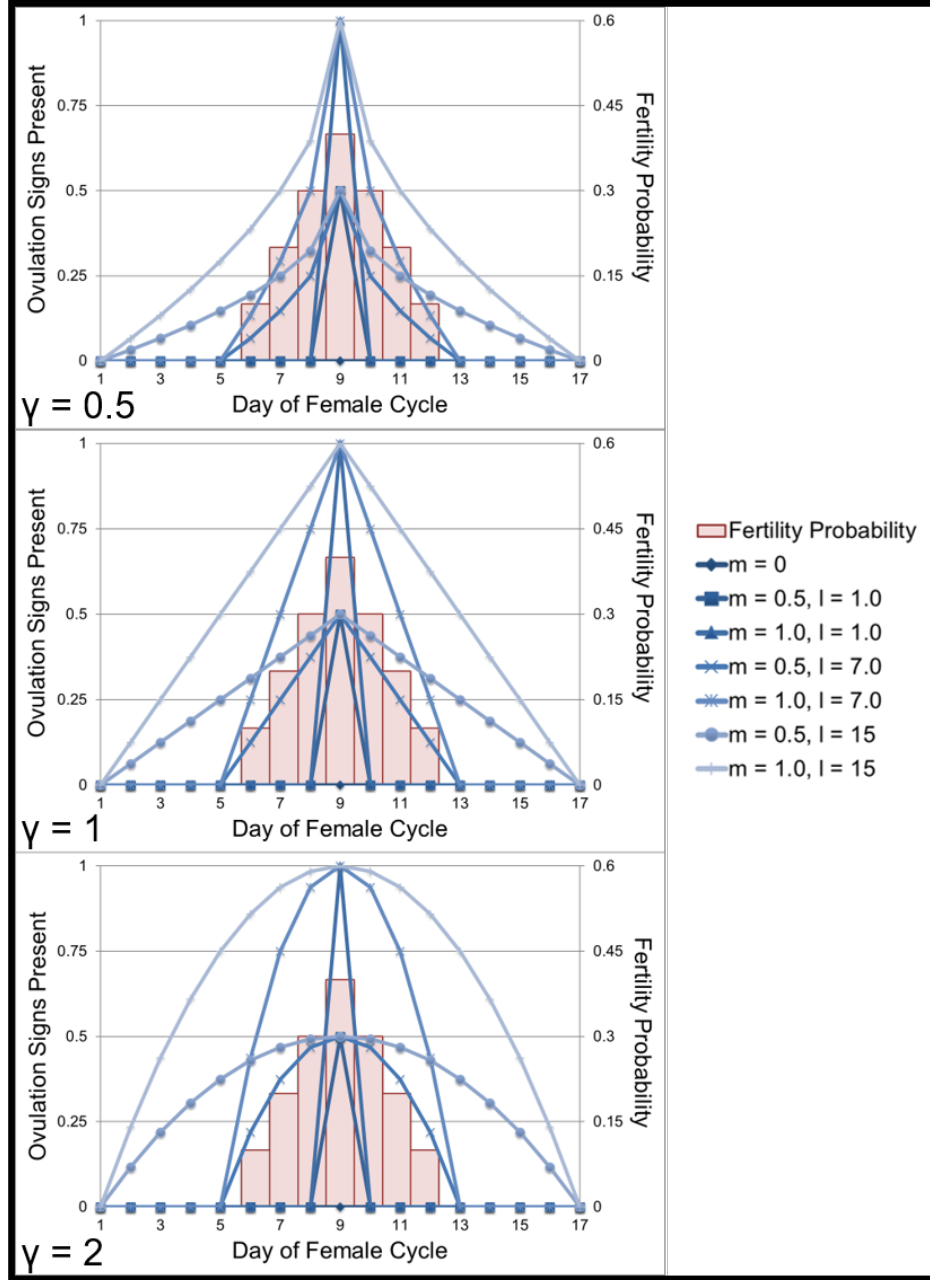


Figure 1.7: For each day d of a female cycle, sample values for visual ovulation signs present, $x_i(d)$, are displayed for $\gamma = 0.5, 1, 2$ (from top to bottom). Each graph includes all combinations of $\ell_i = 1, 7, 15$ and $m_i = 0, 0.5, 1$. Note $C = 7$ in this example, with fertility probabilities depicted by the red shaded bars. $x_i(d) = 0$ on a red shaded day means the female has no signs present on day d , despite still having some positive probability of fertilization. Conversely, positive $x_i(d)$ values *outside* the shaded days indicate visual ovulation signs being present despite a zero probability of fertilization.

with parameter $\omega > 0$ controlling the amount of disparity among males in their corresponding effectiveness within a group. Recall a larger value of ω indicates more disparity among males.

8.1.5. Female Fitness Functions

The female fitness function for female i in the model is as follows:

$$w_i = w_0 + (1 - \eta)y_{m,i} + \frac{\eta}{D} \sum_{\text{all mates } j} y_{p,j} + \sum_{j=1}^N [f(j)g(p_{i,j})] - c\bar{x}_i, \quad (17)$$

where recall η is the weighting on female fitness between GC and NGC, $y_{m,i}$ is the actual benefit from female-female competition (i.e. the GC of the specific male who won the contest of paternity for that particular female's offspring), $y_{p,j}$ the NGC coming from each male j a female mated with throughout her cycle, $p_{i,j}$ perceived paternity probability, and $\bar{x}_i = \frac{1}{D} \sum_{d=1}^D x_i(d)$ the female's average visual ovulation signs present.

Notice for the case when $\alpha = 0$ (i.e. without the effects of infanticide), the fitness function above collapses into:

$$w_i = w_0 + (1 - \eta)y_{m,i} + \frac{\eta}{D} \sum_{\text{all mates } j} y_{p,j} - c\bar{x}_i. \quad (18)$$

Note in this $\alpha = 0$ (no infanticide) case, parameters β , κ , τ , and ω all become unnecessary.

8.2. List of All Parameters

Parameters used in the model are outlined in Table 1.1. For each parameter, one value listed implies the parameter remained fixed throughout all simulations, and multiple values indicates each of those parameter values was tested in all combinations with all other parameters.

Table 1.1: List of All Parameters

Parameter Name	Parameter Description
$G = 400$	Number of groups
$T = 100000$	Time (number of generations)
$D = 29$	Number of days in each female's cycle
$C = 7$	Number of days each female has non-zero probability of fertilization
$C_v = \{0.125, 0.25, 0.375, 0.5, 0.375, 0.25, 0.125\}$	Probability of fertilization on each of those $C = 7$ days
$w_0 = 1$	Baseline female fitness
$N = 4, 8, 16$	Number of males and number of females in each group
$b = 0.1, 0.2, 0.4$	Variation in male quality
$c = 0.1, 0.2, 0.4$	Cost of having ovulation signs present
$\eta = 0.1, 0.2, 0.4$	Weighting of GC vs. NGC on female fitness
$\hat{y} = 1$	Mean male GC
$\epsilon_m = 0, 0.25, 0.5$	Parameter determining the amount of reproductive stochasticity among males
$\epsilon_f = 0, 0.01, 0.05$	Parameter determining the amount of reproductive stochasticity among females
$\gamma = 0.5, 1, 2$	Parameter determining the shape of the function for the amount of visual ovulation signs present
$\alpha = 0, 0.2, 0.6, 1$	Parameter controlling the maximum benefit of protecting against infanticide (note $\alpha = 0$ corresponds to the case with NO infanticide)
$\beta = 1.25, 1.5, 1.75$	Parameter controlling the relative maximum cost of infanticide in the paternity probability effect function
$\tau = 1, 2, 4$	Exponent in the paternity probability effect function g
$\omega = 0, 1, 2$	Parameter in the male effectiveness function $f(j)$
$\kappa = 0, 0.5, 1$	Parameter determining the weight males put on the amount of visual ovulation signs in their paternity estimate
$\nu = 1$	Migration rate (between zero and one)
$\mu = 0.001$	Probability of mutation (per gene per generation)

8.3. Effects of All Parameters

Table 1.2: Effects of All Parameters

Found in Figure:	Increasing Parameter:	Results Primarily In:	Rationale:
1.4(a), 1.8	N	<i>increasing</i> the ovulation traits	Increased competition for high-GC mates
	b	<i>increasing</i> the ovulation traits	Increased variation in male quality
	c	<i>decreasing</i> the ovulation traits	Increased costs of having signs
1.4(b), 1.9, 1.11	α	<i>increasing</i> the ovulation traits	Increased effects of infanticide
	β	<i>increasing</i> the ovulation traits	Increased effects of infanticide
1.4(b), 1.9	κ	<i>increasing</i> the ovulation signs magnitude trait and <i>decreasing</i> the ovulation signs length trait	Increased competition for high-GC mates increases m , while the higher m goes, the lower ℓ goes, due to fitness cost trade-offs
1.4(c), 1.8, 1.9, 1.10	η	<i>decreasing</i> the ovulation traits	Increased weighting on GC vs. NGC
1.4(c), 1.10	ϵ_m	<i>decreasing</i> the ovulation traits	Decreased likelihood of obtaining a high-quality male even when winning female-female competition
	ϵ_f	<i>increasing</i> the ovulation signs magnitude trait and <i>decreasing</i> the ovulation signs length trait	Effects dependent on ϵ_m , but in general the more reproductive stochasticity among females, the more females concentrate their signal stronger and on fewer days
1.10	γ	<i>decreasing</i> the ovulation traits	Increased visual ovulation signs for any given values of m and ℓ
1.11	τ	<i>increasing</i> the ovulation traits	Increased effects of infanticide
	ω	<i>increasing</i> the ovulation signs magnitude trait and <i>decreasing</i> the ovulation signs length trait	Increased competition for high-GC mates increases m , while the higher m goes, the lower ℓ goes, due to fitness cost trade-offs

See Table 1.2 for a summary of effects for all main parameters used in the model. These effects are all illustrated in Figures 1.4,1.8,1.9,1.10,1.11.

Note the intuition behind the effects of N , b , c , α , β , κ , η , ϵ_m , and ϵ_f are discussed in the main text of this chapter. Increasing γ results in decreasing the ovulation signs traits. This is expected since γ changes the shape of the ovulation signs curve (see Fig. 1.7). With a larger γ , for any fixed m_i , ℓ_i , and d , $x_i(d)$ will be larger. Thus with a larger γ , to get the same $x_i(d)$ for all d , one would now need *smaller* m_i , ℓ_i .

Increasing τ increases the benefit a female obtains from protection against infanticide, and hence (as discussed in the main text of the chapter) also the ovulation signs traits.

Finally, increasing ω results in increasing ovulation magnitude (m) and decreasing ovulation length (ℓ) because increasing ω results in a larger disparity among males in terms of their effectiveness within a group. As this disparity, and hence competition for high-GC males, increases, it becomes in females' best interests to allocate more energy/resources towards ovulation signs magnitude vs. ovulation signs length. This difference is because having more ovulation signs present than any other female in the group, even if just present on one day, will make it more likely for that female to secure a copulation with a high-quality male.

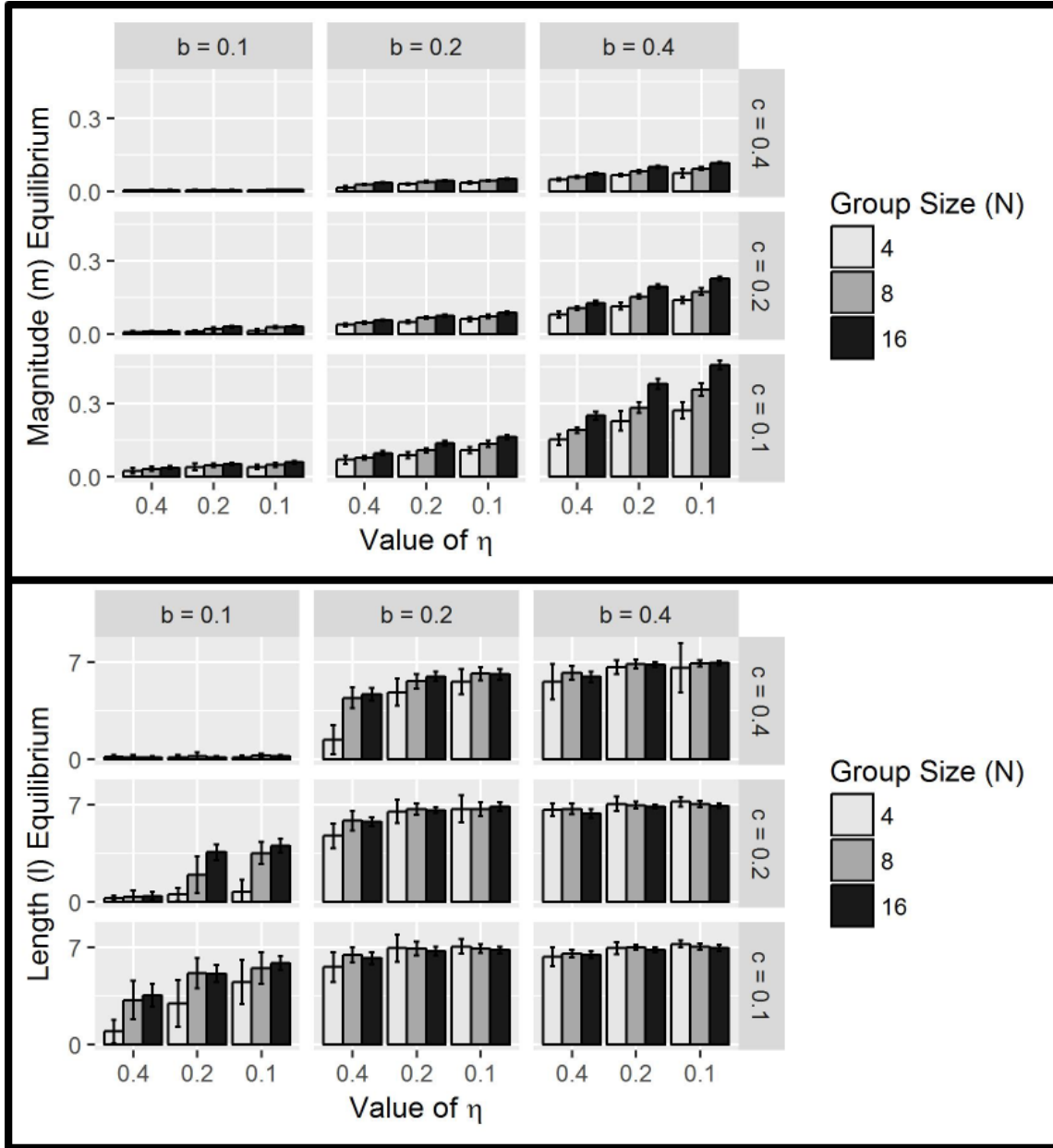


Figure 1.8: The effects of parameters N (group size), b (variation in male quality), c (costs of having ovulation signs), and η (weighting of GC vs. NGC) on the average equilibria values of magnitude m (top graph) and length ℓ (bottom graph). Equilibria are obtained by averaging over 16 initial condition runs (with standard deviation indicated by error bars). Notice ovulation signs become stronger with increasing N , increasing b , decreasing c , and decreasing η . All other parameters were held constant: $\epsilon_m = 0.25$, $\epsilon_f = 0.01$, $\gamma = 1$, $\alpha = 0$.

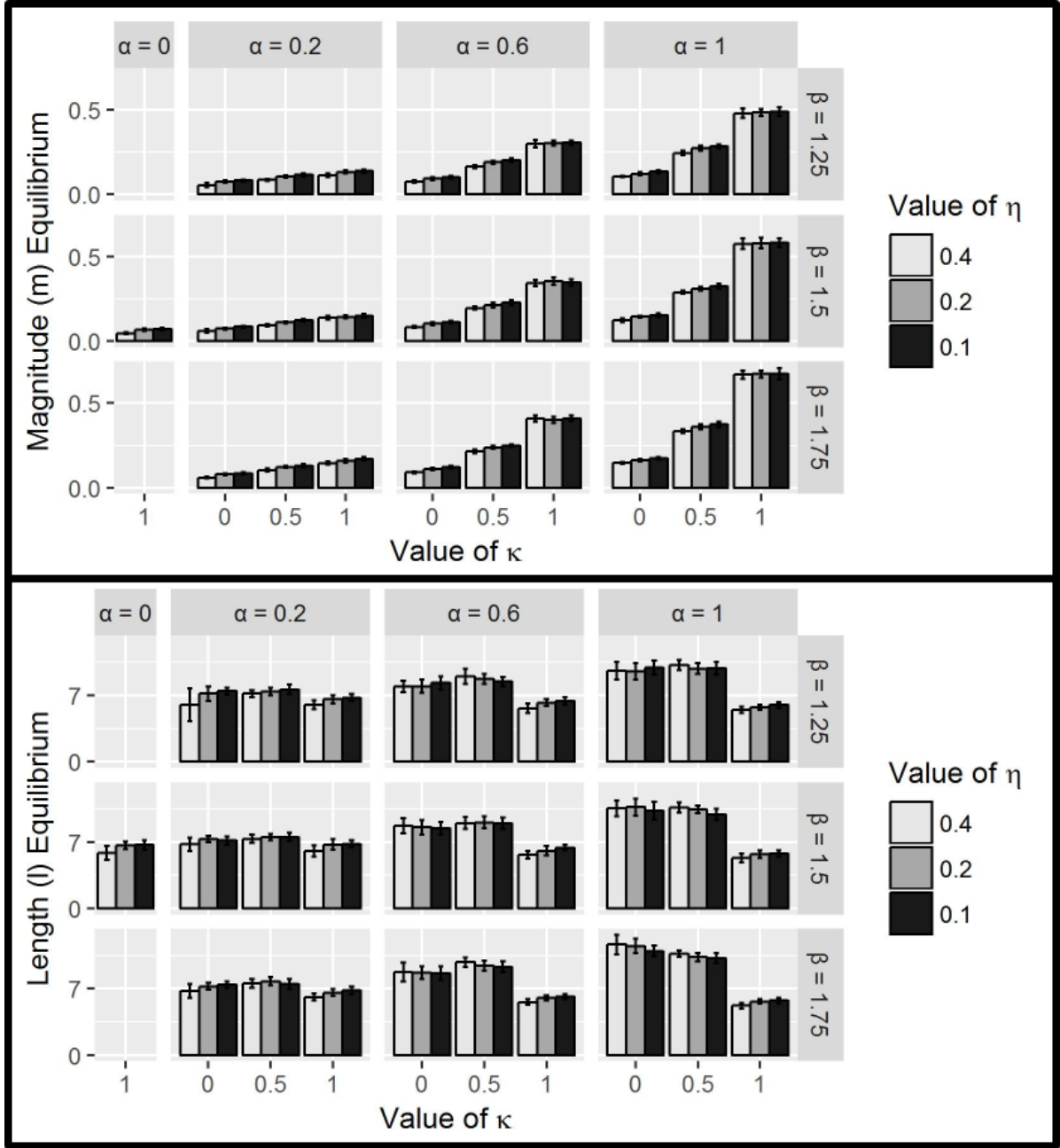


Figure 1.9: The effects of parameters α (maximum benefit of infanticide), β (proportional to the maximum cost of infanticide), κ (weight males put on females having ovulation signs visible), and η (weighting of GC vs. NGC) on the average equilibria values of both magnitude m (top graph) and length ℓ (bottom graph). Equilibria are obtained by averaging over 16 initial condition runs (with standard deviation indicated by error bars). Notice ovulation signs become stronger with increasing α , increasing β , and increasing κ , and increasing η . All other parameters were held constant: $N = 8, b = 0.2, c = 0.2, \epsilon_m = 0.25, \epsilon_f = 0.01, \gamma = 1, \kappa = 1$.

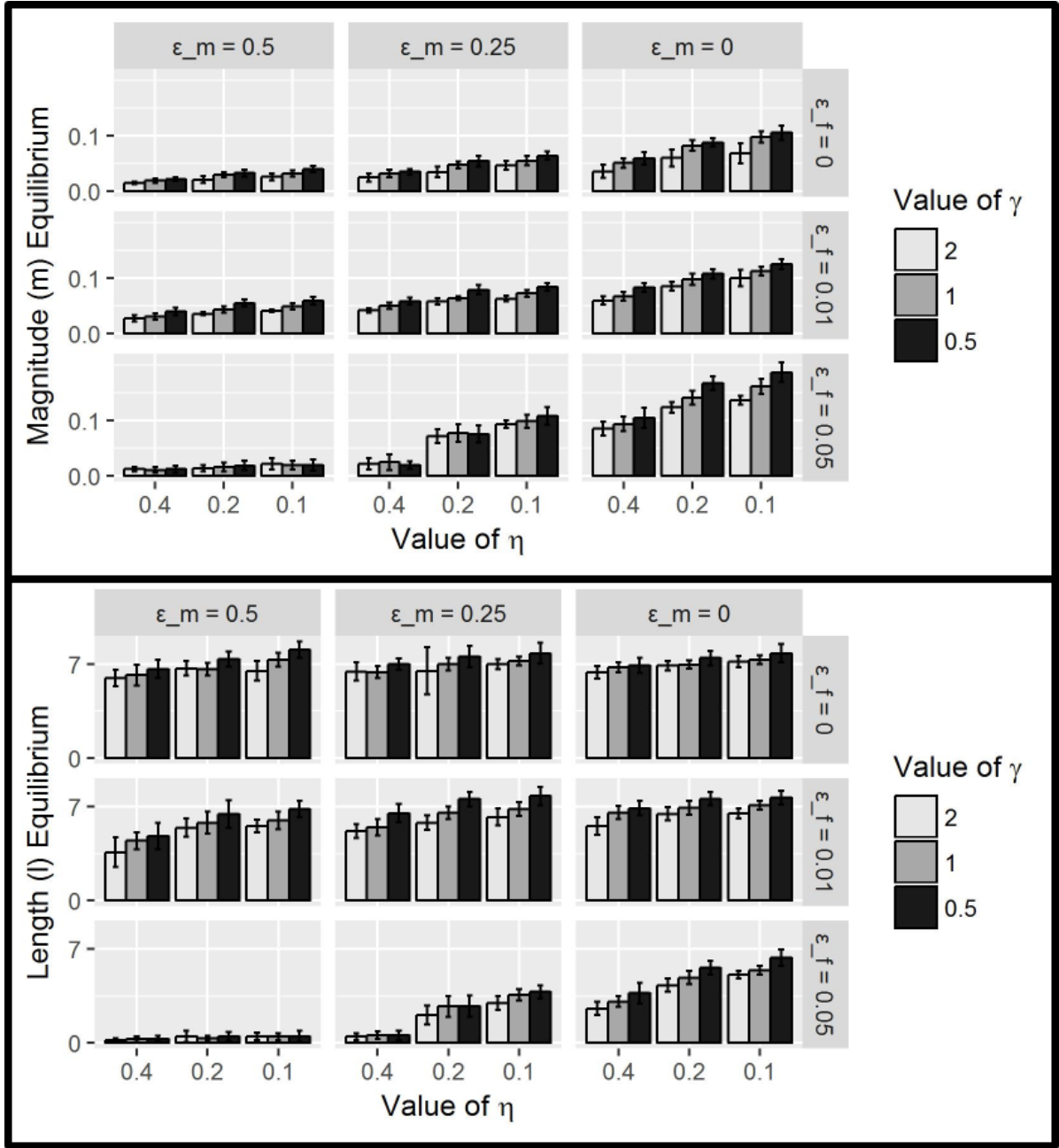


Figure 1.10: The effects of parameters ϵ_m (reproductive stochasticity among males), ϵ_f (reproductive stochasticity among females), γ (determines shape of x_i curve), and η (weighting of GC vs. NGC) on the average equilibria values of both magnitude m (top graph) and length ℓ (bottom graph). Equilibria are obtained by averaging over 16 initial condition runs (with standard deviation indicated by error bars). Notice ovulation signs become stronger with decreasing ϵ_m , γ , and η . All other parameters were held constant: $N = 8$, $b = 0.2$, $c = 0.2$, $\alpha = 0$.

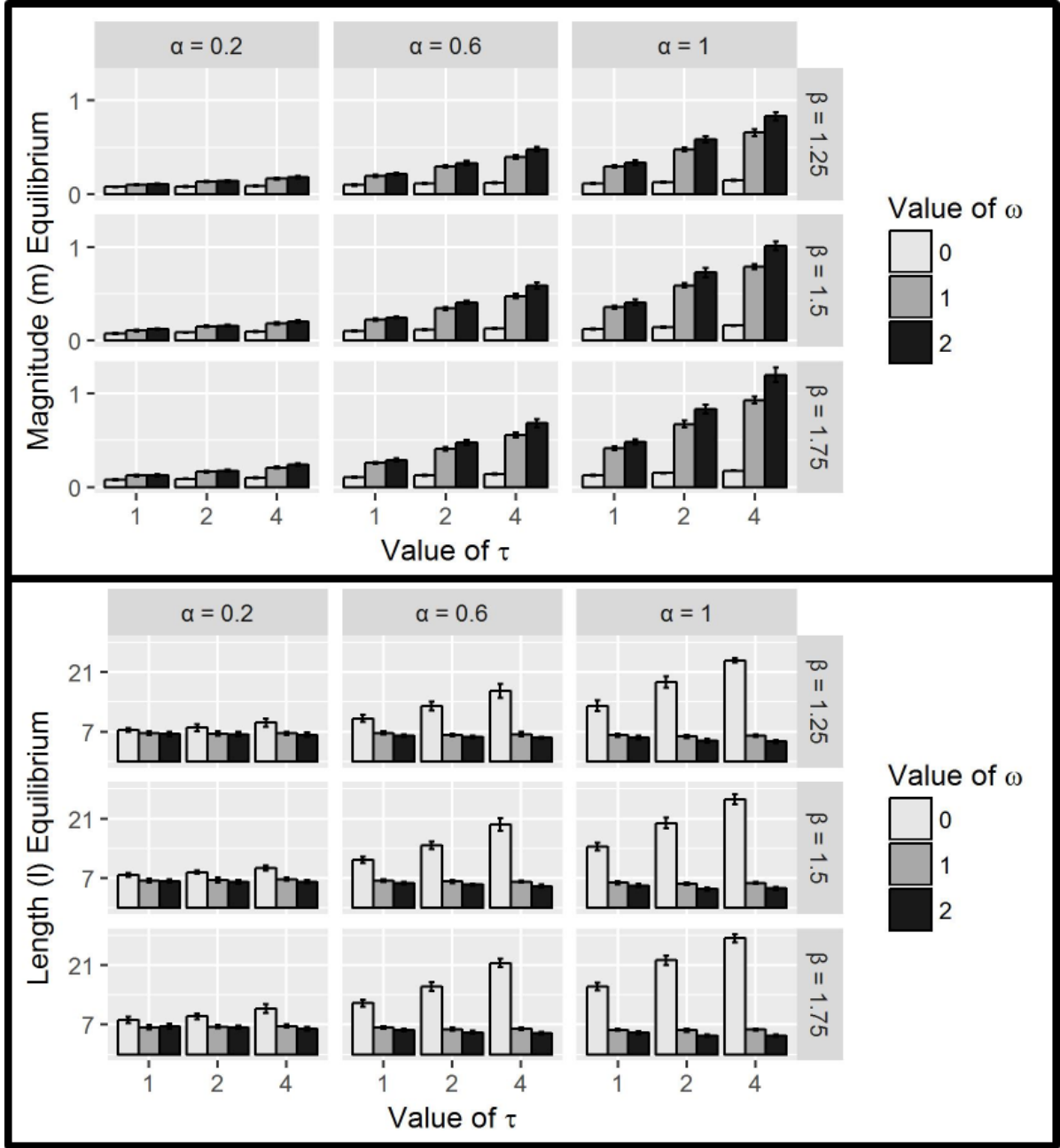


Figure 1.11: The effects of parameters α (maximum benefit of infanticide), β (proportional to the maximum cost of infanticide), τ (non-linearities in g), and ω (determines shape of $f(j)$) on the average equilibria values of both magnitude m (top graph) and length ℓ (bottom graph). Equilibria are obtained by averaging over 16 initial condition runs (with standard deviation indicated by error bars). Notice ovulation signs become stronger with increasing α , increasing β , and increasing τ . All other parameters were held constant: $N = 8, b = 0.2, c = 0.2, \epsilon_m = 0.25, \epsilon_f = 0.01, \gamma = 1, \kappa = 1, \eta = 0.2$.

8.4. Empirical Data

8.4.1. Statistical Tests

Two empirical comparisons were outlined in our main text between: (1) visible ovulation signs and group size, and (2) visible ovulation signs and risk of infanticide. Information for the phyloANOVA test for (1) is presented comprehensively in the main text.

We can also test both (1) and (2) using more powerful Bayesian methods. An overview, as applied to infanticide risk, is included in the main text. Model comparison for this test uses 5 different models: **bm1** (single-rate Brownian motion), **bms.root** (Brownian motion with different rate parameters for each state on a tree, taken from the root), **bms.means** (Brownian motion with different rate parameters for each state on a tree, taken from the means), **ou1** (Ornstein-Uhlenbeck model with a single optimum for all species), and **oum** (Ornstein-Uhlenbeck model with different state means).

For example, comparing models **ou1** and **oum** in our test of group size, model **ou1** being favoured implies there is no difference in group size means between species with “Absent” and “Present” ovulation signs, while model **oum** being favoured implies there IS a difference in the two group size means for species with “Absent” and “Present” ovulation signs. The values in Table 1.3 give the normalized AICw (relative likelihoods) for each of these five models for each of our two tests.

Table 1.3: Results of Statistical Tests

	bm1	bms.root	bms.means	ou1	oum
Group Size	0.043	0.17	0.17	0.17	0.45
Infanticide Risk	<0.001	<0.001	<0.001	0.38	0.62

8.4.2. Raw Data

Starting on the following page is Table 1.4 depicting the empirical data used in constructing Fig. 1.5 of the main text. The first two columns give the Family and Species Names, respectively. The third column depicts the species’ mating system, as classified by Opie et.al. (2013). The fourth column (“Visible Ovulation Signs”) gives the amount of ovulation signs visible (“Absent”, “Slight”, or “Present”). The fifth column (“Group Size”) gives the average group size for each species.

The sixth, seventh, and eighth columns (“Lactation Length”, “Gestation Length”, “Lactation / Gestation”) give the average length of lactation, average length of gestation, and ratio of “Lactation Length” to “Gestation Length”, respectively. Sources for all data in columns 4-7 are listed in the main text. Any cell left empty signifies missing data from these sources for that particular species/trait.

Table 1.4: Complete Raw Primate Data

Family Name	Species Name	Mating System	Ovulation Signs	Group Size	Weaning Age (L)	Gestation Length (G)	L/G
Cercopithecini	Allenopithecus nigroviridis	Polygynandrous	Present	40.00			
Lemuriformes	Allocebus trichotis	Monogamous		1.00			
Atelidae	Alouatta caraya	Polygynous/Polygynandrous	Absent	8.00	325.00	187.00	1.74
Atelidae	Alouatta palliata	Polygynous/Polygynandrous	Slight	12.00	325.00	186.00	1.75
Atelidae	Alouatta pigra	Polygynous/Polygynandrous		6.60			
Atelidae	Alouatta sara	Polygynous/Polygynandrous			371.00	191.00	1.94
Atelidae	Alouatta seniculus	Polygynous/Polygynandrous	Slight	7.10	372.00	191.00	1.95
Cebidae	Aotus azarae	Monogamous	Absent	3.10			
Cebidae	Aotus lemurinus griseimembra	Monogamous	Absent		75.00	133.00	0.56
Cebidae	Aotus nancymaeae	Monogamous	Absent	3.90	75.00	131.00	0.57
Cebidae	Aotus trivirgatus	Monogamous	Absent	2.90	75.00	133.00	0.56
Loridae	Arctocebus aureus	Polygynous			115.00	134.00	0.86
Loridae	Arctocebus calabarensis	Polygynous		1.00	105.00	135.00	0.78
Atelidae	Ateles belzebuth	Polygynandrous	Absent	22.13			
Atelidae	Ateles fusciceps	Polygynandrous	Absent		486.00	226.00	2.15
Atelidae	Ateles geoffroyi	Polygynandrous	Absent	15.00	750.00	225.00	3.33
Atelidae	Ateles geoffroyi panamensis	Polygynandrous	Absent				
Atelidae	Ateles geoffroyi vellerosus	Polygynandrous	Absent				
Atelidae	Ateles geoffroyi yucatanensis	Polygynandrous	Absent				
Atelidae	Ateles paniscus	Polygynandrous	Absent	18.20	760.00	230.00	3.30
Lemuriformes	Avahi laniger	Monogamous		3.00	150.00	136.15	1.10
Lemuriformes	Avahi occidentalis	Monogamous		3.50			
Atelidae	Brachyteles arachnoides	Polygynandrous	Absent	20.90	638.00	233.00	2.74
Hominoidea	Bunopithecus hoolock	Monogamous	Absent/Slight				
Pitheciidae	Callicebus donacophilus	Monogamous	Absent	1.00			
Pitheciidae	Callicebus moloch	Monogamous	Absent	3.30	60.00	164.00	0.37
Cebidae	Callimico goeldii	Polygynous/Monogamous	Absent	7.20	65.00	151.00	0.43
Cebidae	Callithrix argentata	Polygynous/Monogamous	Absent	9.50			
Cebidae	Callithrix aurita	Polygynous/Monogamous	Absent	7.50			
Cebidae	Callithrix emiliae	Polygynous/Monogamous	Absent				
Cebidae	Callithrix geoffroyi	Polyandrous/Monogamous	Absent	5.00			
Cebidae	Callithrix humeralifera	Polygynous/Monogamous	Absent	8.50			
Cebidae	Callithrix jacchus	Polyandrous/Monogamous	Absent	11.00	60.00	148.00	0.41
Cebidae	Callithrix kuhlii	Polygynous/Monogamous	Absent	5.00			
Cebidae	Callithrix penicillata	Polygynous/Monogamous	Absent	8.00			
Cebidae	Callithrix pygmaea	Monogamous	Absent	2.00	90.00	137.00	0.66
Cebidae	Cebus albifrons	Polygynandrous	Absent	25.10	269.00	155.00	1.74
Cebidae	Cebus apella	Polygynandrous	Absent	12.90	261.00	154.00	1.69
Cebidae	Cebus capucinus	Polygynandrous	Absent	16.40	510.00	162.00	3.15
Papionini	Cercocebus agilis	Polygynandrous	Present	21.50			
Papionini	Cercocebus galeritus	Polygynandrous	Present	18.20			
Papionini	Cercocebus torquatus	Polygynandrous	Present	26.90			
Papionini	Cercocebus torquatus atys	Polygynandrous	Present	90.00			
Cercopithecini	Cercopithecus ascanius	Polygynous	Absent	28.80	146.00	172.00	0.85
Cercopithecini	Cercopithecus campbelli lowei	Polygynous		14.00	362.00	180.00	2.01
Cercopithecini	Cercopithecus cephus	Polygynous	Absent	8.00	362.00	170.00	2.13
Cercopithecini	Cercopithecus diana	Polygynous	Absent	24.00			
Cercopithecini	Cercopithecus hamlyni	Polygynous	Absent				
Cercopithecini	Cercopithecus lhoesti	Polygynous	Absent	17.40			
Cercopithecini	Cercopithecus mitis	Polygynous	Absent	18.60	692.00	140.00	4.94
Cercopithecini	Cercopithecus mona	Polygynous	Absent	4.80			

Table 1.4 Cont'd: Complete Raw Primate Data

Family Name	Species Name	Mating System	Ovulation Signs	Group Size	Weaning Age (L)	Gestation Length (G)	L/G
Cercopithecini	Cercopithecus neglectus	Polygynous/Monogamous	Absent	4.50	365.00	165.00	2.21
Cercopithecini	Cercopithecus nictitans	Polygynous	Slight	13.00			
Cercopithecini	Cercopithecus petaurista	Polygynous		11.30			
Cercopithecini	Cercopithecus preussi	Polygynous		3.00			
Cercopithecini	Cercopithecus solatus	Polygynous		10.00			
Lemuriformes	Cheirogaleus crossleyi	Monogamous			45.00	70.00	0.64
Lemuriformes	Cheirogaleus major	Monogamous	Slight	1.00	70.00	71.00	0.99
Lemuriformes	Cheirogaleus medius	Polygynous/Monogamous	Slight	1.00	61.00	62.00	0.98
Cercopithecini	Chlorocebus aethiops	Polygynandrous	Absent	19.50			
Cercopithecini	Chlorocebus pygerythrus	Polygynandrous	Slight		201.00	163.00	1.23
Colobinae	Colobus angolensis	Polygynandrous	Absent/Slight	18.25			
Colobinae	Colobus guereza	Polygynous	Absent	12.00	330.00	170.00	1.94
Colobinae	Colobus polykomos	Polygynandrous	Absent/Slight	11.00	215.00	170.00	1.26
Lemuriformes	Daubentonia madagascariensis	Polygynandrous	Slight	1.00	170.00	164.00	1.04
Cercopithecini	Erythrocebus patas	Polygynous	Slight	28.20	213.00	167.00	1.28
Lemuriformes	Eulemur coronatus	Polygynandrous		9.25			
Lemuriformes	Eulemur fulvus albifrons	Polygynandrous			135.00	120.00	1.13
Lemuriformes	Eulemur fulvus albocollaris	Polygynandrous			135.00	120.00	1.13
Lemuriformes	Eulemur fulvus collaris	Polygynandrous		7.00	135.00	120.00	1.13
Lemuriformes	Eulemur fulvus fulvus	Polygynandrous		8.90	135.00	120.00	1.13
Lemuriformes	Eulemur fulvus mayottensis	Polygynandrous			135.00	120.00	1.13
Lemuriformes	Eulemur fulvus rufus	Polygynandrous		9.17	135.00	120.00	1.13
Lemuriformes	Eulemur fulvus sanfordi	Polygynandrous		5.00	135.00	120.00	1.13
Lemuriformes	Eulemur macaco	Polygynandrous	Slight	10.00	135.00	129.00	1.05
Lemuriformes	Eulemur mongoz	Monogamous		2.93	152.00	129.00	1.18
Lemuriformes	Eulemur rubriventer	Monogamous		3.00			
Galagonidae	Euoticus elegantulus	Polygynandrous		1.00			
Galagonidae	Galago alleni	Polygynous		6.00			
Galagonidae	Galago moholi	Polygynandrous		1.00	84.00	123.00	0.68
Galagonidae	Galago senegalensis	Polygynandrous	Slight	1.00	98.00	142.00	0.69
Galagonidae	Galagoides demidoff	Polygynous/Monogamous		10.00	45.00	110.00	0.41
Galagonidae	Galagoides zanzibaricus	Polygynous/Monogamous		1.00	59.00	126.00	0.47
Hominoidea	Gorilla beringei	Polygynous	Slight	11.10			
Hominoidea	Gorilla gorilla	Polygynous	Slight	7.10	1278.00	260.00	4.92
Lemuriformes	Hapalemur aureus	Monogamous		3.00	140.00	138.00	1.01
Lemuriformes	Hapalemur griseus griseus	Polygynous/Monogamous		4.40	120.00	140.00	0.86
Lemuriformes	Hapalemur simus	Polygynandrous		7.50			
Hominoidea	Homo sapiens	Polygynous/Monogamous	Absent	17.49	730.00	267.00	2.73
Hominoidea	Hylobates agilis	Monogamous	Slight	4.20			
Hominoidea	Hylobates klossii	Monogamous	Slight	3.00	330.00	210.00	1.57
Hominoidea	Hylobates lar	Monogamous	Slight	3.30	548.00	205.00	2.67
Hominoidea	Hylobates moloch	Monogamous	Slight	2.15			
Hominoidea	Hylobates muelleri	Monogamous	Slight	3.20			
Hominoidea	Hylobates pileatus	Monogamous	Slight	3.25			
Lemuriformes	Indri indri	Monogamous		3.20	363.00	159.00	2.28
Atelidae	Lagothrix lagotricha	Polygynandrous	Absent	33.10	315.00	223.00	1.41
Lemuriformes	Lemur catta	Polygynandrous	Slight	17.00	120.00	141.00	0.85
Cebidae	Leontopithecus chrysomelas	Polygynous/Monogamous	Absent	6.70			
Cebidae	Leontopithecus chrysopygus	Monogamous	Absent	3.60			
Cebidae	Leontopithecus rosalia	Polygynous/Monogamous	Absent	5.40	90.00	129.00	0.70
Lemuriformes	Lepilemur dorsalis	Polygynous		1.00			

Table 1.4 Cont'd: Complete Raw Primate Data

Family Name	Species Name	Mating System	Ovulation Signs	Group Size	Weaning Age (L)	Gestation Length (G)	L/G
Lemuriformes	Lepilemur_edwardsi	Monogamous		2.00			
Lemuriformes	Lepilemur_leucopus	Polygynous		1.00	121.66	130.00	0.94
Lemuriformes	Lepilemur_microdon	Polygynous		1.00			
Lemuriformes	Lepilemur_mustelinus	Polygynous		1.00	75.00	135.00	0.56
Lemuriformes	Lepilemur_ruficaudatus	Monogamous		1.00	119.00	150.00	0.79
Lemuriformes	Lepilemur_sahamalazensis	DD		1.00			
Lemuriformes	Lepilemur_septentrionalis	Polygynous			120.97	134.00	0.90
Papionini	Lophocebus_albigena	Polygynous/Polygynandrous	Present	15.80	210.00	186.00	1.13
Papionini	Lophocebus_atterimus	Polygynous/Polygynandrous	Present	17.50			
Loridae	Loris_hydekkerianus_malabaricus	Polygynandrous			135.00	167.00	0.81
Loridae	Loris_tardigradus	Polygynandrous/Monogamous	Slight	2.00	170.00	166.00	1.02
Papionini	Macaca_arctoides	Polygynandrous	Absent	31.10	393.00	178.00	2.21
Papionini	Macaca_assamensis	Polygynandrous	Slight	21.00			
Papionini	Macaca_brunnescens	Polygynandrous	Present				
Papionini	Macaca_cyclopis	Polygynandrous	Present	20.20	206.00	162.00	1.27
Papionini	Macaca_fascicularis	Polygynandrous	Present	27.50	330.00	160.00	2.06
Papionini	Macaca_fuscata	Polygynandrous	Slight	53.70	365.00	173.00	2.11
Papionini	Macaca_hecki	Polygynandrous	Present				
Papionini	Macaca_maura	Polygynandrous	Present				
Papionini	Macaca_mulatta	Polygynandrous	Slight	40.70	192.00	165.00	1.16
Papionini	Macaca_nemestrina	Polygynandrous	Present	18.20	234.00	167.00	1.40
Papionini	Macaca_nigra	Polygynandrous	Present	63.80			
Papionini	Macaca_nigrescens	Polygynandrous	Present	14.50			
Papionini	Macaca_ochreata	Polygynandrous	Present				
Papionini	Macaca_radiata	Polygynandrous	Absent	30.00	365.00	162.00	2.25
Papionini	Macaca_silenus	Polygynandrous	Present	19.60	365.00	180.00	2.03
Papionini	Macaca_sinica	Polygynandrous	Absent	24.50			
Papionini	Macaca_sylvanus	Polygynandrous	Present	18.20	210.00	165.00	1.27
Papionini	Macaca_thibetana	Polygynandrous	Slight	38.30	561.00	170.00	3.30
Papionini	Macaca_tonkeana	Polygynandrous	Present				
Papionini	Mandrillus_leucophaeus	Polygynandrous	Present	17.00			
Papionini	Mandrillus_sphinx	Polygynandrous	Present	13.90	348.00	175.00	1.99
Lemuriformes	Microcebus_murinus	Polygynandrous	Slight	1.00	40.00	60.00	0.67
Lemuriformes	Microcebus_myoxinus	Polygynandrous		1.00			
Lemuriformes	Microcebus_rufus	Polygynandrous		1.00	40.00	57.00	0.70
Cercopithecini	Miopithecus_talapoin	Polygynandrous	Present	66.50	180.00	162.00	1.11
Lemuriformes	Mirza_coquereli	Polygynandrous	Slight	1.00	136.00	87.00	1.56
Colobinae	Nasalis_larvatus	Polygynous	Slight	12.00	210.00	166.00	1.27
Hominoidea	Nomascus_concolor	Monogamous	Absent/Slight	4.00			
Hominoidea	Nomascus_gabriellae	Monogamous	Absent/Slight	1.00			
Hominoidea	Nomascus_leucogenys	Monogamous	Absent/Slight	1.00			
Loridae	Nycticebus_coucang	Polygynous	Slight	1.00	135.00	170.00	0.79
Loridae	Nycticebus_pygmaeus	Polygynous		1.00			
Galagonidae	Otolemur_crassicaudatus	Polygynandrous	Slight	6.00	135.00	135.00	1.00
Galagonidae	Otolemur_garnettii	Polygynandrous		1.00	140.00	132.00	1.06
Hominoidea	Pan_paniscus	Polygynandrous	Present	85.10	1080.00	240.00	4.50
Hominoidea	Pan_troglodytes	Polygynandrous	Present	63.10	1680.00	235.00	7.15
Hominoidea	Pan_troglodytes_schweinfurthii	Polygynandrous	Present		1680.00	235.00	7.15
Hominoidea	Pan_troglodytes_verus	Polygynandrous	Present		1680.00	235.00	7.15
Papionini	Papio_anubis	Polygynandrous	Present	57.70	584.00	180.00	3.24
Papionini	Papio_cynocephalus	Polygynandrous	Present	55.00	365.00	173.00	2.11

Table 1.4 Cont'd: Complete Raw Primate Data

Family Name	Species Name	Mating System	Ovulation Signs	Group Size	Weaning Age (L)	Gestation Length (G)	L/G
Papionini	Papio_hamadryas	Polygynous/Polygynandrous	Present	66.10	561.00	170.00	3.30
Papionini	Papio_papio	Polygynandrous	Present	50.00			
Papionini	Papio_ursinus	Polygynandrous	Present	34.70			
Loridae	Perodicticus_potto	Polygynous	Slight	2.00	150.00	170.00	0.88
Colobinae	Ptilocolobus_badius	Polygynandrous	Present	49.40			
Hominoidea	Pongo_abelii	Polygynandrous	Absent	2.00	720.00	250.00	2.88
Hominoidea	Pongo_pygmaeus	Polygynandrous	Absent	2.00	720.00	250.00	2.88
Colobinae	Presbytis_melalophos	Polygynous/Polygynandrous		14.50			
Colobinae	Procolobus_verus	Polygynous/Polygynandrous	Present	6.30			
Lemuriformes	Propithecus_coquereli	Polygynandrous		5.50			
Lemuriformes	Propithecus_diadema	Polygynandrous/Monogamous		5.38	183.00	178.00	1.03
Lemuriformes	Propithecus_edwardsi	Polygynandrous		4.60	210.00	179.00	1.17
Lemuriformes	Propithecus_tattersalli	Polygynandrous		4.10	153.00	170.00	0.90
Lemuriformes	Propithecus_verreauxi	Polygynandrous	Slight	5.00	180.00	162.00	1.11
Colobinae	Pygathrix_nemaeus	Polygynous/Polygynandrous	Slight	9.30			
Colobinae	Rhinopithecus_avunculus	Polygynous	Absent/Slight	30.00			
Colobinae	Rhinopithecus_bieti	Polygynous	Absent/Slight	50.00			
Colobinae	Rhinopithecus_roxellana	Polygynous	Absent/Slight	65.00			
Cebidae	Saguinus_fuscicollis	Polygynous/Polyandrous/Monogamous	Absent	5.10	90.00	150.00	0.60
Cebidae	Saguinus_geoffroyi	Polygynous/Monogamous	Absent	6.30	55.00	145.00	0.38
Cebidae	Saguinus_imperator	Polygynous/Polyandrous/Monogamous	Absent	7.00			
Cebidae	Saguinus_midus	Monogamous	Absent	5.55	70.00	127.00	0.55
Cebidae	Saguinus_oedipus	Polygynandrous/Monogamous	Absent	5.80	50.00	168.00	0.30
Cebidae	Saimiri_boliviensis	Polygynandrous	Slight	60.00			
Cebidae	Saimiri_oerstedii	Polygynandrous	Slight	55.30			
Cebidae	Saimiri_sciureus	Polygynandrous	Absent	38.00	168.00	170.00	0.99
Colobinae	Semnopithecus_entellus	Polygynous/Polygynandrous	Absent	30.20	249.00	184.00	1.35
Hominoidea	Symphalangus_syndactylus	Monogamous	Absent	4.00	639.00	232.00	2.75
Tarsiidae	Tarsius_bancanus	Polygynous/Monogamous	Slight	1.00	79.00	178.00	0.44
Tarsiidae	Tarsius_syrichta	Polygynous/Monogamous	Slight	1.00	82.00	180.00	0.46
Papionini	Theropithecus_gelada	Polygynous	Present	10.00	540.00	170.00	3.18
Colobinae	Trachypithecus_auratus	Polygynous		14.50			
Colobinae	Trachypithecus_cristatus	Polygynous	Absent	32.40	365.00	195.00	1.87
Colobinae	Trachypithecus_francoisi	Polygynous	Absent/Slight	8.90			
Colobinae	Trachypithecus_johnii	Polygynous	Slight	10.00			
Colobinae	Trachypithecus_obscurus	Polygynous	Slight	10.20			
Colobinae	Trachypithecus_phayrei	Polygynous	Absent/Slight	12.90	305.00	205.00	1.49
Colobinae	Trachypithecus_pileatus	Polygynous	Absent/Slight	7.80			
Lemuriformes	Varecia_rubra	Polygynandrous/Monogamous		4.19	89.00	102.00	0.87
Lemuriformes	Varecia_variegata	Polygynandrous/Monogamous	Slight	2.80	89.00	102.00	0.87

Chapter II

On the evolution of female sexual receptivity

Publication Statement: A version of this Chapter will be submitted soon for publication by its co-authors, Kelly Rooker and Sergey Gavrilets. The modeling and analytical work for this manuscript was primarily done by K. Rooker, and the manuscript also principally composed by K. Rooker.

On the evolution of female receptivity

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Abstract

There has been much interest in female primate reproductive cycles. Some primate species are like humans, sexually receptive to mating throughout their entire estrus cycle, while other species are the opposite, receptive for mere hours out of their several-week cycles. Why is there such prominent variation in sexual receptivity length among primate species? Here we examine the evolutionary trade-offs associated with sexual receptivity length using mathematical modeling. Our model predicts how various factors, including having ovulation signs present vs. concealed ovulation, female physiological costs, the weighting of genetic vs. non-genetic benefits coming from males, and group size, each influence the length of females' receptive period. Our model also predicts that increasing the impacts of infanticide will increase the length of the female receptive period, emphasizing the possible importance of paternity confusion. Overall, our work serves as the first quantitative look at receptivity length in female primates, and sheds light on the evolution of such receptivity length.

1. Introduction

Research on the unique behavioral changes associated with the estrus cycle of female mammals has been of great interest for over a century. As early as 1900, scientists were interested in when and why females were willing to receive male mates, noting that in most species there was a “special period of sexual desire...and only at that time, the female is willing to receive the male” (Heape, 1900, p.6).

1.1. Definitions

In general, there are three main characteristics used to describe the changes through which female mammals go when they are in estrus: sexual attractivity, proceptivity, and receptivity (Beach, 1976). Although the focus of this paper is on female sexual receptivity, much of the work surrounding the estrus cycles of female mammals has instead been in relation to *sexual attractivity*, that is non-behavioral cues, such as sexual swellings, bright colorations, etc. around the female’s genital region, that are used to signal probable times of ovulation. Concealed ovulation, also of much interest, is the lack of any such visual signs around a female’s time of ovulation. *Proceptivity* refers to behavioral cues displayed by females in order to initiate and/or maintain sexual interactions with males. Finally, *receptivity* is the willingness of a female to accept a male and allow copulation/intravaginal ejaculation with him to occur (Dixon, 2012).

Hence, attractivity can be thought of as the value to a male of a female being a sexual stimulus, and typically gets measured for primates in the field via male behaviors: frequency of approaches by males to a female, frequency of attempts by males to mount a female, etc. (Hrdy and Whitten, 1987). Proceptivity instead gets measured via female behaviors: counts of affiliative behaviors (e.g. female moving to sit near a male or to stay in his vicinity), more direct sexual solicitation (e.g. female presenting her hindquarters to a male or making species-specific vocalizations, facial expressions, and/or gestures like lip-smacking in baboons or head-bobbing in rhesus monkeys), investigation of the male’s anogenital region, female grooming of the male, etc. (Beach, 1976). (See (Dixon, 2012, p.133) for a more complete list of proceptive behaviors in female primates.)

Receptivity is harder to measure in the field since it requires a male approaching/wanting

to mate, but can be seen by the female shifting to a specific stance allowing for penile insertion/ejaculation, remaining still in that position, etc. (Hrdy and Whitten, 1987). Receptivity, even when the female is most fertile, is not passive. Females may avoid or actively refuse male mount attempts, or terminate the mount prior to ejaculation. In the field, receptivity in primates often gets measured by counting such refusals, acceptances, and terminations of male mounts by females (Dixson, 2012).

Also, it is important to note that even in species with long lengths of receptivity, matings will not necessarily be evenly distributed throughout such lengths of receptivity. Things like female desire and female-initiated matings may increase around the time of fertility, despite a female still being receptive for longer proportions of her cycle (Hrdy, 1997).

1.2. Receptivity in Non-Primate Mammals

Receptivity as an aspect of primate behavior is much different from the vast majority of non-primate mammals (Beach, 1976). In most non-primate mammals, receptivity is limited to the peri-ovulatory (i.e. around the time of ovulation) phase of the female's estrus cycle, and strongly dependent on both hormones estrogen and progesterone. Prosimian primates (lemurs, galagos, etc.) also tend to be more like non-primate mammals in that they only have limited periods of sexual receptivity during their peri-ovulatory phase (Dixson, 2012). Not only is this time of receptivity typically brief in non-primate mammals and prosimians, but in many species (including galagos), the vagina is actually fused shut, or otherwise completely covered, outside the female's receptive period, making mating outside of this time physically impossible (Hrdy and Whitten, 1987).

In addition, in many non-primate mammals and prosimian primates, ovariectomy (surgical removal of the ovaries and hence also removal of the ovary-produced hormones estrogen and progesterone) results in a complete lack of sexual receptivity in the female, with females even attacking males attempting to copulate with them in the case of galagos. This is quite different from the anthropoid primates (including humans), in which copulations will still occur, even after removal of the ovaries (Dixson, 2012).

1.3. Variation in Receptivity among Primates

Among all species of primates, there is great variability in the duration of the estrus cycle where the female is receptive. Some species are like humans or bonobos in which individual females are receptive throughout their entire cycle, while other species are like gorillas or the prosimians, in which individual females are receptive exclusively during the middle of their cycles around the time of ovulation (Beach, 1976). In some species, females will initiate the majority of all matings (e.g. in *Gorilla*, *Ateles*, *Alouatta*, *Cebus*, and *Rhinopithecus*), while in other species females initiate 0% of all mounts (e.g. in greater galagos and owl monkeys). Other primate species lie more in between, with females initiating 18% of all mounts in free-ranging chimpanzees, 50% in vervet monkeys, and 69% in lion-tailed macaques (Dixon, 2012).

1.4. Other Aspects of Receptivity

Species whose females are receptive throughout their entire cycle are referred to as being ‘continuously receptive’. Many species engage in what is called ‘situation-dependent receptivity’, i.e. when a female becomes receptive despite not being in the middle of her cycle. This most frequently occurs when the female encounters an unfamiliar male. For example, non-receptive females (including in some cases even pregnant females) in gray langurs and gelada baboons will display both proceptive and receptive behaviors within days of new males invading their group (Hrdy and Whitten, 1987).

Longer receptivity is clearly *less* important in species where forced copulations are the norm (Clarke et al., 2009). However, in many primate species, the female takes an active role in mating via proceptive behaviors and many males wait for such behaviors prior to attempting to mate (Beach, 1976). On the other hand, longer receptivity could be *more* important in select species where ‘food-for-sex’ is the norm (i.e. the female receives provisionings or other resources from a male in exchange for her copulating with him) (Stockley and Bro-Jorgensen, 2011). Similarly - and more commonly - the female could instead be trading copulations with a male for protection of herself against predators, harassment, etc., which could also provide selection pressure on the lengthening of receptivity. Alternatively, females could increase their fitness by mating with males more likely to provide paternal care to their offspring (Clutton-Brock, 2016). Note all these different costs and

benefits of receptivity can be thought of as encapsulated together in a single ‘benefit-to-cost’ ratio, what we term later the ‘benefit of paternal male quality’.

1.5. *Female Competition for Preferred Mates*

As stated in a review paper on female-female competition, “Female competition for the sperm of preferred (or competitively successful) males could be a potentially widespread but previously overlooked evolutionary force” (Stockley and Bro-Jorgensen, 2011, p.361). Indeed, receptivity may function less in securing females *any* mate, but rather in securing them their *preferred* mate (i.e. a high-quality male). In these situations, females are trying to *concentrate paternity* of their offspring in that preferred mate. At least in some contexts, sperm limitation may play a role (Clutton-Brock, 2016). In both gelada baboons and hamadryas baboons, there is a negative relationship between conception rate and the ratio of estrus females to males. Similarly, in gorillas, females will receive fewer copulations when other females are in estrus simultaneously (Stockley and Bro-Jorgensen, 2011). Note that such preference in females for ‘high-quality males’ can be thought of not only as a preference for good genes, but also in maturity, rank, fertility, protection, investment, vocal or visual displays, etc. (Clutton-Brock, 2016).

In general, there exists a trade-off between the benefits of a female being receptive for a longer amount of time vs. a female being receptive for shorter. A female with a shorter period of receptivity would have less time in her cycle to mate, which helps both concentrate paternity in high-quality males and also avoid the physiological costs of having longer receptivity. On the other hand, a female with a longer period of receptivity would have more time to mate with multiple males in one cycle (van Schaik et al., 1999). Possible benefits of mating with multiple mates in one cycle include reduced risk of infertility, increased protection of offspring against predators, increased investment of offspring by multiple males (but see (Soltis and McElreath, 2001)), and genetic diversity (Clutton-Brock, 2016).

1.6. *Infanticide*

Another important possible benefit to females from both paternity concentration and paternity confusion - and hence also a possible effect on female receptivity length - comes from infanticide.

Infanticide, the killing of a female's offspring prior to weaning, is extremely detrimental to a female's fitness (van Schaik et al., 2004; Hrdy, 1979). However, committing an act of infanticide may benefit any male who is not the father of that particular offspring, and hence infanticide is often seen when a new male takes over a group (Pradhan and van Schaik, 2008). Since many female primates have prolonged lactational amenorrhoea (van Schaik et al., 1999), a male killing a female's offspring results in bringing that female back to fertility sooner by prematurely ending her lactation (Lovejoy, 2009; Palombit, 1999). The infanticidal male can then subsequently mate with this female, meaning his offspring will be able to be born sooner than would otherwise be possible had he never committed infanticide (Stallman and Froehlich, 2000). Although infanticidal events are typically rare, they have been documented in many primate species (Hrdy, 1979; Opie et al., 2013), and constitute up to 64% of all infant mortality in some primate species (van Schaik et al., 1999).

Indeed, since male decisions about whether or not to help or harm an infant are primarily based on their mating history, one prominent counterstrategy used by females to protect against infanticide is the manipulation of males' probabilities of paternity, either actual or perceived. For example, if infanticide is likely to only be committed by one male in a group, it may be in females' best interests to concentrate their future offspring's paternity in him. This would help ensure that he believes he is the father of her offspring, and hence he would be much less likely to kill the offspring, and may even go out of his way to help protect it should any other male attempt to commit infanticide against that offspring (van Schaik et al., 1999). Observational and genetic data both confirm that infanticide is rarely committed by the father of that offspring (Clutton-Brock, 2016).

Conversely, paternity confusion could also play a role in protecting an offspring from infanticide. If infanticide may be committed by multiple males in the group, or any new outsider male who takes over the group, it would instead likely be in the female's best interest to mate with as many of those males during one cycle as she possibly can, in order to help confuse paternity, i.e. make multiple males think they may be the father of her offspring (van Schaik et al., 1999). Indeed, infanticide could have provided the main selection pressure favoring situation-dependent receptivity, including continuous receptivity. Such a female with the capacity to facultatively and opportunistically solicit

sexual behavior would be better equipped to manipulate males' probable paternities, thus lowering the risk that her infant might be killed (Hrdy, 1979).

1.7. Research Questions

“Anthropologists have long been concerned with explaining why human females are continuously sexually receptive” (Hrdy and Whitten, 1987, p.384). However, while there has been much interest in why *humans* evolved to be continuously receptive, this question ignores the fact that many other diverse primate species are also continuously receptive, or at least willing to mate at times outside their fertile periods (Hrdy and Whitten, 1987). As Alan Dixson says, “Since there is no fossil record of sexual behavior and no direct means of studying the origins of primate sexuality, the comparative approach might indeed provide the only means of shedding light upon these questions” (Dixson, 2012, p.128).

Although there have been at least two studies utilizing this comparative approach as it relates to primate female sexual receptivity (see (Hrdy and Whitten, 1987; Stockley, 2002)), to our knowledge there has yet to be any mathematical modeling work done on the evolution of primate sexual receptivity. The lack of quantitative research on this topic is important since there is no fossil record or other empirical evidence as to past primate sexuality. In addition, as discussed above, there are very clear evolutionary trade-offs whose respective influences can easily be investigated using mathematical modeling. As such, it is our aim to address this gap. Here, we develop a mathematical model to investigate the evolution of female sexual receptivity in primates. In particular, we are interested in the questions: How does length of receptivity contribute to female reproductive success? What factors facilitate the evolution of continuous receptivity and/or very short lengths of receptivity? What role could infanticide have in the evolution of receptivity? How is receptivity linked to the evolution of sexual attractiveness, namely a female having visual ovulation signs or concealed ovulation?

2. Introduction to the Model

2.1. Model Set-Up

Similar to Chapter 1, we construct an agent-based model using a population of individuals living in a large number of multi-male, multi-female groups. See Methods for full details of the

model. We assume generations to be discrete and non-overlapping. Females can differ genetically in both their visible ovulation signs present and their length of receptivity, while males differ in their quality (benefit) to females. Such quality is split into both a genetic component (GC) and a non-genetic component (NGC). GC is the genetic benefit to a female's offspring of her mating with a male with good genes, while NGC can be thought of as any non-genetic benefit to a female from mating with that male. For instance, NGC could be protection provided to the female herself (i.e. not her offspring), increased food/provisioning/access to resources, and/or paternal care (see Ref. (Clutton-Brock, 2016) for further description of such benefits). Note that GC and NGC are inversely related such that a male with higher GC will have lower NGC and vice versa.

We account for group size (N), variation in male quality (scaled by parameter b), the costs of having ovulation signs visible (scaled by parameter c), the costs of receptivity (c_r), the weighting of NGC vs. GC (η), and the effects of infanticide. Our goal is to determine how female receptivity length evolves over time, both with and without visual ovulation signs simultaneously evolving. Female fitness is given by:

$$\begin{aligned}
\text{female fitness} &= \text{baseline female fitness} \\
&+ (1 - \eta) * (\text{benefit of male GC}) \\
&+ \eta * (\text{total average benefit of male NGC}) \\
&\pm \text{benefit/cost due to infanticide from each male} \\
&- \text{cost of having visible ovulation signs} \\
&- \text{cost of receptivity.}
\end{aligned}$$

2.2. The Female Cycle

Our model explicitly accounts for the female cycle, which is split into discrete units of time. While such units could be hours, minutes, etc., we choose to call such units 'days' (for example, with 29 days in one female cycle). Each female is characterized by one genetically controlled trait, r , the length of time the female is receptive to mating (a non-negative, integer value). In addition, females may also be characterized by their visual ovulation signs, denoted $x(d)$ for each day d of the cycle,

thought of as overlapping curves (Dixon, 2009). Ovulation magnitude trait m is the maximum amount of ovulation signs a female has visible during her cycle (a non-negative, continuous value), while ovulation length trait ℓ is the number of days a female has *some* amount of ovulation signs visible (a non-negative, integer value). For an example of what receptivity length r and ovulation signs $x(d)$ could look like across a cycle, see Fig. 2.1.

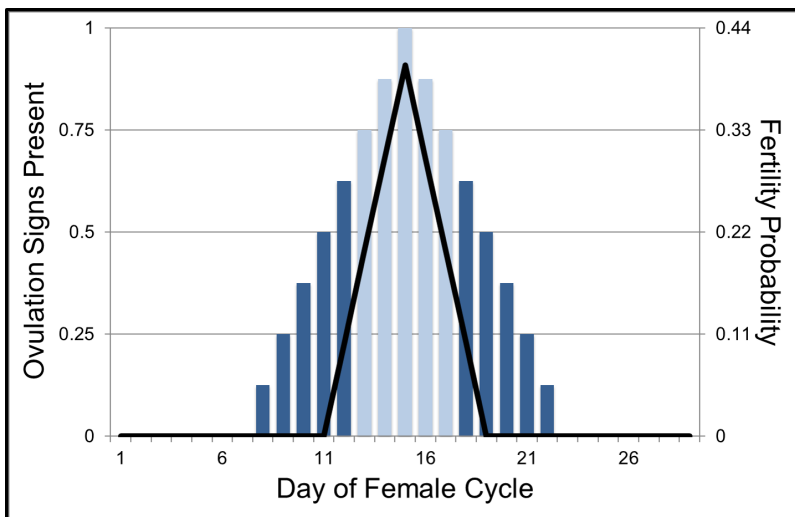


Figure 2.1: Example ovulation signs for one female with $r = 5$, $m = 1$, $\ell = 15$. Fertility probability is depicted by the solid black line and visible ovulation signs by the blue bars. Light blue bars indicate ovulation signs on the female’s days of receptivity, while dark blue bars indicate days on which the female still has ovulation signs present but is not receptive to mating. In this example, ovulation is assumed to happen on day 15, the female’s day of peak fertility.

For each female, the distribution of probabilities of fertilization across the cycle has the same shape but is randomly centered (meaning cycle synchrony may only occur probabilistically). Visual ovulation signs and receptivity are correlated with these days of fertility, but can each still evolve independently. This means we assume there exists at least some reliability of the ovulation signal (since a female’s peak day of fertility will also be her day of peak ovulation signs *and* her median day of receptivity), and that such reliability is the same in all females.

2.3. Male-Female Mating

For every unit of time in the cycle, all males and all receptive females in each group enter the competition for mates. For computational simplicity, we assume each individual to mate no more than once on each unit of time (e.g. day). Males of higher GC are more likely to mate with receptive

females with stronger ovulation signs visible (higher $x(d)$), i.e. when the female is more likely to be most fertile. Males of lower GC mate with receptive females who have fewer ovulation signs visible, i.e. when the female is less likely to be most fertile.

The degree of reproductive stochasticity in males and females is controlled by parameters $\epsilon_m \geq 0$ and $\epsilon_f \geq 0$, respectively. The higher ϵ_m is, the more likely low-GC males are to be able to mate with females with the strongest signal. The higher ϵ_f is, the more unreliable the female signal is in winning high-GC mates. With $\epsilon_m = \epsilon_f = 0$, there is no reproductive stochasticity and mating pairs are formed deterministically so that the highest-GC male mates with the female with the most ovulation signs visible, and so on). (See Methods for a full description of these parameters.)

Mating is followed by offspring production and dispersal. For any female's potential offspring, every male in the group will have an associated probability of paternity, ranging anywhere from zero to one. This quantity is determined by both the number of times that particular male mates with the female during her cycle, as well as her probability of fertilization on the days of each mating event.

2.4. *Infanticide*

Modeling infanticide is similar to as in Chapter 1. Any male is able to help or harm (note 'harm' here could simply mean not helping) an offspring prior to the infant being weaned, although we typically assume that some males (e.g. those larger and/or stronger) are able to do so more effectively. Our model does not distinguish between the threat of infanticide via an outsider male or a previously-subordinate male. Infanticide's effects on offspring viability are scaled by parameters α (maximum benefit) and β (relative maximum cost). An additional parameter $0 \leq \kappa \leq 1$ determines the extent to which males take visible female ovulation signs into account when estimating their paternity. $\kappa = 0$ means males estimate their probability of paternity solely by the *number* of matings with an offspring's mother, while $\kappa = 1$ means males instead estimate paternity exclusively on the basis of the female's visible ovulation signs during their time of mating.

3. Methods

3.1. General Model

We consider a population of individuals interacting in G groups, each comprised of N males and N females. Males are ordered according to their GC, denoted y_j such that $y_1 > y_2 > \dots > y_N$ for each male j . Intuitively, females will gain more fitness benefit from mating with males of higher GC. Such GC is randomly drawn from the normal distribution $\mathbb{N}(\hat{y}, b)$; parameter $\hat{y}$ characterizes mean male GC and $b > 0$ the extent of variation in male GC. Small b implies small potential benefits to females from mating with even high-GC males. [For simplicity, the distribution of y in the population remains constant, e.g. as a result of mutation-selection balance.] Male NGC is then equal to the reflection of the male's GC over the center of the normal distribution, the vertical line $y = \hat{y}$.

Our model explicitly accounts for the female cycle by using D discrete units of time. Without loss of generality, we refer to these units of time as ‘days’ (e.g. $D = 29$ days). We assume each female to mate once on every day on which she is receptive, and each male to be able to mate with no more than one female on any given day. Mating is followed by offspring production and dispersal.

For each day of the cycle, every female will have an associated probability of fertilization. We assume each fertile period to last $C \leq D$ days (e.g. $C = 7$). For all days lying outside these C fertile days, females are assumed to have zero probability of fertilization but can still mate if receptive on those days. We assume a triangular shape for the fertility function to be identical in all females, but with midpoints randomly distributed so as cycle synchrony occurs only probabilistically. Visual ovulation signs and receptivity are both correlated with these days of fertility (so that a female's day of peak ovulation signs will align with both her median day of receptivity and her peak day of fertility), but can each still evolve freely.

Let r be the number of days of the cycle a female is receptive to mating (a non-negative, discrete value). We treat visual ovulation signs, $x(d)$ for each day d of the cycle, as overlapping graded curves. Each female's curve is characterized by two traits: magnitude (m) and length (ℓ). m is the maximum amount of ovulation signs a female has visible during her cycle (a non-negative,

continuous value), while ℓ is the number of days a female has *some* amount of ovulation signs visible (a non-negative, discrete value).

Considering one focal group, for any female i , together $m_i \geq 0$ (ovulation signs magnitude) and $\ell_i \in \mathbb{Z}, \ell_i \in [0, D]$ (ovulation signs length) determine the amount $x_i(d)$ of ovulation signs visible on any day d of the cycle:

$$x_i(d) = \begin{cases} m_i \cdot \left[1 - \left(\frac{2|d - \lceil \ell_i/2 \rceil|}{\ell_i + 1} \right)^\gamma \right] & \text{for } d = 1, 2, \dots, \ell_i \\ 0 & \text{otherwise} \end{cases} \quad (19)$$

for parameter $\gamma > 0$ and d measured from the start of ovulation signs being visible. Note m_i represents the peak of $x_i(d)$ and ℓ_i the width of the non-zero portion of $x_i(d)$. Mutations in m_i are continuous, whereas mutations in ℓ_i and r_i are discrete, representing adding or subtracting exactly one day to or from a female's time of having ovulation signs visible or being receptive, respectively.

On each unit of time, mating pairs are formed by first randomly perturbing both the male trait y and female trait x by adding to each an independent, normally distributed random variable with standard deviation ϵ_m and ϵ_f , respectively. Males and receptive females are then sorted according to these perturbed values and mating occurs between individuals of the same order. This means if there are R receptive females on any given day of the cycle, there will only be R males able to mate with a female on that day. Stochasticity in male ordering reflects stochasticity in the outcome of their competition for mating success; a large ϵ_m means lower reproductive inequality. Stochasticity in female ordering can reflect a lack of opportunity to mate with a higher quality male/female, disinterest in mating with a specific male (female) regardless of quality (signs), variability in signal reliability, etc.

For any female's potential offspring, every male in the group will have an associated probability of paternity. This quantity is determined by both the number of times that particular male mates with the female during her cycle, as well as her probability of fertilization on the days of each mating event. Selection occurs for males via mating success and for females via fertility selection.

Without considering the effects of infanticide, we have female fitness:

$$w_i = w_0 + (1 - \eta)y_{m,i} + \frac{\eta}{D} \sum_{\text{all mates } j} y_{p,j} - c \cdot \bar{x}_i - c_r \cdot \bar{r}_i. \quad (20)$$

Here, w_0 is baseline fitness, $y_{m,i}$ the benefit from female-female competition (i.e. the GC of the specific male who won the contest of paternity for that particular female's offspring), $y_{p,j}$ the NGC coming from each male j a female mated with throughout her cycle, $c \cdot \bar{x}_i$ the costs to a female of supporting her visual ovulation signs with $\bar{x}_i = \frac{1}{D} \sum_{d=1}^D x_i(d)$, a female's average visual ovulation signs, and $c_r \cdot \bar{r}_i$ the costs to a female of receptivity with $\bar{r}_i = \frac{r}{D}$, a female's proportion of the cycle being receptive. Note our choice of the first cost term implies there are fitness trade-offs between a female's m and ℓ traits so that having large values of both traits could imply very large fitness costs.

3.2. Modeling Infanticide

Each male in a female's group has the potential to affect her fitness directly, due to infanticide. This happens with probability $f(j)$ for each male j , typically assuming that males of higher GC will be more effective in helping or harming an offspring (due to body size, strength, etc.). Male j 's contribution $g(p_{i,j})$ to female fitness (positive via helping protect the offspring or negative via harming/not helping) depends on his perceived probability of paternity $p_{i,j}$ for an offspring born to female i . The overall effect of infanticide on fitness for female i (that gets added to the right-hand side of Eq. 20) is $\sum_j f(j)g(p_{i,j})$, summing over all males j .

Following Chapter 1, we define $g(p_{i,j}) = \alpha[1 - \beta(1 - p_{i,j})^\tau]$, with parameters $\alpha, \beta > 0$ and $\tau \geq 1$. Note α determines the maximum benefit a female can obtain from a male protecting her offspring from infanticide, while $\alpha(1 - \beta)$ determines the maximum cost a female can incur from a male *not* protecting her offspring from infanticide. Whenever $\alpha = 0$, there are no effects of infanticide.

A male in general would not know his actual paternity probability and would instead have to estimate it. We define this male-estimated ("perceived") probability by male j of being the father of an offspring born to female i by $p_{i,j} = \sum_{\# \text{ of matings}} (x_i(d))^\kappa$, where $\kappa \in [0, 1]$ represents the weight males put on visual ovulation signs when estimating paternity. Note $\kappa = 0$ represents males estimating their paternity solely on the number of matings with that mother, while $\kappa = 1$ represents

males putting full weight instead on her visual ovulation signs. For each male r , these perceived paternity probabilities get normalized such that $\sum_{i=1}^N p_{i,j} = 1$.

Male effectiveness is set proportional to an exponential function: $f(j) \sim e^{-\omega j}$ with parameter $\omega > 0$ controlling the amount of disparity among males in their corresponding effectiveness. Note a larger value of ω indicates more disparity, and $\omega = 0$ equality. More specific model details can be found in the Supplemental Information.

3.3. Simulations

For this model, we consider 3 different cases regarding selection:

- (1) Evolution of receptivity (r) given fixed concealed ovulation ($m, \ell = 0$)
- (2) Evolution of receptivity (r) given fixed ovulation signs present ($m = 0.9, \ell = 4$), and
- (3) Evolution of receptivity (r), ovulation magnitude (m), and ovulation length (ℓ).

For (1) and (2), we use the following 10 initial conditions: $r = 1, 4, 7, 10, 13, 16, 19, 22, 25, 28$. For (3)'s initial conditions, we use every combination of $r = 7, 21$, $m = 0, 0.9$, and $\ell = 1, 4$. Parameters used for all the above simulations include: $G = 400$; $T = 100,000$; $D = 29$; $C = 7$ (with probabilities of fertilization on each of these days being 0.125, 0.25, 0.375, 0.5, 0.375, 0.25, 0.125, respectively); $w_0 = 2$; $N = 4, 8, 16$; $b = 0.1, 0.2, 0.4$; $c = 0.1, 0.2, 0.4$; $c_r = 0.1, 0.2, 0.4$; $\eta = 0.1, 0.2, 0.4$; $\gamma = 1$; $\hat{y} = 1$; $\epsilon_m = 0, 0.25, 0.5$; $\epsilon_f = 0, 0.01, 0.05$; $\alpha = 0, 0.2, 0.6, 1$; $\beta = 1.25, 1.5, 1.75$; $\tau = 2$; $\omega = 1$; and $\kappa = 0, 0.5, 1$.

This model was implemented in C++. Migration occurs with only females migrating between groups, although simulations were also run with only males migrating between groups and the results were unaffected. Mutation occurs with a mutation rate per gene per generation of 10^{-3} . Mutations for m are selected from $N(0, 0.1)$, while mutations for ℓ and r independently from the set $\{-1, 1\}$ with equal probability. $2N$ offspring are created in each group every generation, with N female and N male, determined randomly. The expected number of offspring per female is 2; the actual number is random, proportional to each female's fitness.

4. Results

For this model, we consider 3 different cases regarding selection:

- (1) Evolution of receptivity (r) given fixed concealed ovulation ($m, \ell = 0$)
- (2) Evolution of receptivity (r) given fixed ovulation signs present ($m = 0.9, \ell = 4$), and
- (3) Evolution of receptivity (r), ovulation signs magnitude (m), and ovulation signs length (ℓ).

For each of these 3 different selection cases, we investigate our model both with and without the effects of infanticide. Numerical simulations of our model in all cases show that the average values of the receptivity trait (r) (and ovulation signs magnitude trait (m) and ovulation signs length trait (ℓ), if included) converge to the unique equilibria r^* (and m^*, ℓ^* , if included). A more complete investigation of the effects of all parameters can be found in the Supplemental Information.

4.1. Case 1: Evolution of receptivity (r) given fixed concealed ovulation ($m, \ell = 0$)

Results from Case 1 can all be seen in the white bars of Fig. 2.2. Under conditions of concealed ovulation, we intuitively expect that increasing the receptivity costs parameter c_r will decrease receptivity length equilibrium r^* . We also expect that increasing η will result in increased r^* . Increasing η increases the benefit going to females from NGC. Since such benefit is non-genetic, females can obtain it throughout their cycle, leading to the continuous receptivity outcomes seen with larger values of η .

We expect that increasing b will increase receptivity length r^* , because increasing b increases the benefit coming to females from winning the female-female competition for higher-GC mates. Since there are no visible ovulation signs present in this case, fertile females have no way of “attracting” high-GC males, and such matings will only happen by chance. Thus, increased female-female competition for high-GC males results in longer lengths of receptivity. Note these results are particularly intuitive when thinking of parameter b as the benefit coming from food-for-sex exchanges or increased protection/defense by males. Indeed, these intuitive patterns do hold in our simulations, and are all visible in the white bars in Fig. 2.2(a,c).

We also see increased receptivity length r^* with increased effects of infanticide (i.e. increased α and β). These results are intuitive since with stronger effects of infanticide comes an increased

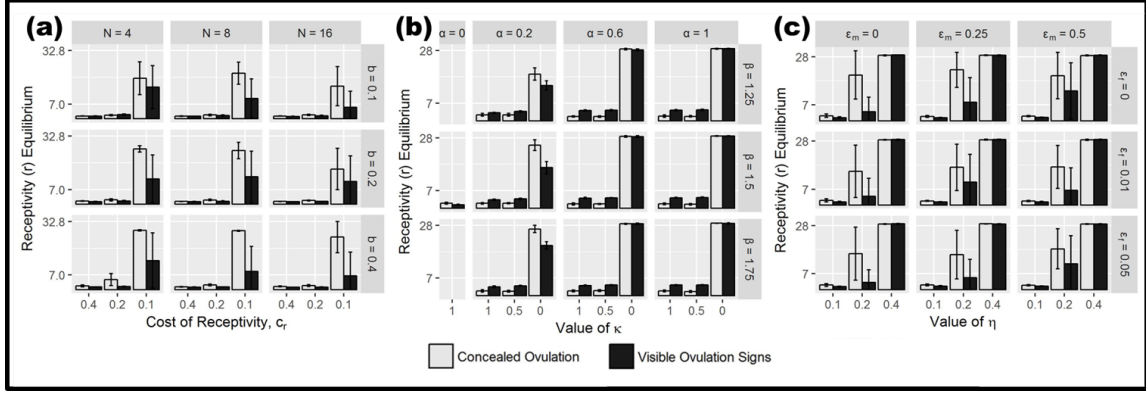


Figure 2.2: The effects of varying parameters on the average equilibria values of receptivity (r^*) in the case when only r is allowed to evolve. White bars indicate Case 1 (assuming concealed ovulation: $m, \ell = 0$), while black bars indicate Case 2 (assuming fixed visible ovulation signs: $m = 0.9, \ell = 4$). Equilibria are obtained by averaging over 10 initial condition runs (with standard deviation indicated by error bars). (a) Varying N (group size), b (variation in male quality), and c_r (costs of receptivity) in the case of no infanticide ($\alpha = 0, c = 0.2, \epsilon_m = 0.25, \epsilon_f = 0.01, \eta = 0.2$). (b) Varying α (maximum benefit of infanticide protection), β (maximum cost of infanticide), and κ (weight males put on visible ovulation signs in estimating paternity) in the case of with infanticide ($N = 8, b = 0.2, c = 0.2, c_r = 0.2, \epsilon_m = 0.25, \epsilon_f = 0.01, \eta = 0.2$). (c) Varying ϵ_m (male reproductive stochasticity), ϵ_f (female reproductive stochasticity), and η (weighting of NGC vs. GC on female fitness) in the case of no infanticide ($\alpha = 0, N = 8, b = 0.2, c = 0.2, c_r = 0.2$).

benefit to females of mating with multiple males. Having a longer length of receptivity allows females more time to be able to mate with multiple males, which in turn makes multiple males think they may be the father of her offspring and hence help protect (or at least not harm) that offspring.

Finally, recall parameter κ controls how much males take visible ovulation signs into account when estimating their paternity. In the concealed ovulation case with $\kappa = 0$, decreasing κ results in increased receptivity length r^* . Even with concealed ovulation, if males only take the number of matings with a female into account when calculating their perceived probability of paternity, females are able to get benefit from mating at any point of their cycle. Hence there becomes an increased benefit to females for having longer lengths of receptivity. These effects are all visible in the white bars in Fig. 2.2(b).

4.2. Case 2: Evolution of receptivity (r) given fixed ovulation signs present ($m = 0.9, \ell = 4$)

Results from Case 2 can all be seen in the black bars of Fig. 2.2. Looking at receptivity length under conditions of fully visible ovulation signs, we see the same effects as in Case 1 of parameters b, c_r, α, β , and η . The parameter κ also has the same effects as in Case 1 for similar reasons. As

males put less weight in the amount of visible ovulation signs a female has when mating with her, females are able to get more benefit in mating with multiple males by mating at times of their cycle when they have fewer (or even no) signs visible. Hence there again becomes an increased benefit to females for having longer lengths of receptivity with smaller κ . These effects are all visible in the black bars of Fig. 2.2(a,c) (no infanticide) and Fig. 2.2(b) (with infanticide).

4.3. Case 3: Evolution of receptivity (r), ovulation magnitude (m), and ovulation length (ℓ)

We now allow for each of receptivity length (r), magnitude of visible ovulation signs (m), and length of visible ovulation signs (ℓ) to independently evolve. We compare these results with not just each other, but also with results for m^* and ℓ^* when r does not evolve and continuous receptivity is instead assumed. These results can all be found in Fig. 2.3 (a,c, without infanticide and b, with infanticide). [Note full results for the evolution of m and ℓ with continuous receptivity assumed can be found in Chapter 1.] In general, in Fig. 2.3(b) we see receptivity length equilibria (r^*), ovulation signs magnitude equilibria (m^*), and ovulation signs length equilibria (ℓ^*) increase with the impacts of infanticide α and β . As discussed above and in Chapter 1, these results are intuitive. In addition, in Fig. 2.3(a) we see the costs of having visual ovulation signs c to have similar effects as in Chapter 1 on m^* and ℓ^* , both as expected.

We also begin to see a trade-off between receptivity length and ovulation signs magnitude. For example, with increasing benefit from males b , we see in Fig. 2.3(a) increased ovulation signs magnitude and length (as expected), but *decreased* length of receptivity. Increasing b increases the female-female competition for high-quality mates, which increases ovulation signs magnitude in order to help concentrate paternity in high-GC males. However, it also becomes in females' best interests to limit their length of receptivity in order to further concentrate paternity. If a female is receptive longer than the length of time she has strong ovulation signs visible, she would end up mating with lower-GC males and hence would not be concentrating paternity for her offspring primarily in high-GC males.

Similarly, we see in Fig. 2.3(b) increasing κ (parameter controlling how much males take visible ovulation signs into account when estimating their paternity) increases m^* and decreases r^* . Again, this is due to the importance of paternity concentration. The stronger the magnitude of the

ovulation signal, the more beneficial it is to the female to only be receptive around those day(s) of having a peak signal, thus concentrating paternity in high-quality males. We also see this pattern with η in Fig. 2.3(c): as we increase η , r^* increases and m^* decreases. This makes sense since increasing η is increasing the weight being put on NGC in female fitness. Hence, with larger η , females will get more benefit from being receptive for longer, even longer than they are fertile.

In the extreme case, when $\kappa = 0$, males are only taking the *number* of matings with a female into account when estimating their paternity, thus ignoring any visible ovulation signs present for their paternity estimate (but still using such signs to find the most likely fertile mates). As the effects of infanticide increase, females gain increasing benefit from mating with multiple males during any one cycle. With $\kappa = 0$, a female is able to make males think they might be the father of her offspring by mating with them even when she has zero ovulation signs visible. Hence it becomes advantageous for obtaining multiple matings (and female fitness, provided the effects of infanticide are large enough) to have as long a length of receptivity as possible, in order to be able to mate with the most males possible.

We also compared these results to the case when receptivity is assumed to be continuous (i.e. females are willing and able to mate on every day of their cycle). The continuous receptivity results are displayed as the white bars in Fig. 2.3, and the evolvable receptivity results as the black bars in Fig. 2.3. In general, introducing evolvable receptivity strongly decreases m^* and ℓ^* in the case of no infanticide, and increases m^* and slightly decreases ℓ^* in the case of with infanticide.

When not considering the effects of infanticide, the only benefit to a female in our model comes from the female mating with high-GC males. This means when continuous receptivity is fixed, the length of a female's visible ovulation signs should be no longer than her length of fertility (i.e. $\ell^* \leq 7$). However, with evolvable receptivity, females are now able to concentrate paternity by having a short length both of receptivity *and* of visible ovulation signs. Then considering the effects of infanticide, paternity confusion becomes important to females in addition to paternity concentration, leading to those longer lengths of visible ovulation signs (and matching receptivity lengths). In addition, since infanticide increases the stakes of the female-female competition, females are now able to devote more resources towards strong ovulation signs magnitude for those fewer

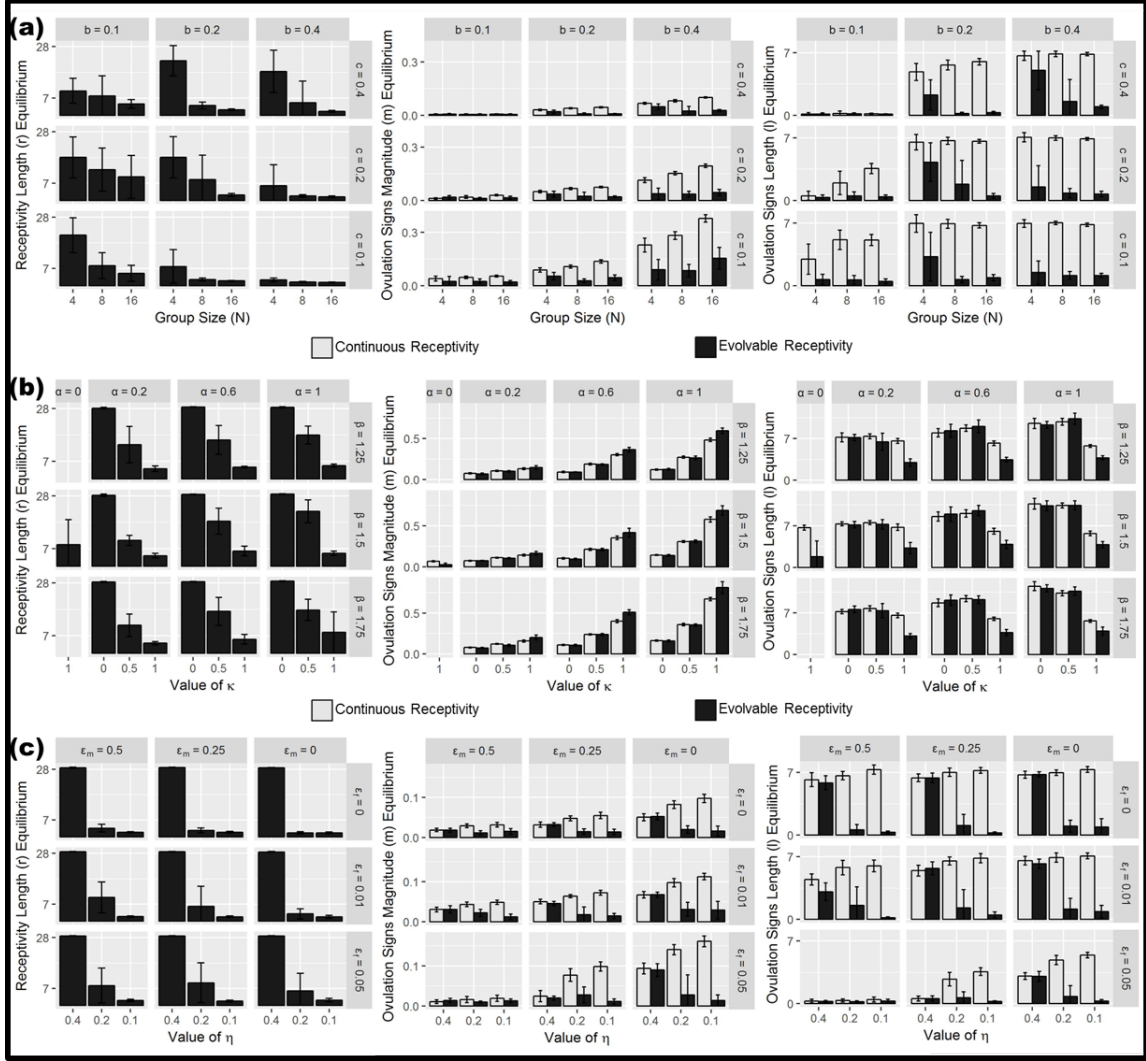


Figure 2.3: The effects of varying parameters on the average equilibria values of receptivity length (r^* , left graphs), ovulation signs magnitude (m^* , middle graphs), and ovulation signs length (l^* , right graphs), with both continuous receptivity assumed (white bars) and evolvable receptivity (black bars). Equilibria are obtained by averaging over 8 initial condition runs (with standard deviation indicated by error bars). (a) Varying N (group size), b (variation in male quality), and c (costs of having ovulation signs present) in the case of no infanticide ($\alpha = 0$, $c_r = 0.2$, $\epsilon_m = 0.25$, $\epsilon_f = 0.01$, $\eta = 0.2$). (b) Varying α (maximum benefit of infanticide protection), β (maximum cost of infanticide), and κ (weight males put on visible ovulation signs in estimating paternity) in the case of with infanticide ($N = 8$, $b = 0.2$, $c = 0.2$, $c_r = 0.2$, $\epsilon_m = 0.25$, $\epsilon_f = 0.01$, $\eta = 0.2$). (c) Varying ϵ_m (male reproductive stochasticity), ϵ_f (female reproductive stochasticity), and η (weighting of NGC vs. GC on female fitness) in the case of no infanticide ($\alpha = 0$, $N = 8$, $b = 0.2$, $c = 0.2$, $c_r = 0.2$).

days of their receptivity, the precise pattern we see in Fig. 2.3(b).

4.3.1. ANOVA Results

Finally, for both with and without infanticide in Case 3, we run an analysis of variance (ANOVA) to determine which of receptivity length r , ovulation magnitude m , and ovulation length ℓ are affected most by which parameters. Full results of these tests can be found in the Supplementary Information, but we do find that considering linear interactions only is sufficient. In general though, what we find in the without infanticide case, is that (intuitively) costs of receptivity c_r primarily affects r (negatively), while costs of having visible ovulation signs c mainly affects m and ℓ (negatively). Also intuitively, we find that η has the largest effect on r (positively). We also find that the benefit of male quality b strongly increases both m and ℓ and only slightly decreases r . Finally, group size N does not have much effect, but does decrease r . In general for the with infanticide case, we find that β has little effect (although what effect it does have is always non-negative), and α positively affects each of r, m, ℓ , although m, ℓ more so than r . κ strongly increases m and strongly decreases both ℓ and r . Note that all of these results reaffirm the patterns discussed above.

With regards to the reproductive stochasticity parameters ϵ_m and ϵ_f , we find no effects on receptivity length r . As in Chapter 1, increasing ϵ_m will result in decreased m^* (since increasing ϵ_m makes the ‘prize’ for females of winning the female-female competition less valuable) and increasing ϵ_f will result in decreased ℓ^* .

5. Discussion

It was our goal here to investigate primate sexual receptivity. In particular, we were interested in under what conditions were continuous receptivity and/or very short lengths of receptivity able to evolve. We were also interested in the effects infanticide could have on the evolution of receptivity, as well as how the evolution of sexual attractiveness (visual ovulation signs vs. concealed ovulation) could affect the evolution of receptivity.

From the results above, our model has produced four main results of interest in answering our research questions.

5.1. *Receptivity Length and Female Reproductive Success*

First, how does length of receptivity contribute to female reproductive success? Female reproductive success here can be thought of as a trade-off between paternity concentration and paternity confusion (van Schaik et al., 1999). A female concentrating paternity in high-GC males allows her offspring to have higher quality, and hence increases the female's fitness. Conversely, a female mating with multiple males during one cycle will confuse paternity, i.e. make multiple males think they may be the father of her subsequent offspring. Paternity confusion can also increase a female's fitness, for example by reducing her offspring's risk of infanticide.

Our modeling work shows this trade-off is manifested in females' length of receptivity. In situations where paternity concentration is favored, short lengths of receptivity are seen. Conversely, in situations where paternity confusion is favored (like strong effects of infanticide), longer lengths of receptivity are seen. Short lengths of receptivity allow females to only mate on days where they have strong visual ovulation signals, meaning on those days where they are able to attract the highest-GC mates. By only mating on those few days of peak signal, such females are able to concentrate paternity of their offspring in high-GC males. On the other hand, long lengths of receptivity give females ample time to mate with as many males as possible, thus allowing them to confuse paternity. It is in this way that receptivity is able to contribute to both paternity concentration and paternity confusion, and hence also female reproductive success.

Even in situations when paternity confusion is not beneficial, as long as paternity concentration is also not particularly beneficial, long lengths of receptivity can evolve. In particular, such long receptivity lengths evolve when η is larger. A large η means increased weight on NGC, i.e. non-genetic benefit. Hence, females are able to obtain such benefit throughout their cycle (not just when they are fertile), leading to longer lengths of receptivity when NGC is favored.

5.2. *Receptivity Length and Infanticide*

Second, what role could infanticide have in the evolution of receptivity? When infanticide is introduced, our model shows that receptivity increases as the effects of infanticide get stronger (i.e. larger α, β). Introducing the threat of infanticide increases the importance of paternity confusion, and as explained above, longer lengths of receptivity allow for more matings with multiple males,

meaning increased paternity confusion. Thus, increasing the effects of infanticide increases the equilibria length of receptivity.

Under conditions of infanticide, we also find that increasing the importance for males estimating their paternity of the female having signs present when mating leads to decreased receptivity length. Note this is equivalent to increasing the parameter κ , i.e. moving away from males estimating their paternity simply mating with a female. Increasing κ increases the weight males put on a female's ovulation signs when evaluating their estimated paternity. This means with increased κ , males are putting *more* weight on females having visual ovulation signs present, which means simply mating with more males may not benefit the female at all; it may only be of benefit to mate when she *does* have strong ovulation signs visible, leading to increasing κ decreasing equilibria receptivity length.

5.3. Continuous Receptivity

Third, what factors facilitate the evolution of continuous receptivity? Our model shows three general ways for continuous receptivity to evolve. The first way is the most general way, applicable to most any situation. As discussed earlier, as the value of η increases, meaning as the non-genetic benefit to females from mating increases, continuous receptivity can result. This means one way continuous receptivity can result is if genetic benefits from mating become less important, and females are able to obtain benefit throughout the length of their cycle. The second way only occurs under conditions of concealed ovulation without any threat of infanticide, while the third only under conditions of infanticide.

Without infanticide, the only benefit to females from mating is coming from the quality (genetic or otherwise) of the males with whom she mates. In particular, continuous receptivity is more likely to evolve under these conditions with smaller group sizes (N), greater variation in male quality (b), and lower costs of receptivity (c_r). The influence of c_r is quite intuitive (i.e. lower costs make its evolution more likely), while the influences of N and b have to do with female-female competition. This competition is typically determined by sexual attractiveness (i.e. amount of ovulation signs visible), but in the case of concealed ovulation, that is not possible. However, larger b still makes the 'prize' of winning such female-female competition more valuable, as does smaller N . Comparing $N = 4$ and $N = 16$, we would expect the gap between the quality of the best and second-best males

to be smaller with 16 males in a group, compared to only 4. Again, just as with larger b , smaller N is making that ‘prize’ of winning the female-female competition that much more valuable, leading to females entering the competition on every single day of their cycle, i.e. have continuous receptivity.

Alternatively, such female-female competition for high-quality males could also be manifested in terms of spite. Biologically, spite occurs when an individual is willing to incur a fitness cost in order to inhibit the reproductive success of a peer (Stockley and Bro-Jorgensen, 2011). An example of spite in terms of receptivity is a female incurring the fitness cost of being receptive for *more* time out of her cycle in order to monopolize a high-quality male on those days. There is a cost to the female of being receptive longer than she is fertile (assuming no other potential benefits, like from the threat of infanticide), but by monopolizing a high-quality male on her non-fertile days, she is able to keep a potentially-fertile peer from mating with that high-quality male on that particular day. This means that despite *her* fitness decreasing by being receptive longer, she might keep a fertile peer from increasing her fitness, meaning the original spiteful female may in fact have higher *relative* fitness as a result. Hence, under certain conditions, longer periods of female receptivity could have evolved out of spite.

In particular, the benefits of such spite are easier to obtain with a smaller group size N . For example, with $N = 4$, it is entirely possible for each of the 4 females to have non-overlapping days of high fertility. By non-fertile females entering the competition for mates, they would only need to ‘beat’ that one female with high-fertility in the female-female competition for mates. Conversely, with $N = 16$, beating in competition the likely multiple high-fertility females would be much tougher. Indeed, as stochasticity in reproductive pair formation increases, continuous receptivity evolving in this concealed ovulation case becomes more likely (see Supplemental Information). However, in general, ϵ_m and ϵ_f have little effect on female receptivity length.

Continuous receptivity can also evolve under conditions of infanticide, regardless of whether ovulation signs are visible or not. As explained earlier, κ is an important parameter influencing receptivity length. At its extreme, $\kappa = 0$, males are not putting *any* weight on a female’s visual ovulation signs; rather, the male when estimating his paternity of an offspring takes into account *only* the number of matings with its mother during that cycle. In this case, with $\kappa = 0$, a female is

able to confuse paternity throughout her entire length of receptivity, regardless of when, if, or how many visual ovulation signs she has present. Hence, when $\kappa = 0$, as expected we can see continuous receptivity evolve.

5.4. *Receptivity Length and Sexual Attractiveness*

Fourth, how is receptivity linked to the evolution of sexual attractiveness? Recall that in the case of fixed visible ovulation signs (i.e. with only r evolving, see Fig.2.2), we postulated visible ovulation signs to have a fixed length of 4 days. Other than the continuous receptivity cases discussed above, we do indeed see receptivity length equilibria around 4 days as well when there is infanticide present, and around 1-2 days when there is no infanticide. Hence with infanticide present, receptivity is evolving to equal the ovulation signs length. Conversely, without infanticide present, there is no benefit to females of paternity confusion, meaning it is now in females' best interests to concentrate paternity by only mating for a very short portion of their cycle (i.e. those 1-2 days).

Next considering the case with each of m , ℓ , and r evolving, we see similar patterns. With fixed continuous receptivity and sufficient male quality (i.e. high-enough quality males worth competing over), females will typically have length of ovulation signs visible matching their length of fertility (i.e. 7 days in Fig. 2.3). Allowing variable receptivity will shorten these ovulation signs lengths (e.g. to about 2-3 days in the case without infanticide), with ovulation signs length equilibria approximately matching the evolved receptivity lengths equilibria.

Thus, in general, we do not see receptivity length equilibria longer than a female's fertility length. This makes sense since outside of the continuous receptivity cases discussed earlier, with a cost (c_r) associated with being receptive, females should typically only be receptive during the portion of their cycle where they are actually able to conceive. In turn, we also typically see a female's ovulation signs length equilibria match her receptivity length equilibria, meaning both receptivity and a female's length of ovulation signs being visible will typically not be longer than her length of fertility. Such continuity between receptivity length and ovulation signs length is intuitive, since again, if females are spending energy on being receptive, they should also only be spending energy on advertising their fertility when they are indeed receptive to mating.

Finally, there also appears to be a trade-off between receptivity length and ovulation signs magnitude (see Fig. 2.3). When each of receptivity (r), ovulation signs magnitude (m), and ovulation signs length (ℓ) are allowed to evolve, we find that with long lengths of receptivity (i.e. at or near being continuously receptive), ovulation signs magnitude (m) is very small. This is suggestive of the possible relationship between concealed ovulation and continuous receptivity.

5.5. Conclusion

Thus, while much work still needs to go into understanding the evolution of sexual receptivity in female primates, our work takes a useful step in this research, by introducing the first theoretical work completed on the subject. In particular, our work suggests several pathways by which longer receptivity could have evolved or diminished. While it was our hope to test our model's predictions on the limited data available on female primate sexual receptivity length (e.g. see (Hrdy and Whitten, 1987; van Schaik et al., 1999; Stockley, 2002)), such data turned out to be too sparse, especially when only considering polygynandrous mating systems as does our model, and also at times variable between sources. In addition, there can be intraspecific variation in receptivity length as well, which our model does not specifically address (for example, receptivity lengthening when a new male takes over a group) (van Schaik et al., 1999). More empirical data would be needed in order to do an effective comparative empirical analysis. Testing the general insights from our model empirically is still our hope, although we recognize further detailed information on fitness components and ecological factors would likely have to be obtained empirically first.

6. Acknowledgments

We thank Susan Perry and Sarah Hrdy for comments and suggestions. K.R. was supported by a National Science Foundation Graduate Research Fellowship. S.G. was supported by the National Institute for Mathematical and Biological Synthesis through National Science Foundation awards EF-0832858 and DBI-1300426, with additional support from The University of Tennessee. All numerical simulations were run on the Volos cluster, which is supported by National Institutes of Health award GM56693 (S.G.).

7. Supplementary Information: Analytical Results

In addition to the numerical results obtained, some analytical results were also possible, albeit with more limiting assumptions. These analytical results hold for the evolution of female sexual receptivity in the case where concealed ovulation is fixed for every female in the population, there is no selection pressure due to infanticide, there are no perturbations in either male or female quality, and there are no non-genetic benefits of mating. In terms of comparison to the numerical work, this means assumptions include $m = 0, \ell = 0, \alpha = 0, \epsilon_m = 0, \epsilon_f = 0$, and $\eta = 0$.

7.1. Analytical Model Set-up

Let $r \in [0, 1]$ be the proportion of the cycle in which a female is *receptive* to mating. Biologically, this still means the proportion of her cycle when approached by a male to mate, she will. We still assume all females' fertile and receptive periods to line up over their times of ovulation, meaning the receptive period must always be covered by the fertile period and/or the fertile period by the receptive period, although not necessarily equal.

Assume there are N females in each group. Let $b > 0$ be the maximum benefit of good genes coming from mating with the 'best' male. We assume male quality comes from the uniform distribution on $[0, b]$. Let $c_r r \geq 0$ be the total cost to a female of being receptive for proportion r of her cycle. Finally, let all females have receptivity trait equal to r , except for one mutant ("focal") female with receptivity trait equal to $\tilde{r}$.

7.2. Constructing the Invasion Female Fitness Function

In the case of concealed ovulation, our female fitness function will be:

$$w_F(r) = w_0 + y_m - c_r r, \quad (21)$$

where y_m is the male quality of a female's actual mate. Hence we need to find a way to approximate this y_m .

From our one focal female's perspective, we can approximate y_m using:

$$y_m \approx \sum_{k=1}^N [\mathbb{P}(1 \text{ of } k \text{ females receptive}) \cdot \mathbb{E}(\text{benefit from good genes given } k \text{ females receptive})], \quad (22)$$

where the first term means the probability that our focal female is exactly 1 of k females receptive, which can range anywhere from $k = 1$ (meaning she is the *only* female receptive) to $k = N$ (meaning *every* female is receptive).

Then for any $1 \leq k \leq N$, the probability of our focal female being 1 of k total receptive females in her group is given by:

$$\mathbb{P}(1 \text{ of } k \text{ females receptive}) = \tilde{r} \binom{N-1}{k-1} r^{k-1} (1-r)^{N-k}, \quad (23)$$

meaning the probability that the focal female is receptive times the probability of there being exactly $k - 1$ other receptive females out of the $N - 1$ other females in her group.

Note all females are considered equal in “quality” here because we are assuming concealed ovulation for all females, meaning males can only mate with receptive females but have no preference between any of the receptive females. Then letting Y_i be the i^{th} order statistic of the uniform distribution on $[0, b]$, we have:

$$\mathbb{E}(\text{benefit from good genes given } k \text{ females receptive}) = \frac{b}{k} \sum_{i=N-k+1}^N \mathbb{E}(Y_i) \quad (24)$$

$$= \frac{b}{k} \sum_{i=N-k+1}^N \left(\frac{i}{N+1} \right) \quad (25)$$

$$= \frac{b}{k(N+1)} \left[k \left(\frac{N + N - k + 1}{2} \right) \right] \quad (26)$$

$$= \frac{b}{N+1} \left(N + \frac{1-k}{2} \right). \quad (27)$$

Thus, plugging Eqs. 3 and 7 into Eq. 2, we obtain:

$$y_m \approx \sum_{k=1}^N \left[\left(\tilde{r} \binom{N-1}{k-1} r^{k-1} (1-r)^{N-k} \right) \left(\frac{b}{N+1} \left(N + \frac{1-k}{2} \right) \right) \right] \quad (28)$$

$$= \frac{b\tilde{r}}{N+1} \sum_{k=1}^N \left[\left(\binom{N-1}{k-1} r^{k-1} (1-r)^{N-k} \right) \left(N + \frac{1-k}{2} \right) \right] \quad (29)$$

$$= \frac{b\tilde{r}}{N+1} \left(\frac{1}{2} (r - N(r-2)) \right) \quad (30)$$

$$= \frac{b\tilde{r}}{2(N+1)} (r - N(r-2)). \quad (31)$$

7.2.1. Invasion Female Fitness

Hence we now have the female invasion fitness function:

$$W_F(\tilde{r}|r) = w_0 + \frac{b\tilde{r}(r - N(r - 2))}{2(N + 1)} - c_r\tilde{r}. \quad (32)$$

7.2.2. Invasion Fitness Gradient

We can now take a partial derivative to get the invasion fitness gradient:

$$\frac{\partial W_F(\tilde{r}|r)}{\partial \tilde{r}} = \frac{b(r - N(r - 2))}{2(N + 1)} - c_r. \quad (33)$$

7.2.3. Equilibria

We can then solve this for r^* and obtain a closed-form equation for any equilibria:

$$r^* = \frac{2(bN - c_r(N + 1))}{b(N - 1)}. \quad (34)$$

7.3. Analytical Results

Note there are exactly three parameters in the above r^* equilibria equation: group size (N), benefit of good genes (b), and cost of receptivity (c_r). See Fig. 2.4 for a depiction of these equilibria values with varying b (x-axis), c_r (y-axis), and N (graphs). Only values satisfying $0 \leq r^* \leq 1$ are plotted in color here, with all other values displayed as white space. Darker shades of red correspond to values closer to $r^* = 0$, and lighter shades of yellow to $r^* = 1$.

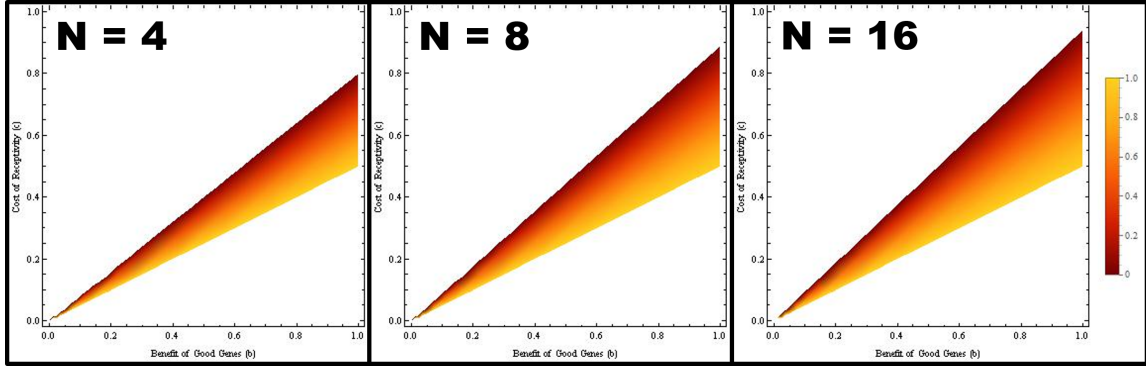


Figure 2.4: Graphs of equilibria r^* (given by Eq. 34) for the above analytical model. Each graph corresponds to a different group size (N), while each of the three graphs displays different values for benefit of good genes (b , x-axis) and costs of receptivity (c_r , y-axis). Only equilibria values satisfying $0 \leq r^* \leq 1$ are displayed in color here.

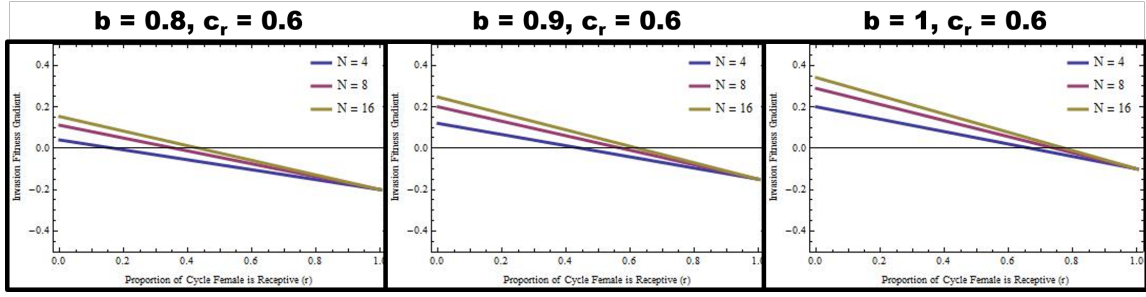


Figure 2.5: Graphs of invasion fitness gradients for the above analytical model. Each graph varies group size (N), while each of the three graphs gives different values of benefit of good genes (b) while keeping costs of receptivity (c_r) constant. Note r^* (x-intercepts) increase with increasing b and increasing N .

In general, we can see that the equilibria value r^* is predicted to increase with *increasing* benefit of good genes (b) and *decreasing* cost of receptivity (c_r), both intuitive results. We also see here that the equilibria value r^* is predicted to increase with *increasing* group size (N). Finally, note that all values of receptivity *are* possible here as equilibria, ranging from no receptivity ($r^* = 0$) to continuous receptivity ($r^* = 1$) for each group size.

Fig. 2.5 shows more explicitly the effects of benefit of good genes (b) on the resulting r^* equilibria (i.e. where the invasion fitness gradient is equal to zero), and Fig. 2.6 similarly the effects of costs of receptivity (c_r). Note we have increased r^* for increased b , decreased c_r , and increased N . Finally, Fig. 2.7 looks at what happens in the ‘white space’ of Fig. 2.4. The left graph of Fig. 2.7 explores an example from the lower white space ($b = 0.95, c_r = 0.4$) and the right graph from the upper white space ($b = 0.95, c_r = 0.95$). In the left graph, we find continuous receptivity and in the right graph, no receptivity at all.

7.4. Comparison to Numerical Results

Comparing these analytical results to the numerical results presented in the main text, we see that they do in fact line up. Recall we are comparing the cases when concealed ovulation is fixed ($m, \ell = 0$), only receptivity r is evolving, there is no infanticide ($\alpha = 0$), and there is no reproductive stochasticity ($\epsilon_m, \epsilon_f = 0$).

From our numerical work in such a case, we know the receptivity equilibria is expected to increase with (i) increasing group size (N), (ii) increasing male benefit (b), and (iii) decreasing

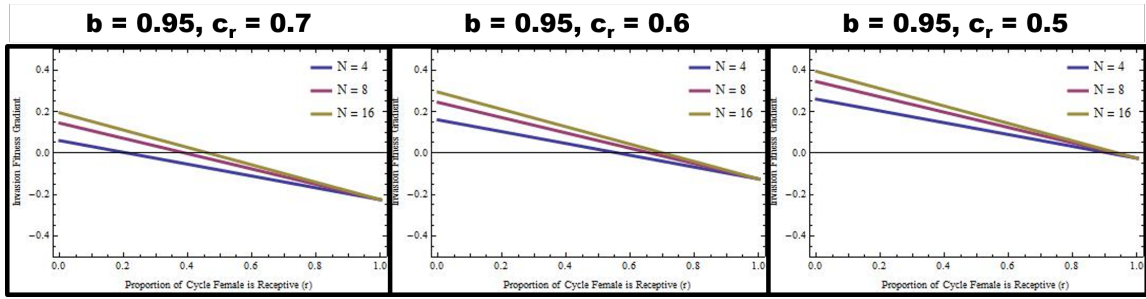


Figure 2.6: Graphs of invasion fitness gradients for the above analytical model. Each graph varies group size (N), while each of the three graphs gives different values of costs of receptivity (c_r), while keeping benefits of good genes (b) constant. Note r^* (x-intercepts) increase with decreasing c_r and increasing N .

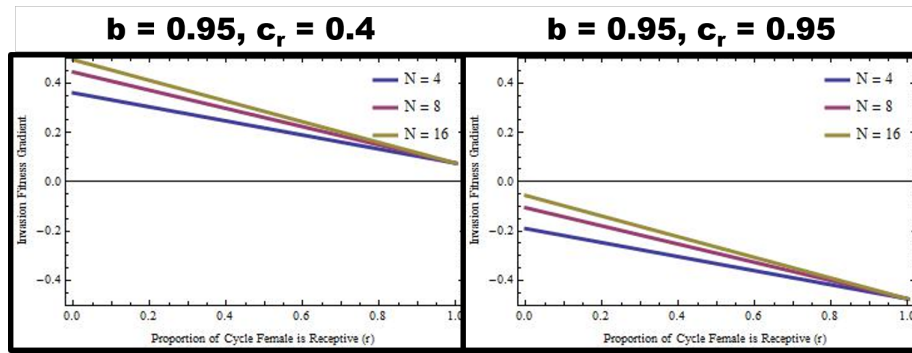


Figure 2.7: Graphs of invasion fitness gradients for the above analytical model. Each graph varies group size (N), while each of the two graphs gives different values of benefit of good genes (b) and costs of receptivity (c_r). In the left graph, we see continuous receptivity (i.e. $r^* = 1$) and in the left graph, no receptivity ($r^* = 0$).

costs of receptivity (c_r). Note such patterns are identical to those predicted by our numerical simulations, providing some validation for our numerical model.

8. Supplementary Information: Numerical Results

8.1. Further Details of the Model

8.1.1. The Female Cycle

Female estrous cycles are divided into D discrete units of time (e.g. $D = 29$ days) with $C \leq D$ days of fertility (e.g. $C = 7$). Although the form of such fertile periods will be identical in all females, the timing of each fertile period will be randomly distributed among females. For instance, when $C = 7$ (calling each of these fertile days $C1, C2, \dots, C7$) and $D = 29$ (calling each of these days $D1, D2, \dots, D29$), $C1$ in every female has an equal probability of landing on any of $D1, D2, \dots, D29$. Since C is a cycle, we similarly assume $C7$ can land on any such day. For example, if $C1$ landed on $D24$, then $C7$ would ‘wrap around’ the cycle to land on $D1$. Moreover, for all females we assume ovulation happens directly in the middle of this C cycle. Hence, for $C = 7$ we assume ovulation happens on $C4$ and the probabilities of fertilization increase from $C1$ to $C4$ and then decrease from $C4$ to $C7$.

Each female i will have $r_i \leq D$ days of receptivity. These days of receptivity line up with the female’s days of fertility, such that a female’s median day of fertility will equal her median day of receptivity. Similarly, since having visible ovulation signs at least loosely correlates with a female’s fertility, we assume the day(s) of having maximum visual ovulation signs also align with the day(s) of that female’s maximum fertilization probability, and align these cycles accordingly (note in most cases, $C \neq r_i \neq \ell_i$).

Recall that $x_i(d)$ denotes the amount of visual ovulation signs present on each day d of the female cycle, with $x_i(d)$ defined by the function in the Methods section of the main text. This function is determined by m_i (ovulation magnitude) and ℓ_i (ovulation length). Note the exact function $x_i(d)$ is chosen such that (i) m_i will denote the peak of the curve, (ii) ℓ_i will denote the width of the non-zero portion of the curve, (iii) m_i, ℓ_i remain independent of each other, and (iv) m_i, ℓ_i can both decrease all the way to zero. Example values of r_i are displayed in Fig. 2.8 via the

light blue shading, for $m_i = 1$, $\ell_i = 7$, and $C = 7$. Ovulation signs on each day are depicted by the red shaded bars, while fertility probabilities are given by the black lines.

8.1.2. Determining Male-Female Mate Pairs

We assume that, for every day of the cycle, males of higher GC will preferentially mate with females with more ovulation signs visible. We also assume each individual to mate at most once on each day of the cycle. Females will only mate on days on which they are receptive, and males will only mate if there is a receptive female with whom to mate. Parameters ϵ_m and ϵ_f control the amount of stochasticity in the process of mating pair formation. With $\epsilon_m, \epsilon_f \rightarrow \infty$, matings are determined completely randomly.

8.1.3. Calculating Probabilities of Paternity

To calculate the probability of paternity for each male, first the fertility of each female with which that male has mated is summed up for every day a particular male mates with her. These values are normalized across males for each female in the group, meaning a male who mates with the female on a day where she has a higher fertility probability will have a higher paternity probability. The actual father for that female's offspring is then determined randomly, proportional to each male's probability of paternity for her offspring.

Note these *actual* probabilities of paternity are different from the $p_{i,j}$, *perceived* probabilities of paternity, as detailed in the Methods section of the main text. We make this distinction because males will not know any female's probability of fertility at the time he mates with her; males will only know how many visible ovulation signs she had present. Separating these quantities allows actual probabilities of paternity (which males do not know) to be calculated using females' probabilities of fertility, and perceived probabilities of paternity (which males *do* know) to be calculated using females' visible ovulation signs.

8.1.4. Infanticide

We define the exponential function of male effectiveness to be:

$$f(j) = \frac{e^{-\omega j}}{\sum_{k=1}^N e^{-\omega k}}, \quad (35)$$

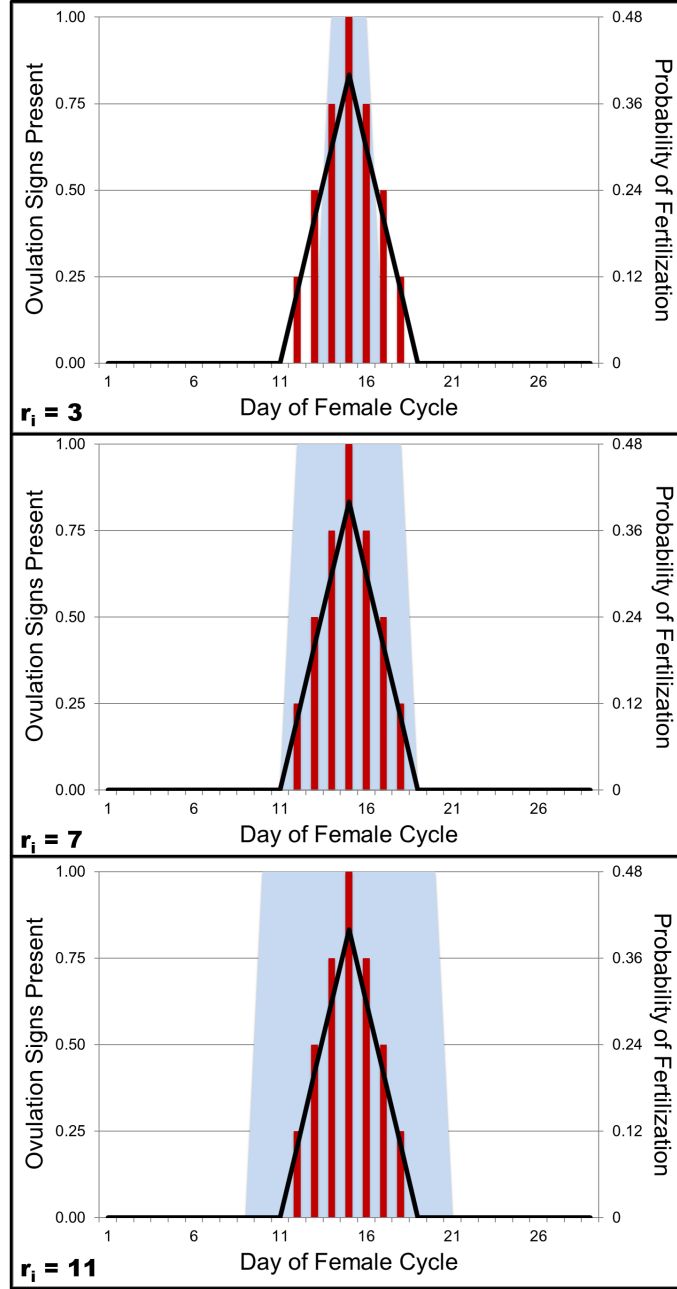


Figure 2.8: Sample receptivity lengths are displayed for $r_i = 3, 7, 11$ (from top to bottom) via the light blue shading. Each graph also includes ovulation signs present (with $m_i = 1, \ell_i = 7$) via the red bars and fertilization probabilities (with $C = 7$) via the black lines. In the top graph, we see that a female can be fertile and/or have ovulation signs visible on days where she is not receptive. In the bottom graph, we instead see a female can be receptive on days where she does not have any ovulation signs visible and/or is not fertile. The middle graph depicts the situation where visible ovulation signs, fertility, and receptivity all line up with lengths of 7 days.

with parameter $\omega > 0$ controlling the amount of disparity among males in their corresponding effectiveness within a group. Recall a larger value of ω indicates more disparity among males.

8.1.5. Female Fitness Functions

The female fitness function for female i in the model is as follows:

$$w_i = w_0 + (1 - \eta)y_{m,i} + \frac{\eta}{D} \sum_{\text{all mates } j} y_{p,j} + \sum_{j=1}^N [f(j)g(p_{i,j})] - c\bar{x}_i - c_r \frac{r_i}{D}, \quad (36)$$

where recall $y_{m,i}$ is the actual benefit from female-female competition (i.e. the GC of the specific male who won the contest of paternity for that particular female's offspring), $p_{i,j}$ perceived paternity probability, $\bar{x}_i = \frac{1}{D} \sum_{d=1}^D x_i(d)$ the female's average visual ovulation signs present, η the weighting on female fitness between GC and NGC, and $y_{p,j}$ the NGC coming from each male j a female mated with throughout her cycle.

Notice for the case when $\alpha = 0$ (i.e. without the effects of infanticide), the fitness function above collapses into:

$$w_i = w_0 + (1 - \eta)y_{m,i} + \frac{\eta}{D} \sum_{\text{all mates } j} y_{p,j} - c\bar{x}_i - c_r \frac{r_i}{D}. \quad (37)$$

Note in this $\alpha = 0$ (no infanticide) case, parameters β , κ , τ , and ω all become unnecessary.

8.2. List of All Parameters

Parameters used in the model are outlined in Table 2.1. For each parameter, one value listed implies the parameter remained fixed throughout all simulations, and multiple values indicates each of those parameter values was tested in all combinations with all other parameters.

Table 2.1: List of All Parameters

Parameter Name	Parameter Description
$G = 400$	Number of groups
$T = 100000$	Time (number of generations)
$D = 29$	Number of days in each female's cycle
$C = 7$	Number of days each female has non-zero probability of fertilization
$C_v = \{0.125, 0.25, 0.375, 0.5, 0.375, 0.25, 0.125\}$	Probability of fertilization on each of those $C = 7$ days
$w_0 = 2$	Baseline female fitness
$N = 4, 8, 16$	Number of males and number of females in each group
$b = 0.1, 0.2, 0.4$	Variation in male quality
$c = 0.1, 0.2, 0.4$	Cost of having ovulation signs present
$c_r = 0.1, 0.2, 0.4$	Cost of being receptive to mating
$\eta = 0.1, 0.2, 0.4$	Weighting of GC vs. NGC
$\hat{g} = 1$	Mean male genetic quality
$\epsilon_m = 0, 0.25, 0.5$	Parameter determining the amount of reproductive stochasticity among males
$\epsilon_f = 0, 0.01, 0.05$	Parameter determining the amount of reproductive stochasticity among females
$\gamma = 1$	Parameter determining the shape of the function for the amount of visual ovulation signs present
$\alpha = 0, 0.2, 0.6, 1$	Parameter controlling the maximum benefit of protecting against infanticide (note $\alpha = 0$ corresponds to the case with NO infanticide)
$\beta = 1.25, 1.5, 1.75$	Parameter controlling the relative maximum cost of infanticide in the paternity probability effect function
$\tau = 2$	Exponent in the paternity probability effect function g
$\omega = 1$	Parameter in the male effectiveness function $f(j)$
$\kappa = 0, 0.5, 1$	Parameter determining the weight males put on the amount of visual ovulation signs in their paternity estimate
$\nu = 1$	Migration rate (between zero and one)
$\mu = 0.001$	Probability of mutation (per gene per generation)

8.3. Effects of All Parameters

See Table 2.2 for a summary of effects for all main parameters used in the model. These effects are all illustrated for simulations with concealed ovulation fixed (Figures 2.9,2.10,2.11), visible ovulation signs fixed (Figures 2.12,2.13,2.14), and each of receptivity length, ovulation signs magnitude, and ovulation signs length evolving (Figures 2.15,2.16,2.17,2.18,2.19,2.20,2.21,2.22,2.23).

Table 2.2: Effects of All Parameters

Traits Evolving:	Found in Figure:	Trait Investigated:	Parameters Investigated:
Only receptivity r , with concealed ovulation fixed ($m = 0, \ell = 0$)	2.9	Receptivity r	N, b, c_r, η
	2.10	Receptivity r	$\alpha, \beta, \kappa, \eta$
	2.11	Receptivity r	$\epsilon_m, \epsilon_f, c_r, \eta$
Only receptivity r , with fixed visual ovulation signs ($m = 0.9, \ell = 4$)	2.12	Receptivity r	N, b, c_r, η
	2.13	Receptivity r	$\alpha, \beta, \kappa, \eta$
	2.14	Receptivity r	$\epsilon_m, \epsilon_f, c_r, \eta$
Receptivity length r , ovulation signs magnitude m , and ovulation signs length ℓ	2.15	Receptivity r	N, b, c, c_r, η
	2.16	Ovulation signs magnitude m	N, b, c, c_r, η
	2.17	Ovulation signs length ℓ	N, b, c, c_r, η
	2.18	Receptivity r	$\alpha, \beta, \kappa, \eta$
	2.19	Ovulation signs magnitude m	$\alpha, \beta, \kappa, \eta$
	2.20	Ovulation signs length ℓ	$\alpha, \beta, \kappa, \eta$
	2.21	Receptivity r	$\epsilon_m, \epsilon_f, c_r, \eta$
	2.22	Ovulation signs magnitude m	$\epsilon_m, \epsilon_f, c_r, \eta$
	2.23	Ovulation signs length ℓ	$\epsilon_m, \epsilon_f, c_r, \eta$

8.3.1. ANOVA Results

The results of our ANOVA tests were discussed in the main text. The following tables (Tables 2.3, 2.4, 2.5) give the detailed results of these tests.

Table 2.3: Effects of N, b, c_r, c, η on receptivity length R and ovulation signs magnitude M and length L

	R	M	L
N	-0.011	0.012	0
b	-0.008	0.309	0.133
c_r	-0.472	-0.112	-0.292
c	0	-0.178	-0.021
η	0.256	0	0.083
error	0.252	0.383	0.470

Table 2.4: Effects of $\alpha, \beta, \kappa, \eta$ on receptivity length R and ovulation signs magnitude M and length L

	R	M	L
α	0.023	0.343	0.214
β	0	0.0195	0
κ	-0.296	0.401	-0.438
η	0.460	-0.015	0.079
error	0.221	0.222	0.261

Table 2.5: Effects of $\epsilon_m, \epsilon_f, c_r, \eta$ on receptivity length R and ovulation signs magnitude M and length L

	R	M	L
ϵ_m	0	-0.181	0
ϵ_f	0	0	-0.110
c_r	-0.517	-0.284	-0.390
η	0.220	0	0.081
error	0.257	0.512	0.399

8.3.2. Supplemental Figures

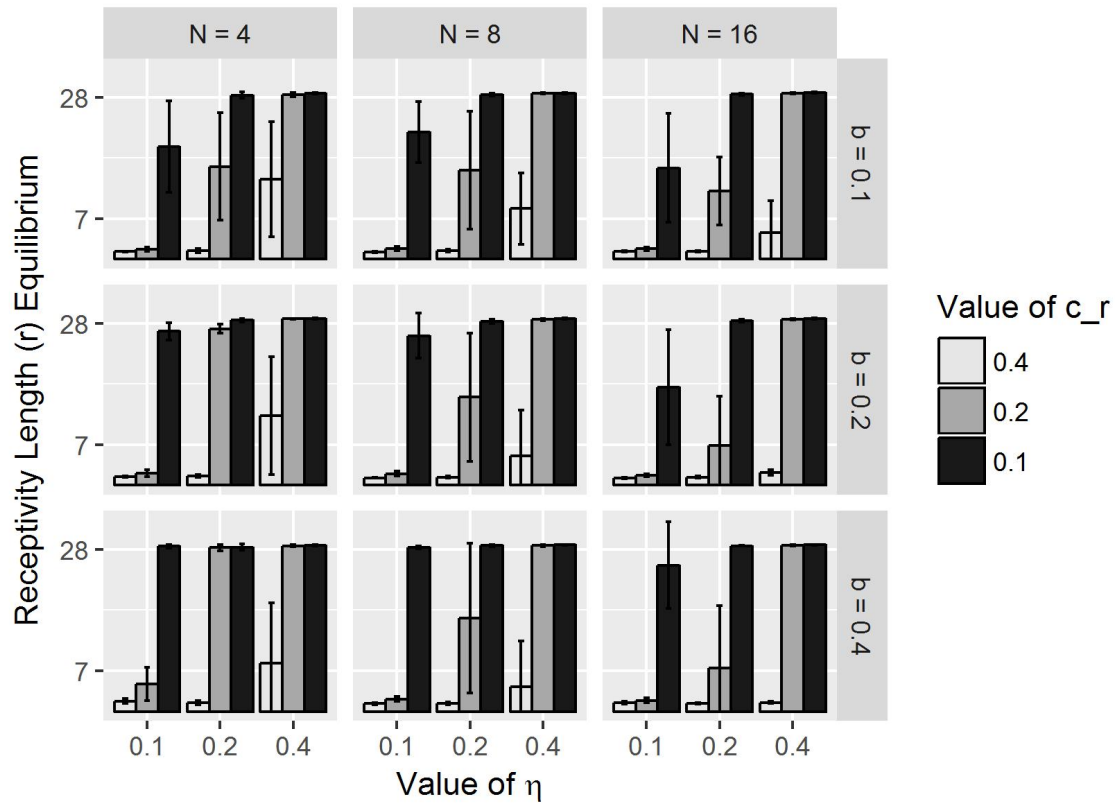


Figure 2.9: The effects of parameters N (group size), b (benefit of male genetic quality), c_r (cost of receptivity length), and η (weight put on genetic vs. non-genetic male benefits) on receptivity length (r), when concealed ovulation is fixed ($m = 0, \ell = 0$ for all females). Equilibria are obtained by averaging over 10 initial condition runs (with standard deviation indicated by error bars). Notice receptivity length becomes longer with decreasing N , increasing b , decreasing c_r , and increasing η . All other parameters were held constant: $\alpha = 0, c = 0.2, \epsilon_m = 0.25, \epsilon_f = 0.01$.

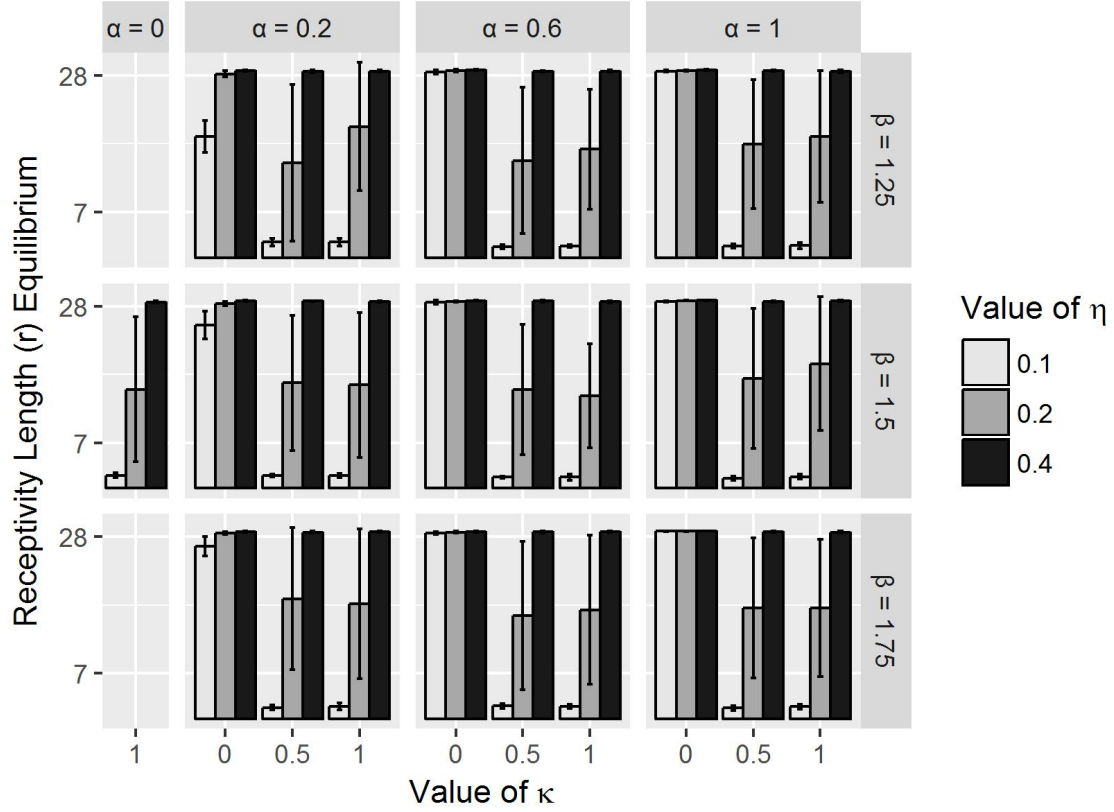


Figure 2.10: The effects of parameters α (maximum benefit of protecting offspring from infanticide), β (maximum cost of not protecting offspring from infanticide), κ (weight males put on visible ovulation signs when estimating their paternity), and η (weight put on genetic vs. non-genetic male benefits) on receptivity length (r), when concealed ovulation is fixed ($m = 0, \ell = 0$ for all females). Equilibria are obtained by averaging over 10 initial condition runs (with standard deviation indicated by error bars). Notice receptivity length becomes longer with increasing α , increasing β , decreasing κ , and increasing η . All other parameters were held constant: $N = 8, b = 0.2, c = 0.2, c_r = 0.2, \epsilon_m = 0.25, \epsilon_f = 0.01$.

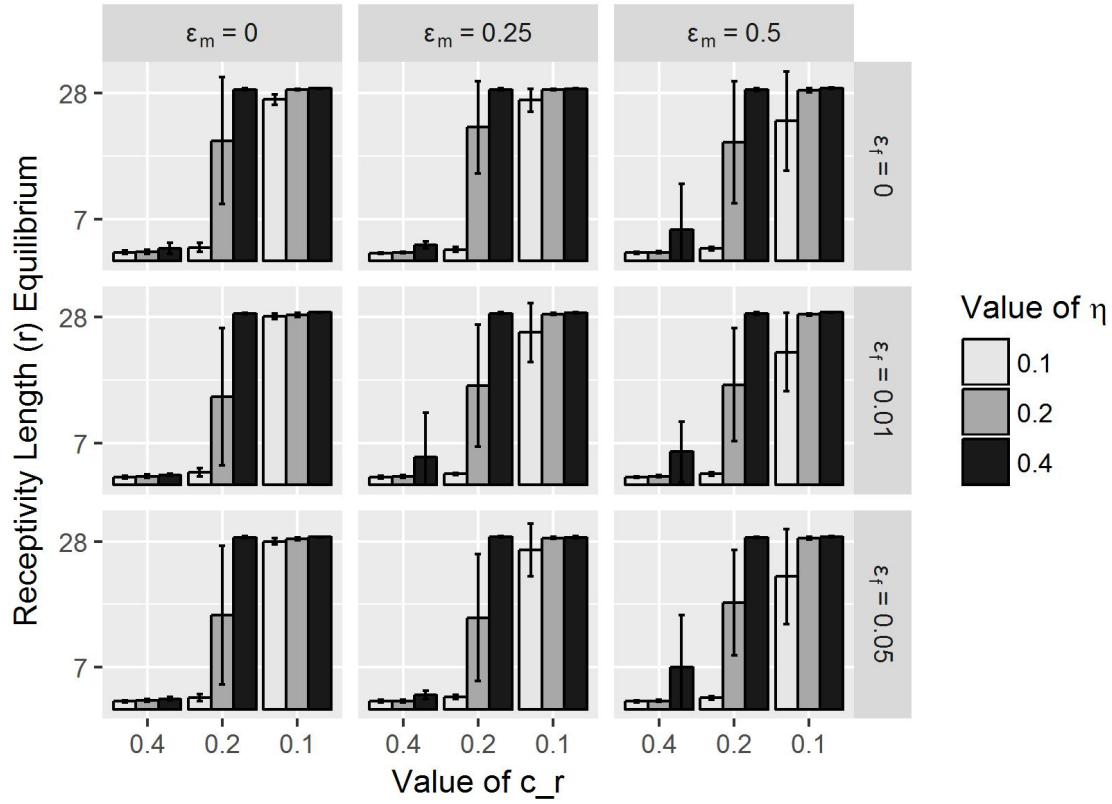


Figure 2.11: The effects of parameters ϵ_m (male reproductive stochasticity), ϵ_f (female reproductive stochasticity), c_r (cost of receptivity length), and η (weight put on genetic vs. non-genetic male benefits) on receptivity length (r), when concealed ovulation is fixed ($m = 0, \ell = 0$ for all females). Equilibria are obtained by averaging over 10 initial condition runs (with standard deviation indicated by error bars). Notice receptivity length becomes longer with decreasing ϵ_m , decreasing ϵ_f , decreasing c_r , and increasing η . All other parameters were held constant: $\alpha = 0, N = 8, b = 0.2, c = 0.2$.

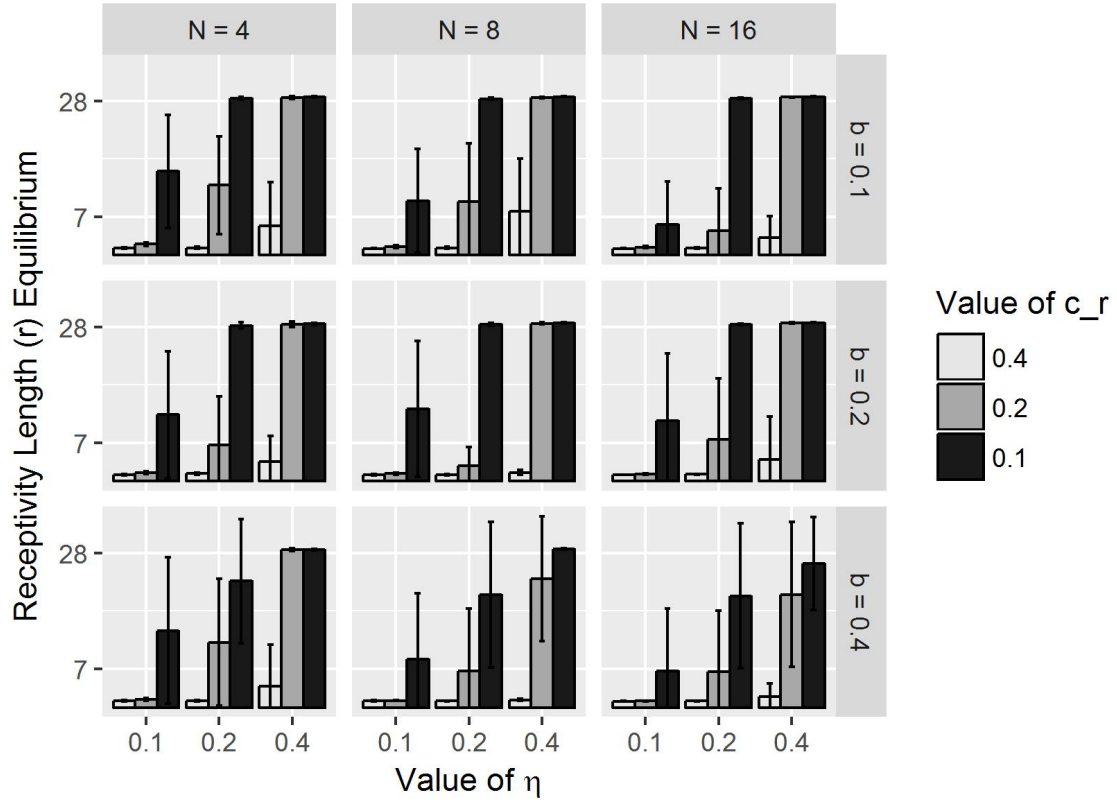


Figure 2.12: The effects of parameters N (group size), b (benefit of male genetic quality), c_r (cost of receptivity length), and η (weight put on genetic vs. non-genetic male benefits) on receptivity length (r), when visible ovulation signs are fixed ($m = 0.9, \ell = 4$ for all females). Equilibria are obtained by averaging over 10 initial condition runs (with standard deviation indicated by error bars). Notice receptivity length becomes longer with decreasing N , increasing b , decreasing c_r , and increasing η . All other parameters were held constant: $\alpha = 0, c = 0.2, \epsilon_m = 0.25, \epsilon_f = 0.01$.

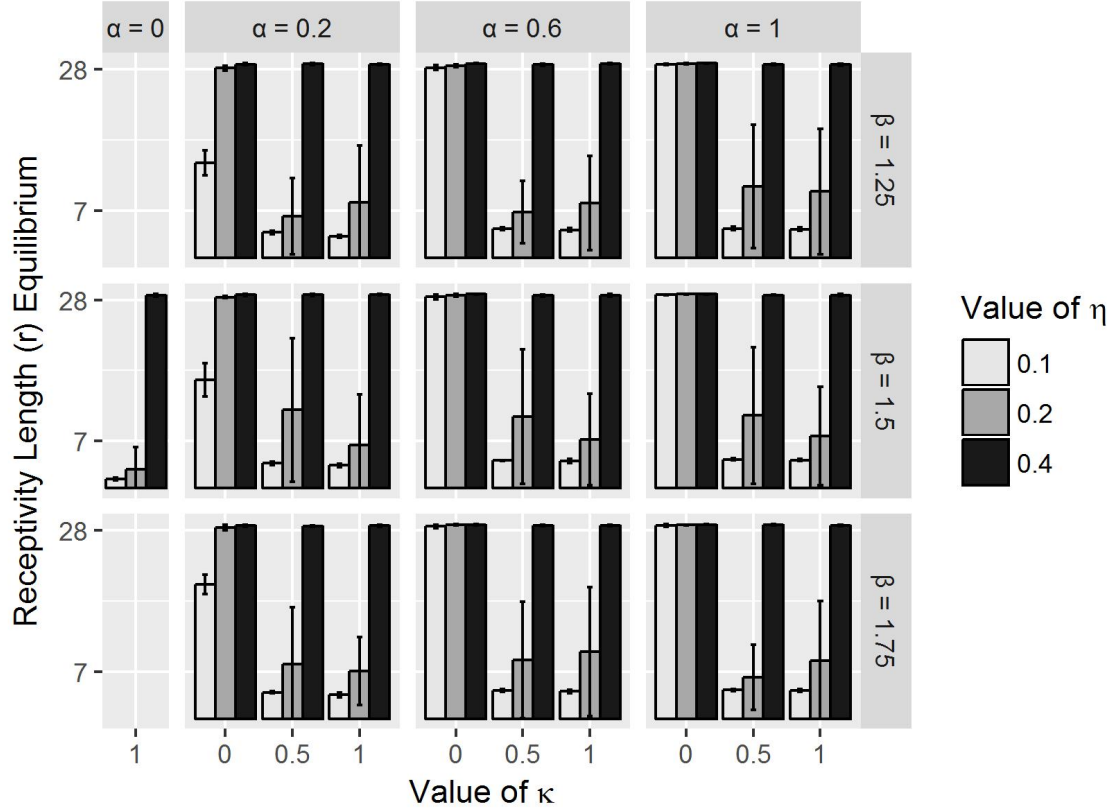


Figure 2.13: The effects of parameters α (maximum benefit of protecting offspring from infanticide), β (maximum cost of not protecting offspring from infanticide), κ (weight males put on visible ovulation signs when estimating their paternity), and η (weight put on genetic vs. non-genetic male benefits) on receptivity length (r), when visible ovulation signs are fixed ($m = 0.9, \ell = 4$ for all females). Equilibria are obtained by averaging over 10 initial condition runs (with standard deviation indicated by error bars). Notice receptivity length becomes longer with increasing α , increasing β , decreasing κ , and increasing η . All other parameters were held constant: $N = 8, b = 0.2, c = 0.2, c_r = 0.2, \epsilon_m = 0.25, \epsilon_f = 0.01$.

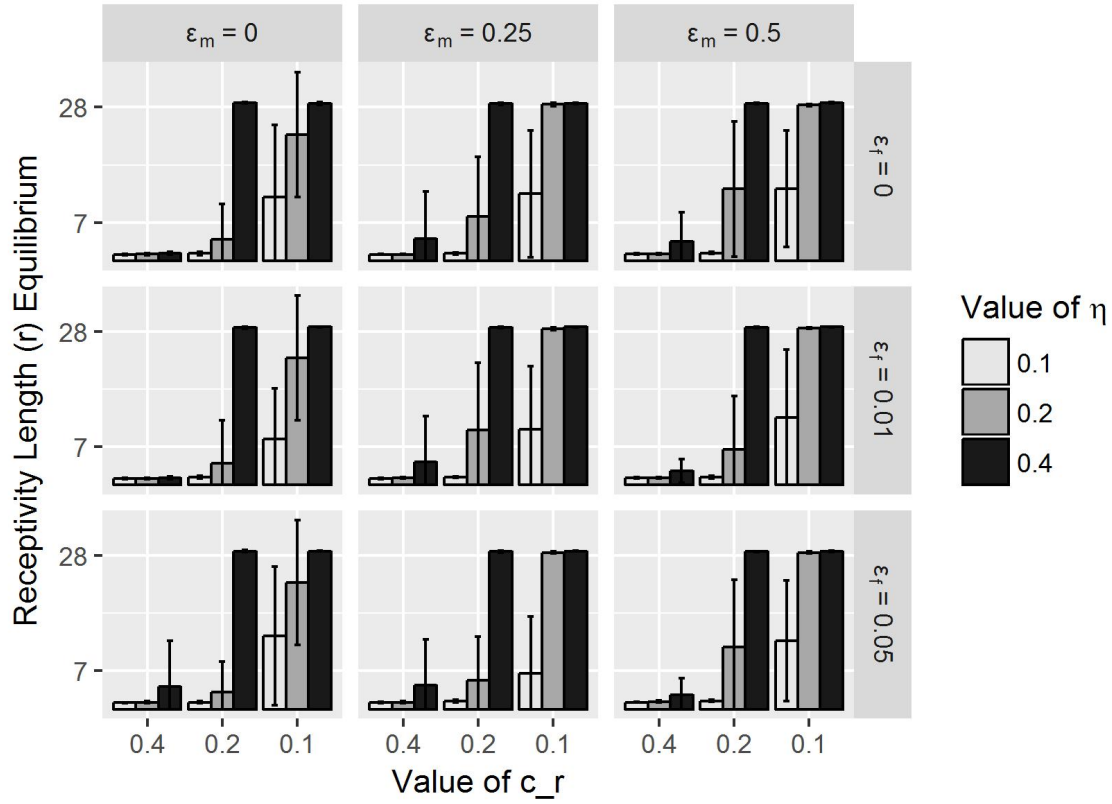


Figure 2.14: The effects of parameters ϵ_m (male reproductive stochasticity), ϵ_f (female reproductive stochasticity), c_r (cost of receptivity length), and η (weight put on genetic vs. non-genetic male benefits) on receptivity length (r), when visible ovulation signs are fixed ($m = 0.9, \ell = 4$ for all females). Equilibria are obtained by averaging over 10 initial condition runs (with standard deviation indicated by error bars). Notice receptivity length becomes longer with decreasing ϵ_m , decreasing ϵ_f , decreasing c_r , and increasing η . All other parameters were held constant: $\alpha = 0, N = 8, b = 0.2, c = 0.2$.

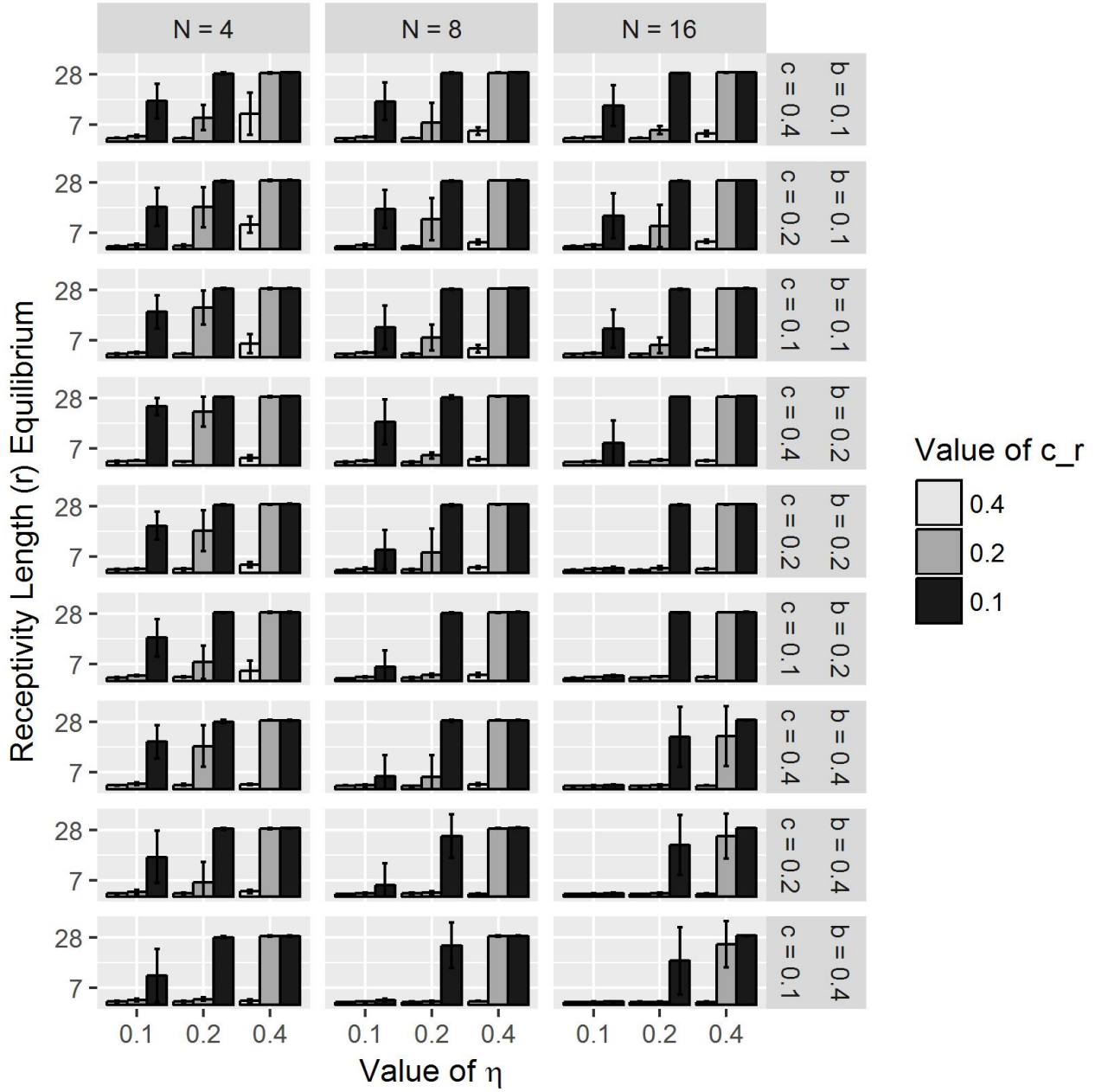


Figure 2.15: The effects of parameters N (group size), b (benefit of male genetic quality), c (cost of visible ovulation signs), c_r (cost of receptivity length), and η (weight put on genetic vs. non-genetic male benefits) on receptivity length (r). Equilibria are obtained by averaging over 27 initial condition runs (with standard deviation indicated by error bars). Notice receptivity length becomes longer with increasing N , increasing b , decreasing c , decreasing c_r , and increasing η . All other parameters were held constant: $\alpha = 0$, $\epsilon_m = 0.25$, $\epsilon_f = 0.01$.

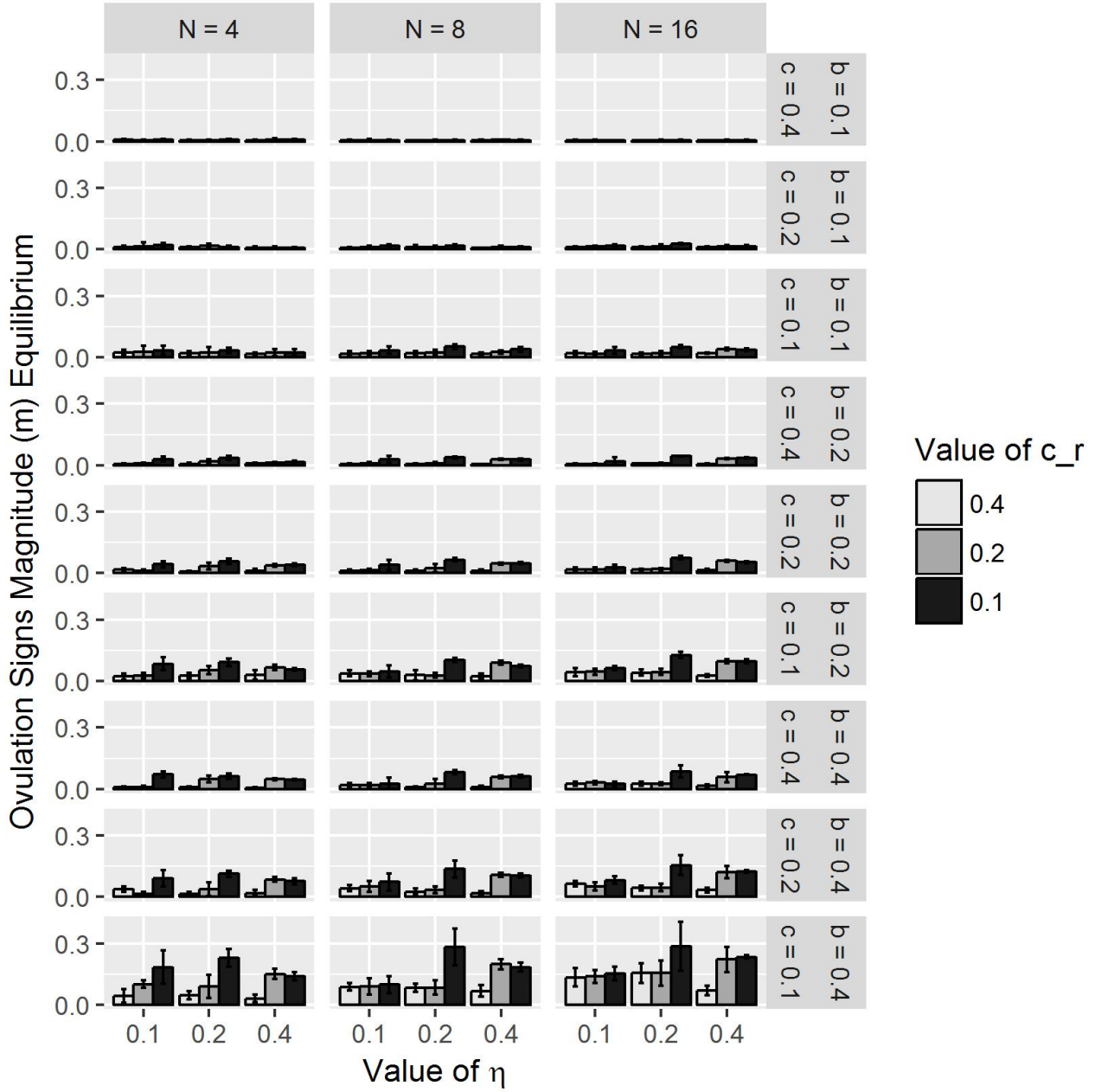


Figure 2.16: The effects of parameters N (group size), b (benefit of male genetic quality), c (cost of visible ovulation signs), c_r (cost of receptivity length), and η (weight put on genetic vs. non-genetic male benefits) on ovulation signs magnitude (m). Equilibria are obtained by averaging over 27 initial condition runs (with standard deviation indicated by error bars). Notice ovulation signs become stronger with decreasing N , increasing b , decreasing c , and decreasing η . All other parameters were held constant: $\alpha = 0$, $\epsilon_m = 0.25$, $\epsilon_f = 0.01$.

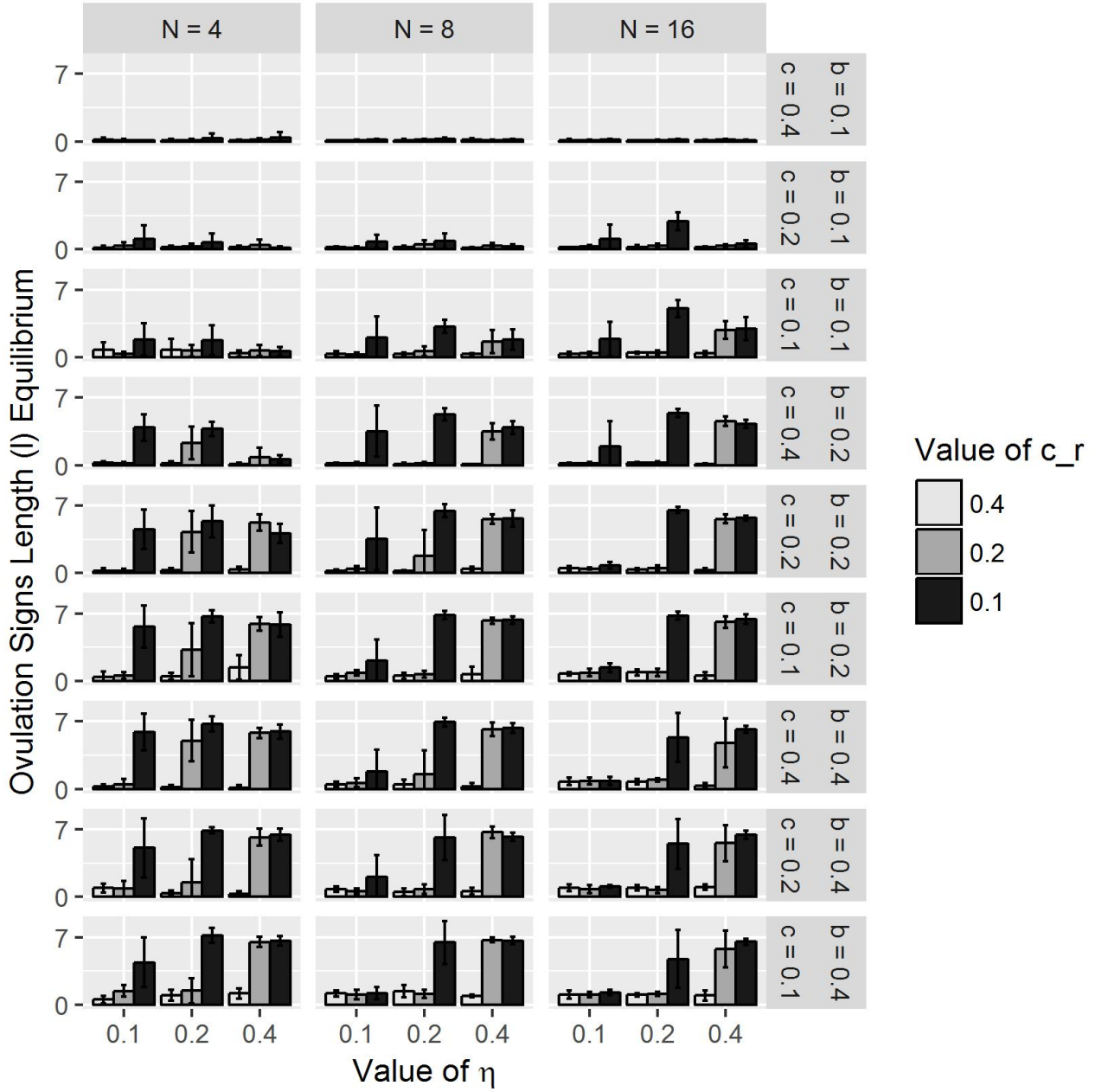


Figure 2.17: The effects of parameters N (group size), b (benefit of male genetic quality), c (cost of visible ovulation signs), c_r (cost of receptivity length), and η (weight put on genetic vs. non-genetic male benefits) on ovulation signs length (ℓ). Equilibria are obtained by averaging over 27 initial condition runs (with standard deviation indicated by error bars). Notice ovulation signs length become longer with decreasing N , increasing b , decreasing c , and increasing η . All other parameters were held constant: $\alpha = 0, \epsilon_m = 0.25, \epsilon_f = 0.01$.

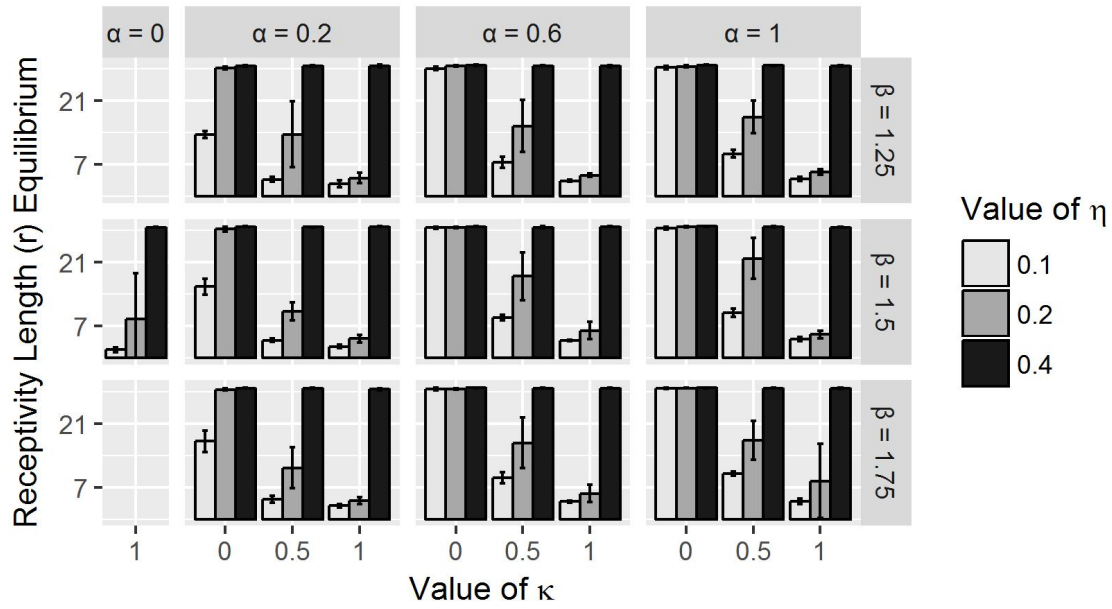


Figure 2.18: The effects of parameters α (maximum benefit of protecting offspring from infanticide), β (maximum cost of not protecting offspring from infanticide), κ (weight males put on visible ovulation signs when estimating their paternity), and η (weight put on genetic vs. non-genetic male benefits) on receptivity length (r). Equilibria are obtained by averaging over 27 initial condition runs (with standard deviation indicated by error bars). Notice receptivity length becomes longer with increasing α , increasing β , decreasing κ , and increasing η . All other parameters were held constant: $N = 8$, $b = 0.2$, $c = 0.2$, $c_r = 0.2$, $\epsilon_m = 0.25$, $\epsilon_f = 0.01$.

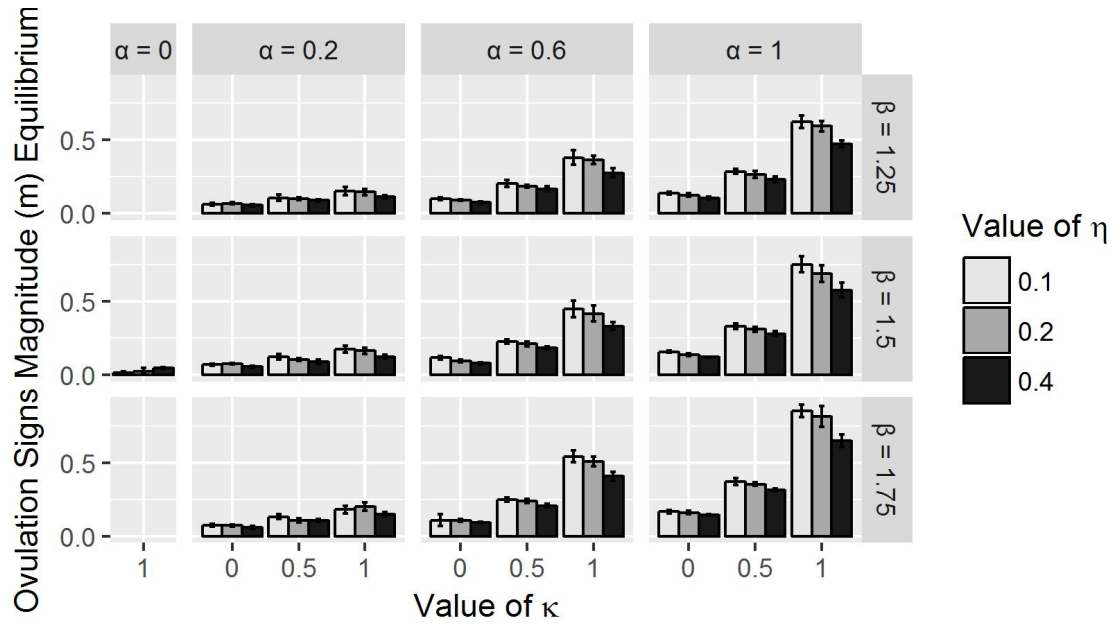


Figure 2.19: The effects of parameters α (maximum benefit of protecting offspring from infanticide), β (maximum cost of not protecting offspring from infanticide), κ (weight males put on visible ovulation signs when estimating their paternity), and η (weight put on genetic vs. non-genetic male benefits) on ovulation signs magnitude (m). Equilibria are obtained by averaging over 27 initial condition runs (with standard deviation indicated by error bars). Notice ovulation signs become stronger with increasing α , increasing β , decreasing κ , and decreasing η . All other parameters were held constant: $N = 8$, $b = 0.2$, $c = 0.2$, $c_r = 0.2$, $\epsilon_m = 0.25$, $\epsilon_f = 0.01$.

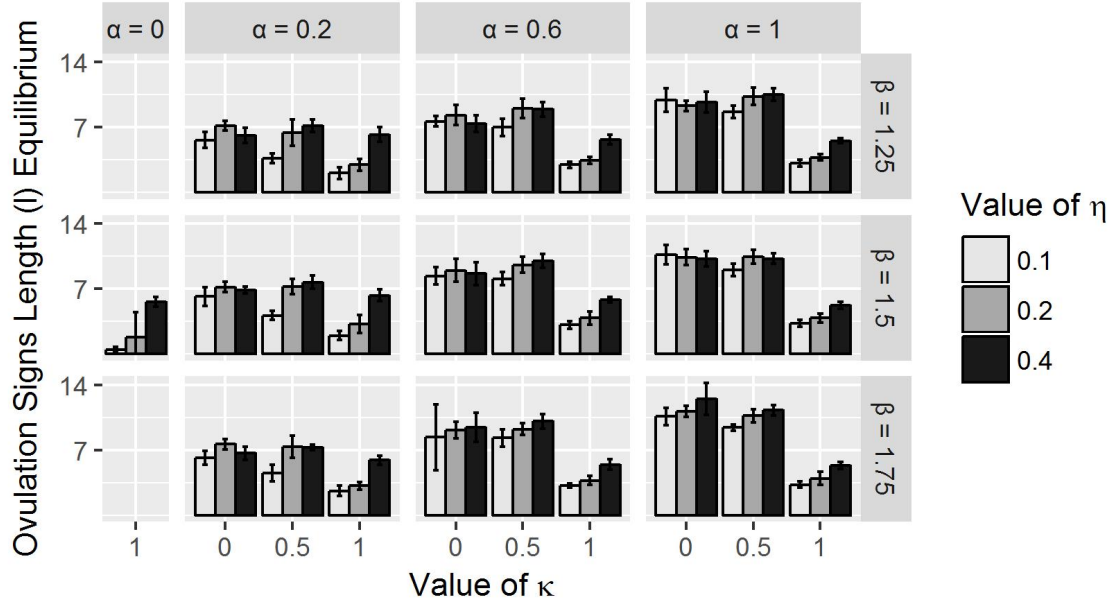


Figure 2.20: The effects of parameters α (maximum benefit of protecting offspring from infanticide), β (maximum cost of not protecting offspring from infanticide), κ (weight males put on visible ovulation signs when estimating their paternity), and η (weight put on genetic vs. non-genetic male benefits) on ovulation signs length (ℓ). Equilibria are obtained by averaging over 27 initial condition runs (with standard deviation indicated by error bars). Notice ovulation signs length become longer with increasing α , increasing β , and decreasing η . All other parameters were held constant: $N = 8, b = 0.2, c = 0.2, c_r = 0.2, \epsilon_m = 0.25, \epsilon_f = 0.01$.

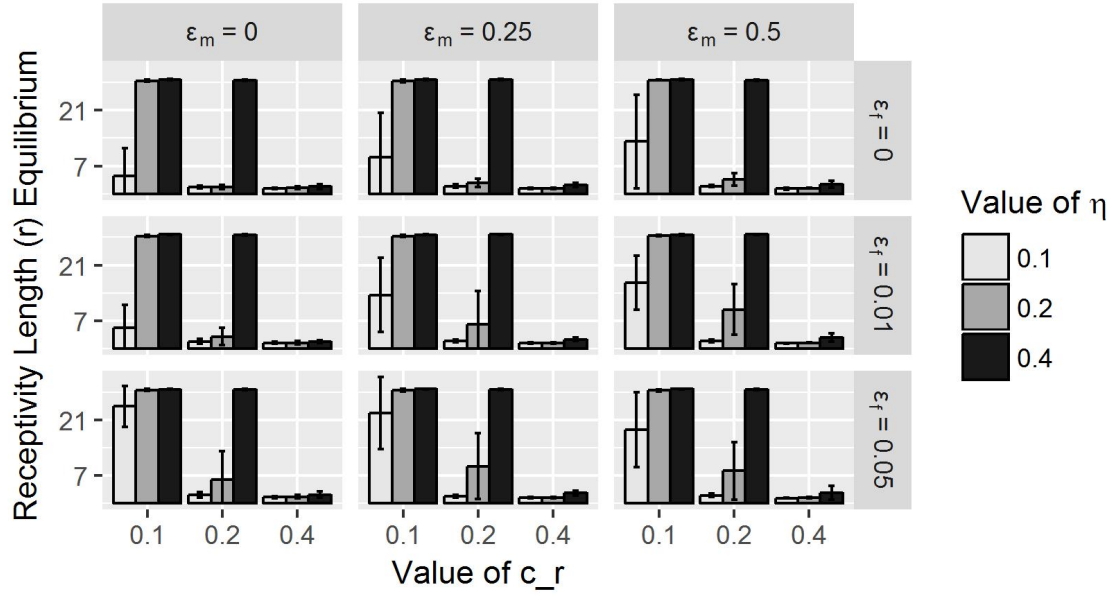


Figure 2.21: The effects of parameters ϵ_m (male reproductive stochasticity), ϵ_f (female reproductive stochasticity), c_r (cost of receptivity length), and η (weight put on genetic vs. non-genetic male benefits) on receptivity length (r). Equilibria are obtained by averaging over 27 initial condition runs (with standard deviation indicated by error bars). Notice receptivity length becomes longer with decreasing ϵ_m , decreasing ϵ_f , decreasing c_r , and increasing η . All other parameters were held constant: $\alpha = 0$, $N = 8$, $b = 0.2$, $c = 0.2$.

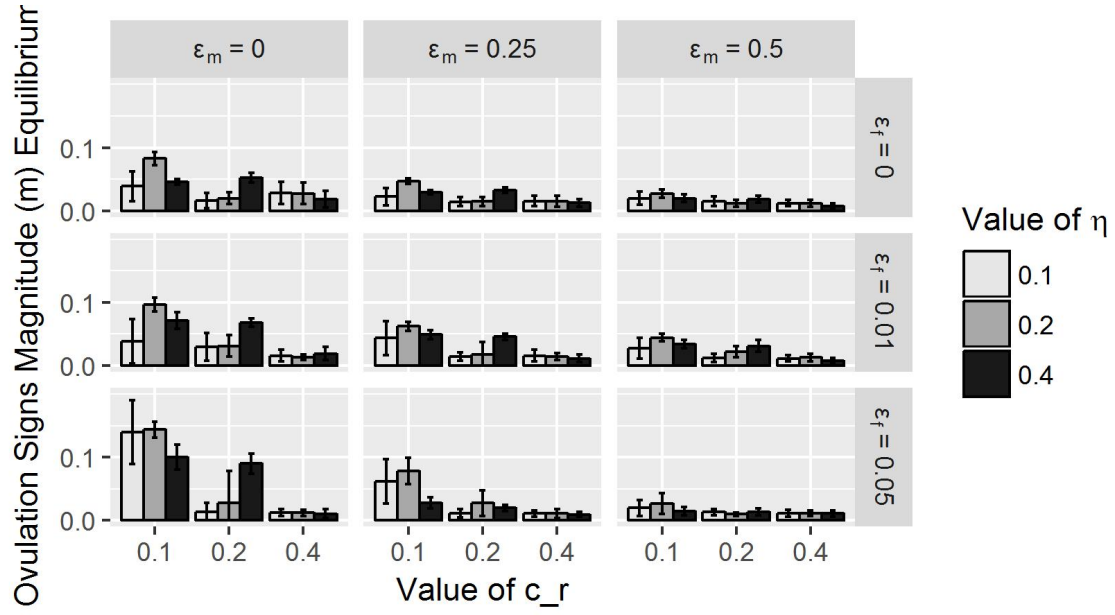


Figure 2.22: The effects of parameters ϵ_m (male reproductive stochasticity), ϵ_f (female reproductive stochasticity), c_r (cost of receptivity length), and η (weight put on genetic vs. non-genetic male benefits) on ovulation signs magnitude (m). Equilibria are obtained by averaging over 27 initial condition runs (with standard deviation indicated by error bars). Notice ovulation signs become stronger with decreasing ϵ_m , increasing ϵ_f , increasing c_r , and decreasing η . All other parameters were held constant: $\alpha = 0$, $N = 8$, $b = 0.2$, $c = 0.2$.

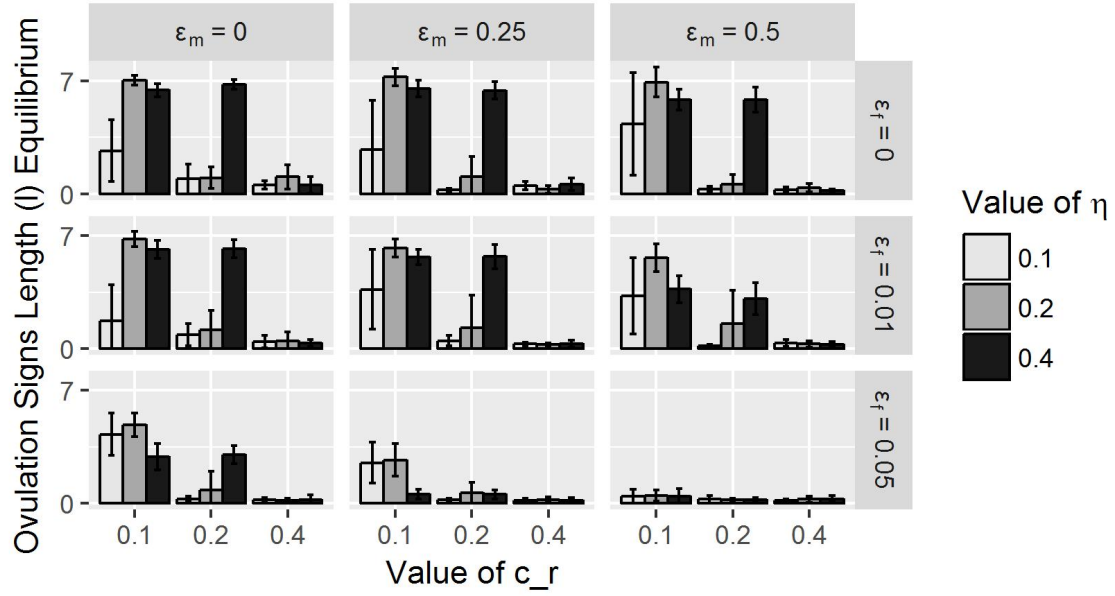


Figure 2.23: The effects of parameters ϵ_m (male reproductive stochasticity), ϵ_f (female reproductive stochasticity), c_r (cost of receptivity length), and η (weight put on genetic vs. non-genetic male benefits) on ovulation signs length (ℓ). Equilibria are obtained by averaging over 27 initial condition runs (with standard deviation indicated by error bars). Notice ovulation signs length become longer with decreasing ϵ_m , decreasing c_r , and decreasing η . All other parameters were held constant: $\alpha = 0$, $N = 8$, $b = 0.2$, $c = 0.2$.

Chapter III

On the evolution of female sexual signal reliability

Publication Statement: A version of this Chapter will be submitted soon for publication by its co-authors, Kelly Rooker and Sergey Gavrilets. The modeling and analytical work for this manuscript was primarily done by K. Rooker, and the manuscript also principally composed by K. Rooker.

On the evolution of female visual sexual signal reliability

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Abstract

Sexual signal reliability has been increasingly of interest to primatologists and evolutionary biologists. Among primates, why do some species have relatively reliable signals of ovulation, while other species have incredibly unreliable signals of ovulation? Even closely-related species like chimpanzees and bonobos vary drastically in such reliability, with chimpanzees having largely reliable signals of ovulation and bonobos having large variation in their signals. Here, we use mathematical modeling techniques to help answer these questions. In particular, our modeling results show that ovulation signal reliability increases with (1) increasing variation in male genetic quality, (2) increasing reproductive skew among males, and (3) increasing the importance of non-genetic benefits coming from mating. Overall, our work introduces a novel model into the field of primate sexual signal reliability and hopes to spark new comparative empirical work in the field.

1. Introduction

1.1. Background

Work on sexual conflict is nothing new (Parker, 1979; Arnqvist and Rowe, 2005), including in primates (Dixon, 2012). One example of known sexual conflict occurring in nature is via signaling. Such signals, whether through an individual's coloration, size, vocalizations, behavior, or pheromones, can help other individuals in deciding with whom to mate (Bradbury and Vehrencamp, 2011). While such mating signals are typically assumed to be characteristics of males allowing females to choose mates of high quality, mating signals can (and are) also used by females (see Ref. (Clutton-Brock, 2009) for a review and Ref. (Servedio and Lande, 2006) for modeling work).

A signal is simply any act that alters the behavior of another organism, has evolved because of this effect, and is effective because the receiver's response has also evolved (Maynard-Smith, 2003). An individual will produce a signal only if doing so will benefit his/her fitness, while other individuals will only receive such signals if doing so instead benefits their interests (Searcy and Nowicki, 2005).

In the case of mate quality, males and females each share the same interest of finding mates and reproducing. However, males and females are each trying to assess the mating quality of the other, while at the same time attempting to bias their quality to appear higher than others. Hence, despite the similar goal for both males and females in sexual signaling, there is still conflict present (Johnstone, 1997). Indeed, we see that males and females can prefer different outcomes (i.e. males wanting fertile females and females wanting choice in the father of their offspring), while both also have the overriding interest of wanting offspring produced (Maynard-Smith, 2003).

Such conflict can manifest itself via signalers attempting to manipulate receivers to the signaler's advantage via their signals, while receivers try to anticipate this manipulation and respond in such a way that their own fitness gets maximized. Such actions may ultimately result in 'arms races' (Clutton-Brock, 2016).

1.2. Honesty in Signals

For costly signals to exist within any species, both the signaler and receiver must benefit to some degree. If a signal contained absolutely no accurate information, then it would no longer be in the

receiver's interests to consider the signal. Similarly, if receivers simply ignored all signals, then it would also no longer be in the sender's interests to signal in the first place. Hence we know that, at a minimum, costly signals must contain *some* amount of useful information (Clutton-Brock, 2016).

Deception occurs when a signal's reception benefits the signaler at the expense of the receiver, regardless of the surrounding conditions (Bradbury and Vehrencamp, 2011). However, in order for dishonesty to evolve, the majority of signals need only be 'honest enough', meaning if signals in general are honest, then an occasional deceitful signal would be able to find possible success (Johnstone, 1997). 'Honest enough' can be thought of as 'honest on average', meaning it contains enough information, sufficiently often, that the receiver is better off considering the signal than ignoring it, on average (Searcy and Nowicki, 2005; Semple and Higham, 2013).

1.3. Signals Relating to Female Primate Ovulation

Due to the comparative ease in studying them, much of our understanding of mammalian communication and signaling is a result of studies of vocal communication. This does not mean vocal communication has been deemed more important than visual or olfactory signals, rather just a bias in past studies conducted (Clutton-Brock, 2016). In addition, the signal displays of many different species often use more than one signal component, and may even combine these together into multiple signals indicating one signal function. Multiple signals can help provide to other individuals more (and/or different) information about the signaler, or they could give more modalities in order to ensure signal detection (Johnstone, 1997).

Indeed, primate ovulation may be one example of multiple different signals being used. These could include behavioral signals (e.g. proceptive approaches and presentations), visual signals (e.g. skin color and sexual swellings), auditory signals (e.g. copulation or estrous calls), and/or olfactory signals (e.g. body odors and vaginal secretions) (Higham et al., 2008b, 2009; Daspre et al., 2009; Rigaiil et al., 2013; Rigaiil, 2014). Considering any of these mechanisms as signals is not a new idea, nor is debating them as being honest/reliable (Stallman and Froehlich, 2000; Gouzoules and Gouzoules, 2002). For example, experiments with visual sexual swellings as signals have shown males to be sexually stimulated by female sexual swellings (Gouzoules and Gouzoules, 2002) and males to use changes in sexual swellings to decide mating activity (Reichert et al., 2002; Deschner

et al., 2004; Barelli et al., 2008; Gesquire et al., 2007; Brauch et al., 2005).

While males are typically not thought of as the ‘choosy sex’, primate males still do make choices about which female(s) to mate with, and whether or not to guard them. It is increasingly accepted that males suffer some amount of sperm limitation, with the costs of sperm production not trivial (Dewsbury, 1982; Stallman and Froehlich, 2000). For males, knowing when copulation will lead to conception is important since that allows males to concentrate their mating efforts on fertile females, hence considerably reducing the effort and costs associated with reproduction. Indeed, in most mammalian taxa, females will exhibit a short period of sexual activity only, which males can relatively easily detect (Engelhardt et al., 2005).

With ovulation being a momentary occurrence and female visual sexual signals lasting for sometimes weeks, it is hard to imagine such signals pinpointing the precise moment of ovulation. This is particularly true when considering that the lifetime of sperm is typically much shorter than the maximum swelling length in most species (Stallman and Froehlich, 2000). Still, there is often some amount of probabilistic association between a female’s time of peak sexual swellings and ovulation, as predicted by costly signals needing to be ‘honest enough’ (Gouzoules and Gouzoules, 2002). However, as a female’s maximum swelling length increases past her length of fertility, and as variability in the timing of ovulation becomes greater, the less precisely the female’s sexual swellings are going to indicate her timing of ovulation (Douglas et al., 2016).

Indeed, in different primate species, the timing of ovulation has been found to be highly variable (e.g. (Heistermann et al., 1996, 2001; Reichert et al., 2002; Engelhardt et al., 2005; Douglas et al., 2016)). In wild Hanuman langurs, for example, ovulation was found to occur anywhere from a female’s first day of receptivity to her last (Heistermann et al., 2001). In wild long-tailed macaques, despite females’ fertile periods always being inside females’ period of having sexual swellings, the timing of such 4-day fertile periods (and hence ovulation) varied considerably within the average 15-day maximum swelling period (Engelhardt et al., 2005). Bonobos, which also show large sexual swellings, have even more extreme patterns, with ovulation occurring during the female’s maximum swelling period in only 53% of cycles observed in one study. Such timing of ovulation in that study was found to vary greatly both between individuals and also within individuals between cycles

(Douglas et al., 2016).

On the other hand, sexual swelling size has also been found to be a relatively reliable indicator of ovulation in other species, with male mating behavior being well-timed to such female sexual swellings and ovulation (e.g. (Deschner et al., 2003, 2004; Mohle et al., 2005; Barelli et al., 2007; Brauch et al., 2005; Gesquire et al., 2007; Higham et al., 2008a,b; Daspre et al., 2009; Dubuc et al., 2009; Higham et al., 2012; Rigai et al., 2013)). For example, in free-ranging Barbary macaques, sexual swelling size was found to increase predictably towards the time of ovulation and reach maximum size during the female's fertile phase. In addition, increasing male ejaculatory copulation rates aligned with the increased sexual swelling sizes, leading to maximum rates during maximum swellings (Brauch et al., 2005). In wild chimpanzees, sexual swellings do not provide precise information about a female's time of ovulation, but do give an informative *window* of time during which ovulation is expected to occur (Deschner et al., 2003). Similarly, in wild white-handed gibbons, ovulation will typically happen during the maximum swelling period, although the timing within that period is quite variable (Barelli et al., 2007).

1.4. *Comparing Bonobos and Chimpanzees*

Comparing bonobos and chimpanzees (both *Pan* species) may be particularly informative due to their phylogenetic closeness and differences in social and reproductive behaviors (Paoli et al., 2006). In bonobos, females have been found to have ovulation occur outside of the period of maximum sexual swellings in over 30% of ovulatory cycles (captive: (Reichert et al., 2002), wild: (Douglas et al., 2016)). Bonobos also have a very low day-specific probability of ovulation (Douglas et al., 2016), especially as compared to chimpanzees (Deschner et al., 2003). For instance, in order for a male to guard a female long enough to ensure she has had a 50% chance of reaching ovulation, a male chimpanzee would only need to guard the female for 3 days, whereas a male bonobo would have to guard an equivalent female for 10 days (Deschner et al., 2003).

1.5. *Hypotheses Related to Primate Signal Reliability*

Such questions of female sexual signaling have long been of interest to primatologists and evolutionary biologists, including their applications to humans (Thornhill and Gangestad, 2008). There

have been several different hypotheses proposed to explain such signal variability among species including variation in male competition (Higham et al., 2012), constraining mate-guarding efforts of fertile females by males (Douglas et al., 2016), and to obtain higher paternal contributions to offspring (Daspre et al., 2009).

First, ovulation signal reliability could be a function of male competition. In species with high sexual dimorphism and high reproductive skew (each indicating males fighting for dominance), females would do better to concentrate paternity in those high-quality males winning the competition and hence reliable ovulation signals are favored. Conversely, in species with low sexual dimorphism and reproductive skew (indicating males are in scramble competition and will instead queue for dominance), females have less benefit in concentrating paternity and hence favor unreliable ovulation signals for paternity confusion (Higham et al., 2012).

Second, ovulation signal reliability could instead be a function of trying to control mate-guarding efforts. The more unreliable an ovulation signal is, the less beneficial it is to males to guard females around their time of maximum swellings. In this way, females may be able to combat male mate-guarding efforts by having more unreliable ovulation signals (Douglas et al., 2016).

Third, ovulation signal reliability could be a function of paternal investment in offspring. When there exist high paternal contributions to offspring fitness, there should be greater selection in the female for higher signal reliability, since now females would have a greater interest in aiding paternity certainty (Daspre et al., 2009). Hence there exists a trade-off for females between signal reliability (which aids paternity certainty) and signal variability (which aids paternity confusion) (van Schaik et al., 1999).

With not many comparative studies involving these hypotheses, little is currently known about which are favored, and in particular in what species and/or under what circumstances. As Tim Clutton-Brock recently said, “A broader approach to signal evolution is needed that incorporates quality-dependent viability trade-offs and explores the consequences of individual variation in the efficiency of signal production” (Clutton-Brock, 2016).

1.6. Research Questions

Here, we are interested in exploring the question of why is there so much variation in the reliability of female visual sexual signaling among primate species? Why do some species have visual sexual signals reliably indicating a female’s time of ovulation, while other species have the same visual sexual signals but instead with a highly variable timing of ovulation?

It is our aim here to test these hypotheses using a mathematical model. In addition to looking at the above hypotheses, we are also interested in the effects on signal reliability of group size, infanticide, and sexual signal reliability’s relationship to female sexual receptivity length. While there has been much mathematical modeling work done with regards to biological signaling in general, and also with honesty and deception in biological signals (for example, see Refs. (Grafen, 1990; Johnstone and Grafen, 1992a,b; Krakauer and Johnstone, 1995; Johnstone, 1995; Payne and Pagel, 1996; Getty, 1998; Szamado, 2000; Kokko and Johnstone, 2002), to our knowledge there has yet to be any mathematical modeling work done with ovulation signaling. It is our aim here to fill that gap.

2. Introduction to the Model

2.1. General Model Set-Up

We consider a population of individuals living in a large number of multi-male, multi-female groups. We assume generations are discrete and non-overlapping. Females differ genetically in signal reliability, while males differ in their quality. Such ‘quality’ is split into a genetic component (GC) and a non-genetic component (NGC). GC can be thought of as the genetic benefit to a female of mating with a male with good genes, while NGC can be thought of as any non-genetic benefit to a female from protection provided to the female herself (i.e. not her offspring), increased food/provisioning/access to resources, or paternal care (see Ref. (Clutton-Brock, 2016) for further description of such benefits). Note that GC and NGC are inversely related such that a male with higher GC will have lower NGC and vice versa.

We account for group size (N), variation in male quality (scaled by parameter b), and weighting of NGC vs. GC (η). (See Methods for further details, explicit fitness functions, and full definitions

of equations and parameters.) Our goal is to understand how female sexual signal reliability evolves over time.

We build an agent-based model such that:

$$\begin{aligned}
\text{female fitness} &= \text{baseline female fitness} \\
&+ (1 - \eta) * (\text{benefit of male GC}) \\
&+ \eta * (\text{total average benefit of male NGC}) \\
&\pm \text{benefit/cost due to infanticide from each male.}
\end{aligned}$$

2.2. The Female Cycle

Our model explicitly accounts for the female cycle, which is split into discrete units of time. While such units could be hours, minutes, etc., we choose to call such units ‘days’ (for example, with 29 days in one female cycle). We treat visual ovulation signs, $x(d)$ for each day d of the cycle, as overlapping curves (Dixon, 2009). Each female is characterized by two traits: magnitude (m) and length (ℓ). m is the maximum amount of ovulation signs a female has visible during her cycle (a non-negative, continuous value), while ℓ is the number of days a female has *some* amount of ovulation signs visible (a non-negative, integer value).

For each female, the distribution of probabilities of fertilization across the cycle has the same shape but is randomly centered (meaning cycle synchrony may only occur probabilistically). Signal reliability p is a genetically controlled trait depicting the number of days either before or after a female’s peak day of having ovulation signs present (i.e. the day she has m signs visible) where the female will have her maximum probability of fertility. Note with $p = 0$, the medians of a female’s fertility length and visible ovulation signs length will be the same.

2.3. Male-Female Mating

For every unit of time in the cycle, all males and all receptive females in each group enter the competition for mates. For computational simplicity, we assume each female to mate exactly once on every unit of time (e.g. day) she is receptive. Males of higher GC are more likely to mate with females with stronger ovulation signs on that particular day (i.e. higher $x(d)$). Males of lower GC mate with females who have fewer or no ovulation signs visible.

The degree of reproductive stochasticity in males and females is controlled by parameters $\epsilon_m \geq 0$ and $\epsilon_f \geq 0$, respectively. The higher ϵ_m is, the more likely low-GC males are to be able to mate with females with the strongest signal. The higher ϵ_f is, the more unreliable the female signal is in winning high-GC mates. With $\epsilon_m = \epsilon_f = 0$, there is no reproductive stochasticity and mating pairs are formed deterministically so that the highest-GC male mates with a female with the most ovulation signs visible, and so on). (See Methods for a full description of these parameters.)

Mating is followed by offspring production and dispersal. For any female's potential offspring, every male in the group will have an associated probability of paternity, ranging anywhere from zero to one. This quantity is determined by both the number of times that particular male mates with the female during her cycle, as well as her probability of fertilization on the days of each mating event.

2.4. *Evolution of Multiple Traits*

Notice that in the above we only allow trait p to evolve, holding females' ovulation signs traits (m, ℓ) and receptivity length (r) constant. In addition to this case, we also evolve each of p, m, ℓ, r together, in order to see possible interactions between these. r is the length of time the female is receptive to mating (a non-negative, integer value).

Our female fitness function in this case becomes:

$$\begin{aligned}
 \text{female fitness} &= \text{baseline female fitness} \\
 &+ (1 - \eta) * (\text{benefit of male GC}) \\
 &+ \eta * (\text{total average benefit of male NGC}) \\
 &\pm \text{benefit/cost due to infanticide from each male} \\
 &- \text{cost of having visible ovulation signs} \\
 &- \text{cost of receptivity,}
 \end{aligned}$$

where we now account for the costs of having visible ovulation signs (scaled by parameter c) and costs of being receptive (scaled by parameter c_r).

2.5. Infanticide

Modeling infanticide is handled similarly to Chapters 1 and 2. Any male is able to help or harm (note ‘harm’ here could simply mean not helping) an offspring prior to the infant being weaned, although we typically assume that some males (e.g. those of higher quality or strength) are able to do so more effectively. Our model does not distinguish between the threat of infanticide via an outsider male or a previously-subordinate male. Infanticide’s effects on offspring viability are scaled by parameters α (maximum benefit) and β (relative maximum cost). An additional parameter $0 \leq \kappa \leq 1$ determines the extent to which males take visible female ovulation signs into account when estimating their paternity. $\kappa = 0$ means males estimate their probability of paternity solely by the *number* of matings with an offspring’s mother, while $\kappa = 1$ means males instead estimate paternity exclusively on the basis of the female’s visible ovulation signs during their time of mating.

3. Methods

3.1. General Model

We consider a population of individuals interacting in G groups comprised of N males and N females. Males are ordered according to their GC to females, denoted y_j such that $y_1 > y_2 > \dots$ for each male j . Intuitively, females will gain more fitness benefit from mating with males of higher GC. Such GC is randomly drawn from the normal distribution $\mathbb{N}(\hat{y}, b)$; parameter $\hat{y}$ characterizes mean male GC and $b > 0$ the extent of variation in male GC. Small b implies small potential benefits to females from mating with even high-GC males. [For simplicity, the distribution of y in the population remains constant, e.g. as a result of mutation-selection balance.] Male NGC is then equal to the reflection of GC over the center of the normal distribution, the vertical line $y = \hat{y}$.

We explicitly incorporate the female estrous cycle using D discrete units of time (e.g. $D = 29$ days). For each of these D days, every female will have an associated probability of fertilization. We assume each fertile period to last $C \leq D$ days (e.g. $C = 7$). For all days lying outside those C fertile days, females are assumed to have zero probability of fertilization but can still mate (see SI for example curves). We assume a triangular shape for the fertility function to be identical in all

females, but with midpoints randomly distributed so as the only cycle synchrony occurring within groups is probabilistic.

For all females, we assume ovulation to happen directly in the middle of the C fertile days. For any female with trait $p = 0$, we assume maximum visual ovulation signs to happen directly in the middle of her C fertile days as well, and hence align perfectly with ovulation (signal reliability). For any female with $p \neq 0$, the female will instead have her day of maximum visible ovulation signs be p days away from her middle day of fertility (signal variability). Mutations in p are discrete, representing adding or subtracting exactly one day to or from the distance between ovulation (i.e. the median day of a female's fertility period) and having maximum ovulation signs visible. Note the full range of p is limited to ensure visible ovulation signs still stay within the female's cycle.

Considering one focal group, for any female i with traits $m_i \geq 0$ (ovulation signs magnitude) and $\ell_i \in \mathbb{Z}, \ell_i \in [0, D]$ (ovulation signs length), the amount $x_i(d)$ of ovulation signs visible on any day d of her cycle is given by:

$$x_i(d) = \begin{cases} m_i \cdot \left[1 - \left(\frac{2|d - \lceil \ell_i/2 \rceil|}{\ell_i + 1} \right)^\gamma \right] & \text{for } d = 1, 2, \dots, \ell_i \\ 0 & \text{otherwise} \end{cases} \quad (38)$$

for $\gamma > 0$ and d measured from the start of ovulation signs being visible. Note m_i represents the peak of $x_i(d)$ and ℓ_i the width of the non-zero portion of $x_i(d)$.

On each day, mating pairs are formed by first randomly perturbing both the male trait y and female trait x by adding to each an independent, normally distributed random variable with standard deviation $\epsilon_m \geq 0$ and $\epsilon_f \geq 0$, respectively. Males and females are then sorted according to these perturbed values and mating occurs between individuals of the same order.

This stochasticity in male ordering reflects stochasticity in the outcome of their competition for mating success; a large ϵ_m means lower reproductive inequality. Stochasticity in female ordering reflects a lack of opportunity to mate with a higher quality male/female, disinterest in mating with a specific male (female) regardless of quality (signs), etc. Note that males will not always choose to mate with the receptive female with the largest visual sexual signal (e.g. Ref. (Fitzpatrick et al.,

2015)), which can be accounted for in our model with $\epsilon_f > 0$.

In the case of no infanticide and only p evolving, we have female fitness $w_i = w_0 + (1 - \eta)y_{m,i} + \frac{\eta}{D} \sum_{\text{all mates } j} NGC_j$, where w_0 is baseline fitness, $y_{m,i}$ the benefit from female-female competition (i.e. the GC of the specific male who won the contest of paternity for that particular female's offspring), and NGC_j the NGC coming from each male j a female mated with throughout her cycle.

3.2. Modeling Infanticide

Each male in a female's group can affect her fitness directly, due to infanticide, although males will differ in their actions' effectiveness (e.g. due to strength, rank, alliances, etc.). We assume each male's effectiveness to be proportional to $f(j)$ for each male j (sorted by male GC) via an exponential function: $f(j) \sim e^{-\omega j}$ with parameter $\omega > 0$ controlling the amount of disparity among males in their corresponding effectiveness within the group. Note a larger value of ω indicates more disparity, and $\omega = 0$ equality.

A male's contribution $g(r)$ to female fitness (positive via helping protect the offspring, or negative via not helping and/or harming the offspring) depends on his perceived probability of paternity r . Adapted from Ref. (van Schaik et al., 2004), we define $g(r) = \alpha[1 - \beta(1 - r)^\tau]$, with parameters $\alpha, \beta > 0$ and $\tau \geq 1$. Note α determines the maximum benefit a female can obtain from a male protecting her offspring from infanticide, while $\alpha(1 - \beta)$ determines the maximum cost a female can incur from a male *not* protecting her offspring from infanticide. The overall effect of infanticide on female fitness (that gets added to the right-hand side of the fitness function listed above) is $\sum_j f(j)g(r_j)$, summing over all males j . Note whenever $\alpha = 0$, there are no effects of infanticide.

A male in general would not know his actual paternity probability and would instead have to estimate it. We define this male-estimated ("perceived") probability by male j of being the father of an offspring born to female i by $r_{i,j} = \sum_{\# \text{ of matings}} (x_i(d))^\kappa$, where $\kappa \in [0, 1]$ represents the weight males put on visual ovulation signs when estimating paternity. Note $\kappa = 0$ represents males estimating their paternity solely on the number of matings with that mother, while $\kappa = 1$ represents males putting full weight instead on her visual ovulation signs. For each male j , these perceived paternity probabilities get normalized such that $\sum_{i=1}^N r_{i,j} = 1$.

3.3. With Multiple Traits Evolving

With multiple traits evolving, we have female fitness:

$$w_i = w_0 + (1 - \eta)y_{m,i} + \frac{\eta}{D} \sum_{\text{all mates } j} y_{p,j} + \sum_{\text{all males } j} f(j)g(r_j) - c \cdot \bar{x}_i - c_r \cdot \bar{r}_i. \quad (39)$$

Here, w_0 is baseline fitness, $y_{m,i}$ the benefit from female-female competition (i.e. the GC of the specific male who won the contest of paternity for that particular female's offspring), $y_{p,j}$ the NGC coming from each male j a female mated with throughout her cycle, $c \cdot \bar{x}_i$ the costs to a female of supporting her visual ovulation signs with $\bar{x}_i = \frac{1}{D} \sum_{d=1}^D x_i(d)$, a female's average visual ovulation signs, and $c_r \cdot \bar{r}_i$ the costs to a female of receptivity with $\bar{r}_i = \frac{r}{D}$, a female's proportion of the cycle being receptive. Note our choice of the first cost term implies there are fitness trade-offs between a female's m and ℓ traits so that having large values of both traits could imply very large fitness costs.

3.4. Simulations

This model was implemented in C++, with both only p evolving and with each of p, m, ℓ, r evolving. Migration occurs with only females migrating between groups, although simulations were also run with only males migrating between groups and the results were unaffected. Mutation occurs with a mutation rate per gene per generation of 10^{-3} . Mutations for p are drawn from the set $\{-1, 1\}$ with equal probability and ensuring they stay between $p \in [-\frac{D}{2}, \frac{D}{2}]$. Mutations for m are selected from $\mathcal{N}(0, 0.1)$, while mutations for ℓ and r independently from the set $\{-1, 1\}$ with equal probability and ensuring they stay between $\ell, r \in [0, D]$. $2N$ offspring are created in each group every generation, with N female and N male, determined randomly. The expected number of offspring per female is 2; the actual number is random, proportional to each female's fitness.

The model with only p evolving uses 10 different initial conditions in all simulations: $p = -9, -7, -5, -3, -1, 1, 3, 5, 7, 9$. The model with each of p, m, ℓ, r evolving uses 16 different initial conditions, namely every combination of: $p = -2, 2$; $m = 0, 0.9$; $\ell = 1, 4$, and $r = 7, 21$. Parameters used include: $G = 400$; $T = 100,000$; $D = 29$; $C = 7$ (with probabilities of fertilization on each of these days being 0.125, 0.25, 0.375, 0.5, 0.375, 0.25, 0.125, respectively); $w_0 = 2$; $N = 4, 8, 16$;

$b = 0.1, 0.2, 0.4$; $c = 0.1, 0.2, 0.4$; $c_r = 0.1, 0.2, 0.4$; $\eta = 0.1, 0.2, 0.4$; $\gamma = 1$; $\hat{g} = 1$; $\epsilon_m = 0, 0.25, 0.5$; $\epsilon_f = 0, 0.01, 0.05$; $\alpha = 0, 0.2, 0.6, 1$; $\beta = 1.25, 1.5, 1.75$; $\tau = 2$; $\omega = 1$; and $\kappa = 0, 0.5, 1$. Further information on parameter values tested and each of their effects is available in the SI.

4. Results

For this model, we consider the evolution of signal variability p both by itself and with the evolution of each of ovulation signs magnitude (m), ovulation signs length (ℓ), and receptivity length (r).

For each of these cases, we run numerical simulations of our model. Numerical simulations of the model in all cases show that the average values of the signal variability trait (p) (and ovulation signs magnitude trait (m), ovulation signs length trait (ℓ), and receptivity length trait (r), if included) converge to the unique equilibria p^* (and m^*, ℓ^*, r^* , if included). A more complete investigation of the effects of all parameters can be found in the Supplemental Information.

In general, we were able to test three main predictions with our model, which can be seen in both Fig. 3.1 and Fig. 3.2:

1. Ovulation signal reliability is a function of variation in male quality: signal reliability (i.e. decreasing p) will increase with increasing b .
2. Ovulation signal reliability is a function of male competition and reproductive skew: signal reliability (i.e. decreasing p) will increase with decreasing ϵ_m .
3. Ovulation signal reliability is a function of non-genetic contributions to offspring fitness: signal reliability (i.e. decreasing p) will increase with decreasing η .

In addition, in Fig. 3.1, we see that increasing group size N increases signal reliability (i.e. decreases p). The results displayed in Fig. 3.2 are from simulations that add ovulation signs magnitude (m), ovulation signs length (ℓ), and receptivity length (r) as all able to evolve in addition to signal variability (p). The main result from this case is that there is a connection between visible ovulation signs and signal reliability. Whenever a parameter combination results in concealed ovulation (i.e. both m^*, ℓ^* close to zero), it also results in larger signal variability (p^*). Conversely, parameter combinations with high m^* (i.e. strength of the visible ovulation signal) will have much

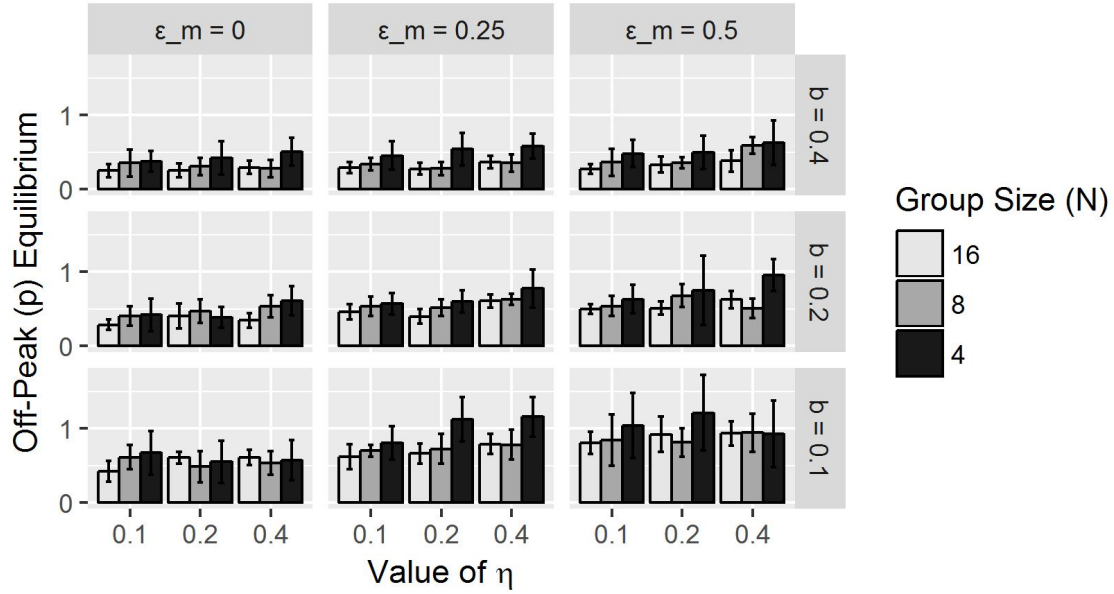


Figure 3.1: The effects of N (group size), b (variation in male quality), ϵ_m (male reproductive stochasticity), and η (weighting of NGC vs. GC) on the average equilibria values of the off-peak equilibria p^* . Equilibria are obtained by averaging over 10 initial condition runs (with standard deviation indicated by error bars). All other parameters held constant: $c = 0$, $c_r = 0$, $\epsilon_f = 0.01$, $\alpha = 0$.

more reliable signals (p^* close to zero).

4.1. ANOVA Results

Finally, for both with and without infanticide in the case of multiple traits evolving, we run an analysis of variance (ANOVA) to determine which of receptivity length r , ovulation magnitude m , ovulation length ℓ , and signal reliability p are affected most by which parameters. Full results of these tests can be found in the Supplementary Information, but we do find that considering linear interactions only is sufficient. In general though, we find that the ANOVA results reaffirm the patterns discussed above.

Traits r , m , and ℓ behave much the same way as in Chapter 2: η positively affects each of r , m , ℓ in the infanticide case, as do parameters α, β in the infanticide case. κ positively affects m , while negatively affecting r, ℓ . b positively affects ovulation signs (m, ℓ) and negatively affects receptivity r . Parameter c will not increase any of m, ℓ, r . Finally, ϵ_m has no effect on receptivity length r , and negatively affects each of m, ℓ .

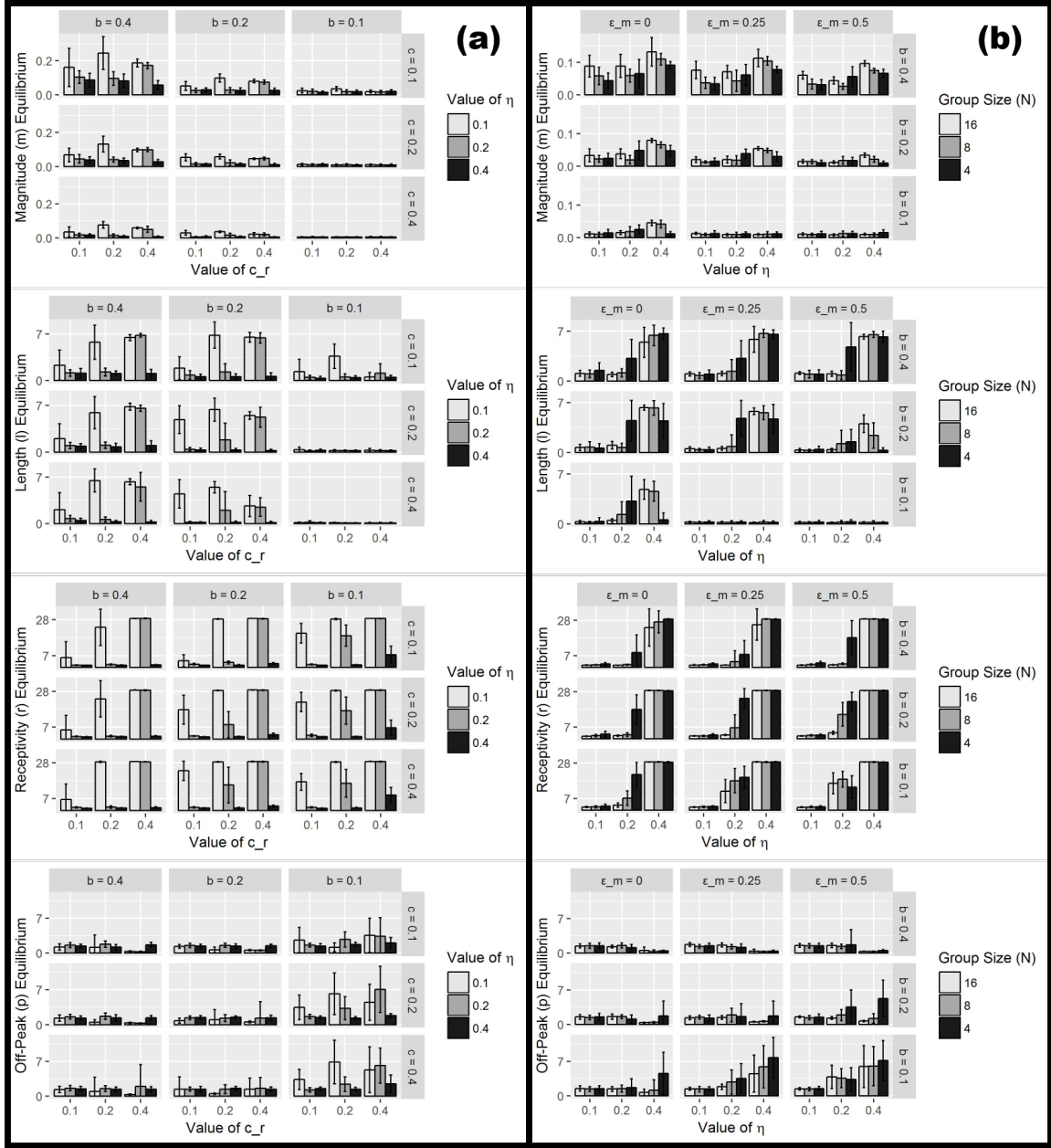


Figure 3.2: The effects of varying parameters on the average equilibria values of receptivity length (r^* , left graphs), ovulation signs magnitude (m^* , middle graphs), and ovulation signs length (ℓ^* , right graphs), with both continuous receptivity assumed (white bars) and evolvable receptivity (black bars). Equilibria are obtained by averaging over 10 initial condition runs (with standard deviation indicated by error bars). (a) Varying N (group size), b (variation in male quality), and c_r (costs of receptivity) in the case of no infanticide ($\alpha = 0$). (b) Varying α (maximum benefit of infanticide protection), β (maximum cost of infanticide), and κ (weight males put on visible ovulation signs in estimating paternity) in the case of with infanticide ($N = 8, b = 0.2, c_r = 0.2$). All other parameters held constant: $c = 0.2, \epsilon_m = 0.25, \epsilon_f = 0.01$.

In terms of parameters' effects on signal reliability p , they are similarly as expected. Most importantly, p will decrease (i.e. the ovulation signal becoming *more* reliable) as b increases, and increase as ϵ_m, η increase.

5. Discussion

It was our goal here to investigate the reliability of primate sexual signals. In particular, we were interested in the effects on signal reliability of group size, male genetic benefits, reproductive stochasticity/skew, the weighting of male genetic vs. non-genetic benefits, and sexual signal reliability's relationship to both female sexual receptivity length and female visual sexual signals.

The first four of these results are quite intuitive. First, in Fig. 3.1, we see that increasing group size N increases signal reliability (i.e. decreases p). Indeed, larger group sizes imply higher competition among females for high-GC mates. Increased competition means females will have fewer opportunities to mate with high-GC males, and hence it is in their best interests for increased honesty in their signal. Otherwise, with fewer opportunities *and* a dishonest signal, a female would likely get no benefit at all from mating with a high-GC male.

Second, we see that p will decrease with increasing b (variation in male GC). Similar to increasing group size, as b increases, the difference between the highest-GC male and the average-GC male will also increase, thus increasing the value of winning the female-female competition for high-GC mates. Again, increased competition means fewer opportunities, and hence a more reliable signal (smaller p).

Third, we see that as ϵ_m (reproductive stochasticity among males) decreases, p also decreases, meaning signal reliability increases. As reproductive stochasticity *decreases*, reproductive inequality is *increasing*, meaning reproductive skew is also increasing. As reproductive stochasticity decreases, females have more incentive to win the female-female competition for high-GC mates. This is because with lower reproductive stochasticity, the genetic quality of the male with whom the winning female gets to mate is likely to be higher than if there were high reproductive stochasticity. Hence, we see signal reliability increase with decreasing reproductive stochasticity.

Fourth, we see increased signal reliability with decreasing η . As η decreases, the weighting of

male genetic benefits (compared to their non-genetic benefits) increases. As such, there is increased benefit to females from having high-quality males be the father of any offspring. Since females are most likely to attract such high-quality males only when their visible ovulation signal is strongest, it is in females' best interests to have a very reliable ovulation signal in order to obtain those genetic benefits from mating with such high-quality males (by having them win the contest for paternity).

When evolving multiple traits simultaneously, the connection between visible ovulation signs and signal reliability is quite intuitive as well. Females having strong visible ovulation signs is costly (due to increased energy expenditure, predation, water retention, etc.). However, if having such visible ovulation signals helps females win copulations with high-GC mates, then that benefit coming from paternity concentration may help offset such costs. If these females also have large signal variability, then winning the competition for high-GC mates would not necessarily afford them the genetic benefit (if they are not also highly fertile at the time of their peak signal, when they would be most attractive to the high-GC males). Conversely, females with strong ovulation signs also having very reliable signals would help make sure that after investing so much energy and resources in their signal, they will be able to gain some benefit (in the form of GC) from having the signal.

Variation in signal reliability among primates has been an increasingly-researched topic in the empirical literature. While much work still needs to go into understanding the evolution of sexual signal reliability in female primates, our work takes a novel step in this research, by introducing the first theoretical work completed on the subject. In particular, our work suggests several pathways by which signal reliability could have evolved or diminished. While it would be wonderful to test our model's predictions on available data, much more empirical data would be needed in order to do any kind of effective empirical analysis between species. We hope for this theoretical research to inspire further comparative empirical work on the subject.

6. Acknowledgments

K.R. was supported by a National Science Foundation Graduate Research Fellowship. S.G. was supported by the National Institute for Mathematical and Biological Synthesis through National

Science Foundation awards EF-0832858 and DBI-1300426, with additional support from The University of Tennessee. All numerical simulations were run on the Volos cluster, which is supported by National Institutes of Health award GM56693 (S.G.).

7. Supplementary Information

7.1. Further Details of the Model

7.1.1. The Female Cycle

Female estrous cycles are divided into D discrete units of time (e.g. $D = 29$ days) with $C \leq D$ days of fertility (e.g. $C = 7$). Although the form of such fertile periods will be identical in all females, the timing of each fertile period will be randomly distributed among females. For instance, when $C = 7$ (calling each of these fertile days $C1, C2, \dots, C7$) and $D = 29$ (calling each of these days $D1, D2, \dots, D29$), $C1$ in every female has an equal probability of landing on any of $D1, D2, \dots, D29$. Since C is a cycle, we similarly assume $C7$ can land on any such day. For example, if $C1$ landed on $D24$, then $C7$ would ‘wrap around’ the cycle to land on $D1$. Moreover, for all females we assume ovulation happens directly in the middle of this C cycle. Hence, for $C = 7$ we assume ovulation happens on $C4$ and the probabilities of fertilization increase from $C1$ to $C4$ and then decrease from $C4$ to $C7$.

Each female i will have $r_i \leq D$ days of receptivity. Each female i will also be characterized by trait $p_i \geq 0$, the number of days separating a female’s day of ovulation (i.e. her median day of fertility) and her peak day of having visible ovulation signs present. The larger p is, the more unreliable the female’s ovulation signal in signaling ovulation and her fertility. Similarly, the closer p is to zero, the more honest is the female’s ovulation signal.

Recall that $x_i(d)$ denotes the amount of visual ovulation signs present on each day d of the female cycle, with $x_i(d)$ defined by the function in the Methods section of the main text. This function is determined by m_i (ovulation magnitude) and ℓ_i (ovulation length). Note the exact function $x_i(d)$ is chosen such that (i) m_i will denote the peak of the curve, (ii) ℓ_i will denote the width of the non-zero portion of the curve, (iii) m_i, ℓ_i remain independent of each other, and (iv) m_i, ℓ_i can both decrease all the way to zero. Example values of p are displayed in Fig. 3.3. Fertility probabilities are denoted by the light blue shading, while visible ovulation signs on each day of

the female's cycle are depicted by the red shaded bars. Note the shapes of each visible ovulation signs and fertility probability remain the same ($m_i = 1$, $\ell_i = 7$, and $C = 7$), while p (the difference between a female's day of peak visible ovulation signs and her day of peak fertility). Note Chapters 1 and 2 of this dissertation assume $p = 0$.

7.1.2. Determining Male-Female Mate Pairs

We assume that, for every day of the cycle, males of higher GC will preferentially mate with females with more ovulation signs visible. We also assume each individual to mate at most once on each day of the cycle. Females will only mate on days on which they are receptive, and males will only mate if there is a receptive female with whom to mate. Parameters ϵ_m and ϵ_f control the amount of stochasticity in the process of mating pair formation. With $\epsilon_m, \epsilon_f \rightarrow \infty$, matings are determined completely randomly.

7.1.3. Calculating Probabilities of Paternity

To calculate the probability of paternity for each male, first the fertility of each female with which that male has mated is summed up for every day a particular male mates with her. These values are normalized across males for each female in the group, meaning a male who mates with the female on a day where she has a higher fertility probability will have a higher paternity probability. The actual father for that female's offspring is then determined randomly, proportional to each male's probability of paternity for her offspring.

Note these *actual* probabilities of paternity are different from the $r_{i,j}$, *perceived* probabilities of paternity, as detailed in the Methods section of the main text. We make this distinction because males will not know any female's probability of fertility at the time he mates with her; males will only know how many visible ovulation signs she had present. Separating these quantities allows actual probabilities of paternity (which males do not know) to be calculated using females' probabilities of fertility, and perceived probabilities of paternity (which males *do* know) to be calculated using females' visible ovulation signs.

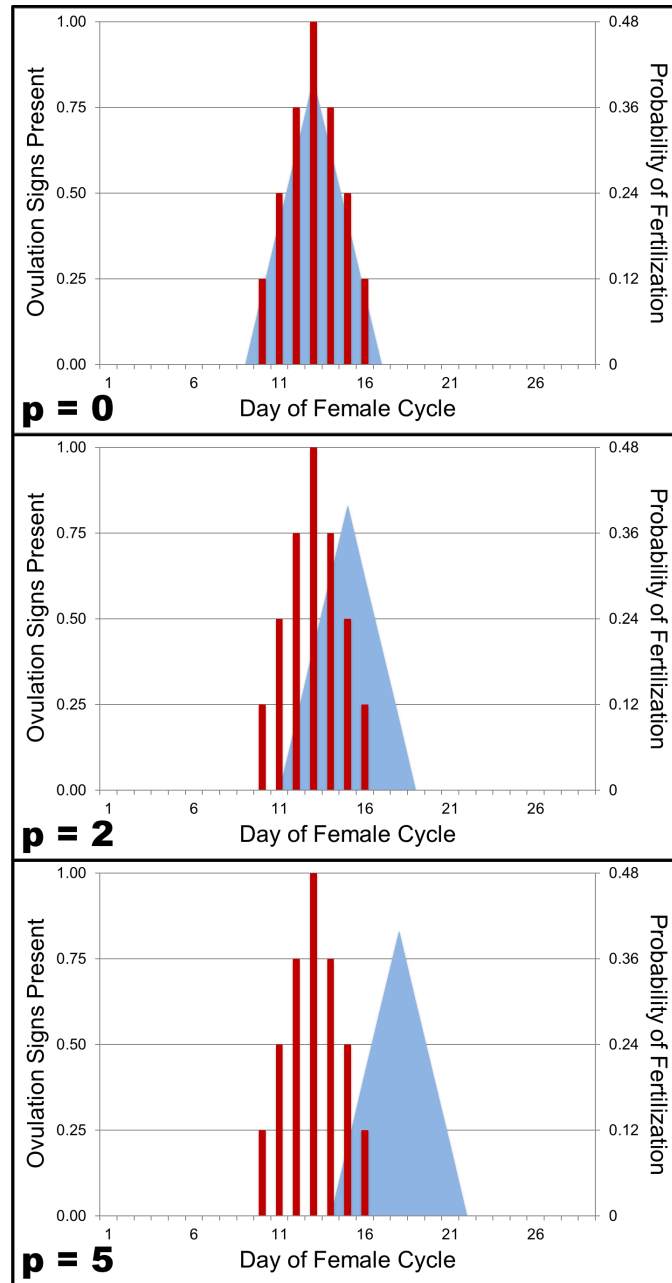


Figure 3.3: Sample values of p ($p = 0, 2, 5$, from top to bottom). Each graph includes visible ovulation signs present (with $m_i = 1, \ell_i = 7$) via the red bars and fertilization probabilities (with $C = 7$) via the light blue shaded region. In the top graph, we see that a female's day of ovulation will also be her day of having peak ovulation signs visible. In the bottom two graphs, we instead see that a female's day of ovulation can occur on a day where she has a lower probability of fertilization, or even a zero percent chance of fertilization.

7.1.4. Infanticide

We define the exponential function of male effectiveness to be:

$$f(j) = \frac{e^{-\omega j}}{\sum_{k=1}^N e^{-\omega k}}, \quad (40)$$

with parameter $\omega > 0$ controlling the amount of disparity among males in their corresponding effectiveness within a group. Recall a larger value of ω indicates more disparity among males.

7.1.5. Female Fitness Functions

The female fitness function for female i in the model is as follows:

$$w_i = w_0 + (1 - \eta)y_{m,i} + \frac{\eta}{D} \sum_{\text{all mates } j} y_{p,j} + \sum_{j=1}^N [f(j)g(r_{i,j})] - c\bar{x}_i - c_r \frac{r_i}{D}, \quad (41)$$

where recall $y_{m,i}$ is the actual benefit from female-female competition (i.e. the GC of the specific male who won the contest of paternity for that particular female's offspring), $p_{i,j}$ perceived paternity probability, $\bar{x}_i = \frac{1}{D} \sum_{d=1}^D x_i(d)$ the female's average visual ovulation signs present, η the weighting on female fitness between GC and NGC, and $y_{p,j}$ the NGC coming from each male j a female mated with throughout her cycle.

Notice for the case when $\alpha = 0$ (i.e. without the effects of infanticide), the fitness function above collapses into:

$$w_i = w_0 + (1 - \eta)y_{m,i} + \frac{\eta}{D} \sum_{\text{all mates } j} y_{p,j} - c\bar{x}_i - c_r \frac{r_i}{D}. \quad (42)$$

Note in this $\alpha = 0$ (no infanticide) case, parameters β , κ , τ , and ω all become unnecessary.

7.2. List of All Parameters

Parameters used in the model are outlined in Table 3.1. For each parameter, one value listed implies the parameter remained fixed throughout all simulations, and multiple values indicates each of those parameter values was tested in all combinations with all other parameters.

Table 3.1: List of All Parameters

Parameter Name	Parameter Description
$G = 400$	Number of groups
$T = 100000$	Time (number of generations)
$D = 29$	Number of days in each female's cycle
$C = 7$	Number of days each female has non-zero probability of fertilization
$C_v = \{0.125, 0.25, 0.375, 0.5, 0.375, 0.25, 0.125\}$	Probability of fertilization on each of those $C = 7$ days
$w_0 = 2$	Baseline female fitness
$N = 4, 8, 16$	Number of males and number of females in each group
$b = 0.1, 0.2, 0.4$	Variation in male quality
$c = 0.1, 0.2, 0.4$	Cost of having ovulation signs present
$c_r = 0.1, 0.2, 0.4$	Cost of being receptive to mating
$\eta = 0.1, 0.2, 0.4$	Weighting of GC vs. NGC
$\hat{y} = 1$	Mean male genetic quality
$\epsilon_m = 0, 0.25, 0.5$	Parameter determining the amount of reproductive stochasticity among males
$\epsilon_f = 0, 0.01, 0.05$	Parameter determining the amount of reproductive stochasticity among females
$\gamma = 1$	Parameter determining the shape of the function for the amount of visual ovulation signs present
$\alpha = 0, 0.2, 0.6, 1$	Parameter controlling the maximum benefit of protecting against infanticide (note $\alpha = 0$ corresponds to the case with NO infanticide)
$\beta = 1.25, 1.5, 1.75$	Parameter controlling the relative maximum cost of infanticide in the paternity probability effect function
$\tau = 2$	Exponent in the paternity probability effect function g
$\omega = 1$	Parameter in the male effectiveness function $f(j)$
$\kappa = 0, 0.5, 1$	Parameter determining the weight males put on the amount of visual ovulation signs in their paternity estimate
$\nu = 1$	Migration rate (between zero and one)
$\mu = 0.001$	Probability of mutation (per gene per generation)

7.3. ANOVA Results

The results of our ANOVA tests were discussed in the main text. The following tables (Tables 3.2, 3.3, 3.4) give the detailed results of these tests.

Table 3.2: Effects of N, b, ϵ_m, η on receptivity length R , ovulation signs magnitude M and length L , and signal reliability P

	R	M	L	P
N	-0.022	0.030	0	0
b	-0.015	0.570	0.167	-0.210
ϵ_m	0	-0.083	-0.033	0.067
η	0.859	0.134	0.411	0.037
error	0.099	0.183	0.385	0.661

Table 3.3: Effects of $\alpha, \beta, \kappa, \eta$ on receptivity length R , ovulation signs magnitude M and length L , and signal reliability P

	R	M	L	P
α	0.018	0.318	0.165	0
β	0	0.022	0	0
κ	- 0.310	0.429	- 0.489	0.150
η	0.441	- 0.017	0.067	- 0.096
error	0.231	0.214	0.271	0.714

Table 3.4: Effects of b, c, c_r, η on receptivity length R , ovulation signs magnitude M and length L , and signal reliability P

	R	M	L	P
b	- 0.021	0.344	0.140	- 0.221
c	0	- 0.179	0	0
c_r	- 0.491	- 0.114	- 0.278	0
η	0.250	0	0.081	0
error	0.237	0.357	0.484	0.723

7.4. Supplemental Figures

The following figures are all supplemental to the text. Fig. 3.4 illustrates the effects of parameters N (group size), b (benefit of male genetic quality), ϵ_m (male reproductive stochasticity), ϵ_f (female reproductive stochasticity), and η (weight put on genetic vs. non-genetic male benefits) on signal reliability (p), when ovulation signs and receptivity are fixed in the population ($m = 0.9, \ell = 4, r = 29$ for all females). Fig. 3.5 illustrates the effects of parameters α (maximum benefit from protection against infanticide), β (proportional to the maximum cost of infanticide), κ (weight males put on visible ovulation signs when estimating their paternity probabilities), and η (weight put on genetic vs. non-genetic male benefits) on ovulation signs magnitude (m), ovulation signs length (ℓ), receptivity length (r), and signal reliability (p). Fig. 3.6 illustrates the effects of parameters N (group size), b (benefit of male genetic quality), c (costs of having visible ovulation signs), and η (weight put on genetic vs. non-genetic male benefits) on ovulation signs magnitude (m), ovulation signs length (ℓ), and signal reliability (p). Fig. 3.7 illustrates the effects of parameters N (group size), ϵ_m (male reproductive stochasticity), ϵ_f (female reproductive stochasticity), and η (weight put on genetic vs. non-genetic male benefits) on ovulation signs magnitude (m), ovulation signs length (ℓ), and signal reliability (p). Finally, Fig. 3.8 illustrates the effects of parameters α (maximum

benefit from protection against infanticide), β (proportional to the maximum cost of infanticide), κ (weight males put on visible ovulation signs when estimating their paternity probabilities), and η (weight put on genetic vs. non-genetic male benefits) on ovulation signs magnitude (m), ovulation signs length (ℓ), and signal reliability (p).

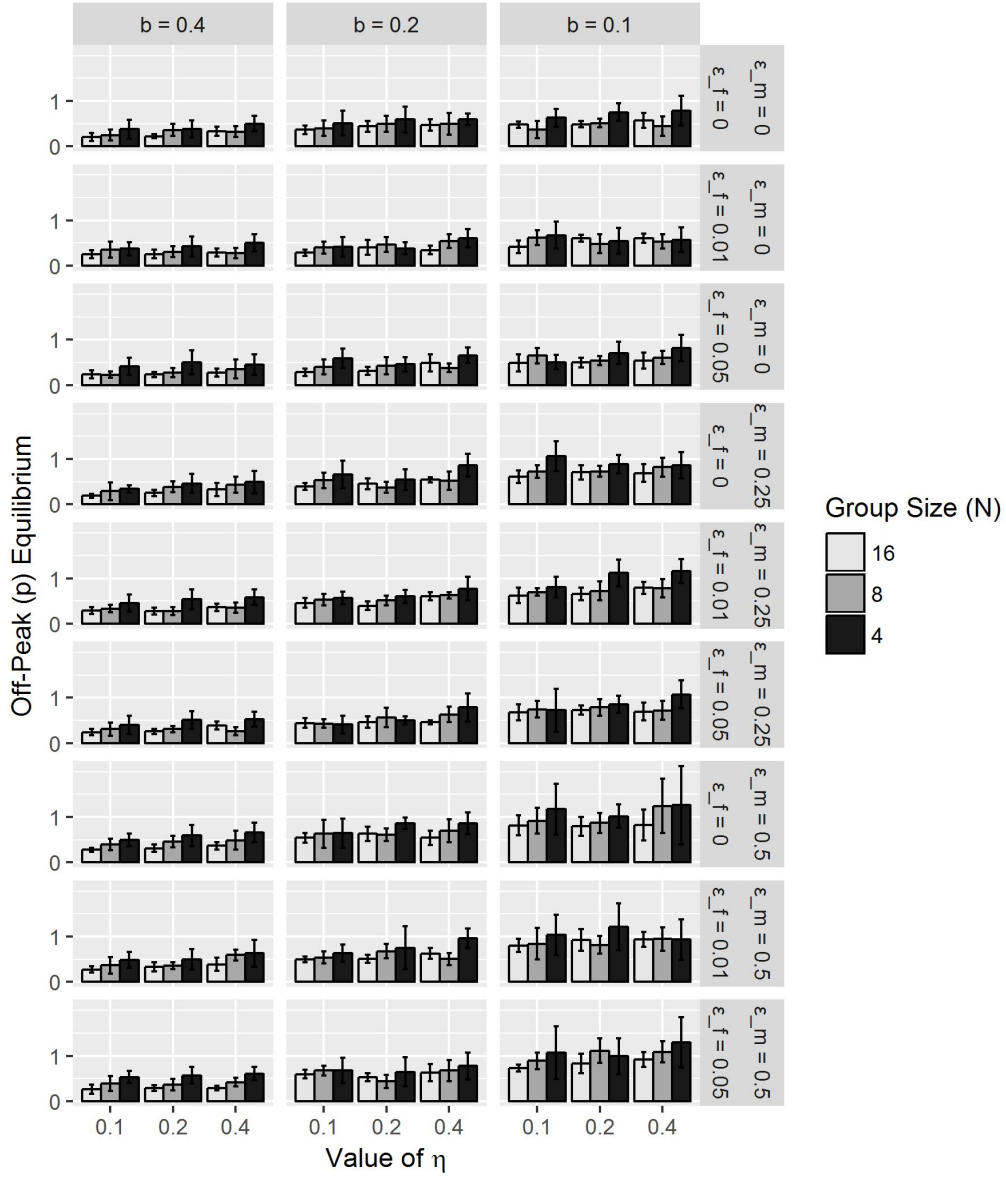


Figure 3.4: The effects of parameters N (group size), b (benefit of male genetic quality), ϵ_m (male reproductive stochasticity), ϵ_f (female reproductive stochasticity), and η (weight put on genetic vs. non-genetic male benefits) on signal reliability (p), when ovulation signs and receptivity are fixed in the population ($m = 0.9, \ell = 4, r = 29$ for all females). Equilibria are obtained by averaging over 7 initial condition runs (with standard deviation indicated by error bars). All other parameters were held constant: $\alpha = 0, c = 0.2, c_r = 0.2$.

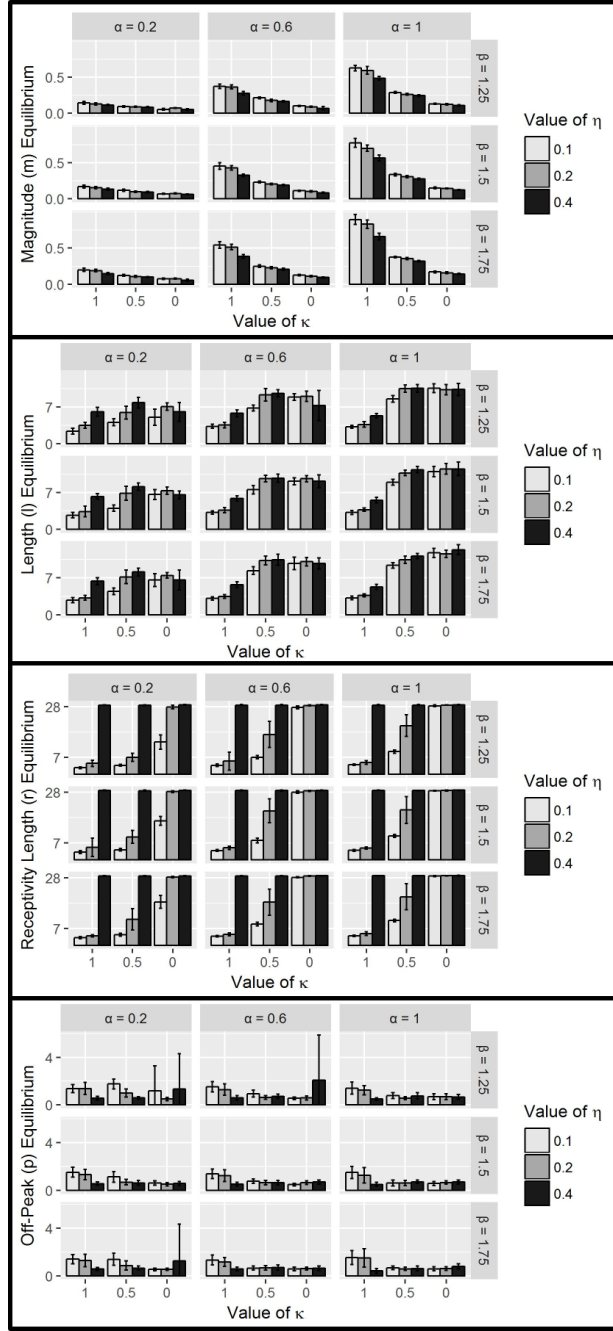


Figure 3.5: The effects of parameters α (maximum benefit from protection against infanticide), β (proportional to the maximum cost of infanticide), κ (weight males put on visible ovulation signs when estimating their paternity probabilities), and η (weight put on genetic vs. non-genetic male benefits) on ovulation signs magnitude (m), ovulation signs length (ℓ), receptivity length (r), and signal reliability (p). Equilibria are obtained by averaging over 16 initial condition runs (with standard deviation indicated by error bars). All other parameters were held constant: $N = 8, b = 0.2, c = 0.2, c_r = 0.2, \epsilon_m = 0.25, \epsilon_f = 0.01$.

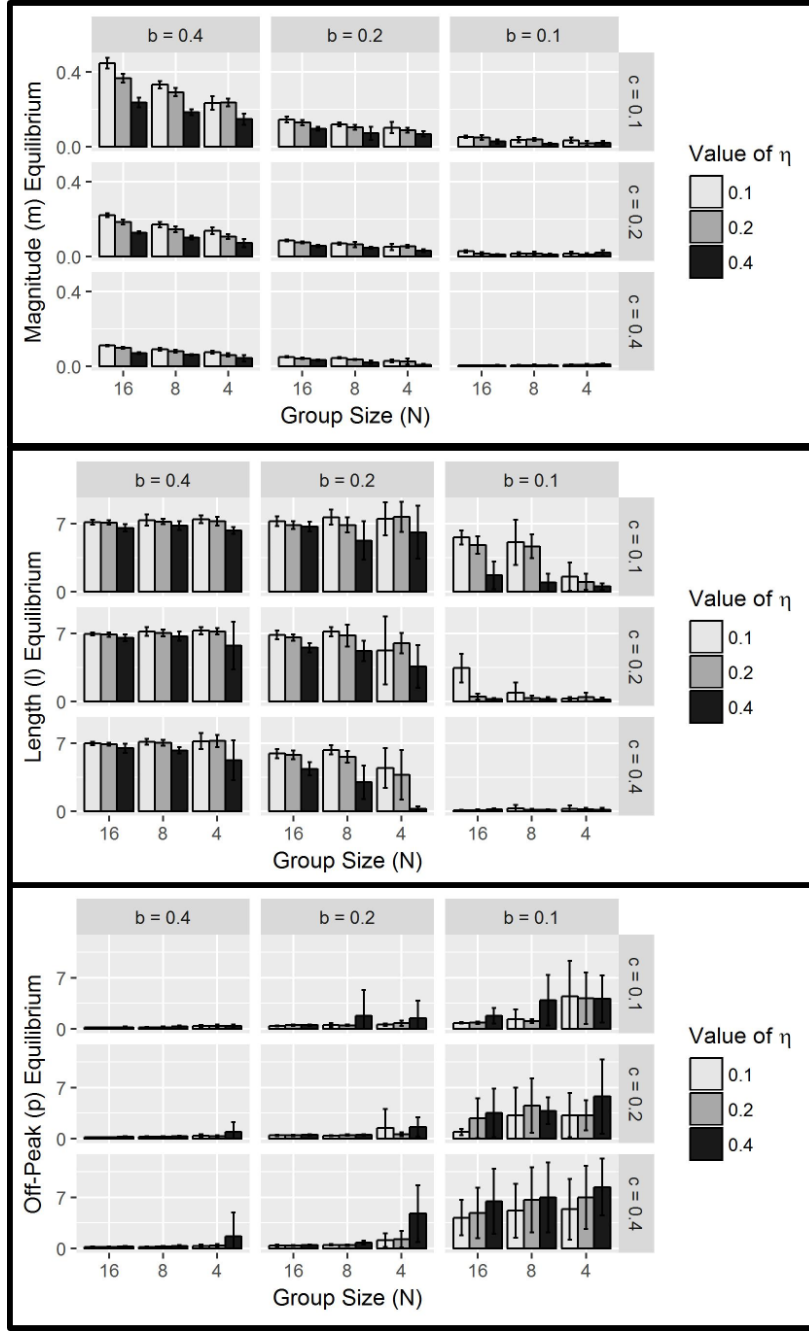


Figure 3.6: The effects of parameters N (group size), b (benefit of male genetic quality), c (costs of having visible ovulation signs), and η (weight put on genetic vs. non-genetic male benefits) on ovulation signs magnitude (m), ovulation signs length (ℓ), and signal reliability (p). Equilibria are obtained by averaging over 8 initial condition runs (with standard deviation indicated by error bars). All other parameters were held constant: $\alpha = 0$, $c_r = 0.2$, $\epsilon_m = 0.25$, $\epsilon_f = 0.01$.

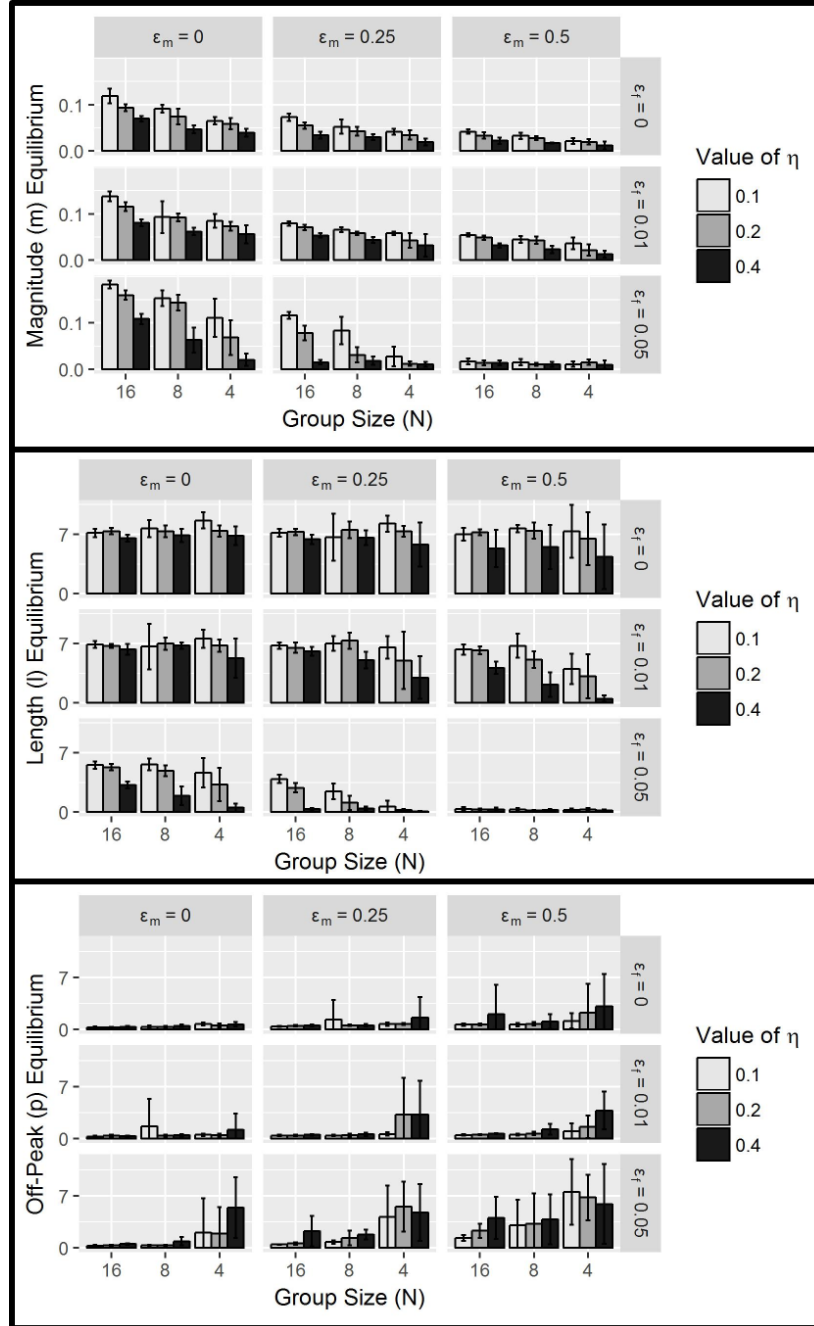


Figure 3.7: The effects of parameters N (group size), ϵ_m (male reproductive stochasticity), ϵ_f (female reproductive stochasticity), and η (weight put on genetic vs. non-genetic male benefits) on ovulation signs magnitude (m), ovulation signs length (ℓ), and signal reliability (p). Equilibria are obtained by averaging over 8 initial condition runs (with standard deviation indicated by error bars). All other parameters were held constant: $\alpha = 0$, $b = 0.2$, $c = 0.2$, $c_r = 0.2$.

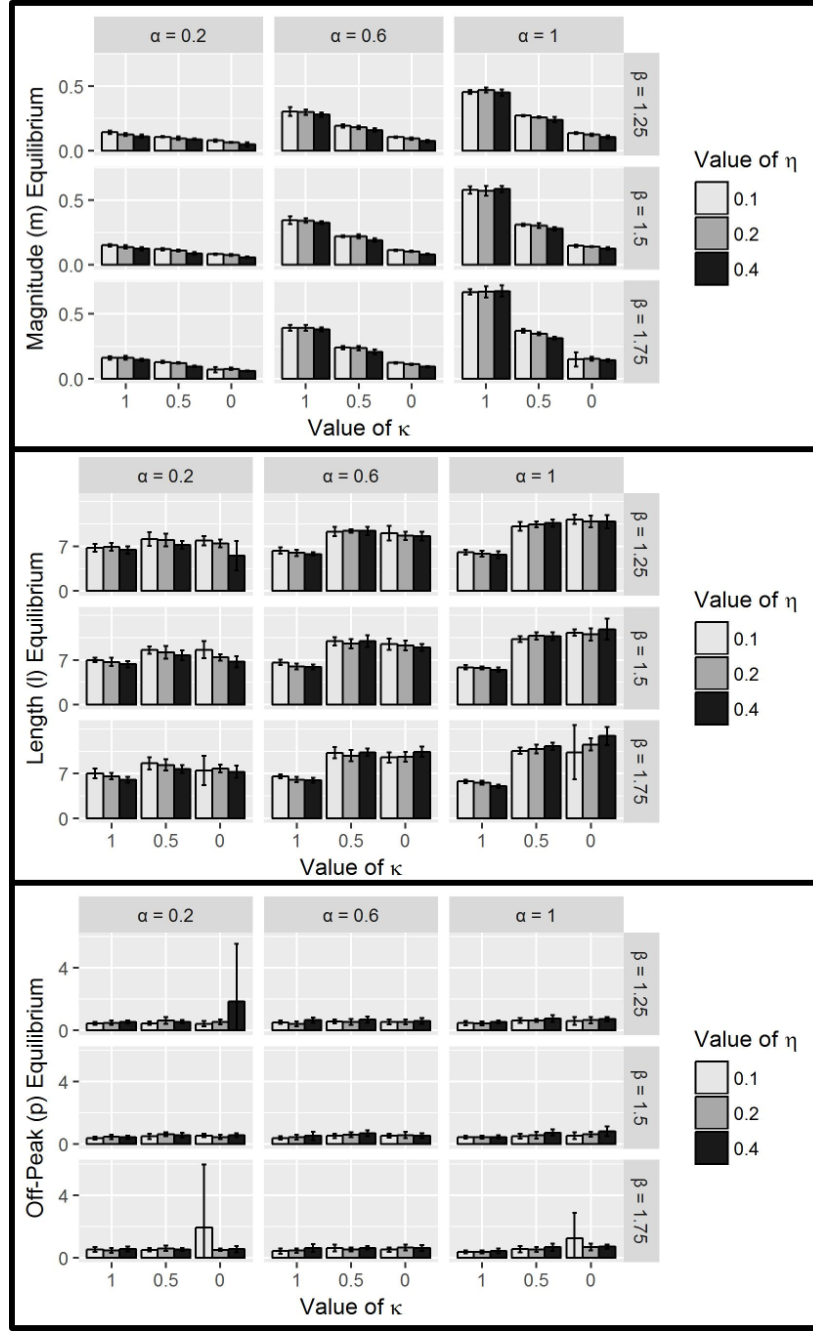


Figure 3.8: The effects of parameters α (maximum benefit from protection against infanticide), β (proportional to the maximum cost of infanticide), κ (weight males put on visible ovulation signs when estimating their paternity probabilities), and η (weight put on genetic vs. non-genetic male benefits) on ovulation signs magnitude (m), ovulation signs length (ℓ), and signal reliability (p). Equilibria are obtained by averaging over 8 initial condition runs (with standard deviation indicated by error bars). All other parameters were held constant: $N = 8, b = 0.2, c = 0.2, c_r = 0.2, \epsilon_m = 0.25, \epsilon_f = 0.01$.

Chapter IV

On the evolution of long-term pair-bonding in humans

Publication Statement: A version of this chapter was originally published by its co-authors, Kelly Rooker and Sergey Gavrilets. 2016. “Evolution of long-term pair-bonding in humans.” *Encyclopedia of Evolutionary Psychological Science*. The text of this chapter was principally composed by K. Rooker.

On the evolution of long-term pair-bonding in humans

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Abstract

Long-term pair-bonding occurs when one breeding male and one breeding female share a common territory, associating with each other for more than just one breeding season regardless of whether or not they currently have any offspring together. While such a scenario occurs in many different species - in particular among birds - it also occurs among several mammals, including among primates. There have long been questions raised as to how long-term pair-bonding evolved independently so many different times, in particular among the primates. For humans, there is not even consensus on from where long-term pair-bonding originated: from promiscuous groups, groups with harems, multi-level societies, or strict monogamy. The various hypotheses which have been historically proposed to explain the evolution of long-term pair-bonding in humans are here grouped into 4 main categories: ecological, protection, provisioning, and conflict hypotheses. While we do not model these here nor how they may relate to females having visible ovulation signs or concealed ovulation, it is our hope that such work will be conducted in the future.

1. Introduction

All three previous chapters deal with some aspect of visual ovulation signals - and hence concealed ovulation as well. One of the oft-proposed hypotheses as to why concealed ovulation evolved has to do with long-term pair-bonding. Long-term pair-bonding occurs when one breeding male and one breeding female share a common territory, associating with each other for more than just one breeding season regardless of whether or not they currently have any offspring together. In humans, long-term pair-bonding can occur via monogamous or polygamous relationships.

While the evolution of each concealed ovulation and long-term pair-bonding have both been labeled “mysteries” in humans, many researchers also think they may have evolved together (Thornhill and Gangestad, 2008). While I do not model that possible coevolution in this dissertation, this chapter will set the problem up as potential related future work. Building off our base model presented in Chapter 1, we could then add the evolution of long-term pair-bonding in order to investigate some of the questions posed by this chapter. It is our hope for this work to be done in the future, and we think such an addition to the literature would be of great interest to psychologists, anthropologists, evolutionary biologists, etc.

1.1. Background

Humans have previously been called the “uniquely unique species” (Alexander, 1990). Despite there being many different factors contributing to this uniqueness (e.g. large brain, language, cumulative culture), one of the most important is humans having a multi-male, multi-female social organization and a long-term pair-bonding mating system (Flinn et al., 2005; Geary and Flinn, 2001; Hill et al., 2011). Most species living in groups composed of multiple males and multiple females are promiscuous, while most pair-bonded species live in groups comprised of only one male and one female. There are no primates other than humans where multiple reproductive pairs live together (de Waal and Gavrilets, 2013). Nuclear and extended families, as well as parental investment in offspring, are characteristics found in all human societies (Geary and Flinn, 2001); such characteristics clearly set us apart from our African ape ancestors (de Waal and Gavrilets, 2013; Flinn et al., 2005). The question then is, how could humans have evolved such a unique

combination of social organization and mating system?

In fact, long-term pair-bonding in humans has always been considered an evolutionary mystery. Why is this practice so common in human societies around the world? How did long-term pair-bonding first evolve in humans? What were the evolutionary pressures making it beneficial to humans? Although these questions have all been asked repeatedly for decades, even centuries, there has yet to be any real consensus on their answers. Still, much progress has been made.

1.2. Defining monogamy

Monogamy and pair-bonding often get used interchangeably, but “monogamy” can mean many different things. For instance, *cultural monogamy* refers to human institutions such as marriage. *Sexual monogamy* is in reference only to sexual relations; one male and one female will mate exclusively with one another, but have no or few interactions outside of that sexual intercourse. Similarly, *genetic monogamy* is when all offspring produced by one male or female in that pair have that same mother and father. Rates of genetic monogamy can be tested in the field by looking at the genetic make-up of all offspring produced (Reichard and Boesch, 2003).

Finally, there is *social monogamy*, or what is more commonly called “*pair-bonding*.” In social monogamy, one male and one female exclusively assort with each other. This assortment is not just in terms of copulations or the genetics of their offspring, but also in terms of with whom they live, where they sleep, and most generally where they spend their time (Reichard and Boesch, 2003). In other words, under social monogamy one breeding male and one breeding female will share a common territory, associating with each other for more than just one breeding season, regardless of whether or not they currently have any offspring together (Lukas and Clutton-Brock, 2013).

It should be noted that many researchers do not restrict the word “pair-bonding” to involving *only* one male and one female. Rather, the term is often used more broadly to describe any lasting reproductive relations between particular males and females. For example, in polygyny where one male is having such lasting reproductive relations with multiple females, some would say that male is just having multiple distinct pair-bonds, occurring simultaneously (Reichard and Boesch, 2003).

Using this terminology, humans as a whole are very much considered having long-term pair-bonds since every known human society has practiced either polygamy or monogamy (and hence

long-term pair-bonding) (Chapais, 2008). Considering only the raw number of human societies throughout the world (vs. also the populations of each of these societies), only about 17% of such societies practice social monogamy. The vast majority of the rest (>80%) practice polygyny. Even among the polygynous societies though, only a small minority of the men in each are actually polygynous; most males lack sufficient resources needed to support multiple female mates (Reichard and Boesch, 2003).

True genetic monogamy is rare in humans, even in the socially monogamous cases (Reichard and Boesch, 2003). However, despite the widespread prevalence of extra-pair matings, there still has yet to be a human society with sexual promiscuity as the main form of mating system (Chapais, 2008). Hence, for the rest of this article, “long-term pair-bonding” will be synonymous with “social monogamy”, thus including both monogamy and polygamy. For cases where one male and one female *do* remain exclusively with each other, this article instead refers to as *strict monogamy*.

In addition to all these different types of monogamy, the word “monogamy” can also imply differences in duration. *Long-term monogamy* implies the one male and one female which form a pair will remain together across many breeding seasons. *Life-long monogamy* is then a different term used to describe when a male and female’s long-term monogamy in fact lasts for the rest of each of their lives. Conversely, *short-term monogamy* refers to monogamy that typically lasts for only one breeding season or year; one male and one female will be monogamous to each other, but only within that limited amount of time. Cases where pairs practice short-term monogamy throughout their lives, but in each instance of short-term monogamy have a different partner, are instead referred to as practicing *serial monogamy* (Reichard and Boesch, 2003).

For example, many species of birds will be monogamous throughout each breeding season (or other period of time), but then each individual will have a different mate come next breeding season (Reichard and Boesch, 2003). This means such birds are monogamous *within* breeding seasons, but not monogamous *between* breeding seasons (i.e. serial monogamy). In addition, human societies where divorce is common, as well as remarriage for both men and women following divorce, have mating systems resembling serial monogamy (Reichard and Boesch, 2003).

Monogamy from a female’s perspective often makes a lot of sense. In mammal reproduction,

only females must go through the costly periods of internal gestation and lactation (Opie et al., 2013). Not only are such activities quite energetically expensive for females, they make predation, death, and other injury to the female more likely. In addition, due to relatively long gestation and lactation periods, female mammals are also much more limited than males are in the quantity of offspring they are able to have in a lifetime. While males are limited only in the number of copulations they are able to obtain with fertile females, females have these other main constraints of resources, time, and energy (Kappeler, 2013).

In a monogamous mating system, having a male remain with/around a female could help the female in any number of ways, be it protection of her and/or her offspring/resources, help in securing meat or other valuable resources, and/or help in caring for any offspring produced from the union. All of these would help loosen the female's main constraint (time) either by allowing a female's interbirth interval to be shortened (e.g. by a male helping with parental care of the older offspring), improving her health which leads to longer times of fertility (e.g. by provisioning the female with valuable foods), and/or lengthening her life and hence her fertile period (e.g. by protecting her from other males or predators) (Kappeler, 2013).

On the other hand, the puzzling part when considering the evolution of long-term pair-bonding is why *males* would ever play their role in a socially monogamous mating system. In mammals, males have much greater potential for producing more offspring throughout their lives than females (Kappeler, 2013), due to their aforementioned traits of internal gestation and lactation (Opie et al., 2013). This means that males mating with only one female throughout their lives are sacrificing all that "extra" reproductive potential. Thus for long-term pair-bonding to evolve, there would have to be some fitness compensation present to make up for that loss in male reproductive potential (Kappeler, 2013). The rest of this article is primarily concerned with considering the many possible forms of such "fitness compensation".

1.3. Phylogeny of social monogamy

Although certainly not prevalent, long-term pair-bonding is widespread throughout the animal kingdom. Long-term pair-bonding is most notably found in birds and mammals. Among mammals, about 3% of species practice long-term pair-bonding (Kleiman, 1977). Even the newer, upper

estimate of 9% of mammals practicing social monogamy (Lukas and Clutton-Brock, 2013) pales in comparison to the 90% of birds estimated to practice social monogamy. However, these 3-9% of mammal species are found in many different clades of mammals, and long-term pair-bonding is believed to have evolved independently upwards of *sixty* different times in mammals (Lukas and Clutton-Brock, 2013).

Such pair-bonding in mammals may appear to be strictly behavioral, but neurophysiology has also been shown to play a role. Certain neuropeptide pathways (e.g. oxytocin, arginine vasopressin, and prolactin) have been shown to have been co-opted to form pair-bonding reward pathways in several species of monogamous mammals, including primates (Lovejoy, 2009). Compared to birds, social and genetic monogamy in mammals are tightly correlated, meaning mammals' rates of extra-pair copulations are relatively low (Lukas and Clutton-Brock, 2013).

Rates of long-term pair-bonding of course vary significantly among mammal species. While some mammalian Orders contain zero socially monogamous species, about 29% of primate species are socially monogamous (Lukas and Clutton-Brock, 2013). In addition, primates are unique among mammals in that social monogamy has evolved independently in every major primate clade, and there are relatively few transitions away from monogamy once it has evolved (Opie et al., 2013).

Among just anthropoid primates, long-term pair-bonding is believed to have evolved independently seven different times, three in *Platyrrhini* (New World Monkeys) and four in *Catarrhini* (apes and Old World Monkeys). Indeed, socially monogamous species among anthropoid primates today include species as varied as New World Monkeys (e.g. marmosets, tamarins), Old World Monkeys (e.g. De Brazza's Monkeys, Sumatran surilis), and apes (e.g. gibbons, humans) (Sillen-Tullberg and Moller, 1993). Despite social monogamy not being ubiquitous among primates, its widespread prevalence indicates there must be some significant benefit to species practicing social monogamy. Although this article focuses on humans, many of the hypotheses and ideas presented below could similarly apply to many of these other primate species as well.

1.4. *Outline*

It is our aim here to first discuss the transition *to* long-term pair-bonding: what were human societies like prior to long-term pair-bonding becoming the norm? We will then discuss the many

hypotheses which have been given to explain the evolution of long-term pair-bonding in humans. We will go into both empirical and theoretical work as it pertains to each of these hypotheses. Finally, we will discuss other work which takes a broader look at the question of long-term pair-bonding in humans.

2. Pathways for the transition to monogamy in groups

Part of the debate surrounding the evolution of long-term pair-bonding in humans is over the ancestral social organization and mating system of genus *Homo*. Were human ancestors monogamous? Or promiscuous? Did they live solitary? In one-male groups? Or in multi-male, multi-female groups? These ancestral-state questions are still very much debated, with many researchers having wildly different, and often directly contradicting, views (Duda and Zrzavy, 2013).

Humans, and many other primate species, are group-living. Such groups of individuals come in various sizes, but all with their associated benefits and costs (Hill et al., 2011). For instance, groups afford members protection against both predators and other groups. Such protection could come from group members alarm-calling, alerting other group members of some impending danger, or in the form of an alliance against a predator or invading group. Groups can also facilitate resource-sharing, whether it be with meat, other valuable foods, or other resources. Individuals in groups are better guaranteed a steady supply of nourishment, with food-sharing in groups especially helping with food/resource scarcity in the event of a drought or other severe ecological condition. In the case of species with alloparenting, group-living can help with child-rearing and allowing females to have more offspring than they would be able to otherwise. Finally, group-living may also help with acquiring mates. By living in groups with at least one male and one female, potential mates are always nearby and mating attempts likely easier to facilitate (Flinn et al., 2005).

On the other hand, group-living can also come with potential costs to all group members, primarily from ecological competition (for resources, mates, etc.) (Hill et al., 2011). Living in groups can result in conflict between group members. Such conflict can be energetically costly to those group members, even resulting in injury or death if fighting is the outcome. Living in close proximity to others can increase theft of resources, food, mates, etc., making group members

expend more energy and time guarding such resources, and again potentially fighting over them. Finally, visibility by a predator, disease, and other parasites become more prevalent when living in the higher population densities that come with groups. Such risk would be absorbed by all group members, not only potentially resulting in increased susceptibility to predation, lasting disability, and death, but also potentially in loss of resources and other vulnerabilities (Flinn et al., 2005).

Humans' closest living relative, the chimpanzee, is certainly a group-living species. Chimpanzees live in highly promiscuous multi-male, multi-female societies. Most evidence does not point to monogamy being the ancestral state of the chimpanzee-human last common ancestor. Rather, their ancestral state was likely some type of promiscuity, with monogamy in humans being a derived trait. However, this broad framework still leaves much room for debate in terms of what that ancestral human social organization, and resulting mating system, specifically looked like (Chapais, 2013). The following are the four main pathways which have been proposed for such an ancestral state.

2.1. Promiscuity

First, the *Pan*-human last common ancestor could have been very much like *Pan* (chimpanzees and bonobos). In this case, the ancestral state for *Homo* would be individuals living in fully promiscuous (i.e. with no stable mating bonds) multi-male, multi-female groups. From this backdrop, humans would have retained this multi-male, multi-female social organization, but then evolved to a socially monogamous mating system from the promiscuous *Pan*-like one (Chapais, 2013).

2.2. Harems

An often-favored alternative to the above is if the *Pan*-human last common ancestor had a harem-style mating system, similar to that of gorillas. In this case, the social organization would have consisted of groups made up of one male with many females (his "harem"), and then lone roaming males outside of any group. The one male in the harem would mate with all of the females in the harem and also provide protection/guard them, while the roaming males got few, if any, copulation opportunities. From this backdrop, humans would have re-organized into a social organization consisting of multiple males and multiple females making up any one group, while still keeping their long-term pair-bonding structure (Chapais, 2013). Evidence for this pathway

includes humans' *Australopithicines* ancestors having large body size sexual dimorphism (larger than either chimpanzees, modern-day humans, or later human ancestors), which is indicative of fierce one-on-one male competition, as seen in gorillas (Geary and Flinn, 2001). In addition, one mathematical modeling approach found this pathway to be the most likely one leading to humans (Nakahashi and Horiuchi, 2012).

2.3. *Multi-level societies*

A third alternative discussed is the idea of multi-level societies. Here, the chimpanzee-human last common ancestor would have lived with a social organization and mating system similar to hamadryas baboons. Under this framework, males still have harems (i.e. groups made up of one male and multiple females, all of whom that one male mates with and protects) but then multiple harems live together in larger groups, often referred to as "clans". Under this backdrop, these larger groups would already be practicing long-term pair-bonding. However, from this humans could have even evolved strict (one-male, one-female) monogamy, since "clans" could contain "harems" of one male and precisely one female. If the size of the male harem was reduced to only female per male, strict monogamy would be the effective result (Chapais, 2013).

2.4. *Strict monogamy*

Finally, although considered unlikely by many, it is also possible that strict monogamy was the ancestral mating system for humans. Similar to gibbons and other primates with such monogamy, monogamous pairs would have been spaced out geographically and not had any kind of group social organization. What evolved from this backdrop would then be the group social organization: monogamous pairs moving closer to each other, ultimately forming multi-male, multi-female groups while still retaining their monogamous mating system. Again, although this pathway is certainly a logical transition, there is little evidence supporting the chimpanzee-human last common ancestor being strictly monogamous (Chapais, 2013; Sillen-Tullberg and Moller, 1993).

3. Hypotheses for the evolution of social monogamy

Regardless of from where exactly it came, there is no doubt that long-term pair-bonding evolved at some point in humans' past. Similar to the debate over the ancestral state of *Homo*, there is

also much debate over how long-term pair-bonding first evolved in humans. Again, there have been many different hypotheses proposed, some directly contradicting each other, and relatively little headway made in finding concrete answers as to which hypothesis is the most likely. Despite no consensus after decades of research, progress has still been made. Note some hypotheses presented below are unique to humans while others are more widely applicable to many primates, or even mammals in general. Some hypotheses are also highly dependent on one or more of the pathways discussed above, while other hypotheses could still be possible regardless of the specific pathway from which early humans evolved. Also note that some hypotheses below only work with regards to strict monogamy (these will be noted), while most do not specify the type of long-term pair-bonding needed.

In general, all the hypotheses discussed below can be grouped into four main categories. The first is ecological. This hypothesis has gained much support for the evolution of social monogamy among non-human mammals, but is widely discredited as being a factor in the evolution of social monogamy in humans. The second group of hypotheses include those having to do with protection, of either the offspring, the mate, or one's self. The third group of hypotheses relates to food: provisioning of the mate, the offspring, or in cooperation. Finally, the fourth group of hypotheses involves conflict, either strictly between males fighting over mates, group-related, or among siblings produced by the same male. It should be noted that most of these hypotheses are not mutually exclusive, but rather two or more could have simultaneously contributed selection pressure to the evolution of long-term pair-bonding in humans.

3.1. Ecological hypothesis

The first hypothesis discussed here is the “ecological” hypothesis (Komers and Brotherton, 1997). Females are assumed to be solitary and relatively spaced out geographically (due to ecological competition), while males are roaming, looking for mates. However, because of the females' geographic spacing, it may become increasingly lucrative for a male to stick to just one female's range. Doing so will save males energy resources expended in searching for females, and also better guarantee males a mate. If they instead wandered, males would have to travel long distances between females with no guarantee of, first, even finding another female, and, second, her desiring

to mate with said male once found (Komers and Brotherton, 1997).

A recent study found the most-likely ancestral state for all non-human mammals to be solitary females and roaming males with overlapping ranges (Lukas and Clutton-Brock, 2013). They then found social monogamy to have evolved in non-human mammals under conditions involving intense female-female competition, low female density, and where breeding females were intolerant of sharing their ranges. Under these conditions, males would likely be unable to defend access to more than one female. Among all non-human mammals, socially monogamous species today do in fact live at significantly lower densities than solitary species, even after controlling for body size differences and despite the fact that both socially monogamous and solitary species share similar home-range sizes (Lukas and Clutton-Brock, 2013).

Although considered by many to be a favored hypothesis for the evolution of social monogamy in non-human mammals, this hypothesis is much less accepted with regards to human evolution since humans did not likely come from solitary ancestors. Each of the three pathways which assume humans had non-strictly-monogamous ancestors (i.e. three of the four discussed above) all center on early humans living in groups of more than two individuals (Kappeler, 2013). However, it is certainly possible that changes in early human dietary patterns (even within groups) lowered female densities, making it much harder for one male to guard multiple females. In this way, a transition from polygyny to strict monogamy could still have been facilitated in humans under the ecological hypothesis (Lukas and Clutton-Brock, 2013).

3.2. Protection hypotheses

The following three hypotheses all deal with protection as the main selection pressure on early humans for the evolution of long-term pair-bonding.

The first hypothesis under this category deals with protection of one's offspring, namely protecting them against the risk of infanticide. Infanticide, the practice of killing an infant not one's own prior to it being weaned, has been widely documented across primates (van Schaik and Dunbar, 1990). In most instances, infanticide is committed by a new alpha male who has just taken over a group. By killing any infants not his in the group prior to their weaning, he is forcing their mothers to return to fertility sooner, increasing the probability of him having offspring with one or more of

those ex-mothers and hence increasing his overall fitness. Such evolutionary benefit for males has already been well-established (van Schaik and Dunbar, 1990).

Having an offspring killed in its first year of life is clearly very costly; the biological mother and father would each get zero fitness benefit from said offspring, despite the mother (and possibly the father as well) investing much time and energy into the pregnancy and early life of the offspring. As such, it may behoove the father to stick around that offspring up until the point it gets weaned in order to protect it from any infanticidal attacks by other males. In doing so, such a male has effectively formed the groundwork for having a pair-bond with the mother of his offspring (van Schaik and Dunbar, 1990).

Infanticide is quite common among primates, in large part due to altriciality (and in turn long durations of lactation), which makes the infants more susceptible to having infanticide committed against them (Opie et al., 2013). These facts would hold for humans as well. In addition, Opie et al. found that in primate phylogeny, infanticide by males typically precedes social monogamy (Opie et al., 2013). They concluded that not only is social monogamy in primates much more likely to evolve in the presence of high male infanticide, once social monogamy *does* evolve, it is more likely for there to be a subsequent decline in the amount of male infanticide than an increase in the level of polygyny (Opie et al., 2013).

There is still much debate over the role of infanticide in the evolution of long-term pair-bonding in non-human mammals. Contrary to the Opie et al. work discussed above, other prominent researchers (Lukas and Clutton-Brock, 2013) found “the available evidence suggests that male infanticide is unlikely to be the principal mechanism for the evolution of social monogamy in mammals.” Despite finding rates of male infanticide among socially monogamous species (9%) to be lower than rates among solitary species (27%), they concluded from their analysis that this difference was most likely due to “an independent evolution of the two traits” (Lukas and Clutton-Brock, 2013).

One key factor considered there was that in most socially monogamous mammals, the duration of female lactation does not typically exceed her gestation period. However, it is in cases where such lactation duration *does* exceed gestation length that a benefit would be expected for males killing other males’ offspring (Lukas and Clutton-Brock, 2013). This is because female mammals

will not typically have two unweaned offspring concurrently. Hence if lactation duration exceeds gestation length, there is benefit to the male in killing one offspring so the next (his) can be born sooner and the female will still only have one unweaned offspring at any given time (Opie et al., 2013).

For example, in modern-day humans one study found 50% of nursing women to return to normal estrous cycling within ten months of giving birth and 100% of the women by twenty months, while the cross-cultural average age of weaning in humans is thirty months (Quinlan, 2008). In chimpanzees, offspring are typically weaned around 4 years of age, while females on average only have one offspring every 5 years, meaning female chimpanzees will also only have one offspring to care for at a time (Pillsworth and Haselton, 2006).

The second hypothesis under the category of protection deals with mate-guarding, or a male protecting his mate. In most formulations, this hypothesis centers on the idea of paternity certainty, or an male protecting his sperm after it is deposited into a female. After mating with a female, males can either stay around that one female (“guarding” her) or leave her in search of new mates. By guarding her, a male can prevent other males from mating with that female (which can increase his fitness by reducing or eliminating completely any sperm competition), as well as offer protection to her physical well-being (discussed later). Roaming males attempting to copulate with a guarded female normally face a high cost-to-benefit ratio in any fight with a male guard, and the guards typically win these fights (Reichard and Boesch, 2003).

In general, a male must choose between acquiring *more* mates and increasing his paternity certainty with *one* mate. If conditions make the latter more important to a male’s fitness than the former, one could expect pair-bonding to evolve, as the male constantly remains near his one mate to help ensure paternity certainty, which would increase his fitness (Hawkes, 2004). However, note that mathematical models of this hypothesis have not supported mate-guarding as a strong enough selection pressure for long-term pair-bonding to evolve (e.g. see (Gavrilets, 2012a)).

Among socially monogamous species, levels of mate-guarding vary drastically. Kokko and Morrell (2005) constructed a theoretical model and found that males will evolve to guard little if females are either very faithful or very unfaithful. The intuition behind this finding is again that in gaining

paternity, males face a trade-off between having more mates and increasing paternity certainty in their one mate. Hence, if a female is highly faithful, her mate would be able to gain more mates by guarding little, with little risk to the paternity certainty of his mate's offspring. Similarly if a female is highly unfaithful, her mate would likely have little to lose by trying to gain more mates by guarding little since even if he spent his time/energy instead guarding her, she would likely still sneak copulations outside this mating bond. In addition, Kokko and Morrell found that attractive males are predicted to guard less, with the intuition being they are more likely to find *more* mates, and hence paternity certainty becomes less crucial in the trade-off (Kokko and Morrell, 2005).

A related hypothesis to mate-guarding, involving the direct physical protection of one's mate mentioned earlier, is the bodyguard hypothesis (Chapais, 2008). Rather than paternity certainty being the driving selection force behind the evolution of long-term pair-bonding, it could instead be physical protection of the female. If a group is such that a female must be constantly worried about harassment/violence from other males, her (and her future offspring's) fitness would be increased by having the male stick around her. However, in doing so he would also be increasing his paternity certainty, especially if the harassment she was receiving was sexual in nature, meaning the mate-guarding and bodyguard hypotheses are largely indistinguishable (Chapais, 2008).

The last hypothesis in this category deals with protection of one's self, namely against the threat of sexually transmitted diseases (STDs) (Kokko et al., 2002). This hypothesis hinges on the fact that STDs were increasingly problematic among early humans, as group size and population density increased (i.e. as humans became increasingly ecologically dominant). As these STDs became more widespread and lethal, the costs of having sexual relations with multiple partners greatly increased. If such costs increased sufficiently enough, it could have been better in terms of a male's fitness to have *fewer* mates rather than more. If that was the case, individuals would have switched to a strategy favoring remaining faithful to one or two mates rather than promiscuity, and hence long-term pair-bonding could have evolved (Kokko et al., 2002).

In addition, using the same reasoning, STDs could influence female choice. A female may view a dominant male, or a male who has mated with many females, as too risky to mate with due to the threat of getting an STD from him. Hence, females may actively begin rejecting mating with

such males. Mathematical models have supported this hypothesis as a way for mating systems to change and long-term pair-bonding to become more prevalent (Kokko et al., 2002).

A more recent paper (Bauch and McElreath, 2016) explicitly models how the threat of STD's could have provided sufficient selection pressure for the evolution of strict monogamy. In particular, the paper considers this hypothesis from a cultural evolution perspective (cultural evolution is discussed in further detail below). Under this interpretation of the hypothesis then, groups with individuals who punish promiscuity will do better over time when STD's are prevalent than groups with more promiscuous or polygamous behavior present. Hence, in conditions where STD's are prevalent, this socially-imposed form of strict monogamy will become more predominant in the long run (Bauch and McElreath, 2016).

3.3. Provisioning hypotheses

These next three hypotheses all fall under the category of food or other provisionings as the key selection pressure on the evolution of long-term pair-bonding in early humans.

The first hypothesis under this category, food for sex, deals with males provisioning their mates, or in particular, males provisioning females in exchange for copulations. As hunting became more important in early human history, and also the nutrients obtained from meat more valuable, females could increase their fitnesses by mating with males in order to obtain more high-benefit foods or other valuable resources. Females were able to increase their fitnesses through such beneficial provisionings, while males increased their fitnesses by increasing their likelihoods of obtaining mates. Provided such benefits were strong enough, and these exchanges consistently occurring between the same male and female, long-term pair-bonding could ultimately be selected for (Reichard and Boesch, 2003).

Such exchanges of important foods for copulations are routinely seen in the genus *Pan* (chimpanzees and bonobos). "Important foods" include meats and fruits high in fats and/or proteins, as well as foods requiring increased search time (Lovejoy, 2009). Looking at primates, there is an association between the evolution of social monogamy and a reliance on relatively scarce foods (Lukas and Clutton-Brock, 2013). Fruit, compared to foods like gum, bark, and fungi, offers much higher nutritional value while also being a more limited resource. Indeed, fruit constitutes a main

part of the diet in about 90% of socially monogamous primate species and only 28% of solitary primate species. Conversely, foods like gum, bark, and fungi constitute the main part of the diet in about 40% of socially monogamous primate species and 78% of solitary primate species (Lukas and Clutton-Brock, 2013).

In addition, a hypothesis related to both the above and the ecological hypothesis, the resource-defense hypothesis, posits that social monogamy could have evolved from a male defending a highly-valued (e.g. due to food resources) territory, able to keep all intruders out. In this case, a female may want to remain in that territory permanently, not so much because of the male himself, but because of the valuable territory he is able to control (Reichard and Boesch, 2003).

The next hypothesis in this group, paternal care, looks at a different type of male provisioning, this time to his offspring (Benshoof and Thornhill, 1979). Parental investment is any expenditure benefiting an individual's offspring at some cost to that individual (even if just an inability to invest that energy into other fitness components) (Quinlan, 2008). Offspring fitness is increased when a male is around to help provision said offspring. Provided this helping male is indeed the offspring's biological father, the male will be helping his own fitness as well (Wittenberger and Tilson, 1980). 59% of socially monogamous mammal species have been found to have regular carrying or provisioning of offspring by males, whereas only *three* non-monogamous mammal species have such carrying or provisioning (Lukas and Clutton-Brock, 2013). Mammals in particular may require the extra help from offspring provisioning because of that special energy-intensive (i.e. 670 kcal/day in humans) lactation period in a female mammal's life (Quinlan, 2008).

In addition, some researchers believe that as the human brain got bigger and human infants became increasingly dependent on others (altricial) during the early years of their lives, mothers were no longer able to care for their offspring alone (Benshoof and Thornhill, 1979). For instance, children cross-culturally consume more calories than they produce until at least age fifteen; contrast this with chimpanzees who typically produce more calories than they consume by age five (Conroy-Beam et al., 2015). Over the past 4 million years, human ancestors underwent a doubling of the length of their developmental period (Geary and Flinn, 2001). Among modern-day hunter-gatherers, females on average have one child every 3-4 years, meaning most women will have multiple dependent

offspring to care for at the same time, unlike in chimpanzees and other primates (Pillsworth and Haselton, 2006). In addition, parental investment in offspring does not just stop; rather, it often continues all the way through to become ‘grandparental investment’ (i.e. alloparenting) (Conroy-Beam et al., 2015).

If more than one adult was needed to care for an offspring, fitness-wise it made sense that that “other adult” would be the offspring’s biological father, since no other adult (besides the mother) would have as much genetic material invested in the offspring. As hunting became more important in the early human diet, males (the hunters) were increasingly needed as meat-providers. Hence it could have been some combination of increasingly-altricial offspring, the importance of hunting, and bipedality (meaning humans were more easily able to carry offspring and/or the provisionings) that could have helped long-term pair-bonding to evolve, provided paternity certainty was sufficiently high (Benshoof and Thornhill, 1979). However, two separate phylogenetic analyses suggest that paternal care is more likely to be a secondary adaptation occurring either with or after the evolution of long-term pair-bonding, rather than a main driver of this evolution (Lukas and Clutton-Brock, 2013; Opie et al., 2013).

The next hypothesis under this category, related to both parental investment and food-for-sex, is that of economic interdependence (Conroy-Beam et al., 2015). Much research has gone into the evolution of a division of labor among early humans, namely why, how, and when did men and women become solely responsible for specific and different tasks or chores? The economic interdependence hypothesis hinges on the fact that, on average, men have more strength/endurance needed for hunting large game than women do. In addition, during pregnancy and lactation women are already expending more energy in their daily lives than men and would likely encounter increased risk during large-game hunts (Conroy-Beam et al., 2015). For example, a pregnant female requires 8-10% more in caloric intake than a non-pregnant female, and a lactating female about 26% more (Pillsworth and Haselton, 2006).

Under this hypothesis, males and females would then engage in economic partnerships, whereby each sex would focus on that resource acquisition they could accomplish most efficiently. Hence, males would provide meat for a female and her offspring and the female would share with him the

foods she had produced or gathered. Not only does this give females easier access to valuable meat, it also provides males with protection in the event that he is unsuccessful in hunts (Conroy-Beam et al., 2015).

A related idea to this involves the idea of cooking (Wrangham et al., 1999). Cooking food is beneficial in terms of (1) calorie intake, (2) protection from disease or other sickness due to food poisoning, and (3) increasing the edible range of plant foods through digestibility. While men went out to hunt, women were left at the camp and hence became in charge of cooking. While males relied on women then for the cooked food each night they came back to camp, women in turn also relied on the men to help protect these valuable cooked foods as well as any foods which had already been gathered but yet to be cooked. It is in this way that such a relationship benefited both the male and female, and could have fostered the formation of long-term pair-bonds (Wrangham et al., 1999).

Once producing one's own food became prevalent among early human societies, there were two main strategies each individual could adopt, producing vs. scrounging. An individual following the producing strategy would be focused on producing his/her own food, and then storing such food for later consumption. On the other hand, an individual following the scrounging strategy would instead not be focused on producing his/her own food, but rather finding already-produced food elsewhere. Since females were naturally less mobile (due to the demands and restrictions of pregnancy, lactation, and parental care) and males naturally stronger, a logical division of labor resulted where females followed the producing strategy and males the scrounging strategy. However, this means males were often stealing already-produced foods from females, making it in females' best interests to 'buy protection' in any way they could. Since one such form of payment could be the female giving one male some of her cooked food as well as her exclusive reproductive rights, long-term pair-bonds could have evolved under this selection pressure (Wrangham et al., 1999).

3.4. Conflict hypotheses

Finally, this last category of hypotheses all deal with conflict as the main selection pressure for the evolution of long-term pair-bonding in early humans.

The first hypothesis in this category, one specifically relating to humans, has to do with the

idea of leveling (Chapais, 2013). As tool and weapon use became increasingly common across early humans, male size/strength dimorphism became less important in fights. Just because an alpha male is bigger and stronger than a lower-ranked male does not necessarily give that alpha male an advantage in a physical confrontation if the lower-ranked male has more and/or better tools/weapons available to him than the alpha male (Chapais, 2013). This meant that any fights between two now-evenly-matched males were much more costly to both males involved and also more likely to end without a winner despite the larger cost involved. Hence it was harder and more costly for an alpha male to protect any “harem” of females, making pairs of one male and only one female increasingly common until only strict monogamy was left (Chapais, 2008).

Males fighting over mates can be very costly to a group as a whole. If males’ time and energy is being spent in intragroup conflict, there will be less time and energy available to devote to intergroup conflict. As such, this second hypothesis under the category of conflict predicts that groups with more monogamous pairs will be more successful in the long term than promiscuous groups which routinely have intragroup fighting over mates. This hypothesis posits that since cooperation is key to early human survival and long-term pair-bonding supports such cooperation, long-term pair-bonding could have been able to evolve (Chapais, 2013).

There is little doubt that cooperation is in fact key to early human survival. Humans are a cooperative group-living species and coalitions are often formed in human social competition (Geary and Flinn, 2001). Among contemporary hunter-gatherers, cooperation is shown in ways far exceeding that of other primate species. Such examples include sharing food across all members in the group, groups having high levels of alloparental offspring care, cooperative hunting or other food acquisition strategies, construction and maintenance of living spaces, transportation of children or resources, contribution to public goods, etc. (Hill et al., 2011).

There is some evidence for this hypothesis from the palaeontological record (Lovejoy, 2009), albeit rather controversial. Fossils 4.4 million years old of *Ardipithecus ramidis*, a species occurring relatively soon after the hominin/chimpanzee split, indicate reduced sexual size dimorphism and reduced upper canine teeth size, both of which are indicative of a decline in male-male conflict (Lovejoy, 2009). *Homo erectus* appeared about 1.5-2 million years ago with males about 20%

heavier than females (similar to the sexual dimorphism seen in today's humans). Contrast this with the 60% heavier males found in the *Australopithecine* species from up to 5 million years ago (Geary and Flinn, 2001). If reduction in intragroup conflict was a driving force behind early human evolution, it would be expected that related characteristics like sexual dimorphism would evolve quickly in humans' ancestral line. The above fossil record provides support for this idea.

One intriguing alternative related to this hypothesis is if long-term pair-bonding evolved via cultural evolution (i.e. as an institution via marriage) instead (Henrich et al., 2012). This means that instead of relying on the evolution of *gene(s)* relating to long-term pair-bonding, it is instead *cultural* traits (norms) that are important in this evolution. Humans and their ancestors are still sexually dimorphic in size (as discussed above), an indication that the species should perhaps not be considered genetically monogamous and that it is culture playing the crucial role instead (de Waal and Gavrilets, 2013). However, note these cultural hypotheses would be operating on a much shorter time scale than the genetic hypotheses (i.e. tens of thousands vs. hundreds of thousands of years) (Fortunato and Archetti, 2010).

Henrich et al. question how one-male, one-female (strict) monogamy came to be when approximately 85% of societies (meaning raw number, not considering each society's population size) in the anthropological record allow for polygyny. They reason that strict monogamy was favored by cultural evolution because of how much it benefits groups (i.e. cultural group selection). Similar to the above *gene* evolution, because of those benefits, groups with strict monogamy would do better in the long term than their counterpart groups with polygamy (Henrich et al., 2012).

So where do such benefits come from? As already discussed, monogamy can lower male-male competition within a group; it can also reduce the number of un-mated males within the group. This is true regardless of the mating system of the comparative group (i.e. promiscuous vs. polygynous). Having fewer un-mated males within a group can reduce crime rates (for murder, rape, assault, robbery, etc.) and other personal abuses which are harmful to groups. Males low enough in rank/status that they get no mating opportunities will have essentially zero fitness and thus, evolutionarily, nothing to lose. This creates males who are more willing to take risks in order to get copulations, and hence creates conflicts within their groups (Henrich et al., 2012). Again,

groups who impose long-term pair-bonding as the social order will do better than those that do not. Under this hypothesis, even if selection on the actual genes is not strong enough to produce long-term pair-bonding, such evolution is still possible, just considering instead evolution on the related cultural traits.

Finally, the last hypothesis considered here deals with conflict between a male's various offspring, especially if produced by different females (Fortunato and Archetti, 2010). As it became the norm for humans for property and/or other resources to be passed on to the next generation, more clearly defined familial lines became increasingly beneficial. A father's fitness can be decreased by having to split his wealth (resources) among the more offspring he would have by having more mates. Strict monogamy would allow male/female mating bonds to be stable over time, and hence all the male's resources to be given to his offspring, all full siblings. In this way, strict monogamy could simply be a strategic behavior on behalf of males and females in allocating their resources to the next generation (Fortunato and Archetti, 2010).

In particular, Fortunato and Archetti (2010) constructed a mathematical model and found two conditions which would make strict monogamy (or "monogamous marriage" in their words) the most beneficial cultural mating system. First, when there is unigeniture, meaning only one offspring inheriting all of a parent's wealth, and second, when women are more faithful in a monogamous bond than a polygynous one. If the latter is the case, while men would still have that fitness loss from not mating with more than one woman, they *would* get a benefit from that one woman being more faithful than otherwise (i.e. increased paternity certainty). Note the female of this long-term pair-bond would also benefit, in that her male mate would be investing his resources exclusively in her children for the next generation (Fortunato and Archetti, 2010).

This hypothesis could help explain why monogamous marriage is so common in societies with intensive agriculture (e.g. plow or irrigation agriculture of much of Europe and Asia, as opposed to hoe agriculture or pastoralism of much of sub-Saharan Africa). In intensive agriculture, land is often the limiting resource, so splitting land among multiple heirs can only go so far. Hence, this hypothesis would be predicted to have much greater selective pressure under intensive agriculture than other forms of agriculture (Fortunato and Archetti, 2010).

4. Comparative work

Most of the research discussed above focuses on only one of these hypotheses. There is relatively little comparative work done pitting such hypotheses against each other. However, note Opie et al. (2013) and Lukas and Clutton-Brock (2013), both discussed at length earlier, each take a comparative empirical look at different hypotheses, using phylogeny and other data (Opie et al., 2013; Lukas and Clutton-Brock, 2013).

On the other hand, one example of comparative theoretical work is Gavrillets (2012). In this paper, Gavrillets constructed models for several of the above hypotheses in order to look at how long-term pair-bonding could have evolved in early humans' past. In particular, he found that long-term pair-bonding was rarely predicted to evolve in (1) the baseline scenario ("communal care"; males allocate some amount of effort towards helping *all* offspring in their group), (2) with mate-guarding, (3) with food-for-sex, and (4) with "mate-provisioning" (equivalent to the food-for-sex hypothesis except males are only able to provision one female and females to be provisioned by one male) (Gavrillets, 2012a).

However, long-term pair-bonding *was* able to evolve provided two things were present: (1) inequalities between males in their fighting abilities (i.e. rank among males in a group), and (2) differences in females in both "faithfulness" (how faithful any given female will be to her mate) and choosiness (for a male who will provision her more). These results imply that it is likely that more than just mate-guarding or food-for-sex are needed in order for long-term pair-bonding to evolve. Not only is male rank important (since lower-ranked males may initially be the only ones who do better pursuing a pair-bonding mating strategy), but females' ability to choose mates (namely those who provision more and/or provide more care) is also important in allowing long-term pair-bonding to evolve (Gavrillets, 2012a).

The above study looked at individual fitnesses, meaning how different mating systems could have evolved by benefiting the individuals within such a system (Gavrillets, 2012a). Another comparative theoretical study instead took a group selection approach, looking at how different mating systems could have evolved by benefiting *groups* as a whole (Nakahashi and Horiuchi, 2012). These authors constructed a mathematical model looking at the evolution of the alpha male strategy, as well as

female mating and grouping strategies. In particular, they find that a human-type mating system (i.e. one in which groups consist of multiple females and multiple males, male-male competition is weak, and there is no female promiscuity) can evolve when having a larger group is beneficial (e.g. in defense against other groups) and when the cost of female promiscuity is large (e.g. due to the many types of human venereal diseases) (Nakahashi and Horiuchi, 2012).

5. Conclusion

Despite all the debate and curiosity surrounding the evolution of long-term pair-bonding in humans, few concrete answers have yet to be obtained. Perhaps future mathematical modeling work will be able to help. In particular, there is still debate over the ancestral mating system in early humans, as well as the main selection pressure(s) resulting in humans switching their mating system to one of long-term pair-bonding and over what role (if any) concealed ovulation may have played. Indeed, researchers still contradict each other over whether the chimpanzee-human last common ancestor had a promiscuous mating system (like chimpanzees), a harem-style polygynous mating system (like gorillas), or multi-level societies (like hamadryas baboons). Many of the hypotheses for the evolution of long-term pair-bonding in humans have more support under only one of these mating system ancestral states, making this a related, and equally important, research question.

In terms of how long-term pair-bonding evolved and whether strict monogamy was a culturally and/or genetically evolved trait in humans, these are both still very much unanswered research questions. Many different researchers have contributed much to this ever-growing body of literature spanning over a century, but few agreed-upon answers have yet been obtained. Some hypotheses have certainly gained more support throughout the years than others, but there are still so many being actively considered. The time is ripe for researching human origins, and progress will likely be made in this upcoming decade. Being human ourselves, it is only natural to ask the question of from where did our species come and to investigate those traits that make us the “uniquely unique species” (Alexander, 1990). The human universal of forming long-term pair-bonds is just one such trait.

6. Acknowledgments

K.R. was supported by a National Science Foundation Graduate Research Fellowship. S.G. was supported by the National Institute for Mathematical and Biological Synthesis through National Science Foundation awards EF-0832858 and DBI-1300426, with additional support from The University of Tennessee.

Conclusion

In this dissertation, I have analyzed several aspects of primate non-verbal sexual communication, using mathematical models. Doing so has contributed a new, and important, body of work to the literature already written on the subject.

0.1. Recap of Chapters

In Chapter 1, I investigated the effects of certain ecological factors on visual ovulation signaling among female primates. I showed that increasing group size, increasing variation in male quality, decreasing the costs of having visible ovulation signs, decreasing male reproductive stochasticity, and increasing the weight of genetic vs. non-genetic effects could each result in increased visual ovulation signs.

I also showed that visual ovulation signs would increase as the risk of infanticide increased in the population. I discussed how such a finding is intuitive, since pronounced visible ovulation signs help a female concentrate paternity, and hence gain possible protection for her infant from infanticide.

Finally, I was able to test two of these predictions empirically, and found empirical support for both. First, as predicted by our model, species with larger group sizes had more visible ovulation signs present. Second, also as predicted by our model, species with a higher risk of infanticide had more visual ovulation signs present.

In Chapter 2, I investigated the evolution of female sexual receptivity. I showed female continuous receptivity and concealed ovulation to be correlated, and how only in groups with visible ovulation signs present would one expect to find a relatively short length of time when the female will be receptive to mating.

I discussed the trade-offs of paternity concentration vs. paternity confusion, and how each relates to infanticide. Where paternity concentration is favored, so are shorter lengths of sexual receptivity. Conversely, where paternity confusion is favored (for example, with an increasing threat from infanticide), so are longer lengths of sexual receptivity, including continuous receptivity. I also discuss other ways for continuous receptivity to evolve, including when increased weight gets put on non-genetic benefits to females coming from mating, compared to any genetic benefits. In such a situation, it can behoove females to mate throughout their entire cycle.

In Chapter 3, I investigated the evolution of sexual signal reliability, evaluating under what conditions will a female's visual sexual signaling line up perfectly (or not) with her peak time of fertility. I found that signal reliability could increase with increasing group size, increasing variation in male quality, decreasing reproductive stochasticity, and increasing weight being put on genetic vs. non-genetic male benefits.

In addition, I find the intuitive relationship between the evolution of signal reliability and that of visual ovulation signs: females with strong visual ovulation signs also have highly reliable signals, and vice versa. Females with both strong visual ovulation signs and very reliable signals are better able to gain some benefit (in the form of male genetic quality) from having the costly signal. Females instead with both large signal variability and strong visible ovulation signs would not necessarily gain any genetic benefit (if they are not also highly fertile at the time of their peak signal, when they would be most attractive to the high-quality males), while still having the large costs associated with strong visible ovulation signs (due to increased energy expenditure, predation, water retention, etc.).

Finally, in Chapter 4, I outlined a future, related biological problem (the evolution of long-term pair-bonding in humans). The problem in general was described, as well as the possible evolutionary pathways to long-term pair-bonding in humans (from promiscuous groups, harems, multi-level societies, or strict monogamy), the various hypotheses for how it evolved (ecological, protection, provisioning, and conflict), and also comparative work which has already been done.

In addition, I outlined the potential connection between the evolution of long-term pair-bonding and the evolution of concealed ovulation. Such a problem could easily be addressed with many of the same mathematical methods/models used in the earlier chapters. While modeling such a phenomenon has been saved as future work here, it is certainly a research question of prime importance to those in the field.

0.2. Future Work

There is also other possible future work which would be of great interest and could build off the work presented here. This dissertation does not address any male evolving strategies nor the possible co-evolution of any male and female strategies. Addressing this limitation is important

future work which could be done. For example, this dissertation does not explicitly include mate guarding; introducing a male strategy allowing time spent mate guarding to evolve would likely influence the evolution of female ovulation signs.

This dissertation is also only concerned with the evolution of primate *non-verbal* sexual communication. However, there has been much research done regarding female sexual *vocal* communication among primates, for example copulation calls. Copulation calls have also been linked to the trade-offs in female sexual strategies between favoring paternity concentration vs. paternity confusion. Loud, prominent copulation calls benefit paternity concentration, while silent mating benefits paternity confusion. Modeling work similar to that presented here could be used again for researching paternity concentration vs. paternity confusion strategies, except now for verbal ones, such as female copulation calls.

In addition, there has been much recent interest in multi-modal sexual signal communication among primates. This field seems ripe for future research, especially theoretical. While much modeling work has been done with multi-modal communication in general, there is still much room to explore these models more specifically as they relate to multi-modal sexual communication among female primates.

Finally, there are also remaining questions as to the importance of male genetic vs. non-genetic benefits to females. While this dissertation includes the parameter η which weights the benefits of each genetic and non-genetic effects on female fitness, it has no effect on the *males* themselves. For instance, are males of higher genetic quality more likely to also have higher non-genetic quality? Or are males of the highest genetic quality the *least* likely to have high non-genetic quality? Or are genetic and non-genetic quality determined completely independently of each other? These questions would be easy to address using the modeling framework presented here. Rather than males' genetic quality being drawn from a normal distribution and then non-genetic quality generated deterministically (as is the case for the Chapters presented here), one could instead draw males' genetic and non-genetic quality from a bivariate normal distribution with correlation parameter ρ . In this case, $\rho < 0$ corresponds to the case when male genetic and non-genetic qualities are inversely related to each other (for instance, when powerful males will invest in obtaining more

mates vs. any paternal care). $\rho > 0$ instead corresponds to the case when male genetic and non-genetic quality are positively correlated with each other (for instance, when being a more powerful male implies being better at provisioning). $\rho = 0$ indicates the last case: when male genetic and non-genetic qualities are completely independent of one another.

0.3. Discussion

Overall, what I have shown in this dissertation is that primate non-verbal sexual communication can be affected by many different things. While so much interest has been on the evolutionary mystery of exaggerated primate sexual swellings, and in particular concealed ovulation, we have shown here that such evolution, in both the exaggerated and concealed directions, could have evolved for a variety of reasons. While so many researchers historically have been out to find ‘the one’ reason for such evolutionary trajectories, we have here demonstrated that perhaps there is no ‘the one’. Perhaps different species, under different selection pressures, simply ended up with the same results.

I have also shown in this dissertation that there exist trade-offs between receptivity length and ovulation signs *and* between signal reliability and ovulation signs. Both of these trade-offs relate to paternity concentration vs. confusion. When it is in females’ best interests to concentrate paternity, they benefit from having short receptivity, strong ovulation signs, and a highly reliable signal. Conversely, when it is in females’ best interests to confuse paternity, they instead benefit from long lengths of receptivity (including continuous receptivity), weak ovulation signs (including concealed ovulation), and an unreliable signal. Indeed, the modeling work presented here has shown concealed ovulation and continuous receptivity to be correlated, and strong ovulation signs to be correlated with reliable signaling. Hence, as predicted by primatologists, each of visible ovulation signs, receptivity length, and signal reliability play a role in balancing female sexual strategies between paternity concentration and paternity confusion.

While such work is informative by itself, I truly hope for these models to get tested in the future. More data is needed to do robust comparative tests, but I hope for such data to become available in the future. I hope the work here will serve as inspiration for future empirical work, allowing empiricists to look at these hypotheses and their own ideas in potentially different ways. Regardless

of such future use, the results presented in this dissertation have used mathematics to give new insight into primate evolution and to help resolve old mysteries surrounding primate non-verbal sexual communication.

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Vita

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