Reproductive Suppression in Female Primates: A Review

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Reproductive performance is the currency of evolution. All things being equal, an organism should reproduce as often as possible. The puzzling questions in evolutionary biology, therefore, are not how and why an organism *does* reproduce, but rather how and why an organism *does not* reproduce. It is difficult to understand why any individual, particularly a female, might forestall reproduction when one of the biggest limitations for female mammalian reproduction is time (that is, reproductive lifespan).[1] The answer, now widely cited throughout behavioral ecology is quite simple: Reproductive suppression can be an adaptive strategy.[2]

Mammalian female reproduction is a costly endeavor that may have a high chance of failure under suboptimal circumstances. Therefore, over evolutionary time, natural selection should have favored females that are sensitive to environmental cues and able to suppress their own reproduction when current prospects for reproductive success are poor. This idea, known as the reproductive suppression model,[2] maintains that females may increase their lifetime reproductive success by restricting reproduction to coincide with favorable conditions. Thus, the general rule of thumb is this: If prevailing conditions are poor, delay reproduction to preserve energy for future reproduction; if conditions are likely to remain the same or get worse, carry on with reproduction.

Several assumptions are associated with this model.[2] First, female reproduction should be sensitive to all cues that are predictive of poor reproductive outcomes and natural selection should favor females that are able to translate these cues correctly into reproductive decisions. Second, females should suppress reproduction *as soon as* a cue is available because the earlier the loss, the less it affects a female's subsequent reproduction. Third, females should use only cues that *reliably* predict poor reproductive performance because it is costly to suppress a reproductive event that would otherwise have been successful. Fourth, reproductive suppression should be regulated by the female herself, and thus differs from the more general term "reproductive failure," which also includes externally imposed sources of loss, such as disease, injury, or infanticide.

A comprehensive review of the reproductive suppression model is not possible here. Therefore, we restrict this review in three important ways. First, we focus on within-population comparisons and examine why one female might reproduce successfully while another in the same population might not. Between-species comparisons that explore life-history evolution across taxa have received excellent coverage elsewhere.[1]

Second, although we briefly review the energetic causes of reproductive suppression, we focus primarily on social causes (Fig. 1). Certainly, a female's physiology should be sensitive to all cues that indicate harsh reproductive conditions: their own energy balance and health, the physical and genetic status of their embryo or fetus, and the local environmental conditions.[3] But social pressures are among the most dramatic and least understood factors affecting female reproduction.

Third, in the spirit of this special issue, we would like to highlight the work by Jeanne Altmann and Sarah Hrdy, who have substantially furthered our understanding of reproductive suppression. In her groundbreaking work with wild yellow baboons in Amboseli National Park, Kenya, Jeanne Altmann has demonstrated that living in a resource-poor and unpredictable environment can have dramatic consequences on female fitness across the life span.[4–9] Similarly, Sarah Hrdy has established much of the groundwork for testing hypotheses related to social suppression in female primates through her empirical work with the

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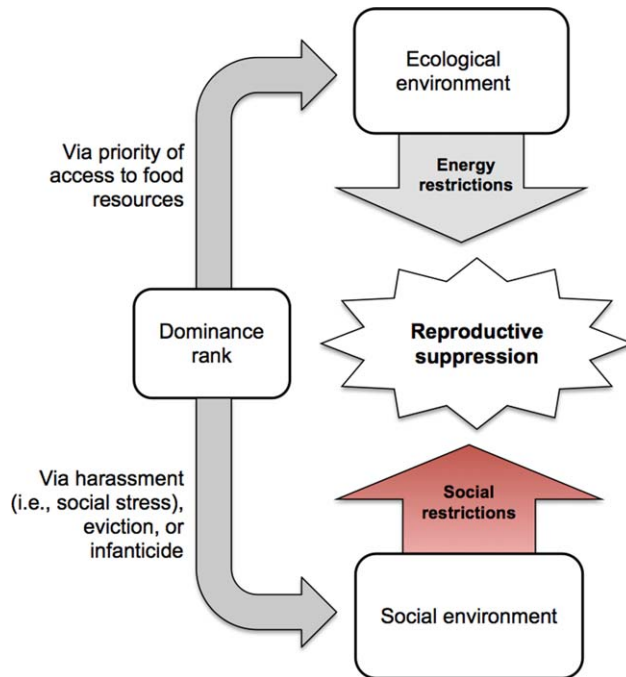
Figure 1. Routes to reproductive suppression. It has been notoriously difficult to separate rank-related reproductive dysfunction caused *directly* by elevated levels of social stress from that caused *indirectly* by nutritional insufficiencies. Here, we focus mainly on empirical studies in primates that have demonstrated a direct link between the social environment and female reproductive failure (red arrow). Note that although dominance rank is a component of the social environment, it can mediate fitness in two ways: through the ecological environment (for example, by restricting energy intake, increasing energy expenditure, or both) and through the social environment (for example, via social stress or social suppression of subordinates). (Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.)

ENERGETIC SUPPRESSION

Mammalian female reproduction requires a great deal of energy.[16] When food is limited or entails high acquisition costs, organisms must make allocation decisions to prioritize the partitioning of metabolic fuels. For many long-lived organisms, individual survival is paramount thus, when energy is at a minimum, basic cellular processes critical for survival take precedence over processes that can be compromised, such as reproduction.[17] However, even when survival is not at stake, females should abandon reproductive efforts as soon as they detect that they do not have enough energy to complete them. Energetic suppression is defined as the self-imposed inhibition of reproductive physiology in otherwise fertile individuals in response to an internal metabolic cue that indicates a poor reproductive outcome. To be clear, "self-imposed inhibition" does not involve any conscious decision by the female, but is mediated by a metabolic signal triggered in response to an appropriate cue. The specifics that underlie this metabolic pathway have yet to be fully identified, but it is becoming increasingly clear that integrative control of energy balance and reproduction is carried out by multiple metabolic and neuroendocrine signals. Research on *how* females suppress reproduction in response to energetic signals is still rapidly developing, and we refer readers to recent reviews of this topic.[17,18]

The question of *why* females suppress reproduction in response to limited energy is easier to tackle. Energetic suppression is common in species in which physical condition must meet a threshold before initiating reproduction (capital breeders).[19] This energetic threshold presumably indicates whether a mother will be able to partition the necessary energy to reproduction on an ongoing basis. In contrast, other species exclusively rely on external cues to initiate reproduction (income breeders). These external cues enable females to schedule more energetically demanding stages of reproduction with a reliable and predictable abundance of resources.[19] Few

Hanuman langurs of Mt. Abu, India,[10,11] and her theoretical ideas about infanticide.[12–14]

In what follows, we provide an overview of reproductive suppression in female primates, paying particular attention to two different levels of analysis: an ultimate (functional) and a proximate (mechanistic) perspective (Fig. 2). From a functional perspective, we ask why females suppress their own reproduction. That is, what are the fitness benefits? To answer this question, we review the consequences of reproducing under conditions in which the prospects for success are poor. Second, from a proximate perspective, we ask how females suppress their own reproduction. That is, what mechanisms are involved? There are two ways to answer this question. We can explore the cues themselves or how we can examine these cues are translated into reproductive sup-

pression via neuroendocrine mechanisms. In some cases, these are one and the same; the factors that serve as proximate cues indicating a suboptimal environment are the same ones that trigger reproductive suppression (for example, poor energetic condition). In most cases, however, the cues that reliably predict reproductive failure are not inherently linked to reproductive suppression (for example, the arrival of a new male). In the interest of bridging function and mechanism, we focus on both the cues as well as the specific neuroendocrine mechanisms. We ask "why" and "how" questions as two related lines of inquiry. Just as the blind man cannot completely describe an elephant by holding onto its tail, we specifically hope to emphasize how integrating answers at different levels can help us emerge with a better understanding of reproductive suppression in female primates.
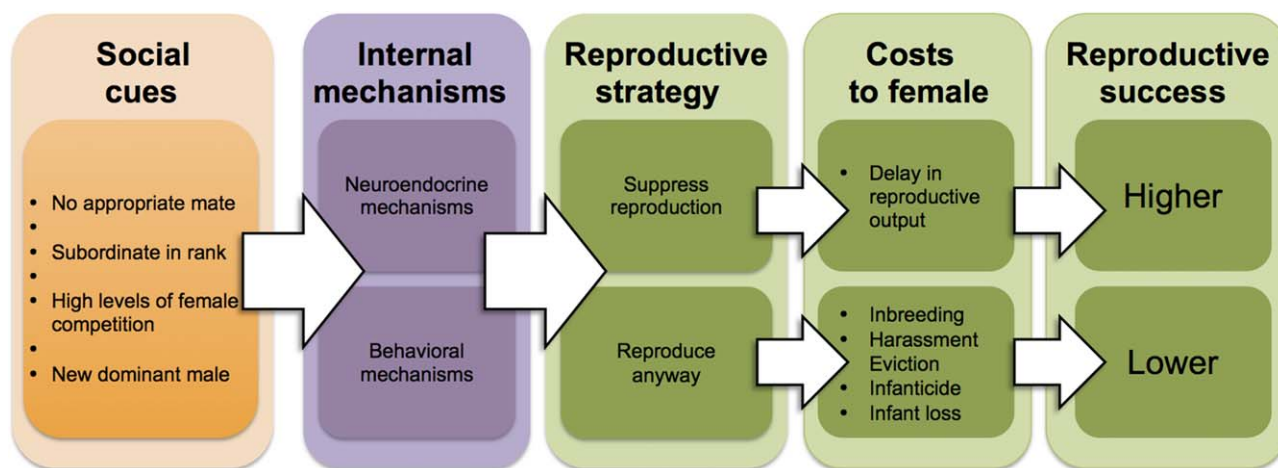
Figure 2. Overview of proximate (orange and purple) and functional (green) pathways that produce social suppression of reproduction in female primates. In this model, selection should favor females that translate external social cues appropriately. Note that the costs caused by reproductive suppression could lead to *lower* reproductive success if social cues become unreliable and females that "reproduce anyway" are successful. (Color figure can be viewed in the online issue, which is available at wileyonlinelibrary.com.)

primates are strictly capital or income breeders, and primates without a strict breeding season probably use a combination of internal and external cues. Therefore, with all the uncertainty that comes with a lengthy investment period, most primates should be able to abandon reproduction at any time.

Captive studies of primates have unambiguously confirmed that inadequate maternal energy is associated with reproductive dysfunction. For example experimental manipulations involving food deprivation were able to induce amenorrhea in common marmosets[20] or pregnancy loss in rhesus macaques.[21] Similarly, strenuous exercise without a corresponding increase in caloric intake resulted in amenorrhea in rhesus macaques.[22]

However, captive studies do not provide the multi-dimensional aspects of the natural lives of female primates where a multitude of social and environmental factors contribute to variable reproductive outcomes. Some of the best examples of the adaptive significance of energetic suppression in wild primates derive from Jeanne Altmann's long-term studies on Amboseli yellow baboons. Altmann and her colleagues found that a resource-rich environment can dramatically improve female reproductive outcomes. In one group, baboons supplemented their diet with scraps from the garbage of a nearby tourist lodge[23], while in

another group, severe habitat degradation, forced individuals to move to a new home range with more abundant food resources.[9] In both cases, female baboons in the improved foraging environments matured earlier and exhibited higher fertility.[8,9] Another study on the Amboseli baboons provided direct support for the role of energetics in female reproductive suppression. During drought periods, females were much less likely to ovulate; if they ovulated, they were less likely to conceive and if they conceived, they were less likely to carry the pregnancy to term.[24]

Studies from Amboseli and other wild primate populations have demonstrated that female fertility declines as the number of competitors for resources goes up.[24–26] For example, female yellow baboons in larger groups exhibited longer interbirth intervals than did females in smaller groups.[9] Similarly, during drought periods, the probability of conception decreased as the number of females in the group increased.[24]

Numerous studies have shown that the costs of living in a primate group may not affect all females equally, particularly in species with strict dominance relationships, such as cercopithecines. Across cercopithecines, low maternal rank is often associated with a later age at maturation,[27–29] longer interbirth intervals,[9,26,31,32] and later age at first birth.[26,28] Possibly media-

ting the link between energetic condition and reproductive rate, one study on female yellow baboons reported that (for females of all ranks), successful implantation was facilitated by relatively high levels of progesterone shortly after ovulation.[33] However, the progesterone threshold for proper implantation was higher in subordinate females than in dominants. Thus, an elevated threshold for implantation may actually serve as a "reproductive filter," reducing the probability of conception in females most likely to be affected by harsh environmental conditions (i. e. subordinate).

The relationships among group size, dominance rank, and fertility may be indirectly mediated by access to resources, directly mediated by social stress, or a combination of both. Separating the relative contributions of each mechanism is critical for making predictions about which populations are likely to exhibit rank-related or group-size differences in female reproduction and under what conditions. Social variables are likely to interact with energetic ones to generate an adaptive reproductive "decision." To date, social suppression of reproduction has largely been ignored in many primates, despite evidence that social interactions can have major impacts on female fitness. For this reason, we focus the remainder of our review on social suppression.

SOCIAL SUPPRESSION

Social suppression is said to occur when reproduction in otherwise fertile individuals is delayed, inhibited, or terminated in response to social cues.[2] It is independent from other factors that may influence reproduction, such as energy balance, physical health, and day length. Sarah Hrdy was one of the first to highlight the fact that social factors can have dramatic effects on female reproductive success. Her landmark work on infanticide in langurs and other primates[10,12,13] stimulated interest in how females can reduce the costs of such interactions.

A FUNCTIONAL PERSPECTIVE ON SOCIAL SUPPRESSION: WHY IS REPRODUCTIVE SUPPRESSION ADVANTAGEOUS?

Delayed Maturation to Avoid Inbreeding

In most taxa, males or females do not remain in their natal group long enough for inbreeding to occur. However, among primates there are some notable exceptions. For example, in the cooperatively breeding callitrichids (marmosets and tamarins), daughters routinely remain in their natal groups well past the age of puberty,[34] possibly due to a shortage of productive territories and/or the inclusive fitness benefits or experience gained from helping to raise siblings in the natal group.[35] Another example comes from our own research on a wild population of geladas in the Simien Mountains National Park, Ethiopia. Geladas live in polygynous one-male units in which leader males occasionally maintain extensive reproductive tenures (maximum mate tenure, 6.9 years) that are well past the age at puberty for females in this population (manage at puberty 4.7 years). Yet for both callitrichids and geladas, females are able to avoid inbreeding because (for the most part) they are able to delay puberty under certain conditions.

Life-history theory predicts that the timing of puberty should reflect a balance between the *costs* of diverting energy from growth and the *benefits* of reproducing earlier. However, social factors, such as the availability of suitable mates, may influence this calculus. When only kin are "available" as mates, the costs of inbreeding might shift the balance toward investing in growth and delaying the onset of reproduction. In rodents, the presence of unrelated males is able to accelerate puberty in juvenile females,[36] a phenomenon known as the Vandenbergh effect. The Vandenbergh effect encompasses two levels of control: the *inhibition* of reproduction when social cues indicate high competition or no appropriate mates, and the *stimulation* of reproduction once these inhibitory cues are lifted. Such social inhibition and stimulation of puberty clearly operate in callitrichids and may explain the timing of puberty in some catarrhines, like geladas.

Among callitrichids (with a few exceptions[37,38]), female daughters that remain in their natal group exhibit some degree of ovulatory suppression in the presence of the dominant breeding female (usually the mother).[34] Subordinate females remain anovulatory until social conditions change. The social conditions necessary for the full onset of puberty in subordinates, including ovulation, mating, and conception, varies by species. For example, puberty in at least two species of marmosets requires only the removal of the dominant female;[38,39] while puberty in cotton-top tamarins requires the introduction of a novel male as well[40,41] (but see French, Abbott, and Snowdom[42] and Heistermann and coworkers[43]). Occasionally, subordinate marmoset daughters ovulate in the presence of their mothers.[39] Remarkably, the best predictor of this "escape" from reproductive suppression is the relationship of these daughters with their mother. Daughters that do not show submission to their mothers are the ones that ovulate.[39]

Puberty is a complex process that is affected by both life-history parameters and social cues. For instance, female callitrichids are able to ovulate only a few short weeks after removal from the dominant female[44] and pairing with a novel male,[40] which presumably is too short a time to encompass the entire maturational process. Moreover, ovulating females will sometimes quickly revert to an anovulatory condition at the first signs of social subordination.[45] Consequently, it has been proposed that suppressed females are not delaying puberty at all; that is, the reproductive axis undergoes sexual maturation, but puberty is masked by immediate ovulatory suppression.[40] Thus, a more accurate characterization in callitrichids may be that subordinate females are released from or inhibited by family-induced fertility suppression.

Among catarrhines, we know of only two cases suggesting that males have an effect on female puberty. The first is an anecdote from captive hamadryas baboons, a species that lives in relatively "closed" one-male polygynous units. The arrival of a new male in a captive group was followed by the sudden, early maturation of four young females, as determined by their first signs of perineal sexual swellings.[46] The second case is from our population of geladas, who also exhibit closed reproductive one-male units.[47] When we examined the timing of puberty for gelada females, we found two striking patterns. First, puberty was disproportionately more likely to occur the week after the arrival of an unrelated male.[48] An examination of the timing of female maturation found that there was an 11-fold increase in maturations the week after a new male arrived as compared to the background rate in the population (N = 33 females across 17 units, Fig. 3). Second, no females matured while their fathers were the only breeding male, even though many were old enough to do so (N = 40). Yet 60% of these females subsequently matured in the two months after an unrelated male arrived (Fig. 4). Taken together, these data strongly suggest that social conditions can affect the timing of puberty in geladas; new males appear to trigger puberty and father-only groups appear to delay it.

To our knowledge, these are the only two reports of delayed puberty in a catarrhine primate in association
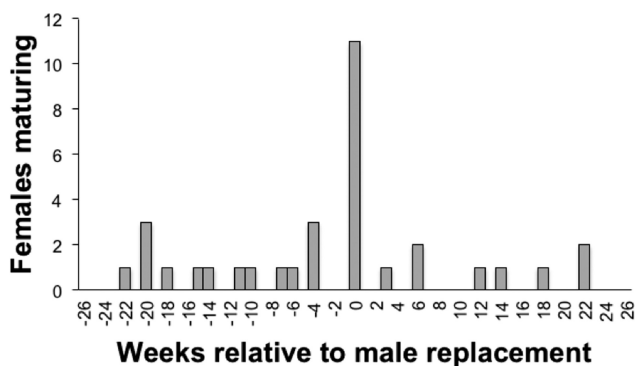
Figure 3. Wild female geladas (*Theropithecus gelada*) exhibit external signs of maturation (based on the first signs of sexual swelling) disproportionately more often during the week after a male replacement. Shown are the number of females reaching maturity in groups where males were replaced relative to the timing of those replacements (N=33 females). The zero point on the x-axis indicates the week after a male replacement.

with a social factor. As yet, we have no data on the sexual maturation of the reproductive axis relative to the putative suppression imposed by the female's father. Furthermore, because many catarrhine primates, (e.g. yellow baboons),[4] have a period of "adolescent sterility" between puberty and first reproduction, we do not yet know whether accelerated or delayed puberty in a catarrhine necessarily corresponds to an earlier or later age at first reproduction.

Reproductive suppression to Minimize Female Competition

Primate females may also suppress reproduction to minimize competition with other females. In some species, dominants may reduce the fitness of subordinates by imposing costs (for example, through harassment) or reducing the likelihood of success (for example, through infanticide). Thus, dominants can create an environment in which subordinates would do better

to suppress reproduction until competition becomes less fierce, (e.g. when food is abundant or when the dominant is not breeding). In some societies, the predictable nature of social inequalities between dominants and subordinates may have selected for complete or partial ovulatory suppression in subordinates.[49]

Infanticide.

Females are known to kill other females' offspring across a wide variety of mammalian taxa.[12,50] However, female infanticide is relatively rare among primates, having been reported only in ring-tailed and brown lemurs,[51] yellow baboons,[52,53] chimpanzees,[54,55] and common marmosets.[56] Female infanticide is hypothesized to result from competition for scarce resources. That is, by killing the young of other females, the perpetrator and her offspring eliminate current and future competitors for alloparents and/or food.[13,50] Such intense competition among females has almost certainly had strong selection on the evolution of reproductive suppression in subordinates.[50]

The callitrichid social system may provide the most extreme example of social suppression because, generally, only the dominant female breeds. Most callitrichid females give birth to twins, which must be carried continuously for the first few weeks of life. Soon after, conception occurs again.[57] Such high rates of reproductive invest require that nonbreeding group members ("helpers") aid in rearing the dominant female's offspring.[57] The ubiquity of helpers has led some to suggest that they are mandatory for successful rearing of twins.[58] Competition for helpers is therefore intense. Perhaps as a result, among marmosets, dominant females kill the offspring of subordinate females[59] and among tamarins, dominant females evict subordinate females that give birth.[60,61] These actions allow the dominant female to maintain "reproductive sovereignty." As in many cooperatively breeding mammals, selection appears to have favored predictable ovulatory suppression in subordinate callitrichid females, sometimes even in the presence of an appropriate male mate.[39]
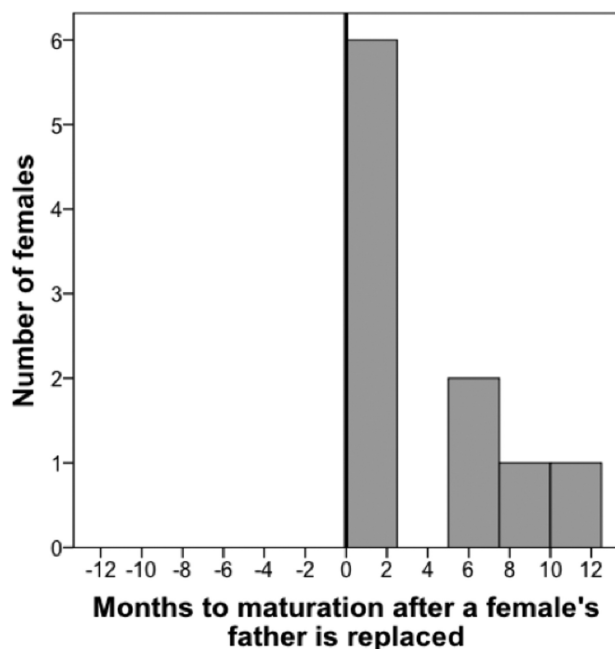

Figure 4. Shown is the number of female geladas reaching maturation (based on the first signs of sexual swelling) relative to the timing of their genetic fathers being replaced as the leader male (N=10 females across 20 groups). The dark vertical line represents the father's replacement. Only females whose fathers were present are shown. Note that no females matured while their father was still the dominant male.

Harassment

In contrast with the predictable ovulatory suppression of callitrichids, individual females in many other primate taxa suppress ovulation in a more probabilistic fashion. Is in callitrichids most of these cases are associated with high levels of female competition when resources are scarce. Females that are persistently harassed may delay reproduction until a more opportune time.

The most widely cited study of female social harassment documents female coalitionary attack behavior in a wild population of yellow baboons.[53] Such attacks were disproportionately directed toward in reproductive phases most susceptible to disruption (i.e., ovulation and early pregnancy). However, the results of this study have been criticized, mainly because the frequency of attacks (rather than individual females) was the unit of analysis.[63] Other results consistent with the social suppression hypothesis derive from studies on captive rhesus macaques,[64] wild chacma baboons,[65] and wild geladas.[66,67] In each case, ovulatory females received the highest rates of aggression from other females. However, the study on yellow baboons was the only one to also record changes in reproductive parameters following aggression, with females receiving the most attacks exhibiting more cycles to conception and a higher rate of miscarriage than their dominant counterparts.[68] Moreover, a follow-up study on captive geladas confirmed that attacks by dominant females can indeed directly inhibit ovulation in subordinates.[69]

Reproductive suppression to Counter Infanticide by Males

Male infanticide has been documented in nearly every major taxon of primates and is perhaps the most pervasive and extreme threat to female reproductive success.[70] Incoming dominant males kill offspring that are not their own. When they do, the mothers of these infants return to a fertile state, allowing the infanticidal male to sire offspring much sooner than if he had to wait for the previous infants to be weaned. In response to this threat, females have developed a variety of counter-strategies.[71] Some of these strategies appear to prevent infanticide altogether, while other strategies appear to mitigate the costs of infanticide.

Prevention strategies typically involve behavioral tactics by females that act as either deterrents (for example, enlisting the protection of other males) or manipulations (for example, obscuring the cues that trigger infanticidal behavior by males). Prevention strategies *per se* are not examples of female reproductive suppression. Mitigation strategies, in contrast, cause vulnerable females to immediately terminate all further investment in their current infant to allow investment in a future and presumably more successful infant. Such "loss-cutting" strategies fall squarely within the framework of the reproductive suppression model. Mitigation strategies inflict reproductive costs on females because all previous investment in the current infant is lost.

Strategies for pregnant females

Pregnancy termination may be one of the most dramatic mitigation strategies available to primate females that encounter the threat of infanticide during the prepartum period. If females that maintain their pregnancies after a new dominant male enters the group routinely lose their infants to infanticide, then selection should weed out this strategy. Alternatively, if females that miscarry following the arrival of a new male are able to quickly and successfully reproduce with the new male, then females with the physiological ability to terminate pregnancies in response to a novel male will predominate until such a trait becomes relatively fixed in a population. In support of this scenario, our data from geladas indicates that terminating a pregnancy is indeed an adaptive strategy for females whose infants might be susceptible to infanticide.[72]

The hypothesis that pregnancy termination might be adaptive for females builds on decades of experimental studies of rodents. In 1959, Hilda Bruce published the first demonstration of what would later be called a "Bruce effect," a phenomenon in which pregnant females terminated their pregnancies after exposure to novel males.[73] The Bruce effect was first documented in mice and later extended to numerous other rodent species.[74] However, all of these demonstrations were conducted under laboratory conditions, leading to speculation that the Bruce effect might simply be a laboratory artifact.[75] However, our recent research on geladas has since provided clear evidence of an adaptive Bruce effect in a wild popuation.[72]

Using five years of demographic and hormonal data from geladas living in the Simien Mountains National Park, Ethiopia, we found that more than 80% of pregnant females spontaneously miscarried when the dominant male was replaced by another male ("takeover"). Because nearly half of all infants fall victim to infanticide after takeovers,[76] the context and high frequency of the Bruce effect in geladas support its connection to infanticide. Most surprising, females miscarry the same day that the new male arrives. Because of the immediacy of the effect and because males rarely harass or attack the females that miscarry, the Bruce effect in geladas is unlikely to be mediated by generalized stress.

Anecdotal observations from captive hamadryas baboons also suggest that a Bruce effect may be a female reproductive strategy to mitigate infanticide. A pregnant female spontaneously aborted immediately after the introduction of a new male, even though this male exhibited no aggression towards her.[46] In contrast, with geladas and hamadryas baboons, in at least two other cases, pregnancy loss following male takeovers was directly associated with heightened aggression and harassment from the incoming male. For example, in yellow baboons, although miscarriages are rare and infanticide is uncommon,[24,77] three abortions were observed within two weeks of the immigration of an extremely aggressive adult male.[78] Similarly, spontaneous abortions following takeovers in Hanuman langurs were accompanied by harassment and aggression directed at pregnant females or their infants.[79] Therefore, we suspect that

there may be two different mechanisms mediating pregnancy termination following male takeovers. One is an adaptive strategy to a highly predictable threat as, for example, in geladas and hamadryas; the other is an outcome of a generalized stress response to a novel threat as, for example, in yellow baboons.

Strategies for lactating females

Lactating females may also use strategies to mitigate the effects of infanticide. Mothers could abandon infants that are vulnerable to infanticide, thus reducing the costs of "wasted" investment and hastening the resumption of fertility. However, abandonment is uncommon and it has only been reported for Hanuman langurs[12] and ursine colobus monkeys.[80] In contrast to complete abandonment, mothers could wean older infants somewhat earlier than usual after a takeover. Early weaning might reduce the infant's risk of being killed if it hastens the mother's return to fertility or prevents males from using nursing behavior as a cue in their decision to attack infants. Accelerated weaning after the arrival of a new male has been documented in several primate species, including wild ursine colobus monkeys,[81] wild white-headed leaf monkeys,[82] and captive vervet monkeys.[83] Premature weaning has also been observed in wild geladas, but it is unclear whether this was associated with an earlier return to fertility.[84] Finally, following the introduction of new males in a captive colony of hamadryas baboons, all six lactating females resumed fertile sexual cycles within two weeks, regardless of the age of their infants, (suggesting accelerated weaning) although only the oldest infant (nearly one year old) survived.[46] Thus, the combined data from wild and captive studies lends support to the hypothesis that accelerated weaning can be a successful strategy, particularly for mothers of older infants.

Alternatives to Reproductive Suppression

Based on the preceding examples, reproductive suppression can entail short-term, costs for females, (i.e., delay in reproduction, that nevertheless reap the long-term benefits of avoiding harassment or infanticide), but are there other options? Certainly, there is ample evidence that many female primates can use behavioral strategies in lieu of reproductive suppression, particularly with respect to infanticide avoidance.[71] Although such "prevention" strategies are beyond the scope of this review, we do focus on one such strategy.

Across many primate species, pregnant and lactating females exhibit nonconceptive sexual behavior or display external signs of fertility such as sexual swellings. These false signals of fertility may manipulate or obscure cues that infanticidal males use to identify their victims. For example, males may refrain from killing infants of females they have mated with in the past. Routine nonconceptive mating is common in many primates.[71] However, "conditional" nonconceptive mating observed after male takeovers has been reported only for Hanuman langurs, sifakas, brown capuchins, and blue monkeys.[71] In addition, among chimpanzees,[85] red colobus monkeys,[86] hamadryas baboons,[87] and geladas[88] such nonconceptive mating is accompanied by "deceptive" sexual swellings. Intriguingly, for hamadryas baboons and geladas, the infants of lactating females that produced postpartum sexual swellings were less likely to be killed by new males than were the infants of other females.[87,88] However, false signals do not seem to provide effective protection to infants in all cases.[71]

A PROXIMATE PERSPECTIVE ON SOCIAL SUPPRESSION; HOW DO FEMALES SUPPRESS REPRODUCTION?

Activation of the Stress Response

Almost 75 years ago, Hans Selye discovered that reproduction in female rats could be inhibited through activation of the (HPA) axis, the neuroendocrine pathway that regulates an organism's short-term response to stress.[89] Although conclusive studies from wild primate populations are rare, experimental work has documented the negative effects of stress on reproductive function in a variety of primate and other mammalian species.[17,90]

All vertebrate reproductive activity is coordinated through the hypothalamic–pituitary–gonadal (HPG) axis (Box 1). Gonadotropin-releasing hormone (GnRH) neurons in the hypothalamus release GnRH into the median eminence, from which it is transported through the hypothalamic-hypophysial portal system to the anterior pituitary. There, GnRH regulates the secretion of the gonadotropins, follicle-stimulating hormone (FSH), and luteinizing hormone (LH), which are released into general circulation, where they act on the ovary to stimulate ovulation.

Although activation of the stress axis could potentially influence reproduction at any level of the HPG axis, the majority of studies suggests that the effects of stress downregulate the release of GnRH from neurons in the hypothalamus.[17] Identifying the precise signals that relay HPA activity to GnRH neurons has been challenging, but recent research points toward corticotropin releasing factor (CRF) as one likely candidate.[91] Other hormones released into circulation during stress, such as glucocorticoids, have been proposed to mediate stress effects to the hypothalamic-pituitary unit.[90] Lending support to this idea, several studies have shown that administration of glucocorticoids can, at least in part, inhibit the secretion of LH from the anterior pituitary.[90] Although the precise signals that relay the effects of stress to the HPG axis are not completely known, they are likely to include multiple neuroendocrine pathways that vary depending on the species, sex, and hormonal or social status of the subject.[17,90]

Many mammalian studies show that early pregnancy can be terminated by stress.[92] Under stressful conditions, the adrenal cortex secretes substantial quantities of androgens, which readily metabolize

Box 1. Reproductive Function and Dysfunction in Female Primates

Reproductive function in female primates. The reproductive cycle of female primates is controlled by the HPG axis. Release of GnRH from the hypothalamus into the median eminence and its transport through the hypothalamic-hypophysial portal system controls the secretion of gonadotropins, (FSH, and LH) from the anterior pituitary. (FSH and LH) travel via systemic circulation to the ovaries, where they stimulate follicle growth (which secretes estrogens) and eventual ovulation. After ovulation, the ruptured follicle tissue becomes the corpus luteum, which secretes progesterone. The HPG-axis is involved in each reproductive stage: puberty followed by regular ovulatory cycles (ovulation) and finally offspring production.

Puberty. The normal process of sexual maturation in most female primates is tightly linked with growth and energy balance. Female puberty occurs shortly after a transition in energy allocation: Energy previously invested in growth is shunted to reproduction. On a physiological level, the HPG-axis inhibits reproduction in pre-pubertal females at both the pituitary and gonadal level, keeping reproductive hormones (that is, estradiol and progesterone) low. As growth slows, increasing energy reserves in the peri-pubertal female cause this inhibition to lift, leading to a gradual rise in hormones. The rise in estradiol, in particular, stimulates development of the reproductive tract and eventually facilitates functional menstrual cycles and sexual behavior.

Ovulation. This is the process whereby a mature egg is released from the female ovary into the oviduct, where it is available to become fertilized. For successful ovulation, GnRH is released by the hypothalamus in brief pulses, stimulating the anterior pituitary to release gonadotropins. Gonadotropins cause one or more ovarian follicles to develop. As the follicle develops, it produces estrogens and progestins. Through a positive feedback mechanism, increasing levels of estrogens stimulate more gonadotropins to be released. Ovulation occurs when a spike in the amount of gondadotropins causes eggs to be released from the developing follicles.

Conception. If females successfully ovulate, the released eggs still need to be fertilized and undergo successful implantation. Thus, conception necessarily involves both a behavioral component (mating with a fertile male) and a physiological one (successful implantation). Implantation is an early stage of pregnancy at which the embryo must successfully adhere to the wall of the uterus. This process is highly dependent on progesterone coming from the corpus luteum.

Gestation. If successful implantation occurs, the embryo proceeds to develop within the protective confines of the uterus. Gestation begins the moment of implantation and ends with parturition, and is the second most energetically expensive reproductive phase for mammalian females. The maintenance of gestation is dependent on threshold levels of progesterone and estrogens. At the beginning of pregnancy, these hormones are secreted by the ovary; however, once pregnancy is established, the fetal placenta takes over this process. Minute changes in the concentrations of either steroid can disrupt gestation and lead to pregnancy failure.

Lactation. Parturition is immediately followed by lactation, during which the neonate is dependent on only maternal milk as a source of nutrition. Lactation is the most energetically expensive reproductive phase for mammalian females. The ability to sustain the costs of lactation and avoid infant mortality is therefore highly dependent on maternal body condition. The HPG axis is quiescent during this period, returning to cyclic activity only when suckling bouts have subsided and maternal energy balance has returned to positive.

Reproductive suppression. This is defined broadly as the inhibition of reproductive physiology and/or reproductive behavior in an otherwise fertile individual in response to specific environmental or physiological conditions. Reproductive suppression generally results from external and internal cues that predict poor reproductive outcomes. Such cues may come in the form of negative energy balance, in which, energy output exceeds energy input; physiological stress, or elevated circulating concentrations of glucocorticoids; or either chemosignals or social signals from conspecifics. These signals are mediated by specialized neuroendocrine mechanisms.
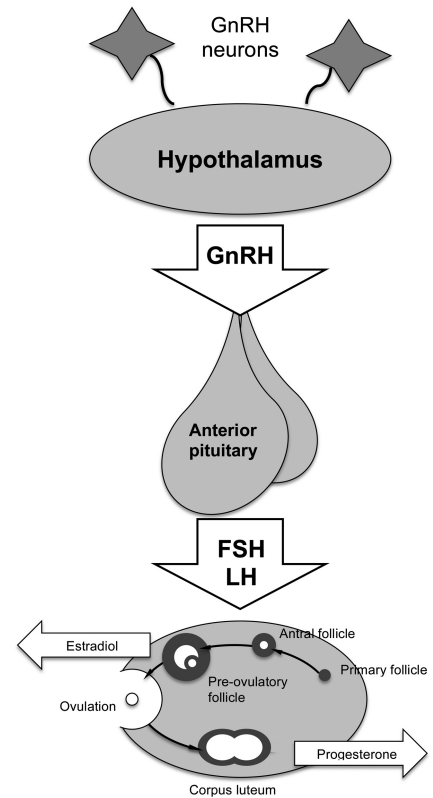
Generally, the common endpoint for all of these mechanisms is disruption of the secretion of GnRH in the hypothalamus, which curtails all downstream products of the HPG axis.

Reproductive suppression has been documented in females across a wide range of primate taxa and may involve inhibition at any reproductive stage. Importantly, the costs and benefits of reproductive suppression change with respect to the stage at which it occurs, with suppression at early stages entailing fewer costs and more benefits, while suppression at late stages generates higher costs and fewer benefits. Reproductive suppression can be divided into two "types" based on the cues thought to mediate reproductive inhibition.

Energetic suppression: The inhibition of reproductive physiology in otherwise fertile individuals in response to inadequate availability of maternal energy.

Social suppression. The inhibition of reproductive physiology or behavior in otherwise fertile individuals directly in response to their interactions with other conspecifics.

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to estrogens. Consequently, in addition to glucocorticoids, acute stress leads to an elevation in estradiol. Although estradiol is an essential hormone for preparing the uterus for pregnancy, even very small elevations above optimal levels can interfere with blastocyst implantation.[93] Elevations in estradiol may also be an important component of reproductive control outside of physiological stress.[93]

Although most of what we know about the effects of stress on reproduction comes from captive studies on rodents and domestic animals, a few primate studies have successfully connected the dots between a stressful social environment, a neuroendocrine stress response, and reproductive suppression in one form or other. For example, high rates of aggression and elevated levels of cortisol in subordinate females were linked to failure to produce the LH surge required for ovulation in captive talapoin monkeys[94] and a longer time to conception in captive geladas.[69] Further, subordinate female cynomolgus monkeys experienced higher rates of aggression, lower rates of grooming, increased cortisol responses to an ACTH infusion, and impaired reproductive function.[95,96] These results suggest that many examples of social suppression in female primates may be mediated by a generalized stress response.

Remarkably, physiological stress does not appear to play a role in callitrichid reproductive suppression.[49] Indeed, in cooperatively breeding societies, subordinate individuals tend to be characterized by glucocorticoid levels that are equal to or lower than those of dominants.[97] As a result, it is now accepted that the extreme reproductive suppression in cooperatively breeding species is probably not mediated by social stress.

Chemosensory or Other Signals

Chemical signals (or chemosignals) also alter the reproductive physiology and behavior of conspecifics. Although this term is often used interchangeably with "pheromone," it is rare among mammals for a chemosignal to meet the strict criteria for a pheromone: (which is a unitary, species-typical substance that is both necessary and sufficient for an experience-independent behavioral or physiological response).[98] In contrast, mammalian chemosignals contain multiple compounds that interact to produce a "chemical image" that generally contains priming or releasing agents (serving essentially as pheromones)[99] as well as a chemical signature used to recognize an animal as an individual or as a member of a particular social group.[100] Therefore, in mammals, social information has the potential to dramatically alter the effects of particular chemosignals.

Chemosignals in nonprimates

In mammals, chemosignals have the potential to prime reproductive physiology so that individuals become sexually mature and active at times when reproduction is most probable and suppress it when it is not. For example, chemosignals within the urine of male rodents can accelerate puberty in juvenile females (the Vandenbergh effect)[101] and stimulate ovulation in anovulatory adult females (the Whitten effect).[102] This pro-ovulatory "male effect" has also been found in anestrous domestic sheep and goats.[103] The same chemosignals that stimulate reproduction in cycling females may also disrupt gestation in pregnant females (the Bruce effect),[73] suggesting that each of these effects may be controlled by the same mechanism.[104] Remarkably, after females learn the olfactory signature of a male, (that is, they recognize his odor) his urine no longer induces such effects.

Currently, two hypotheses exist for the "stimulating agent" within male urine.[98] The longstanding hypothesis is that a combination of volatile compounds in male urine activates a neuroendocrine pathway in the vomeronasal olfactory system (VNS) of females and inhibits the prolactin release necessary for maintaining pregnancy.[99] The alternative hypothesis does not require mediation by neural events but is instead directly mediated by estradiol in an unknown male urine. Exogenous estradiol is directly absorbed by females and binds to receptors along the reproductive tract, inducing puberty or disrupting pregnancy.[93] Both hypotheses support the idea that the signal is androgen-dependent and must be bound to nonvolatile

proteins to facilitate odor individuality.[98] The VNS detects and translates chemical signals in urine that individually identify males.[105] Importantly, in most rodents, these urinary constituents are both necessary and sufficient for inducing these effects.[106]

Urine of group-housed female mice can delay puberty in juvenile females (another form of the Vandenbergh effect)[107] and prolong or completely suppress ovarian cyclicity in adult females (the Lee-Boot effect).[108] In both cases, the "delaying agent" in female urine is adrenal-dependent. Females suppress ovulation when signals from conspecifics indicate a high population density, suggesting increased competition for resources.[98]

Chemosignals in primates

In primates, chemosignals are used in a variety of contexts.[109] However, most of what we know about the role of chemosignals in reproductive suppression derives from research on captive callitrichids. It has become clear that chemosignals in the scent secretions of a dominant female play a key role in both *initiating* and *maintaining* reproductive inhibition in subordinate marmosets and tamarins.[41,110,111] (Additionally at least one study on cotton-top tamarins suggests that related males can have the same suppressive effect.[112]) For example, common marmoset females rendered anosmic (unable to detect chemosignals) were able to avoid ovulatory suppression after exposure to a dominant female.[113] Furthermore, anovulatory marmoset and tamarin females kept only within scent contact of the dominant female exhibited a significant delay in the onset of ovulation.[41,110,111]

However, despite a delay in ovulation, the effectiveness of the odor cues alone expired after a few weeks,[114] indicating that chemosignals from the dominant female are *not sufficient* for maintaining ovulatory suppression. Supporting this argument, odor from an unfamiliar dominant female was completely ineffective at maintaining ovulatory suppression, suggesting that chemical signals from dominants are effective only if they are paired with recognition of the signaler.[115] In this regard, reproductive control by dominants may be mediated simply through the learned association by subordinates between the dominant female's cues and the behavioral subordination she imposes.[116]

In addition, scent secretions from dominant females are *not necessary* for maintaining ovulatory suppression in callitrichids. In another version of the experiment above, subordinate marmoset females rendered anosmic remained anovulatory as long as they were housed with a known dominant female.[114] Furthermore, even after physical separation from the dominant female, her visual cues alone were enough to maintain anovulation in anosmic subordinates.[114] Yet neither visual nor chemical signals were able to maintain suppression indefinitely, indicating that social cues from the dominant female may be the predominant stimulus controlling ovulatory suppression.[49]

The role that novel males (or their chemosignals) might have in stimulating ovulation in callitrichid females is less straightforward. First, whether males themselves are even necessary for stimulating ovulation is variable across species, with only cotton-top tamarins requiring both the removal of the dominant female and the introduction of a novel male.[40,41] Second, in cotton-top tamarins, males were required for initiating ovulation in previously acyclic females, but were not required for maintaining.[117] Finally, male chemosignals alone were not sufficient for stimulating ovulation in anovulatory female tamarins,[112] although combining these chemosignals with visual and auditory cues did stimulate cyclicity.[117] To our knowledge, no study has examined whether male chemosignals are necessary for ovulatory stimulation.

We know even less about the specific neuroendocrine mechanisms involved. Anovulatory, mature, female callitrichids dwelling in their natal groups exhibit acyclic levels of gonadotropins and ovarian hormones.[49,111,118] Further, at the first signs of subordination, previously cycling females exhibit a sudden drop in gonadotropins, followed shortly afterward by complete ovulatory suppression.[45,119] Although the precise mechanism for this is unknown, it has been suggested, in common marmosets, that social subordination causes a dissociation between GnRH and pituitary secretion of gonadotropins, possibly via reduced pituitary sensitivity to GnRH.[116]

In catarrhine primates, we understand almost nothing of the proximate mechanisms stimulating changes in female reproductive condition in response to social cues. In addition to a Bruce effect and a possible Vandenbergh effect in female geladas, we also see signs of false fertility in lactating females after new males enter the group. (Intriguingly, all three have also been reported for hamadryas baboons.)[46,87] None of these is consistent with a stress-mediated mechanism. In both gelada and hamadryas studies, new males did not appear to harass, intimidate, or injure females on takeover,[76] and the speed with which pregnant females terminated their pregnancies does not suggest the outcome of stress-induced inhibition of the HPG axis. Instead, it resembles the chemosensory-mediated Bruce and Vandenbergh effects in rodents. However, the VNS in catarrhine primates has become vestigial, indicating a diminished role for olfaction and chemosignals.[99] Without a functional VNS, catarrhines presumably lack the ability to detect non-volatile chemosensory compounds. So how are these effects mediated?

At present, several possibilities may explain the reproductive changes observed in female geladas and hamadryas baboons following takeovers. First, although unlikely, male chemosignals could be transmitted to females via the main olfactory system, dermal contact, or semen. Second, as in callitrichids, the trigger for reproductive suppression could come from visual, tactile, or other social cues. These cues alone or in combination with

chemosignals could stimulate neuro-endocrine activity within the female, causing prolactin inhibition and/or a rise in estradiol, both of which can significantly effect a developing fetus. Regardless of the mechanism, it is possible that the same neuroendocrine pathway controls all three different reproductive responses to novel males. Understanding the details of this pathway is therefore crucial for determining how such responses have been shaped by evolution.

CONCLUSIONS

Throughout this review, we have taken the stance that reproductive suppression is an evolved strategy mediated by females. Females translate predictable and reliable signals into a "decision" to continue or abandon reproduction at a mechanistic level. But to what extent is this assumption justified? In other words, could reproductive suppression simply be the outcome of physiological constraints in the same way that genetic abnormalities may cause a pregnancy to fail?

Reproduction can fail in one of two ways: *indirectly*, as the outcome of an evolved mechanism that responds appropriately to a proximate cue indicating suboptimal conditions, (as has been suggested for social suppression), or *directly*, as the outcome of a mechanism that responds to the suboptimal conditions themselves, (as has been suggested for energetic suppression). For example, high rates of aggression inflicted by one unusually aggressive yellow baboon male in Amboseli[78] almost certainly caused the subsequent abortions observed in several females, perhaps as a result of hemorrhage or stress. One could argue that an acute stress response by these females acts the same way that injury, disease, or pathology does to cause reproductive failure (a non-adaptive response). Alternatively, one could argue that females have evolved to suppress reproduction in response to high levels of generalized stress, regardless of the nature of this stress. Both of these arguments stand in contrast to the

data emerging from geladas, where systematic abortions following takeovers appear to be the rule rather than the exception. So how do we distinguish adaptive reproductive suppression from more general maladaptive reproductive failure?

We suggest three ways to resolving this. First, we place reproductive failures along a continuum that ranges from adaptive strategies at one end (for example, a Bruce effect in geladas) to non-adaptive outcomes at the other (for example a, infant death by disease). The adaptive end requires reliable and predictable cues; the non-adaptive end is imposed by purely stochastic events. Stress-mediated reproductive failure falls in the middle of this continuum, where the generalized stress response is adaptive, but the events triggering the response are stochastic.

Second, we emphasize the importance of identifying the proximate cues associated with the immanent failure. If the proximate cue, (such as the arrival of a new male) is not also the mechanism for reproductive failure, this is more likely to be evolved reproductive strategy than if the cue is intricately tied to the mechanism itself (such as negative energy balance then this more likely).

Third, we encourage research that merges mechanism and function to test adaptive hypotheses. For example, we know a great deal about the mechanisms mediating reproductive suppression in callitrichids, but we know much less about their adaptive significance. Why do most subordinate callitrichid females completely suppress their own reproduction? What is the likelihood that they will eventually become the dominant breeding female? Or is their advantage solely one of kin selection? In contrast, in catarrhines we are beginning to understand of why some forms of reproductive suppression are adaptive, but we have little to no data on the mechanisms mediating these strategies. It is paramount that primate biologists integrate both functional and mechanistic approaches for us to fully understand the why and how behind reproductive suppression in female primates.

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